THE AUTHORITATIVE EVIDENCE-BASED GUIDE FOR ANAESTHETISTS

OXFORD HANDBOOK OF ANAESTHESIA

EDITED BY Rachel Freedman | Lara Herbert
Aidan O'Donnell | Nicola Ross

Quick reference guide to providing anaesthesia for surgical procedures

Emergency advice on anaesthetic management and conduct for emergency surgeries, procedures, and the complex trauma patient

Incorporates revised and new material including the health and well-being of anaesthetists, patient safety, and perioperative medicine

FIFTH EDITION 5 FIFTH EDITION

OXFORD HANDBOOK OF

Anaesthesia

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Preface

Welcome to the fifth edition of the *Oxford Handbook of Anaesthesia*. This edition sees a new editorial team. Rachel Freedman, Lara Herbert, Aidan O'Donnell, and Nicki Ross have taken over from Keith Allman and Iain Wilson, who have continued to provide advice and encouragement during the development of this edition. Keith and Iain started this book from scratch and have been at the wheel for almost 20 years. Now they have handed us the keys and let us drive—we promise to try not to crash into anything.

We must begin by acknowledging the thousands of hours of work contributed by the authors that have been involved in the previous editions. The handbook is a success because of the work that has gone before.

Our aim with the fifth edition is to make this the most useful anaesthetic handbook in the world. The layout of the book has been changed to bring disparate topics together, and to make the flow of the book more aligned with the sequence of a patient's perioperative journey. We have maintained an emphasis on patient safety, practicality, and good practice throughout. Familiar sections have been completely revised or expanded, including Preoperative considerations, Regional anaesthesia, and Major trauma. New sections have been added, including sensible advice about looking after yourself and what to do when things go wrong. We have welcomed a large number of new contributors with a wider international spread to enrich the content. We have tried to do all this while keeping the size of the book manageable.

The *Oxford Handbook of Anaesthesia* remains a comprehensive and practical guide to anaesthesia. It has proven popular in many countries throughout the world. It has been translated into Polish and Romanian. A low-cost edition is available in many countries across Asia (Afghanistan, Bangladesh, Bhutan, Cambodia, China, India, Indonesia, Laos, Malaysia, Myanmar, Nepal, Pakistan, Philippines, Syria, Taiwan, Thailand, Turkey, Vietnam, and Yemen) and Africa (Libya, Egypt).

We hope that you will enjoy this latest edition of the handbook and find the changes helpful. We welcome your feedback, so that we can keep improving the book. Despite all our efforts, it is possible that an occasional error exists; please be careful.

We would like to thank our partners for their long-suffering support. Our own families have grown larger, and we welcome Andrew and Joshua, who were born while this book was gestating. We thank our many contributors for their expertise and hard work; and Keith and Iain, without whom the book would not exist. Unlike Keith and Iain, our editorial team is located in four different cities in two different countries, so we have not had the luxury of meeting over a beer. We have instead sat through many hours of discussion in a virtual public house we fondly refer to as *The Skype Arms*. If Iain were to buy us one drink for each time he told us this book was nearly finished, we would have a merry night indeed.

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We hope this edition will augment the already fantastic work anaesthetists are doing all around the world to keep their patients safe. In the words of Lewis Carrol: 'One of the deep secrets of life is that all that is really worth the doing is what we do for others'.

Rachel, Lara, Aidan and Nicki
(www.oup.com/uk/medicine/handbooks)
February 2021

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Abbreviations

2D	two-dimensional
3D	three-dimensional
5-HT	serotonin (5-hydroxytryptamine)
AAA	abdominal aortic aneurysm
AADR	anaesthetic adverse drug reaction
AAGA	accidental awareness during general anaesthesia
ABC	airway, breathing, circulation
ABG	arterial blood gas
AC	alternating current
ACC	American College of Cardiology
ACE	angiotensin-converting enzyme
ACF	antecubital fossa
ACT	activated clotting time
ACTH	adrenocorticotrophic hormone
ADH	antidiuretic hormone
ADHD	attention-deficit hyperactivity disorder
ADP	adenosine diphosphate
AEC	airway exchange catheter
AED	automated external defibrillator
AF	atrial fibrillation
AFE	amniotic fluid embolism
AFOI	awake fibreoptic intubation
AHI	apnoea–hypopnoea index
AIC	Aintree intubation catheter
AIDS	acquired immune deficiency syndrome
AKI	acute kidney injury
ALF	acute liver failure
ALI	acute lung injury
ALP	alkaline phosphatase
ALS	advanced life support
ALT	alanine aminotransferase
a.m.	ante meridiem (before noon)
AMI	acute myocardial infarction
AoA	Association of Anaesthetists
AP	anteroposterior
APAGBI	Association of Paediatric Anaesthetists of Great Britain and Ireland
APL	adjustable pressure-limiting
APTT	activated partial thromboplastin time
AR	aortic regurgitation
ARB	angiotensin receptor blocker
ARDS	acute respiratory distress syndrome
AS	aortic stenosis
ASA	American Society of Anesthesiologists
ASD	atrial septal defect
ASIS	anterior superior iliac spine
AST	aspartate transaminase
AT	anaerobic threshold
ATLS®	Advanced Trauma Life Support®
AV	atrioventricular
A–V	arteriovenous
AVM	arteriovenous malformation
AVR	aortic valve replacement
AVSD	atrioventricular septal defect
BCIS	bone cement implantation syndrome
bd	*bis diem* (twice daily)
BiPAP	bilevel positive airway pressure
BIS	bispectral index
BLS	basic life support
BMI	body mass index
BMS	bare-metal stent
BNF	British National Formulary
BNFc	British National Formulary for Children
BNP	brain natriuretic peptide
BP	blood pressure
bpm	beats per minute
BURP	backward, upward, and rightward pressure
Ca^{2+}	calcium ion
CABG	coronary artery bypass grafting
CAD	coronary artery disease
CBF	cerebral blood flow
CC	creatinine clearance
CCF	congestive cardiac failure
CEA	carotid endarterectomy
CF	cystic fibrosis

CFTR	cystic fibrosis transmembrane regulator
Ch	Charrière (French) gauge (also FG or Fr)
CHD	congenital heart disease
CI	confidence interval
CICO	can't intubate/can't oxygenate
CK	creatine kinase
CKD	chronic kidney disease
CLD	chronic liver disease
cm	centimetre
cmH_2O	centimetre of water
CMV	cytomegalovirus
CNB	central neuraxial block
CNS	central nervous system
CO	carbon monoxide; cardiac output
CO_2	carbon dioxide
COHb	carboxyhaemoglobin
COPD	chronic obstructive pulmonary disease
COX	cyclo-oxygenase
CPAP	continuous positive airway pressure
CPB	cardiopulmonary bypass
CPET	cardiopulmonary exercise testing
CPP	cerebral perfusion pressure
CPR	cardiopulmonary resuscitation
CPSP	chronic postsurgical pain
CRA	continuous regional analgesia
CREST	calcinosis, Raynaud's, oesophageal dysfunction, sclerodactyly and telangiectasia
CRP	C-reactive protein
CRPS	complex regional pain syndrome
CRT	cardiac resynchronisation therapy
CS	Caesarean section
CSE	combined spinal/epidural
CSF	cerebrospinal fluid
CSHT	context-sensitive half-time
C-spine	cervical spine
CT	computed tomography
cTnT	high-sensitivity troponin T
CTZ	chemoreceptor trigger zone
CVC	central venous catheter
CVE	cerebrovascular event
CVP	central venous pressure
CVS	cardiovascular system
CXR	chest X-ray
d	day
Da	dalton
DAPT	dual antiplatelet therapy
DAS	Difficult Airway Society
dB	decibel
DBD	donation after brain death
DBS	double-burst stimulation
DCD	donation after circulatory death
DCR	damage control resuscitation
DCS	damage control surgery
DES	drug-eluting stent
DHCA	deep hypothermic circulatory arrest
DHS	dynamic hip screw
DIC	disseminated intravascular coagulation
dL	decilitre
DLCO	diffusing capacity of the lungs for carbon monoxide
DLT	double-lumen (endobronchial) tube
DM	diabetes mellitus
DMARD	disease-modifying antirheumatic drug
DNAR	Do Not Attempt Resuscitation
DVT	deep vein thrombosis
EBM	evidence-based medicine
EBV	estimated blood volume
ECF	extracellular fluid
ECG	electrocardiogram
ECMO	extracorporeal membrane oxygenation
ECT	electroconvulsive therapy
ED	emergency department
EEG	electroencephalogram
EF	ejection fraction
eFAST	extended focused assessment with sonography for trauma
eFONA	emergency front of neck access
eGFR	estimated glomerular filtration rate
EMA	European Medicines Agency
EMG	electromyogram
EMLA®	Eutectic Mixture of Local Anaesthetics®
ENT	ear, nose, and throat

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EO	external oblique
EPO	erythropoietin
ERAS	enhanced recovery after surgery
ERCP	endoscopic retrograde cholangiopancreatography
ERPC	evacuation of retained products of conception
ESP	erector spinae plane
ESR	erythrocyte sedimentation rate
ETAG	end-tidal anaesthetic gas (monitoring)
$ETCO_2$	end-tidal carbon dioxide
ETO_2	end-tidal oxygen
ETT	endotracheal tube
EUA	examination under anaesthesia
EuroSCORE	European System for Cardiac Operative Risk Evaluation
EVAR	endovascular aneurysm repair
EVD	external ventricular drain
FAST	focused assessment sonogram in trauma
FBC	full blood count
FES	fat embolism syndrome
FEV_1	forced expiratory volume in 1s
FFP	fresh frozen plasma
FGF	fresh gas flow
FiO_2	fractional inspired oxygen content
FOB	flexible optical bronchoscope
Fr	French gauge (also FG or Ch)
FRC	functional residual capacity
FTc	corrected flow time
FTSG	full-thickness skin graft
FVC	forced vital capacity
g	gram
G	Gauge (standard wire gauge)
G&S	group and screen
G6PD	glucose-6-phosphate dehydrogenase
GA	general anaesthetic/ anaesthesia
GCS	Glasgow Coma Scale
GDFT	goal-directed fluid therapy
GFR	glomerular filtration rate
GI	gastrointestinal
GP	general practitioner
GTN	glyceryl trinitrate
GUCH	grown-up (patient with) congenital heart (malformation)
h	hour
H^+	hydrogen ion
HAS	human albumin solution
Hb	haemoglobin
HbA1c	glycosylated haemoglobin
HCM	hypertrophic cardiomyopathy
HCO_3^-	bicarbonate ion
Hct	haematocrit
HDL	high-density lipoprotein
HDU	high dependency unit
HELLP	haemolysis, elevated liver enzymes, low platelets
HFmrEF	heart failure with mid-range ejection fraction
HFNO	high-flow nasal oxygen
HFO	high-frequency oscillation
HFpEF	heart failure with preserved ejection fraction
HFrEF	heart failure with reduced ejection fraction
Hib	*Haemophilus influenzae* type b
HIT	heparin-induced thrombocytopenia
HIV	human immunodeficiency virus
HLA	human leucocyte antigen
HME	heat and moisture exchanger
HPA	hypothalamic–pituitary–adrenal
HR	heart rate
HRT	hormone replacement therapy
HTLV	human T cell lymphotropic virus
HUS	haemolytic uraemic syndrome
Hz	hertz
I:E ratio	inspired:expired ratio
IABP	intra-aortic balloon pump
IBW	ideal body weight
ICD	implantable cardioverter–defibrillator
ICP	intracranial pressure
ICU	intensive care unit
ID	internal diameter
IE	infective endocarditis
IGF	insulin-like growth factor
IHD	ischaemic heart disease
IL	interleukin
ILD	interstitial lung disease
iLMA	intubating laryngeal mask airway
IM	intramuscular(ly)
in	inch
INR	international normalised ratio
IO	internal oblique/intraosseous

IOP	intraocular pressure
IP	in-plane
iPACK	infiltration between the popliteal artery and capsule of the knee
IPP	interpectoral plane
IPPV	intermittent positive pressure ventilation
ISO	International Organization for Standardization
ITP	idiopathic thrombocytopenic purpura
IU	international unit
IV	intravenous(ly)
IVC	inferior vena cava
IVI	intravenous infusion
IVRA	intravenous regional anaesthesia
J	joule
JVP	jugular venous pressure
K^+	potassium ion
kcal	kilocalorie
KCl	potassium chloride
kg	kilogram
km	kilometre
kPa	kilopascal
L	litre
LA	local anaesthetic/anaesthesia
LAST	local anaesthetic systemic toxicity
LAVH	laparoscopically assisted vaginal hysterectomy
LBW	low birthweight
LCNT	lateral cutaneous nerve of thigh
LDH	lactate dehydrogenase
LFT	liver function test
LIA	local infiltration analgesia
LMA	laryngeal mask airway
LMWH	low-molecular weight heparin
LRTI	lower respiratory tract infection
LSCS	lower-segment Caesarean section
LV	left ventricle/ventricular
LVEDP	left ventricular end-diastolic pressure
LVEDV	left ventricular end-diastolic volume
LVEF	left ventricular ejection fraction
LVH	left ventricular hypertrophy
m	metre
M	molar
mA	milliampere
MABL	maximal allowable blood loss
MAC	minimum alveolar concentration
MACE	major adverse cardiac events
MAO	monoamine oxidase
MAOI	monoamine oxidase inhibitor
MAP	mean arterial pressure
MCV	mean corpuscular volume
MELD	model for end-stage liver disease
MEN	multiple endocrine neoplasia
MEOWS	Modified Early Obstetric Warning Scoring
MET	metabolic equivalent of task
mg	milligram
Mg^{2+}	magnesium ion
MH	malignant hyperthermia
MHRA	Medicines and Healthcare Products Regulatory Agency
MHz	megahertz
MI	myocardial infarction
MILS	manual in-line stabilisation
min	minute
MINS	myocardial injury after non-cardiac surgery
mL	millilitre
mm	millimetre
mmHg	millimetre of mercury
mmol	millimole
mo	month
mol	mole
mOsmol	milliosmole
MPFL	medial patellofemoral ligament
mph	miles per hour
MR	mitral regurgitation
MRI	magnetic resonance imaging
MRSA	meticillin-resistant *Staphylococcus aureus*
ms	millisecond
MSU	midstream urine
mT	millitesla
MTC	major trauma centre
MTP	massive transfusion protocol
MUA	manipulation under anaesthesia
MW	molecular weight
6MWT	6-minute walk test
N	newton
Na^+	sodium ion

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NaCl	sodium chloride
NAP	National Audit Project
NAP4	National Audit Project 4
NCA	nurse-controlled analgesia
NDD	neurological determination of death
NDMR	non-depolarising muscle relaxant
NELA	National Emergency Laparotomy Audit
NG	nasogastric
NGT	nasogastric tube
NHS	National Health Service
NIBP	non-invasive blood pressure
NICE	National Institute for Health and Care Excellence
NJR	National Joint Registry
NK	natural killer (cells)
nm	nanometre
NMB	neuromuscular blockade
NMBA	neuromuscular-blocking agent
NMDA	*N*-methyl-*D*-aspartate
nmol	nanomole
NNT	number needed to treat
NO	nitric oxide
N_2O	nitrous oxide
NSAID	non-steroidal anti-inflammatory drug
NYHA	New York Heart Association
O&D	oesophagoscopy and dilatation
O_2	oxygen
OELM	optimal external laryngeal manipulation
OGT	orogastric tube
OHS	obesity hypoventilation syndrome
OIH	opioid-induced hyperalgesia
OLV	one-lung ventilation
OOP	out-of-plane
OPA	oropharyngeal airway
OPCABG	off-pump coronary artery bypass grafting
OR	odds ratio
ORIF	open reduction and internal fixation
OSA	obstructive sleep apnoea
OS-MRS	Obesity Surgery Mortality Risk Score
Pa	pascal
PA	posterior to anterior; pulmonary artery
PABA	para-aminobenzoic acid
$PaCO_2$	arterial partial pressure of carbon dioxide
PAFC	pulmonary artery flotation catheter
PaO_2	arterial partial pressure of oxygen
PAO_2	alveolar partial pressure of oxygen
PAOP	pulmonary artery occlusion pressure
PAP	pulmonary artery pressure
P_{aw}	airway pressure
PCA	patient-controlled analgesia
PCC	prothrombin complex concentrate
PCEA	patient-controlled epidural analgesia
PCI	percutaneous coronary intervention
PCV	packed cell volume
PD	Parkinson's disease
PDA	persistent ductus arteriosus
PDE	phosphodiesterase
PDGF	platelet-derived growth factor
PDPH	postdural puncture headache
PE	pulmonary embolism
PEA	pulseless electrical activity
PEEP	positive end-expiratory pressure
PEFR	peak expiratory flow rate
PENG	pericapsular nerve group
PFT	pulmonary function test
pg	picogram
PICC	peripherally inserted central catheter
p.m.	*post meridiem* (after noon)
pmol	picomole
PNB	peripheral nerve block
PNS	peripheral nerve stimulator
PO	per os (oral/orally)
POCD	postoperative cognitive dysfunction
POCT	point-of-care testing
POD	post operative delirium
POND	post operative neurocognitive disorder
PONV	post operative nausea and vomiting
PORC	postoperative residual curarisation

PPI	proton pump inhibitor
ppm	parts per million
PPV	pulse pressure variation
PR	per rectum
PRBC	packed red blood cell
PRN	*pro re nata* (as required)
PSG	polysomnography
PSIS	posterior superior iliac spine
PSP	pectoserratus plane
PT	prothrombin time
PTH	parathyroid hormone
PV	polycythaemia vera
PVR	pulmonary vascular resistance
qds	*quater die sumendus* (four times daily)
RA	rheumatoid arthritis
RAE	Ring–Adair–Elwyn (tube)
RAST	radioallergosorbent test
RBC	red blood cells
RCoA	Royal College of Anaesthetists
RCT	randomised controlled trial
REM	rapid eye movement
RFA	radiofrequency ablation
rFVIIa	recombinant factor VIIa
RIMA	reversible inhibitor of monoamine oxidase A
ROCSM	restriction of cervical spine motion
ROSC	restoration of spontaneous circulation
ROTEM®	rotational thromboelastometry
RR	relative risk; respiratory rate
RSC	rectus sheath catheter
RSI	rapid sequence induction
rt-PA	recombinant tissue plasminogen activator
RUQ	right upper quadrant
RV	right ventricle/ventricular
RWMA	regional wall motion abnormality
s	second
SAD	substance abuse disorder; supraglottic airway device
SAH	subarachnoid haemorrhage
SaO_2	arterial oxygen saturation
SC	subcutaneous(ly)
SCD	sickle-cell disease
SCIWORA	spinal cord injury without radiographic abnormality
SCM	sternocleidomastoid muscle
$ScvO_2$	central venous oxygen saturation
SGA	supraglottic airway; small for gestational age
SIADH	syndrome of inappropriate antidiuretic hormone (secretion)
SIDS	sudden infant death syndrome
SIRS	systemic inflammatory response syndrome
SLE	systemic lupus erythematosus
SLT	single-lumen tube
SNARI	serotonin noradrenergic reuptake inhibitor
SOBA	Society for Obesity and Bariatric Anaesthesia
SORT	Surgical Outcome Risk Tool
SpO_2	peripheral oxygen saturation
SR	sinus rhythm
SSG	split skin graft
SSRI	selective serotonin reuptake inhibitor
STOP	suction termination of pregnancy
SV	spontaneous ventilation
SVC	superior vena cava
SVR	systemic vascular resistance
SVT	supraventricular tachycardia
SVV	stroke volume variation
T	tesla
T_3	liothyronine (tri-iodothyronine)
T_4	levothyroxine
TA	transversus abdominis
TA-GvHD	transfusion-associated graft-versus-host disease
TAP	transversus abdominis plane
TAPSE	tricuspid annular plane systolic excursion
TAVI	transcatheter aortic valve implantation
TB	tuberculosis
TBSA	total body surface area
TBW	total body water
TCA	tricyclic antidepressant
TCI	target-controlled infusion
tds	*ter die sumendus* (three times daily)
TEG®	thromboelastography
TENS	transcutaneous electrical nerve stimulation

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TIA	transient ischaemic attack
TIC	trauma-induced coagulopathy
TIPSS	transjugular intrahepatic portosystemic shunt
TIVA	total intravenous anaesthesia
TLCO	transfer factor of the lungs for carbon monoxide
TMJ	temporomandibular joint
TNF	tumour necrosis factor
TOE	transoesophageal echocardiography
ToF	tetralogy of Fallot
TOF	train-of-four
TOFR	train-of-four ratio
TRALI	transfusion-related acute lung injury
TTL	trauma team leader
TTP	thrombotic thrombocytopenic purpura
TURP	transurethral resection of the prostate
U&E	urea and electrolytes
UFH	unfractionated heparin
UK	United Kingdom
UPPP	uvulopalatopharyngoplasty
URTI	upper respiratory tract infection
US	United States
USS	ultrasound scanning
V/Q	ventilation/perfusion
V	volt
VAE	venous air embolism
VATS	video-assisted thoracoscopic surgery
VC	vital capacity
vCJD	variant Creutzfeldt–Jakob disease
VEGF	vascular endothelial growth factor
VF	ventricular fibrillation
VIP	vasoactive intestinal peptide
VL	videolaryngoscope
VO_2	oxygen consumption
VP	venous pressure
VRIII	variable-rate intravenous insulin infusion
vs	versus
VSD	ventricular septal defect
V_T	tidal volume
VT	ventricular tachycardia
VTE	venous thromboembolism
VTOP	vacuum termination of pregnancy
vWF	von Willebrand factor
w	week
W	watt
WCC	white cell count
WHO	World Health Organization
WPW	Wolff–Parkinson–White
X-match	cross-match
y	year

Chapter 1

Good practice and safety

Rachel Freedman, Lara Herbert, Aidan O'Donnell, Nicola Ross and Mincho Marroquin-Harris

Responsibilities of the anaesthetist *2*
Looking after the patient *3*
Looking after yourself *15*

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Responsibilities of the anaesthetist

The first general anaesthetic was administered in 1846. Today, around 313 million anaesthetics are given worldwide every year, with over 3 million in the United Kingdom (UK) alone. Anaesthetists use highly specialised skills to provide anaesthesia, analgesia and life-sustaining care for patients undergoing operations and procedures. They also lead the way in safety, crisis response and education.

Other roles for anaesthetists include the following:

- Preoperative assessment and optimisation of patients
- Intensive care medicine
- Pain medicine—both acute and chronic
- Obstetric anaesthesia and pain relief in labour
- Resuscitation and stabilisation of patients in the emergency department (ED) and on the ward
- Transport of acutely ill and injured patients
- The provision of anaesthesia or sedation to facilitate a wide range of medical, dental and radiological interventions and investigations
- Prehospital emergency care
- Managerial and organisational roles both inside and outside the theatre environment
- Education, training and simulation.

Anaesthetists form the largest specialty group of doctors in the National Health Service (NHS). They work closely with all other hospital specialties in locations throughout the hospital. They deliver individualised care, usually on a one-to-one basis and at times when patients are at their most vulnerable. Anaesthetists are therefore well positioned to be patient safety advocates at local, national and international levels. Indeed, underpinning all the other roles and responsibilities of the anaesthetist is the ingrained commitment to keep patients safe.

Looking after the patient

Perioperative safety

Anaesthetists are recognised worldwide as leaders in patient safety. Although modern anaesthesia is very safe, new challenges that impact on patient safety continue to arise, such as an ageing population, the prevalence of medication error, and burnout among anaesthetists. Improvements in perioperative safety have been greatest in high-income countries, and although outcomes have improved overall worldwide, there is an urgent need to address the global inequalities that still exist in perioperative safety. From a global surgical perspective, it is estimated that 313 million surgical procedures are performed each year. From elective procedures alone, around 17% of patients suffer harm and 0.5% die.[1] Much of this harm may be preventable.

Why do complications occur?

Accidents are inevitable in systems such as health care because of situational complexity and the latent factors that set up humans to fail.[2] It is therefore unsurprising that errors occur in the dynamic, and sometimes stressful, operating theatre environment. Deficiencies in human factors, such as communication, leadership, teamworking, decision-making and situational awareness, have been shown to contribute substantially to patient harm.

Safety culture

- The new approach to patient safety seems to be much less about individual projects, and more about changing the culture and creating systems that enable reliable delivery of care.
- Even with the most flawless systems in place, however, human factors can still lead to error.
- A safety culture starts with leaders taking ownership of safety and exemplifying it in both actions and words on a daily basis.
- Leaders must steer the difficult balance between holding people accountable and nurturing a supportive environment where individuals and teams both have opportunities to learn from their mistakes and feel empowered to voice their concerns.
- Communication is key. This may be difficult when teams change continually. It is well recognised that steep hierarchies between professions may impede effective communication. In poorly functioning teams, members of staff may not feel able to raise concerns, even when patient safety is compromised.

Strategies to improve patient safety

Below are a number of strategies that contribute towards improving patient safety. Many of these approaches are based on the understanding that humans make mistakes. They encourage both learning from errors and the creation of robust systems to prevent them:

- Reporting an error is fundamental to error prevention. Practitioners need to feel safe to report their mistakes, knowing that the aim is to improve safety, rather than to blame individuals.
- Patient safety groups champion and promote patient safety and learning, such as the Safe Anaesthesia Liaison Group (SALG).

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- Technology and engineering have facilitated big gains in patient safety, e.g. pin-indexing of cylinders, mandatory SpO_2 and capnography, advanced intubating devices and ultrasound for vessel and nerve visualisation.
- Use of guidelines, protocols and good practice standards, e.g. National Institute for Health and Care Excellence (NICE), Royal College of Anaesthetists (RCoA) and Association of Anaesthetists (AoA) guidance. In the UK, the RCoA is leading the way with programmes for excellence, such as Anaesthesia Clinical Services Accreditation (ACSA), where participating anaesthesia departments measure their performance against a robust set of standards.
- Outcome measures may be immediate (complication rate), medium term (length of stay, 30d mortality) or long term (quality of life improvements and functional improvement over 1y, National Emergency Laparotomy Audit (NELA)[3]). We improve what we measure and what we measure must be important to the patient.
- Quality improvement, audit and research: a growing trend is where data from small quality improvement projects conducted by local hospital teams are pooled at a national level, thereby giving the data greater validity. The National Audit Projects (NAPs) and the Sprint National Anaesthesia Projects (SNAPs) have generated information on large numbers of patients, giving evidence-based insight into deficits in care and incidence of problems.[4,5] The Healthcare Quality Improvement Partnership is a huge resource of strategies for examining and instituting best practice.
- Morbidity and mortality reviews are important, both locally and nationally (e.g. National Confidential Enquiry into Perioperative Deaths and the maternal morbidity and mortality reviews). They examine themes that lead to harm and aid delivery of better care through education, training and constant review.[6,7]
- There is increasing evidence that simulation-based team training to improve technical and non-technical skills improves both team performance and patient outcomes.[8]
- Coordinated, safe care requires high-quality handovers. There is growing evidence that these can make a significant difference, not only to patient morbidity, but also to mortality.
- Checklists have been shown to decrease human error, improve patient safety and teamwork and increase the quality of care, e.g. the World Health Organization (WHO) checklist and checklists for handovers, procedures and emergency situations.[9]
- Patient education (e.g. joint schools) and assessment (e.g. preassessment clinics) allow amelioration of risk through optimisation and planning (for detail, see ➔ pp. 49–52).

The WHO checklist

The WHO Second Global Patient Safety Challenge addressed surgical and anaesthesia safety and looked at the evidence base behind surgical complications. The result was a comprehensive clinical review and a 19-point WHO Surgical Safety Checklist (Fig. 1.1) intended to focus on key parts of the surgical journey.[10]

Before induction of anaesthesia → Before skin incision → Before patient leaves operating room

Before induction of anaesthesia

(with at least nurse and anaesthetist)

Has the patient confirmed his/her identity, site, procedure, and consent?
- ☐ Yes

Is the site marked?
- ☐ Yes
- ☐ Not applicable

Is the anaesthesia machine and medication check complete?
- ☐ Yes

Is the pulse oximeter on the patient and functioning?
- ☐ Yes

Does the patient have a:

Known allergy?
- ☐ No
- ☐ Yes

Difficult airway or aspiration risk?
- ☐ No
- ☐ Yes, and equipment/assistance available

Risk of >500ml blood loss (7ml/kg in children)?
- ☐ No
- ☐ Yes, and two IVs/central access and fluids planned

Before skin incision

(with nurse, anaesthetist and surgeon)

- ☐ **Confirm all team members have introduced themselves by name and role.**
- ☐ **Confirm the patient's name, procedure, and where the incision will be made.**

Has antibiotic prophylaxis been given within the last 60 minutes?
- ☐ Yes
- ☐ Not applicable

Anticipated Critical Events

To Surgeon:
- ☐ What are the critical or non-routine steps?
- ☐ How long will the case take?
- ☐ What is the anticipated blood loss?

To Anaesthetist:
- ☐ Are there any patient-specific concerns?

To Nursing Team:
- ☐ Has sterility (including indicator results) been confirmed?
- ☐ Are there equipment issues or any concerns?

Is essential imaging displayed?
- ☐ Yes
- ☐ Not applicable

Before patient leaves operating room

(with nurse, anaesthetist and surgeon)

Nurse verbally confirms:
- ☐ The name of the procedure
- ☐ Completion of instrument, sponge and needle counts
- ☐ Specimen labelling (read specimen labels aloud, including patient name)
- ☐ Whether there are any equipment problems to be addressed

To Surgeon, Anaesthetist and Nurse:
- ☐ What are the key concerns for recovery and management of this patient?

This checklist is not intended to be comprehensive. Additions and modifications to fit local practice are encouraged.

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Fig. 1.1 World Health Organization Surgical Safety Checklist. Reproduced with permission from the World Health Organization. http://whqlibdoc.who.int/publications/2009/9789241598590_eng_Checklist.pdf © World Health Organization 2009.

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- It provides a framework to ensure that crucial steps are not omitted in operating room care. It is a team communication tool and is not intended as a tick-box exercise.
- The WHO checklist was trialled in four high- and four middle- and low-income countries and resulted in a reduction in surgical complications and mortality.[11]
- Hospitals are encouraged to modify the checklist to reflect local practice; eye surgery is different to cardiac surgery. Care should be taken to avoid making it too complex or simplistic.
- Successful introduction of the WHO checklist requires a shift in theatre culture where leadership from senior clinicians, nurses and allied health practitioners is key.

Briefings and debriefings

Team discussion before the start and end of an operating list improves communication, teamworking, theatre efficiency, planning and patient safety. A debrief at the end of the list allows the team to both acknowledge good practice and identify areas requiring improvement.

- When briefing and debriefing are combined with the WHO Surgical Safety Checklist, a strong safety culture is engendered (Table 1.1).

Documentation

The General Medical Council (GMC) guidance on good medical practice states that in providing care, the anaesthetist must keep clear, accurate, legible and contemporaneous records that report the relevant clinical findings, the decisions made, the information given to patients and any drugs prescribed or other investigation or treatment.[12]

- Good documentation in anaesthetic practice is essential for maintaining excellent clinical care, facilitating handover of care between health care professionals, aiding interspecialty communication and enabling patient identification without risk or error.
- Electronic patient and anaesthetic records are becoming more widespread. In many ways, they improve patient safety, but they come with their own set of problems. Avoid 'copying and pasting' information, as this can lead to errors. Be meticulous in saving records under your name and protecting your passwords. Electronic prescriptions should be checked in the same vigilant manner you would apply if you were writing the prescription. Do not assume a computer will prevent you from prescribing the wrong drug at the wrong time in the wrong amount.
- Previous anaesthetic charts are valuable resources for anaesthetists during their preoperative assessment of patients. They are also useful for education and retrospective clinical governance.
- The outcome of medicolegal cases is often dependent on the anaesthetic record. An untidy, illegible, scantily completed chart may be taken as indirect evidence of shoddy or inattentive care.[13]
- There is no standard anaesthetic record in the UK. However, the RCoA and AoA have set out what they regard as the minimum data set for the anaesthetic record.[14]

Table 1.1 Suggested components of briefing and debriefing

Briefing	Introduction of team members and roles
	Patients and procedures planned
	Confirmation of order of list
	Specific equipment (anaesthesia and surgery) required
	Any concerns relevant to the day
Debriefing	Thanks to team members for specific actions
	Factors that went well that were useful lessons
	Factors that could be improved for next time

Drug safety

Anaesthetists are at high risk of making drug errors due to the frequency and rapidity of drug administration. Prospective data suggest errors may occur in 1 in every 133 anaesthetics.[15,16]

- ⚠ Any drug can be harmful if given in the wrong quantity, wrong dilution or by the wrong route.
- The appearance of ampoules changes with alarming frequency as hospitals reduce costs by sourcing the cheapest formulation. Do not rely on pattern recognition. Pay particular attention to ampoules of drugs that look alike or sound alike.
- Avoid distractions. Do not talk to someone else or allow other distractions while drawing up drugs. Drug error is more likely when fatigued, busy or distracted or when multiple anaesthetists work together.
- Do not assume that the drug in the ampoule is the same as the drug labelled on the outside of the box.
- Many drugs are available in different concentrations in the same hospital (e.g. propofol, ketamine, heparin, insulin, atropine). Before drawing up any drug, always check the generic name, concentration, volume and expiry date on the ampoule, preferably with a second person.
- Label syringes immediately after drawing up a drug; do not put a syringe containing a drug down until it is labelled.
- Consider a second person to check drugs prone to errors (potassium, insulin, heparin, inotropes).
- If you have never given the drug before, or not by that route, check with someone who has.
- Always check controlled drugs. Sign for what you have given and what is discarded.
- Dilution of medications for paediatric patients is very anaesthetist-dependent (e.g. suxamethonium 50mg/mL or 10mg/mL, fentanyl 50 micrograms/mL or 10 micrograms/mL). Confirm with your team which dilutions are being used and write these on the syringes clearly. It is easy to make a mistake by a factor of 10 if you are in a rush. Work out doses before the patient arrives and if in doubt, check with a second person.

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- ISO26825 recommendations for pre-printed colour-coded labels should be adhered to.
- Ensure any writing on labels is legible and clear, particularly the concentration of the drug in the syringe. Use standard units and dilutions wherever possible.
- It is preferable to use Tallman lettering (Box 1.1) to help differentiate drug names that are similar. Tallman lettering involves a combination of upper and lower case lettering to emphasise key differences in drugs with similar names.
- Where available, use special red-coloured syringes to draw up muscle relaxants.
- For high-risk drugs such as muscle relaxants, incorrectly timed administration can be minimised by adding additional labelling covering both the syringe and the sheathed drawing-up needle, such that this additional label must be torn prior to administering the drug.
- Additional ISO preprinted self-adhesive labels can be placed on the distal end of infusion lines to easily identify the drug being infused.
- ⚠ **Avoid** having drugs/fluids poured into unlabelled pots onto a sterile field, particularly when neuraxial or other regional procedures are being performed. Drugs and 0.9% sodium chloride should be double-checked and drawn up directly into a syringe with the aid of an assistant. Completely separate chlorhexidine and other antiseptics from other drugs that are being drawn up when preparing for neuraxial or regional procedures.
- Drugs used for different routes of administration should be kept separately.
- Do not administer a drug if you are unsure of what it is or its concentration.
- It is good practice to check electronic postoperative orders twice or by another person to ensure the correct patient and order has been given.
- Admit errors; usually there is an opportunity to minimise harmful effects and this should not be missed.
- Inform the patient of any error in a timely fashion and apologise. You have a duty of candour.

There are multiple electronic safety systems being integrated into anaesthetic practice around the world to reduce drug error. For example, drugs can be checked against allergies and comorbidities via radiofrequency or barcoded drug labels which are scanned prior to administration. Electronic medical records can also reduce error by providing reminders for safe drug dosages. Similarly, software has been integrated into drug delivery devices such as 'smart pumps' which warn users when dosages fall out of safe ranges and allow cross-checking with electronic patient data such as age, allergies and comorbidities.

Box 1.1 Examples of Tallman lettering

- DOBUTamine
- ePHEDrine
- hydrALAZINE
- fentaNYL
- cefaLEXin
- DOPamine
- EPINEPHrine
- HYDROmorphone
- SUFentanil
- cefaZOLin

Vigilance and crisis management

Anaesthetic emergencies may develop rapidly into life-threatening conditions that cannot be managed effectively by an individual and require a team response. In a theatre setting, the anaesthetist is likely to be the team member with the specialist knowledge and skills to deal with the event. This can give rise to intense pressure. Always call for help early. A fresh pair of hands and eyes is invaluable assistance, even if only to reassure you that you are already dealing with the situation appropriately. Critical incident protocols, cognitive aids and drills have been designed to aid in the management of common and uncommon emergencies. Recognition of a problem is the first step in any management strategy, and all members of a team should be empowered to voice their concerns without fear of a derisory or dismissive reception.

The unanticipated crisis

- ⚠ Declare problems clearly and early to the whole theatre team.
- ⚠ Call for help early.
- Resuscitation should be ongoing while you figure out the diagnosis.
- A repeated and systematic ABC approach helps render the patient 'safe' and buys thinking time.
- A good team leader is able to step back from the situation to consider the whole picture. This can only be achieved through task delegation. It is more effective if the leader knows the team members' names (WHO briefing), as well as their roles and expertise.
- Do not dwell on whatever or whoever 'caused' the crisis; use objective and non-judgemental communication.
- To communicate effectively, your messages or commands must be:
  - Addressed: ask specifically named individuals to perform tasks
  - Heard: reduce background noise (e.g. turn off the radio)
  - Understood: if you make a complex request, ask the recipient to repeat it back to you (closed loop communication).
- If the cause of the problem is unknown, say so. Voice your thoughts and reasoning and your team is more likely to be able to help you. Encourage others to contribute.
- Reappraise the situation regularly. Update the rest of the team with new information.
- Often you will not be the team leader, but an equally valuable member of the team nonetheless—a team player. A good team player is adaptable, understands other team members' roles, assumes complete responsibility for delegated problems and feeds back information to the rest of the team.

Handling serious events and conflict in theatre

Debriefing after adverse events in theatre is recognised as a tool to facilitate improved teamworking. A structured approach is often used and many hospital trusts have teams to assist with these formal debriefs. You should also seek personal support, contact your medical defence organisation and identify any areas where training or return-to-work programmes may be beneficial.

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Bullying and conflict are detrimental cultures within health care. Campaigns such as 'Knock it Out' within anaesthesia and 'Hammer it Out' and 'Cut it Out' from our orthopaedic and surgical colleagues, respectively, aim to create a positive workplace culture where individuals are empowered to speak out against unacceptable behaviours and promote exemplary behaviours. 'Civility Saves Lives' is a collective voice for the importance of respect, professional courtesy and valuing each other.[17]

Keeping up-to-date

Continuing professional development refers to any learning outside of undergraduate education or postgraduate training which helps doctors to maintain and improve their performance. It covers the development of knowledge, skills, attitudes and behaviours across all areas of a doctor's professional practice. It includes both formal and informal learning activities.[18] Since 2012, all UK licensed doctors have to undertake 5-yearly revalidation to demonstrate to the GMC that they are up-to-date and fit to practise. For trainees, this process is incorporated into the Annual Review of Career Progression (or similar). Supporting information will include:

- A diary of CPD
- Quality improvement activities
- Significant events
- Feedback on your practice from patients and colleagues
- Review of compliments and complaints.

Evidence-based medicine (EBM)

Defined as conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. It teaches how to ask a specific and relevant question arising from clinical practice, how to access and critically appraise up-to-date knowledge ('evidence') (Table 1.2) and then, using clinical experience and judgement, how to determine whether the evidence is applicable to a clinical setting. Use of proven effective treatments should improve patient outcome.

- EBM depends on well-designed studies producing reliable results, with an emphasis on randomised controlled trials (RCTs).
- Random assignment to treatment group and objective assessment of outcome are the best methods of avoiding bias. A consistent finding from several RCTs is very convincing, and so the pooled results of such trials constitute high-level evidence.
- Small RCTs are prone to type 2 error—incorrectly accepting the null hypothesis, and so a beneficial (or harmful) effect of treatment might be missed.
- Large RCTs are needed to provide sufficient study power to identify effective treatments.
- Large, multicentre RCTs and meta-analyses of numerous RCTs can include a broad range of patients and health care settings to better reflect everyday clinical practice.
- Most studies in anaesthesia are too small to detect effective treatments that can prevent adverse outcomes; too often, they focus only on surrogate endpoints.

Table 1.2 Levels of evidence

Level	Description
1	Meta-analysis of RCTs (with homogeneity) or individual RCT with narrow confidence interval
2	Low-quality RCT or cohort studies
3	Case-control study
4	Case series (and poor-quality cohort and case-control studies)
5	Expert opinion or based on basic science research

Statistical analysis: the basics

Binary outcomes

The effect on binary outcomes (complication/no complication) can be summarised by the relative risk (RR) (Table 1.3) or odds ratio (OR).

- RR is the probability of an event occurring in the exposed group vs the non-exposed group. In order to use the RR which is based on the incidence of an event, the exposure status of subjects must be known. The probability (risk) of rolling a 6 on one die is 1/6.
- Odds is a different way of considering probability, comparing the specified outcome against the remaining possible outcomes. In this way, the odds of rolling a 6 on one die is 1:5, for 6 possible outcomes. The OR is the ratio of the odds of an event occurring in the exposed group vs the odds of it occurring in the non-exposed group. The OR will approximate the RR for uncommon events but will otherwise overestimate the risk and should be avoided if the RR can be used.
- An RR of 1.0 indicates no difference in risk between the groups being compared. RR <1 ↓ risk, and RR >1 ↑ risk.

Numerical outcomes

Numerical outcomes (e.g. cardiac index or opioid consumption) can be summarised as a weighted mean difference.

Table 1.3 Interpreting relative risk

Relative risk (RR)	Effect on risk
0.25	75% reduction in risk
0.5	50% reduction in risk
1.0	No effect
1.5	50% increase in risk
2.0	100% (or 2-fold) increase in risk

p-values

A null hypothesis states that there is no relationship between the two variables being studied and any discrepancy in results is due to chance, rather than being a significant finding. Levels of statistical significance are described by the p-value. The smaller the p-value, the stronger the evidence that the null hypothesis should be rejected. A p-value of 0.05 is taken as

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expressing statistical significance (that there is only a 5% chance that the results obtained are due to chance alone). p-values support research hypotheses by rejecting null hypotheses but cannot prove that the research hypothesis is true.

95% confidence interval (CI)

The 95% CI is the range in which we can be 95% certain the true number lies. The width of the CI indicates the precision or reliability of the estimate. If either 95% confidence limit were the true effect, and if such a finding would change the conclusion of the study, then we are left with uncertainty regarding the outcome of the study.

How to interpret a meta-analysis

A systematic review is the process of analysing similar studies and a meta-analysis is the statistical method used to pool those results. A meta-analysis may be done using either a fixed effect model, which assumes that the individual study results are correlated with one another and probably represent similar study populations, or a random effects model, which does not require this assumption. The latter should be used if there is study heterogeneity.

There are some weaknesses with a meta-analysis, e.g. publication bias (negative studies are less likely to be published), duplicate/repeated publication, heterogeneity and inclusion of outdated studies; they probably inflate the risk of type 1 error (incorrectly rejecting a true null hypothesis). Meta-analyses that have minimal heterogeneity, narrow CIs and a large number of study events and that include at least one large RCT tend to be more reliable.

Forest plot: the basics

A forest plot can be used to graphically represent the individual studies contributing to a meta-analysis.

Fig. 1.2 summarises four trials comparing paravertebral block (PVB) with epidural analgesia to reduce pulmonary complications. The PVB group in column 1 is the intervention group and the epidural group in column 2 is the control group.

- The estimated effect (in this case, RR) of each trial is represented by the box and its 95% CI. The size of the box reflects the size of the study, and this is quantified by the study weight (%). The horizontal lines through the boxes illustrate the length of the CI. The longer the lines, the wider the CI and the less reliable the study results. The vertical line is the line of no effect (i.e. the position at which there is no clear difference between the intervention group and the control group). If the CI crosses the vertical line, then the individual study is not statistically significant.
- Pooled data are represented by the diamond at the bottom, whose area and width are also proportional to the pooled data size and 95% CI, respectively. If the diamond touches the vertical line (RR of 1), the overall combined result is not statistically significant.
- In this meta-analysis, the pooled RR is 0.41, indicating a 59% reduction in risk of pulmonary complications in the treatment group (PVB), compared to the control group (epidural). The CI of this estimated RR ranges from 0.17 (83% risk reduction) to 0.95 (5% risk reduction), which is weakly statistically significant ($p = 0.04$).

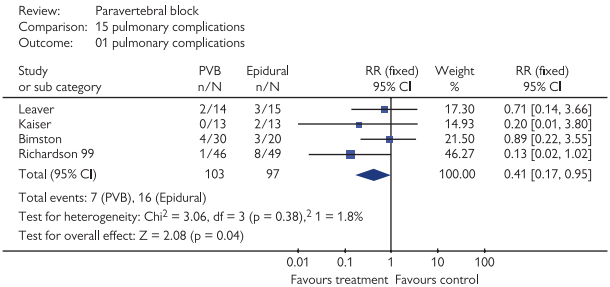

Fig. 1.2 Example forest plot showing four trials comparing paravertebral block with epidural analgesia to reduce pulmonary complications. See text for details. Source: data from Davies RG, Myles PS, Graham JM (2006). A comparison of the analgesic efficacy and side-effects of paravertebral vs epidural blockade for thoracotomy—a systematic review and meta-analysis of randomized trials. *Br J Anaes*, **96**, 418–426.

Finding the evidence

Many helpful guides to performing literature searches are available on the Internet. Medical librarians can also be helpful and may even perform a literature search for you. A selection of useful literature search resources is below.

Resources on EBM

- The Oxford Centre for Evidence-Based Medicine. How to practise EBM. http://www.cebm.net
- Bandolier. Premier EBM site with a focus on pain. http://www.bandolier.org.uk
- The UK National Institute for Health and Care Excellence. Produces evidence-based clinical guidelines. http://www.nice.org.uk
- Turning Research into Practice (Trip). Search engine linking databases of systematic reviews. http://www.tripdatabase.com

Literature searches

- PubMed. Free search engine accessing primarily the MEDLINE database. http://www.ncbi.nlm.nih.gov/pubmed
- OvidSP. Medical literature platform giving access to medical databases such as Embase, Ovid MEDLINE and Cochrane.

Preappraised summaries of the evidence

- Cochrane Collaboration. Global network of systematic reviews with links to a teaching resource for meta-analysis. http://www.cochrane.org
- Database of Abstracts of Reviews of Effects (DARE). Evaluates the effects of health care interventions and health service organisation and delivery. It contains over 35 000 abstracts, reviews and protocols. http://www.crd.york.ac.uk/CRDWeb

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- PROSPERO. An international database of prospectively registered systematic reviews. It aims to provide a centralised resource in which proposed systematic reviews can be collated, so avoiding duplication. http://www.crd.york.ac.uk/PROSPERO

Clinical trials and meta-analysis

- The James Lind Library. Collection of essays on the development of 'fair tests of treatments'. http://www.jameslindlibrary.org

Looking after yourself

⚠ 'First, do no harm' begins with yourself.

Self-care and resilience

Anaesthesia is a demanding occupation—from exams, shift work and moving around for training rotations to the clinical challenges our patients pose and the need to maintain our skills and keep up-to-date. It is increasingly recognised that taking time for self-care is vital and building our own resilience is required. We need to try to stay physically and mentally healthy: exercise, a healthy diet, hobbies, social activities, awareness of potential for addictive or destructive behaviour and seeking help when indicated. All are easier said than done, and building a support network around you is very important.

Recognising fatigue and burnout

The 2015 tragic death of an anaesthetic trainee driving home after a series of night shifts prompted a national survey of fatigue in trainee anaesthetists.[19] This was followed by a 2018 survey of anaesthesia and critical care consultants,[20] showing the vast majority of respondents reported work-related fatigue impacting on all areas of life.

The Fight Fatigue campaign recommends a shift in culture to ensure awareness of the importance of guarding against fatigue in maintaining patient safety and staff well-being. This requires detection, education and prevention. Practical tips for anaesthetists on night shifts have been publicised and improvements to rest facilities while on shift have been recommended.

Burnout is a syndrome characterised by depersonalisation, emotional exhaustion and loss of sense of achievement.[21] The clinical symptoms and signs of burnout are often non-specific and can include depression, irritability, insomnia, tiredness and anger. There are individual, environmental and organisational risk factors for the development of burnout and tools are available for practising clinicians to improve well-being, increase resilience and reduce risk of burnout. Burnout is associated with significant morbidity and mortality and the management of burnout syndrome involves a multidisciplinary approach. Beware of signs of burnout or emotional distress in yourself and colleagues. If you are struggling, seek help urgently, and if you know of someone else who is struggling, offer support.

Resources

- **DocHealth** is a confidential, not-for-profit psychotherapeutic consultation service for all doctors. It is delivered by consultant medical psychotherapists based at BMA House in London. https://www.dochealth.org.uk/
- **Royal Medical Benevolent Fund** provides financial help for medical students, doctors and their families who are facing financial hardship. https://rmbf.org/
- **Practitioner Health Programme** is an award-winning, free and confidential NHS service for doctors and dentists with mental illness and addiction problems who are working or looking to return to clinical practice. http://php.nhs.uk/
- **BMA support pages** are devoted to well-being services and support. https://www.bma.org.uk/advice-and-support/your-wellbeing#wellbeing-support-services

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- **Doctor Support Service** provides confidential phone and face-to-face support for doctors facing GMC fitness to practise hearings. http://www.bma.org.uk/support-at-work/doctors-well-being/doctor-support-service
- **Doctors' Support Network** provides peer support group for doctors with mental health concerns, including stress, burnout, mood and eating disorders. https://www.dsn.org.uk/
- **The Sick Doctors Trust** is an early intervention programme for addiction, which facilitates treatment in appropriate centres, arranges funding for inpatient treatment and provides advocacy and representation when required. http://sick-doctors-trust.co.uk/
- **The Couch (doctors.net)** This service is available to doctors registered with doctors.net. There is a forum for mutual support and advice, with the option of anonymous posting and a long list of doctors around the UK happy to help colleagues in distress. http://www.doctors.net.uk
- **Association of Anaesthetists wellbeing & support pages**. https://anaesthetists.org/Home/Wellbeing-support
- **Tea & Empathy** is an online peer-to-peer support group. https://www.facebook.com/groups/1215686978446877; Twitter: @tea_empathyNHS

Drug addiction and suicide in anaesthesia

Anaesthesia can be an extremely rewarding, interesting and enjoyable career, but it can also be stressful, exhausting and emotionally draining. The relative availability of drugs of abuse means that unfortunately, anaesthetists have one of the highest risks of developing a drug addiction and of suicide. Care for your colleagues and care for yourself. If you are feeling overwhelmed or disconnected, or you think a colleague may be at risk, seek help. It is important that you comply with local guidance for recording controlled drugs. If you suspect medications are going missing or someone is showing signs of medication misuse, then you must report this to a senior colleague. Do not confront anyone or speculate with others. The ease of availability of these medications can be a burden and a temptation to some; seek help from occupational health or your general practitioner (GP) if needed.

The pregnant anaesthetist

Women make up a significant proportion of the anaesthesia workforce and the number of ♀ anaesthetists likely to be working when pregnant is increasing year-on-year.[22] Anaesthetic work poses certain risks to the mother and fetus which are hard to quantify: anaesthetic agents, ionising radiation, exposure to infections, manual handling, prolonged standing and shift work.

Individual risk assessments should be completed for all pregnant anaesthetists, and those with pre-existing medical problems or pregnancy-related complications will need greater input from their occupational health physician. Your department should support you while pregnant (e.g. reducing or stopping on-calls, avoiding radiation, etc.).

If you feel well during pregnancy, you should be able to do most things normally, but you should not put yourself at risk or carry on working if you are fatigued or unwell. Departments and training programme directors will be grateful for early notification to allow them the maximum time to cover your maternity leave.

Personal protective equipment (PPE)

Employers have a duty to protect you from avoidable risks in the workplace.
⚠ Keep yourself safe, first and foremost.

- Before undertaking any procedure, you should assess the likelihood of exposure to potentially harmful agents and wear appropriate PPE. There will be local policies to guide you. Examples of PPE include: gloves (sterile and non-sterile), aprons, masks of varying protection (e.g. fluid-resistant, respirator, aerosol-resistant), laser goggles, lead shields, aprons and radiation badges.
- PPE is only as good as the person putting it on. Make sure it is appropriate for the task, fits you, you know how to don and doff it correctly and you know how to dispose of it safely.

Blood and bodily fluid exposure

Exposure to blood and bodily fluids is a daily hazard for the anaesthetist. Develop safe practice from the very beginning of your career to minimise your likelihood of inoculation injury. It is hard to change your habits later in your career.

Prevention

Ensure your vaccinations are up-to-date; always wear PPE as per local policies, and dispose of sharps correctly.

Exposure

Broken skin or mucous membranes can be contaminated with blood or bodily fluid. Apply protective dressings to broken skin.

First aid

Remove the contaminant. If the skin is contaminated, wash with soap and water. If the skin is breached, encourage bleeding if possible. Irrigate contaminated mucous membranes with copious volumes of 0.9% sodium chloride; then dry and cover if possible. Remove contact lenses if eyes are contaminated. If in doubt about the seriousness of the injury, attend the ED.

Report

Report to your supervisor and fill out a Reporting of Injuries, Diseases and Dangerous Occurrences Regulations (RIDDOR) form or your local equivalent.

Risk assessment

Should not be done by the recipient (contaminated person). The type of incident, infective state of the donor (usually the patient), lifestyle risks and vulnerability of the recipient are all important. A hollow needle that breaks the skin carries the greatest risk of transmission (1:3 hepatitis B, 1:30 hepatitis C, 1:300 human immunodeficiency virus (HIV)).

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Occupational health

(Out of hours, notify the ED.) Post-exposure prophylaxis may be required (hepatitis B and HIV), and baseline bloods should be taken from the recipient. A third party needs to obtain informed consent from the donor before blood can be sent for testing.

Follow-up

This can be very stressful. Follow-up with occupational health is important. The recipient may be temporarily suspended from carrying out exposure-prone procedures until transmission is confirmed as negative.

Manual handling

Look after yourself by using the correct equipment and technique and attending any training offered. Do not compromise your safety or that of your team or the patient for the sake of rapidity. It is usual for the anaesthetist to take charge of movement of the patient in theatre; use clear commands to protect both the patient and staff from injury.

- The anaesthetist is responsible for protecting unconscious patients from harm when they are being moved. Pay attention to connected drips, wires, airway tubing, catheter bags, surgical drains and other potential hazards to avoid problems that can cause accidental injury to patients or staff.
- If you are carrying an injury, contact occupational health for advice before musculoskeletal injuries are exacerbated.

Anaesthetising colleagues and their relatives

During your career, you may be asked to anaesthetise a colleague, a friend or their relative. The initial flattery of being approached is invariably replaced quickly by anxiety. It may be something you are expert in or do routinely, but the added stress of knowing the patient can be a potential hazard. You may feel pressurised into doing something you would not normally do at personal request or be less thorough with your consent, having assumed understanding. Only agree if you feel happy to do so and if you have any concerns, politely decline or ask for senior help. If you are faced with this situation in an emergency, be aware of potential pitfalls; remain vigilant for differences from your normal practice, and ask for senior help if needed.

Dealing with a complaint/coroner's inquest

Unfortunately, many of us will have to deal with a complaint, an investigation or a coroner's inquest regarding aspects of our care during our careers. This is an upsetting and stressful time and should not be faced alone. Seek advice from colleagues, mentors and friends, along with your medical defence organisation and your hospital's legal team if needed.

- Sometimes the complaint occurs in your presence, but more often, it is via written communication to a third party. Coroner's inquests and investigations may take many months or years to come to a conclusion, during which time you may or may not be aware of the process.
- If you have any concerns about an event in which you have been involved, or think there is a possibility further investigation may occur, it is worthwhile contacting your medical defence organisation. Most have a 24h helpline and the advice, support and reassurance they offer is very helpful.

- When you hear you are involved in a complaint/investigation, inform your supervisor or head of department.
- If you feel that the complaint/investigation is having an adverse effect on your ability to do your job, inform your supervisor immediately.
- A complaint should be answered promptly with an honest explanation of the events. Ask a senior colleague, member of the hospital legal team or defence organisation to help you.
- Resolution of a complaint involves focus on the complainant's needs; offer a sincere apology, explanation/investigation or assurance it will not happen again. You should act fairly and openly and remain accountable for, and reflect on, your actions. Try to put things right if possible.
- If appropriate, apologise sincerely to the complainant—often this is all that is needed. It is advisable for both parties to have a support person present for any meetings. Document clearly what is said.
- If you are aware of a complaint, or are involved in an event that may warrant further investigation, make comprehensive, factual and contemporaneous notes. A list of personnel present is very useful. If it has been some time since the event, ask to see the notes, but do not make a copy; ensure you comply with data protection law.
- Your own separate contemporaneous record can differ from patient notes by also containing your thoughts, feelings, conversations and beliefs. These notes should contain no personal identifiers. Make as full a record as possible, as early as possible, so you are able to refer back at a later date. Always review the data protection policies to ensure that your record-keeping and storage comply with the law.

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Chapter 2

Preoperative considerations

Tom Blincoe, Ian Densham and David Kotwinski

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Better perioperative care

Perioperative medicine, also known as perioperative care, is the practice of patient-centred, multidisciplinary and integrated medical care of patients from the moment of contemplation of surgery until full recovery.[1] The aim is to reduce variation, increase patient satisfaction and improve outcomes.

- The number of surgical procedures performed each year is increasing. Patients are older and more comorbid. They are undergoing more major and increasingly complex procedures.[2]
- Perioperative medicine represents a concept of improved, more efficient care using integrated care pathways that focus on patients at the higher-risk end of the spectrum.
- The centre for perioperative care distinguishes four distinct episodes of any perioperative pathway, each with key elements[3] (Fig. 2.1).

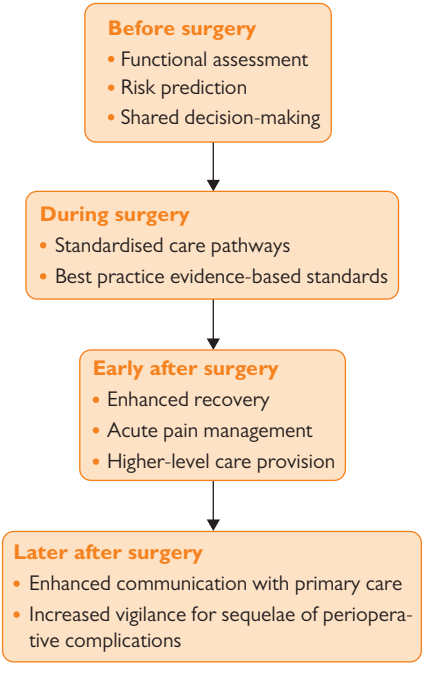

Fig. 2.1 Key elements of any perioperative pathway.

Preoperative assessment

All patients should be assessed prior to anaesthesia. There are two main parts to a preoperative assessment.

For planned surgery:

- A nurse-led, protocol-driven triage process may be the only assessment for fit patients prior to surgery, or this may be followed by a face-to-face consultation with a preoperative specialist nurse.
- Less fit patients undergoing major surgery require a comprehensive preoperative assessment. This should be performed by an anaesthetist or a perioperative physician who will engage in a meaningful, shared decision-making process.
- The second part occurs on the day of surgery. It involves a brief systemic enquiry for new or progressive symptoms, a check of fasting status, medications, allergies and a relevant examination.
- It is also an opportunity to discuss the planned anaesthetic technique, to establish rapport and to alleviate concerns.

For emergency or urgent surgery in the patient with an unplanned admission:

- The two parts described above merge into a more focused risk assessment.

Objectives of preoperative assessment

- Take a full history.
- Perform an examination.
- Establish optimal management of concomitant disease.
- Establish functional status/exercise capacity.
- Establish and discuss risk.
- Establish and discuss possible anaesthetic plans.
- Discuss concerns and expectations.
- Build rapport.
- Discuss immediate perioperative measures.
- Provide written information for further consideration.
- Document the process.
- Prevent cancellation on the day of surgery.

Further reading

There are robust guidelines for the delivery of preoperative assessment services and recommendations for the management of comorbidities, available from the AoA, RCoA, NICE and The Preoperative Association.[4,5,6,7]

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Preoperative history

Preoperative history taking in the preoperative assessment clinic should be comprehensive and cover the following areas.

Anaesthetic history

- Previous adverse events related to anaesthesia
- Airway difficulties (review of old charts)
- Anaphylaxis
- Postoperative nausea and vomiting (PONV)
- Difficult vascular access
- Unplanned critical care admission
- Family history of anaesthetic problems
- Malignant hyperthermia (MH) and suxamethonium apnoea.

Surgical history

- Indication for current procedure
- Patient's attitude towards condition
- Duration and progression of symptoms
- Effect of symptoms on the patient's life
- Trial of conservative measures/alternative options
- Previous surgical procedures:
  - May increase complexity/duration of proposed procedure
  - May have implications for regional anaesthesia/analgesia
  - Will inform risk–benefit discussion.

Medical history

A systemic enquiry is necessary to identify symptoms where further treatment or investigation may be required. Key areas of concern are:

Respiratory

(For detail, see ➔ p. 164.)

- Asthma, chronic obstructive pulmonary disease (COPD), obstructive sleep apnoea (OSA)
- Admissions and management of respiratory diseases
- Comparison of current and previous lung function tests.

Cardiovascular

(For detail, see ➔ Chapter 5.)

- Ischaemic heart disease (IHD), hypertension, pulmonary hypertension, myocardial dysfunction, valvular disease, arrhythmias
- Anaemia, previous transfusions and attitudes towards transfusion.

Endocrine

(For detail, see ➔ Chapter 9.)

- Diabetes.

Neurology

(For detail, see ➲ Considerations for the older patient, pp. 89–94; ➲ Chapter 12; ➲ Chapter 41.)

- Acute or chronic pain
- Frailty and cognitive impairment
- Previous cerebrovascular event (CVE)/transient ischaemic attack (TIA) and residual deficit.

Gastrointestinal

- Gastro-oesophogeal reflux disease (see ➲ p. 58)
- Nutritional status (see ➲ p. 50; ➲ p. 82).

Renal

(For detail, see ➲ Chapter 7.)

- Level of impairment/cause.

Musculoskeletal

- Considerations for positioning
- Considerations for regional anaesthesia.

Social history

- Occupation or retired occupation
- Family members, relationships and local support
- Accommodation arrangements
- Level of independence and ability with activities of daily living
- Alcohol consumption/misuse
- Smoking
- Recreational/illicit drug use.

Drug history and allergy status

- Record a full list of current medications and details of significant historical use, e.g. recent steroids, disease-modifying antirheumatic drugs (DMARDs) and chemotherapy.
- Careful perioperative management of certain medications, such as anticoagulants and diabetic medications, is required (see ➲ pp. 269–75; ➲ pp. 216–21).
- It is essential that instructions regarding medications are clear.
- Most medications can and should be continued until the morning of surgery.
- Be attentive for drugs that interact with anaesthetics (Table 2.1).

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Table 2.1 Common drugs that interact with anaesthetic agents

Drug group	Interaction
Cardiovascular system	
Angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs)	Potentially severe hypotension
β-blockers	Severe hypotension ± bradycardia
Calcium channel blockers	Severe hypotension, potential potentiation of neuromuscular blockade (NMB)
Diuretics	Electrolyte disturbance, potentiation of arrhythmias
Central nervous system	
Anticonvulsants	Liver enzyme induction
Benzodiazepines	Potentiation of anaesthetic agents
Tricyclic antidepressants (TCAs)	Inhibition of catecholamine metabolism
Monoamine oxidase inhibitors (MAOIs)	Potentiation of hypertension with vasopressors
Lithium	Potentiation of non-depolarising NMB
Other	
Steroids	Adrenocortical suppression (see pp. 230–1)
Aminoglycosides	Potentiation of NMB
Oestrogens	Increased risk of venous thromboembolism (VTE)

Preoperative examination

The completeness of the physical examination as part of a preoperative assessment will depend on the patient and the procedure. The following aspects of physical examination, however, are usually relevant.

General appearance

- Body habitus
- Vital signs
- Fluid status
- Nutritional status
- Mobility/aids.

Airway assessment

(For detail, see ➲ pp. 363–7.)

- Mouth opening/Mallampati score
- Jaw protrusion
- Dentition
- Neck range of movement
- Facial hair.

Respiratory

(For detail, see ➲ p. 164.)

- Presence of dyspnoea and/or cyanosis
- Medical interventions (oxygen (O_2), nebulisers, chest drain)
- Clarity of lung fields
- Scarring/previous surgery.

Cardiovascular

(For detail, see ➲ Chapter 5.)

- Arterial pulses and rhythm
- Vascular access options
- Heart sounds
- Peripheral oedema
- Presence of cardiac implantable electronic devices.

Neurological

(For detail, see ➲ Chapter 12.)

- Any pre-existing deficit.

Musculoskeletal

- Range of movements relevant to positioning (e.g. shoulder joints for proning)
- Examination of potential regional block sites.

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Preoperative investigations

- Preoperative investigations should be conducted according to individual patient circumstances.
- National guidelines exist[8] and should be taken into account to prevent unnecessary tests and target those in whom investigations will inform discussion of risk and support shared decision-making.
- Standardised referral pathways (with specific fitness for referral criteria) between 1° and 2° care should minimise duplication of investigations.
- NICE guidelines[8] make recommendations for investigations based on the grade of surgery and American Society of Anesthesiologists (ASA) score (Table 2.2). They also recommend the following:
  - Pregnancy testing in all women with childbearing potential
  - HbA1c testing only if diabetic and no test within last 3mo
  - No other tests should be offered routinely. These include chest X-ray (CXR), urine dipsticks, resting echocardiography and tests for sickle cell disease or trait.

Table 2.2 Recommendations for preoperative investigations

Grade of surgery	ASA grade		
	ASA 1	ASA 2	ASA 3/4
Minor			Consider renal function in those at risk of acute kidney injury (AKI) Consider electrocardiogram (ECG)
Intermediate		Consider renal function in those at risk of AKI Consider ECG	Check renal function and ECG Consider full blood count (FBC), coagulation screen and lung function tests
Major/complex	Perform FBC Consider renal function in those at risk of AKI Consider ECG if >65y	Perform FBC, renal function and ECG	Perform FBC, renal function and ECG Consider coagulation screen and lung function tests

Specialist investigations

- Some specialist investigations and functional assessment (see pp. 32–5) may be performed at preoperative assessment clinic. Some require referral.
- Detail on these investigations is provided in the relevant specialty chapters. A brief overview is provided here.

Cardiac investigations

(See ➲ Chapter 5, in particular pp. 104–5.)

Valvular disease

- Patients with clinically suspected valvular disease (> moderate stenosis or regurgitation) should undergo preoperative echocardiography if there is:
  - No prior echocardiogram within 1y
  - A significant change in clinical status or physical examination since the last examination.

Assessment of left ventricular function

- ↓ left ventricular (LV) systolic function is associated with perioperative complications, particularly postoperative heart failure.
- The association is greatest in patients with high predicted perioperative mortality.
- In non-cardiac surgery, consider echocardiography in patients who:
  - Have unexplained dyspnoea
  - Have known heart failure and worsening dyspnoea, or who have not had assessment of their LV function within 1y
  - Are potential candidates for solid organ transplant.
- Patients with significant LV impairment should be referred for specialist cardiology opinion and optimisation of cardiac function if urgency of surgery allows.

Cardiac stress testing

- Echocardiography at rest will not detect stress-inducible ischaemia.[9]
- Exercise ECG or pharmacological stress testing has a high negative predictive value for postoperative myocardial infarction (MI) or cardiac death.
- Local variation will exist as to the type of stress testing preferred.
- If cardiopulmonary exercise testing (CPET) is available (and the patient is able to exercise), then this should be considered, as it gives an assessment of functional capacity and cardiorespiratory fitness. (For CPET, see ➲ pp. 33–5.)

Cardiac implantable electronic device testing

- The function of cardiac implantable electronic devices should be checked prior to surgery if this has not been done routinely within 12mo for permanent pacemakers or 6mo for implantable cardioverter–defibrillators (ICDs).[10]

Respiratory investigations

(See ➲ p. 164.)

Spirometry

- Spirometry has not been shown to be predictive of postoperative outcomes.
- Preoperative spirometry can be used to:
  - Aid in the diagnosis of unexplained dyspnoea
  - Monitor lung function in those with known chronic lung disease
  - Assess lung function in patients with neuromuscular disorders
  - Predict postoperative lung function in those undergoing lung resection surgery.

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Lung ultrasound

- Particularly useful in the context of urgent and emergency surgery. Its uses include:
  - Exclusion of pneumothorax
  - Assessment of lung parenchyma
  - Diagnosis of pleural effusion.

Sleep apnoea screening

(For sleep apnoea, see ➲ pp. 73–5; ➲ p. 186.)

- Patients at high risk of OSA should be screened preoperatively.

Other

Gastric ultrasound

(For fasting, see ➲ pp. 57–8.)

- The objective of point-of-care gastric ultrasound is to aid decision-making when planning an airway management strategy.
- It can be considered where there is uncertainty over the fasting status of patients or where there may be delayed gastric emptying (e.g. pregnancy, diabetes, pain, obesity, etc.).
- Gastric ultrasound has been shown to be highly sensitive (1.0) and specific (0.975) to detect or rule out a full stomach.[11]

Further reading

American College of Cardiology (ACC) and American Heart Association (AHA) (2014). *2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery*. http://www.onlinejacc.org/content/accj/64/22/e77.full.pdf?_ga=2.166163326.1996434336.1588937684-1866901098.1588937684

European Society of Cardiology and European Society of Anaesthesiology (2014). *ESC/ESA guidelines on non-cardiac surgery: cardiovascular assessment and management*. https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/ESC-ESA-Guidelines-on-non-cardiac-surgery-cardiovascular-assessment-and-managem

Consultation models

Consultation models provide a structure for complex interactions that occur between patients and doctors. They may also enable more effective communication to take place. Many consultation models exist. One that is frequently applied to preoperative consultation is the Calgary–Cambridge model.[12] This consists of the following elements.

Initiating the session

- Preparation
- Establishing initial rapport
- Identifying the reason(s) for the consultation.

Gathering information to explore the case fully

- Biomedical perspective (symptoms, signs, clinical investigations)
- Patient's perspective
- Background information.

Building the relationship

- Using appropriate non-verbal behaviour
- Developing rapport
- Involving the patient.

Providing structure to the consultation

- Making the consultation organisation overt (summarising at the end of a specific line of enquiry and progressing from one section to another, using signposting and transitional statements)
- Attending to flow (structuring the consultation in a logical sequence and attending to timing).

Explanation and planning

- Providing the correct amount and type of information
- Aiding accurate recall and understanding
- Achieving a shared understanding and incorporate the patient's perspective
- Planning with shared decision-making.

Closing the session

- Ensuring appropriate point of closure
- Forward planning.

While these elements tend to be sequential, 'building the relationship' and 'providing structure' continue throughout the consultation.

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Functional assessment

- Evaluation of functional capacity is integral to preoperative risk assessment; a fundamental prerequisite for safe perioperative care.
- Objective functional capacity assessments are required to support preoperative risk stratification. Unfortunately, functional assessment tools suffer from a paucity of evidence and a lack of unified definition regarding the outcomes that are of most importance to patients (see p. 39).
- Subjective assessments have been shown to be less accurate at identifying those with poor cardiopulmonary fitness or predicting postoperative complications.[13]
- There are physical and non-physical functional assessment tests.

Tests of functional capacity

6-minute walk test (6MWT)

- Formal conduct of the 6MWT has been defined by the American Thoracic Society.[14] It involves the patient walking as many laps as possible in 6min of a flat, indoor 30m course.
- It has the advantage of being simple and cheap.
- Recent evidence suggests weak correlation between the 6MWT and CPET, and moderate correlation between the 6MWT and Duke Activity Status Index (DASI) scores.[15]

Exercise ECG and pharmacological stress testing

(See pp. 104–5.)

- For the detection of myocardial ischaemia and identification of those in whom percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) are indicated prior to major surgery.
- Exercise ECG also provides an indication of functional capacity.
- Those with pre-existing ECG abnormalities may undergo pharmacological stress testing or a myocardial perfusion scan.

Perioperative cardiac biomarker screening[16]

- There is likely a significant amount of undiagnosed/asymptomatic perioperative myocardial ischaemia contributing to 30d mortality.
- Cardiac biomarker screening represents a promising alternative to, or in conjunction with, other risk stratification methods to predict postoperative cardiovascular morbidity and mortality.
- Tests consist of preoperative natriuretic peptides (brain natriuretic peptide (BNP), N-terminal proBNP (NT-proBNP)) and early postoperative troponins.
- Postoperative troponin surveillance may help identify high-risk patients, particularly those urgent and emergency surgical patients lacking detailed preoperative investigation.
- Relatively simple, well-established medical risk reduction strategies (including antiplatelets, statins, β-blockade and direct oral anticoagulants) exist for those identified with abnormal results at high risk of morbidity and mortality.
- Cardiac biomarker testing is less expensive and resource-intensive than cardiac imaging, with promising predictive value for both cardiac and non-cardiac morbidity and mortality.
- More evidence is required prior to widespread implementation.

Cardiopulmonary exercise testing

- CPET is a non-invasive, objective assessment of exercise capacity.
- It is a dynamic, integrated evaluation of the cardiorespiratory system which reflects the physiological reserve of an individual and can be helpful in predicting postoperative outcomes.
- CPET is based on the premise that major surgery increases baseline O_2 consumption and that there is a correlation between reduced capacity for O_2 delivery and postoperative organ dysfunction.
- Consensus clinical guidelines on indications, organisation, conduct and physiological interpretation of CPET exist to standardise practice across centres and to build on the existing evidence base.[17]

Indications for CPET

- Prognostication of surgical outcome
- To inform shared decision-making
- Identification of patients suitable for prehabilitation
- To guide and monitor effects of preoptimisation
- To monitor effects of neoadjuvant chemotherapy
- To guide intraoperative anaesthetic management
- To guide levels of postoperative care
- Identification of pathology and causes of exercise intolerance.

Preparation and consent

- Patients should receive information on the rationale for testing, the risks, benefits and the process itself.
- A full medical history should be taken, with detail on cardiorespiratory comorbidity and medications.
- A pretest haemoglobin (Hb) is necessary.

CPET equipment and the exercise protocol

- The basic CPET requirements are full resuscitation facilities, a cycle ergometer, a metabolic cart and ancillary equipment for serial monitoring of arterial oxygen saturation (SaO_2), non-invasive blood pressure (NIBP) and ECG. Two staff members are advisable.
- The metabolic cart contains a calibrated gas analyser, a computer and screens which display the continuous 12-lead ECG with ST-segment analysis and graphical displays of the physiological changes occurring during exercise.
- Gas flows and volumes are quantified by a pressure differential pneumotachograph attached to the patient's mouthpiece.
- An incremental ramp protocol is used up until the limit of exercise tolerance is reached.
- A ramp slope (W/min) is selected, using patient gender, age, height and weight. It can then be adjusted according to comorbidity and functional status to achieve a testing duration of between 8 and 12min of ramped exercise (Table 2.3).

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Table 2.3 Calculating work rate increment (W/min)

Variable	Unit	Sex	Formula
Peak VO_2	mL/min	♂	Height (cm) – age (y) × 20
		♀	Height (cm) – age (y) × 14
VO_2 unloaded	mL/min		150+ (6 × weight (kg))

Work rate increment = (peak VO_2 – VO_2 unloaded)/100

VO_2 = O_2 consumption.

Test conduct

There are four stages to an exercise test:

- Rest (3min):
  - To record resting baseline data.
- Unloaded cycling (3min):
  - For the patient to become accustomed to a cadence of 60/min.
- Ramp exercise (8–12min):
  - Cycling continues until failure to maintain cadence of 60/min.
  - A Borg score (rating of perceived exertion) should be recorded.
- Recovery (5min):
  - Cycling continues unloaded for a short period.
  - Monitor the patient until variables return to near baseline values.

Key exercise response variables[17]

Three main variables of cardiorespiratory function are reported in relation to surgical outcome. Impairments in these indices are associated with an ↑ risk of postoperative morbidity and mortality following major non-cardiac surgery.

- VO_2 peak in mL/kg/min is the highest O_2 uptake (VO_2) achieved on a rapid incremental test at end-exercise. It differs from VO_2 max in that it represents the patient's best effort, rather than a physiological endpoint. Measured values are compared to predicted values to determine abnormality:
  - >80% predicted = not abnormal
  - 71–80% predicted = mildly reduced
  - 51–70% predicted = moderately reduced
  - <50% = severely reduced.
- Anaerobic threshold (AT) in mL/kg/min. This is the VO_2 above which arterial lactate first begins to increase systematically during incremental exercise, reflecting a metabolic switch to ↑ glycolysis.
  - A low AT has a predictive value for morbidity and mortality in many surgical cohorts, with a value of <11mL/kg/min widely used to delineate higher-risk patients.
  - This value should be interpreted in the context of other variables and the broader clinical picture, rather than used as an absolute threshold for decision-making.

- Ventilatory equivalents for carbon dioxide (VE/VCO_2) are indicators of ventilatory efficiency, representing the ratio of minute ventilation to carbon dioxide (CO_2) output at any point in time.
  - High values suggest hyperventilation and/or ↑ physiological dead space.
  - Values >34 at AT have been associated with adverse perioperative outcomes.

Caution should also be applied to absolute values indexed to body weight for VO_2 peak and AT, as they overestimate risk for patients with high body mass index (BMI) and underestimate risk for patients with a low BMI.

Interpreting CPET reports

- More holistic reporting and interpretation of CPET should guide decision-making away from that based purely on thresholds of specific exercise response variables.
- A CPET report consists of an objective assessment of exercise capacity and a more subjective review of the implications of this exercise capacity for the suggested intervention.
- A CPET report should be viewed alongside other risk assessment tools to inform the shared decision-making process.
- The key elements of CPET interpretation are as follows:[17]
  - Determine the reason for the CPET.
  - Review the patient's medical history and laboratory results.
  - Note the test quality–patient effort ± reason for termination.
  - Present data in tabular and graphical forms.
  - Report exercise capacity using AT and VO_2 peak.
  - Report other indices related to risk such as VE/VCO_2.
  - Evaluate the 1° cause of exercise limitation.
  - Comment on perioperative risk implications and further investigations advised.

Duke Activity Status Index

- The DASI[18] is a questionnaire which defines an individual's exercise capacity through the use of metabolic equivalents of task (METs) where 1 MET is the resting O_2 consumption of 3.5mL/kg/min for a 40y old 70kg ♂ (Box 2.1).
- DASI scores were the only assessment of functional capacity to be associated with the 1° outcome measure of death or MI within 30d in a large prospective cohort study.[13]
- Patients who cannot sustain 4 METs of physical activity frequently have adverse outcomes following high-risk surgery.

Box 2.1 Metabolic equivalents of common tasks

1–4 METs	Eating, dressing, dishwashing, walking around the house
5–10 METs	Climbing a flight of stairs, walking on level ground at >6km/h, running briefly, playing golf
>10 METs	Strenuous sports: swimming, singles tennis, football

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Risk assessment

- Surgical and anaesthetic intervention decisions are made by balancing the benefits against the risks.
- The actual risks and benefits of any given intervention are unique to individuals, the procedure, the type of anaesthesia and an individual's perception of what is risky and what a good outcome is.
- Meaningful outcomes for individual patients should be central to any discussion on risk and the context will guide specific risk disclosure.
- Risk assessment, shared decision-making (➲ p. 40) and informed consent (➲ p. 41) are closely related, but distinct entities and should be treated as such.

Estimating risk

- Reliably informing patients about risk remains a challenge.[19]
- Accurate and early risk stratification to identify high-risk patients supports the introduction of targeted measures to reduce modifiable risk via prehabilitation and optimisation, robust intraoperative management plans and appropriate postoperative support.
- It is essential for minimising adverse outcomes and relies on high-quality data at individual, institutional and national levels.
- It empowers patients and informs shared decision-making, facilitates communication and underpins the legal consent process.
- There are a variety of tools for risk assessment. They vary widely in their ease of use, accuracy and what cohort or risk they refer to.
- The process of risk assessment has been shown to positively influence patients' care, regardless of level of risk.[20]

Risk can be estimated using

- Biomarkers (see ➲ p. 32; ➲ p. 104)
- Functional assessment (see ➲ pp. 32–5)
- Risk assessment tools:
  - Scoring systems such as ASA, Lee's Revised Cardiac Risk Index and the Assess Respiratory Risk in Surgical Patients in Catalonia (ARISCAT) risk index
  - Prediction models such as Surgical Outcome Risk Tool (SORT), Portsmouth-Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity (P-POSSUM), the National Emergency Laparotomy Audit (NELA) risk prediction tool and the American College of Surgeons National Surgical Quality Improvement Programme (ACS NSQIP).
- Improving risk estimation tools, so that they predict outcomes that are important to patients, remains a key challenge.
- Being able to predict outcomes that are meaningful to patients will facilitate more informed decision-making prior to surgery.

Discussing risk

- Disclosure of risk has changed (following Montgomery vs Lanarkshire Health Board[21]) from the practice of medical professionals informing the patient of what they consider to be appropriate, to what a reasonable patient would want to know.
- This reflects the legal and moral shift away from paternalism towards autonomy and self-determination with respect to decision-making about health.
- Clinicians need to explore what patients consider to be relevant:
  - It may not be appropriate to detail very serious, very rare complications to a young, fit patient having a minor procedure, but this should not be assumed.
  - The focus of discussion for an elderly, comorbid patient listed for major complex surgery would likely focus on survival benefit and complications that may lead to loss of independence or a decline in functional status. This, however, cannot be assumed.
- Ideally, comprehensive written information ± direction to online resources should be provided and documented.
- The entire risk conversation is subject to bias and interpretation.
- Clinicians should seek to minimise such influences and support patients in their decision-making by providing them with information which is tailored to their individual needs.
- Factors that may alter the perception of a given risk include:
  - Exposure bias: under- or over-publicity
  - Regional bias: geographical variation
  - Severity: anxiety and fixation on serious complications, or serious events perceived as higher risk, despite rarity
  - Vulnerability: denial, 'it won't happen to me'
  - Controllability: more accepting of risk, more understanding of complications
  - Certainty: knowledge and understanding of events
  - Familiarity: experience of prior interventions
  - Presentation: spin on absolute vs relative risks, positive vs negative presentation of numbers, e.g. '90% survival' rather than '10% mortality'.
- Words, pictures or numbers can be used to communicate risk.
- Evidence suggests considerable variation in the numerical translation of verbal probability expressions such as 'negligible', 'low', 'moderate' and 'high' risk.[22]
- Patient leaflets and clear information, including an infographic of anaesthetic risks and probability phrases, are available on the RCoA website.[23] (See Table 2.4 for summary.)

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Table 2.4 Common events and risks in anaesthesia

Very common: >1 in 10	
Equivalent to one person in your family	
Shivering	>1 in 10
Temporary memory loss (mainly in over 60s)	>1 in 10
Sickness	>1 in 10
Thirst	>1 in 10
Sore throat	>1 in 10
Bruising	>1 in 10
Common: between 1 in 10 and 1 in 100	
Equivalent to one person in a street	
Pain at the injection site	>1 in 10 <1 in 100
Minor lip or tongue injury	>1 in 10 <1 in 100
Uncommon: between 1 in 100 and 1 in 1000	
Equivalent to one person in a village	
Minor nerve injury	>1 in 100 <1 in 1000
Rare: between 1 in 1000 and 1 in 10 000	
Equivalent to one person in a small town	
Peripheral nerve damage that is permanent	1in 000
Corneal abrasion	1in 2,800
Damage to teeth requiring treatment	1in 4500
Anaphylaxis (severe allergic reaction to a drug)	1in 10 000
Very rare: between 1 in 10 000 to 1 in 100 000 or more	
Equivalent to one person in a large town	
Awareness during an anaesthetic	1in 20 000
Loss of vision	1in 100 000
Death as a direct result of anaesthesia	1in 100 000

Source: data from Common events and risks in anaesthesia infographic, Royal College of Anaesthetists (rcoa.ac.uk/media/1946)[23]

Outcomes

- Audit, quality improvement and research can be used to review and analyse patient outcome data to improve patient care.
- Routinely collecting patient outcome data and using these to guide local practice is increasingly widespread in UK centres. For example, Patient Reported Outcome Measures (PROMs) following hip and knee replacement surgery have been collected since 2009.
- Measurement of outcome after surgery is essential. Well-defined outcome measures enable comparison between interventions, individual practitioners and institutions, and enhance risk stratification.
- Outcome measures can be considered as either clinical outcomes or patient-reported outcomes.
- Clinical outcomes include mortality and morbidity; 30d and 1y mortality are routinely and relatively easily collected.
- Morbidity has a much higher prevalence than mortality in the general surgical population and can be assessed using various tools such as the Postoperative Morbidity Survey (POMS)[24] or the Clavien-Dindo classification.[25]
- Recent emphasis has focused on the use of patient-reported outcomes as the more traditional and crude measures, such as 30d mortality, unsurprisingly fail to adequately determine the impact of interventions on patients' lives.
- There is a need for more meaningful measures that occur more frequently such as short-term complications and longer-term mortality.
- More common outcomes also yield more statistically meaningful comparisons.[26]
- The Perioperative Quality Improvement Programme (PQIP)[27] was established in 2016 and aims to look at perioperative care of patients undergoing major non-cardiac surgery. It measures complication rates, failure to rescue and patient-reported outcomes. Participating hospitals are provided with a live dashboard of their results, along with quarterly and annual reports.
- The Standardised Endpoints in Perioperative Medicine (StEP) initiative aims to define measures used in future studies to enable comparison between studies and consolidate the evidence base.[28]

Shared decision-making

- The context in which clinical decisions are made is wide-ranging.
- Patients vary considerably in their attitudes towards their health, their ability to make informed decisions and their understanding of options and possible outcomes of those options.
- The legal and moral landscape in which health care decisions are made is also changing (see Risk assessment, pp. 36–8).
- If possible, establish patients' values and preferences, as these should determine the significance of various risks.[29]
- Shared decision-making is the process in which clinicians and patients work together to select tests, treatments, management or support packages using both clinical evidence and the patient's informed preferences.[30]

Essential components of shared decision-making include:

- Reliable, balanced, evidence-based information that outlines options, outcomes and uncertainties
- Decision support counselling with a clinician to clarify options and preferences
- Recording, communicating and implementing patient preferences
- Shared decision-making recognises two forms of expertise, as shown in Box 2.2.[30]

Box 2.2 Sharing expertise

Clinician's expertise	Patient's expertise
• Diagnosis	• Experience of illness
• Disease aetiology	• Social circumstances
• Prognosis	• Attitude to risk
• Treatment options	• Values
• Outcome probabilities	• Preferences

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Decision aids

- Most clinical decisions are not straightforward, with multiple options and outcomes as possibilities. Just as clinicians are supported by clinical guidelines, patients also require decision aids.
- Decision aids are tools that not only provide information on options, but also help patients to decide which option to choose.
- Examples of decision aids include the following, with guidelines for assessing the quality of any given resource: patient information sheets, computer programs, smart phone or social media applications, interactive websites and filmed patient/clinician interviews.[31]
- The use of decision aids has been shown to result in: greater knowledge and understanding, more accurate risk perception, greater comfort with decisions, less regret with complications, better adherence to treatment, no increase in anxiety and fewer patients choosing major surgery.[32]

Consent

- The legal stance on consent is changing, and professionals need to keep abreast of evolving legal developments in the jurisdiction in which they practise.
- The key principles of biomedical ethics, autonomy and self-determination require clinicians to obtain consent prior to medical intervention.
- Health professionals who carry out procedures without valid consent are liable to legal action and investigation by professional bodies.[33]

Guidance on the process of consent[34,35]

- Information should be:
  - Provided as early as possible
  - Written or online for future reference
  - Tailored to the individual.
- Discussion should:
  - Include risks, benefits and alternatives
  - Include opportunity for questions
  - Be fully documented.
- Exceptions to the requirement for providing information:
  - Patients consistently express desire not to know
  - Informing the patient would pose a serious threat
  - Emergency treatment in the patient's best interests.
- In the UK, separate written consent is not required for anaesthesia for another procedure, although it is recommended for independent procedures.
- Consent is an ongoing process and should be obtained by either the individual performing the procedure or a suitable delegate. It may require repeated discussion and/or confirmation, with documentation at every stage.
- Patients can qualify consent by refusing aspects of treatment.[35]

Capacity

- Valid consent requires that the patient has capacity.
- To have capacity for consent, the patient must understand and recall the information provided, weigh up the risks and benefits, consider the consequences of not having the procedure and communicate a decision without coercion.
- It is the responsibility of the clinician obtaining the consent to establish whether or not an individual has capacity.
- Capacity is not absolute. It is time- and decision-specific, and is subject to change.
- The Mental Capacity Act (MCA) 2005[36] sets out the action required by law when a patient over the age of 16y lacks capacity. The main principles of the MCA 2005 are that an individual must:
  - Be assumed to have capacity unless proven otherwise
  - Be given all practicable help to make a decision before being treated as lacking capacity
  - Not be treated as lacking capacity merely because they make an unwise decision.

- An intervention or decision made on behalf of a person lacking capacity must:
  - Be in their best interests
  - Cause the least restriction of their rights and freedom of action to achieve the stated purpose.

Best interests

- Acting in a patient's best interests involves consideration of social, psychological and medical factors, and must be guided by the patient's own opinions, if possible.
- Family or close friends should be consulted, although this should not compromise care if emergent.
- The Independent Mental Capacity Advocacy Service (IMCAS) can provide support for patients without friends or family in England and Wales.[36]
- Many competent individuals anticipate a loss of capacity in the future and legally binding advance decisions (advance directives or living wills) can be written which specify refusal of certain life-sustaining treatment in the event of incapacity.
- Alternatively, individuals may have made a Lasting Power of Attorney (LPA), a legal document appointing a person with capacity to act on their behalf with health decisions, should they lose capacity in the future (England and Wales).[35]

Children and young people

(See also ➲ p. 915.)

- Competent young people (16 and 17y olds) can give consent for any treatment without obtaining separate consent from a parent or guardian.
- Those under 16y who demonstrate that they fully appreciate the risks and benefits of the planned intervention can also give consent.
- On the other hand, children and young people who refuse treatment may have their decision overridden by a parent or the court if they are likely to suffer irreversible harm as a result of their refusal.
- When a child lacks capacity for consent, parental consent should be sought. If such a child refuses treatment, judgement needs to be exercised by the parent and the doctor as to the level of restraint that is acceptable. When faced with a child who is uncontrollable for whatever reason, the anaesthetist should consider postponing the case until adequate premedication can be given.[35]

Qualified consent

- Qualified consent applies to patients who, for religious or personal reasons, may consent to treatment in general but refuse certain aspects of this treatment, e.g. Jehovah's Witness patients who refuse blood transfusion (see ➲ p. 462).[35] The details of the exact nature of the patient's restriction and the explanation of the risks should be carefully documented.

Planning postoperative care

- Crucial to preoperative planning is anticipating postoperative needs and where those needs will be best met.
- About 80% of surgery is done on ambulatory, day case pathways, but for more complex operations and comorbid patients, inpatient care is required.
- Local variation exists, but preoperative predicted mortality of >5% should warrant consideration of higher levels of postoperative care (Table 2.5).
- Intraoperative complications or unexpected findings may mean a patient requires a higher level of care than predicted preoperatively.
- Early communication with the surgical team, bed management and clinicians responsible for higher-level care is recommended to ensure patients' care needs are met.

Table 2.5 Levels of postoperative care

Level	Description
0	Patients whose needs can be met through normal ward care in an acute hospital
1	Patients at risk of their condition deteriorating, or those recently relocated from higher levels of care whose needs can be met on an acute ward, with additional advice and support from the critical care team
2	Patients requiring more detailed observation or intervention, including support for a single failing organ system or postoperative care and those 'stepping down' from higher levels of care
3	Patients requiring advanced respiratory support alone or basic respiratory support, together with support of at least two organ systems. This level includes all complex patients requiring support for multiorgan failure

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Enhanced recovery after surgery

- Enhanced recovery after surgery (ERAS) is the name given to a programme that is designed to get patients back to normal health as quickly as possible after major operations.
- All patients undergoing major surgery should follow an appropriate ERAS programme.
- It is an evidence-based approach to the perioperative pathway that has been shown to improve outcomes and ↓ length of hospital stay. (See Box 2.3 for the key ERAS principles.)
- The details of individual ERAS pathways vary, depending on the type of surgery and local needs. Example pathways are available on the ERAS society website.[37]

Box 2.3 Key ERAS principles

- Patient preparation and counselling
- Optimisation of nutrition and minimisation of fasting times
- Standardised anaesthetic and analgesic regimes
- Regional and neuraxial techniques to minimise opioid usage
- Minimally invasive surgery and ↓ use of nasogastric tubes (NGTs) and drains
- Early mobilisation and enteral nutrition

Ongoing care

- The surgical team is responsible for patients not requiring higher-level care once they leave the recovery area.
- An anaesthetist must be available to review and provide advice for patients with any problems/complications potentially related to anaesthesia and related procedures.

Enhanced postoperative care

- Centres in the UK are now setting up enhanced care areas that provide care between levels 1 and 2 (sometimes called 'level 1.5 units') for those higher-risk patients who do not require admission to a high dependency unit (HDU) but need a level of support higher than that offered on a ward.
- Enhanced care is a relatively new and evolving concept, with the aim of providing a higher level of observation, monitoring and interventions than on the general ward.
- Enhanced advice and support from the critical care team should be more easily accessible.
- Patients are under the combined care of the anaesthetic and surgical teams.

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Chapter 3

Preoptimisation

Tom Blincoe, David Kotwinski, and Ian Densham

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Preoptimisation

The role of the preoperative team has evolved over the past decade and the focus is now not only on assessment, but also on patient optimisation. This is where a collaborative approach is taken to get patients as fit for surgery as possible in the time frame available.

Preoptimisation includes both lifestyle and medical optimisation of comorbidity. The preoperative team works together with hospital specialists and GPs to identify and minimise risk factors for postoperative complications. With the facts, patients are then empowered to be fully involved in shared decision-making.

Patients' attitudes towards surgery are likely to shift in the future. Far from being passive participants on the 'conveyor belt' to surgery, patients are being encouraged to motivate and engage in physical and psychological training, so as to 'get fit' for their operations, an approach analogous to how an athlete would prepare for a marathon.

Prehabilitation

Habilitation is derived from the Latin '*habilitare*', meaning 'to make suitable' or 'to enable'. Prehabilitation is the practice of enhancing a patient's functional capacity before surgery, with the aim of improving postoperative outcomes (Fig. 3.1).[1] This may include lifestyle and nutritional advice, structured physical exercise and psychosocial support.

Physical exercise

Physical inactivity is one of the leading risk factors for death worldwide (WHO, 2018). In 2015/16, 26% of UK adults were classified as inactive (<30min of physical activity per week).

- There is extensive evidence that exercise prolongs life and reduces CVS disease in a dose-related fashion.
- A sedentary lifestyle is associated with twice the risk of premature death, compared to physically active people.[2]
- Patients should be encouraged to undertake a minimum of 150min of moderate exercise or 90min of vigorous exercise per week.
- Social prescribers can signpost patients to local physical activity initiatives. GPs may be able to prescribe free or subsidised gym memberships.
- Well-designed studies have demonstrated that preoperative exercise can improve exercise capacity.
- There is limited evidence to suggest preoperative exercise programmes reduce length of stay and postoperative pain and improve physical function following major surgery.[3]
- Large multicentre RCTs are under way, aiming to demonstrate a reduction in morbidity and mortality.
- Exercise programmes need to be tailored to individual patients, according to baseline fitness levels and the time frame available.

Psychological support

There may be a role for preoperative psychological support in those at risk of developing persistent and difficult-to-control postoperative pain.

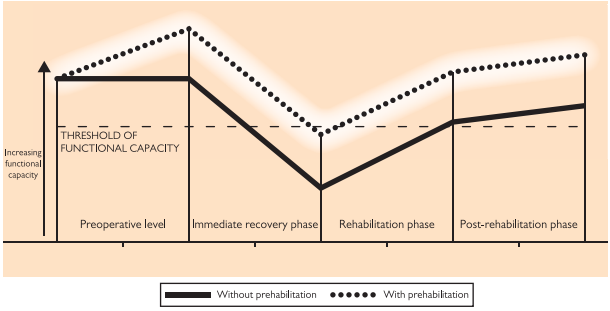

Fig. 3.1 Optimisation of surgical outcomes with prehabilitation.

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Nutrition

(See ➔ p. 82 for the malnourished patient.)

Patients who are at high risk of being undernourished should have formal dietetic referral prior to surgery (Fig. 3.2).

- Consider delaying their surgery to allow time for improvement in nutritional state.
- All patients having major surgery should have minimal fasting times and/or complex carbohydrate loading (including non-insulin-dependent diabetic patients).[4]

Behavioural change

Making Every Contact Count (MECC) is a behavioural change consensus statement published by NHS Health Education England in 2016. It recognises that telling people what to do is not the most effective way to change behaviour.

- Staff across health, local authority and voluntary sectors have thousands of contacts with individuals every day.
- MECC describes using these opportunities to raise awareness, encourage change and signpost to supporting agencies using an 'Ask, Advise, Assist' structure to promote health and healthy lifestyles.[5]

Alcohol

Alcohol is the most consumed recreational drug in Great Britain; 57% of adults drink alcohol regularly and nearly 10% drink on ≥5d per week.[6]

- Established liver cirrhosis presents a significant risk for perioperative morbidity and mortality, but even moderate consumption of alcohol is associated with ↑ rates of postoperative infection.
- Abstinence from alcohol for 6–8w preoperatively has been demonstrated to significantly reduce morbidity.[7]
- Using screening tools, such as the Fast Alcohol Screening Test and the Alcohol Use Disorders Identification Test, can identify those at risk of alcohol dependence/withdrawal and allow referral to specialist services for detoxification programmes.

Smoking

(See ➔ p. 173.)

In the UK, 14.4% of adults smoke regularly (2018).[8]

- Smoking is strongly associated with higher rates of significant postoperative complications (relative risk 1.3–2.5).[9]
- Smoking cessation should occur at least 4w prior to surgery to produce a significant reduction in risk. The longer the period of abstinence, the greater the risk reduction.
- A meta-analysis of preoperative smoking cessation concluded that intense behavioural interventions significantly reduced postoperative complications and long-term abstinence, compared to brief interventions.[10]

Step 1 BMI score + **Step 2** Weight loss score + **Step 3** Acute disease effect score

BMI kg/m²	Score
>20 (>30 Obese)	= 0
18.5–20	= 1
<18.5	= 2

Unplanned weight loss in past 3–6 months

%	Score
<5	= 0
5–10	= 1
>10	= 2

If patient is acutely ill **and** there has been or is likely to be no nutritional intake for >5 days **Score 2**

If unable to obtain height and weight, see full MUST document for alternative measurements and use of subjective criteria

Acute disease effect is unlikely to apply outside hospital. See 'MUST' Explanatory Booklet for further information

Step 4
Overall risk of malnutrition

Add Scores together to calculate overall risk of malnutrition
Score 0 Low Risk Score 1 Medium Risk Score 2 or more High Risk

Step 5
Management guidelines

0
Low Risk
Routine clinical care

- Repeat screening
  Hospital – weekly
  Care Homes – monthly
  Community – annually
  for special groups
  e.g. those >75 yrs

1
Medium Risk
Observe

- Document dietary intake for 3 days
- If adequate – little concern and repeat screening
  - Hospital – weekly
  - Care Home – at least monthly
  - Community – at least every 2–3 months
- If inadequate – clinical concern – follow local policy, set goals, improve and increase overall nutritional intake, monitor and review care plan regularly

2 or more
High Risk
Treat*

- Refer to dietitian, Nutritional Support Team or implement local policy
- Set goals, improve and increase overall nutritional intake
- Monitor and review care plan
  Hospital – weekly
  Care Home – monthly
  Community – monthly

* Unless detrimental or no benefit is expected from nutritional support e.g. imminent death.

All risk categories:

- Treat underlying condition and provide help and advice on food choices, eating and drinking when necessary.
- Record malnutrition risk category.
- Record need for special diets and follow local policy.

Obesity:

- Record presence of obesity. For those with underlying conditions, these are generally controlled before the treatment of obesity.

Re-assess subjects identified at risk as they move through care settings

See *The 'MUST' Explanatory Booklet* for further details and *The 'MUST' Report* for supporting evidence.

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Fig. 3.2 The Malnutrition Universal Screening Tool (MUST). The 'Malnutrition Universal Screening Tool' ('MUST') is reproduced here with the kind permission of BAPEN (British Association for Parenteral and Enteral Nutrition). For further information on 'MUST' see www.bapen.org.uk Copyright © BAPEN 2012.

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Obesity

(See ➔ p. 70.)

UK obesity prevalence ↑ from 15% in 1993 to 27% in 2015. In the UK, it is the 2nd leading risk factor for developing cancer.

- Patients with BMI >40 and obesity-related comorbidities are particularly at risk of postoperative complications. For example, obesity doubles the risk of joint infection after hip and knee arthroplasty.[11]
- Preoperative weight loss not only reduces perioperative risk,[12] but may also reduce the need for surgery.

Patient blood management, anaemia and iron deficiency

Patient blood management

Patient blood management (PBM) is a WHO-endorsed, multidisciplinary, evidence-based approach to the care of patients who are at risk of requiring a perioperative allogeneic blood transfusion.

- Effective PBM programmes are associated with not only ↓ blood transfusions, but also improved surgical outcomes, with ↓ infection rates, perioperative MI and CVE and ↓ 30d mortality, as well as reduced length of stay, readmission rates and costs.[13]
- PBM comprises three pillars of care:
  - Optimisation of haematopoiesis
  - Minimisation of blood loss
  - Management of postoperative anaemia.
- The detection and management of anaemia are central to the first pillar—optimisation of haematopoiesis. This is outlined below. (For more information on PBM, see ➔ pp. 458–9.)

Preoperative anaemia

(See also ➔ pp. 254–6.)

The prevalence of anaemia was found to be 36% in a large multicentre cohort of patients undergoing major surgery.[14]

- Perioperative anaemia and allogeneic blood transfusion are independent risk factors for poor postoperative outcomes such as ↑ length of stay, complications and mortality.[15]
- New recommendations quantify anaemia as Hb <130g/dL in adult surgical patients, irrespective of gender. Unlike the WHO definition of anaemia, this is not gender-specific because women have smaller circulating volumes and during surgery are just as likely to bleed as men. They are therefore at ↑ risk of requiring a blood transfusion.[16]
- The presence of anaemia should be investigated in all surgical procedures with expected moderate to high blood loss (>500mL).[16]
- Anaemia investigation and treatment should start as soon as the decision to undertake surgery is made. This gives the longest possible time for any treatments to be efficacious.
- Major non-urgent surgery should be postponed to allow the treatment of anaemia and iron deficiency.

Iron deficiency

- Iron deficiency affects >2 billion people worldwide and remains the leading cause of anaemia.[17]
- Iron is an essential human bioelement with roles in erythropoiesis, cellular respiration, O_2 flux, gene regulation and immunity.
- Symptoms of iron deficiency are non-specific and independent of anaemia. They include pallor, fatigue, exhaustion, palpitations, shortness of breath, 'brain fog', hair loss and muscle pain.
- Iron deficiency anaemia occurs in cases of severe iron deficiency.

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Iron physiology

- About 65% of iron is stored in Hb within red blood cells (RBCs) ± 15–20% is stored in macrophages, myoglobin, tissue enzymes and cytochromes, and ± 15–20% within the liver, spleen and marrow as haemosiderin and ferritin.
- Circulating (ferric) iron is primarily transported on transferrin, with typically ± 30% of the binding sites being saturated.
- Systemic iron homeostasis is primarily regulated by the hepatically produced peptide hormone hepcidin, which inhibits the movement of iron into the circulation. In addition to high systemic iron levels, hepcidin levels increase in response to infection, inflammation and malignancy.
- Iron used for the synthesis of new RBCs is mainly from macrophage-recycled senescent RBCs.
- Normally, daily iron losses are balanced by gastrointestinal (GI) absorption.

Aetiology

The causes can be broadly classified into causes of absolute and functional iron deficiency:

- Absolute iron deficiency is caused by ↑ iron requirements, limited supply (insufficient intake or ↓ absorption) and chronic blood loss.
- Functional iron deficiency is caused by ↑ hepcidin concentrations, which may be either genetic or caused by a chronic inflammatory state.

Investigating iron deficiency

- The hallmark blood test for iron deficiency is serum ferritin (sF). Normal sF levels are >100 micrograms/L, with sF of <15 micrograms/L considered pathognomonic of iron deficiency.
- Transferrin saturation (T_{sat}) values of >20% are required for normal erythropoiesis.
- The main problem with sF is that it is an acute phase reactant, so iron deficiency can exist with normal sF levels.
- In the presence of inflammation and/or T_{sat} <20%, an sF level <100 micrograms/L is indicative of iron deficiency.[16]
- GPs should be informed of any new diagnosis of iron deficiency anaemia made by the preassessment service, so that the cause can be fully investigated.
- Fig. 3.3 shows an example algorithm for the diagnosis of iron deficiency anaemia.

Treatment of iron deficiency anaemia

- The aim of preoperative iron treatment is replenishing iron stores (sF >100 micrograms/L) and achieving Hb ≥130g/dL, irrespective of gender.
- Despite the focus on patients with a high transfusion risk, it is considered good practice to treat all surgical patients with iron deficiency anaemia.
- Patients with iron deficiency (without anaemia) undergoing high-transfusion risk surgery should also be considered for supplementation.
- Treatment options are either oral (PO) or intravenous (IV) iron preparations.

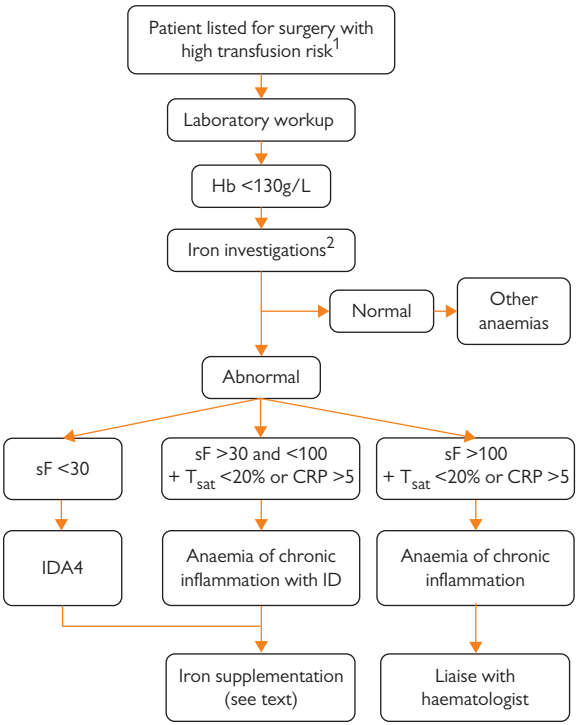

Fig. 3.3 Algorithm for management of patients with iron deficiency anaemia. CRP, C-reactive protein (mg/L); sF, serum ferritin (micrograms/L); Tsat, transferrin saturation (%).

Notes:

[1] Transfusion risk >10% or estimated blood loss >500mL. Includes CABG, cardiac valve procedures, colorectal resection, cystectomy, nephrectomy, 1° and revision hip or knee replacement, open carotid artery procedures and other open aortic/iliac vascular operations.

[2] International consensus suggests iron investigations include sF, T_{sat} and CRP (or equivalent inflammatory marker). However, some centres use reticulocyte Hb content.

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Oral iron

- If >6w until surgery, then consider a trial of PO iron (40–60mg daily or 80–100mg alternate days) and check Hb at 4w before surgery. If an inadequate response or intolerant (gastric upset), then switch to an IV treatment strategy.
- Effective in many but requires time and education to work.

IV iron

- If <4–6w to surgery, give IV iron.
- Highly efficacious, with symptom relief achieved at d3 and an Hb response at d5 (maximal at 2–3w).[18]
- Even giving 1d preoperatively has been shown to aid postoperative recovery.[19]
- Side effects: irreversible skin discoloration with extravasation (use an infusion pump), fishbone reaction—a self-limiting reaction with flushing, chest tightness and myalgia, and without hypotension, wheezing, stridor or oedema (pause infusion for 15min), hypersensitivity <1/25 000 (less frequent with newer preparations).
- Expert haematology and pharmacist advice should be sought when developing local policies and considering which preparations to use.
- Whilst it is recommended practice, and considered safe and effective, to treat patients with iron deficiency preoperatively, it is important to acknowledge the recently published PREVENTT trial. This was a UK-based randomised controlled trial of 487 patients that concluded that preoperative IV iron was no better than placebo in reducing the need for blood transfusion or improving 30-day mortality in anaemic patients undergoing major abdominal surgery.[20]

Postoperative iron deficiency anaemia

- All patients who had major surgery with preoperative anaemia or moderate to severe blood loss should be screened for iron deficiency anaemia postoperatively for a minimum of 3d.
- Early high-dose IV iron is recommended as 1st-line therapy.
- High blood loss requiring blood transfusion may also require postoperative supplementary IV iron.

Further reading

Muñoz M, Acheson AG, Bisbe E, *et al*. (2018). An international consensus statement on the management of postoperative anaemia after major surgical procedures. *Anaesthesia*, **11**, 1418–31. doi:10.1111/anae.14358

Fasting

Preoperative fasting is defined as the restriction of fluid or food intake prior to general anaesthesia (GA) or sedation.

Prolonged periods of fasting are unnecessary and have been associated with deleterious effects, including distress, confusion, dehydration, CVS instability, electrolyte disturbance, PONV, hypoglycaemia, insulin resistance, worse outcomes and morbidity.

Preoperative fasting

The aim of preoperative fasting, by reducing the volume and nature of gastric content, is to mitigate the risk of peri-procedural pulmonary aspiration.

- Patients should receive fasting guidance, including information on the underlying rationale, during the anaesthetic preassessment.[21]
- Fasting times should be minimised, with specific consideration given to the management of routine medication.
- ASA guidance (Table 3.1) is consistent with well-established practice.[21]
- The ongoing administration of clear fluids should be addressed continuously over the course of an operating list. A 2h fast for clear fluids can translate into significantly longer periods in reality.
- In 2018, an international consensus statement was issued, advising a reduced fasting period for clear fluids to 1h (with a maximum volume of 3mL/kg) for children, in the absence of contraindications, undergoing elective procedures[22] (see ➲ pp. 915–16). There is mounting pressure for adult guidelines to adopt this reduced time frame.
- Examples of clear fluids include water, non-particulate juice, black tea or coffee and carbohydrate nutritional drinks, but not alcohol.
- The ASA considers carbonated beverages to be clear fluids, but this is not universally accepted—local guidelines should be followed.
- Non-human milk is considered solid, as when mixed with gastric juices, it congeals. Small amounts (<20%) of milk in tea and coffee divide opinion[23]—local guidelines should be followed.
- Normal gastric emptying of solids is slower and more variable than that of fluids. Light meals (e.g. toast) have faster gastric emptying times than foods with a high-fat or meat content.
- Patients should not have their surgery delayed for chewing gum, sucking a boiled sweet or smoking a cigarette immediately before coming to theatre.[23]
- A vital, if obvious, practical point is to ensure that chewing gum is removed before anaesthesia to prevent it from being aspirated.

Table 3.1 Summary of 2017 ASA guidance for adult preoperative fasting periods

Ingested material	Minimum fast (h)
Clear liquids	2
Human breast milk	4
Light meal and non-human milk	6
Fried fatty food and meat	8

Source: data from *Anesthesiology*. 2017(Mar);**126**(3):376–93.

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Delayed gastric emptying

- Causes: metabolic, e.g. poorly controlled diabetes mellitus (DM), renal failure, sepsis; ↓ gastric motility, e.g. head injury; pyloric obstruction, e.g. pyloric stenosis; gastro-oesophageal reflux; ↑ intra-abdominal pressure, e.g. pregnancy, obesity; opioids; trauma.
- Err on the side of caution when estimating time for an empty stomach in trauma patients. The time taken to return to normal gastric emptying after trauma has not been established and varies, depending upon the degree of trauma and the level of pain.
- Anxiety has not been shown to have any consistent effect on gastric emptying.

Gastric ultrasound

Emerging work on the use of bedside ultrasound to ascertain residual gastric content is promising.[24] It is used to influence modification of the induction technique in the context of urgent or emergent surgery or for those with delayed gastric emptying.[25]

Intraoperative management of the unfasted patient

- Check the patient's fasting status at the time of the procedure.
- If delayed gastric emptying, consider prolonged fasting periods, premedication and/or modification of the anaesthetic induction.
- When preoperative fasting has not been achieved, the benefit of proceeding should be balanced against the risk of pulmonary aspiration. Considerations should include the type and volume of ingested material, as well as the urgency of surgery.[21]
- Consideration of gastric decompression (e.g. via wide-bore orogastric tube (OGT) or NGT) should be made on a case-by-case basis.
- Without a clear indication for an ongoing requirement, any OGT or NGT placed during surgery should be removed before emergence from anaesthesia.

Postoperative resumption of oral intake

- Oral fluids should be offered post-procedurally when patients are fully awake and free from nausea, unless prohibited by specific instructions from the surgeon or anaesthetist.
- If an airway has been topicalised with a local anaesthetic (LA) (e.g. awake tracheal intubation), a patient should remain fasted until airway anaesthesia has worn off and airway reflexes have returned.
- Return to normal diet is a central component of ERAS protocols and postoperative surgical instructions should address this.

Venous thromboembolism prophylaxis

- Venous thromboembolic disease (VTE) is a common source of perioperative morbidity and mortality.[26]
- Pulmonary embolism (PE) is a potentially avoidable cause of postoperative death and typically results from a deep vein thrombosis (DVT) originating from within lower limb venous plexuses.
- DVTs carry their own morbidity related to post-thrombotic chronic venous insufficiency and venous ulceration.
- Without VTE prophylaxis, historical data indicate the incidence of VTE in surgical patients is high, with up to 50% developing a DVT and 10% a PE.[27]

Risk assessment for VTE and bleeding

- The risks of VTE and bleeding are influenced by patient, surgical and anaesthetic factors (Table 3.2).
- Contributing towards the risk of VTE is the Virchow's triad of pathophysiological factors: hypercoagulability, abnormal venous blood flow and endothelial damage.
- In the UK, one of the most commonly used assessments is the Department of Health VTE Risk Assessment Tool.[28] This indicates that all adult surgical patients should be assessed:
  - On hospital admission and at 24h post-admission
  - Each time their clinical situation changes (e.g. intraoperatively, and both immediately and later postoperatively).
- Surgical patients with ≥1 VTE risk factor should be considered for prophylaxis.
- Intermediate- or high-risk patients ideally should have a combination of mechanical and pharmacological prophylaxis.[29]
- Risk scoring tools are available with online risk calculators and recommended prophylactic strategies such as The Caprini Score.[30]
- Patients should be given an explanation of their assessment and the risk vs benefit of any suggested prophylactic strategy.

Prophylactic management

- Prophylaxis should normally be started as soon as possible (usually <14h after a non-elective admission)—unless patient, surgical or anaesthetic considerations preclude this.[29]
- Some hospitals have incorporated VTE prophylaxis into an amended WHO Surgical Safety Checklist to address intra- and postoperative management.
- The type, timing, dose and duration of postoperative VTE prophylaxis should be agreed between the surgeon and the anaesthetist, and ideally prescribed before the patient leaves theatre.
- Prophylactic management include:
  - Non-pharmacological measures—mechanical and inferior vena cava (IVC) filters
  - Pharmacological measures.

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Table 3.2 VTE and bleeding risk factors[28]

VTE risk	Bleeding risk
Patient factors	
Thrombophilia History of VTE (personal or family) Obesity (BMI >30kg/m²) Malignancy (active or treatment) Oestrogen-containing contraceptive* Hormone replacement therapy (HRT)* Pregnancy or <6w postpartum Age >60y Phlebitic varicose veins Dehydration Other significant medical disease	Active bleeding Acquired bleeding disorders (e.g. acute liver failure) Anticoagulant use (e.g. warfarin with international normalised ratio (INR) >2) Acute CVE Thrombocytopenia (platelets <75 × 10⁹g/L) Hypertension >230/120mmHg Untreated inherited bleeding disorders (e.g. haemophilia, von Willebrand disease)
Surgical factors	
Surgery causing reduced mobility Hip fracture Hip or knee replacement Acute inflammatory/intra-abdominal condition	Neurosurgery Spinal surgery Ophthalmic surgery Procedures with high bleeding risk
Anaesthetic factors	
Total anaesthetic time >90min Total anaesthetic time >60min for pelvic or lower limb surgery Critical care admission	Neuraxial technique planned in next 12h Neuraxial technique performed within previous 4h

More than one VTE risk should prompt thromboprophylaxis. The risk of bleeding should be balanced against the risk of VTE when deciding whether to offer pharmacological thromboprophylaxis to surgical and trauma patients.[4]

* For oestrogen-containing contraceptive and HRT, see p. 64.

Source: data from Risk assessment for venous thromboembolism (VTE), Department of Health, https://webarchive.nationalarchives.gov.uk/20130123195034/http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_088215

Non-pharmacological VTE prophylaxis

Graduated compression stockings

- Should provide graduated compression of 14–15mmHg at the calf.
- Contraindicated in peripheral artery disease, peripheral artery bypass grafts, peripheral neuropathy, severe leg oedema, significant limb deformity, local vulnerable skin conditions and acute CVE.[29]
- Skin integrity should be monitored and if compromised, the stockings should be removed and consideration given to the use of intermittent pneumatic compression devices.[29]

Intermittent pneumatic compression devices

- Intermittent pneumatic compression devices compress the leg (35–40mmHg) for ± 10s every minute, promoting venous flow.

- There is some evidence that they are as effective as heparin in reducing the incidence of DVT and are typically used in preference to graduated compression stockings for higher-risk patients.[31]
- They are used for patients with known ↑ risk for DVT and in major surgery and/or prolonged surgery.

IVC filters

- The evidence for the use of IVC filters in patients with a contraindication for pharmacological and mechanical thromboprophylaxis is not robust.[31]
- IVC filter-associated complications often outweigh a potential benefit. They are therefore not routinely used in the perioperative period. However, they could be considered for insertion in high-risk patients (e.g. recent proven DVT with non-deferrable major surgery) when other methods are contraindicated, but only on a case-by-case basis and in discussion with the patient and the multidisciplinary team.

Pharmacological prophylaxis

- Low-molecular weight heparin (LMWH) is a commonly used agent (adjusted for both weight and renal function), e.g. for a 50–100kg patient, enoxaparin 40mg/24h subcutaneously (SC) or 20mg if creatinine clearance (CC) <30mL/min.
- Other agents can be used but are usually surgery- (or patient-) specific and reflect current evidence. These include unfractionated heparin (UFH), rivaroxaban, apixaban, dabigatran, fondaparinux, warfarin and aspirin.[6] Local, national or international guidelines should be followed.
- Anaesthetic assessment should address the timing and management of any pre- and postoperative dosing, specifically with regard to any anaesthetic procedures (e.g. neuraxial or regional techniques).
- If pharmacological prophylaxis is contraindicated, then mechanical prophylaxis in isolation should be commenced (preferably with intermittent pneumatic compression devices).[31]

Paediatrics

(See ➲ Chapter 36 for general paediatric anaesthesia information.)

The majority of paediatric surgical patients do not require VTE prophylaxis. However, the Association of Paediatric Anaesthetists of Great Britain and Ireland (APAGBI) Guidelines[32] recommends that children aged ≥13y should be risk-assessed for VTE (and bleeding) and managed accordingly. Specifically they suggest:

- Children ≥13y and >40kg, with an expected anaesthetic time >60min, should be fitted with intermittent pneumatic compression devices intraoperatively.
- Central venous catheter (CVC) lines are the commonest risk factor for paediatric VTE.
- Other VTE risk factors are similar to those for an adult.
- Paediatric patients are classed as low (no risk factors), moderate risk (one risk factor) or high risk (≥2 risk factors).
- Consider graduated compression stockings in moderate-risk patients (if >40kg and appropriate size).
- High-risk patients should be assessed for potential LMWH.

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Antibiotic prophylaxis

- Antibiotic prophylaxis is administered to patients as part of a care bundle to mitigate the risk of surgical site (wound) infection.[33]
- Other anaesthetic components of this bundle include maintaining patient homeostasis such as normothermia, adequate oxygenation, tissue perfusion and targeted glycaemic control.

Surgical antibiotic prophylaxis[34]

- Antibiotic prophylaxis is recommended before:
  - Clean surgery involving prosthesis or implants
  - Clean-contaminated surgery
  - Contaminated surgery.
- It is not recommended for clean, non-prosthetic, uncomplicated surgery.
- A patient's antibiotic allergy and meticillin-resistant *Staphylococcus aureus* (MRSA) status should be identified during preoperative assessment, and prophylactic antibiotic choices tailored accordingly.
- Best practice is to inform patients preoperatively if they will need antibiotic prophylaxis. They should also be informed postoperatively if they received antibiotics during the operation.
- A course of antibiotic treatment should be given to patients having surgery on dirty or infected wounds or to those with proven or suspected systemic sepsis.

Timing of prophylactic antibiotics

- The WHO recommends that antibiotic prophylaxis is given within 60min of surgical incision.
- One exception is with infected patients where it is planned to take surgical samples prior to administering antibiotics.
- It is recommended that prophylactic antibiotics are administered on starting anaesthesia; however, earlier prophylaxis may be needed for procedures requiring a tourniquet.[34]
- Antibiotics with long infusion durations should be commenced, so that the infusion is complete before whichever is the earlier of knife to skin or tourniquet inflation (e.g. vancomycin and fluoroquinolones are infused over 60–120min).
- Repeat antibiotic doses should be administered for procedures >2–4h (typically where the duration >2 half-lives of the antibiotic) or with associated significant blood loss (>1.5L).[33,35]
- It is important to note that certain patient factors, such as extensive burns or renal failure, may impact on the half-life of the antibiotic.
- Decisions on the choice of antibiotics and redosing should follow local guidelines or the advice of a microbiologist.

Decolonisation

- For procedures where *S. aureus* is a likely cause of surgical site infection, consider preoperative decolonisation, e.g. with a course of nasal mupirocin and chlorhexidine body wash.
- Screening for both MRSA and meticillin-sensitive *S. aureus* can help guide decision-making for decolonisation and subsequent prophylactic antibiotic choice.

Infective endocarditis prophylaxis[36,37]

- Infective endocarditis (IE) is rare but has a high mortality rate.
- Guidelines have not been in agreement about antibiotic prophylaxis in the prevention of IE.
- The European Society of Cardiology and the American Heart Association have each restricted prophylactic antibiotics to those patients with the highest risk of adverse outcomes.
- In contrast, NICE went even further and advised against prophylactic antibiotics altogether. The exception to this is patients at risk of IE who are undergoing a GI or genitourinary procedure at a site of suspected or proven infection should receive antibiotics that cover organisms causative of IE.
- There is no global consensus for patients traditionally considered high risk. Box 3.1 outlines cardiac conditions considered at risk of IE by NICE.

Box 3.1 Cardiac conditions at risk of IE[36]

- Previous IE
- Acquired heart valve disease with stenosis or regurgitation
- Heart valve replacement
- Hypertrophic cardiomyopathy
- Structural congenital heart disease, excluding isolated atrial septal defect (ASD), fully repaired ventricular septal defect (VSD) or fully repaired persistent ductus arteriosus (PDA) and closure devices that are thought to be endothelialised

General recommendations

- Antibiotics indicated for surgical procedures should be given as normal.
- For 'infected surgery' in patients at risk of IE, appropriate antibiotic prophylaxis should be administered.
- In patients at high risk of IE, the decision to provide prophylactic antibiotics for dental treatment should be made by treating clinicians after discussion with the patient.
- Administration of antibiotics is not risk-free, and local protocols should be followed.

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Perioperative medications

Prescribed, over-the-counter, herbal and recreational medications need to be carefully considered at the time of the preoperative assessment. Individualised plans that take into account patient and surgical factors (and based on local guidelines) should be made and clearly communicated (in written form) to the patient and teams involved in their perioperative care.

Some specific considerations include:

- Anticoagulants and antiplatelet therapies (see ➔ pp. 269–79)
- Diabetic medications (see ➔ pp. 216–21)
- Antipsychotics (see ➔ p. 334)
- Renin–angiotensin system antagonists (see ➔ Chapter 5, in particular ➔ p. 107, p. 113 and p. 116)
- β-blockers (see ➔ Chapter 5, in particular ➔ p. 107, p. 113 and p. 116)
- Antiepileptics (see ➔ pp. 300–2) and anti-Parkinson's medications (see ➔ pp. 292–5).

Statins

- Statins should be continued due to their effects on improved endothelial function, enhanced stability of atherosclerotic plaques and reduced vascular inflammation, causing a decrease in perioperative cardiac events.[38]

Oestrogen therapy

- NICE guidance[39] recommends that women consider stopping oestrogen therapy (contraception or HRT) 4w before elective surgery due to the theoretical ↑ risk of VTE.
- There is, however, no consensus on the perioperative advice to give patients due to insufficient evidence for ↑ perioperative VTE risk and the risks of unwanted pregnancy, or the recurrence of troublesome menopausal symptoms. These should be considered in a patient's decision-making process.
- Patients should be advised on a case-by-case basis. If contraception is stopped, advice must be given about alternative contraceptive measures and discussions documented.

Herbal medicines and anaesthesia

- About 5–14% of patients take perioperative herbal medication.
- Of these, 70% do not disclose this fact to their doctor.
- Content of herbal remedies may vary dramatically.
- Most herbal remedies are harmless, but some may have important consequences for anaesthesia (Table 3.3).
- The ASA recommends that patients stop herbal medications 2w before surgery.

Table 3.3 Herbal medicines which may affect anaesthesia

Drug (common name)	Potential uses	Perioperative concerns
Echinacea (purple coneflower root)	Boosts immune system (stimulates cell-mediated immunity)	Immunosuppression in long-term use. Avoid in transplant surgery. May cause hepatoxicity[40]
Ephedra (Ma huang)	Promotes weight loss. Used for asthma and bronchitis (direct and indirect sympathomimetic)	↑ risk of cardiac arrhythmias, hypertension, CVE and MI.[41] May cause ventricular arrhythmias with halothane. Life-threatening interaction with MAOIs[40]
Garlic	Treatment of hypertension, hyperlipidaemia and atherosclerosis	↑ risk of bleeding due to antiplatelet effects[42]
Ginkgo (duck foot tree, maidenhair tree, silver apricot)	Used to improve mental alertness (antiplatelet activity)	↑ risk of bleeding when combined with anticoagulant and antithrombotic medication[43]
Ginseng (American, Asian, Chinese, Korean ginseng)	Aimed at increasing physical and mental stamina	May lower blood concentration of warfarin. May cause hypoglycaemia.[40] May see tachycardia and hypertension
Kava-kava (intoxicating pepper, kawa)	Anxiolytic and muscle relaxant	Can increase sedative effect of anaesthetic [40]
St John's wort (amber, goat weed, hardhay, Hypericum, klamatheweed)	Antidepression, anxiolytic and used in sleep disorders (inhibits neurotransmitter reuptake)	Induction of cytochrome P450 liver enzymes. Decreases serum digoxin levels.[40] Avoid in transplant surgery.[44]Associated with hypertensive crisis[45]
Valerian (All heal, garden heliotrope, vandal root)	Sleeping aid	Potentiates anaesthetic agents[40]

Further reading

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Premedicants

- Premedicants are given before the induction of anaesthesia.
- The aims of premedication are to improve patient comfort and reduce perioperative risk.
- Changes in anaesthetics agents and short postoperative stays have led to the decline in premedicant prescription.
- Commonly used premedicants are outlined below. (See ➲ p. 917 for paediatric premedicants.)

Analgesics

- Paracetamol. Can be considered in all patients unless contraindicated. Single dosing >1g for higher body weight patients is safe if max 4g/day. PO dose 1–2g.
- NSAIDs. Commonly prescribed (avoid in patients with a history of gastric ulceration and renal impairment, and caution in older patients). Suggested PO doses: ibuprofen 400mg, diclofenac 50–100mg.
- Topical LAs (e.g. prilocaine, EMLA®), often used in paediatrics (see ➲ p. 916).

Anxiolytics

(See ➲ p. 419 for sedation.)

- Benzodiazepines. Most commonly used anxiolytics. Longer-acting agents may delay recovery. Effects range from mild anxiolysis to deep sedation, depending on dose. Temazepam PO 10–20mg, lorazepam PO 1–2mg.
- Opioids. Strong analgesic agents that produce sedation more than anxiolysis. Less commonly used as premedicants. PO/intramuscular (IM) dose of morphine10–20mg.
- Ketamine. Produces rapid sedation and analgesia. Particularly useful in reduced/non-compliant patients. Co-administration with benzodiazepines may reduce hallucinations. Suggested doses: PO 5–7mg/kg, IM 2–5mg/kg, IV 0.5–1mg/kg.

Antacids and prophylaxis of aspiration pneumonitis

- Sodium citrate (PO 30mL of 0.3M solution). Often given prior to GA for Caesarean section (CS), increases pH but does not decrease volume of gastric contents.
- Proton pump inhibitors decrease volume and increase pH of gastric contents. Also used to decrease gastric irritant effects of NSAIDs. Omeprazole PO 20–40mg.
- Histamine (H2) receptor antagonists decrease gastric acid secretion. Ranitidine can be given IV or PO (IV 50mg, diluted in 20mL of 0.9% sodium chloride, given over 2min; PO 150mg).
- Metoclopramide increases gastric emptying and decreases gastric volume, and also has antiemetic effects. Given as 10mg PO/IV.

Antisialagogues

- Anticholinergic. Glycopyrronium bromide often used prior to awake tracheal intubation. Dose: IV 200–400 micrograms.

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Chapter 4

At-risk populations

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Obesity introduction

The prevalence of obesity is increasing worldwide and so the number of obese surgical patients increases. Recent UK government statistics, published in 2019, suggest that the majority of adults are either overweight or obese (64%).[1] Obesity prevalence ↑ steeply between 1993 and around 2000, with a slower rate of increase after that. In 2017, the proportion of adults who were obese in the UK was 29%. Obesity is more common in women (30%) than in men (27%). Morbid obesity has also ↑, with <1% in 1993, to nearly 4% in 2017.[1] The rise in obesity applies not only to adults, but also to children. Globally, the obesity rate in the United States (US) is the highest at 40%; however, countries such as Japan and Korea have rates lower than 10%.[2]

Anaesthesia and surgery may entail considerable risk for obese patients. Obesity is a multisystem disorder, particularly involving the respiratory system and CVS; therefore, a multidisciplinary approach is required.

Definitions

Obesity is defined by the BMI. BMI is defined as weight (in kg) divided by the height (in m squared) (Table 4.1).

Table 4.1 BMI definitions

BMI (in kg/m^2)	Obesity class
<19.9	Underweight
20–24.9	Normal
25–29.9	Overweight
30–39.9	Obese
40–49.9	Morbidly obese
50–59.9	Super obese
60–69.9	Super super obese
>70	Hyperobese

Common comorbidities in obesity

Respiratory system

- OSA (see ➲ pp. 73–5; ➲ p. 186).
- Obesity hypoventilation syndrome (OHS):
  - OHS is at the severe end-stage of OSA in obese patients, characterised by diurnal variation in ventilation and arterial partial pressure of CO_2 ($PaCO_2$) >5.9 kPa. Sensitivity to CO_2 is reduced due to loss of central drive, leading to hypoventilation.
  - O_2 consumption is ↑ by metabolically active adipose tissue and the workload of supporting muscles, with associated increase in CO_2 production.
- Functional residual capacity (FRC) is reduced in the awake obese patient and decreases significantly following induction, which may encroach upon the closing capacity. Pulmonary compliance is ↓ due to a heavy chest wall and a splinted diaphragm. O_2 desaturation occurs rapidly in the obese, apnoeic patient.
- Obese patients may have clinical signs that appear to be asthma. In many cases, the bronchoconstrictive symptoms experienced by the obese are often due to airway closure without affecting the calibre of the airways.[3] This increase in airway closure is a direct effect of obesity and is not due to any intrinsic lung disease. It is not improved by bronchodilator therapy.

Cardiovascular system

- The excess adipose tissue exerts increasing metabolic demands on the CVS, leading to ↑ cardiac output (CO), myocardial demand and arterial pressure, and ↑ blood volume.
- Hypertension is more prevalent in obesity. This results in LV dilation and hypertrophy, ultimately leading to elevated LV end-diastolic pressure (LVEDP).
- Blood volume is ↑ and this, coupled with ↑ LVEDP, increases the risk of developing heart failure.
- Right ventricular (RV) failure (see ➲ Chapter 5) is common in morbidly obese patients. It can be due to LV changes, OSA and/or OHS, which leads to pulmonary vasoconstriction and pulmonary hypertension. It is difficult to diagnose, requires investigation and increases the risk of surgery.
- Obese patients are at ↑ risk of arrhythmias.
- In addition to all these factors, the ↑ incidence of DM and reduced level of activity increase the risk of developing IHD in the obese population.

Endocrine system

Insulin resistance and diabetes

- Insulin resistance with hyperinsulinaemia is characteristic of obesity and is present before the onset of hyperglycaemia.
- Hyperinsulinaemia is caused by both insulin resistance and an impairment in glucose removal.
- Peripheral resistance to the effects of insulin on glucose and fatty acid utilisation leads to type 2 diabetes.
- Many obese patients will be on complex antidiabetic drug regimens. Normal protocols may not work in the obese, and their insulin requirements appear to be greater than those of the non-obese.

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- Bariatric surgery, particularly gastric bypass, often has a profound effect on type 2 diabetes control immediately postoperatively, before any weight loss occurs. Complete resolution of type 2 diabetes occurs within a year in up to 80% of patients following gastric bypass.

Metabolic syndrome

- This is defined as the occurrence of several metabolic risk factors for both type 2 diabetes and CVS disease (abdominal obesity, hyperglycaemia, dyslipidaemia and hypertension). The National Cholesterol Education Program/Adult Treatment Panel III (NCEP/ATP III) is the most widely used, and its criteria include the presence of any three of the following five traits:
  - Abdominal obesity, defined as a waist circumference in men ≥102cm and in women ≥88cm
  - Serum triglycerides ≥1.7mmol/L or drug treatment for elevated triglycerides
  - Serum high-density lipoprotein (HDL) cholesterol <1mmol/L in men and <1.3mmol/L in women, or drug treatment for low HDL cholesterol
  - BP ≥130/85mmHg or drug treatment for elevated BP
  - Fasting plasma glucose ≥5.6mmol/L, or drug treatment for elevated blood glucose.
- Hyperinsulinaemia and hyperglycaemia associated with obesity, along with adipokines, may lead to vascular endothelial dysfunction, an abnormal lipid profile, hypertension and vascular inflammation, all of which promote the development of atherosclerotic disease.[4]
- The prevalence of obese patients admitted to critical care is increasing. Outcome data show predominantly either equal or lower mortality in obese patients than in normal-weight, critically ill patients.[5] This phenomenon—the obesity paradox[6]—has been observed in obese patients with heart failure and sepsis too. There is also no evidence that surgical outcomes in obese patients are worse than in normal-weight patients.

Gastrointestinal

These patients are more likely to have raised intra-abdominal pressures and are at greater risk of developing a hiatus hernia, contributing to an ↑ risk of reflux and aspiration.

Venous thromboembolic disease

(See also ➲ p. 59.)

There is a significantly ↑ risk for DVT and PE in obese subjects. In addition, there is an ↑ risk of recurrent VTE once anticoagulation treatment has been withdrawn.[7]

- Obesity also appears to contribute to further increasing the risk of VTE in a number of other settings:
  - Smoking and age in patients with Factor V Leiden mutation
  - Long-duration air travel
  - Women taking oral contraceptives.

Obstructive sleep apnoea

Sleep apnoea[8] is defined as the cessation of airflow at the mouth and nose for at least 10s during sleep. Sufferers develop intermittent respiratory arrest and hypoxaemia during rapid eye movement (REM) sleep. Respiration resumes due to hypoxic stimulation.

Apnoeas may be obstructive, central or mixed. OSA, the most prevalent (85%), occurs commonly in patients with obesity and results from obstruction of the upper airway. Central apnoea (5%) is due to intermittent loss of respiratory drive.

General considerations

- OSA patients have an ↑ perioperative risk. This is likely due to ↑ upper airway collapse and OSA-related comorbidities.
- OSA is an independent risk factor for serious neurocognitive, endocrine and CVS morbidity and mortality in all age groups.
- In children, OSA is most commonly associated with adenotonsillar hypertrophy, but the severity of OSA is not always proportional to the size of the tonsils and adenoids.
- Patients with OSA are at risk of perioperative airway obstruction and respiratory failure while under the effects of sedatives and opioid analgesics.

Diagnosis and treatment

- OSA is often diagnosed on history and examination. Overweight, middle-aged men who present with snoring and periods of apnoea will almost certainly have OSA. However, for standardisation and qualitative diagnosis, more formal investigations are required.
- The gold standard for diagnosis is polysomnography (PSG). PSG is a scarce, lab-based, labour-intensive and expensive study.
- During PSG, recordings of ECG, electroencephalogram (EEG), eye movements and electromyogram (EMG) are taken. Respiratory effort, oronasal airflow, peripheral SpO_2 and body position are commonly also recorded. After a night's sleep, the recordings are analysed in 30s intervals called epochs. The stages of sleep are recorded with help of the EEG. The apnoea–hypopnoea index (AHI) and oxygen desaturation index (ODI) are calculated, along with the arousal index and level of sleep disturbance.
- The AHI is the number of apnoeas per hour of sleep. The severity of OSA can be classified according to the AHI:
  - AHI <5—normal
  - AHI ≥5, but <15/h—mild
  - AHI ≥15, but <30/h—moderate
  - AHI ≥30/h—severe.
- The ODI is the number of times per hour of sleep that the blood O_2 level drops ≥4% from the baseline. The severity of OSA has been related to the ODI with the same criteria as with the AHI above, with severe OSA relating to an ODI ≥30/h.
- The numerous monitors can make performing PSG in children difficult. Compared to adults, due to developmental and physiological differences, children require age-adjusted scoring and interpretation. For example, obstructive apnoeas and hypopnoeas are scored if they are at least two breaths' duration, even if they are <10s duration.

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- Home sleep apnoea testing is accessible and cost-effective, and includes overnight SpO_2 (giving the ODI) or SpO_2 and airflow and abdominal movement monitoring (giving the ODI/AHI).
- Several tools are available to assess OSA. Commonly used are the Epworth Sleepiness Scale and the STOP-Bang assessment (Box 4.1).
- Patients with significant sleep apnoea should be treated with continuous positive airway pressure (CPAP) overnight using a nasal or face mask.

Box 4.1 STOP-Bang assessment

Score (<5, low risk of OSA; ≥5, high risk of OSA):

- Snoring loudly
- Tired: daytime somnolence
- Observed apnoeic episodes
- Pressure: hypertension
- BMI >35kg/m²
- Age >50
- Neck circumference >15.75in
- Gender: ♂

Adapted with permission from Wolters Kluwer Health, Inc.:Chung F, *et al*. STOP Questionnaire: A Tool to Screen Patients for Obstructive Sleep Apnea, *Anesthesiology* 2008:**108**(5);812–21, ©2008. https://pubs.asahq.org/anesthesiology/article/108/5/812/8377/STOP-QuestionnaireA-Tool-to-Screen-Patients-for

Perioperative consideration

Preoperative assessment

- OSA is undiagnosed in 80% of patients. Preoperative evaluation should include a review of previous medical records, a history from the patient and/or family and a physical examination. In a recent Canadian study, in 267 preassessed patients with moderate to severe OSA, 92% and 60% were not diagnosed by their surgeons and anaesthetists, respectively.[9]
- Check for airway difficulty with previous anaesthetics, hypertension and other CVS problems. Use a screening tool such as STOP-Bang.
- OHS is the extreme end of OSA. These patients are morbidly obese, have significant daytime somnolence and breathlessness and are particularly sensitive to anaesthesia and opioids. It is important to identify them preoperatively.
- Note any current treatment and compliance such as with CPAP.
- Day surgery may be reasonable, depending on OSA severity, comorbidities, analgesic requirements and nature of the proposed surgery.
- In patients with a new diagnosis of OSA treated with CPAP, it is advisable to delay surgery for 1–3mo. However, consideration for the urgency of surgery and patient preference should be given.

Investigations

- In known OSA, perform FBC (polycythaemia), SpO_2 and ECG (right heart strain). If the ECG shows right heart strain, echocardiography is indicated to exclude RV hypertrophy.
- If hypoxic on air, consider performing an arterial blood gas (ABG).

Conduct of anaesthesia

- Avoid night sedation and sedative premedication.
- Anticipate that mask ventilation and intubation may be difficult, and prepare for this.
- A local or regional anaesthetic technique is preferred where suitable. Use short-acting anaesthetic/analgesic agents where possible.
- GA preceded by preoxygenation, with tracheal intubation and mechanical ventilation, is preferred to sedation or GA with spontaneous ventilation (SV).
- Give NSAIDs and paracetamol.
- At the end of the procedure, ensure NMB is fully reversed and extubate fully awake in the sitting position.

Postoperative care

- Nurse the patient sitting up whenever possible.
- High-risk patients may require admission to HDU/intensive care unit (ICU).
- Administer supplementary O_2, and ensure continuous SpO_2 monitoring on the ward.
- Unless contraindicated, CPAP should be administered continuously to patients who were using it preoperatively.
- Aim to maintain the O_2 saturation that the patient had preoperatively, titrating O_2 to the minimum required.
- Minimise opioid analgesics. If used, consider postoperative location, level of staff training and need for ↑ monitoring.

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Airway considerations in obesity

The RcoA's National Audit Project 4 (NAP4) report in 2011 indicates airway difficulties in the obese patient are twice those in the general population, and even higher in the morbidly obese. Problems included an ↑ frequency of aspiration, difficult bag–mask ventilation and tracheal intubation and airway obstruction during emergence or recovery. When rescue techniques were necessary in obese patients, they failed more often than in the non-obese. Obesity needs to be recognised as a risk factor for airway difficulty, and plans modified accordingly.[10] A BMI >30 is an independent predictor of difficult bag–mask ventilation.

The Society for Obesity and Bariatric Anaesthesia (SOBA) has issued guidance on how to manage these patients. This includes:[11]

- Preoxygenation and intubation in the obese patient should be performed with the patient in the head-up or ramped position. This improves the efficacy of preoxygenation, maximises the time before desaturation, reduces the risk of reflux and reduces the incidence of difficult intubation close to that of the non-obese population.
- Obesity is a weak risk factor for difficult intubation, and predictors of difficulty are generally the same as for normal-weight patients.
- ↑ work of breathing and early airway closure occurring during tidal ventilation suggest that obese patients should not be allowed to breathe spontaneously for anything other than the shortest procedure. These patients will desaturate rapidly, so the time interval from induction of anaesthesia to assisted ventilation of the lungs should be minimised. Caution should be exercised using high-flow O_2 delivery systems to increase the apnoea time of obese patients, as the evidence of safety is lacking.
- Caution should be taken with the use of supraglottic airway devices (SADs) in patients with a BMI >35kg/m^2.
- Tracheal extubation in an obese patient should likewise be performed in the head-up position, with the patient awake.
- Problems usually occur because of poor planning, choice of technique or inadequate preparation. Morbidly obese patients should be managed by an experienced anaesthetist as part of an experienced team.

Videolaryngoscopy has a high success rate in morbidly obese patients. Second-generation SADs should be available in case of failed intubation.

Practicalities with obese patients

The patient's high BMI should be discussed at the safety brief, along with the specific preparations required:

- Trained staff
- Number of staff
- Manual handling
- Operating table and adjuncts
- Monitoring
- Patient position
- Airway management.

Patients, if able, can walk directly into theatre and anaesthesia is induced on the operating table, reducing manual handling risk. Operating tables with the appropriate maximum weight allowance must be used.

- A hover mattress underneath the patient is very helpful to facilitate patient transfer post-surgery.
- There must be enough trained and experienced staff in theatre to assist with moving the patient, should it become necessary.
- Standard monitoring should include a correct-sized BP cuff. This may be impossible in the normal upper arm position in many patients. Using a forearm cuff is virtually always possible instead and, although not validated, is effective and used by many bariatric anaesthetists as standard.
- Invasive arterial catheters are rarely needed, unless for CVS indications.
- Venous cannulation is often difficult. CVCs may be necessary.
- Calf compression devices should be used, and particular care given to pressure areas to prevent sores and nerve injury.

Patient positioning is very important. 'Sniffing the morning air' position may be difficult to achieve due to the large soft tissue mass of the neck and chest wall. A ramped position is ideal, and many adjuncts to achieve this are commercially available.

- Preoxygenation in the head-up position will slow the rapid desaturation that can occur when supine.
- Awake fibreoptic intubation (AFOI) should be considered in any patients who have a history or clinical signs suggestive of airway problems. If the patient is preoxygenated to an end-tidal O_2 (ETO_2) >90%, then minimal bagging will be needed while waiting for the muscle relaxant to take effect.
- High-flow O_2 delivery systems can be used safely for preoxygenation but offer no advantage to face mask oxygenation to an ETO_2 >90%.
- Use of short-acting anaesthetic agents, such as remifentanil, or a total intravenous anaesthesia (TIVA) technique with propofol and remifentanil, aids rapid recovery from anaesthesia and minimise postoperative hypoventilation and hypoxaemia.
- Monitoring of NMB is essential, as incomplete reversal of neuromuscular-blocking agents (NMBAs) is problematic in the obese.
- The use of rocuronium should be considered because it can be fully reversed with sugammadex.

A single sheet guideline (Fig. 4.1) on anaesthesia for the obese patient has been produced by the SOBA.

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Anaesthesia for the Obese Patient

Society for Obesity and Bariatric Anaesthesia

Pre-operative Evaluation

Red Flags

- Poor functional capacity
- Abnormal ECG
- Uncontrolled BP, CCF or IHD
- SpO2 <94% on air
- If bicarbonate >27, OHS likely
- Previous DVT/PE
- STOP-BANG ≥5
- OS-MRS >3
- Metabolic Syndrome
- High NSQIP ACS Risk

Yes →

Consider:
- Preoperative CPAP
- Blood Gases/Sleep Studies
- Echocardiogram
- Cardiorespiratory referral
- Experienced Anaesthetist
- Book HDU Bed

No →

- May be suitable for day-case surgery

Central Obesity
(waist > half height)
- Difficult airway/ventilation more likely
- Greater risk of CVS disease/thrombosis
- Higher risk of metabolic syndrome

Peripheral Obesity
(Fat outside body cavity)
- Less co-morbidity
- Lower risk

OS-MRS Calculator
tools.farmacologiaclinica.info

NSQIP ACS Risk Calculator
riskcalculator.facs.org/RiskCalculator

STOPBANG Calculator
www.stopbang.ca

Intra-operative Management

Suggested Equipment:
- Suitable bed/trolley and operating table
- Positioning
- Video laryngoscopy
- Table extensions/arm boards
- Forearm cuff or large BP cuff
- Device or equipment for ramping
- Step for anaesthetist
- Difficult airway equipment
- Videolaryngoscope
- Ventilator capable of PEEP & pressure modes
- Hover mattress or equivalent
- Intravenous access and vascular needles
- Ultrasound machine
- Appropriately sized calf compression devices
- Depth of anaesthesia monitoring
- Neuromuscular monitoring
- Sufficient staff to move patient

Ramping

- Tragus level with sternum
- Reduces risk of difficult laryngoscopy
- Improves ventilation and pre-oxygenation

Anaesthetic Technique:
- Consider premed antacid & analgesia
- Consider gastrokinetic control
- DVT prophylaxis
- Self-position on operating table
- Preoxygenate and intubate in ramped/sitting position
- Consider CPAP and HFNO
- Minimal induction to ventilation time
- Commence maintenance promptly
- Tracheal intubation recommended
- Avoid non-depolarising NMBs
- Avoid prolonged ventilation, use PEEP
- Use short-acting inhalationals or TIVA
- Short-acting opioids & multimodal analgesia
- PONV prophylaxis
- Ensure full NMB reversal
- Extubate and recover sitting up

Lean Body Weight: This exceeds ideal body weight in the obese and plateaus at:
- ≈100kg for a man
- ≈70kg for a woman

Ideal Body Weight: Broca formula
- Men: height (in cm) -100
- Women: height (in cm) -105

If in doubt, titrate and monitor effect

Suggested dosing for anaesthetic drugs

Lean Body Weight (Males Max 100Kg Females Max 70Kg)	Adjusted Body Weight (Ideal plus 40% excess)	Total Body Weight
• **Propofol induction** • **Thiopentone** • **Fentanyl and Alfentanil** • **Morphine** • **Non-depolarising NMBDs** • **Paracetamol** • **Local Anaesthetics**	• **Propofol Infusion** • **Neostigmine** (max 5mg) • **Sugammadex** (read pack insert) • **Antibiotics**	**Suxamethonium** **LMWHs** (titrate dose with Xa levels)

Post-operative Care

PACU discharge:
- Usual discharge criteria should be met
- SpO_2 should be maintained at pre-op levels with minimal O_2therapy
- No evidence of hypoventilation

OSA or Obesity Hypoventilation Syndrome:
- Sit up and avoid sedatives and post-op opioids
- Reinstate patient's own CPAP if applicable with additional time in recovery until free of apnoeas without stimulation
- Patients untreated, intolerant of CPAP or ineffectively treated (persistent symptoms) are at risk of hypoventilation
- In these cases, IV opioids should be avoided but where necessary, patient should have continuous SpO_2 monitoring and level 2 care must be considered

General good ward level practice includes:
- Multimodal analgesia
- Caution with long-acting opioids and sedatives
- Early mobilisation
- Robust thromboprophylaxis regime
- Experienced Consultant Review

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Fig. 4.1 Anaesthesia for the obese patient, SOBA. The SOBA Single Sheet Guideline. 2019 edition, released Dec 2019. Modified with permission from SOBA single sheet Guideline, © SOBA UK 2020. https://www.sobauk.co.uk/guidelines-1

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Pharmacology in obese patients

- Recommendations on which weight to use for calculating drug doses in various classes of drug are found on the SOBA one-page guideline (see also ➔ p. 79).
- Lean body weight is a part of the body composition that is defined as the difference between total body weight and body fat weight. This exceeds the ideal body weight (IBW) in the obese and plateaus at 100kg for a man and at 70kg for a woman.
- The IBW in kg can be calculated by the Broca formula:
  - Man: height in cm minus 100 ± 10%
  - Woman: height in cm minus 105 ± 15%.
- Volume of distribution for drugs is altered due to a smaller proportion of total body water (TBW), a greater proportion of adipose tissue, ↑ lean body mass and ↑ blood volume and CO.
- Hydrophilic drugs (e.g. competitive neuromuscular blockers such as rocuronium, vecuronium and atracurium) have similar absolute volumes of distribution, clearance and elimination half-lives. Base the dose on the lean body weight.
- Lipophilic drugs (e.g. thiopental, propofol, opioids and benzodiazepines) have ↑ volumes of distribution, normal clearance and ↑ elimination half-lives. Titrate to CO, which equates to the lean body weight in a fit patient.
- ↑ plasma cholinesterase activity. Suxamethonium dose should be based on the total body weight to a maximum of 200mg.

Suxamethonium may appear to be the NMBA of choice for induction of anaesthesia in obese patients,[12] due to their reduced safe apnoea time; its rapid onset allows rapid tracheal intubation. However, one study demonstrated that the use of suxamethonium was associated with more rapid desaturation, when compared to rocuronium, for RSI.[13]

Thromboprophylaxis in obesity

(See also ➔ p. 59; ➔ p. 72.)

Obesity and surgery are known risk factors for VTE, but there is limited information about the independent effects of obesity on the incidence of postoperative VTE. The Million Women Study[14] concluded that VTE risk increases with increasing BMI and the associated excess risk is much greater following surgery than without surgery. In the UK, NICE has offered guidance on VTE prophylaxis in its publication *Venous thromboembolism in over 16s: reducing the risk of hospital-acquired deep vein thrombosis or pulmonary embolism*, last updated in August 2019.[15]

Start mechanical VTE prophylaxis on admission. Choose any one of:

- Antiembolism stockings (thigh or knee length)
- Intermittent pneumatic compression devices (thigh or knee length).

Continue mechanical VTE prophylaxis until the patient no longer has significantly reduced mobility.

Add pharmacological VTE prophylaxis for patients who have a low risk for major bleeding, taking into account individual patient factors and according to clinical judgement.

This should continue until the patient no longer has significantly reduced mobility (minimum 7d). Some bariatric surgical centres in the UK use extended prophylaxis for up to 3w and others use a higher dosage. There has been no guidance on the duration or dosing for the obese. Therefore, local guidelines must be consulted when considering VTE prophylaxis in this group.

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The malnourished patient

Malnutrition affects a large proportion of patients with chronic disease—25% of those with COPD and 80% of patients with pancreatic or head and neck malignancy.[16,17] It affects many hospitalised patients in the western world, as well as being a major global issue.[18]

Definition: imbalance between supply of essential nutrients and energy and body's requirements for cellular growth, maintenance and function.[18]

It is associated with ↑ morbidity and infection, poor wound healing, pressure ulceration, prolonged hospital stay, an ↑ incidence of ICU requirement, readmission to hospital and mortality.[16] The causes of malnutrition are outlined below (Table 4.2).[17,18]

Table 4.2 Causes of malnutrition

Starvation	Environmental: poverty, food scarcity, poor access, war, natural disasters Patient: inability to eat (e.g. maxillofacial deformities)
Cachexia	COPD, heart failure, cancer (e.g. pancreatic, head and neck), chronic kidney disease, liver disease, rheumatoid arthritis, Alzheimer's disease, cystic fibrosis, infectious diseases
Mental health	Depression, schizophrenia, anorexia nervosa
Absorption problems	Inflammatory bowel conditions, short gut syndrome, coeliac disease, hyperemesis, intestinal worms
Critical illness	Post-surgery, ICU admission, burns

(See also Obesity introduction, p.70.)

Cachexia related to chronic illness or malignancy

Excessive weight loss in the setting of ongoing disease, with disproportionate muscle wasting. Highly predictive of mortality. ↑ proinflammatory cytokines[19] lead to ↓ muscle protein synthesis, ↑ proteolysis, ↑ lipolysis, a negative energy balance and weight loss. Not corrected by nutritional support alone. Affects multiple organ systems. Patients require careful assessment and management (Boxes 4.2 and 4.3).[19,20]

Complications[18]

- Central nervous system (CNS): ↓ cognitive function, fatigue, weakness
- Metabolic/GI: gastric distension and delayed emptying time. ↑ cortisol and catecholamines. Hypoglycaemia when BMI falls <13kg/m^2
- Musculoskeletal: abnormal bone density and ↑ risk of fractures, hypothermia, ↓ muscle mass
- Respiratory: ↓ muscle function and strength, spontaneous pneumothorax
- Haematological: pancytopenia (anaemia and immunodeficiency)
- CVS: ↓ CO and LV ejection fraction (EF), ↓ BP, ↓ heart rate (HR), loss of cardiac muscle, mitral prolapse, ↑ corrected QT interval, ↑ vagal tone, ST depression/T wave inversion
- Renal: ↓ glomerular filtration rate (GFR), proteinuria, TBW higher, ↑ urea.

Box 4.2 Diagnostic criteria for cachexia

- Unintentional weight loss ≥5%
- BMI:
  - <20kg/m^2 in those aged <65y
  - <22kg/m^2 in those aged ≥65y
- Albumin <35g/L
- Low fat-free mass
- Evidence of cytokine excess, e.g. ↑ C-reactive protein (CRP)

Reproduced from Morley JE *et al*. (2006). Cachexia: pathophysiology and clinical review. *American Journal Clinical Nutrition*, **83**, 735–43 with permission from Oxford University Press.

Box 4.3 European Society for Clinical Nutrition and Metabolism risk factors for severe undernutrition

At least one of the following:

- Weight loss >10–15% within 6mo
- BMI <18.5kg/m^2
- Subjective Global Assessment Grade C (severely malnourished)
- Serum albumin <30g/L (no hepatic/renal dysfunction)

Source: data from Hirose K, *et al*. (2014). Perioperative management of severe anorexia nervosa. *BJA*, **112** (2), 246–54. https://www.espen.org/files/ESPEN-guideline_Clinical-nutrition-in-surgery.pdf

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Anaesthetic approach[18]

Preoperative

- Nutritional assessment:
  - History: disease, infections, surgery, recent weight loss, caloric intake, relevant drugs
  - Examination: appearance, hydration status, CVS examination, BMI
  - Investigation: bloods (FBC, urea and electrolytes (U&E), liver function tests (LFTs), calcium (Ca^{2+}), phosphate, magnesium (Mg^{2+}), glucose, transferrin, albumin), urine (protein and ketones), ECG, echo if appropriate
  - Tools: Nutritional Risk Assessment Scale, Subjective Global Assessment.[5] Practically, these can be time-consuming.
- Nutritional optimisation where possible, especially in leucopenia or hypoalbuminaemia. In severe malnutrition (Box 4.3), 7–14d of preoperative parenteral nutrition is advisable, especially if gut function is unlikely to return to normal postoperatively.[16,20] Only consider a delay to surgery if severe malnutrition is present.[16]
- Evidence suggests that preoperative carbohydrate drinks preserve insulin sensitivity postoperatively and reduce length of stay. Limit perioperative fasting where possible (induces metabolic stress and impairs mitochondrial function and insulin sensitivity)[16].
- Correct electrolytes, including phosphate. Hydrate the patient.
- Monitor and maintain blood glucose.
- Prokinetic and antacid recommended. Consider NGT.

Intraoperative

- Monitoring:
  - Have a low threshold for invasive cardiac monitoring (susceptibility to cardiac arrhythmia or collapse).
  - Consider continuous central temperature monitoring.
  - Consider calibrated neuromuscular monitoring to minimise muscle relaxant dose and monitor for prolonged effect.
- Venous access may be challenging. Central access may be required for monitoring and nutritional support postoperatively.
- Induction may cause CVS collapse if underfilled.
- Rapid sequence induction (RSI) recommended. Treat as having a full stomach.
- Protect pressure areas and exposed nerves (reduced muscle mass and soft tissue).
- Bony fragility; take care when moving/positioning.
- Avoid hypothermia with warm fluids, forced air warming blanket, heat and moisture exchanger (HME).
- Avoid hyperventilation—can lower potassium (K^+) further.

Pharmacology

- Pay attention to patient weight, lean and total body mass, albumin and volume of distribution. Adjust doses appropriately.
- Muscle bulk may be reduced (reduce water-soluble drug doses).
- Consider regional techniques to spare opioids and other drugs metabolised by the liver.

Postoperative

- Extubation may be difficult (respiratory weakness and impaired upper airway reflexes).
- Postoperative nutritional support: encourage early enteral feeding.
- Correct pH and electrolytes. Limit excessive fluids and opioids. Prokinetics are recommended.
- Have a low threshold for ICU if available.
- Refeeding syndrome is a risk (see also p. 87).
- Parenteral nutrition is reserved for those who cannot meet their calorific needs within 7–10d postoperatively (GI dysfunction, ileus, bowel discontinuation).

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Anorexia nervosa

Chronic, severe, multisystem disorder with the highest morbidity and mortality rate of any psychiatric disorder (up to 20%). BMI <17.5kg/m² is a diagnostic criterion. Severe cases have BMI <13kg/m².[20]

Complications

- Metabolic/GI: hypoglycaemia, liver injury, delayed gastric emptying. Excessive loss of sodium (Na^+), K^+, chloride and hydrogen ions (H^+) from the stomach, hypochloraemic metabolic alkalosis and hypokalaemia. Severe hypokalaemia is uncommon. Correct with caution preoperatively. Hypocalcaemia may accompany hypokalaemia.
- Musculoskeletal: osteopenia, osteoporosis, ↑ risk of fractures and hypothermia.
- Respiratory: loss of lung elasticity, higher airway pressures.
- Haematological: pancytopenia (anaemia and immunodeficiency).
- CVS: sinus bradycardia, ↓ LV forces, orthostatic hypotension, mitral prolapse, prolonged corrected QT interval, ↑ vagal tone, pericardial effusion, congestive heart failure, arrhythmia (very sensitive to catecholamines or neostigmine). Cardiac arrest can occur 2° to corrected QT interval prolongation or cardiac arrhythmia.
- Renal: ↓ GFR, proteinuria.
- Immunological: 1° cause of death is severe sepsis (especially very low BMI). ►► Granulocyte stimulating factor can be used.
- Endocrine: amenorrhoea, impaired thyroid function and glycaemic control. May mimic panhypopituitarism.

Anaesthetic approach

Preoperative

- Full nutritional assessment (see also ➲ p. 84), including psychiatric assessment.
- Correct hypoglycaemia (cautiously and gradually; beware rebound hyperinsulinaemia and profound hypoglycaemia).
- Acute refeeding can be risky. There is evidence that delaying surgery to achieve BMI >14.5kg/m² improves hypoglycaemia and leucopenia, which decreases mortality.[20]
- Correct other electrolytes, especially K^+ and phosphate. Replace Ca^{2+} and vitamin D.

Intraoperative

- Short, minimally invasive operations are better due to cardiac risk.
- RSI recommended. Fasting does not guarantee an empty stomach.
- Reduce doses of NMBAs. Prolonged recovery 2° to hypokalaemia and hypocalcaemia. Monitor neuromuscular block. Consider sugammadex over neostigmine to reduce risk of cardiac arrhythmia.
- Carefully consider drug doses. Take into account patient weight, hypoalbuminaemia (reduced protein binding) and renal and liver function (reduced metabolism and excretion).
- Carefully maintain body temperature with forced air warming devices and warmed infusions.
- Beware of positioning due to risk of pressure necrosis and nerve palsies.
- Do not overfill. Can lead to pulmonary oedema and cardiac failure.

Postoperative

- Beware refeeding syndrome (hypophosphataemia, hypokalaemia, hypomagnesaemia and hypoglycaemia in response to feeding after a period of starvation) and use an appropriate refeeding regime with nutritional support, cardiac monitoring, and electrolyte monitoring and supplementation.
- As per the NICE-SUGAR study, avoid 'intensive' glucose control.[20]
- Be wary of postoperative infection, with rigorous attention to the surgical site infection bundle and care of vascular access.

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Short gut (bowel) syndrome

Removal of half or more of the small bowel due to congenital or acquired disease (necrotising enterocolitis, congenital defect, meconium ileus, intussusception, Crohn's disease, ischaemic or traumatic bowel injury, malignancy) or failure of existing gut to function.[21,22]

Complications

Dehydration, severe malnourishment, anaemia, electrolyte disturbance, chronic pain, thrombosis and long-term anticoagulation, high-output stoma, complications relating to feeding lines or tubes, liver failure, kidney or gallstones, lactic acidosis.[21,22]

Nutritional deficits depend on the site of non-functioning bowel:

- Duodenum: iron, water-soluble B vitamins, vitamin C
- Jejunum: carbohydrate, fat, protein, folate
- Ileum: bile salts, fat-soluble vitamins A, D and E, vitamin B_{12}, Ca^{2+}, magnesium, zinc (terminal ileum).

Treatment

Enteral, oral and parenteral nutrition, repletion of fluids and electrolytes, supplemental vitamins. Intestinal transplant is offered to those who have <1m of functioning small bowel who develop complications of parenteral nutrition.

Anaesthetic approach

These are usually high-risk patients who are often home or hospital-bound. Assessment should be carried out via a multidisciplinary team to assess CVS fitness, risk profile and vascular access strategy.[21] Intestinal transplants take place in specialist tertiary centres. Patients requiring other surgical procedures need to be handled with a similar level of attention to detail and a considered approach.

Considerations for the older patient

'Elderly' arbitrarily refers to patients >65y. They comprise the most rapidly expanding demographic of the surgical population. Age-related physiological and cognitive decline, comorbidity and frailty all contribute to the higher risk of perioperative morbidity and mortality among older patients. Great care should be taken when prescribing or giving drugs to older patients, as polypharmacy is common and both pharmacokinetics and pharmacodynamics are influenced by age.

Ageing is associated with progressive functional deterioration in all systems, the effects of which may be compounded by organ-specific comorbidity.

Cardiovascular

- Significant CVS disease is present in 50–65% of patients.
- Myocardial fibrosis and ventricular wall thickening occur, reducing ventricular compliance. Small changes in filling may have major effects upon CO and BP.
- Atrial fibrillation (AF) is common and reduces stroke volume through loss of the atrial component of ventricular filling.
- Maximal CO with exercise decreases by ~1% per year from the 5th decade.
- Reduced arterial compliance causes systolic hypertension and widened pulse pressure.
- Reduced autonomic responsiveness impairs CVS responses to hypotension. The hypotensive effect of anaesthetic agents is therefore likely to be more pronounced.
- Capillary permeability is ↑, leading to a greater risk of pulmonary oedema.
- The thirst response to reduced extracellular fluid (ECF) volume and ↑ plasma osmolality is reduced, increasing susceptibility to fluid depletion.

Respiratory

- Ventilatory responses to hypoxia and hypercapnia decline. Postoperative apnoea is more common. Ventilatory reserve declines.
- O_2 consumption and CO_2 production fall by 10–15% by the 7th decade. Patients are able to tolerate a longer period of apnoea following preoxygenation and minute volume requirement is reduced.
- Loss of elastic recoil increases pulmonary compliance, but chest wall compliance falls due to degenerative changes in joints. Therefore, total thoracic compliance may fall.
- Loss of septa increases the alveolar dead space. Closing volume increases to exceed the FRC in the upright posture in patients >65y, resulting in venous admixture. Thus, normal arterial partial pressure of oxygen (PaO_2) falls steadily [(13.3 – age/30) kPa or (100 – age/4) mmHg].
- Airway protective reflexes decline, increasing the risk of postoperative pulmonary aspiration.
- In edentulous patients, maintaining a patent airway and face mask seal can be difficult. Leaving false teeth *in situ* may help.

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Renal

- Renal mass and the number of glomeruli fall progressively (by 30% in the 8th decade), resulting in reduced GFR. CC falls comparably, although serum creatinine may not rise because of ↓ production from reduced muscle mass (see ➲ p. 192).
- Tubular function deteriorates, leading to reduced renin–aldosterone response, antidiuretic hormone (ADH) sensitivity and concentrating ability. As a result, all renal homeostatic functions deteriorate, rendering elderly patients more susceptible to both fluid overload and hypovolaemia. Hypo- and hypernatraemia are also more likely.
- Reduced clearance of renally excreted drugs necessitates dose adjustment. Particular care must be taken with potentially nephrotoxic drugs such as aminoglycosides.

Hepatic

- Hepatic mass and blood flow fall by up to 40% by the 9th decade. Although cellular function is relatively well preserved in healthy patients, the reduction in size reduces clearance and prolongs the effect of drugs that are metabolized and excreted by the liver. These include opioids, propofol, benzodiazepines and NMBAs.

Central nervous system

- Brain size and neuronal mass decrease. The average brain weight falls by 18% between the ages of 30 and 80y. Mild cognitive impairment may progress to dementia, which affects 10% of patients over 65y of age, and 20% of those over 80y. It is important to distinguish between dementia and reversible confusional states due to hypoxia, sepsis, pain, metabolic derangement and depression. The hospital environment can precipitate anxiety and confusion.
- The elderly have fewer requirements for opioids and sedatives and are more susceptible to opioid-induced depressed consciousness and respiration. This is likely to be due to both pharmacodynamic and pharmacokinetic effects. Pain threshold may be ↑.
- Postoperative delirium (POD) and postoperative neurocognitive disorder (POND, formerly postoperative cognitive dysfunction (POCD)) are common in the elderly, occurring in >10% of patients. Disturbances of cerebral perfusion and cellular oxygenation are likely to be contributory factors. Potentially reversible risk factors for POD include severe pain, infection, malnutrition, electrolyte imbalance, dehydration, environmental disturbances and substance withdrawal (alcohol, medication).
- POND describes significant perioperative decline in mental status or awareness, occurring up to 30d (delayed neurocognitive recovery) or 12mo (POND) after surgery, and which may persist beyond that. It is associated with ↑ mortality, impaired quality of life and loss of employment.
- POND is more common after major surgery, cardiac surgery and emergency surgery. Multifactorial causes are likely, related to inflammatory reactions, altered hormonal homeostasis and/or direct anaesthetic agent toxicity. Focused anaesthesia interventions (treatment of hypoxaemia and hypotension), guided by the depth of anaesthesia and cerebral saturation monitoring, may ameliorate these.

Endocrine

- Glucose loading is increasingly poorly tolerated in elderly patients. The incidence of DM rises to 25% in patients >80y.

Haematology and the immune system

- Hypercoagulability and DVT are more common with advancing age.
- Anaemia is more common, and the response of the marrow to anaemia is impaired.
- Immune responses are reduced in the elderly, putting them at ↑ risk of infection. This is due to reduced bone marrow and splenic mass with loss of the thymus.

Thermoregulation

- Thermoregulation is impaired, increasing the risk of hypothermia.
- Postoperative shivering increases skeletal muscle O_2 consumption, while vasoconstriction increases myocardial work and O_2 demand.

Pharmacology

- TBW is reduced, while fat percentage is ↑. The volume of distribution of water-soluble drugs is reduced, reducing dose requirements, while that of lipid-soluble drugs is ↑, which may prolong clearance.
- The initial volume of distribution falls because of reduced CO. This reduces the dose requirement and is particularly relevant for induction agents. Arm–brain circulation time is prolonged, increasing the time taken for induction agents to take effect.
- Reduced plasma albumin concentration decreases the dose requirement of drugs such as benzodiazepines and opioids, which are bound to albumin.
- The minimum alveolar concentration (MAC) of inhaled agents decreases steadily with age (6% reduction per decade) and is reduced by around 40% by the age of 80y (see pp. 411–12). This may be related to a reduction in neuronal mass. Reductions in blood/gas partition coefficient and CO in the elderly result in shorter onset time.
- The risk of GI bleeding due to NSAIDs is ↑. These agents may also contribute to the development of AKI in the presence of impaired renal perfusion. ACE inhibitors exacerbate this risk. Fluid retention due to NSAIDs may precipitate heart failure in susceptible patients.

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Anaesthesia for older patients

Perioperative mortality increases with age, ASA status and the type and urgency of surgery. The 30d mortality after hip fracture surgery is ~6.5% in the UK (see also ➲ pp. 642–4), and after emergency laparotomy ~4% in those aged 50, rising by ~4% per decade. Outcome is improved by thorough multidisciplinary preoperative assessment, choice of an anaesthetic technique appropriate to the patient's condition and meticulous perioperative care aimed at minimising physiological disturbance.

Preoperative assessment and management

- A systematic review is vital. In patients who have sustained a fracture, an underlying medical cause for a fall should be sought.
- Day surgery is particularly appropriate for fit patients undergoing minor surgery, as it minimises the disorientation associated with a change of environment.
- The level of physical activity that can be sustained is a useful indicator of cardiorespiratory fitness but is often limited by joint disease.
- The mental state should be evaluated. The abbreviated mental test or Mini-Mental State Examination may be useful in differentiating dementia from acute confusional states.
- Consideration should be given to preoptimisation of medical conditions. This may require cross-specialty involvement and high dependency care. The benefits of delaying surgery, while this takes place, should be balanced against the risks, particularly in non-elective surgery. In patients with lower limb fractures, a delay in mobilisation may increase the risk of pressure sores, DVT and pneumonia.
- With the exception of oral hypoglycaemics, regular medications should be continued until the time of surgery. Alcohol should not be withheld the day before surgery, and nicotine patches may be helpful in smokers. Sedative premedications should generally be avoided, particularly benzodiazepines, centrally acting anticholinergics and pethidine. Antacid prophylaxis should be considered. Maintaining β-blockade may reduce the risk of MI.

Frailty

- Frailty defines patients with reduced physiological capacity to compensate effectively for external stressors such as illness and surgery.
- Frail patients are at greater risk of adverse outcomes, including prolonged hospital stay, institutionalisation, worsening disability and death.
- Frailty is more common in older people (10% >65y, 40% >85y), but age is only one of numerous factors (e.g. comorbidity, strength, cognition) used to derive predictive scoring tools.
- Several scoring tools can be used to identify frailty and monitor changes in frailty over time (e.g. Rockwood and Edmonton). These can be useful prognostic tools and can be used to provide older, vulnerable patients with information and reassurance about perioperative care based on their individual needs.

Nutrition

- Nutritional status is frequently poor in older people, under-recognised by clinicians and compounded by a lack of appetite resulting from surgery, pain and nausea.
- Perioperative complications and length of hospital stay may be reduced by nutritional supplementation prior to major surgery.

Perioperative management

- The type of anaesthesia appears less important than the care with which it is given with regard to the patient's physiological condition. However, regional anaesthesia may reduce bleeding, risk of DVT, respiratory infection and cognitive dysfunction (particularly if given without/with minimal sedation). MAC should be age-adjusted or, if using TIVA, infusions should be target-controlled (TCI).
- Careful monitoring is necessary to detect hypotension during GA induction and shortly after spinal anaesthesia administration. Consideration should be given to invasive BP and depth of anaesthesia monitoring. Prolonged arm–brain circulation time delays the onset of IV induction agents; flush the drugs with 0.9% sodium chloride and remain patient to avoid an inadvertent overdose.
- Temperature should be measured, and hypothermia prevented using fluid warmers, active body-warming devices and elevation of ambient temperature.
- Prolonged surgery and periods of hypotension increase the risk of pressure sores. Care should be taken to reduce pressure with soft padding. During long procedures, it is advisable to relieve pressure and massage vulnerable areas intermittently.

Postoperative management

- High dependency facilities should be considered if this is likely to reduce morbidity or mortality significantly, or if an identifiable organ support is required.
- Fluid balance, vital signs, serum electrolytes and haematology must be carefully monitored and treated appropriately. Patients with CVS disease may need to have their Hb kept >90–100g/L.
- Reversible factors should be sought if the patient exhibits delirium (pain, hypoxaemia, distended bladder, myocardial/cerebral ischaemia, electrolyte disorder, drugs).
- Pain is common but undertreated in elderly surgical patients, particularly if cognitively impaired. Regular paracetamol prescription and regional analgesia should always be considered and are preferable to opioids and NSAIDs.
- Anaesthetists should facilitate postoperative patient remobilisation and 're-enablement' through age-appropriate anaesthesia, fluid therapy, thermoregulation, analgesia and good communication.

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When not to operate on older people

Heroic curative surgery may not be appropriate if the chance of benefiting the patient is felt to be very low. Decisions regarding futility of surgery are difficult and should be multidisciplinary and taken at consultant level, with the involvement of the patient and their family. Palliative procedures to improve the quality of life should be considered if the patient is adequately prepared. These decisions must be carefully documented.

- Older patients must be assumed to have the mental capacity to make decisions about their treatment.
- Access to surgical or critical care should not be rationed on the basis of age.

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Learning disabilities and/or autism

The term learning disability encompasses a broad range of conditions characterised by impairment in information processing.[23]

Approximately 1.5 million people in the UK have a mild, moderate or severe learning disability.

These patients form a heterogeneous group, often with congenital or acquired comorbidities, including craniofacial anomalies, congenital cardiac disease and epilepsy. Comorbidity and other factors, such as ↑ regurgitation, aspiration and some patients' sensitivity to opioids (e.g. those with cerebral palsy), explain a relatively high risk of respiratory complications perioperatively.

Patients with learning disabilities have ↑ health care needs and also often have behaviours that can challenge. An over-reliance on physical or pharmacological restraint fails to address behavioural issues, which may be exacerbated over repeated encounters with health professionals. The aim must be to adopt the least restrictive approach to succeed. Reasonable adjustments may have to be made to standard care pathways at any point in the perioperative period to aid patient cooperation.

Communication for learning disability patients

- There should be clear signage within the hospital and ease of access ensured for those with mobility problems.
- Difficulty in communication is the source of most problems in the care of learning disability patients. Some patients are non-verbal. Others have difficulties with the speech, social or emotional domains of communication.
- Hospital learning disability liaison services are an invaluable source of knowledge and support for patients and carers. They serve to facilitate communication and logistics for patients with learning disability.
- Examine and perfect your own non-verbal communication. Most patients will read it very well.
- Use clear, precise instructions. Avoid metaphors, abstraction, jargon and acronyms, as these are often not understood. Use signing systems, such as Makaton®, with the aid of parents or carers if necessary. Intensive interaction, such as mirroring, may reduce anxiety.
- Behavioural training can be effective in the preoperative period:
  - Using text or visual descriptions, such as Easy Read and social stories or objects of reference, helps to break down a new task into a series of achievable objectives.
  - Desensitisation visits allow patients to attend the hospital and leave without any procedures being performed. Ideally this leaves the patient with a positive view of the hospital for future care.
  - Prior use of three-dimensional (3D) immersive videos enables patients to take a virtual tour of the journey to theatre and the anaesthetic room and can reduce anxiety on the day of admission.

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Autistic spectrum disorder

- Autistic spectrum disorder (ASD) has been defined as a persistent deficit in social communication, interaction and imagination.[23]
- About 50% of patients with ASD will have a learning disability.
- ♂:♀ is now recognised as closer to 3:1. Girls are more difficult to diagnose, as they often display social camouflaging. ASD has a prevalence of 1.5% and an onset usually by 3y, but diagnosis is often delayed.
- Restrictive, repetitive behaviours are often recognised first.
- No clear aetiology, although a number of genetic variations and perinatal risk factors have been identified.
- Described as a 'theory of mind' deficit. ASD patients display an inability to understand the impact of their behaviour on others and lack insight into the perspectives or intentions of others.
- Patients typically have difficulties with verbal communication and/or non-verbal communication, particularly with social or emotional aspects of interactions.
- ASD patients often have significant hyper- or hyposensory issues.

Communication points for patients with autistic spectrum disorder

- Allow time for questions to be digested, and avoid causing confusion with repeated questioning.
- Reduced abstract thinking ability means non-literal communication is often not understood.
- Patients often benefit from detailed, factual descriptions of each stage of anaesthesia being broken down into small, achievable steps.
- Patients may become distressed if prevented from performing certain repetitive, self-stimulatory behaviour such as hand-flapping, rocking, spinning or verbal repetition.
- Sensory dysregulation can have profound effects on autistic patients. They can be deeply disturbed by touch and eye contact. Keep noise down (including that from adjacent rooms); restrict numbers in the room, and consider dimming the lights. They may not tolerate adhesive dressings while awake. It may be necessary to attach monitoring immediately after induction.
- Debunking myths about ASD patients—they do feel pain, are often tricky **because** they are scared, do tell jokes and can engage with guided imagery.

Capacity and consent

- All patients should be presumed to have capacity unless an assessment has shown a lack of capacity.
- Regardless of the level of capacity, every effort should be made to provide information to patients in a format they can understand.
- Consenting requires detailed knowledge of the Mental Capacity Act (2005)/Adults with Incapacity Act (2000) in Scotland.
- Elective best interest meetings are held to gather the views of family and carers, so as to understand the patient's perceptions and beliefs and the conditions that will be acceptable. If there is nobody suitable

to help make decisions about medical treatment, an Independent Mental Capacity Advocate will be required. Best interest meetings enable discussion about additional risks and reasonable adjustments to treatment.
- In an emergency, two clinicians involved with the patient's care may reach a medical best interests decision.
- The appropriate consent form must be used for a patient who lacks capacity, signed by the 1° decision-maker for the procedure, not necessarily by the clinician delivering it.

Preoperative preparation

- Hospitals must provide training and policies specific to learning disability and ASD patients.
- Patients with a learning disability or ASD should not be denied treatment **because** of their disability if that treatment is in their best interests.
- Systems must identify patients early to create bespoke care plans, with multidisciplinary and liaison team involvement.
- Combining multiple planned treatments or investigations under a single GA is beneficial, e.g. imaging, dental procedures and vaccinations.
- Parents and carers should be actively involved. They are an invaluable source of information on specific anxieties, triggers and beneficial behavioural modifications.
- Request care 'passports' which contain individualised information.
- Comprehensive preoperative assessment and history are essential. Use opportunistic observations for airway/physical assessments.
- Many congenital conditions associated with learning disability have multisystem sequelae, so a comprehensive systems enquiry is essential.

Perioperative plan

- Patients with a learning disability or ASD should ideally be first on the operating list, with steps taken to reduce delays and waiting times.
- Maintain a calm, quiet environment. Where possible and safe, adjustments should be made to accommodate patient preferences such as dimmed lighting, own clothes and parents/carers supporting them in the anaesthetic room.
- A sedative/anxiolytic premedication or clinical holding may be advisable. Use an appropriate technique, dependent on your team's experience. Some suggested doses are shown in Table 4.3.
- Midazolam is beneficial for its anxiolytic effect but may not be sedative, especially in ASD. Ketamine is useful for dissociation in adult patients when more sedation is required. It should be used with caution in epilepsy due to reduced seizure thresholds.
- Best interest meetings may identify some patients who require premedication at home or in the hospital car park. Risk:benefit assessments take into account constraints, e.g. monitoring pertaining to these locations.
- Inhalational induction is often preferable, avoiding the stress of awake IV cannulation. Maintenance of anaesthesia may then be switched to the IV route. Indeed, there is increasing evidence of reduced emergence delirium or ASD rages (recovery meltdown) with TIVA.
- Multimodal analgesia optimises pain control and reduces opioid use.

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Table 4.3 Premedication regimes

Effect	Drug	Route	Dose	Max dose
Anxiolysis	Midazolam	PO/IM	0.5mg/kg	30mg
Sedation ++	Clonidine (inpatient)	PO/IM	2–4 micrograms/kg	150micrograms
Sedation and dissociation	Ketamine (day case)	PO/IM	2–3mg/kg	300mg

Postoperative

- As for induction, emergence from anaesthesia should involve minimal physical or sensory stimulation. Early presence of carers/family in recovery is often invaluable.
- IV cannulae and selected monitoring should be removed as soon as it is safe to do so, to reduce patient distress.
- Communication difficulties frequently impede pain assessment.
- Always consider pain (while excluding hypoxia) as a source of ↑ agitation. Behavioural serial pain scoring systems are often beneficial.
- ASD patients may experience explosive behavioural 'meltdowns'. Triggers include disorientation, sensory or communication issues, feeling out of control and anxiety. Be aware of each individual's warning signs and minimise stimulation on waking.
- Modify discharge criteria (e.g. eating/drinking) to allow patients to return to their own routine and environment as soon as possible.
- Discharge documentation, instructions and prescriptions should be completed in advance to facilitate a potentially abrupt discharge.

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Chapter 5

Cardiovascular disease

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- Narrow complex tachycardia pp. *1062–1064*
- Broad complex tachycardia p. *1062*

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Ischaemic heart disease

Current Hospital Episode Statistics data suggest that ~35 000 patients die each year in the UK within 30d of surgery. Major adverse cardiac events (MACE) are the largest cause of perioperative deaths, accounting for at least 30% of deaths.

Perioperative risk of myocardial ischaemia

CVS risk is influenced by both patient and surgical factors. Patient factors are summarised in Table 5.1.[1]

Table 5.1 Patient factors influencing perioperative risk of myocardial ischaemia

Major risk predictors (markers of unstable CAD)	MI <2mo prior to surgery
	Unstable or severe angina
	Ongoing ischaemia after MI (clinical symptoms or non-invasive testing)
	Symptomatic heart failure with EF <40%
	Significant arrhythmias (e.g. high-grade AV block, symptomatic arrhythmias, or supraventricular arrhythmias with uncontrolled ventricular rate)
	Severe valvular heart disease
	PCI (BMS <30d, DES <1y, angioplasty <2w)
Intermediate risk predictors (markers of stable CAD)	History of IHD
	Asymptomatic heart failure, reduced EF
	Heart failure with preserved EF (diastolic dysfunction)
	History of cerebrovascular disease
	Abnormal renal function
	Diabetes
Minor risk predictors (↑ probability of heart disease)	Advanced age
	Abnormal ECG
	Rhythm other than sinus
	Low functional capacity
	Uncontrolled systemic hypertension
	Smoking history

AV, atrioventricular; BMS, bare-metal stent; CABG, coronary artery bypass grafting; CAD, coronary artery disease; DES, drug-eluting stent; ECG, electrocardiogram; EF, ejection fraction; IHD, ischaemic heart disease; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Functional capacity

Cardiopulmonary fitness is commonly used to estimate perioperative risk. The physiological response to major surgery increases O_2 demand by up to 40%, requiring a subsequent increase in O_2 delivery. The ability to exercise is an indicator of CVS reserve. It is expressed in metabolic equivalents of task (METs) (Box 5.1). One MET is the resting O_2 consumption of a 40y-old 70kg ♂ (3.5mL/kg/min). Patients who cannot sustain 4 METs of physical activity frequently have adverse outcomes following high-risk surgery (see also ➲ p. 35).

METs are most commonly assessed subjectively, based on patient history, which may result in substantial misclassification of risk. More objective measures, such as the Duke Activity Status Index (a self-reported questionnaire about usual physical activities) or CPET, have been shown to improve identification of patients at risk of perioperative MACE and death.[2]

Surgical factors are also important in determining perioperative risk (Table 5.2).[3]

Box 5.1 Metabolic equivalents of common tasks

1–4 METs	Eating, dressing, dishwashing and walking around the house
4–10 METs	Climbing a flight of stairs, walking on level ground at >6km/h, running briefly, playing golf
>10 METs	Strenuous sports: swimming, singles tennis, football

Table 5.2 Risk of ischaemic heart disease by type of surgery

High risk: >5% death/non-fatal MI	Major emergency surgery
	Aortic/major vascular surgery
	Prolonged surgery with large fluid shifts
Intermediate risk: <5% death/non-fatal MI	CEA/EVAR
	Head and neck surgery
	Intraperitoneal and intrathoracic surgery
	Orthopaedic surgery
	Prostatic surgery
Low risk: <1% death/non-fatal MI	Minimally invasive endoscopic surgery
	Cataract extraction
	Superficial surgery (including breast)

CEA, carotid endarterectomy; EVAR, endovascular aneurysm repair.

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Clinical risk indices

Risk indices integrate patient and surgical factors, providing a quantitative estimate of perioperative risk. The Revised Cardiac Risk Index (Table 5.3) is the most validated clinical risk index for prediction of major perioperative cardiac complications.

Table 5.3 Revised Cardiac Risk Index

Variable	Points
History of ischaemic heart disease	1
History of congestive heart failure	1
History of cerebrovascular disease	1
Use of insulin therapy for diabetes	1
Preoperative serum creatinine >177 micromoles/L	1
High-risk surgery	1

Total points: none = risk of MACE 3.9%; 1 = risk of MACE 6%; 2 = risk of MACE 10.1%; >2 = risk of MACE 15%.

Source: data from Lee TH *et al. Circulation*. 1999;100:1043–9.

Perioperative testing

Evaluation of patients with IHD depends on the planned surgery, facilities and time available. Special investigations are discussed below. Precise recommendations remain controversial, but careful history, examination and practical application of preoperative screening tests are important. Little advantage is gained from complex examinations which will not alter management. The positive predictive value of most tests is low. In high-risk patients, alternative surgical procedures or medical management may need to be considered, with input from anaesthetists, cardiologists, surgeons and the patient.

Special investigations

12-lead electrocardiogram

A preoperative ECG should be performed on patients over 65y undergoing intermediate-risk surgery and anyone with risk factors for IHD. Patients with IHD may have a normal or non-specific resting ECG.

Cardiac biomarkers

- Preoperative measurement of BNP and cardiac troponins in the perioperative period predicts myocardial injury after non-cardiac surgery (MINS) in patients undergoing non-cardiac surgery.
- Biomarker assessment may complement risk prediction in patients at high risk of MACE.[3,4]
- Daily troponin measurements and postoperative ECGs for 48–72h after non-cardiac surgery in patients at high risk of MACE may be recommended to detect perioperative MINS, facilitating monitoring and medical management.

Assessment of LV function

- Echocardiography is the most widely available test for diagnosis and serial assessment of patients with dyspnoea or known LV dysfunction.
- Cardiac MRI provides reliable assessment of LV function, as well as coronary artery evaluation.

Coronary CT angiography

A non-invasive alternative to coronary angiography. Calculates the Coronary Artery Calcium Score which estimates risk of IHD and need for invasive coronary angiography, but overestimates the risk of MACE, so is not recommended for routine cardiac risk estimation.

Exercise ECG

A functional test in patients with known coronary artery disease (CAD). Detects myocardial ischaemia through changes in ST-segment morphology. Interpretation is difficult if there are pre-existing ST-segment abnormalities and if the patient fails to achieve target HR. High prevalence of false positives and negatives, so no longer used for CAD diagnosis. Of limited use for risk prediction for postoperative MACE.

CPET

(See also ➔ pp. 33–5.)

CPET is an integrative assessment of cardiovascular, respiratory and musculoskeletal systems. It provides an objective assessment of functional capacity. It takes 30min to perform but requires expensive equipment. It has a low positive predictive value but may be used to guide perioperative SDM in high-risk patients.

Pharmacological stress testing

Moderate/large areas of reversible ischaemia are associated with ↑ risk of MACE. Pharmacological stress tests provide a dynamic assessment of myocardial perfusion. A normal study has a high negative predictive value. High-risk patients should be tested if testing will change management. Patients who have large areas of reversible ischaemia should be considered for coronary angiography.

- Myocardial perfusion imaging (thallium scintigraphy) uses a coronary vasodilator (dipyridamole or adenosine) and a radioisotope, thallium-201, that rapidly accumulates within perfused myocardium. Comparisons are made between resting and stress images. Changes in uptake reflect reversible ischaemia, while fixed defects represent irreversible infarcted/scarred myocardium.
- Dobutamine stress echocardiography uses an increasing dose of dobutamine (max 40 micrograms/kg/min), with simultaneous two-dimensional (2D) precordial echocardiography. New or worsening regional wall motion abnormality (RWMA) is an indicator of impaired perfusion.
- Stress cardiac MRI can incorporate dobutamine and vasodilator stress testing principles to assess for RWMA and myocardial perfusion abnormalities, helping predict the risk of future MACE.

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Preoperative coronary artery revascularisation

Revascularisation may be percutaneous (angioplasty/stent insertion) or surgical.

Percutaneous coronary intervention

- Indicated preoperatively in patients with unstable IHD.
- Recent PCI is associated with ↑ MACE and 30d mortality.
- PCI causes trauma to the vessel wall, rendering the endoluminal surface thrombogenic until the vessel wall has healed or the stent has re-endothelialised. Therefore, dual antiplatelet therapy (DAPT) with aspirin and a P2Y12 inhibitor (e.g. clopidogrel or ticagrelor) is necessary to prevent stent thrombosis.
- If a patient treated with DAPT post-PCI requires an operation where bleeding may be problematic (e.g. intracranial surgery, spinal surgery, open aortic surgery), and the operation cannot be deferred for an appropriate time period, consider stopping clopidogrel/ticagrelor/prasugrel for 5–7d preoperatively. Cardiology advice may include antiplatelet bridging with a short-acting platelet IIb/IIIa glycoprotein receptor antagonist (tirofiban, eptifibatide).
- Duration of DAPT is dependent on type of stent and associated acute coronary syndrome at the time of stent insertion.
- Interruption of DAPT perioperatively is associated with a high cardiac complication rate and requires multidisciplinary team discussion.
- Recommendations for interrupting DAPT for non-cardiac surgery[5] are: bare-metal stent (BMS): delay for 1mo; drug-eluting stent (DES): delay depends on type of DES, so liaise with cardiology. Continue aspirin perioperatively whenever possible.
- If PCI is required prior to surgery, careful consideration should be given to the type of stent inserted. For procedures that can be delayed by 12mo, it is reasonable to use a DES. If the procedure can be delayed for 6–8w, a BMS can be used. If the condition requires surgery to be performed in the next 2–4w, consideration should be given to performing balloon angioplasty.

Coronary artery bypass grafting

Indications for preoperative CABG are identical to those for CABG on medical grounds, i.e. >50% left main stem stenosis, >70% two- or three-vessel disease (including the proximal left anterior descending artery) and/or LV systolic dysfunction. Elective surgery should be postponed for a minimum of 6w post-CABG.

Perioperative MACE risk modification

Patients are at risk of ischaemic events and MI in the perioperative period. MI can be classified into five types:

- Type 1: caused by an acute atherothrombotic coronary event
- Type 2: heterogeneous aetiology, resulting from a condition other than CAD contributing to an imbalance between O_2 supply and demand
- Types 3: relates to sudden unexplained cardiac death
- Type 4: relates to MI associated with PCI
- Type 5: relates to MI associated with cardiac surgery.

About 75% of perioperative MIs are type 2,[6] and the other 25% are type 1. Reduction of the risk of MACE requires a multimodal approach, dependent on time available and patient engagement, but involves lifestyle changes as well as pharmacological therapy.

- Medical therapy should be continued perioperatively to protect against ischaemic stress. If GI absorption is impaired or the patient is kept nil by mouth, drugs should be given IV if possible.
- Aspirin alone has not been shown to reduce MACE or 30d mortality in patients undergoing non-cardiac surgery.[3]
- Long-term β-blockade should be continued perioperatively due to the cardioprotective effects (decrease myocardial O_2 demand).
- Initiation of β-blockade within 24h prior to surgery is not recommended.[4]
- There is limited evidence to suggest that high-risk patients who demonstrate inducible ischaemia on preoperative stress testing may benefit from carefully titrated β-blockade (to a HR of 60–80bpm) started at least 1w prior to surgery. Low-dose bisoprolol is commonly used. Dose titration is challenging.
- Nitrates should be continued perioperatively, IV or transdermally, if necessary. There is no evidence that prophylactic administration decreases the risk of perioperative cardiac complications.
- Calcium channel blockers should be continued preoperatively and resumed as soon as possible postoperatively. Verapamil has been shown to confer a small measure of cardiac protection in small RCTs but must be avoided in LV impairment.
- ACE inhibitors and angiotensin II receptor blockers (ARBs) improve survival following MI and in patients with LV dysfunction. They do not protect against perioperative MACE. ACE inhibitors and ARBs have been shown to increase the risk of intraoperative hypotension, but not MACE. Other studies have shown clinically important hypotension to be independently associated with an ↑ risk of death, MI and CVE, but the highest risk comes from hypotension 24h after surgery. It has been suggested[4] that withholding ACE inhibitors and ARBs 24h before surgery and recommencing them 2d after surgery may be appropriate, but the quality of evidence is currently low and these drugs are very important in the management of these patients. Others suggest stopping them only if used to treat hypertension.[3] In those with severe LV dysfunction, it may be prudent to continue ACE inhibitors throughout the perioperative period. Cardiology advice may be needed.
- Perioperative statin administration has been shown to improve both short-term and long-term cardiac outcome following non-cardiac and coronary bypass surgery.[3,4] Statins enhance plaque stability, making plaque rupture less likely.

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Anaesthetic considerations

The main goal is to maintain O_2 demand and supply matching.

- Consider 5-lead ECG monitoring.
- Invasive cardiovascular monitoring (arterial line ± central venous pressure ± CO monitoring) should be considered for high-/intermediate-risk patients.
- Point-of-care ultrasound can provide real-time assessment of cardiovascular and respiratory function.
- Avoid hypotension, hypertension and tachycardia to minimise myocardial demand and supply mismatching. Anticipate phases of sympathetic stimulation (e.g. intubation, emergence) and consider using a short-acting β-blocker to attenuate this.
- Pain causes sympathetic stimulation: provide effective pain control.
- Central neuraxial and regional blocks can be very effective, but not always possible in the context of DAPT.
- Liberal transfusion triggers (Hb 90–100g/L) reduce the risk of MACE.[7]
- Consider admission to HDU postoperatively for close monitoring.

Perioperative acute myocardial ischaemia and infarction

Acute myocardial infarction (AMI)

MI is defined pathologically as myocardial cell death due to prolonged ischaemia. AMI is universally defined as acute myocardial injury with clinical evidence of acute myocardial ischaemia and detection of a dynamic change in high-sensitivity cardiac troponin (cTnT) values. At least one other ischaemic feature (symptoms, new ECG changes, new RWMA, new coronary thrombus)[8] is required for diagnosis. Perioperative AMI occurs in 4% of adult inpatients undergoing non-cardiac surgery. Of these, nearly 10% will die within 30d of surgery.[9] Typical ischaemic symptoms of AMI may be masked by the effects of drugs and distracting surgical pain.

Serial cardiac troponin measurement is a cornerstone for AMI diagnosis. Development of cTnT assays has improved overall diagnostic accuracy. Myocardial injury releases cardiac troponins into the systemic circulation, with cTnT levels rising within 3–4h after onset of myocardial injury. Levels remain ↑ for 10–14d. A rise or fall (dynamic change) in cTnT implies an acute event. At least one cTnT level above the 99th percentile of a normal reference population is required for diagnosis of AMI.

Immediate management

- Ensure you confirm the diagnosis: perform ECG, send cTnT blood sample and consider point-of-care ultrasound if available.
- Reduce O_2 demand: control HR to low normal. Consider use of short-acting β-blocker (esmolol 50–200 micrograms/kg/min loading dose, then 0.05–0.2 micrograms/kg/min). If awake, opioids can reduce pain associated with ischaemic symptoms.
- Optimise O_2 delivery: maintain normotension. Attempt coronary artery vasodilation with glyceryl trinitrate (GTN), either sublingual or IV. Provide supplemental O_2. Ensure adequate Hb concentration.
- In the anaesthetised patient, consider the need to abandon surgery.
- Consider administration of aspirin (NGT may be required).
- Consult cardiologist. Urgent/emergent PCI may be warranted.

Myocardial injury after non-cardiac surgery

MINS is defined as prognostically relevant myocardial injury due to ischaemia that occurs during or within 30d after non-cardiac surgery, including, but not limited to, patients who have had an MI. MINS occurs in nearly 20% of patients undergoing inpatient non-cardiac surgery and is an independent predictor of postoperative adverse cardiac events and death.[8,10] More than 90% of MINS events occur in the absence of clinical signs or symptoms of ischaemia[11] and only 20% will fulfil the universal definition for MI.

Diagnostic criteria[8,10]

- cTnT level ≥65 nanograms/L (or 20–64 nanograms/L, with absolute change of 5 nanograms/L) or non-cTnT ≥30 nanograms/L
- Occurring within 30d of non-cardiac surgery
- Not due to non-ischaemic aetiology (e.g. rapid AF, anaemia, sepsis)
- Ischaemic symptoms or ECG changes not necessary for MINS.

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Routine postoperative surveillance

Daily cTnT measurements on d1, 2 and 3 after non-cardiac surgery, while the patient is in hospital, in at-risk patients (e.g. age ≥65y or with a history of atherosclerotic disease).[12]

Medical management[8]

- Consider starting low-dose aspirin and statin therapy.
- Consider starting ACE inhibitor if there is hypertension.
- There is some evidence that initiation of anticoagulation (MANAGE trial) may reduce the risk of major vascular complications.[12]
- Role of coronary angiography and revascularisation is unclear.
- Arrange postoperative follow-up with cardiologist as an outpatient.

Heart failure

Heart failure is the commonest cause of admission to hospital in those aged >65y. Incidence rises with increasing age, with a 5y mortality of ~50%. Pertinent to the anaesthetist, indices estimating perioperative MACE and mortality include heart failure as an independent prognostic variable, with unstable or decompensated heart failure bestowing a greater burden of risk.

Heart failure is a clinical syndrome characterised by typical symptoms (breathlessness, ankle swelling and fatigue) with or without signs (raised jugular venous pressure (JVP), pulmonary crackles and peripheral oedema), caused by structural and/or functional cardiac abnormality, resulting in a reduced CO and/or raised intracardiac pressures at rest or during stress.[8]

LV systolic and diastolic dysfunction can be asymptomatic precursors of heart failure.[8] Terminology has been based on left ventricular ejection fraction (LVEF), but symptoms may not fit with the degree of reduction in LVEF. Current terminology used to describe heart failure is as follows: heart failure with reduced ejection fraction (HFrEF), heart failure with preserved ejection fraction (HFpEF) and heart failure with mid-range ejection fraction (HFmrEF) (is treated as per HFpEF) (Table 5.4).

Heart failure can be graded by severity of symptoms ± exercise intolerance or structural disease by the New York Heart Association (NYHA) functional classification (Table 5.5) or the American College of Cardiology/American Heart Association stages of heart failure (Table 5.6).

Other classification systems used to describe heart failure include:

- Chronicity: acute, subacute and chronic
- Symptoms: stable and unstable (deterioration from stable symptoms is referred to as decompensation)
- Affected side(s): left- and/or right-sided ventricular failure
- Diastolic and/or systolic dysfunction.

Table 5.4 Criteria required to define HFpEF, HFmrEF and HFrEF

	HFpEF	HFmrEF	HFrEF
Symptoms ± signs*	Yes	Yes	Yes
LVEF	≥50%	40–49%	<40%
BNP or NT-proBNP	Elevated	Elevated	
At least one additional criterion	LVH/left atrial enlargement or diastolic dysfunction	LVH/left atrial enlargement or diastolic dysfunction	

BNP, brain natriuretic peptide; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; NT-proBNP, N-terminal pro-brain natriuretic peptide.

* Signs may not be present in early stages or if treated.

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Table 5.5 New York Heart Association functional classification

Class	
1	Cardiac disease, no symptoms or limitation to ordinary physical activity
2	Mild symptoms and slight limitation during ordinary activity
3	Significant limitations in activity due to symptoms. Comfortable at rest
4	Symptoms at rest. Severe limitations

Source: data from Criteria Committee, New York Heart Association, Inc. *Diseases of the Heart and Blood Vessels. Nomenclature and Criteria for diagnosis*, 6th edition Boston, Little, Brown and Co. 1964, p 114.

Table 5.6 American College of Cardiology and American Heart Association stages of heart failure

Stage	
A	At high risk of heart failure, but without symptoms or structural heart disease
B	Structural heart disease, but without signs or symptoms of heart failure
C	Structural heart disease, with prior or current symptoms of heart failure
D	Refractory heart failure requiring specialised interventions

Source: data from Hunt, S.A. *et al.*, Focused update incorporated into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults, *Journal of the American College of Cardiology*, 2009;**53**(15):e1–e90.

Left ventricular heart failure (LVHF)

A note on terminology: although heart failure terminology has changed (HFpEF, HFmrEF, HFrEF), the terms systolic and diastolic left ventricular dysfunction are still widely used. In essence, systolic dysfunction refers to HFrEF, and diastolic dysfunction to HFpEF. HFmrEF is a grey zone in the middle. Symptoms can be present, independent of the degree of preservation of LVEF.

- Orthopnoea and paroxysmal nocturnal dyspnoea are the most sensitive symptoms.
- Elevated JVP is the most sensitive clinical sign.

Other common symptoms include:

- Presence of peripheral oedema
- Uncontrolled hypertension, tachycardia or arrhythmias
- Limited exercise tolerance and exertional dyspnoea.

Echocardiography is the mainstay of LVHF investigation. Echocardiographic changes include:

- ↓ LVEF
- ↓ LV filling pressure, mitral valve inflow velocity
- Evidence of left ventricular hypertrophy (LVH) or left atrial enlargement.

Given a normal LVEF in HFpEF, symptoms, signs, ECG (AF, LVH) and plasma natriuretic peptides (BNP) all help with the diagnosis.

Management[8, 13]

Aims to delay progression of symptoms, optimise symptom management and functional capacity and reduce mortality. Modifying risk factors (e.g. hypertension, glycaemic control) may delay disease progression.

Medical treatment of HFrEF

The evidence for best management is clearest for HFrEF.

- ACE inhibitors reduce morbidity and mortality. Beneficial effects include reduction in pre-/afterload and neurohormonal antagonism of the renin–angiotensin–aldosterone system. Titrate to maximum tolerated dose. ARBs are recommended in patients intolerant of ACE inihibitors.
- The combination of neprilysin inhibitor with ARB (sacubitril/valsartan) reduces cardiovascular mortality by 20% and all-cause mortality by 16%. The neprilysin inhibitor increases the bioavailability of natriuretic peptides. This leads to ventricular unloading and vasodilatory effects. This combination may be used instead of ACE inhibitor or ARB in refractory cases.
- β-blockers (carvedilol, bisoprolol) reduce HR and myocardial O_2 demand. β-blockers should be continued throughout the perioperative period. Use with caution in new-onset or decompensated heart failure.
- Mineralocorticoid receptor antagonists, e.g. spironolactone, reduce mortality in symptomatic patients already treated with ACE inhibitors. Use with caution in patients with impaired renal function. Other diuretics may be added for symptom control.
- Inotropes: digoxin may be used to control AF with rapid ventricular rate in symptomatic heart failure or in patients in SR with severe heart failure.
- Anticoagulation: if in AF or at risk of VTE.
- Calcium channel blockers are not safe for use in HFrEF.

Device treatment

- ICDs may be indicated for high-risk symptomatic patients or after unstable ventricular arrhythmias. They reduce the risk of sudden cardiac death.
- Cardiac resynchronisation therapy (CRT) for those with prolonged QRS duration. Biventricular pacing wires are placed. This aims to improve symptoms and reduce mortality in select patients.

Medical treatment of HFpEF and HFmrEF

No specific treatments have demonstrated a reduction in morbidity or mortality in these groups.[14]

- Diuretics are used as the mainstay of symptom management. They probably reduce morbidity and mortality.
- Spironolactone reduces heart failure hospitalisations.
- β-blockers and ACE inhibitors may be used for BP control.
- Manage AF in accordance with established clinical guidelines.

Right-sided heart failure[15]

RV dysfunction is an independent predictor of MACE after non-cardiac surgery. Right heart failure can be broadly divided into three categories, depending on its causation:

- 2° to pulmonary hypertension
- 2° to pericardial disease
- 2° to ventricular or valvular pathology.

Pulmonary hypertension is most commonly caused by LV failure, but other causes include chronic lung disease, OSA and PE.

Table 5.7 lists the causes of RV failure.

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Table 5.7 Causes of right heart failure

	↓ RV contractility	RV volume overload	RV pressure overload
Acute	Sepsis Myocardial injury/MI Myocarditis	Excessive transfusion LVHF	Acidosis Hypoxia, IPPV, ARDS PE LVHF
Chronic	RV cardiomyopathy Ebstein anomaly	Pulmonary or tricuspid regurgitation	Pulmonary hypertension Pulmonary stenosis Left-sided valvular disease Restrictive cardiomyopathy

ARDS, acute respiratory distress syndrome; IPPV, intermittent positive pressure ventilation; LVHF, left ventricular heart failure; MI, myocardial infarction; PE, pulmonary embolism.

Signs and symptoms of RHF

- Signs of reduced CO: hypotension, tachycardia, cyanosis
- Peripheral oedema—the commonest clinical sign
- Elevated JVP and left parasternal heave
- Ascites and hepatic/renal dysfunction
- Arrhythmias—common.

Investigations

- ECG may show right axis deviation and RV strain (S deflection in I, Q deflection in III and inverted T in III).
- Check blood for anaemia and renal and thyroid dysfunction.
- Raised BNP and troponin reflect stress and injury. Along with raised liver enzymes and prolonged international normalised ratio (INR), they reflect poor prognosis.
- CXR may show signs of causative pathology, e.g. LVHF.
- Transthoracic echocardiogram. Tricuspid annular plane systolic excursion (TAPSE) is a measurement of displacement of the lateral tricuspid annulus towards the apex during systole. It is used as an assessment of global RV function. TAPSE <16mm indicates RV systolic dysfunction. Alternatives include transoesophageal echocardiography (TOE), radionuclide imaging and cardiac MRI.
- Cardiac catheterisation may help if significant coronary or valvular heart disease is suspected. Right heart catheterisation provides an accurate assessment of pulmonary hypertension.

Medical management

Treat the underling condition (e.g. myocarditis, LVHF, PE) and aim to optimise RV preload, afterload and contractility.

- Diuretics usually require high doses. Do not discontinue in a hypotensive patient if there is evidence of volume overload. Renal replacement therapy may be required.
- Correct physiological causes of raised pulmonary vascular resistance (PVR) (e.g. hypoxia, hypercapnia, acidosis).

- Reduce PVR with non-selective vasodilators (e.g. GTN or sodium nitroprusside) or selective pulmonary vasodilators (e.g. prostacyclins, nitric oxide, phosphodiesterase (PDE)-5 inhibitors). Pulmonary vasodilators improve exercise tolerance and MACE in patients with right heart failure 2° to pulmonary hypertension.
- Consider dobutamine or milrinone, which have combined inotropic and vasodilator properties. Vasopressors may also be required.
- In chronic right heart failure, consider sodium restriction.
- Patients with biventricular dysfunction should be managed according to current guidelines for LVHF.

Perioperative management

Some patients may be deemed unfit for the proposed surgery. Consideration of individual patient and surgical risk is required. Multidisciplinary discussion will direct best patient care.

- History and examination should identify present or recent episodes of decompensated heart failure.
- Continue medical management in the preoperative period. If concerned, consult a cardiologist to see if changes in therapy are needed to minimise symptoms of ventricular dysfunction.
- Tachy- and bradyarrhythmias may occur. Consider direct current cardioversion (DCCV) in haemodynamically unstable patients. Correct electrolyte abnormalities. For chronic arrhythmias, aim for HR ~80bpm. Use local or regional techniques for peripheral procedures.
- Ensure adequate ventilation to maintain oxygenation and normocapnia. Careful use of positive end-expiratory pressure (PEEP) will reduce PVR in patients with pre-existing RV dysfunction.
- Invasive cardiovascular monitoring (arterial line, CVC, TOE) should be considered for all major surgery and high-risk patients.
- Perioperative decompensation may require treatment with vasodilator and inotropic therapy.
- Careful monitoring of fluid balance is essential.
- All patients should have supplemental O_2 following surgery.
- Good postoperative analgesia is essential to minimise detrimental effects of catecholamine release in response to pain.
- Consider ICU/HDU.

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Hypertension

About 25% of the adult UK population has hypertension, defined as systolic blood pressure (BP) >140mmHg and diastolic BP >90mmHg (stage 1 hypertension). Triggers for consideration for treatment in 1° care are ≥160/100mmHg or average ambulatory BP ≥150/95mmHg (stage 2 hypertension).[16] The link between elevated arterial pressure and CVS disease is well established, with the greatest risk associated with the highest arterial pressures.

Evidence that moderate hypertension is associated with ↑ perioperative risk is limited, although CVS lability may be ↑. In adults undergoing non-cardiac surgery, maintaining systolic arterial pressure >100mmHg and mean arterial pressure (MAP) >60–70mmHg may reduce the risk of perioperative myocardial injury, AKI and death. The degree of injury is dependent on the severity and duration of hypotension. The association of hypertension with end-organ damage (IHD, heart failure, renal failure) contributes to the likelihood of perioperative CVS complications.

Perioperative management[17]

Ambulatory NIBP measurement is the optimal method to establish baseline values. Preoperative readings are unlikely to be an accurate representation of long-term BP control.

- 1° (essential) hypertension accounts for 95% of all cases, but consider the possibility of 2° hypertension (e.g. renal parenchymal disease, thyroid dysfunction, hyperaldosteronism, phaeochromocytoma). 2° hypertension is more likely with early-onset or malignant/accelerated hypertension and has specific anaesthetic implications.
- Do not defer surgery on the basis of a single BP reading on admission to hospital. Refer to ambulatory BP reading or contact GP for a more accurate baseline record.
- Preoperative BP targets are unclear for those on antihypertensives, but both preoperative hypotension and hypertension are associated with an increase in perioperative risk.
- There is insufficient evidence to support the introduction of hypertensive medical management in the immediate preoperative period to reduce perioperative risk.
- There is currently no upper limit of MAP that necessitates medical management. Current evidence is contradictory, but expert opinion recommends postponement of elective surgery in patients with arterial pressure exceeding 180mmHg systolic and/or 110mmHg diastolic.
- The presence of end-organ dysfunction (including coronary or cerebrovascular disease, impairment of renal function, signs of LVH and heart failure) increases perioperative risk. These conditions may require further investigation and/or treatment, in addition to the control of elevated arterial BP.
- Withhold ACE inhibitor/ARB 24h prior to surgery to reduce the risk of intraoperative cardiovascular instability (use caution in heart failure).
- Continue β-blockers, calcium channel blockers and thiazide diuretics in the perioperative period.

- Intraoperative BP targets may be described as absolute thresholds (i.e. MAP >65mmHg) or relative thresholds (within 20% of baseline). Absolute thresholds are usually easier to target, especially given the uncertainty of accuracy of preoperative BP values.
- Patient-specific BP targets should be set based on patient and surgical factors. Certain surgeries (e.g. intracranial and carotid endarterectomy (CEA)) will require a narrower BP range to minimise postoperative complications.
- Postoperatively[18] maintain systolic arterial BP >90mmHg. Postoperative BP below this value is associated with ↑ risk of MINS, CVE and death. Risk is ↑ by prolonged periods of hypotension. Injury may occur at higher values in patients with pre-existing hypertension.
- Postoperative hypertension is associated with ↑ risk of MINS, CVE, arrhythmias, heart failure and surgical site bleeding.

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Valvular heart disease

Valvular heart disease is found in 4% of patients over the age of 65y. Patients with a known valve problem may already be under the care of a cardiologist. Valvular lesions most commonly occur in isolation, but multivalvular disease (most commonly degenerative in nature) occurs in ~10–20% (defined by moderate dysfunction in at least two cardiac valves).

In each case:

- Assess the significance of the pathology for the proposed surgery
- Assess for presence and progression of symptoms
- In the absence of a recent echocardiogram within 1y, or a significant change in clinical status, obtain an echocardiogram to provide an objective assessment of form and function. In the non-elective setting, point-of-care ultrasound may be useful to guide perioperative management
- In a minority of cases, treatment of the valvular pathology is more urgent than the surgery itself.

The patient with an undiagnosed murmur

Most heart murmurs do not signify cardiac disease. Many are related to physiological increases in blood flow. Assess the functional capacity (DASI; see ➲ p. 35) and for the presence or absence of symptoms. Many asymptomatic children and young adults with a murmur can safely undergo anaesthesia and surgery if they have a good functional capacity and are asymptomatic.

Elderly patients may have an asymptomatic 'aortic' systolic murmur related to sclerotic aortic valve leaflets. Aortic sclerosis is now considered to be an early form of aortic stenosis (AS) (~15% of patients with aortic sclerosis will develop AS within 7y) but should not cause clinical problems until progression to AV obstruction occurs.

Factors that differentiate early asymptomatic sclerosis from stenosis include:

- Good exercise tolerance (>4 METs)
- No history of exertional symptoms (angina/breathlessness/syncope)
- Absence of slow-rising pulse (normal pulse pressure)
- Absence of LVH/LV strain pattern on ECG.

The auditory volume of the murmur does not help.

Take a full history and examine the ECG and CXR. Patients able to manage 4 METs, with a normal ECG and CXR, should tolerate minor and intermediate surgery but should have an echocardiogram prior to major surgery. Conversely, poor functional capacity or an abnormal ECG (such as LVH or a prior MI) should be investigated by echocardiography. For urgent or emergent surgery, point-of-care ultrasound enables objective assessment of cardiac valves, ventricular function and intravascular status.

Prosthetic valves

- Bioprosthetic (tissue) valves do not require long-term anticoagulation due to low risk of thrombosis and thromboembolism.
- Mechanical valves require lifelong anticoagulation with warfarin (target INR 2–3.5, depending on type of valve).
- Direct oral anticoagulants are not currently recommended for mechanical prosthetic valves.[19]
- Aspirin is recommended, in addition to warfarin, if low risk of bleeding.
- Anticoagulation may need to be withheld, depending on the type of surgery (warfarin usually stopped for 5d preoperatively).
- Bridging with LMWH (or UFH if renal failure) may be required.
- Requirement for bridging therapy is dependent on risk of thromboembolism (e.g. mechanical mitral valve, older mechanical AV, history of thromboembolism, presence of AF).
- Newer-generation bileaflet mechanical AV has <4% per annum risk of thromboembolism, so bridging is not required in the absence of additional patient-specific risks for thromboembolism.
- Consult local policies on bridging regimes.[20]

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Aortic stenosis

AS is the commonest valvulopathy in Europe and North America. The prevalence increases exponentially with age and 2% of the general population have a congenital bicuspid valve, a risk factor for AS. The annual mortality rate in patients with symptomatic severe AS is 25%.[21] The risk of perioperative MACE is high due to anaesthetic and surgical stresses.

AS is commonly due to AV calcification. Degenerative AV sclerosis is an antecedent to clinically significant AS. Progressive valve narrowing increases afterload, leading to concentric LVH and reduced diastolic compliance. Elevated filling pressures and SR are required to fill the non-compliant LV. Normal LVEDP may reflect hypovolaemia.

Atrial contractions contribute up to 40% to the left ventricular end-diastolic volume (LVEDV) in AS (normally only 20–30%). Arrhythmias will reduce CO. There is a high risk of myocardial ischaemia due to O_2 supply–demand mismatch. Increasing LV mass increases O_2 demand. Resultant increases in systolic wall tension and intraventricular cavity pressure compress subendocardial vessels during systole, making the subendocardium vulnerable to ischaemia.

Even with normal coronary vessels, 30% of patients with AS experience angina. Tachycardia (stress, pain) further increases O_2 demand and shortens diastole, increasing compromise of coronary perfusion and increasing ischaemia. Diastolic BP is crucial to maintain coronary perfusion.

History and examination

Symptoms do not correlate well with the severity of stenosis, but exertional angina, dyspnoea and pre-/syncope may be present. On examination, there may be a slow-rising pulse with narrow pulse pressure. An ejection systolic murmur is typically maximal at the 2nd intercostal space, right sternal edge, and radiates to the neck.

Investigations

- ECG: LVH and strain (with 2° ST–T wave abnormalities).
- CXR: post-stenotic dilation of the aorta, calcified aortic annulus and signs of LV heart failure.
- Echocardiography: assesses AS severity (Table 5.8) and LV function.
- Cardiac catheterisation allows measurement of AV gradient.

Table 5.8 Aortic stenosis severity grading

	Peak velocity (m/s)	Mean gradient (mmHg)	Valve area (cm^2)
Sclerosis	<2.5	Normal	Normal
Mild	2.5–3	<20	>1.5
Moderate	3–4	20–40	1–1.5
Severe	>4	40	<1

Perioperative management[1,3,21]

Risk

- Guidelines prioritise the presence and progression of symptoms in decision-making for elective non-cardiac surgery.
- Irrespective of symptoms, a large meta-analysis demonstrated higher rates of MACE, but not mortality, in patients with AS undergoing non-cardiac surgery. Patients with AS and CAD probably represent a higher-risk population.
- With appropriate haemodynamic monitoring, it is reasonable for asymptomatic patients with severe AS to undergo elective, intermediate-risk non-cardiac surgery.
- Symptomatic patients with severe AS may be considered for valvular intervention prior to elective surgery.

Haemodynamic goals

- Low-normal HR
- Maintain SR
- Adequate preload
- Maintain systemic vascular resistance (SVR) and avoid hypotension.

Perioperative

- Patients with severe AS have a fixed CO. They cannot compensate for a reduced SVR, resulting in hypotension, myocardial ischaemia and a downward spiral of reduced contractility.
- Balance myocardial O_2 supply–demand by maintaining afterload and avoiding tachycardia ('slow and tight').
- An arterial line is extremely useful—place prior to induction.
- Titrate drugs carefully and treat hypotension promptly and aggressively. Insertion of a CVC allows reliable administration of vasoconstrictor therapy. Intraoperative TOE may be useful.
- Arrhythmias must be treated promptly, or haemodynamic collapse may ensue. Consider the role of DCCV.
- Effective analgesia avoids catecholamine-induced tachycardia and hypertension. Central neuraxial blocks (CNBs) must be used with caution due to significant reduction in afterload. Regional blocks can be used alone or in conjunction with GA.
- Have a low threshold for admission to ICU/HDU for observation and possible need for vasopressors.

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Aortic regurgitation

Aortic regurgitant lesions are better tolerated than stenotic lesions. Asymptomatic patients with normal LVEF have a favourable prognosis.

Pathophysiology

Aortic regurgitation (AR) results from diseases involving the aortic leaflets themselves, or aortic root/annulus incompetence.

- Aortic leaflet: degenerative disease, IE, rheumatic heart disease, connective tissue disease, bicuspid valve
- Aortic root/annulus: Marfan's syndrome, connective tissue disease, aortic dissection.

Acute AR (e.g. endocarditis or acute type A aortic dissection)

With a large regurgitant volume, the LV becomes overwhelmed acutely. LV diastolic pressure rises, approaching aortic diastolic pressure. Untreated, mitral regurgitation (MR) follows, leading to pulmonary oedema and circulatory failure. Treatment is emergency valve surgery.

Chronic AR

The return of blood back to the ventricle during diastole contributes to preload. ↑ LVEDV produces a compensatory increase in force of contraction and stroke volume. Aortic systolic pressure rises, but aortic diastolic pressure is reduced due to regurgitation.

- Eccentric LVH followed by LV dilation prevents significant increases in LV filling pressures despite large regurgitant volume. Progressive LV dilation increases wall stress. Initially, LVEF is maintained, followed by dilation. LV dysfunction may be masked by raised preload.
- Decompensation can be caused by further increase in LV wall stress, leading to ↓ diastolic compliance and LV systolic dysfunction. Heart failure symptoms ensue.

History, examination and investigations

Most of the symptoms are 2° to heart failure in decompensated AR, but patients may experience palpitations or angina due to reduced coronary perfusion pressure in the setting of ↑ LV wall stress.

- On examination, a collapsing ('waterhammer') pulse illustrates a wide pulse pressure resulting in: visible neck pulsation (Corrigan's sign), head nodding (de Musset's sign) or visible capillary pulsations in the nail beds (Quincke's sign). A diastolic murmur may be heard at the 2nd intercostal space, right sternal edge.
- CXR: cardiomegaly, boot-shaped heart.
- ECG: non-specific LVH.
- Echocardiogram provides quantitative analysis of the aortic leaflets and aortic root. Doppler studies allow estimation of regurgitant severity. LV size and function are assessed.

Perioperative risk[1]

- AR increases cardiac risk during non-cardiac surgery, especially if LV function is reduced.
- In asymptomatic patients with severe AR, elective intermediate-risk non-cardiac surgery is reasonable with appropriate perioperative haemodynamic monitoring.
- Patients with poor functional capacity should be considered for valve replacement surgery prior to elective surgery.

Haemodynamic goals

- High-normal HR (~90bpm)
- Adequate preload
- Low SVR
- Maintain SR
- Maintain contractility.

The selected anaesthetic technique aims to promote forward systemic flow. Maintaining a faster HR reduces diastolic regurgitation time. A reduction in SVR to the low-normal range decreases afterload while maintaining coronary perfusion pressure ('full, fast and forward').

- Spinal and epidural anaesthesia is well tolerated.
- Intra-arterial pressure monitoring is useful for major surgery and/or high-risk symptomatic patients. Non-invasive CO monitoring is inaccurate. TOE, if available, may guide optimisation of LV function.
- Treat perioperative supraventricular tachycardia (SVT)/AF promptly with synchronised DCCV (see p. 149), particularly if associated with hypotension. Persistent bradycardia may need to be treated with β-agonist or anticholinergic agents.

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Mitral stenosis

Rheumatic fever is the commonest cause. A minority have isolated stenosis; the majority have mixed mitral valve disease (stenosis and regurgitation).

- Mitral valve stenosis (MS) causes underfilling of the LV and increasing pressure and volume upstream of the valve (Table 5.9). The LV functions normally but is small and poorly filled.
- Pulmonary vascular pressures are initially maintained by left atrial dilation. As disease progresses, the pulmonary artery pressure (PAP) increases. Reactive pulmonary vasoconstriction contributes to development of pulmonary hypertension.
- Adaptive RV hypertrophy fails to compensate for volume and pressure overload, leading to progressive RV dilation and failure.
- The pressure gradient across the narrow mitral orifice increases with the square of the CO. Rapid HRs, especially with AF, decrease the diastolic filling time and markedly decrease the CO.
- Chronic left atrial dilation greatly increases the risk of AF.
- Intracardiac thrombus, either in the left atrium or left atrial appendage, may develop due to low-velocity blood flow. If present, anticoagulation is required.
- Critical MS is a fixed, low CO state.

Table 5.9 Valve area in mitral stenosis

Normal valve surface area	4–6cm^2
Symptom-free until	1.6–2.5cm^2
Moderate stenosis	1–1.5cm^2
Severe stenosis	<1.0cm^2

History

Patients complain of dyspnoea, haemoptysis and recurrent bronchitis. Fatigue and palpitations are common.

Examination

- Mitral facies: malar flush on cheeks
- Peripheral cyanosis
- Signs of right heart failure
- Tapping apex beat. Loud 1st heart sound, opening snap (if in SR) and low-pitched diastolic murmur heard best at the apex (with the bell of the stethoscope).

Investigations

- ECG: P mitrale (left atrial enlargement) if SR. AF common
- CXR: valve calcification. Large left atrium (lateral film). Double shadow behind the heart on posterior-to-anterior (PA) film. Splaying of the carina. Kerley B lines indicating pulmonary congestion
- Echocardiogram measures the gradient and valve area (Table 5.9). Allows assessments of RV function. Assesses for presence of intracardiac thrombus.

Perioperative risk[1]

Patients with MS who meet standard criteria should undergo valvular intervention prior to elective surgery (open or percutaneous mitral commissurotomy).

- In asymptomatic patients with severe MS, elective intermediate-risk non-cardiac surgery is reasonable with appropriate perioperative hsemodynamic monitoring.

Haemodynamic goals

- Low-normal HR 50–70bpm
- Maintain SR. Cardiovert if AF occurs perioperatively
- Adequate preload
- High-normal SVR
- Avoid hypercapnia, acidosis and hypoxia, which may exacerbate pulmonary hypertension.

Anaesthetic goals aim to maintain the delicate balance of adequate LV filling, while minimising decompensation of chronically ↑ pulmonary pressures.

- Tachycardia should be avoided, as ↑ HR reduces time for LV filling. Short-acting β-blockers may be used for HR control.
- Maintain an adequate afterload. However, fluid overload may precipitate acute pulmonary oedema. Measurement of CVP and pulmonary capillary occlusion pressure and TOE, if available, will guide intravascular volume management and ventricular function.
- Inotropic support may be required to optimise RV function.
- Spinal and epidural anaesthesia may be hazardous.

ALGRAWANY

Mitral regurgitation

Mitral regurgitant lesions are better tolerated than stenotic lesions. Asymptomatic patients with normal LVEF have a favourable prognosis. MR results from leaflet, chordal or papillary muscle abnormalities or as a consequence of LV dysfunction (functional MR).

- Leaflet: complication of endocarditis, rheumatic disease or mitral valve prolapse, myxomatous degeneration
- Chordal: chordae rupture after AMI or after bacterial endocarditis
- Papillary muscle: ischaemic posterior papillary muscle dysfunction
- LV failure leads to dilation of mitral valve annulus.

The left atrium is subjected to volume and pressure overload—adaptive dilation occurs gradually. As much as 50% of the LV volume flows into the dilated left atrium before the aortic valve opens. LVEF is ↑ and over time, LV dysfunction may occur. The degree of regurgitation is determined by the afterload, size of the regurgitant orifice and HR. A moderately ↑ HR (>90bpm) decreases the time for regurgitation in systole and decreases the time for diastolic filling, reducing LV overload. Chronic MR will lead to pulmonary vascular congestion, followed by pulmonary hypertension and right heart failure.

History, examination and investigations

- Fatigue, dyspnoea, palpitations, symptoms of right heart failure. Acutely, MR may present with flash pulmonary oedema
- Displaced and forceful apex due to LVH, soft S_1, apical pansystolic murmur radiating to the axilla and loud S_3
- ECG: left atrial enlargement. AF is common
- CXR: left atrial and LV enlargement. Mitral annular calcification
- Echocardiogram gives quantitative estimate of regurgitant fraction, including measuring the vena contracta (narrowest cross-sectional area of a fluid jet) which corresponds to the regurgitant orifice area and MR severity. Assess ventricular function. TOE particularly useful as the left atrium is adjacent to the oesophagus.

Mitral valve prolapse

- Common (incidental finding in 5% of population)
- Usually asymptomatic, but may be associated with atypical chest pain, palpitations, syncope and emboli
- Mid-systolic click and late diastolic murmur
- Echocardiogram shows enlarged redundant mitral valve leaflets prolapsing into the left atrium during mid- to late systole, causing arrhythmias and regurgitation
- Antiarrhythmics must be continued perioperatively.

Perioperative risk[1]

MR increases cardiac risk during non-cardiac surgery, especially if LV function is reduced.

- In asymptomatic patients with severe MR, elective intermediate-risk non-cardiac surgery is reasonable with appropriate perioperative haemodynamic monitoring.
- Patients with poor functional capacity should be considered for valve repair/replacement surgery prior to elective surgery.

Haemodynamic goals

- High-normal HR (~90bpm).
- Adequate preload.
- Low SVR.
- Low PVR.
- Maintain SR.

Anaesthetic goals aim to promote forward systemic flow. Maintaining a faster HR reduces systolic regurgitation time. Bradycardia will increase LV diastolic filling, leading to LV distension.

- A reduction in SVR to the low-normal range decreases afterload while maintaining coronary perfusion pressure. Spinal and epidural anaesthesia is well tolerated.
- Intra-arterial pressure monitoring is useful for major surgery and/or high-risk symptomatic patients.
- Preload can be difficult to estimate; for major non-cardiac surgery, a pulmonary artery (PA) catheter and/or TOE, if available, may be useful to guide intravascular volume management.
- In advanced disease, pulmonary hypertension is common. Avoid factors that increase the PAP (hypoxia, hypercapnia, high inspiratory pressures, acidosis).
- Inotropic support may be required to optimise ventricular function.

ALGRAWANY

Pericardial disease

Acute pericarditis

- Inflammatory disease of the pericardium.
- May be idiopathic, infective, non-infective (e.g. post-cardiac surgery) or autoimmune. Usually presents with chest pain. The diagnosis is supported by widespread saddle-shaped ST-elevation on ECG.
- Frequently occurs with myocarditis, which may increase the likelihood of arrhythmia and sudden death.
- Elective surgery should be postponed for at least 6w.

Constrictive pericarditis

- Chronic inflammation of the pericardium causes thickening and reduced compliance.
- Common causes include post-infective and autoimmune diseases (e.g. systemic lupus erythematosus (SLE). The only effective treatment is pericardiectomy which may be dramatically effective.
- Pulsus paradoxus (exaggerated fall in systolic BP with inspiration) may be seen due to the thickened pericardium preserving intrapericardial pressure. The normal maximum fall is 10mmHg.
- Systolic function of the myocardium is well maintained, but diastolic function is severely impaired. When exercise tolerance is reduced, GA carries a significant risk.
- Bradycardia and reduced cardiac filling are poorly tolerated.
- Elevations in intrathoracic pressure, such as during intermittent positive pressure ventilation (IPPV), can result in profound hypotension.
- If anaesthesia is unavoidable, and regional block is not possible, then a spontaneously breathing technique is preferable to IPPV. Preload should be maintained, and tachycardia avoided.

Pericardial effusion

- Excessive fluid accumulation within the pericardial space.
- Effusion may be transudate, exudate, haemorrhagic or purulent, depending on the aetiology.
- Progressive accumulation may lead to compression of cardiac chambers and tamponade.
- Non-specific symptoms include dyspnoea, orthopnoea, chest pain and tachycardia.
- Clinical signs of tamponade include Beck's triad: raised JVP, hypotension and muffled heart sounds.
- Echocardiography is a useful diagnostic tool.
- Urgent pericardiocentesis or a pericardial window may be required.
- Elective surgery should be postponed for at least 6w.

Cardiomyopathy

'A heterogenous group diseases of the myocardium associated with mechanical and/or electrical dysfunction which usually exhibit inappropriate ventricular hypertrophy or dilation.[22]

Cardiomyopathies are either confined to the heart (1°) (Table 5.10) or a manifestation of a systemic disease (2°), e.g. amyloidosis, haemochromatosis, sarcoidosis or thyroid disease.[22] Regardless of the aetiology, there is a potential final common pathway of myocardial injury, leading to ventricular dysfunction and clinical heart failure. Often, no disease-specific treatment is available. Management focuses on heart failure therapy.

There is little evidence on perioperative risk evaluation in patients with non-ischaemic cardiomyopathy undergoing non-cardiac surgery.[1,23] Markers of disease severity include poor functional capacity and reduced LVEF. Other factors associated with poor prognosis include resting tachycardia, low BP, interventricular conduction abnormalities and LV diastolic dysfunction.

Table 5.10 Causes of primary cardiomyopathy

Genetic	Hypertrophic cardiomyopathy, haemochromatosis, arrhythmogenic RV dysplasia, dilated cardiomyopathy variants, Fabry's disease
Acquired	Tachycardia-induced, stress-induced, drug-related
Mixed	Idiopathic dilated cardiomyopathy, restrictive

Stress-induced cardiomyopathy

- Also known as Takotsubo and apical ballooning syndrome.
- Reversible cardiomyopathy characterised by transient systolic dysfunction indistinguishable from AMI, but in the absence of CAD.
- Responsible for ~2% of all acute coronary syndrome events.
- Differing mechanisms have been suggested, but possibly due to catecholamine release.
- Commonly precipitated by sudden, emotionally (occasionally physiologically) stressful events.
- Commonest in women aged 58–75y (90%).
- RWMAs are beyond single vascular territory.
- Patients are often critically ill acutely: congestive cardiac failure (CCF), arrhythmias, cardiogenic shock, left ventricular outflow tract obstruction.
- Mortality is in the region of 1%.
- Symptoms resolve quickly; about 10% may reoccur.
- Treatment is supportive.

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Restrictive cardiomyopathy

- Rare condition. The commonest cause is myocardial infiltration by amyloid.
- Characterised by stiff ventricles that impair ventricular filling. Right heart failure is often prominent. Echo shows diastolic dysfunction.
- Anaesthesia is potentially associated with high risk.
- Peripheral vasodilation, myocardial depression and reduced venous return may cause catastrophic cardiovascular decompensation and may precipitate cardiac arrest.
- Venous return may be further compromised by positive pressure ventilation. Wherever possible, maintain spontaneous respiration.
- Ketamine may be useful, as it increases myocardial contractility and peripheral resistance.
- Fluids should be given to maintain elevated right heart pressures.

Haemodynamic goals

- Maintain SR
- Adequate volume loading
- High-normal SVR
- Avoid myocardial depression.

Dilated cardiomyopathy

This manifests as cardiac failure with impaired systolic function and dilation of both ventricles, predominantly the LV. Functional mitral and tricuspid incompetence occurs commonly, due to dilation of the valve annulus, exacerbating heart failure.

- The commonest problems are heart failure, arrhythmias and embolic phenomena.
- Heart failure is treated according to established guidelines (see ➔ pp. 111–15). Medical management includes ACE inhibitors, β-blockers and mineralocorticoid antagonists. Patients are frequently anticoagulated. Synchronised biventricular pacing may be used. Some patients may require an ICD for prevention of sudden cardiac death (see ➔ pp. 830–1 and p. 161).
- Peripheral nerve blocks (PNBs) minimise sympathetic activation. CNB reduces afterload. Associated hypotension must be avoided.
- Invasive monitoring with arterial and central venous catheters should be used. Intraoperative TOE, where available, will guide fluid management and ventricular support.
- Inotropic support may be provided with a variety of β-agonists and inodilators.

Haemodynamic goals

- Maintain SR. Avoid tachycardia.
- Adequate volume preload.
- Normal SVR. Avoid increases in afterload.
- Avoid myocardial depression; inotropic support is frequently required with dobutamine or PDE inhibitors.

Hypertrophic obstructive cardiomyopathy

- Hypertrophic obstructive cardiomyopathy is predominantly an autosomal dominant genetic disorder in ~60% of cases. Symptoms similar to AS. Sudden death is common.
- Accounts for two-thirds of hypertrophic obstructive cardiomyopathy patients. Hypertrophy can occur in any part of the ventricle, but the interventricular septum is commonest.
- Causes dynamic obstruction of the LV outflow during systole.
- Main feature is asymmetric hypertrophy of the interventricular septum, which obstructs the LV outflow tract during systole.
- Ventricular systole is associated with movement of the anterior mitral valve leaflet towards the septum (systolic anterior motion), causing further obstruction of the outflow tract. In some patients, this causes MR.
- As with AS, hypertrophic obstructive cardiomyopathy results in pressure overload of the LV. Diastolic dysfunction is evident on echocardiography.
- Patients are prone to arrhythmias which are refractory to medical treatment. Insertion of an ICD may be warranted for 1° prevention of sudden cardiac death.
- Symptom management includes β-blockers or verapamil, due to their negative inotropic and rate-controlling properties.
- ECG shows evidence of LVH.
- Echocardiography or cardiac MRI estimate the degree of functional obstruction, asymmetric LVH and systolic anterior motion of the mitral valve.
- SR is crucial to maintaining ventricular filling. Invasive haemodynamic monitoring is indicated. Where available, TOE is a useful guide for intravascular volume management.
- Inotropes are contraindicated as LV obstruction is exacerbated by ↑ myocardial contractility.

Haemodynamic goals

Maintain a 'large ventricle', since dynamic obstruction is reduced.

- Low-normal HR
- Maintain SR
- Adequate volume loading
- High-normal SVR
- Low ventricular contractility.

ALGRAWANY

Patients with a transplanted heart

Heart transplantation is increasing in frequency, and patients may present to a non-specialist centre for non-cardiac surgery. Anaesthesia requires attention to:

- Altered physiology
- Effects of immunosuppression
- Medications
- Associated risk factors.

Altered physiology

- The heart is denervated; resting HR is usually around 90–110bpm due to the absence of vagal tone.
- Intrinsic properties and autoregulatory functions are maintained:[21] normal Frank–Starling pressure–volume relationship, intact adrenoreceptors and normal impulse generation and conduction.
- The heart is preload-dependent. Compensation for hypovolaemia and hypotension is delayed due to denervation. An increase in catecholamines will result in an increase in chronotropy and inotropy, but this may take 5–6min.
- Reinnervation is an area of ongoing controversy. Some studies have demonstrated partial, and rarely complete, reinnervation.
- Pharmacodynamic effects are affected by denervation.[21] Direct-acting agents should be used—atropine has no effect on the denervated heart; the effect of ephedrine is reduced and unpredictable, and hydralazine and phenylephrine produce no reflex tachy- or bradycardia in response to their 1° action. Adrenaline, noradrenaline, isoprenaline and β- and α-blockers act as expected. Due to the potential for reinnervation, reversal of NMB with neostigmine should be performed with caution.
- Some patients may have experienced temporary bradyarrhythmias after transplantation. A pacemaker may be *in situ*.
- Contractility of the heart is close to normal, unless rejection is developing. In the absence of sympathetic innervation, the age-predicted maximal HR is reduced.

Immunosuppression

Three classes of drugs are used:

- Calcineurin inhibitors (ciclosporin, tacrolimus) prevent cytokine-mediated T-cell activation and proliferation.
- Nucleic acid synthesis inhibitors (azathioprine) block lymphocyte proliferation.
- Steroids block the production of inflammatory cytokines, lyse T-lymphocytes and alter the function of the remaining lymphocytes.

Effects of treatment

- Anaemia and thrombocytopenia, as well as leucopenia, may result, requiring treatment before surgery.[24]
- Ciclosporin is associated with renal dysfunction and is the most likely cause of the hypertension that affects 40% of heart–lung transplant recipients. It may also prolong the action of non-depolarising muscle relaxants (NDMRs).

- Calcium antagonists increase ciclosporin levels variably and are used in some centres to reduce the ciclosporin dose in an attempt to reduce side effects. The effect on blood concentrations must be remembered if calcium antagonists are omitted for any reason perioperatively.
- Renal dysfunction is also commonly caused by tacrolimus.
- Steroid supplementation may be required if large doses of prednisolone are being used.

Associated risk factors

- Previous, and often repeated, use of central and peripheral vessels can make IV and arterial access difficult.
- Cough may be impaired due to a combination of phrenic and recurrent laryngeal nerve palsies. This increases the risks of sputum retention and postoperative chest infection.
- Heart–lung recipients will have a tracheal anastomosis. It is desirable to avoid unnecessary intubation, but if it is necessary, use a short tube and carefully monitor the tracheal cuff pressure. Disrupted lung lymphatic drainage increases the risk of pulmonary oedema.[21]
- The transplanted heart may still develop CAD.

Choice of technique

There is no evidence to support one anaesthetic technique above another.

- Peripheral surgery under regional block is likely to be well tolerated.
- Subarachnoid or epidural block may result in marked falls in BP because of absent cardiac innervation.
- Strict asepsis must be used with all invasive procedures.

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Congenital heart disease and non-cardiac surgery

Congenital heart disease (CHD) is common (1 in 150 births), with >85% of affected children reaching adult life. Although many will have undergone corrective surgery, residual problems may be present. There is a higher incidence of adverse perioperative events in CHD patients undergoing non-cardiac surgery, although risk varies markedly across the cohort.

General considerations

Procedures for CHD aim to improve the patient's haemodynamic status, although complete normalisation is not always achieved. They may broadly be divided as follows:

- Corrective procedures: the lesion is completely repaired, with no long-term sequelae (e.g. PDA and ASD closure). In some cases, a residual defect may be present (e.g. free pulmonary regurgitation after tetralogy of Fallot repair (ToF); see also ➲ p. 137), requiring repeat interventions and/or reducing life expectancy.
- Palliative procedures: these patients may have abnormal circulations and physiology but avoid the consequences of untreated CHD. Life expectancy is not normal, but many survive to adulthood (e.g. Fontan procedures; see also ➲ p. 137).

Preoperative assessment

Aim to understand the patient's cardiac anatomy and pathophysiology, with a view to risk stratification and perioperative planning.

- History: define the severity of the lesion in terms of impact on normal activities and growth. Consider relevant comorbidity (e.g. CHARGE syndrome). Check current medication and obtain a recent cardiology review.
- Examination: check for cyanosis and signs of infection/failure. Consider neurological examination for cyanotic patients.
- Investigations: CXR/ECG/echocardiogram. Review cardiac catheter data if available. Baseline peripheral O_2 saturation (SpO_2) in air. Lab tests depend on the proposed surgery, but most will require FBC and electrolytes.
- Consider: whether surgery should proceed, in view of the risks vs potential benefit, if the patient can or should be moved to a cardiac centre and if postoperative ICU/HDU will be required.
- Anticipate the likely impact of anaesthetic interventions (e.g. the effect of high fractional inspired oxygen content (FiO_2) on PVR, anaesthetic drugs on myocardial function and SVR, positive pressure ventilation on systemic venous return).

Factors suggestive of increased perioperative risk

- Pulmonary hypertension, particularly if RV pressures are systemic or near-systemic; any further rise in PVR can cause acute RV decompensation and cardiac arrest. In this context, the presence of an ASD or VSD can be protective; in the event of a pulmonary hypertension crisis, a source of right-to-left shunt may allow CO to be maintained (albeit at the expense of oxygenation).
- Severe ventricular outflow obstruction (e.g. Williams syndrome).

- 'Duct-dependent' or single ventricle physiology.
- Cardiomyopathy (e.g. HCM).
- Severe hypoxaemia (e.g. SpO_2 <75% on air) or polycythaemia (e.g. haematocrit (Hct) >60%).
- Severe or worsening CCF or evidence of myocardial ischaemia.
- Other markers of haemodynamic insufficiency: syncope, arrhythmias or recent CVE.

Specific considerations

- Cyanosis results from shunting of blood from the right to the left side of the heart through a focal defect (e.g. ToF) or complete intracardiac mixing (e.g. single ventricle lesions, complete atrioventricular septal defect (AVSD)). Adaptations to chronic hypoxaemia include polycythaemia and ↑ blood volume. Blood viscosity is ↑, impairing tissue perfusion. Thrombosis (e.g. cerebral/renal) due to polycythaemia and coagulopathy due to platelet dysfunction and fibrinogen deficiency may occur. ↑ tissue vascularity may worsen bleeding.
- Air emboli: any intracardiac shunt (particularly right-to-left) poses the risk of air entering the coronary or cerebral circulation, with potentially catastrophic consequences. Meticulous 'bubble precautions' should be taken.
- 'Duct-dependent' or single ventricle physiology. In order to be compatible with life, some lesions (e.g. tricuspic atresia, hypoplastic left heart syndrome, severe aortic coarctation) require a patent ductus arteriosus to maintain blood flow to either pulmonary or systemic vascular beds; they will require a continuous prostaglandin infusion to achieve this. These patients should be managed in specialist centres as they are extremely sensitive to changes in PVR/SVR, myocardial contractility and preload. Aim to keep physiological parameters (e.g. SpO_2) as close to what is normal for the patient as far as possible. Patients dependent on a Blalock–Taussig shunt (see ➲ p. 137) to maintain pulmonary perfusion share similar characteristics.
- Eisenmenger's syndrome. Irreversible elevation in PVR occurring in response to chronic high pulmonary flow (i.e. any large left-to-right shunt that is left untreated). Shunt reversal occurs, with consequent cyanosis. The degree of shunting depends on the PVR:SVR ratio. Increasing SVR or decreasing PVR leads to higher arterial SpO_2. Avoid reductions in SVR (neuraxial anaesthesia) and rises in PVR (hypoxia/hypercapnia /acidosis). Associated with ↑ morbidity and mortality.

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Specific congenital heart disease lesions

There are >100 forms of CHD, but the commonest lesions are: ASD, VSD, PDA, ToF, AS, coarctation of the aorta and transposition of the great arteries. Lesions not amenable to biventricular repair are usually managed palliatively by producing a Fontan circulation (also known as a total cavopulmonary connection).

Atrial septal defect (secundum type)

- A defect in the membrane of the fossa ovalis, accounting for 80% of ASDs. Often asymptomatic.
- Results in a left-to-right shunt with right heart volume overload.
- Can be closed surgically or transcatheter.

Atrial septal defect (primum type)

- Endocardial cushion (or partial atrioventricular (AV) canal) defect—may involve AV valves.
- The more severe form AVSD is associated with Down's syndrome and results in severe pulmonary hypertension if left untreated (see ➲ pp. 139–44).
- Surgical repair of these lesions may result in complete heart block.

Ventricular septal defect

- Commonest form of CHD, resulting in a left-to-right shunt which, in turn, causes ↑ pulmonary blood flow and left heart volume overload. As with any lesion causing excessive pulmonary blood flow, they are at risk of developing pulmonary hypertension and shunt reversal (see ➲ p. 135, Eisenmenger's syndrome).
- A small single VSD may be asymptomatic with a small left-to-right shunt (pulmonary:systemic flow (Q_p:Q_s) ratio <1.5:1).
- A moderate VSD (Q_p:Q_s ratio 2–3:1) may present with mild CCF.
- Patients with a large VSD have equal pressures in their right and left ventricles and present at around 2mo of age with severe CCF. They require early repair. However, if they need anaesthesia for another procedure prior to definitive cardiac surgery, they can be challenging. They should be intubated for all but the most minor procedures, and increases in the left-to-right shunt should be avoided (e.g. avoid hyperventilation and high FiO_2). Care should be taken with fluid administration, and inotropic support may be required.
- Those unsuitable for definitive repair (e.g. very small babies, multiple muscular VSDs) may require PA banding to protect the pulmonary circulation, preventing the development of pulmonary hypertension. This band tightens as the child grows, leading to RV pressure loading and progressive cyanosis. The band is removed when the child is large enough for full repair or the defects have closed spontaneously.
- May be closed transcatheter in certain circumstances.

Patent ductus arteriosus

- Represents 10% of CHD lesions. Incidence is 20–30% in preterm neonates. Produces a left-to-right shunt, resulting in CCF and raised PVR. Diastolic run-off from the proximal aorta to the PA can compromise distal organ perfusion (e.g. gut, kidneys).
- Can be closed surgically or transcatheter.

Tetralogy of Fallot

- Pulmonary stenosis, VSD, overriding aorta and RV hypertrophy.
- May be prone to cyanotic episodes ('tet spells') due to intermittent RV infundibular spasm causing ↑ right-to-left shunting. If this occurs under anaesthesia, treatment includes: 100% O_2, IV fluid, adequate analgesia/anaesthesia to reduce circulating catecholamines and increasing SVR (e.g. by bringing the knees towards the chest and/or IV vasopressors).
- Before full repair, may be treated medically with β-blockers or surgically via a modified Blalock–Taussig shunt—a Gore-Tex® tube interposed between the subclavian and pulmonary arteries.
- Full repair of ToF is usually undertaken in the first 6mo of life.

Transposition of the great arteries

- Accounts for 7–8% of CHD, in which the aorta arises from the RV and the PA from the LV; a VSD occurs in ~30%.
- In order to be compatible with life, there must be a source of mixing (i.e. PDA, ASD or VSD) to allow oxygenated pulmonary venous blood to reach the systemic circulation.
- Preoperative management usually consists of a prostaglandin infusion ± balloon atrial septostomy if mixing is inadequate.
- Repair is usually with an arterial switch procedure in the first 2w of life.

Hypoplastic left heart syndrome and the Fontan circulation

- Hypoplastic left heart syndrome accounts for around 2% of CHD and carries a significant mortality risk, with ~70% surviving to 5y.
- Patients with hypoplastic left heart syndrome undergo a 3-stage repair, usually involving a neonatal Norwood operation, followed by a Glenn shunt at 3–6mo, and finally a completion Fontan procedure (also called a total cavopulmonary connection) at 3–5y.
- After total cavopulmonary connection, all superior vena cava (SVC) and IVC blood flows directly into the PA, bypassing the right heart. Pulmonary blood flow is thus passively dependent on systemic venous pressure.
- The aim is to volume-offload the RV, while providing acceptable arterial saturations (75–85%); however, the Fontan circulation inevitably leads to high venous pressures, liver congestion, protein-losing enteropathy and pleural and pericardial effusions.
- Hypovolaemia can lead to hypoxia and CVS collapse. IPPV results in a fall in CO, and high ventilatory pressures result in poor pulmonary perfusion.
- These children are particularly vulnerable and should be managed in a specialist centre, whenever possible.

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Adults with congenital heart disease

Anything but the most straightforward situation should be discussed, and the patient referred to a congenital cardiac centre.

Uncorrected disease

- ASDs/VSDs may be small and of no haemodynamic significance. Apart from the risk of paradoxical emboli, small defects present with no anaesthetic issues.
- Large left-to-right shunts will cause progressive pulmonary hypertension and eventual shunt reversal (Eisenmenger's syndrome). Once irreversible pulmonary hypertension has developed, surgical correction is not possible. These patients are at high risk. If surgery is absolutely necessary, it should be performed in a specialist centre.

Corrected disease

- These patients have either had spontaneous resolution or a corrective procedure. They can generally be treated as normal.
- Best assessment of CVS function is generally exercise tolerance.
- Exclude surgical sequelae/continuing disease.
- Exclude any associated congenital abnormalities.

Palliated disease

- These patients have had operations that improve functional capacity and life expectancy but do not restore normal anatomy. Operations include Senning and Mustard for transposition of the great arteries (arterial switch is now preferred) and Fontan pathway for single ventricle lesions (e.g. hypoplastic left heart syndrome and pulmonary atresia).
- An understanding of the underlying physiology is required to avoid complications when anaesthetising these patients. Management is best provided in specialist cardiac centres.
- In patients with a Fontan circulation, blood leaves a single ventricle and passes through the systemic circulation, then through the pulmonary circulation, before returning to the heart. As a result, the systemic venous pressure is high, providing a pressure gradient across the pulmonary vascular bed. Any pulmonary hypertension is poorly tolerated and results in reduced ventricular filling. The high venous pressure can result in life-threatening haemorrhage from mucosal procedures such as adenoidectomy (or nasal intubation!).

Pulmonary hypertension

Pulmonary hypertension describes a situation of elevated pressure in the pulmonary circulation. Pulmonary hypertension has many aetiologies and can complicate most cardiovascular and respiratory conditions (Table 5.11). Although the aetiology may be different, the sequelae of endothelial dysfunction, vasoconstriction and vascular remodelling are common.[25]

Normal mean pulmonary artery pressure (mPAP) is 14mmHg. Pulmonary hypertension is defined as a mPAP ≥25mmHg measured by right heart catheterisation at rest.

- Mild mPAP 25–40mmHg
- Moderate mPAP 41–55mmHg
- Severe mPAP >55mmHg.

Pulmonary artery hypertension (PAH) is distinct from the other classes of pulmonary hypertension and is defined as pre-capillary pulmonary hypertension with PA occlusion pressure ≤15mmHg and PVR >3 Wood units, in the absence of other causes of pre-capillary pulmonary hypertension. PAH accounts for 45% of pulmonary hypertension, with chronic thromboembolic pulmonary hypertension being the second commonest (19%), followed by left heart disease. Pulmonary hypertension is classified into groups of similar pathophysiological mechanisms, clinical presentation and haemodynamic characteristics (Table 5.11).[26]

Table 5.11 Updated clinical classification of pulmonary hypertension

Class	Aetiology	Examples
1	PAH	Idiopathic PAH, drug- or toxin-induced, associated with CHD/portal hypertension/HIV or persistent pulmonary pulmonary hypertension of the newborn
2	Pulmonary hypertension due to left heart disease	Pulmonary hypertension due to heart failure (HFpEF and HFrEF), valvular disease and congenital cardiomyopathies
3	Pulmonary hypertension due to lung disease and/or hypoxia	Obstructive, restrictive or mixed lung disease, hypoxia without lung disease and developmental lung disorders
4	Pulmonary hypertension due to pulmonary artery obstructions	Chronic thromboembolic pulmonary hypertension and other PA obstructions
5	Pulmonary hypertension with unclear mechanism	Haematological disorders, systemic and metabolic disorders, complex CHD

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Symptoms and signs

Symptoms are non-specific and initially exercise induced, mainly related to RV dysfunction and low cardiac output state, such as dyspnoea, fatigue, angina and syncope. Rupture or distension of vessels in the pulmonary vascular bed may cause symptoms, e.g. haemoptysis and hoarse voice (compression of left recurrent laryngeal nerve). Other symptoms related to the causative disease process may also be present. Examination may reveal signs of right heart failure, a pansystolic murmur (tricuspid regurgitation) and a diastolic murmur (pulmonary regurgitation).

Diagnosis of pulmonary hypertension

- Right heart catheterisation is the gold standard for diagnosis as mPAP can be measured directly.
- TOE is often the 1° investigation due to its availability. Pulmonary artery systolic pressure (PASP) is estimated from peak tricuspid regurgitation velocity and right atrial pressure can be used to stratify pulmonary hypertension. Note the stratification is different to that obtained from right heart catheterisation (mild pulmonary hypertension: PASP 35–45mmHg; moderate pulmonary hypertension: PASP 46–59mmHg; severe pulmonary hypertension: PASP >60mmHg).
- The aetiology of pulmonary hypertension may also be diagnosed by echocardiography.
- ECG: may be normal or show P pulmonale, right axis deviatio, RV hypertrophy, right bundle branch block, RV strain or prolonged corrected QT.
- The 6MWT is the most widely used test for prognostication.[27]
- CPET is becoming more widely used for therapeutic decision-making and adds to information from the 6MWT.[28]

Other tests should then be used to delineate the aetiology[25]

- CXR: COPD and sarcoidosis
- Computed tomography pulmonary angiography or V/Q scan: thromboembolic disease
- Sleep studies: OSA
- Liver ultrasound: portopulmonary hypertension
- Cardiac magnetic resonance imaging (MRI): cardiomyopathy and CHD.

Severity

Severity of pulmonary hypertension can be classified by mPAP and PASP (see above) or functionally using the NYHA classification (Table 5.12).

Treatment

PAH remains an incurable condition. Treatment advances in the last decade have slowed progression and improved symptoms, but prognosis remains poor. Pulmonary hypertension-specific therapies (Table 5.13) are licensed for PAH only. In the UK, only pulmonary hypertension specialists are able to prescribe them.

Treatment options for other classes of pulmonary hypertension largely depend on treating the underlying cause. Patients with chronic thromboembolic pulmonary hypertension should be referred for consideration for pulmonary endarterectomy and they may be also considered for some pulmonary hypertension-specific therapies as per PAH.[25]

Table 5.12 New York Heart Association functional classification of pulmonary hypertension

Class	
1	Pulmonary hypertension with no symptoms or limitation to ordinary physical activity
2	Pulmonary hypertension with mild symptoms and slight limitation during ordinary activity
3	Pulmonary hypertension with marked limitation of physical activity. Comfortable at rest
4	Pulmonary hypertension with signs of right heart failure, possibly even at rest. Inability to carry out any activity without symptoms

Table 5.13 Treatment for pulmonary artery hypertension

Treatment	Notes
Non-specific treatment	Anticoagulation (idiopathic PAH only) Diuretics for decompensated right heart failure O_2 reduces PVR, but no evidence for long-term O_2 therapy
Calcium channel blockers	<10% of patients benefit
Synthetic prostacyclin analogues (epoprostenol, iloprost, treprostinil)	Epoprostenol shown to improve survival in idiopathic PAH Treprostinil and inhaled iloprost shown to improve symptoms and reduce clinical events
Endothelin receptor antagonists	Macitentan reduces progression of PAH and death. Ambrisentan and bosentan improve symptoms ± progression
PDE-5 inhibitors	Sildenafil and tadalafil improve exercise capacity and symptoms
Guanylate cyclase stimulation	Riociguat beneficial in PAH
Transplantation	Heart–lung and double-lung transplantation improves survival in idiopathic PAH

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Anaesthesia

(See ➲ pp. 524–5 for cardiac surgery and pulmonary hypertension.)

Evidence is lacking, but postoperative mortality after non-cardiac surgery ranges from 4–20% depending on the series. Although RV contractility can be normal in mild disease, systolic dysfunction is common and progresses with worsening class of pulmonary hypertension. Systemic BP is usually low (disease- and treatment-related). Table 5.14 lists some of the factors that are used to assess the 1y mortality risk.[27] Consider referring any patient in the intermediate- or high-risk categories to a specialist pulmonary hypertension centre for their surgery.

Risk factors associated with increased perioperative mortality and morbidity[25]

- NYHA class ≥2
- 6MWT <300m
- PVR >400dyn.s.cm^{-5}
- Elevated right atrial pressure (>14 mmHg)
- Low cardiac index in right heart catheterisation (<2.4L/m^2)
- History of: CAD, PE, chronic kidney disease (CKD) and RV hypertrophy with severe systolic dysfunction
- Emergency surgery
- Intermediate-/high-risk surgery
- Duration of surgery >3h
- Intraoperative vasopressor requirement.

Table 5.14 Some determinants used to estimate 1y mortality (mainly based on expert opinion)

	Low risk <5%	Medium risk 5–10%	High risk >10%
Clinical signs	Absent	Absent	Present
Progressive symptoms	No	Slow	Rapid
WHO functional class	I	II–III	IV
6MWT	>440m	165–440m	<165m
CPET	Peak VO_2 >65% predicted VE/VCO_2 <36	Peak VO_2 35–65% predicted VE/VCO_2 36–44.9	Peak VO_2 <35% predicted VE/VCO_2 >45
Biochemical markers	BNP <50 nanograms/L NT-proBNP <300 nanograms/L	BNP 50–300 nanograms/L NT-proBNP 300–1400 nanograms/L	BNP >300 nanograms/L NT-proBNP >1400 nanograms/L
Imaging	Right atrial area <18cm^2 No pericardial effusion	Right atrial area 18–26cm^2 No/minimal pericardial effusion	Right atrial area >26cm^2 Pericardial effusion

Source: data from Galiè N *et al*., 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension), *European Heart Journal*, 2016;**37**(1):67–119, https://doi.org/10.1093/eurheartj/ehv317.

Perioperative management[25]

⚠ Maintain RV and pulmonary circulation coupling, and prevent a pulmonary hypertension crisis.

- Many anaesthetic drugs affect RV contractility and PVR (Table 5.15).
- Basic haemodynamic goals:
  - Maintain systolic BP at baseline if possible, or at least a minimum systolic pressure of 90mmHg and 40mmHg above systolic PAP.
  - MAP ≥65mmHg and 20mmHg above mPAP.
  - Lowest right atrial pressure to maintain MAP >65mmHg.
- Give supplementary O_2 (O_2 is a pulmonary vasodilator); avoid hypoxic pulmonary vasoconstriction.
- Avoid hypothermia (causes pulmonary vasoconstriction and V/Q mismatch).
- Invasive arterial monitoring to aid rapid response to changes in BP and allow for intermittent sampling.
- Intraoperative TOE or PA catheter should be considered in all patients with severe pulmonary hypertension or evidence of right heart failure.
- Obtund sympathetic response to laryngoscopy to avoid increase in PVR and atrial tachyarrhythmias, which can lead to right heart failure and death. For rhythm control, amiodarone is the drug of choice; use digoxin if rhythm control not possible. β-blockade is poorly tolerated.

Table 5.15 Effect of anaesthetic drugs in pulmonary hypertension

Medication	Effect
Isoflurane, desflurane, sevoflurane	Marked, dose-dependent reduction in RV contractility Increase in PVR (not sevoflurane)
Propofol	Reduces RV contractility, RV ejection fraction and cardiac index
Thiopental	Reduced RV contractility and SVR
Ketamine	Increases PVR (adults only)
Vasopressors: noradrenaline, vasopressin	Improve perfusion of right coronary artery, reduce PVR/SVR ratio, enhance RV contractility and marginally improve CO
Inotropes: adrenaline, dobutamine, levosimendan	Increase RV contractility
Inodilators: PDE-3 inhibitors (e.g. milrinone)	Reduce PVR and improve PVR/SVR ratio. Reduction in SVR can compromise coronary artery blood flow
IV vasodilator: sildenafil	Can be given PO and IV, so could be an option for patients already on oral therapy
Inhaled pulmonary vasodilators	Nitric oxide, epoprostenol, prostacyclin, iloprost and treprostinil have all been used perioperatively, but in small numbers

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- Use low-dose vasoconstrictor to maintain systolic BP if needed.
- Conventional ventilation with low PEEP is generally well tolerated (PEEP reduces atelectasis, mPAP and shunt but increases RV afterload, as does hypercapnia and acidosis).
- Patients are preload-dependent but tolerate overload poorly. Fluid therapy should be targeted and restrictive. Consider CVP monitoring to estimate baseline preload. Blood loss should be replaced promptly.
- CNB is safe, but acute hypotension must be avoided. Gradual lumbar epidural anaesthesia may be considered to minimise rapid block of sympathetic fibres. Increases in both HR and pulmonary afterload cause reflex increases in RV contractility via thoracic sympathetic fibres. This homeometric autoregulation maintains coupling between the RV and pulmonary circulation. Blocking these fibres with a thoracic epidural could cause a critical reduction in CO, leading to right heart failure.
- Prepare for events during surgery which may affect preload, afterload or pulmonary flow, e.g. cementing during orthopaedic surgery, microemboli after tourniquet release, pneumoperitoneum during laparoscopic surgery, cross-clamping of major blood vessels and change of patient positioning.
- Pain should be planned for and treated aggressively.
- HDU/ICU postoperatively.
- Common causes of postoperative death are right heart failure, arrhythmias, sepsis, PE, respiratory failure and MI.

Pregnancy and pulmonary hypertension[25]

- Mortality is still high and patients should be advised accordingly.
- Most centres favour elective CS over vaginal delivery.
- There is no consensus on whether GA or regional anaesthesia is better; maternal morbidity remains around 20%.
- Deaths occur due to RHF and PE.

Perioperative arrhythmias

(See also ➲ Chapter 39.)

Perioperative cardiac arrhythmias are common and may result in significant morbidity and mortality. In the setting of pre-existing arrhythmia, rate and rhythm control should be optimised prior to surgery, as perioperative stressors can lead to marked deterioration.

Preoperative recognition of potential risk factors and triggers may allow prevention and/or rapid management of arrhythmia.

When providing IV drug treatment for arrhythmia management, ensure a defibrillator is readily available in case of rhythm deterioration.

Risk factors for new-onset perioperative arrhythmia or deterioration of pre-existing arrhythmia may be classified according to patient factors and context-specific triggers (Table 5.16).

Table 5.16 Risk factors for perioperative arrhythmia

Patient factors	Pre-existing cardiac disease (e.g. IHD, conduction abnormality, valvular disease, heart failure) Advanced age Electrolyte disturbance Hormonal imbalance (e.g. thyroid dysfunction, phaeochromocytoma) Due to acute disease process (e.g. LRTI, sepsis, SAH, anaemia, PE) Obstructive sleep apnoea
Anaesthetic triggers	Intubation Drug-induced (e.g. anticholinergics, LA toxicity) Hypoxia Inadequate pain management Electrolyte abnormalities Intraoperative hypotension Insertion of central vascular catheters
Surgical triggers	Cardiothoracic surgery Pneumoperitoneum Activation of ANS reflex (e.g. oculocardiac reflex in ophthalmic surgery, peritoneal traction) Intravascular fluid shifts

ANS, autonomic nervous system; IHD, ischaemic heart disease; LA, local anaesthetic; LRTI, lower respiratory tract infection; PE, pulmonary embolism; SAH, subarachnoid haemorrhage.

Practical diagnosis of arrhythmias

- Assess ABC.
- Ensure appropriate monitoring is applied: SpO_2, ECG and NIBP.
- Provide O_2 if hypoxic.
- Ensure adequate IV access.
- Assess for presence of adverse features (shock, syncope, myocardial ischaemia or heart failure). If present, emergency management is required.

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Arrhythmia assessment is ideally undertaken with a 12-lead ECG. This is often impractical intraoperatively. Alternative leads may be interrogated using a traditional 3- or 5-lead intraoperative ECG configuration.

Determine

- What is the ventricular rate?
- Is the QRS complex of normal duration or widened?
- Is the QRS regular or irregular?
- Are P waves present, and are they normally shaped?
- How is atrial activity related to ventricular activity?

Ventricular rate

- Calculate the approximate ventricular rate (divide 300 by the number of large squares between each QRS complex).
- Tachyarrhythmia: rate >100bpm; bradyarrhythmia: rate <60bpm.

QRS complex

- Supraventricular rhythms include nodal rhythms and arise from a focus above the ventricles. Since the ventricles still depolarise via the normal His/Purkinje system, the QRS complexes are of normal width (<120ms or three small squares) and are termed 'narrow complex rhythms'.
- Arrhythmias arising from a ventricular focus will be 'broad complex' with a QRS width of >0.12s. In the presence of AV or bundle branch block, a supraventricular rhythm may have broad complexes (2%).

Regularity

- Irregular rhythm suggests ectopic beats (atrial or ventricular), AF, atrial flutter with variable block or 2nd-degree heart block with variable block.

P waves

- The presence of P waves indicates atrial depolarisation. Absent P waves with an irregular ventricular rhythm suggest AF, whereas a sawtoothed pattern is characteristic of atrial flutter.

Atrial/ventricular activity

- Normally, there will be one P wave per QRS complex. Any change in this ratio indicates a block to conduction between atria and ventricles.

Narrow complex arrhythmias

Narrow complex arrhythmias are characterised by QRS complexes <0.12s.

Sinus arrhythmia

Irregular spacing of normal complexes associated with respiration. Constant P–R interval, with beat-to-beat change in the R–R interval. Normal finding, especially in young people.

Sinus bradycardia

Rate <60bpm, with normal P–QRS–T complexes. Unnecessary to correct in a fit person with no haemodynamic compromise (usually when HR >40bpm). May be normal in athletic patients or due to vagal stimulation. Other causes include: drugs (e.g. β-blockers, digoxin, anticholinesterases, halothane, suxamethonium), MI, sick sinus syndrome, raised intracranial pressure (ICP), hypothyroidism and hypothermia. Correct the underlying cause, e.g. stop the surgical stimulus. Incremental doses of atropine up to 20 micrograms/kg, or glycopyrronium up to 10 micrograms/kg IV. Patients on β-blockers may be resistant, and an isoprenaline infusion is occasionally required (0.5–10 micrograms/min) (adrenaline is an alternative).

Temporary cardiac pacing is rarely required.

Sick sinus syndrome

May coexist with AV nodal disease. Episodes of AV block, atrial tachycardia, atrial flutter and AF may occur.

- The commonest causes are congenital and advanced age.
- May be asymptomatic or present with dizziness or syncope.
- Consider permanent pacing if symptomatic.

Atrial ectopics

These are common and benign. Causes include: ischaemia/hypoxia, inadequate depth of anaesthesia, sepsis, shock and anaesthetic drugs.

Correct any underlying cause, but treatment unnecessary if solitary.

Long QT syndrome

- Can be genetic or drug-induced and causes prolonged ventricular repolarisation due to abnormalities in the cardiac ion channels. It can lead to polymorphic VT or torsade de pointes and subsequent ventricular fibrillation (VF). Around 60% of patients are symptomatic (syncope, seizure-like episodes, cardiac arrest).
- Patients should have preoperative cardiology assessment, as there is a high risk of perioperative malignant ventricular arrhythmias which may be refractory.
- Avoid medications that have well-documented or moderate association with prolonged QT (e.g. ondansetron, octreotide, macrolides, amiodarone, droperidol) and use caution with drugs with ↑ potential to precipitate torsade de pointes in genetic long QT (e.g. ephedrine, phenylephrine, adrenaline, cocaine).
- Perioperative management includes β-blockade.
- Normalise all electrolytes and prevent sympathetic activation (pain, sedative premedication, laryngoscopy, extubation, hypercapnia, etc.).

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- Invasive monitoring is advisable. Maintain temperature (hypothermia prolongs QT) and adequate analgesia is essential.
- For torsade de pointes management, see ➲ p. 153.
- Consider postoperative ICU.

Regular supraventricular tachycardia

Sinus tachycardia

Rate >100bpm with normal P–QRS–T complexes. Most often an appropriate autonomic response to a physiological stress.

Causes include:

- Inadequate depth of anaesthesia, pain, surgical stimulation
- Fever/sepsis, hypovolaemia, anaemia, heart failure, thyrotoxicosis
- Drugs, e.g. atropine, ketamine, catecholamines.
- Correct the underlying cause where possible.

AV nodal re-entry tachycardia

Often paroxysmal with sudden onset. Rate 150–200bpm.

Caused by re-entry circuit within the AV node comprising two pathways with different conduction speeds. P wave often obscured by QRS complex due to almost simultaneous activation of atria and ventricles.

Terminates with adenosine administration.

AV re-entry tachycardia

Sudden onset. Rate 150–200bpm. Caused by abnormal myocardial fibres that form an accessory pathway bypassing the AV node. Conduction through this pathway may be ante- or retrograde. ECG morphology depends on the direction of electrical travel through the re-entry circuit. In orthodromic conduction (via AV node with retrograde conduction through accessory pathway), P waves occur after narrow QRS complex. In antidromic conduction (via accessory pathway with retrograde conduction through AV node), there are regular wide QRS complexes due to slow ventricular depolarisation. Terminates with adenosine administration.

Wolff–Parkinson–White (WPW) syndrome

A type of congenital AV re-entry tachycardia with evidence of pre-excitation; early activation of ventricles due to bypass of AV node via an accessory pathway. Classic ECG pattern of pre-excitation include: short PR interval (<120ms) and delta wave (Fig. 5.1). Emergent treatment is DCCV.

Fig. 5.1 This patient has WPW syndrome as they have delta waves (slurred QRS upstrokes) in beats 1 and 4 on this rhythm strip. The delta wave both broadens the ventricular complex and shortens the PR interval. If a patient with WPW has AF, avoid AV node blockers such as diltiazem, verapamil and digoxin—but flecainide may be used. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al*. *Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

Atrial flutter

Often caused by a re-entry circuit at the tricuspid valve. Associated with cardiac disease. Atrial depolarisation is ~300bpm. However, ventricular rate is determined by AV conduction, resulting in an AV block. The commonest AV ratio is 2:1. Higher degrees of AV block can occur, usually due to drugs which lower ventricular conduction.

Associated risk of thromboembolism similar to AF.

Acute management entails:

- Synchronised DCCV if adverse features are present or new onset under anaesthesia. Requires sedation or GA, but lower energy (70–120J) may suffice.[29] Nearly 100% conversion.
- If haemodynamically stable: vagal manoeuvres, carotid sinus massage or Valsalva manoeuvre may terminate re-entry SVT and may be helpful in differentiating SVT from atrial flutter and fast AF. Adenosine blocks AV nodal conduction and is especially useful for terminating re-entry SVT. Give 6mg IV rapidly, followed by a large 0.9% sodium chloride flush into a large proximal vein (e.g. antecubital). Record ECG during the injection. If rate slows, look for atrial activity to aid diagnosis. If no effect, give 12mg IV bolus. The effects of adenosine last only 10–15s. It should be used with caution in asthma due to risk of bronchospasm.
- Acute rate and rhythm control: β-blockers (rate) or amiodarone (rhythm), as per treatment for AF below. Digoxin should be avoided as it facilitates conduction through the AV accessory pathway in WPW syndrome and may worsen tachycardia.

Irregular supraventricular tachycardia

Atrial fibrillation

- The most commonly sustained arrhythmia after surgery. Associated with increase in length of stay, incidence of postoperative CVE and in-hospital mortality.[30] Table 5.17 illustrates strategies to prevent perioperative AF.[31]
- Disordered generation of atrial electrical impulses leads to uncoordinated atrial activity that is conducted intermittently and irregularly to the ventricles via the AV node (Fig. 5.2).
- Atrial contraction contributes up to 30% of ventricular filling. The onset of AF, particularly in the setting of rapid ventricular rate, causes a reduction in ventricular filling and CO.
- Thrombi may form within the atria, due in part to blood stasis, and embolise systemically. Annual risk of thromboembolic events and need for anticoagulation may be estimated with prognostic models (i.e. CHA_2DS_2-VASc).
- ECG demonstrates: irregularly irregular rhythm without P waves, narrow QRS (<120ms) unless conduction abnormality (e.g. pre-existing bundle branch block), variable ventricular rate and absence of isoelectric baseline. Aberrancy, or functional conduction delay, refers to the broadening of the QRS as the rate gets faster.

Management of acute atrial fibrillation

- Goal: restore CO and ensure adequate oxygenation.
- Determine presence of adverse features: shock, syncope, myocardial ischaemia and heart failure.[31]

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- In patients with no adverse features present, evaluate underlying causes and correct precipitating factors where possible. Either a rate or a rhythm control management strategy is reasonable.
- If adverse features present, perform urgent synchronised DCCV. Start at 120–150J, and increase incrementally if required.
- Anticoagulation should be started as soon as possible and continued for at least 4w; but ideally lifelong if otherwise not contraindicated. (See Table 5.18.)[32]

Table 5.17 Prevention of new-onset AF or rapid ventricular rate in pre-existing/paroxysmal AF

Preoperative	Assess patient risk factors Formulate plan for perioperative anticoagulation Continue AV nodal-blocking drugs until morning of surgery (e.g. β-blockers, CCBs) Investigate causes of new-onset AF Consider delaying surgery in patients with RVR
Intraoperative	Avoid and treat anaesthetic-/surgery-related triggers Treat unstable AF RVR with electrical cardioversion Treat stable AF RVR with rate control agents Avoid arrhythmogenic drugs (i.e. ketamine, adrenergic vasopressors, glycopyrronium)
Postoperative	Consider CPAP in patients with OSA POCUS in patients with haemodynamic instability Treat unstable AF with electrical cardioversion Treat stable AF with rate control agents Continue rate control agents at discharge until follow-up Antithrombotic therapy when appropriate

AV, atrioventricular; CCB, calcium channel blocker; OSA, obstructive sleep apnoea; POCUS, point-of-care ultrasound; RVR, rapid ventricular rate.

Karamchandani K, Khanna AK, Bose S; et al (2020) Atrial fibrillation: current evidence and management strategies during the perioperative period. *Anesth Anal* 130(1); 2-13.

Table 5.18 Anticoagulation for AF rhythm control

Low risk CHA_2DS_2-VASc 0 <48h duration	Consider anticoagulation prior to DCCV Anticoagulation not needed post-cardioversion
Higher risk CHA_2DS_2-VASc ≥1 <48h duration	Start anticoagulation ASAP prior to DCCV Long-term anticoagulation recommended
>48h or unknown duration	Anticoagulation ≥3w prior to DCCV

Acute ventricular rate control[33]

- β-blockers—esmolol 0.5mg/kg IV bolus over 1min; then 0.05–0.25mg/kg/min or metoprolol 2.5–10mg IV bolus. Caution in HFrEF. Cardioselective β-blockers are unlikely to adversely affect respiratory disease.
- Calcium channel blockers (diltiazem 15–25mg IV or verapamil 2.5–10mg IV, repeated as required). Use with caution in combination with β-blockers. Contraindicated in HFrEF.
- Amiodarone may be used as 1st-line rate control in patients with HFrEF, as digoxin is often ineffective in high adrenergic states.[34]

Acute rhythm control[33]

DCCV restores SR quicker and more effectively than pharmacological cardioversion, but pharmacological cardioversion does not require sedation/GA or fasting. Amiodarone: 300mg IV loading dose (in 5% glucose) via central vein over 1h, followed by 900mg over 23h. May be given via peripheral line in an emergency but has a high incidence of thrombophlebitis. Can be combined with digoxin or β-blockers. May precipitate bradycardia/AV block/hypotension.

Fig. 5.2 (a) AF: note the irregular spacing of QRS complexes and lack of P waves. (b) AF with a rapid ventricular response (sometimes referred to as 'fast AF'). No pattern to QRS complex spacing, and rate >100bpm. (c) Atrial flutter with 2:1 block (two P waves for every one QRS complex). The P waves have the classic 'sawtooth' appearance. Alternate P waves are merged with the QRS complex. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

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Broad complex arrhythmias

Ventricular ectopics

In the absence of structural heart disease, these are usually benign, but in patients with ventricular disease, they may occasionally herald the onset of runs of ventricular tachycardia (VT) (Fig. 5.3).

- Identify and correct any reversible causes (e.g. hypoxia, ↓ K^+, ↑ Mg^{2+}).
- If the underlying sinus rate is slow (<50bpm), then 'ectopics' may be ventricular escape beats. Try increasing the rate using IV atropine or glycopyrronium.

Fig. 5.3 Bigeminy—a normal QRS is followed by a ventricular ectopic beat (*), then a compensatory pause; this pattern then repeats. The ectopic beats have the same morphology as each other, so probably all share an origin. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

Ventricular tachycardia

This is a serious, potentially life-threatening arrhythmia. The QRS is always wide. P waves may be seen if there is AV dissociation (Fig. 5.4). May be monomorphic (a single ventricular focus with uniform QRS complexes) or polymorphic (multiple ventricular foci with an irregular QRS morphology).

Perioperative triggers include:

- Myocardial ischaemia, hypoxia and hypotension
- Fluid overload
- Electrolyte imbalance (low K^+, Mg^{2+}, etc.)
- Injection of adrenaline or other catecholamines
- Drugs which prolong the QT interval (e.g. amiodarone, ondansetron, haloperidol).

Fig. 5.4 VT with a rate of 235/min. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

Management[29,34]

- Confirm the presence of CO (central pulse, arterial line waveform, capnography).
- If no evidence of CO, commence cardiopulmonary resuscitation (CPR) and advanced cardiovascular life support with immediate defibrillation.

- If evidence of CO, assess for adverse features. If present, promptly perform synchronised DCCV (start at 120–150J; increase if required). If the patient relapses into VT, amiodarone may be given to sustain SR.
- If no adverse features present, treat with amiodarone 300mg IV, preferably via a CVC, over 20–60min, followed by 900mg over 23h.

Torsade de pointes

A type of polymorphic VT occurring in the context of prolonged QT. Defined by a characteristic of the QRS complexes twisting around the ECG isoelectric line, with a high risk of degenerating into VF (Fig. 5.5). Often associated with haemodynamic compromise, necessitating immediate defibrillation.[33] Even if there is evidence of CO, defibrillation is performed as synchronisation is not possible.

If stable, give Mg^{2+} 2g IV loading dose over 10–20min. Continue Mg^{2+} infusion at 1–4g/h. Monitor plasma levels. Consult cardiology for ongoing management and preventative therapy.

Do not give amiodarone as QT interval will be prolonged further.

Fig. 5.5 Torsade de pointes tachycardia. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

Supraventricular tachycardia with aberrant conduction

An SVT may be broad complex due to aberrant conduction between the atria and ventricles. This may appear only at high HRs (rate-related aberrant conduction). SVT caused by an abnormal or accessory pathway (e.g. WPW syndrome) will be of normal width if conduction in the accessory pathway is retrograde (i.e. it is the normal pathway that initiates the QRS complex), but broad complex if conduction is anterograde in the accessory pathway. Adenosine may be used diagnostically to slow AV conduction and may reveal the underlying rhythm in atrial flutter or atrial tachycardia. In the case of SVT, it may also result in conversion to SR. In practice, however, all broad complex tachycardias should be treated as VT until proven otherwise.

Ventricular fibrillation

- This results in cardiac arrest. There is chaotic and disorganised contraction of the ventricular muscle, and no QRS complexes can be identified on the ECG (Fig. 5.6).
- Immediate DCCV as per established resuscitation protocol (200J) (see ➲ pp. 1055–7).

Fig. 5.6 VF. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

A L G R A W A N Y

Disturbances of conduction (heart block)

First-degree block

Delay in conduction through the AV node to the ventricles. Prolongation of the P–R interval to >200ms. Normally benign but may progress to 2nd- or 3rd-degree block (Fig. 5.7).

Second-degree block—Mobitz type I (Wenckebach)

Progressive lengthening of the P–R interval and then failure of conduction of an atrial beat. This is followed by a conducted beat, and the cycle repeats. Common in young athletic adults with high vagal tone. May occur during inferior MI and tends to be self-limiting. Asymptomatic patients do not normally require treatment perioperatively but may require long-term pacing, as Wenckebach block may progress to higher degrees of block (Fig. 5.7).

Second-degree block—Mobitz type II

Intermittent atrial depolarisation without a subsequent ventricular beat (2:1 or 3:1 are common forms). This often progresses to complete heart block (Fig. 5.7).

Third-degree block/complete heart block

Complete failure of conduction between the atria and ventricles. Occasionally, a transient phenomenon due to severe vagal stimulation. Very rarely, it may be congenital (Fig. 5.7).

Bundle branch block

If there is a delay in depolarisation of the right or left bundle branches, this will cause a delay in depolarisation of part of the ventricular muscle, with subsequent QRS widening.

Right bundle branch block

Wide complexes with an 'RSR' in lead V_1 (may appear 'M'-shaped) and a small initial negative downward deflection, followed by a larger upward positive wave, and then a 2nd downward wave in V_6. Often benign but may be indicative of myocardial disease (Fig. 5.8).

Left bundle branch block

Septal depolarisation is reversed, with change in the initial direction of the QRS complex in every lead. Left bundle branch block is nearly always representative of a disease process. Further interpretation of the ECG, other than the rate and rhythm, is difficult in the presence of left bundle branch block (Fig. 5.9).

Bifascicular block

Combination of right bundle branch block and block of the left anterior or left posterior fascicle. Right bundle branch block with left anterior hemiblock is more common and appears as an 'RSR' in V_1, together with left axis deviation. Right bundle branch block with left posterior hemiblock is less common and appears as right bundle branch block with an abnormal degree of right axis deviation. However, other causes for right axis deviation should be considered, and it is a non-specific sign (Fig. 5.10).

Trifascicular block
Sometimes used to indicate the presence of a prolonged P–R interval together with bifascicular block.

P P P P

First-degree AV block. P–R interval = 0.28s.

P P P P

Mobitz type I (Wenckebach) AV block. With each successive QRS, the P–R interval increases until there is a non-conducted P wave.

P P P P P

Mobitz type II AV block. Ratio of AV conduction varies from 2:1 to 3:1.

P P P P P P P

Complete AV block with narrow ventricular complex.
There is no relation between atrial and the slower ventricular activity.

Fig. 5.7 Rhythm strips of heart blocks. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

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Preoperative management

- First-degree heart block in the absence of symptoms is common. It needs no specific investigation or treatment.
- Second- or 3rd-degree heart block may need pacemaker insertion. If surgery is urgent, this may be achieved quickly by inserting a temporary transvenous wire prior to definitive insertion.
- Bundle branch, bifascicular or trifascicular block (bifascicular with 1st-degree block) will rarely progress to complete heart block during anaesthesia, and so it is not common practice to insert a pacing wire, unless there have been episodes of syncope.

Indications for preoperative pacing

- Symptomatic 1st-degree heart block
- Symptomatic 2nd-degree (Mobitz type I) heart block
- Second-degree (Mobitz type II) heart block
- Third-degree heart block
- Symptomatic bifascicular block or symptomatic 1st-degree heart block plus bifascicular block (trifascicular block)
- Symptomatic sinus node disease.

Intraoperative heart block

- Atropine is rarely effective but may be administered.
- If hypotension is profound, then an isoprenaline infusion (alternative is adrenaline) can be used to temporise: 1–10 micrograms/min.
  - Dilute 1mg in 50mL of 5% glucose/glucose–0.9% sodium chloride, and titrate to effect (1.5–30mL/h).
- Transcutaneous pacing may be practical intraoperatively if electrodes can be placed.
- Transvenous pacing is both more reliable and effective, and relatively easy. A PA catheter introducer of adequate size to pass the wire is inserted into the internal jugular or subclavian vein.
  - Insert balloon-tipped pacing wire to the 20cm mark.
  - Inflate the balloon and connect the pulse generator at 5V.
  - Advance until ventricular capture. When this happens, deflate the balloon and insert a further 5cm of catheter.
  - If the 50cm mark is reached and the catheter is coiling up or not entering the heart, deflate the balloon; withdraw to the 20cm mark, and try again.

Fig. 5.8 Right bundle branch block—broad QRS, M pattern in V_1 and sloped S wave (with the eye of faith, a 'W' shape) in V_5, MaRRoW = RBBB. Reproduced with permission of Oxford Publishing Limited through PLSclear from Ramrakha P *et al. Oxford Handbook of Cardiology*. © Oxford University Press 2012; © Punit Ramrakha and Jonathan Hill (Editor's contribution) 2012.

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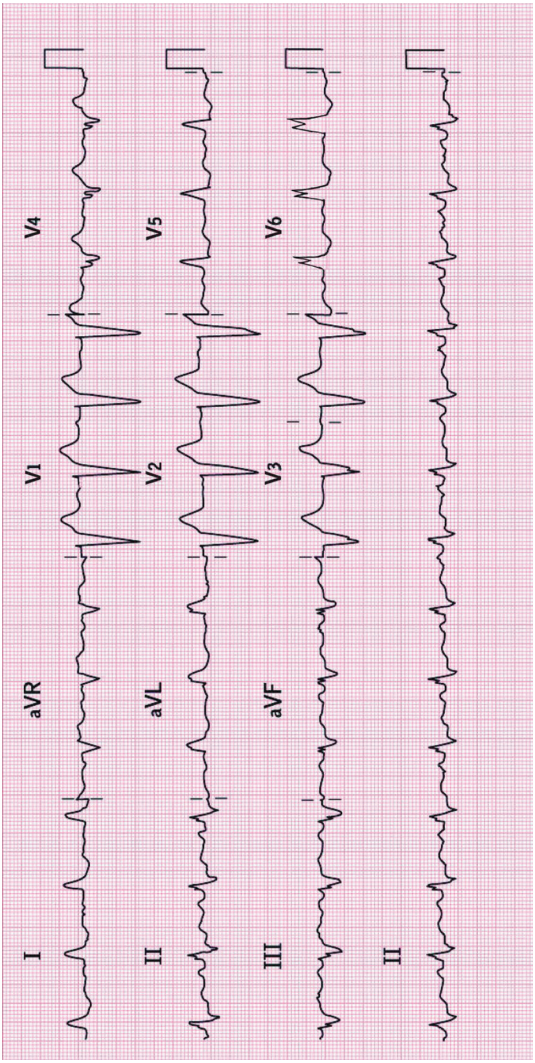

Fig. 5.9 Left bundle branch block: wide QRS with a W pattern in V_1 (slight notching in upstroke of S wave—clearer in V_3) and the M pattern in V_6. WiLLiaM = LBBB. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

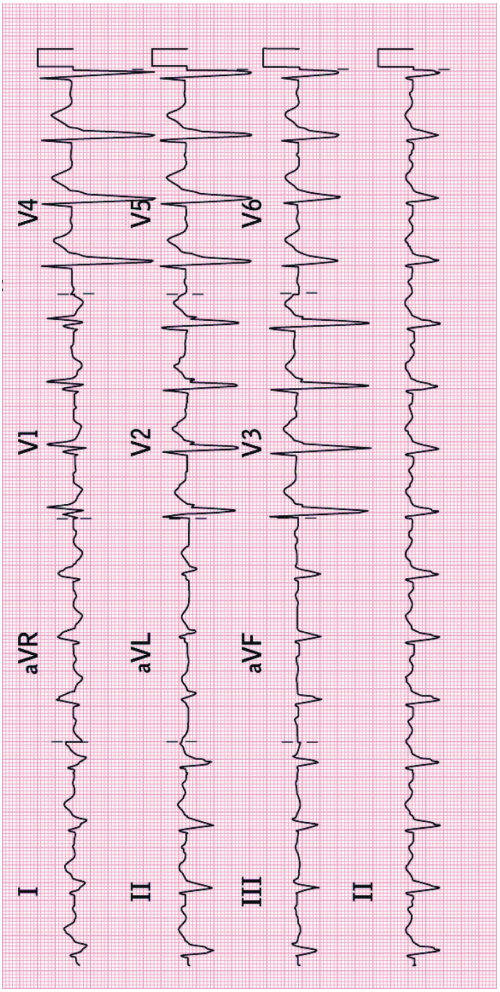

Fig. 5.10 ECG showing bifascicular block. There is a wide QRS complex with an RSR pattern in V_1 and a deep, slurred S wave in V_6 (= RBBB). The QRS in lead 1 is positive and lead aVF negative (= left anterior hemiblock). There is also second-degree AV nodal block (Mobitz type I or Wenckebach). Observing the rhythm strip from the 1st P wave, there is a gradually prolonging PR interval and the P wave that follows the 6th QRS complex is blocked. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

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Pacemakers and defibrillators

Pacemakers are usually used to treat bradyarrhythmias (Fig. 5.11). However, biventricular systems are used to improve the functional capacity and quality of life in selected patients with severe heart failure.

The Heart Rhythm Society and Heart Rhythm UK pacemaker codes are used to describe pacemaker types and function (Box 5.2). The code consists of five letters or positions. The first three describe antibradycardia functions and are always stated. The 4th and 5th positions relate to additional functions.

Box 5.2 US/UK pacemaker positions (and possible settings)

- Position 1: chamber-paced (O/V/A/D)
- Position 2: sensing chamber (O/V/A/D)
- Position 3: response to sensing (O/T/I/D)
- Position 4: programmability or rate modulation (O/P/M/R)
- Position 5: antitachycardia functions (O/P/S/D)

O, none; A, atrial; V, ventricular; D, dual; P, programmable; M, multiprogrammable; R, rate modulation; S, shock; T, trigger; I, inhibit.

Fig. 5.11 ECG of a paced rhythms strip. Reproduced with permission of Oxford Publishing Limited through PLSclear from Wilkinson I *et al. Oxford Handbook of Clinical Medicine*. © Oxford University Press 2017.

Implications for anaesthesia and surgery

The most recent permanent pacemaker check should confirm an adequate battery life, normal function of the pacemaker system and pacemaker dependency. A preoperative ECG will provide confirmation of the expected function, e.g. AV synchronicity, polarity and baseline rate.

- Electromagnetic interference (most commonly diathermy) can cause inappropriate triggering or inhibition of a paced output, asynchronous pacing, reprogramming (usually into a backup mode) and damage to device circuitry. Pacing wires may also act as aerials and cause heating where they contact the endocardium.
- Bipolar diathermy is safe. If unipolar diathermy is necessary, position the plate so that current passes away from the pacemaker and use brief bursts and the lowest possible amplitude.
- Pacemakers should be set to the asynchronous (VOO) mode in permanent pacemaker-dependent patients. A cardiac electrophysiologist may be consulted for temporary reprogramming. Alternatively, a medical-grade magnet may be applied over the pulse generator. Postoperative pacemaker interrogation by a cardiac electrophysiologist will ensure appropriate programming and sensing-pacing thresholds.

Implantable cardioverter–defibrillators

ICDs should be deactivated prior to surgery where diathermy might be used (the ICD will detect the signal as VF and deliver a shock). A magnet placed over the ICD inhibits their function. The patient should be monitored throughout surgery, and then the device reactivated postoperatively. While deactivated, an external defibrillator should be immediately available, with pads applied pre-emptively.

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Chapter 6

Respiratory disease

Sarah Jarvis

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Assessment of respiratory function

A complete history and physical examination are the most important elements of preoperative risk assessment.[1,2] (See ➲ Chapter 2 for generic information about perioperative medicine.)

History

- Identify significant risk factors for postoperative pulmonary complications (see ➲ pp. 168–9).
- Note details of hospital admissions with respiratory disease, particularly any intensive care admissions.
- If chronic lung disease, compare current respiratory function with previous disease trends.
- Explore symptoms such as cough and sputum production. Send a sputum specimen for culture and sensitivity.
- Note past and present cigarette consumption.
- Review current treatment, reversibility of symptoms with bronchodilators and steroid intake.
- There is evidence that screening for sleep apnoea affects surgical complication rates. It is advisable to question obese patients about symptoms suggestive of OSA prior to major surgery (see ➲ pp. 73–5).[3]
- Beware of a history suggesting undiagnosed chronic lung disease, e.g. exercise intolerance or unexplained dyspnoea.
- Using Roizen's classification, undiagnosed dyspnoea of grade II or worse may require further investigation (Table 6.1).

Examination

- Abnormal findings are predictive of pulmonary complications after abdominal surgery.[1]
- Look for evidence of obstructive lung disease. Signs such as ↓ breath sounds, wheeze or ↑ expiratory phase are important.

Investigations

Preoperative respiratory investigations used to inform preoperative risk assessment are outlined in Table 6.2.

Table 6.1 Roizen's classification of dyspnoea

Grade 0	No dyspnoea while walking on the level at normal pace
Grade I	'I am able to walk as far as I like, provided I take my time'
Grade II	Specific street block limitation: 'I have to stop for a while after one or two blocks'
Grade III	Dyspnoea on mild exertion: 'I have to stop and rest, going from the kitchen to the bathroom'
Grade IV	Dyspnoea at rest

Table 6.2 Preoperative respiratory investigations

	Uses/notes	Advantages	Disadvantages
SpO_2	Good screening tool for all patients	Easily available Gives baseline May prompt further investigations Helps to stratify risk Useful before high-risk surgery[4]	Non-specific
Informal stair climb	Immediate qualitative understanding of degree of dyspnoea	Easy to do with a patient in clinic if stairs nearby Informs further investigation	Informal test Impractical if limited mobility
Peak expiratory flow rate (PEFR)	COPD/asthma assessment	Ward-based test/patient's own home record	Technique-dependent
CXR	Investigating dyspnoea/stridor	Easy to do Informative	Small dose of radiation Most findings are chronic/predictable and do not affect management
Spirometry*	Differentiates between restrictive and obstructive lung defects Quantifies severity of defects Valuable test for lung resection surgery candidates	Can be done at bedside/in laboratory Forced expiratory volume in 1s (FEV_1) <1L predicts likelihood of needing respiratory support post-major surgery due to poor cough	Recent evidence that spirometry does not predict pulmonary complications, so it is not a routine test prior to high-risk surgery[5] and not to be used as the 1° reason to deny surgery
Flow–volume loops	To assess extrinsic and intrinsic airway obstruction	More accurate information (than obtained with spirometry) on ventilatory function and upper airway obstruction	Requires specialist respiratory equipment Complex to interpret Poor effort mimics the flow seen in upper airway obstruction
ABGs	To quantify actual or suspected chronic CO_2 retention SpO_2 <92% may influence postoperative SpO_2 target vs baseline	Easy to do in outpatients Instant results Helpful to compare with previous results	Invasive Painful

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	Uses/notes	Advantages	Disadvantages
Transfer factor/ diffusing capacity of the lung for carbon monoxide	Evaluation of restrictive and obstructive lung disease, as well as pulmonary vascular disease	Quick and safe	
CT thorax	Assess extent of lung disease, e.g. bullae/cysts/ fibrosis/mass lesions impinging major airways High-resolution spiral CT for PE/ aortic dissection detection	Informative Now more routinely available	Radiation exposure Need to lie flat may challenge those with both intrinsic and extrinsic respiratory failure
Ventilation/ perfusion (V/Q) scan	Reports likelihood of PE Assesses patients undergoing lung parenchymal resection to predict effect of surgery on pulmonary performance		Radiation exposure Hard to interpret in presence of other pathology
6MWT (see ➔ p. 32)	Assessment of degree of dyspnoea	Correlates well with pulmonary function tests (spirometry and lung volumes) Provides a reliable test of pulmonary function	Also reflects CVS status, cooperation and determination Impractical for those with limited mobility
CPET (see ➔ pp. 33–5)	Used to differentiate dyspnoea of unknown cause (CVS vs respiratory)	Provides data on ventilatory efficiency and response to exercise Helpful in predicting mortality, morbidity and length of stay[6]	As with 6MWT Requires specialist knowledge to interpret

* See Table 6.3.

Table 6.3 Gradation of severity of airflow obstruction

	FEV_1 % predicted
Stage 2: moderate	50–79%
Stage 3: severe	30–49%
Stage 4: very severe	<30% or <50% with respiratory failure

Source: data from National Institute of Clinical Excellence (2018) *Chronic obstructive pulmonary disease in over 16s: diagnosis and management* NICE guideline 115. Updated July 26, 2019.

Effects of surgery and anaesthesia on respiratory function

Effects of surgery

- Upper abdominal operations are associated with pulmonary complications in 20–40% of the general surgical population.[1]
- Incidence with lower abdominal surgery is 2–5%.
- Upper abdominal or thoracic surgery is associated with a profound ↓ in lung volume; vital capacity (VC) is ↓ by 50–60%; FRC is ↓ by about 30%. Diaphragmatic dysfunction, pain and splinting are important contributing factors.
- A ↓ lung volume is not seen with surgery on the extremities.

Effects of anaesthesia

- On induction of anaesthesia, FRC is ↓ by 15–20% (~450mL); the diaphragm relaxes and moves cranially; the rib cage moves inward.
- FRC may be ↓ by 50% of the awake supine value in morbidly obese patients. PEEP may reduce these effects. FRC is relatively maintained during ketamine anaesthesia.
- Under anaesthesia, the closing capacity (the lung volume at which airway closure begins) encroaches upon the FRC, contributing to the risk of atelectasis, pneumonia and V/Q mismatching. This happens more readily in smokers, the elderly and those with underlying lung disease.
- Chest CT shows atelectasis in the dependent zones of the lungs in >80% of anaesthetised subjects. Microatelectasis results in areas of the lungs that are perfused but not ventilated, leading to impaired gas exchange and consequent postoperative hypoxaemia.
- Intubation halves the dead space by circumventing the upper airway.
- The ventilatory response to hypercapnia is blunted, and the acute responses to hypoxia and acidaemia almost abolished by anaesthetic vapours at concentrations as low as 0.1 MAC.
- Inhibition of cough and impairment of mucociliary clearance of secretions contribute to the risk of postoperative infection.
- Most of these adverse changes are more marked in patients with lung disease but usually improve within a few hours postoperatively. After major surgery, they may last several days.

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Predicting postoperative pulmonary complications

Postoperative pulmonary complications include atelectasis, infection, prolonged mechanical ventilation, respiratory failure, exacerbation of underlying chronic lung disease and bronchospasm.[7]

- They are as prevalent as cardiac complications and contribute similarly to morbidity and mortality.
- They are ↓ by preoperative identification of patients with pre-existing respiratory dysfunction.
- Even for patients with severe pulmonary disease, surgery that does not involve the abdominal or chest cavities is inherently of low risk for serious perioperative pulmonary complications.

Predictors of perioperative pulmonary complications

Patient factors

- Increasing age (>50y)
- COPD
- Smoking within 8w of surgery
- OSA[8]
- Pulmonary hypertension[9]
- ASA grade 2 or greater
- CCF
- Functional dependence
- Serum albumin <30g/dL.

Procedure-related factors

- Prolonged surgery
- Residual NMB[10]
- Upper abdominal and thoracic surgery
- Neurosurgery, head and neck surgery
- Vascular surgery, especially aortic aneurysm repair
- Emergency surgery.

Risk calculators

(For risk assessment, see ➲ pp. 36–8.)

There are a number of tools used to evaluate risk of respiratory complications postoperatively:

- Arozullah: two scoring systems exist, one to predict postoperative pneumonia, and the other to predict postoperative respiratory failure
- Assess Respiratory Risk in Surgical Patients in Catalonia (ARISCAT)
- Gupta
- American College of Surgeons National Surgery Quality Improvement Programme (ACS NSQIP) universal calculator.

The Arozullah and ARISCAT indices detailed below provide a simplified approach to preoperative pulmonary risk estimation and are useful tools to stratify risk and identify those patients most likely to benefit from risk reduction strategies.

Arozullah respiratory failure risk index[11]

(See Table 6.4.)

- Procedure-related risk factors dominate this index, with type of surgery and emergency surgery being the most important predictors.
- Scores predict postoperative respiratory failure: <10pts 0.5%, 11–19pts 1.8%, 20–27pts 4.2%, 28–40pts 10.1%, >40pts 26.6%.

Table 6.4 Arozullah respiratory failure risk index

Predictor	Score
Age 60–69y	4
Age ≥70y	6
COPD	6
Partially or fully dependent functional status	7
Blood urea nitrogen >30mg/dL	8
Albumin <3g/dL	9
Neck surgery	11
Neuro, upper GI, peripheral vascular surgery	14
Thoracic surgery	21
Abdominal aortic surgery	27
Emergency surgery	11

Reproduced from Arozullah A *et al*., Multifactorial risk index for predicting postoperative respiratory failure in men after major noncardiac surgery, *Annals of Surgery*, **232**(2), 242–53, with permission from Wolters Kluwer Health, Inc. © 2001.

ARISCAT risk index

(See Table 6.5.)

Scores predict postoperative pulmonary complications: <26pts 1.6%, 26–44pts 13.3%, >45pts 42.1%.

Table 6.5 ARISCAT score for postoperative pulmonary complications

Predictor	Score
Age 51–80	3
Age >80	16
Preoperative SpO_2 91–95%	8
Preoperative SpO_2 ≤90%	24
Respiratory infection within 1mo	17
Preoperative Hb ≤100g/L	11
Upper abdominal incision	15
Intrathoracic incision	24
Surgery 2–3h	16
Surgery ≥3h	23
Emergency	8

Reproduced from Canet J *et al*., Prediction of Postoperative Pulmonary Complications in a Population-based Surgical Cohort, *Anesthesiology*,113(6), 1338–50, with permission from Wolters Kluwer Health, Inc. © 2010. https://pubs.asahq.org/anesthesiology/article/113/6/1338/9603/Prediction-of-Postoperative-Pulmonary.

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Strategies to reduce postoperative pulmonary complications

Preoperative

(See ➲ Chapter 3 for generic information on preoptimisation.)

- Smoking cessation as early as possible[12] (see ➲ p. 50; ➲ p. 173).
- Regular ipratropium or tiotropium for all patients with clinically significant COPD.
- Inhaled β-agonists, as required, for patients with COPD or asthma who have wheeze or dyspnoea.
- Preoperative glucocorticoids for COPD or asthma patients who are not optimised and whose airway obstruction is not maximally ↓.
- Delay elective surgery if respiratory tract infection is present (see ➲ p. 173).
- Antibiotics only for patients with lower respiratory tract infection (LRTI).
- Preoperative inspiratory muscle training and chest physiotherapy. This involves breathing exercises, aerobic exercise, incentive spirometry (deep breathing facilitated by a simple mechanical device), education on active breathing and forced expiration techniques.

Intraoperative

- Procedures <4h duration preferable.
- Regional anaesthesia if appropriate. The benefits of opioid-based analgesia (patient control, mobility and avoidance of bladder catheterisation) should be weighed against the benefits of regional analgesia (avoidance of high-dose systemic opioids, preservation of respiratory function) and discussed with the patient preoperatively.
- Epidural or spinal anaesthesia may confer lower risk than GA, though this remains an area of debate.
- Avoid long-acting muscle relaxants if possible. Residual NMB is associated with hypoventilation which increases postoperative complications.
- Choose laparascopic or endoscopic surgery over open surgery if possible.
- Lung-protective ventilation (low tidal volume (V_T) 6–8mL/kg of IBW, PEEP 6–8cmH_2O and recruitment manoeuvres every 30min) is associated with a reduction in adverse pulmonary events. The IMPROVE study showed the rate of postoperative pulmonary complications after abdominal surgery was significantly lower in the group receiving low V_T ventilation.[13]

Postoperative

- Respiratory performance, FRC and clearance of secretions are improved when sitting or standing, compared with the supine position. Aim for upright posture postoperatively if possible.
- Monitor the respiratory rate (RR) and O_2 saturation. Respiratory deterioration may present in a non-specific way (confusion, tachycardia, fever, malaise). Regular review to trigger urgent investigation and aggressive therapy.

- Seek assessment and advice of the intensive care/outreach team early if the patient does not respond to initial treatment.
- Early postoperative chest physiotherapy aids clearance of secretions and decreases atelectasis.
- Administer supplemental O_2 for up to 72h postoperatively, particularly if the patient is receiving opioids. Anaesthetic agents exert a dose-dependent depression on the sensitivity of central chemoreceptors, reducing the stimulatory effect of CO_2. This effect can occur for up to 72h postoperatively and is commonest at night. Preoperative measurement of PaO_2, SaO_2 and $PaCO_2$ is essential to establish realistic targets for each patient.
- Humidification of O_2 aids physiotherapy and sputum clearance.
- High-flow O_2 may be useful (up to 70L/min). This must be warmed and humidified to reduce drying of secretions and mucous membranes. It can improve oxygenation and ventilation by washing out of pharyngeal dead space, decreasing inspiratory effort, meeting inspiratory flow rates and provision of some CPAP.
- CO_2 retainers (advanced COPD) may be dependent on hypoxaemia as their main ventilatory drive due to downregulation of central chemoreceptors. The concentration of delivered O_2 should be controlled (e.g. by Venturi mask) and titrated in order to optimise oxygenation and prevent hypoventilation. Adequate monitoring should be available, ideally using serial ABG measurement (pulse oximetry shows only SpO_2).
- Accurate management and documentation of fluid balance is essential. Adequate intravascular filling is required to maintain perfusion of vital organs. However, patients with lung disease are at ↑ risk of pulmonary oedema. (A dilated RV may mechanically compromise the function of the LV.) Fluid overload is poorly tolerated in these patients. Maintain a high index of suspicion.
- Good analgesia is essential for the maintenance of efficient respiratory function, compliance with physiotherapy, early mobilisation and minimising cardiac stress. Regular paracetamol and (where not contraindicated) NSAIDs should be prescribed.
- Involve the pain management team to ensure adequate analgesia.

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Postoperative admission to HDU/ICU

- Ideally, admission to ICU or HDU should be planned preoperatively (see ➲ p. 43 for generic information on postoperative planning).
- Patients may require admission for ventilatory support (CPAP, bilevel positive airway pressure (BiPAP), invasive ventilation) or ↑ levels of monitoring and nursing care that are not available on the surgical ward.
- The precipitating reasons for admission to the ICU or HDU are listed in Table 6.6.

Table 6.6 Reasons for postoperative ICU or HDU admission

Predictable	Unpredictable
Borderline or established failure of gas exchange preoperatively	Unexpected perioperative complications (e.g. fluid overload, haemorrhage)
Intercurrent respiratory infection (with urgent surgery)	Inadequate or ineffective regional analgesia with deterioration in respiratory function
Chest disease productive of large amounts of secretions (e.g. bronchiectasis)	Unexpectedly prolonged procedure
Major abdominal or thoracic surgery	Acidosis
Major surgery not amenable to regional analgesia and necessitating systemic opioids	Hypothermia
Long duration of surgery	Depressed conscious level/slow recovery from anaesthetic/poor cough

Smoking and respiratory tract infections

Smoking

- Cigarette smoke contains nicotine, a highly addictive substance, and ≥4700 other chemical compounds, 43 of which are carcinogenic.
- Respiratory tract mucus is produced in greater quantities, but mucociliary clearance is less efficient.
- Smokers are more susceptible to respiratory events during anaesthesia and to postoperative atelectasis/pneumonia. Abdominal or thoracic surgery and obesity increase these risks.
- Carboxyhaemoglobin (COHb) levels may reach 5–15% in heavy smokers, causing reduced O_2 carriage by blood. COHb has a similar absorption spectrum to oxyhaemoglobin and will cause falsely high O_2 saturation readings.

Risk reduction

- Abstinence from smoking for 8w is required to decrease respiratory complications to a rate similar to that of non-smokers.[12]
- Smokers unwilling to stop preoperatively will still benefit by refraining from smoking for 12h before surgery. During this time, the effects of nicotine (activation of the sympathoadrenergic system with raised coronary vascular resistance) and COHb will decrease.
- ↑ airway irritability risks ↑ coughing, laryngospasm and desaturation during induction and airway manipulation. Minimise risk by using a less irritant volatile agent (e.g. sevoflurane) and deepening anaesthesia slowly.
- Maintaining spontaneous breathing via an endotracheal tube (ETT) or supraglottic airway (SGA) may be awkward due to airway irritation. Consider LA to the vocal cords, opioids, NMBAs and IPPV.

Respiratory tract infections

(For respiratory tract infections in children, see ➲ pp. 913–14.)

- Patients who have respiratory tract infections producing fever and cough, with or without chest signs on auscultation, should not undergo elective surgery under GA due to ↑ risk of postoperative pulmonary complications.
- Adult patients with simple coryza are not at significantly ↑ risk of developing postoperative pulmonary problems, unless they have pre-existing respiratory disease or are having major abdominal or thoracic surgery.[14]
- Laryngospasm may be more likely in patients with a recent history of upper respiratory tract symptoms who are asymptomatic at the time of surgery.
- Compared with asymptomatic children, children with symptoms of acute or recent upper respiratory tract infection (URTI) are more likely to suffer from transient postoperative hypoxaemia (SpO_2 <93%). This is most marked when intubation is necessary.[15]

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Asthma

Asthma[16,17,18] is a disorder of variable severity, which causes symptoms resulting from airway obstruction, inflammation and hyper-responsiveness.

- Symptoms are most frequently a combination of shortness of breath, wheeze, cough and sputum production.
- Bronchial wall inflammation is a fundamental component and results in mucus hypersecretion, epithelial damage and an ↑ tendency for airways to constrict.
- It is differentiated from COPD by the presence of childhood symptoms, diurnal variation, specific trigger factors (especially allergic), absence of smoking history and response to previous treatments.

General considerations

- Most well-controlled asthmatic patients tolerate anaesthesia and surgery well. The incidence of perioperative bronchospasm and laryngospasm in routine surgery is <2%, especially if routine medication is continued.
- The frequency of complications is ↑ in patients >50y and in those with active disease.
- Poorly controlled asthmatic patients are at risk of perioperative problems (bronchospasm, sputum retention, atelectasis, infection, respiratory failure).
- Avoid anaesthetising patients with suboptimally controlled asthma for elective surgery.[17]

Preoperative assessment

- The severity of asthma is frequently underestimated, especially if it is long-standing.
- Key indicators of severe disease include a history of frequent exacerbations, hospital visits and, most importantly, prior tracheal intubation and mechanical ventilation to manage a severe attack.
- Document any allergies/drug sensitivities, especially the effect of aspirin/NSAIDs. The prevalence of aspirin-induced asthma (measured by oral provocation) is 21% in adult and 5% in paediatric asthma patients. Much lower rates are quoted if verbal history is used to assess prevalence (3% and 2%, respectively).
- Ask about trigger factors and recent respiratory tract infections. Viral infections are potent triggers of asthma, so postpone elective surgery if symptoms suggest URTI.
- The type, dose, frequency and degree of benefit of therapy provide important clues about the severity and control of the disease.
- Examination is often unremarkable but should focus on detecting signs of acute bronchospasm, active lung infection (which should defer surgery), chronic lung disease and right heart failure.
- Advise patients to stop smoking at least 8w prior to surgery.
- Patients with mild asthma (peak flow >80% of predicted and minimal symptoms) rarely require extra treatment prior to surgery. Consider adding a short-acting β2-agonist just prior to surgery.[18]
- Moderately controlled patients should add inhaled corticosteroids to their β2-agonists 1w prior to surgery.

- Poorly controlled asthmatics (>20% variability in PEFR) may need to add oral corticosteroids to their regimen such as oral prednisolone 20–40mg daily for 1w. Consider preoperative review by a chest physician.
- Start chest physiotherapy preoperatively if major surgery.
- Emphasise the benefits of good compliance with treatment prior to surgery (Table 6.7).

Table 6.7 Perioperative recommendations for asthma medications

Class of drug	Examples	Perioperative recommendation	Notes
β2-agonists	Salbutamol, terbutaline, salmeterol	Convert to nebulised preparation	High doses may lower K^+ and cause tachycardia and tremor
Anticholinergic drugs	Ipratropium	Convert to nebulised form	
Inhaled steroids	Beclometasone, budesonide, fluticasone	Continue inhaled formulation	If >1500 micrograms/24h of beclometasone, adrenal suppression may be present
Oral steroids	Prednisolone	Continue as IV hydrocortisone until taking PO	If >10mg/24h, adrenal suppression is likely (see p. 230–1)
Leukotriene receptor antagonist (anti-inflammatory effect)	Montelukast, zafirlukast	Restart when taking oral medications	
Mast cell stabiliser	Disodium cromoglicate	Continue by inhaler	
Phosphodiesterase inhibitor	Aminophylline	Continue where possible	In severe asthma, consider converting to an infusion perioperatively

Investigations

(For preoperative respiratory investigations, see Table 6.2.)

- Serial home measurements of PEFR are more informative than a single reading. Measure the response to bronchodilators, and look for 'early morning dip' (suggesting suboptimal control).
- Spirometry helps to detect the chronic residual effects of acute asthma and to stratify the severity of the disease.
- Results of peak flow and spirometry are compared with predicted values based on age, sex and height.
- Blood gases are only necessary in assessing patients with severe asthma, particularly prior to major surgery.

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- ECG may show right atrial or RV hypertrophy, acute strain, right axis deviation and right bundle branch block, although these are often associated with acute asthma attacks and are reversible.[19]
- CXRs reveal flattened diaphragms if the lungs are hyperinflated. Useful to evaluate for pulmonary congestion, oedema or infiltrates.

Conduct of anaesthesia

- The overriding goal is to avoid bronchospasm.
- Consider premedicating the patient with nebulised salbutamol 2.5mg and an anticholinergic agent, such as glycopyrronium or atropine, to dry out secretions and suppress upper airway vagal responses.
- Consider the need for an arterial line intraoperatively in high-risk cases to facilitate ABG measurement.
- No definitive evidence shows that one method is superior to another.
- When asthma is poorly controlled, regional techniques are ideal for peripheral surgery. Spinal anaesthesia or plexus/nerve blocks are generally safe, provided the patient is able to lie flat comfortably.
- Where GA is necessary, use short-acting anaesthetic agents. Short-acting opioid analgesics (e.g. alfentanil, remifentanil) are appropriate for procedures with minimal postoperative pain or when a reliable regional block is present (Table 6.8).
- Intubation may provoke bronchospasm. Consider potent opioid cover (alfentanil). LA to the cords may help. Only instrument the airway when the patient is in a deep plane of anaesthesia. The use of an SGA may be preferable to tracheal intubation in asthmatic patients; however, the benefits of these must be weighed up against the risks of an unsecured airway.
- Ventilatory strategies, such as limiting peak inspiratory pressures and V_T and decreasing the inspiratory: expiratory (I:E) ratio, assist in avoiding air trapping and auto-PEEP.
- Caution with/avoid desflurane and histamine-releasing drugs such as morphine, atracurium and mivacurium.
- Inspired gases should be humidified to avoid airway irritation.
- Stimulating manoeuvres, such as airway suctioning, should be avoided or only performed while the patient is deeply anaesthetised.
- Caution with acetylcholinesterase inhibitors in asthmatics due to their muscarinic side effects such as bronchospasm. Consider using rocuronium as this can be reversed with sugammadex if required.
- Prophylactic use of antiemetic agents or antacids should be considered to avoid aspiration, which can trigger severe bronchospasm.

Table 6.8 Drugs considered safe for asthmatics

Induction	Propofol, etomidate, ketamine, midazolam
Opioids	Pethidine, fentanyl, alfentanil, remifentanil
NMBAs	Suxamethonium, vecuronium, rocuronium, pancuronium, cisatracurium
Volatile agents	Halothane, isoflurane, enflurane, sevoflurane
Others	IV lidocaine

Severe bronchospasm during anaesthesia

(See ➲ pp. 1078–9.)

Postoperative care

- Patients with severe asthma undergoing major abdominal or thoracic surgery should be admitted to HDU/ICU for observation.
- If acute bronchospasm persists at the end of the operation, or there are concerns about extubation, consideration should be given to a period of postoperative mechanical ventilation to avoid having to reverse the NMBA and to allow time for airway recovery. Mechanical ventilation is not without risks in this population, however, so liaise with the intensive care team early.
- Ensure all usual medications are prescribed after surgery.
- Following major abdominal or thoracic surgery, good pain control is important and epidural analgesia is frequently the best choice, provided widespread intercostal blockade is avoided.
- If prescribing patient-controlled analgesia (PCA), consider fentanyl if morphine has previously exacerbated bronchospasm.
- Prescribe O_2 for the duration of the epidural or PCA.
- Prescribe regular nebuliser therapy, with additional nebulised bronchodilators as needed.
- Review the dose and route of administration of steroid daily.
- Regular NSAIDs can be used if tolerated in the past. Avoid in brittle and poorly controlled asthmatics.
- If there is increasing dyspnoea and wheeze following surgery, consider other possible contributing factors (LV failure and PEs are potent triggers of bronchospasm). Also consider fluid overload and pneumothorax (check for recent central line).

ALGRAWANY

Chronic obstructive pulmonary disease

COPD[20,21,22,23] is a common, preventable and treatable disease. It is characterised by airflow obstruction that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lungs to noxious particles or gases.

- The more familiar terms 'chronic bronchitis' and 'emphysema' are now included within the COPD diagnosis.[21]
- Chronic bronchitis is defined as a chronic, productive cough for 3mo in each of two successive years in a patient in whom other causes of cough (e.g. bronchiectasis) have been excluded. Small airway inflammation causes obstruction and air trapping, which results in V/Q mismatching and poor respiratory muscle mechanics.
- Emphysema is a histological diagnosis of abnormal and permanent enlargement of the airspaces distal to the terminal bronchioles, without obvious fibrosis. Loss of alveolar structural integrity leads to ↓ gas transfer, as well as V/Q mismatching.
- Often it is not possible to distinguish between the two subtypes, and the relative contribution of each varies from patient to patient.
- The most important risk factor for COPD is cigarette smoking. Other factors associated with COPD include occupational exposure to dusts, atmospheric pollution, poor socioeconomic status, age, ♀ sex, repeated viral infections and α-1 antitrypsin deficiency. Genetic factors may also be implicated.
- A diagnosis of COPD should be considered in patients over the age of 35 who have a risk factor (generally smoking) and who present with exertional breathlessness, chronic cough, regular sputum production, frequent winter 'bronchitis' or wheeze. The presence of airflow obstruction should be confirmed by performing post-bronchodilator spirometry. Airflow obstruction defined by an FEV_1/forced vital capacity (FVC) ratio of <0.7 is used to diagnose COPD.[22]
- There is no single test to assess the severity of COPD. Instead, it is assessed using a range of factors, including measurement of the degree of airflow obstruction, the level of disability, the frequency of exacerbations, FEV_1, TLCO, the degree of breathlessness, exercise tolerance and BMI.

General considerations

- ↑ risk of perioperative pulmonary complications.
- Frequent exacerbations of COPD can occur, causing a rapid and sustained worsening of the patient's symptoms beyond normal day-to-day variations.
- COPD is not an absolute contraindication to any surgery.
- The further the procedure from the diaphragm, the lower the complication rate.
- COPD is associated with a number of comorbidities such as lung cancer, CVS disease and pulmonary hypertension, osteoporosis, anxiety and depression.

Preoperative assessment

- Ask about smoking, dyspnoea, cough and sputum production.
- Establish the exercise tolerance, asking specifically about hills and stairs. A simple exercise test such as stair climbing is safe and simple to perform and correlates well with more formal exercise testing.
- Enquire as to the frequency of exacerbations, timings of the most recent course of antibiotics or steroids, hospital admissions and previous requirements for invasive and non-invasive ventilation.
- Poor nutritional status (albumin <30g/L) is a strong predictor of postoperative pulmonary complications.
- ↓ breath sounds, prolonged expiration and wheeze are predictive of postoperative pulmonary complications.
- If possible, postpone surgery and commence appropriate treatment if symptoms and signs of an active respiratory infection are found.[22]

Investigations

- Preoperative spirometry is useful both to assess severity of disease and to evaluate whether the patient is at their best baseline. Identification of severe airflow obstruction is important in patients who are candidates for upper abdominal or thoracic surgical procedures.
- Check ABGs in patients with moderate to severe COPD. Useful to determine postoperative respiratory parameters.
- ECG may reveal right heart disease (RV hypertrophy or strain) or concomitant IHD. Consider echocardiography.
- CXR is not mandatory. It should be considered if there is clinical evidence of current infection or recent deterioration in symptoms.

Preoperative optimisation

(For generic information on preoptimisation, see ➲ Chapter 3.)

- Every effort should be made to assist patients in stopping smoking.
- All patients with symptomatic COPD should receive daily inhaled ipratropium or tiotropium.[23]
- Inhaled β-agonists should be used, as needed, for symptoms and wheezing in the perioperative period.
- Continue patients' usual inhaled medication perioperatively.
- Patients with COPD, persistent wheeze and functional limitations, despite bronchodilators, should be treated with perioperative glucocorticoids. Ideally, these patients should be reviewed by a respiratory physician preoperatively.
- If patients have severe COPD, postoperative respiratory failure is likely after abdominal or thoracic surgery. Plan for elective HDU/ICU admission.
- Preoperative chest physiotherapy may reduce the incidence of intraoperative bronchial plugging or pneumonitis.
- Pulmonary prehabilitation in the form of patient education, exercise training and behavioural interventions may be useful.[24]
- Consider the need for preoperative nutritional supplementation.

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Conduct of anaesthesia

- GA, and in particular tracheal intubation and IPPV, is associated with adverse outcomes. Such patients are prone to bronchospasm, laryngospasm, CVS instability, barotrauma, hypoxaemia and ↑ rates of postoperative pulmonary complications.
- Consider avoiding GA by using a regional anaesthetic technique. This may be limited by some patients' inability to lie flat.
- Consider using an arterial line for both beat-to-beat BP and repeated blood gas analysis.
- Where GA cannot be avoided, preoxygenate attentively.
- Avoid intubation where possible. Some patients, however, are unsuitable for a spontaneously breathing technique (particularly those who are obese and breathless and require long operations). Patients with heavy sputum production may benefit from endotracheal toilet.
- If using IPPV, consider using PEEP and allowing more time for exhalation by decreasing the RR or the I:E ratio (typically 1:3–1:5). These approaches may help to reduce air trapping and the development of auto-PEEP, both of which can cause an increase in intrathoracic pressure, leading to CVS instability, pulmonary barotrauma, hypercapnia and acidosis.
- Ensure the NMBA is fully reversed and the patient is warm and well oxygenated and has a $PaCO_2$ close to their normal preoperative values prior to extubation.
- Extubate in the sitting position.
- Bronchodilator treatment may be helpful peri-extubation.
- Extubation of the high-risk patient directly onto non-invasive ventilation may reduce the work of breathing and air trapping.

Postoperative care

- Those patients with severe disease or significant comorbidities should be managed in a high dependency setting capable of regular ABG measurements and the provision of non-invasive ventilation.
- Hypoventilation as a result of residual anaesthesia or opioids should be avoided, as this may lead to hypercapnia and hypoxia.
- Encourage early mobilisation.
- Use of 0.9% sodium chloride nebulisation, suctioning and physiotherapy are useful to prevent atelectasis and to encourage sputum production.
- Continue with nebulised salbutamol (2.5mg four times daily (qds)) and ipratropium (500 micrograms qds) until fully mobile. Change back to inhalers at least 24h before discharge.
- Effective analgesia is a significant determinant of postoperative pulmonary function. Epidural anaesthesia is an attractive option, as it reduces the risk of respiratory failure because of excessive sedation from opioids. It should therefore be considered if appropriate.[23]

Bronchiectasis

Bronchiectasis has similar features to COPD. The diagnosis is usually established clinically on the basis of a chronic daily cough with thick, mucopurulent sputum production, and radiographically by the presence of bronchial wall thickening, dilation of the bronchi and bronchioles on chest CT scans.

- Acquired disorder characterised by permanent abnormal dilation and destruction of the bronchial and bronchiolar walls.
- Multiple aetiologies can lead to the pathophysiological processes that cause bronchiectasis, including airway obstruction (e.g. foreign body aspiration), defective host defences, cystic fibrosis (CF), rheumatic diseases, dyskinetic cilia, smoking, pulmonary infections and allergic bronchopulmonary aspergillosis.

General considerations

- Patients with bronchiectasis need to be optimised before undergoing any major surgery which will inhibit coughing and impair respiratory function.
- Once established, bacterial infections can be difficult or impossible to eradicate. *Pseudomonas aeruginosa* is a common pathogen that may be chronic and associated with intermittent exacerbations of respiratory symptoms.
- The mainstay of treatment for bronchiectasis is regular physiotherapy, frequent courses of appropriate antibiotics and treatment of any asthmatic symptoms.

Preoperative assessment

- Consultation with the patient's chest physician is essential.
- Send a sputum sample for culture. A course of IV antibiotics and physiotherapy for 3–10d immediately prior to surgery may be necessary. Use current or most recent sputum cultures, with advice from the microbiologist/local protocols, to guide appropriate prescribing. If in doubt, assume *P. aeruginosa* infection.
- Maximise bronchodilation by converting to nebulised bronchodilators and increase the dose of prednisolone by 5–10mg/24h if on long-term oral steroids.

Investigations and conduct of anaesthesia

- As per COPD (see pp. 179–80). Also send a sputum sample for culture.

Postoperative care

- Arrange regular physiotherapy (three times daily (tds)) and nightly if severely affected.
- Continue appropriate IV antibiotics for at least 3d postoperatively or until discharged.
- Maintain adequate nutrition, especially if any malabsorption.
- Refer to the respiratory physician early if there is any deterioration in respiratory symptoms.

ALGRAWANY

Cystic fibrosis

CF[25,26,27] is a multisystem, autosomal recessive disease and is the commonest lethal genetic disease in Caucasians.

- Caused by mutations in a single gene, the CF transmembrane regulator (*CFTR*) gene on chromosome 7.
- The CFTR is a chloride channel found at the apical border of epithelial cells which line most exocrine glands in the body. All mutations causing CF affect chloride conductance through this channel, resulting in loss of chloride transport and disturbance of the Na^+/chloride balance needed to maintain a normal thin mucus layer.
- In CF, the mucus is viscid and less well cleared by the cilia.
- Clinical manifestations include progressive lung disease (frequent LRTIs, chronic hypoxaemia and cor pulmonale), nasal problems (chronic sinusitis and nasal polyps), hepatobiliary system disease due to obstruction of bile ductules (focal biliary cirrhosis, portal hypertension, multinodular biliary cirrhosis), meconium ileus, recurrent abdominal pain, pancreatic exocrine insufficiency and CF-related diabetes, infertility and osteoporosis.

General considerations

- Patients with severe disease are best managed in a major centre with multidisciplinary input.[25]
- Neonates may present for surgical treatment of meconium ileus.
- Common elective surgical procedures for CF patients include nasal polypectomy, enteral feeding or vascular access device placement.
- Almost all patients with CF have symptoms of bronchiectasis.
- The postoperative complication rate in CF is only 10% (mostly pulmonary), but half of these operations are for minor ear, nose and throat (ENT) procedures.
- Day case surgery is uncommon in CF patients; however, it is possible if disease is stable and there is good baseline function.

Preoperative assessment

- Gain a history of therapy, medications and exacerbations.
- Exclude or treat active chest infection.
- Ascertain the patient's functional ability.
- Note details of the non-respiratory components.
- Always inform the patient's physician of a surgical admission.

Investigations

- Perform FBC, U&E, coagulation study, LFTs and blood glucose.
- Respiratory tests include CXR, baseline ABG analysis and spirometry. Spirometry generally shows an obstructive pattern, with ↓ FEV_1 and FEV_1/FVC ratio.
- In advanced disease, an ECG and echocardiogram are useful to diagnose cor pulmonale.
- A 6MWT or CPET forms part of the prelung transplant workup in many centres. The results of this may be available when patients present for non-transplant surgery.

Conduct of anaesthesia

- Consider placing an arterial line to facilitate frequent ABG analysis.
- Consider using CO monitoring in patients with cor pulmonale who present for major surgery.
- For short/non-abdominal/non-thoracic procedures, an SGA with a spontaneously breathing patient may minimise the adverse effects of GA on respiratory mechanics.
- An ETT allows bronchial toilet and improved ventilatory control.
- Avoid nasal intubation, where possible, due to the high incidence of nasal polyposis.
- Keep airway pressures as low as possible when using positive pressure ventilation. Monitor for pneumothorax.
- Use humidified gases.
- Short-acting drugs should be used, wherever possible, to facilitate rapid emergence.
- Patients are often cachectic, so careful positioning and padding are important.
- Consider a regional anaesthetic technique, where appropriate, to avoid airway manipulation and to optimise postoperative analgesia.

Postoperative care

- Aim to minimise the risk of development of a postoperative respiratory tract infection.
- Aim to extubate early.
- Ensure NMB is fully reversed.
- For patients who use home non-invasive ventilation, ensure that the patient's own equipment is available immediately postoperatively.
- Chest physiotherapy should be resumed as early as possible.
- It is appropriate for patients with advanced disease to be monitored in a high dependency setting.
- For patients with FEV_1 <1L, PaO_2 <9.3kPa or $PaCO_2$ >6.6kPa, consider a period of postoperative ventilation.
- 80% of CF patients have pancreatic malabsorption. Maintaining adequate nutrition after surgery is essential, as is the advice of an experienced dietitian.

ALGRAWANY

Restrictive pulmonary disease

Restrictive lung disease is characterised by a reduction in all lung volumes with preservation of expiratory flow and ↓ compliance on pulmonary function tests (PFTs). The many disorders that cause a decrease in lung volume may be classified into intrinsic and extrinsic pulmonary disease.

Intrinsic disorders or interstitial lung disease

- A diverse group of disorders (often referred to as interstitial lung disease).
- Classified together because of similar clinical, radiographic, physiological or pathological manifestations.
- An initial inflammatory reaction in the alveoli is followed by collagen deposition and fibrosis.
- This diffuse parenchymal disease results in lungs that are smaller in volume with ↓ compliance and impaired gas exchange.
- There are >300 conditions included in the total number of interstitial lung diseases. Some have no cause, idiopathic pulmonary fibrosis being the commonest.
- Some of the causes of interstitial lung disease include:
  - Exposure to occupational agents causing pneumoconiosis (asbestos, coal, cotton)
  - Exposure to environmental agents (dust, fungus, mould)
  - Drug-induced pulmonary toxicity (amiodarone, bleomycin, methotrexate, infliximab, etanercept and paraquat poisoning)
  - Radiation to chest
  - Post-infection
  - Autoimmune disorders (sarcoid, dermatomyositis, rheumatoid arthritis (RA), SLE and scleroderma).
- Infections that can cause interstitial opacities on CXR include fungal, atypical bacterial and viral pneumonias. They often occur in immunocompromised hosts.
- Treatment is usually with oral steroids, but other immunosuppressive therapy may be used. Young patients may be considered for lung transplantation if severely affected.

Extrinsic disorders

- Chest wall components include the bony structures (ribs, spine), respiratory muscles and nerves connecting the CNS with the respiratory muscles.
- Conditions that alter chest wall structure affect the mechanics of ventilation and may result in respiratory compromise. These include ankylosing spondylitis, congenital deformities (pectus excavatum, flail chest, kyphoscoliosis), thoracoplasty, abdominal processes (morbid obesity, ascites) and chest wall tumours.
- Neuromuscular disorders affecting chest wall nerves and muscles decrease the ability of the respiratory muscles to inflate and deflate the lungs, resulting in chronically reduced lung volumes and restrictive physiology. This can be acute (Guillain–Barré) or chronic (multiple sclerosis, myasthenia gravis and amyotrophic lateral sclerosis) (see ➔ p. 309).

Preoperative assessment

- Should focus on determining the degree of respiratory impairment and establishing the extent of involvement of other organs.
- A history of exertional dyspnoea (or at rest) should be evaluated further with ABGs and PFTs.
- Discuss seriously affected patients with a respiratory physician.
- Many patients are stable and only slowly deteriorate over some years. These patients may tolerate surgery relatively well.

Investigations

- ABGs often remain normal until late. Reduced PaO_2 reflects significant disease, and CO_2 retention is a late sign, implying impending ventilatory failure.
- Obtain spirometry (lung volumes are ↓) and gas transfer if these have not been done within the previous 8w. A VC of <15mL/kg is indicative of severe dysfunction.
- Echocardiogram to look for evidence of right heart failure.
- CPET may aid perioperative risk stratification for patients undergoing high-risk intra-abdominal surgery.[28]
- CXR changes will be according to the underlying condition.

Conduct of anaesthesia

- As for other pathologies, consider regional techniques.
- Minimise positive pressure ventilation and airway instrumentation. Spinal disease may preclude subarachnoid or epidural blocks.
- If IPPV is necessary, minimise peak airway pressures using pressure-controlled ventilation with a high rate and low V_T.
- Check need for additional steroid cover for those patients on regular steroid therapy (see ➲ pp. 230–1).
- Maintain a high index of suspicion for pneumothorax.

Postoperative care

- Consider postoperative ICU/HDU admission following major surgery. May be suitable for elective training in non-invasive ventilation techniques preoperatively.
- The work of respiration is optimised by slow deep breaths and is easier in the sitting position. Extubate sitting upright.
- Give supplemental O_2, and maintain SpO_2 >92%.
- Good physiotherapy and analgesia are vital to achieve sputum clearance. With severe disease, minor respiratory complications may precipitate respiratory failure.
- Mobilise early.
- Treat respiratory infection vigorously.
- Ensure steroid cover continues in appropriate formulation.

ALGRAWANY

Sleep-related breathing disorders

Sleep-related breathing disorders are characterised by abnormal respiration during sleep. They occur in both adults and children. There are four major groups of sleep-related breathing disorders:[29]

- Central sleep apnoea syndromes
- OSA disorders (see pp. 73–5)
- Sleep-related hypoventilation disorders
- Sleep-related hypoxaemia disorder.

The hypoventilation/hypoxaemia groups are due to a variety of pulmonary parenchymal, vascular, neuromuscular or chest wall disorders, and are relatively rare.

Central sleep apnoea syndromes

Central sleep apnoea is a disorder characterised by repetitive cessation or decrease of both airflow and ventilatory effort during sleep. The condition can be 1° (idiopathic central sleep apnoea) or 2° (see Table 6.9 for detail). Risk factors are age >65 (1.1% vs 0.4%), ♂ gender, chronic opioid use and comorbidities such as heart failure.

Table 6.9 Primary and secondary central sleep apnoea

Sleep disorder	Causes	Characteristics	History	Investigation
1° central sleep apnoea	Idiopathic Prematurity/ neonates	Repetitive cessation/↓ in airflow and ventilatory effort during sleep	Daytime sleepiness Poor concentration Poor sleep quality Desaturations at night may cause nocturnal angina or paroxysmal nocturnal dyspnoea Morning headache	Laboratory-based PSG is gold standard (see pp. 73–4)
2° central sleep apnoea	Cheyne–Stokes type (CCF or CVE) Drugs High-altitude periodic breathing	Central apnoeas that occur during the decrescendo portion of the cyclic crescendo–decrescendo respiratory pattern	As above Symptoms of the coexisting illness, e.g. orthopnoea, peripheral oedema or asymmetrical weakness History of opioid use or recent time at high altitude	PSG

Sarcoidosis

Multisystem disease of unknown aetiology characterised by the formation of non-caseating granulomata, which occur in any body tissue and heal with fibrosis.

- Affects all ages, with highest prevalence at 20–40y
- More common in black individuals in the US.

General considerations

- Pulmonary changes occur in 50% of cases. Pleural, peribronchial and alveolar granulomata are replaced by fibrosis. Hilar lymphadenopathy may cause bronchial obstruction and distal atelectasis. Infiltration of the bronchial mucosa may cause stenosis. Mucosal infiltration of the nose, nasopharynx, tonsils, palate or larynx can occur.
- Cardiac effects (in 20%). RV failure 2° to lung disease. Myocardial and valvular granulomata are rare. Conduction abnormalities, VT and sudden death have been reported.
- Can also involve skin and cause uveitis/iritis and hypercalcaemia.

Preoperative assessment

- Pulmonary and cardiac features are most important.
- May have extensive pathology, but only minor symptoms.
- Note steroid treatment or other immunosuppressive drugs.

Investigations

- Preoperative PFTs may reveal a restrictive defect. TLCO (DLCO) may be ↓. ABGs will determine the level of hypoxaemia.
- ECG may show RV hypertrophy or arrhythmias.
- Check serum Ca^{2+} for hypercalcaemia (treat with systemic steroids).

Conduct of anaesthesia

- If respiratory function is impaired, consider avoidance of GA and the use of LA/regional anaesthesia.
- Consider regional analgesia for abdominal surgery if significant respiratory disease.
- Give appropriate steroid cover if needed.

Postoperative care

- Nurse the patient sitting upright.
- Good postoperative analgesia.
- Chest physiotherapy/breathing exercises.

ALGRAWANY

Anaesthesia after lung transplantation

(See also ➲ Patients with a transplanted heart, pp. 132–3.)

Indications for surgery after lung transplantation may be:

- Unrelated to the transplant
- Related to the disease precipitating the transplant (emphysema, α-1 antitrypsin deficiency, pulmonary fibrosis, 1° pulmonary hypertension, CF)
- Due to complications with the transplant
- Due to complications from lifelong immunosuppression.

General considerations[30]

- Loss of afferent and efferent innervation distal to bronchial anastomosis results in loss of the cough reflex to stimuli distal to the anastomosis, and neurally mediated changes in the bronchomotor tone.
- Airway reactivity does not appear to be ↑.
- Mucociliary clearance is impaired in the pulmonary allograft, which, together with immunosuppression and impaired cough, places the patient at ↑ risk for perioperative pneumonia.
- Hypoxic vasoconstriction is not impaired, so during an episode of acute rejection, pulmonary blood flow may be directed away from the transplanted lung.
- Lymphatic drainage is severed but then re-established 2–4w post-transplantation. Transplanted lungs are at particular risk of pulmonary oedema, especially in the early postoperative period.
- Allograft function may be compromised at any time by episodes of acute rejection, which are often difficult to distinguish clinically from pulmonary infection.
- In double-lung transplant, the heart may be denervated, has a higher resting HR (90–100bpm) and may be more susceptible to arrhythmias.

Preoperative assessment

- Assess the status of the transplanted lung(s). Have there been any episodes of rejection, immunosuppression or infection?
- All but the most urgent procedures should be delayed if there is a reversible complication present.
- In patients with single-lung transplants, careful attention should be paid to establish the extent of disease and the degree of compromise in the native lung because these factors may have implications for the provision of mechanical ventilation, e.g. differential lung ventilation requiring a double-lumen tube may be required.
- Symptoms such as dry cough and dyspnoea 8–12mo postoperatively may indicate obliterative bronchiolitis, the predominant feature of chronic transplant rejection. This is characterised by progressive narrowing of the small airways.
- Evaluate the extent of any residual systemic disease and the effects of immunosuppressive drugs on other organ function.
- Assess for airway narrowing or compromise. Previous tracheostomy may have caused a degree of subglottic stenosis.
- Coordinate with transplant or respiratory services. Make a plan for whether they will need to review the patient perioperatively.

Conduct of anaesthesia

- The interaction of immunosuppressive drugs (ciclosporin, steroids, azathioprine) with anaesthetic drugs is more theoretical than clinical.
- Stress doses of steroids will be required in most cases.
- Attention to aseptic techniques is important, as immunosuppression occurs with most chemotherapeutic drugs.
- There is no evidence to suggest that placing CVCs on the side opposite the transplanted lung is safer.
- Monitor neuromuscular function and avoid high doses of opioid in order to achieve early extubation.
- To minimise the risk of damage to the tracheal/bronchial anastomosis, intubation should leave the tube just through the cords and the cuff carefully inflated and checked intraoperatively. Place double-lumen tubes under direct vision using a flexible optical bronchoscope (FOB).
- The basic goal in ventilation is ensuring adequate oxygenation and ventilation while minimising peak airway pressures and O_2.
- A mask or SGA is not contraindicated, although there is a risk of silent aspiration in patients with no carinal cough reflex.
- Strict attention to fluid balance is required.
- Aim for early return of pulmonary function and extubation.

Postoperative care

- Postoperative admission to ICU is only indicated when anaesthesia is complicated by inadequate recovery of respiratory function, the surgical condition or the presence of rejection or infection.
- Chest physiotherapy, postural drainage and incentive spirometry in the postoperative period may be beneficial.

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Chapter 7

Renal disease

Quentin Milner

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Glomerular filtration rate and creatinine clearance

Renal dysfunction is an independent risk factor for postoperative morbidity and mortality and has important implications for the clearance of many commonly used drugs. Accurate calculation and recognition of renal dysfunction are therefore required. Estimation of the GFR is largely accepted as the best overall index of kidney function.

GFR is the total filtration rate of all functioning nephrons. It varies with age, sex and body size. Inulin clearance is the gold standard measure of GFR, although this is not used regularly in clinical practice. Instead, GFR is estimated using formulae derived from serum creatinine, a product of skeletal metabolism. GFR has a rectangular hyperbolic relationship with creatinine, meaning it must drop by 50% before serum creatinine begins to rise. For this reason, serum creatinine is not a good marker of GFR on its own.

Creatinine is secreted by the proximal tubule, as well as filtered by the glomerulus, and so creatinine clearance (CC) exceeds GFR slightly. CC can be either measured directly or estimated using formulae. Commonly used equations are the Modification of Diet in Renal Disease (MDRD) and Cockcroft–Gault equation. Both use serum creatinine in combination with age, sex, weight or race to estimate GFR. The Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation is a new equation based on serum creatinine and race.

Kidney function is proportional to kidney size, which is proportional to body surface area. Recent standards suggest estimated glomerular filtration rate (eGFR) should be indexed to the normal mean body surface area for young adults (1.73m^2). The MDRD and CKD-EPI equations both estimate GFR adjusted for body surface area and can therefore be applied to determine kidney function, regardless of a patient's size.

Certain patient groups have dramatically different serum creatinine production or elimination, compared to the normal patient population. Patients with ↑ CC include patients with cystic fibrosis or burns and pregnant women. Patients with lower limb amputations or muscle wasting disorders can have a lower muscle mass, thus reducing serum creatinine. Patients with cirrhosis have less hepatic conversion of creatine to creatinine, so have falsely low serum creatinine levels. Other sources of error include extremes of muscle mass and body size, diet, drugs affecting tubular secretion of creatinine (cimetidine, trimethoprim, fenofibrate), dialysis, antibiotics and interference with creatinine assay (bilirubin, glucose and ketones).

Chronic kidney disease

CKD is defined as kidney damage or abnormalities of kidney structure resulting in GFR <60mL/min/1.73m² for 3mo or more. It is a multisystem disease (Box 7.1). Patients often have complex medical histories, take a multitude of drugs and may have severe systemic complications.

Box 7.1 Main causes of CKD

DM	40%
Hypertension	24%
Glomerulonephritis	7%
Chronic pyelonephritis	5%
Polycystic renal disease	4%
Unknown cause	20%

Classification of CKD

CKD is classified based on the GFR and the level of proteinuria. Patients are classified as G1–G5 based on the eGFR, and A1–A3 based on the albumin:creatinine ratio. The higher the stage (G1–G5) (Table 7.1) and the greater the amount of protein present in the urine (A1–A3), the more 'severe' the CKD.[1]

Table 7.1 Stages of renal dysfunction by GFR

GFR category	GFR (mL/min/1.73m²)	Terms
G1	≥90	Normal or high
G2	60–89	Mild decrease relative to young adult
G3a	45–59	Mild–moderate decrease
G3b	30–44	Moderate–severe decrease
G4	15–29	Severe decrease
G5	<15	Kidney failure

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Preoperative

- Determine the underlying cause, previous surgery, including transplantation, and drug therapy.
- These patients are high risk, particularly diabetics with CKD G3–G5.
- Check for hypertension, DM and anaemia. IHD is common and often silent, especially in diabetics. Incidence of calcific valvular heart disease and LV failure is ↑. Autonomic neuropathy is common. Pericardial effusions are rare if dialysis is effective.
- Check the type of dialysis: peritoneal or haemodialysis? Do they have a central line or fistula?
- Determine the residual urine output per day.
- Examine for fluid overload (dependent oedema, basal crepitations, dialysis record, dry and wet weights) or hypovolaemia (postural hypotension, low JVP, thirst, skin turgor, oliguria).

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- Allow 4–6h to elapse after haemodialysis before surgery. This allows fluid compartment equilibration and metabolism of residual heparin. Indications for urgent dialysis include hyperkalaemia, fluid overload, acute acidosis and symptomatic uraemia. If volume overload occurs postoperatively, the patient will need extra dialysis.
- If major surgery, plan postoperative care with renal and ICU teams.

Investigations

- FBC: usually well-compensated and tolerated normochromic normocytic anaemia due to ↓ erythropoiesis, red cell survival and GI losses. Aim for Hb 80–100g/L; transfusion can worsen hypertension and precipitate heart failure.
- Electrolytes: a recent serum K^+ is essential and if >6.0mmol/L, dialysis will be required. Drugs causing raised K^+ include suxamethonium, NSAIDs, β-blockers, ACE inhibitors, spironolactone, tacrolimus and ciclosporin. Na^+ may be low due to water retention. Hypocalcaemia and hyperphosphataemia are common, but rarely symptomatic. A mild metabolic acidosis is frequent, and the ability to compensate for further acidosis is poor.
- Coagulation: INR, activated partial thromboplastin time (APTT) and platelet count are usually normal. Uraemia affects platelet function and causes a prolonged bleeding time. Dialysis improves coagulation once heparin has worn off. Thrombocytopathy is not corrected by platelet transfusion but may be improved by cryoprecipitate or desmopressin (0.3 micrograms/kg in 30mL of 0.9% sodium chloride over 30min). Tendency to thrombosis in fistula in stage G5 CKD on haemodialysis.

Perioperative care

- Venous access and fistulae: many patients have an upper limb arteriovenous (A–V) fistula. Avoid cannulation and NIBP in this arm. Protect the fistula arm with padding. Try to cannulate the dorsum of the hand to avoid damage to the veins in the forearm and antecubital fossa which may be needed for future fistulae. Use dialysis catheters for IV access only as a last resort, and remember that the dead space may contain high-dose heparin (at least 1000IU/mL). Aspirate and discard. Arterial cannulation may compromise future A–V fistula formation and should only be used where absolutely necessary.
- Fluid and electrolyte balance must be carefully managed. Many patients have some residual renal function and urine output. Maintaining this is essential. Normovolaemia is ideal. Replace fluid losses promptly. Avoid hypotension.
- If large fluid shifts are likely, CVP or oesophageal Doppler monitoring is useful. These patients may have had multiple CVP lines, so use ultrasound. Avoid the femoral vein in patients suitable for transplants and the subclavian vein in those needing dialysis, as the incidence of stenosis is high.
- The type and volume of fluid administered affect renal outcomes. Dynamic measures of fluid responsiveness (straight leg raise, pulse pressure variation) are better at gauging fluid deficit in these patients. There is little evidence on which fluid is best in the perioperative setting; however, current literature supports the use of balanced

crystalloid solutions (rather than high chloride solutions) in critical care environments. Albumin solutions can be indicated in renal patients with high fluid demands to lessen interstitial volume overload.[2]

- Suxamethonium elevates serum K^+ by 0.5mmol/L. Hyperkalaemia is worsened by acidosis, so avoid hypoventilation and hypercarbia.
- Delayed gastric emptying due to autonomic neuropathy and ↑ gastric acidity make gastric reflux more likely. Most patients are on H_2 antagonists/PPIs (cimetidine may cause confusion and should be avoided). In practice, RSI is reserved for patients who are not fasted or who have symptomatic reflux and a normal serum K^+.
- Immunity: sepsis is a leading cause of death in CKD. Inhibition of humoral and cell-mediated immunity occurs. Careful attention to asepsis is required for all invasive procedures.
- Hepatitis B and C are common.

Postoperative

- Any patient with CKD is at ↑ risk of developing AKI. Prevention is most important (see pp. 198–200), but postoperatively, renal function must be checked.
- Liaise carefully with the renal unit about the timing/need for dialysis postoperatively. Use epidurals with caution.
- Prescribe analgesics carefully (see p. 197).
- Pay attention to fluid balance. In oliguria, hourly fluid maintenance should replace fluid losses plus 30mL/h for insensible losses. Avoid nephrotoxic drugs and periods of hypotension.

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Anaesthetic drugs in CKD

Most drugs are excreted by the kidneys, either unchanged or as metabolites. Loading doses of drugs are often unchanged, but maintenance doses should be reduced and/or the dosing interval prolonged. Hypoalbuminaemia and acidosis increase the free drug availability of highly protein-bound drugs (e.g. induction agents). Most anaesthetics reduce renal blood flow, GFR and urine output (Table 7.2).

- Analgesics: most opioids are excreted by the kidney and so have a prolonged duration of action in CKD. The long-acting morphine metabolite morphine-6-glucuronide has far greater potency than morphine itself. Avoid pethidine (meperidine), as its metabolite norpethidine can cause convulsions. Fentanyl has inactive metabolites but accumulates with prolonged use (10% renally excreted). Alfentanil and remifentanil may be used in normal doses. Half-lives of codeine and dihydrocodeine are prolonged five times, so avoid. Oxycodone has active metabolites, so reduce the dose and increase the dose interval. Tramadol and its active metabolites are renally excreted. The manufacturer does not recommend its use in end-stage renal failure.
- PCA morphine or fentanyl (10 micrograms bolus, 5min lockout time) can be used, but with caution. In theory, the reduction in excretion increases the plasma concentration, thus reducing demand.
- Paracetamol is safe in normal doses. Avoid NSAIDs, even if anuric.
- Induction agents: reduce the doses of benzodiazepines, thiopental and etomidate by ~30% because of changes in protein binding, volume of distribution and cardiac function. However, the dose of propofol required for a bispectral index (BIS) of 50 is ↑. Wake-up following propofol infusion is faster.
- The elimination of volatile anaesthetic agents is not dependent on the renal function. Isoflurane, halothane and desflurane are all safe. Sevoflurane is safe for induction but will produce inorganic fluoride ions with prolonged use (avoid >4 MAC hours total).
- Muscle relaxants: suxamethonium is discussed on ➲ pp. 194–5; plasma cholinesterase activity is unchanged in CKD. Atracurium and cisatracurium are logical choices. Vecuronium and rocuronium can be used as single doses, with prolonged duration of action. Mivacurium clearance is ↓. Always use a peripheral nerve stimulator. Sugammadex is excreted in the urine unchanged, but its action does not depend on renal excretion. It appears to be safe to use in severe CKD but is not recommended for GFR <30mL/min, as clearance may take much longer and repeated muscle relaxation with atracurium may be needed.[3] It is unpredictably removed by dialysis.
- The excretion of neostigmine and glycopyrronium is prolonged.
- The duration of action of LAs is reduced. Reduce maximum doses by 25% because of ↓ protein binding and a lower CNS seizure threshold. Epidurals and spinals work well, but consider the ↑ risks of hypovolaemia after dialysis, haemorrhage and spinal haematoma formation.

- Anaesthesia for A–V fistula formation: ask the surgeon where the fistula is to be formed. Local infiltration works well for brachiobasilic fistulae. An axillary/supraclavicular brachial plexus block is recommended for a brachiocephalic fistula, and evidence shows a better fistula outcome. Patients may need extra LA in the axilla and mild sedation. Avoid hypotension to prevent fistula thrombosis. The fistula may be used for dialysis after 3–4w. Synthetic grafts can be used immediately but do not last as long.
- Peritoneal dialysis uses the large surface area of the peritoneum to exchange fluid and metabolites via temporary (hard) or permanent Tenckhoff (soft) catheters in the lower abdomen. It is inefficient but can run continuously. Catheter placement or removal usually requires a mini-laparotomy but can be done under LA ± sedation if necessary. Dialysis fluid should be drained before anaesthesia to prevent respiratory function compromise. Patients can usually omit 24–48h of postoperative dialysis, but a period of haemodialysis may be needed if undergoing bowel surgery.
- Most antibiotics are excreted by the kidney. It is common to use a normal loading dose, with reduced and/or delayed maintenance doses. If in doubt, check in the *British National Formulary* (*BNF*) or with a microbiologist.

Table 7.2 Anaesthetic drugs and CKD

	Safe drugs	Drugs safe in limited or reduced doses	Contraindicated drugs
Premedication	Lormetazepam, midazolam, temazepam		
Induction	Propofol	Ketamine, etomidate, thiopental	
Maintenance	Isoflurane, desflurane, halothane, propofol	Sevoflurane	Enflurane
Muscle relaxants	Suxamethonium, atracurium, cisatracurium	Vecuronium, rocuronium	Pancuronium
Opioids	Alfentanil, remifentanil	Fentanyl, morphine, oxycodone	Pethidine, codeine, tramadol
Local anaesthetics	Bupivacaine, lidocaine (reduce dose by 25%)		
Analgesics	Paracetamol		NSAIDs

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Acute kidney injury

AKI is defined as any of the following:
- Increase in serum creatinine by ≥0.3mg/dL within 48h; or
- Increase in serum creatinine by ≥1.5 times the baseline, which is known or presumed to have occurred within the prior 7d; or
- Urine volume <0.5mL/kg/h for 6h.

AKI is categorized into three stages:
Stage 1 is defined as any one of the following:
- An increase in serum creatinine by ≥26micromol/L within 48h
- A ≥50% rise in serum creatinine within 7d (25% in children)
- Urine volume <0.5mL/kg/h for 6h (>8h in children)

Stage 2: serum creatinine doubles and/or urine <0.5mL/kg/h ≥12h
Stage 3 is defined as any one of the following:
- Serum creatinine triples or >354 micromoles/L
- Anuria ≥12h or <0.3mL/kg/h urine for ≥24h
- Renal replacement therapy required
- eGFR <35mL/min in patients <18y.

Perioperative management of AKI

AKI developing in the perioperative period has a high mortality. Table 7.3 highlights the risk factors for developing AKI.

Table 7.3 Risk factors for perioperative AKI

Pre-existing	CKD, DM, liver disease, cardiovascular disease, previous AKI, neurological or cognitive impairment affecting diet, over 65y
Perioperative	Sepsis, hypotension, hypovolaemia, dehydration
Drugs	Nephrotoxins: antibiotics, NSAIDs, ACE inhibitors, lithium, chemotherapy agents, radiological contrast media
Trauma	Rhabdomyolysis (myoglobinaemia from crush injuries)
Surgery	Emergency, intraperitoneal, biliary surgery in the presence of obstructive jaundice (hepatorenal syndrome)
	Renal and abdominal vascular surgery
Intra-abdominal hypertension	Any cause of abdominal distension
Urinary obstruction	

Considerations

- Preoperative rehydration is essential, and any fluid deficit should be corrected before surgery. Invasive monitoring may be needed.
- Remember that an adequate BP is needed for renal perfusion and this is relative to the patient's baseline. A MAP under 80mmHg for ≥10min has been linked to ↑ risk of AKI,[4] but this may be inadequate in hypertensives. Inotropes may be required.

- Aim to prevent further deterioration of renal function and maintain an adequate urine output (>0.5mL/kg/h). Remember, urinary catheters can block.
- Intra-abdominal hypertension (pressure >20mmHg) can occur following major abdominal surgery and causes anuria by direct compression of the renal pelvis and reduced renal perfusion.
- Seek advice from the renal unit/ICU about postoperative care and dialysis if hyperkalaemic, acidotic, symptomatic uraemia or fluid overloaded or in pulmonary oedema.

Tests

- eGFR and serum creatinine are the main initial measurement for diagnosis. Serum urea is much less specific since it is ↑ in dehydration, GI bleeding, sepsis and excessive diuretic use.
- Check electrolytes, especially serum K^+.
- Check for and treat hyperglycaemia.
- Urinary electrolytes may help differentiate hypoperfusion (Na^+ <20mmol/L, urine osmolality >500mOsmol/kg) from acute tubular necrosis (Na^+ >20mmol/L, urine osmolality <500mOsmol/kg). These results are meaningless if diuretics have been given.

Medications

- Stop nephrotoxic drugs: NSAID, ACE inhibitor/ARB and K^+-sparing diuretic.
- Metformin may accumulate, causing lactic acidosis.
- Aminoglycosides should not be used unless no suitable, less nephrotoxic alternative is available.
- Contrast-induced nephropathy should be predicted and avoided if possible. Volume loading with crystalloid or sodium bicarbonate may be helpful if contrast is used. IV sodium bicarbonate 1.26% 3mL/kg/h for 1h preoperatively, and then 1mL/kg/h until 6h postoperatively. Acetylcysteine PO may also be useful.
- The outcome from polyuric AKI is better than oliguric AKI. Diuretics should be used to manage fluid overload but usually are not indicated in AKI otherwise. Furosemide is given initially as an IV bolus of 20–40mg. In patients with established renal failure, furosemide 250mg may be infused over 1h.
- There is no evidence to support the use of low-dose dopamine.
- Mannitol (0.5g/kg IV) may improve urine flow.

Emergency management of hyperkalaemia

(See ➲ pp. 240–1.)

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Further reading

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Chapter 8

Hepatic disease

Ashleigh Williams and John Christie

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Liver disease

Liver disease can be classified as chronic liver disease (CLD) and acute liver failure (ALF). ALF can be subclassified into hyperacute, subacute, and acute. Common causes of ALF and CLD are listed in Table 8.1.

Chronic liver disease

- CLD is defined as impairment of liver synthetic and metabolic function present for >26w.
- Chronic inflammation leads to fibrosis and cirrhosis (nodular regeneration and disruption of liver architecture), leading to portal hypertension.
- Cirrhosis can be compensated (function preserved) or decompensated (presence of ascites, encephalopathy or oesophageal varices).
- Once there is evidence of decompensation, survival is around 5y, so assessment for transplantation should be considered.
- Portal hypertension in CLD leads to engorgement of the anastomoses between portal and systemic circulations, leading to varices at the gastro-oesophageal junction, haemorrhoids and dilated abdominal wall veins (caput medusae). Patients with varices are at risk of acute bleeding, which is associated with a high mortality rate.
- Impaired hepatic synthetic function leads to low albumin and plasma proteins. This contributes to oedema/ascites and has implications for drug protein-binding.
- Ascites: liver fibrosis leads to portal hypertension and, in combination with salt/water retention 2° to hyperaldosteronism, splanchnic vasodilation and low serum albumin, fluid accumulates in the peritoneal cavity. More commonly seen in CLD. Spironolactone long term may exacerbate electrolyte disturbances and renal dysfunction.

Acute liver failure

- ALF is a process involving rapidly progressing liver necrosis and an inflammatory response with physiological derangement similar to sepsis.
- Patients develop the triad of hepatocellular dysfunction (jaundice), coagulopathy and encephalopathy, without prior known liver disease.
- ALF can be classified by time interval from onset of jaundice to encephalopathy (Table 8.2).
- Hyperacute ALF is typical of paracetamol overdose, although jaundice is often absent (coagulopathy and deranged LFTs used as surrogate for onset time).
- The precipitating cause ultimately results in liver necrosis and an inflammatory response with physiological derangement similar to sepsis. Absence of hepatic encephalopathy, defined as acute liver injury, has a better prognosis.
- For specific ALF management and referral criteria, see ➔ p. 210.

Table 8.1 Causes of liver failure

Cause	Acute	Chronic
Paracetamol	Accounts for majority of cases (>70%) in the UK	
Alcohol	Acute alcoholic hepatitis	Commonest cause in the UK. Chronic alcoholism may be complicated by alcohol withdrawal peri- or postoperatively (see p. 338) or by acute alcoholic hepatitis
Other drugs and toxins	Carbon tetrachloride, *Amanita phalloides* mushrooms, idiosyncratic drug reactions	Isoniazid, methyldopa, methotrexate, amiodarone, carbon tetrachloride, *Amanita phalloides* mushrooms
Viral hepatitis	Commonest cause worldwide. Types A–E, cytomegalovirus, herpes simplex, Epstein–Barr virus, seronegative hepatitis	Hepatitis B: 3% of those infected as an adult develop chronic hepatitis B infection
		Hepatitis C: 75% develop chronic infection. Main risk factor for infection is IV drug abuse

Table 8.2 Subtypes of acute liver failure

Subtype	Onset	Transplant-free survival (%)
Hyperacute	Within 7d	30
Acute	1w to 1mo	33
Subacute	1–3mo	14

Liver disease: a multisystem problem

Renal

Impairment is most commonly due to dehydration, sepsis or nephrotoxic drugs. Renal failure in the context of liver failure confers a high mortality; the precipitating cause should be investigated and treated. Up to 50% of patients presenting with ALF will also have AKI. Ensure adequate hydration and renal blood flow.

Hepatorenal syndrome

A diagnosis of exclusion in patients with cirrhotic liver disease as a consequence of altered renovascular tone. Type 1: rapidly progressive; type 2: slower in onset and associated with diuretic-resistant ascites. Both are associated with poor prognosis and liver transplantation may be the only definitive treatment. Diagnostic criteria: urinary Na^+ <10mmol/L, urine:plasma osmolality and creatinine ratios >1, normal CVP and no diuresis on central volume expansion underlying CLD and ascites.

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Hepatopulmonary syndrome

Occurs when intrapulmonary vascular dilations contribute to hypoxia in liver disease, possibly 2° to ↑ production or ↓ clearance of vasodilators such as nitrous oxide (N_2O). Intrapulmonary shunting further contributes to V/Q mismatch, ↑ A–a gradient and low PaO_2. The only definitive treatment is liver transplantation.

Pulmonary hypertension

A serious complication present in 0.25–4% of all patients with cirrhosis. It is thought to occur due to local pulmonary production of vasoconstrictors that occurs while systemically vasodilation predominates.

Encephalopathy

Toxic metabolites build up (particularly ammonia, due to deranged amino acid metabolism), leading to progressive encephalopathy (Box 8.1). Give regular lactulose to prevent constipation. Encephalopathy in CLD heralds decompensation and a precipitating cause should be sought. Reduced Glasgow coma scale (GCS) and loss of airway reflexes may cause respiratory failure. Patients with grade III/IV encephalopathy need intubation to protect their airway and ventilation permits manipulation of ICP.

Box 8.1 West Haven classification of encephalopathy

Grade 0	Alert and orientated
Grade I	Drowsy and orientated
Grade II	Drowsy and disorientated
Grade III	Rousable stupor, restlessness
Grade IV	Coma: unresponsive to deep pain

Source: data from Conn HO *et al*. Comparison of lactulose and neomycin in the treatment of chronic portal-systemic encephalopathy. A double blind controlled trial, Gastroenterology, 1989;**72**:573–83.

Anaesthetic management of patients with liver disease

CLD presents to the anaesthetist and surgeon more frequently than acute liver disease. While the underlying cause and disease course differ between ALF and CLD, the physiological consequences are similar and will be discussed together. Specific considerations in ALF are discussed at the end.

Patients with liver disease have a high perioperative risk which is proportional to the degree of hepatic dysfunction. All patients should have a thorough preoperative assessment, including signs, symptoms and risk factors for liver disease.

- **Symptoms**: anorexia, malaise, weight loss, easy bruising, itching, right upper quadrant (RUQ) pain
- **Signs**: jaundice, palmar erythema, spider naevi, caput medusae, gynaecomastia, ascites, hepatosplenomegaly, testicular atrophy
- **Risk factors**: alcohol excess, IV drug abuse, obesity, autoimmune conditions, haemodialysis, haemophilia, unprotected sexual intercourse and previous blood transfusion.

Risk assessment

- Assessment of risk for surgery and anaesthesia in CLD may be estimated by the Child's classification (Table 8.3), originally designed to guide mortality in portal decompressive surgery, or by the model for end-stage liver disease (MELD) score, which was designed to predict 3mo survival in transjugular intrahepatic portosystemic shunt (TIPSS) patients.
- MELD scoring requires serum bilirubin, creatinine and INR. Online calculator: www.alchepscores.com
- Acute active hepatitis or liver failure is a contraindication to elective surgery. Elective surgery is contraindicated in Child–Pugh class C and MELD score >15 as mortality is ~40%.
- Child–Pugh class B or MELD score 10–15 should be approached with caution, and Child–Pugh class A or MELD score <10 can proceed with elective surgery.
- Generic illness severity scoring tools, such as SOFA (Sequential Organ Failure Assessment) or APACHE II (Acute Physiology and Chronic Health Evaluation II), may be more accurate in acutely unwell patients with CLD.
- Surgical risk in CLD depends on the extent of hepatic impairment, in addition to the urgency and type of surgery. Common causes of mortality in the perioperative period include sepsis, renal failure, bleeding and worsening liver failure with encephalopathy.

Preoperative laboratory investigations

- FBC and clotting studies: anaemia is contributed to by chronic blood loss, hypersplenism, haemolysis, chronic illness and malnutrition.
- Electrolytes and creatinine: urea is often falsely low due to ↓ hepatic production. Hyponatraemia and hypokalaemia are common. Diuretic use may exacerbate electrolyte disturbances.
- Glucose: impaired glycogen storage and glucose utilisation.
- LFTs.
- Hepatitis screening: hepatitis B and C are highly contagious to clinical staff via parenteral inoculation and universal precautions must be strictly followed.

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Table 8.3 Surgical risk assessment: Child's classification, as modified by Pugh

Mortality	Minimal (<5%)	Modest (5–50%)	Marked (>50%)
Bilirubin (micromoles/L)	<25	25–40	>40
Albumin (g/L)	>35	30–35	<30
PT (s, prolonged)	1–4 (INR <1.7)	4–6 (INR 1.7–2.3)	>6 (INR >2.3)
Ascites	None	Moderate	Marked
Encephalopathy (see p. 204)	None	Grades I and II	Grades III and IV
Nutrition	Excellent	Good	Poor

INR, international normalised ratio; PT, prothrombin time.

Assessment of liver function

- Serum LFTs (Table 8.4) are rarely specific, but prothrombin time (PT), albumin and bilirubin are sensitive markers of overall liver function. Serial measurements are useful and indicate trends. Avoid giving fresh frozen plasma (FFP), unless treating active bleeding, as PT is an excellent guide to overall liver function.
- Liver transaminases (aspartate transaminase (AST), alanine aminotransferase (ALT)) are sensitive to even mild liver damage and have no role in mortality prediction. Levels may decrease in severe disease.
- Alkaline phosphatase (ALP) is raised with biliary obstruction.
- Imaging techniques: ultrasound is the main initial investigation of obstructive jaundice. Other useful investigations include endoscopic retrograde cholangiopancreatography (ERCP), CT and magnetic resonance cholangiography.
- LFTs must always be interpreted alongside a careful history and examination. The liver has a large functional reserve and can often withstand considerable damage before LFTs become deranged.

Preoperative investigations

Cardiac

- ECG is essential as electrolyte abnormalities may precipitate arrhythmias and alcohol excess may result in AF and cardiomyopathy. Prolonged corrected QT is relatively common, and hyperbilirubinaemia may precipitate bradyarrhythmias.
- Echocardiography: cardiomyopathy may develop in association with causes of CLD, particularly alcohol excess. Pericardial effusions and diastolic dysfunction also develop in cirrhosis. Smoking is associated with alcohol excess and is an independent risk factor for CAD.

Respiratory

- CXR
- ABG.

Table 8.4 Liver function tests

Test	Normal range	Raised
Bilirubin	2–17 micromoles/L	Haemolysis Gilbert's syndrome Acute and chronic liver failure Biliary obstruction
AST	0–35IU/L	Non-specific (found in liver, heart, muscle, etc.) Hepatocellular injury
ALT	0–45IU/L	Specific Hepatocellular injury Degree of elevation can point to aetiology: >1000: acute viral hepatitis, drugs, autoimmune hepatitis and ischaemia 100–200: acute viral hepatitis, alcohol and non-alcoholic fatty liver disease
ALP	30–120IU/L	Physiological (pregnancy, adolescents, familial) Bile duct obstruction (stones, drugs, cancer) 1° biliary cirrhosis Metastatic liver disease Bone disease
γ-glutamyl transpeptidase	0–30IU/L	Non-specific (found in heart, pancreas and kidneys) Alcoholic liver disease
Albumin	40–60g/L	Non-specific (affected by nutritional status, catabolism, and urinary and GI losses) Prognostic in chronic liver disease
PT and INR	10.9–12.5s (INR 1.0–1.2)	Non-specific (vitamin K deficiency, warfarin therapy, disseminated intravascular coagulation) However, best prognostic marker in ALF

Nutrition

CLD is associated with a poor nutritional state and predisposes to ↑ incidence of postoperative complications. Preoperative nutrition may help to reduce this in major elective surgical procedures.

Other

The presence of varices in patients with known liver disease must be established, as this is a contraindication for the use of oesophageal Doppler probes/TOE and oesophageal temperature probes.

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Perioperative considerations

Premedication

PPIs or H_2 antagonists should be used preoperatively. RSI will further reduce the risks of gastric aspiration. Sedative medication may precipitate or worsen encephalopathy.

Monitoring

Standard monitoring should be used, with consideration given to invasive arterial and CVP monitoring. CVP monitoring is controversial but allows for centrally administered vasopressors and venous access. Perioperative haemodynamic instability can worsen hepatic function; MAP should be maintained within 10–20% of preoperative levels. Hepatic blood flow and O_2 delivery should be maintained; CO monitoring may be useful, although transthoracic echocardiography or oesophageal Doppler may be contraindicated in varices.

Drug effects

Even in severe liver disease, the problem is usually one of exaggerated effects on the CNS, rather than poor liver metabolism. Hepatic blood flow is altered by anaesthetic drugs (e.g. α- and β-agonists/antagonists), positive pressure ventilation, PEEP and surgical technique. In most cases, anaesthesia reduces hepatic blood flow. (See ➲ Drug metabolism and liver disease, p. 211.) Desflurane best preserves hepatic blood flow, is least metabolised and has a quicker emergence time, but sevoflurane and isoflurane are also acceptable volatiles to use.

Regional techniques

Can be useful adjuncts, but neuraxial techniques are contraindicated in the presence of coagulopathy. Most LAs are metabolised by the liver.

Cardiovascular

Low SVR and arterial pressure, ↑ HR and volume expansion 2° to an activated renin–angiotensin system seen in both ALF and CLD. Vasopressors may be required; maintain MAP >75mmHg. Portosystemic, pulmonary and cutaneous shunting (spider angiomata) contributes to a hyperdynamic, high CO state, often ↑ by up to 50%. Alcohol excess is associated with cardiomyopathy; cirrhosis is associated with a high incidence of cardiac dysfunction ('cirrhotic cardiomyopathy'), and concurrent smoking is a risk factor for CAD. Preoperative assessment, including echo, where indicated. Propranolol to reduce portal pressures.

Respiratory

Hypoxia is common and multifactorial in CLD. Ascites causes splinting of the diaphragm, basal atelectasis and collapse. Excess PEEP will increase hepatic venous pressure and ICP. Tense ascites may affect respiratory mechanics; consider percutaneous drainage.

Pulmonary hypertension

Consider echo if suspicion of pulmonary hypertension (see ➲ pp. 139–44).

Encephalopathy

This may be precipitated by sedatives, GI bleeding, infection, surgical operations, trauma, hypokalaemia, constipation or acute severe liver failure.

Coagulopathy

Very common in liver disease; the liver synthesises all clotting factors, except factor VIII. Coagulopathy is attributed to several mechanisms (↓ synthesis of clotting factors and clearance of activated clotting factors, quantitative and qualitative platelet abnormalities and hyperfibrinolysis). Consider vitamin K. Reversal of coagulopathy with FFP, cryoprecipitate and platelets directed by thromboelastography (TEG®)or similar point-of-care testing (POCT). Ensure adequate provision is made for cross (X)-matched blood and clotting products.

Renal

CO monitoring/goal-directed fluid therapy (GDFT) may be useful. Tense ascites may impair renal blood flow and give a falsely high CVP. Avoid hypotension and nephrotoxic drugs, and aim for urine output >1mL/kg/h.

Portal hypertension

Use of oesophageal Doppler/TOE/oesophageal temperature probes is contraindicated. Treatment with β-blockers may contribute to perioperative hypotension. A low CVP may reduce the risk of variceal or GI bleeding.

Ascites

Consider draining preoperatively as hypotension, hypoventilation and aspiration are all ↑ with tense ascites.

Hypoglycaemia

Patients with liver disease have impaired hepatic glycogen storage and are prone to hypoglycaemia. Check blood glucose levels regularly. Give 10% glucose infusions if <2mmol/L, and monitor plasma K^+.

Immune function

Infections of the respiratory and urinary tracts are common. In the presence of ascites, spontaneous bacterial peritonitis may cause significant sepsis—have a low index of suspicion. Intraoperative antibiotic prophylaxis should be given where indicated.

Fluid resuscitation

Sodium chloride 0.9% is recommended 1st line, and sodium bicarbonate may be considered if significant acidosis. Addition of glucose-containing fluids if hypoglycaemic. Caution is required as over-resuscitation with crystalloid may worsen cerebral oedema.

Other considerations

IM and SC injections risk haematoma formation if coagulopathic or thrombocytopenic. Care with positioning; the skin may be fragile. Muscle wasting may leave patients prone to neuropraxia and pressure damage.

Postoperative considerations

- Patients with advanced liver disease will need postoperative intensive care or high dependency care.
- Postoperative decompensation of CLD carries a high mortality.
- Constipating analgesics, such as opioids, should be prescribed with concurrent lactulose to prevent encephalopathy.
- Postoperative ileus may also precipitate encephalopathy in cirrhotic patients.
- Complications include delayed wound healing, sepsis, renal impairment and bleeding.

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- Fluid balance should be carefully monitored postoperatively, aiming for a urine output of 1mL/kg/h.
- Coagulopathy increases the risk of postoperative bleeding and haematoma formation.

Specific considerations for ALF

⚠ Active acute hepatitis is a contraindication to elective surgery. Due to high perioperative mortality, patients should have all surgery postponed (unless a true emergency) until at least 30d after LFTs have returned to normal.

- Care is largely supportive, with specific treatments depending on underlying cause (acetylcysteine infusion in paracetamol overdose, delivery of fetus if pregnancy-related, penicillin for *Amanita phalloides*, chelating agents and/or haemodialysis in iron overdose, protease inhibitors in acute hepatitis B infection, aciclovir in herpes simplex virus infection and corticosteroids in autoimmune hepatitis).
- Coagulation: reversal of coagulopathy should not be routinely carried out, as PT provides a surrogate marker of hepatic function. Reversal is indicated for invasive procedures or active bleeding.
- Management may be best delivered in an ICU/HDU environment. The presence of grade II encephalopathy or worse should prompt ICU review.
- Patients with encephalopathy, deteriorating INR, hypoglycaemia or acidosis should be discussed urgently with a hepatologist in a specialist liver unit (for King's College criteria for liver transplant, see Box 8.2).
- Orthotopic liver transplantation may be the definitive treatment in some severe cases. Spontaneous recovery is only seen in around 40% of cases.

Box 8.2 King's College criteria for transplant referral in acute liver disease

Paracetamol

- Arterial pH <7.25 following volume resuscitation >24h post-overdose (OD)

Or

- Serum lactate >3.5 following volume resuscitation >12h post-OD

Or

- Serum lactate >2.5 following volume resuscitation >24h post-OD

Or ALL of the following:

- Grade III–IV encephalopathy
- Creatinine >300 micromoles/L
- PT >100s

(or two of the above with non-septic deterioration in organ function, e.g. vasopressors, increasing FiO_2)

Non-paracetamol

- PT >100s

Or

- Presence of encephalopathy AND any three of the following:
  - Age <10 or >40y
  - PT >50s
  - Bilirubin >300 micromoles/L
  - Duration of jaundice before encephalopathy >7d
  - Aetiology: non-A, non-B hepatitis, drug reaction

Source: data from O'Grady JG *et al*. Early indicators of prognosis in fulminant hepatic failure, *Gastroenterology*, 1989;**97**(2):439–45.

Drug metabolism and liver disease

- The vast majority of drugs, including anaesthetic drugs, are metabolised by the liver (Table 8.5).
- Most drugs are initially metabolised by the cytochrome P450 system. In phase I, they are either oxidised or reduced, and in phase II, they are conjugated with a glucuronide, glycine or sulphate to enhance water solubility and excretion in bile or urine.
- In early alcoholic liver disease, the cytochrome P450 system is often induced, leading to rapid metabolism of drugs, whereas this is reversed in end-stage disease.
- The liver has a large functional reserve, so these functions are usually preserved until end-stage disease.
- Pharmacodynamics and the sensitivity of target organs for sedatives and anaesthetics may be altered, with coma easily induced in end-stage liver disease.
- ↓ portal blood flow in hepatic fibrosis reduces 1st-pass metabolism.
- Hypoalbuminaemia increases free drug in plasma.
- There is an ↑ volume of distribution due to ascites and Na^+/water retention.
- Biotransformation enzymes may increase or decrease activity.
- Reduced liver cell mass results in reduced enzyme synthesis (e.g. plasma cholinesterase) activation or deactivation.
- ↓ biliary excretion of drugs due to obstructive jaundice.

Table 8.5 Anaesthetic drugs in liver failure

	Drugs safe in liver failure	Drugs to be used with caution (may need reduced dosage)	Drugs contraindicated in liver failure
Premedication	Lorazepam	Midazolam, diazepam	
Induction	Propofol, thiopental, etomidate		
Maintenance	Desflurane, sevoflurane, isoflurane, N_2O	Enflurane	Halothane (possibly)*
Muscle relaxants	Atracurium, cisatracurium	Rocuronium, vecuronium, suxamethonium	
Opioids	Remifentanil	Fentanyl, alfentanil, morphine, pethidine	
Analgesics	Paracetamol	NSAIDs, lidocaine, bupivacaine	

* Halothane has been rarely reported to cause hepatitis (see p. 213).

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Postoperative liver dysfunction or jaundice

Postoperative jaundice is relatively common; haematoma reabsorption or blood transfusion can result in a high bilirubin load. Significant liver dysfunction is uncommon in previously healthy patients.

Mild elevation of liver transaminases has a varied aetiology and often resolves without treatment. LFTs more than double the normal range suggest hepatocellular injury. Hepatitis due to volatile agents is extremely rare, especially with newer agents, and is largely a diagnosis of exclusion.

- Common causes include reduced hepatic O_2 delivery 2° to hypoxia or hypotension, drugs, trauma or infective causes (Table 8.6).
- Benign postoperative intrahepatic cholestasis mimics biliary obstruction and usually occurs after major abdominal surgery.

Table 8.6 Causes of postoperative liver dysfunction or jaundice

Bilirubin overload (haemolysis)	Blood transfusion Haematoma resorption Haemolytic anaemia (sickle-cell, prosthetic heart valve, glucose-6-phosphate dehydrogenase deficiency)
Hepatocellular injury	Exacerbation of pre-existing liver disease Hepatic ischaemia: hypovolaemia, hypotension, cardiac failure Septicaemia Drug-induced (antibiotics, halothane) Hypoxia Viral hepatitis
Cholestasis	Intrahepatic (benign, infection, drug-induced, e.g. cephalosporins, carbamazepine, erythromycin) Extrahepatic (pancreatitis, gallstones, bile duct injury)
Congenital	Gilbert's syndrome

Halothane hepatitis

The use of halothane has been largely superseded by other volatile agents, so it is becoming a historical phenomenon. Halothane has been linked to postoperative liver dysfunction. Two syndromes are recognised:

- The first is associated with a transient rise in LFTs and low morbidity, often after initial exposure.
- The second is thought to occur after repeated exposure and has an 'immune' mechanism with the development of fulminant hepatic failure and high mortality. It is rare, with an incidence of 1 in 35 000 anaesthetics.
- Antibodies specific to fulminant hepatic failure patients exposed to halothane are found in 70% of such patients. It is postulated that a halothane oxidative metabolite binds to liver cytochromes to form a hapten and induces a hypersensitivity reaction. All patients exposed to halothane have altered liver proteins, but it is unknown why only a few develop liver failure.

Other inhalational anaesthetic agents

- The chance of an 'immune' reaction to a volatile agent occurring is thought to relate to the amount it is metabolised. Halothane is 20% metabolised, enflurane 2% and isoflurane 0.2%. Products of enflurane metabolism have been shown to alter liver proteins and there have been rare case reports linking enflurane with liver damage. There is a theoretical basis for crossreactivity with previous halothane exposure. Isoflurane is considered safe for use in patients at risk of hepatic failure, as are sevoflurane and desflurane.

Intravenous fluids in liver disease

- It is important to maintain adequate peri- and postoperative hydration, as there is a high risk of AKI.
- Glucose 5% or 10% is unsuitable as a resuscitation or maintenance fluid, as it provides little intravascular volume replacement and may exacerbate hyponatraemia and cerebral oedema. It is useful in the correction of hypoglycaemia.
- Sodium chloride 0.9% or Hartmann's solution are both good choices of crystalloid, although Hartmann's may present an external lactate load and the high Na^+ load in 0.9% sodium chloride may worsen ascites.
- Human albumin solution (HAS) 4.5% is a useful colloid, especially if synthetic liver function is impaired and serum albumin is low.
- If oliguria persists despite adequate fluid resuscitation, IV terlipressin 0.5–2mg IV qds, in conjunction with daily HAS, may improve renal function.
- Perioperative removal of ascites will result in postoperative reaccumulation. This should be accounted for in the fluid balance.

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Further reading

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Chapter 9

Endocrine and metabolic disease

Joanna Poole and **Hannah Blanshard**

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See also:
➲ Endocrine surgery pp. *709–21*

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Diabetes mellitus

Insulin is necessary, even when fasting, to maintain glucose homeostasis and balance stress hormones (e.g. adrenaline). It has two classes of action:

- Excitatory: stimulating glucose uptake and lipid synthesis
- Inhibitory (physiologically more important): inhibits lipolysis, proteolysis, glycogenolysis, gluconeogenesis and ketogenesis.

Lack of insulin is associated with hyperglycaemia, osmotic diuresis, dehydration, hyperosmolarity, hyperviscosity predisposing to thrombosis and ↑ rates of wound infection. Sustained hyperglycaemia is associated with ↑ mortality, hospital stay and complication rates.

DM is present in 10–15% of the surgical population.

- Type 1 diabetes (20%): immune-mediated and leads to absolute insulin deficiency. Patients cannot tolerate prolonged periods without exogenous insulin. Glycogenolysis and gluconeogenesis occur, resulting in hyperglycaemia and ketosis. Treatment is with insulin.
- Type 2 diabetes (80%): a disease of adult onset, associated with insulin resistance. Patients produce some endogenous insulin and their metabolic state often improves with fasting. Treatment may be diet control, oral hypoglycaemics and/or insulin.

General considerations

Many diabetic patients are well informed about their condition and have undergone previous surgery; discuss management with them. Hospital diabetes teams can be useful for advice. The overall aims of perioperative diabetic management are to maintain physiological glucose levels (above hypoglycaemic levels, but below those at which deleterious effects of hyperglycaemia become evident) and prevent hypokalaemia, hypomagnesaemia and hypophosphataemia.

Preoperative assessment

- CVS: IHD (sometimes asymptomatic), cerebrovascular disease, MI and cardiomyopathy are all associated with DM. Autonomic neuropathy can lead to tachy- or bradycardia and postural hypotension.
- Renal: 40% of diabetics develop microalbuminuria, which is associated with hypertension, IHD and retinopathy. This may be reduced by treatment with ACE inhibitors.
- Respiratory: perioperative chest infections are more common, especially if other risk factors such as smoking and obesity.
- Airway: thickening of soft tissues (glycosylation) occurs, especially in ligaments around joints, leading to limited joint mobility syndrome. Intubation may be difficult if the neck is affected or there is insufficient mouth opening.
- GI: 50% of patients have delayed gastric emptying ± reflux.
- ↑ risk of wound infection.
- Peripheral neuropathy is common; document any existing sensory loss if regional technique planned.

Investigations

- Measure glycosylated Hb (HbA1c), a measure of recent glycaemic control (normal 20–48mmol/mol, 4–6.5%). If HbA1c is >69mmol/mol (8.5%), refer to the team that manages their diabetes for optimisation. Surgery may then proceed with caution. A value >85mmol/mol (10%) suggests inadequate control. Refer to the diabetes team, and only proceed if surgery is urgent or if they feel the patient's control is as good as it can be.
- Patients with hypoglycaemic unawareness should be referred to the diabetes specialist team, irrespective of HbA1c.

Preoperative management

- Make an individualised diabetes management plan, agreed with the patient, for the preadmission and perioperative period.
- Place the patient 1st on the operating list, if possible.
- Individuals with type 1 diabetes should NEVER go without insulin, as they are at risk of diabetic ketoacidosis.
- The Enhanced Recovery Partnership Programme recommends high-carbohydrate drinks prior to surgery. This may compromise blood sugar control and is not recommended for people with insulin-treated diabetes.
- Patients with a planned short starvation period (no more than one missed meal in total) should be managed by modification of their usual diabetes regime, avoiding a variable-rate IV insulin infusion (VRIII) wherever possible.
- Patients expected to miss >1 meal should have a VRIII.
- For suggested perioperative management of insulin, see Table 9.1. For suggested perioperative management of non-insulin diabetic medication, see Table 9.2.

Perioperative management

- Monitor blood glucose on admission, and hourly during the day of surgery. Aim for blood glucose level of 6–10mmol/L; 4–12mmol/L is acceptable.
- If blood glucose is >12mmol/L either pre- or post-surgery, check capillary blood ketones or urinary ketones. If capillary blood ketones are >3mmol/L or urinary ketones > +++, cancel surgery.
- Consider an RSI if gastric stasis is suspected.
- Regional techniques may be useful for extremity surgery and to reduce the risk of undetected hypoglycaemia.
- Autonomic dysfunction may exacerbate the hypotensive effect of spinals and epidurals.

Patients undergoing surgery with a long starvation period (i.e. two or more missed meals)

- A number of glucose, K^+ and insulin regimes have been described in the past (e.g. Alberti), but the VRIII is widely used and should be commenced on admission.
- If the patient is already on a long-acting insulin analogue, this should be continued at 80% the usual dose, even if planning to use a VRIII through the perioperative period.

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Table 9.1 Perioperative management of insulin therapy when no more than one missed meal

	Morning surgery	Afternoon surgery
	Always check blood glucose on admission	
Once daily in evening	Reduce the night before by 20%	Reduce the night before by 20%
Once daily in morning	Reduce by 20% on day before surgery AND on day of surgery	Reduce by 20% on day before surgery AND on day of surgery
Twice daily: one injection twice a day, i.e. biphasic or ultralong acting	Halve the usual morning dose	Halve the usual morning dose
Twice daily: two injections twice a day, i.e. short and intermediate acting	Calculate total dose of morning insulin(s); give half as intermediate acting only in the morning	Calculate total dose of morning insulin(s); give half as intermediate acting only in the morning
3–5 injections daily	Basal bolus regimens: omit morning and lunchtime short-acting insulins; keep basal unchanged Premixed morning insulin: halve morning dose and omit lunchtime dose	Give usual morning insulin dose(s); omit lunchtime dose

Source: data from Barker P *et al*. (2015) Peri-operative management of the surgical patient with diabetes. *Anaesthesia*, **70**, 1427–40.

- Glucose/insulin infusions should be administered through the same cannula to prevent accidental administration of insulin without glucose. Both infusions should be regulated by volumetric pumps, with an antireflux valve on the IV glucose line.
- Hartmann's solution should be used in preference to 0.9% sodium chloride in those patients not requiring a VRIII.
- The recommended 1st-choice solution for VRIII is 0.45% sodium chloride with 5% glucose, and either 0.15% or 0.3% potassium chloride (KCl); however, this is not always available.
- Glucose 4% and 0.18% sodium chloride; 10% glucose or 5% glucose are acceptable. Whenever giving hypotonic parenteral fluids, beware of hyponatraemia. Preferably give 10% glucose at 60mL/h, rather than 5% glucose at 120mL/h (prevents water overload, particularly in the elderly).
- If K^+ <4.5mmol/L, add 10mmol KCl to each 500mL bag of glucose.
- Start VRIII using a syringe pump. Adjust according to local guidelines for VRIII (see example in Table 9.3). Test blood glucose hourly initially. Patients on >100 units of insulin/day will need higher doses of insulin by infusion.

Table 9.2 Perioperative management of oral diabetic medication when no more than one missed meal

	Morning surgery	Afternoon surgery
	Always check blood glucose on admission	
Acarbose	Omit morning dose if NBM	Give morning dose if eating
Meglitinide (repaglinide or nateglinide)	Omit morning dose if NBM	Give morning dose if eating
Metformin*	Take as normal	Take as normal
Sulfonylurea (e.g. glibenclamide, gliclazide)	Omit morning dose (whether once daily or twice daily)	Omit all (whether once daily or twice daily)
SGLT-2 inhibitors (e.g. dapagliflozin)	Omit	Omit
Pioglitazone	Take as normal	Take as normal
DDP-4 inhibitor (e.g. sitagliptin, vildagliptin, saxagliptin)	Take as normal	Take as normal
GLP-1 analogue (e.g. exenatide, liraglutide)	Take as normal	Take as normal

DPP-4, dipeptidyl peptidase-4; GLP-1, glucagon-like peptide-1; NBM, nil by mouth; SGLT-2, sodium–glucose cotransporter-2.

* If contrast medium is to be used and estimated GFR <50mL/min/1.73², metformin should be omitted on the day of surgery and for the following 48h.

Source: data from Barker P *et al*. (2015) Peri-operative management of the surgical patient with diabetes. *Anaesthesia*, **70**, 1427–1440.

Table 9.3 VRIII infusion

Blood glucose (mmol/L)	Initial rate of insulin infusion (units/h)	Insulin infusion rate if blood glucose not <10mmol/L (units/h)
<4.0	0.5—if a long-acting insulin has been continued, stop and treat as for hypoglycaemia	
4.1–7.0	1.0	2.0
7.1–9.0	2.0	3.0
9.1–11.0	3.0	4.0
11.1–14.0	4.0	5.0
14.1–17.0	5.0	6.0
17.1–20.0	6.0	8.0
>20	Check infusion running, and seek diabetes team or medical advice)	

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Perioperative treatment of hyperglycaemia

For well-controlled patients (HbA1c <69mmol/mol) undergoing surgery with a short starvation period (one missed meal) and preoperative hyperglycaemia (blood glucose >12mmol/L):

- Type 1 diabetes: give SC rapid-acting insulin analogue. Assume that 1 unit will drop blood glucose by 3mmol/L, but take advice from the patient wherever possible. Recheck blood glucose hourly. If surgery cannot be delayed, commence VRIII.
- Type 2 diabetes: give 0.1 units/kg of SC rapid-acting insulin analogue, and recheck blood glucose 1h later to ensure it is falling. If surgery cannot be delayed or the response is inadequate, commence VRIII.

Hypoglycaemia

- Blood glucose <4mmol/L is the main danger to diabetics perioperatively. Fasting, recent alcohol consumption, liver failure and septicaemia commonly exacerbate this.
- Characteristic signs are tachycardia, light-headedness, sweating and pallor. This may progress to confusion, restlessness, incomprehensible speech, double vision, convulsions and coma. If untreated, permanent brain damage will occur, made worse by hypotension and hypoxia.
- Anaesthetised patients may not show any of these signs. Monitor blood sugar preoperatively and then hourly if stable, and suspect hypoglycaemia with unexplained changes in the patient's condition.
- If hypoglycaemia occurs, give 75mL of 20% glucose over 15min or 150mL of 10% glucose, and repeat blood glucose after 15min. Alternatively, give 1mg of glucagon (IM or IV); 10–20g (2–4 teaspoons) of sugar by mouth or an NGT is an alternative.

Transferring from a variable-rate intravenous insulin infusion to subcutaneous insulin or oral treatment

Restarting oral hypoglycaemic medication

- Recommence oral hypoglycaemic agents once the patient is ready to eat and drink.
- Be prepared to withhold or reduce sulfonylureas if the food intake is likely to be reduced.
- Metformin should only be recommenced if the eGFR >50mL/min/1.73^2.

Restarting subcutaneous insulin for patients already established on insulin

- Conversion to SC insulin should be delayed until the patient is able to eat and drink without nausea and vomiting.
- Should take place when the next meal-related SC insulin dose is due.
- Restart the normal presurgical regime. Be aware that insulin requirement may change due to postoperative stress, infection or altered food intake.
- Consult the diabetes team if blood sugar is outside the acceptable range (4–12mmol/L).
- Ensure overlap between the VRIII and the 1st injection of the fast-acting insulin. The fast-acting insulin should be injected SC with the meal, and the VRIII discontinued 30–60min later.

For patients on basal bolus insulin

- If the patient was previously on a long-acting insulin analogue, such as Lantus® or Levemir®, this should have been continued and so the patient only needs to restart their normal short-acting insulin at the next meal.

For patients on continuous subcutaneous insulin

- Commence the SC insulin infusion at their normal basal rate as long as not at bedtime.
- VRIII should be continued until the next meal bolus has been given.

Further reading

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ALGRAWANY

Acromegaly

A rare clinical syndrome caused by overproduction of growth hormone from the anterior pituitary. Patients may present for pituitary surgery (see ➲ pp. 567–8) or require surgery unrelated to their pituitary pathology.

Preoperative assessment

- Cardiac assessment for hypertension (30%), IHD, cardiomyopathy, heart failure, conduction defects and valvular disease.
- Possible difficult airway management and intubation due to large head and tongue, hypertrophy of the larynx and trachea, vocal cord thickening and strictures and chondrocalcinosis of the larynx. Consider direct/indirect laryngoscopy preoperatively if vocal cord or laryngeal pathology is suspected.
- Enlargement of the thyroid (25%) may compress the trachea.
- Twenty-five per cent of patients have diabetes.
- Snoring and daytime somnolence may indicate sleep apnoea (70%). Chronic OSA can cause elevated PAP.
- Kyphoscoliosis may worsen respiratory function.
- Medications: somatostatin analogues (octreotide, lanreotide) may cause vomiting and diarrhoea. Bromocriptine, a long-acting dopamine agonist, is often used to lower growth hormone levels and can cause severe postural hypotension.
- Symptoms and signs of raised ICP or nerve compression (e.g. carpal tunnel syndrome).

Investigations

- ECG as routine. Echocardiogram if patient symptomatic or has murmurs.
- CXR if cardiorespiratory problems.
- Blood glucose.

Conduct of anaesthesia

- Large face masks, long-bladed laryngoscopes and gum elastic bougies may make airway management and intubation easier. Use a videolaryngoscope (VL) if available. AFOI is the technique of choice for patients with anticipated difficult intubation. More problems experienced with extubation than intubation.
- A long table may be required.
- Nerve compression syndromes are common, so take care to protect vulnerable areas (ulnar nerve at the elbow, median nerve at the wrist, and common peroneal nerve below the knee).

Further reading

Menon R, Murphy P, Lindley A (2011). Anaesthesia and pituitary disease. *Contin Educ Anaesth Crit Care Pain*, **11**, 133–7.

Nemergut EC, Dumont AS, Barry UJ, *et al*. (2005). Perioperative management of patients undergoing transsphenoidal pituitary surgery. *Anesth Analg*, **101**, 1170–81.

Thyroid disease

May present for thyroidectomy (see ➲ pp. 710–13) or non-thyroid surgery.

General considerations for non-thyroid surgery

Hypothyroidism

- Commonly due to autoimmune thyroid destruction.
- CVS complications include ↓ blood volume, ↓ CO (up to 50%), bradycardia, hypotension and IHD. Pericardial effusions also occur.
- Respiratory muscle weakness, impaired hypoxic/hypercapnic drive and OSA.
- Also associated with anaemia, hypoglycaemia, hyponatraemia, ↑ thrombotic tendency, impaired hepatic drug metabolism and renal clearance affecting drug clearance.
- Reduced gastric motility and ↑ postoperative ileus.
- If clinical evidence of hypothyroidism, delay elective surgery to obtain a euthyroid state. Liaise with an endocrinologist. Suggest levothyroxine (T_4), starting dose 50 micrograms, increasing to 100–200 micrograms PO over several weeks. The elderly are susceptible to angina and heart failure after thyroxine replacement due to ↑ cardiac work. Start with 25 micrograms T_4, and increase by 25 micrograms at 3- to 4-weekly intervals.
- If surgery is urgent, use liothyronine (T_3), 10–50 micrograms slow IV injection with ECG monitoring (consider half dose if suspected cardiac disease).
- Euthyroid sick syndrome refers to suppression of serum thyroid hormone occurring in euthyroid patients due to concurrent illness. Subclinical hypothyroidism is a risk factor for postoperative AF, but there is no clear evidence hormone replacement should be given in this situation.
- Myxoedema coma is an emergency with mortality of up to 80%. Presents with confusion, drowsiness, bradycardia, hypotension and hypothermia. May be precipitated by infection or cold and require IV thyroxine replacement.

Hyperthyroidism (thyrotoxicosis)

- Typically presents with weight loss, hypertension, sweating and cardiac arrhythmias (especially AF). Treatment is with carbimazole (30–45mg PO daily for 6–8w). This inhibits iodination of tyrosyl residues in thyroglobulin. Occasionally, in severe cases with a large thyroid, Lugol's iodine is substituted 10d preoperatively to reduce gland vascularity.
- β-blockade is started if there are signs of tremor or palpitations. Non-cardioselective β-blockers (propranolol 30–60mg tds) are most effective. β1-adrenergic blockade treats tachycardia and β2-adrenergic blockade prevents the peripheral conversion of T_4 to T_3. Calcium channel blockers are an alternative for those with reactive airway disease.

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Preoperative assessment

- Elective surgery should only occur when patient is euthyroid (HR <80bpm, no hand tremor). Patients with subclinical hypothyroidism usually have no anaesthetic problems and no delay is needed.[5]
- A goitre can cause respiratory obstruction, so look for tracheal deviation and retrosternal extension. Look for evidence of tracheal compression with shortness of breath (may be positional), dysphagia and stridor (occurs with 50% compression). Infiltrating carcinoma may make any neck movement difficult and is an independent predictor of difficult intubation.
- SVC obstruction can occur. Look for distended neck veins that do not change with respiration.
- Check for other autoimmune disorders.

Investigations

(See ➲ pp. 710–11.)

Conduct of anaesthesia

Hypothyroid patients

- Give all drugs slowly. Susceptible to profound hypotension, which may be relatively resistant to the effects of catecholamine therapy.
- Actively warm as low metabolic rate predisposes to hypothermia.
- Tendency to hypoventilate; controlled ventilation is recommended.
- Drug metabolism is slower, so reduce the dose of relaxants and opioids and monitor twitch response.

Hyperthyroid patients

- Continue β-blockade to reduce the possibility of a thyroid storm.
- Consider invasive arterial BP monitoring.

Special considerations

Thyroid storm

- A life-threatening exacerbation of the hyperthyroid state, with evidence of decompensation in one or more organ systems (mortality 20–30%).
- Usually presents 6–24h post-surgery with fever (>40°C), sweating, sinus tachycardia (>140bpm), coma, nausea, vomiting and diarrhoea.
- If presents intraoperatively, may be difficult to distinguish from MH. Higher mixed venous partial pressure of CO_2 and higher creatine kinase (CK) in MH.
- Rehydrate with IV 0.9% sodium chloride and glucose.
- Treat hyperthermia with tepid sponging and paracetamol. Do not give NSAIDs or aspirin, as they displace thyroid hormone from serum binding sites.
- Give propranolol (1mg increments, up to 10mg), with CVS monitoring, to decrease the pulse rate to <90bpm. Alternatively, give esmolol (loading dose 250–500 micrograms/kg, followed by 50–100 micrograms/kg/min).
- Give hydrocortisone (200mg IV qds) to treat adrenal insufficiency and to decrease T_4 release and conversion to T_3 at very high levels.

- Give propylthiouracil (1g loading dose via NGT, followed by 200–300mg qds). This inhibits thyroid hormone release and also decreases the peripheral conversion of T_4 to T_3 (not immediate).
- After blockade by propylthiouracil, give sodium iodide (500mg tds IV), potassium iodide (five drops qds via NGT) or Lugol's iodine (5–10 drops qds via NGT).

Hypothyroid coma

- A rare form of decompensated hypothyroidism (mortality 15–20%), precipitated by infection, trauma, cold and CNS depressants.
- Characterised by coma, hypoventilation, bradycardia, hypotension and severe dilutional hyponatraemia.
- Rehydrate with IV glucose and 0.9% sodium chloride.
- May require cardiorespiratory stabilisation and ventilation.
- Warm slowly. Sudden warming may lead to extreme peripheral vasodilation.
- Give stress-dose steroids (e.g. hydrocortisone 100mg qds IV), in case they have concomitant 1° or 2° adrenal insufficiency, a common result of hypothyroidism.
- Give levothyroxine 200–400 micrograms IV bolus, followed by 100 micrograms the next day, less in patients with CVS disease. Consider a combination of IV T_3 and T_4, particularly if urgent surgery required. The conversion of T_4 to T_3 is suppressed in hypothyroid coma, and T_3 is more active than T_4. In hypothyroid coma give 5–20 micrograms liothyronine sodium IV, every 12h; can be increased to 5–20 micrograms every 4h if required.
- Transfer to ICU.

Further reading

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Bahn RS, Burch HB, Cooper DS, *et al*. (2011). Hyperthyroidism and other causes of thyrotoxicosis: management guidelines of the American Thyroid Association and American Association of Clinical Endocrinologists. *Thyroid*, **21**, 593–646.

ALGRAWANY

Parathyroid disorders

General considerations

The parathyroid glands secrete parathyroid hormone (PTH), which acts on the bones and kidneys to increase serum Ca^{2+} and decrease serum phosphate. It stimulates osteoclasts to release Ca^{2+} and phosphate into the ECF and simultaneously increases phosphate excretion and Ca^{2+} reabsorption in the kidney. Patients may present for parathyroidectomy (see ➔ pp. 714–15) and non-parathyroid-related surgery.

Hyperparathyroidism

- 1° hyperthyroidism: usually an adenoma causing high PTH, high Ca^{2+} and low phosphate. Associated with familial multiple endocrine neoplasia (MEN) type 1. Tumours rarely palpable and are located at surgery. Methylthioninium chloride (methylene blue) up to 1mg/kg is given preoperatively to localise the parathyroid gland.
- Fifty per cent of cases are asymptomatic, and presentation is often subtle. May present with anorexia, dyspepsia, nausea, vomiting and constipation, hypertension, shortened QT interval, polydipsia, polyuria, renal calculi, depression, poor memory and drowsiness.

Secondary hyperparathyroidism

- Results from compensatory parathyroid hypertrophy due to chronic low Ca^{2+}. Complicates CKD.
- Parathyroid hyperplasia causes high PTH, normal or low Ca^{2+} level and a high phosphate level.
- Usually presents as excessive bone resorption (seen earliest in the radial aspect of the middle phalanx of the 2nd digit) or calcification of the vascular system, organs and soft tissues.
- Treat medically with dietary phosphate restriction and calcium and vitamin D supplements. Medical therapy fails in 5–10% of patients on long-term dialysis and surgery becomes necessary.
- Risks of surgery are bleeding, recurrent hyperparathyroidism, hypoparathyroidism and injury to the recurrent laryngeal nerves. Patients should undergo dialysis within 1d of surgery and then 48h postoperatively or as required.
- Watch for postoperative hypocalcaemia and hypomagnesaemia.

Tertiary hyperparathyroidism

- Parathyroid hyperplasia progresses to autonomous secretion, behaving like an adenoma. Excessive secretion of PTH continues, despite correction of renal failure. Only a few cases require operation.

Hypercalcaemic crisis

- Occurs most commonly in the elderly with undiagnosed hyperparathyroidism and with malignant disease. Dehydration results in anorexia and nausea/vomiting which exacerbates the cycle. Characterised by weakness, lethargy, mental changes, abdominal pain and coma.
- Serum Ca^{2+} >4.5mmol/L is life-threatening and can be rapidly, but transiently lowered with phosphate (500mL of 0.1M neutral solution over 6–8h).

- Rehydration is the 1° treatment (4–6L of fluid often required).
- Pamidronate (60mg in 500mL of 0.9% sodium chloride over 4h) is the 1st-line treatment. Bisphosphonates are potent inhibitors of osteoclastic bone resorption. Effect is rapid and long-lasting.
- Calcitonin, 3–4U/kg IV, then 4U/kg SC bd, causes a rapid, but temporary decrease in skeletal release of Ca^{2+} and phosphate.
- The 2nd-line treatment, once volume repletion has been achieved, is forced 0.9% sodium chloride diuresis with furosemide (40mg IV every 4h). Loop diuretics decrease the proximal tubular resorption of Ca^{2+}. Consider central pressure monitoring in the elderly at risk of LV failure.
- Hydrocortisone (200–400mg IV daily) in patients with malignancy.
- Dialysis is reserved for patients with renal failure.

Perioperative plan

(See ➲ p. 715.)

- If serum Ca^{2+} <3mmol/L, with no ECG changes or end-organ impairment, then proceed with the operation.
- If serum Ca^{2+} >3mmol/L, ECG is abnormal or CVS or renal impairment, the operation should be postponed until after treatment.

Hypoparathyroidism

- Usually caused by parathyroidectomy, but post-radiotherapy and idiopathic cases also occur. Patients with a history of extensive neck dissection should have serum Ca^{2+} measured before surgery.
- Results in hypocalcaemia (ionised Ca^{2+} <0.9mmol/L, corrected Ca^{2+} <2.2mmol/L). Trough level usually occurs at 20h following parathyroidectomy and normalises by d3.
- Corrected Ca^{2+} is total Ca^{2+} corrected to albumin concentration; add 0.1mmol/L to Ca^{2+} for each 5g/L albumin is below 40g/L.
- The presenting features are due to low Ca^{2+} levels and manifest as carpopedal spasm, tetany, dysrhythmia, hypotension and prolonged P–R interval on ECG.
- Treat with Ca^{2+} (calcium gluconate 10mL 10% IV over 10min, followed by 40mL in 1L of 0.9% sodium chloride over 8h).
- Low serum magnesium is also common and can be treated with magnesium sulfate (1–5mmol IV slowly).

Further reading

Sasidharan P, Johnston IG (2009). *Parathyroid physiology and anaesthesia. Anaesthesia tutorial of the week.* https://www.frca.co.uk/Documents/142%20Anaesthesia%20&%20the%20parathyroid%20gland.pdf

ALGRAWANY

Adrenocortical insufficiency

Primary adrenocortical insufficiency (Addison's disease)

Destruction of adrenal cortex by autoimmune disease (75%), infection (tuberculosis (TB)), septicaemia, acquired immune deficiency syndrome (AIDS), haemorrhage, metastases, and surgery. Associated with glucocorticoid and mineralocorticoid deficiency.

Secondary adrenocortical insufficiency

Insufficient adrenocorticotrophic hormone (ACTH) to stimulate the adrenal cortex due to pituitary suppression by exogenous steroids or generalised hypopituitarism usually from pituitary or hypothalamic tumours. Associated with glucocorticoid deficiency only.

Clinical features of adrenal insufficiency

Weakness, fatigue (100%), nausea, vomiting, diarrhoea, weight loss (60%), myalgia and joint pain. Some features more pronounced or found only in Addison's disease: skin hyperpigmentation (90%), postural hypotension (90%) and salt craving. Skin pallor found only in 2° insufficiency.

Acute adrenal crisis

Medical emergency characterised by dizziness, weakness, sweating, abdominal pain, nausea and vomiting or coma.

Causes may be inadequate stress-related steroid replacement in patients with chronic adrenal insufficiency, adrenal haemorrhage or pituitary apoplexy (apoplexy is defined as a sudden neurological impairment, usually due to a vascular process, i.e. infarction or haemorrhage).

Investigations

- Serum shows: low glucose, low Na^+ (90%) and raised K^+ (70%). Ca^{2+}, urea and creatinine are raised in Addison's disease only.
- Table 9.4 shows biochemical diagnosis of adrenal insufficiency.

Treatment

- Hydrocortisone, 20mg in the morning and 10mg at night PO.
- Fludrocortisone, 0.1mg PO, to replace aldosterone in Addison's disease.

Perioperative management of patients with Addison's disease

- Joint care with an endocrinologist is advisable.
- Give all medication on the morning of surgery.
- For any nil-by-mouth regime, give IV 0.9% sodium chloride to prevent dehydration and maintain mineralocorticoid stability.
- Hydrocortisone 100mg before surgery, then continuous infusion at rate of 200mg/24h intraoperatively. Postoperatively, the infusion can be reduced to 100mg/24h infusion or give 50mg 6-hourly.
- Once eating and drinking, start double normal dose for 48h, extended to 1w if recovery complicated.
- It is worth noting that 20mg hydrocortisone is equivalent to 0.05mg fludrocortisone, so with hydrocortisone doses of 50mg or more, mineralocorticoid replacement in 1° adrenal insufficiency can be reduced.
- If any postoperative complications arise, e.g. fever, delay the return to normal dose.
- Four-hourly blood glucose and daily electrolytes.

Table 9.4 Biochemical diagnosis of adrenal insufficiency

Test	Normal range	Definite adrenal insufficiency	
		1°	2°
Early morning cortisol	165–680nmol/L	Cortisol <165nmol/L and ACTH >22.0pmol/L	Cortisol <100nmol/L
Early morning ACTH	1.1–11.0pmol/L		Not diagnostic
Standard short Synacthen® test*	Peak cortisol >500nmol/L	Peak cortisol <500nmol/L	Peak cortisol <500nmol/L
Insulin tolerance test**	Peak cortisol >500nmol/L		Peak cortisol <500nmol/L

* Serum cortisol at 0 and 30min after 250 micrograms of Synacthen® IV.

** Serum glucose and cortisol at 0, 15, 30, 45, 60 and 90min after insulin (0.1–0.15 units/kg IV). Test only valid if symptomatic hypoglycaemia (serum glucose <2.2mmol/L) is achieved. Gold standard test—close supervision mandatory.

Adrenal crisis (Addisonian crisis)

- Classically presents as hypotension, hyponatraemia, hyperkalaemia and hypoglycaemia with abdominal pain.
- Can resemble hypovolaemic shock but can also mimic septic shock with fever, peripheral vasodilation and high CO.
- In patients with type 1 diabetes, deterioration of glycaemic control with recurrent hypoglycaemia can be a presenting sign of a crisis.
- O_2 100% and ventilatory support if necessary. Refer to ICU/HDU.
- IV fluids to restore blood volume, 0.9% sodium chloride to replace Na^+ deficit initially at 1000mL/h and glucose for hypoglycaemia.
- Baseline cortisol and ACTH prior to hydrocortisone.
- Hydrocortisone 200mg, followed by 100mg qds. If given IV, no faster than 10mg/min to avoid vascular damage.
- Dexamethasone 4mg IV can be given if the diagnosis has not been confirmed, since this does not interfere with the measurement of cortisol and ACTH stimulation testing.
- Vasopressor resistance may occur before steroid replacement.
- Treat the precipitating cause.

Relative adrenal insufficiency in the critically ill

- Relative hypoadrenalism in ICU patients occurs in ~30–50% of septic patients. Consider in patients with increasing inotrope/vasopressor requirements or prolonged mechanical ventilation. Treat with 200mg hydrocortisone IV.
- Abnormal short Synacthen® test is a poor prognostic indicator.

Further reading

Wass J, Howlett T, Arlt W, *et al*. (2017). Surgical guidelines for Addison's disease and other forms of adrenal insufficiency. *Addison's Disease Self-Help Group Surgical Guidelines*, **A5**. https://www.addisonsdisease.org.uk/surgery

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ALGRAWANY

The patient on steroids

Steroids are commonly used to suppress inflammatory and immunological responses, as well as in treatment of adrenocortical insufficiency. These patients may develop perioperative complications from their underlying disease or from a potentially impaired stress response due to hypothalamic–pituitary–adrenal (HPA) suppression. Recent research suggests that small, physiological replacement doses are adequate. Table 9.5 shows the AoA guidance on recommended doses for intra- and postoperative steroid cover in adults receiving adrenosuppressive doses of steroids (equivalent to ≥5mg prednisolone).

Table 9.5 AoA guidelines on adult glucocorticoid replacement (2020)

	Intraoperative	Postoperative
Major surgery and CS	100mg hydrocortisone at induction, then initiate infusion at rate 200mg/24h. Alternatively, 6–8mg dexamethasone at induction will suffice for 24h	Reduce infusion rate by 50% and normal dose when eating Complicated recovery: continue double normal dose for 2–7d or until complications resolve
Body surface + intermediate surgery	As above	Double regular dose for 2d
Bowel procedures	HPA suppression possible: IV fluids + 100mg hydrocortisone IV for bowel prep and induction HPA suppression unlikely: continue normal dose (equivalent IV dose if nil by mouth)	Double normal dose for 24h
Labour	Hydrocortisone 100mg IV at onset of labour, followed by infusion at rate 200mg/24h	

Source: data from Woodcock, T *et al*. (2020) Guidelines for the management of glucocorticoids during the peri-operative period for patients with adrenal insufficiency. *Anaesthesia*, **75**:654–63.

Hypothalamic–pituitary–adrenal suppression

- Endogenous cortisol (hydrocortisone) production is of the order of 25–30mg/24h (following a circadian pattern). During stress induced by major surgery, it rises to 75–100mg/d and can remain elevated for a variable period of time (up to 72h following cardiac surgery).
- Prednisolone is a synthetic glucocorticoid with the general properties of corticosteroids. Prednisolone exceeds hydrocortisone in glucocorticoid and anti-inflammatory activity, being 3–4 times more potent on a weight basis than the parent hormone; however, it is less active than

hydrocortisone in mineralocorticoid activity. It is often given PO for chronic conditions to limit water retention. In contrast, hydrocortisone can be given IV or PO, making it suitable for use perioperatively (for conversion, see Box 9.1). Its high mineralocorticoid activity and resulting fluid retention make it unsuitable for chronic disease suppression.

- Fludrocortisone is available only in the oral preparation. It may be withheld on the day of surgery and while the patient is receiving stress doses of hydrocortisone.
- Low-dose steroid treatment (<10mg prednisolone per day) usually carries little danger of HPA suppression.
- HPA suppression may occur after PO, topical, parenteral, nebulised and inhaled routes (e.g. 1500 micrograms/d beclometasone).
- HPA suppression can be measured using various methods. In practice, the short Synacthen® (corticotropin) test is reliable and cheap.
- Patients are given 250 micrograms IV Synacthen® (synthetic corticotropin), and serum cortisol is measured at 0, 30 and 60min. Normal peak cortisol (420–700nmol/L) indicate the ability of the patient to mount a stress response. Insulin tolerance tests can be performed under the supervision of an endocrinologist, for equivocal results.

Box 9.1 Steroid equivalence

Prednisolone 5mg equivalent to:	Hydrocortisone 20mg
	Methylprednisolone 4mg
	Betamethasone 750 micrograms
	Dexamethasone 750 micrograms
	Cortisone acetate 25mg
	Deflazacort 6mg
	Triamcinolone 4mg

Further reading

Woodcock T, Barker P, Daniel S, *et al.* (2020). Guidelines for the management of glucocorticoids during the peri-operative period for patients with adrenal insufficiency. *Anaesthesia*, **75**, 654–63.

Liu MM, Reidy AB, Saatee S, *et al.* (2017). Perioperative steroid management: approaches based on current evidence. *Anesthesiology*, **127**, 166–72.

ALGRAWANY

Cushing's syndrome

Cushing's syndrome describes excess plasma cortisol. Commonly caused by overtreatment with steroids. The 2nd commonest cause is a pituitary adenoma (Cushing's disease). Ectopic ACTH (e.g. oat cell carcinoma of lung), adrenal adenoma and adrenal carcinoma are also possible causes.

Clinical features

- Round 'moon' face, truncal obesity, bruised and fragile skin
- Proximal myopathy and osteoporosis
- High Na^+, bicarbonate (HCO_3^-) and glucose; low K^+ and Ca^{2+}.

Diagnosis

- High plasma cortisol with loss of diurnal variation (peak 6 a.m., trough midnight). Normal cortisol range ~140–700nmol/L dependent on time of day and clinical context.
- ↑ urinary 17-(OH)-steroids.
- Loss of suppression with dexamethasone 2mg.
- ACTH level high with ectopic ACTH, normal/high in pituitary disease and low with adrenal disease or ectopic cortisol administration.

Preoperative assessment

- Many patients have ECG abnormalities (high-voltage QRS and inverted T waves), which may make IHD difficult to exclude. These will revert to normal after curative surgery.
- Eighty-five per cent of patients are hypertensive, often poorly controlled, and LVH should be looked for.
- Sleep apnoea and gastro-oesophageal reflux are common and difficult intubation should be anticipated if facial adipose tissue ↑.
- Sixty per cent of patients have diabetes or impaired glucose tolerance and a VRIII may be appropriate (see ➲ p. 219).
- Obesity may make venous access difficult.
- Patients are at risk of peptic ulcer disease, so give prophylactic antacid medication.

Conduct of anaesthesia

- Position the patient carefully intraoperatively due to ↑ risk of pressure sores and fractures 2° to fragile skin and osteoporosis.

Further reading

Nieman LK, Biller BMK, Findling JW, *et al*. (2015). Treatment of Cushing's syndrome: an Endocrine society clinical guideline. *J Clin Endocrinol Metab*, **100**, 2807–31.

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Conn's syndrome

Excess of aldosterone produced from an adenoma (60%) or benign hyperplasia of the adrenal gland (35–40%) or an adrenal carcinoma (rare).

General considerations

Aldosterone promotes active reabsorption of Na^+ and excretion of K^+ through the renal tubules. Water is retained with Na^+, resulting in an increase in ECF volume. To a lesser extent, there is also tubular secretion of H^+ ions and Mg^{2+}, resulting in a metabolic alkalosis.

Clinical features

- Refractory hypertension, hypervolaemia, metabolic alkalosis.
- Spontaneous hypokalaemia (K^+ <3.5mmol/L); moderately severe hypokalaemia (K^+ <3.0mmol/L) during diuretic therapy despite PO K^+.
- Muscle weakness, especially in ethnic Chinese, 2° to hypokalaemia.
- Nephrogenic diabetes insipidus 2° to renal tubular damage.
- Impaired glucose tolerance in ~50% of patients.

Preoperative assessment for adrenalectomy

- Spironolactone, a competitive aldosterone antagonist, is given to reverse the metabolic and electrolyte effects and restore normovolaemia. Doses of up to 400mg/d may be required.
- The patient should have normal serum K^+ and HCO_3^- if possible.
- Hypertension is usually mild and well controlled on spironolactone.
- Calcium channel blockers, such as nifedipine, are effective antihypertensive agents with aldosterone-secreting adenomas.

Investigations

- Aldosterone (pg/mL) to renin (nanograms/mL/h) ratio >400.
- 2° hyperaldosteronism: raised serum aldosterone, but normal ratio.
- Important to distinguish between adenoma and hyperplasia as adenoma is usually treated surgically and hyperplasia medically.
- Adrenal vein sampling, radiolabelling, CT and MRI are all used.

Conduct of anaesthesia for adrenalectomy

Unilateral adrenalectomy should ideally be performed laparoscopically. Handling of the adrenal gland during surgery can cause CVS instability but is not as severe as with a phaeochromocytoma (see ➲ pp. 716–19).

- A short-acting α-blocker should be available (phentolamine 1mg boluses IV).
- Check blood glucose perioperatively.
- Chronic hypokalaemia has an antagonistic action upon insulin secretion/release and may result in abnormal glucose tolerance with the stress of surgery.

Postoperative care

- K^+ supplements and spironolactone should be discontinued postoperatively.
- Give hydrocortisone IV postoperatively until the patient can tolerate oral hydrocortisone and fludrocortisone.
- Hypertension may persist after removal of the adenoma, presumably due to permanent changes in vascular resistance.

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Management of patients with Conn's syndrome for non-adrenal surgery

Such patients usually have bilateral hyperplasia of the zona glomerulosa. Hypertension is usually more severe and may require additional therapy (ACE inhibitors are useful). Try to restore K^+ to normal value preoperatively. Perform CVS assessment as for any hypertensive patient.

Further reading

Funder JW, Carey RM, Mantero F, *et al*. (2016). The management of primary aldosteronism: case detection, diagnosis and treatment: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*, **101**, 1889–916.

Apudomas

Tumours of amine precursor uptake and decarboxylation (APUD) cells which are present in the anterior pituitary gland, thyroid, adrenal medulla, GI tract, pancreatic islets, carotid bodies and lungs. Apudomas include phaeochromocytoma, carcinoid tumour, gastrinoma, VIPomas and insulinoma and may occur as part of the MEN syndrome. In 2017, the European Neuroendocrine Society released guidance on perioperative management of neuroendocrine tumours.[1]

Phaeochromocytoma

(See ➲ pp. 716–19.)

Carcinoid tumours

- Carcinoid tumours are derived from argentaffin cells and produce peptides and amines. They occur in the GI tract (75%), bronchus, pancreas and gonads. Mainly benign, but of those that are malignant, only about a quarter release vasoactive substances into the systemic circulation, leading to the carcinoid syndrome.
- Mediators are metabolised in the liver; carcinoid syndrome thus occurs if: (1) liver metastasis; (2) tumour drainage bypasses the portal circulation; and (3) precursors are so copious they overwhelm hepatic enzyme capacity, e.g. high lymph node metastasis burden.
- Niacin deficiency or pellagra can occur due to the consumption of niacin in serotonin production.
- Vasoactive substances include serotonin, bradykinin, histamine, substance P, prostaglandins and vasoactive intestinal peptide (VIP).
- Patients with an asymptomatic carcinoid tumour have simple carcinoid disease and do not present particular anaesthetic difficulties.
- Patients with carcinoid syndrome can be extremely difficult to manage perioperatively.
- Symptoms such as flushing (multiple episodes a day) are predictive of sequelae such as carcinoid heart disease.

Carcinoid syndrome

- Affects about 10% of patients with carcinoid tumours.
- Patients may have symptoms related to the tumour (e.g. intestinal obstruction or haemoptysis).
- Vasoactive peptides result in intermittent flushing, especially of the head, neck and torso (90%), or diarrhoea (78%), which may lead to dehydration and electrolyte disturbances.
- Other symptoms include bronchospasm (20%), hypotension, hypertension, tachycardia and hyperglycaemia.
- Twenty per cent of patients have heart disease related to carcinoid. Endocardial fibrosis of the pulmonary and tricuspid valves leads to right heart failure, so consider an echocardiogram preoperatively. ECG may show RV hypertrophy.

NT-proBNP has been shown to be predictive of both heart disease and mortality.

- Check FBC, electrolytes, LFTs and clotting if metastases present.
- X-match blood.

(See ➲ pp. 720–1 for conduct of anaesthesia.)

ALGRAWANY

Gastrinoma

- Excess production of gastrin by benign adenoma, malignancy or hyperplasia of D cells of the pancreatic islets.
- Gastrin stimulates acid production from gastric parietal cells. Leads to Zollinger–Ellison syndrome, severe peptic ulceration and diarrhoea.
- May also have GI bleeds, perforation, electrolyte disturbance and volume depletion.
- Treatment includes high-dose PPIs (e.g. omeprazole), H_2 receptor antagonists and octreotide.
- May present for surgery related to gastrinoma, e.g. perforation, or pancreatic resection of the tumour or a totally unrelated pathology. Actively look for anaemia from gastric ulceration and coagulopathy from liver dysfunction.
- Patients require perioperative invasive pressure monitoring for major surgery.
- Continue omeprazole postoperatively (up to 60–80mg/day), as the gastric mucosa may have become hypertrophied, producing excess acid.

VIPoma

- Rare tumour secreting VIP which leads to Verner–Morrison syndrome.
- Characterised by profuse watery diarrhoea, intestinal ileus, abdominal distension, confusion, hypokalaemia, achlorhydria, hypomagnesaemia, hyperglycaemia, metabolic alkalosis and tetany.
- VIP inhibits gastrin release; therefore, give H_2 receptor-blocking drugs preoperatively to prevent rebound gastric acid hypersecretion.
- Will often require a period of resuscitation with correction of fluid losses and electrolytes prior to surgery.
- Treat medically with somatostatin analogues (octreotide). If this fails, try steroids (such as methylprednisolone) and indometacin (a prostaglandin inhibitor).
- Sixty per cent malignant with liver metastases, so all warrant resection.
- Use invasive pressure monitoring for major surgery.
- ABG to check the acid–base status and electrolytes.

Insulinoma

- Rare tumour of β cells of the pancreas which secrete insulin.
- Whipple's triad of clinical presentation: fasting hypoglycaemia, symptoms of hypoglycaemia and relief of symptoms when glucose is given.
- Diagnosis also made by a fasting blood glucose <2.2mmol/L, ↑ insulin, ↑ C-peptide and no plasma sulfonylurea.
- Patients often have to eat high-carbohydrate diets and limit exercise to avoid hypoglycaemia; this predisposes to obesity and its sequelae.
- Medical treatment is used to reduce symptoms. Diazoxide (a non-diuretic benzothiazide which inhibits the release of insulin and stimulates glycogenolysis) has been used but has unpredictable efficacy and is avoided in hypoglycaemic presentations. Can cause significant fluid retention and oedema.
- Octreotide is also used. It binds with somatostatin receptors on insulinomas and decreases insulin secretion in 40–60% of patients; it can, however, worsen hypoglycaemia and should be monitored.

- Tumours rarely malignant, but if so, enucleation or pancreatic resection required.
- Start 10% glucose and K^+ infusion preoperatively, and monitor blood glucose closely perioperatively, particularly at the time of tumour manipulation.
- Almost immediate rise in glucose once tumour resected (IV glucose may be paused temporarily to observe for this).

Glucagonoma

- Tumour of α cells of the pancreas.
- Glucagon stimulates hepatic glycogenolysis and gluconeogenesis, resulting in ↑ blood glucose and DM. Ketoacidosis is rare, since insulin is also ↑.
- Characterised by a rash (necrotising migratory erythema which presents in the groin/perineum and migrates to the distal extremities).
- Often cachectic and may require antibiotics for skin infection.
- Associated with weight loss, glossitis, stomatitis, anaemia and diarrhoea.
- Patients usually have liver metastases at presentation.
- Treatment consists of surgical debulking and somatostatin analogues.
- ↑ incidence of venous thrombosis and PE, so give prophylactic antithrombotic therapy.

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ALGRAWANY

Hypokalaemia

Defined as plasma K^+ <3.5mmol/L.

• Mild	• 3.0–3.5mmol/L
• Moderate	• 2.5–3.0mmol/L
• Severe	• <2.5mmol/L

Causes

- ↓ intake.
- ↑ K^+ loss: vomiting or nasogastric (NG) suctioning, diarrhoea, pyloric stenosis, diuretics, renal tubular acidosis, hyperaldosteronism, Mg^{2+} depletion, leukaemia.
- Intercompartmental shift: insulin, alkalosis (pH increase of 0.1 decreases K^+ by 0.6mmol/L), β2-agonists and steroids.

Clinical manifestations

- ECG changes: T wave flattening and inversion, prominent U wave, ST-segment depression, prolonged P–R interval.
- Dysrhythmias, ↓ cardiac contractility.
- Skeletal muscle weakness, tetany, ileus, polyuria, impaired renal concentrating ability, ↓ insulin secretion, ↓ growth hormone secretion, ↓ aldosterone secretion, negative nitrogen balance.
- Encephalopathy in patients with liver disease.

Management

- Check U&E, creatinine, Ca^{2+}, phosphate, Mg^{2+}, HCO_3^- and glucose if other electrolyte disturbances suspected. Hypokalaemia resistant to treatment may be due to concurrent hypomagnesaemia.
- Exclude Cushing's and Conn's syndromes.
- Oral replacement is safest, up to 200mmol/d, e.g. KCl (Sando-K®) two tablets qds = 96mmol K^+.
- IV replacement: essential for patients with cardiac manifestations or skeletal muscle weakness or where oral replacement not appropriate.
- Aim to increase K^+ to 4.0mmol/L if treating cardiac manifestations.
- Maximum concentration for peripheral administration is 40mmol/L (greater concentrations than this can lead to venous necrosis); 40mmol KCl can be given in 100mL of 0.9% sodium chloride over 1h, but only via an infusion device, with ECG monitoring, in an HDU/ICU/theatre environment and via a central vein. Plasma K^+ should be measured at least hourly during rapid replacement. K^+ depletion sufficient to cause 0.3mmol/L drop in serum K^+ requires a loss of ~100mmol of K^+ from total body store.

Anaesthetic considerations

Predominant issue is risk of arrhythmia. Hypokalaemia is significantly associated with postoperative gastric transit delay, i.e. ileus, its own source of morbidity.[2]

- Classically, K^+ <3.0mmol/L has led to postponement of elective procedures (some controversy exists about this in the fit, non-digitalised patient who may well tolerate chronically lower K^+ levels, e.g. 2.5mmol/L, without adverse events).
- For emergency surgery, if possible, replace K^+ in the 24h prior to surgery. Aim for levels of 3.5–4.0mmol/L. If this is not possible, use an IV replacement regime, as documented earlier, intra-/perioperatively.
- If HCO_3^- is raised, then the loss is probably long-standing with low intracellular K^+ and will take days to replace.
- May increase sensitivity to NMB; therefore, need to monitor.
- ↑ risk of digoxin toxicity at low K^+ levels. Aim for K^+ of 4.0mmol/L in a digitalised patient.

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ALGRAWANY

Hyperkalaemia

Defined as plasma K^+ >5.5mmol/L.

• Mild	• 5.5–6.0mmol/L
• Moderate	• 6.1–7.0mmol/L
• Severe	• >7.0mmol/L

Causes

- ↑ intake, e.g. IV administration, rapid blood transfusion.
- ↑ urinary excretion, e.g. renal failure (acute or chronic), adrenocortical insufficiency, drugs (K^+-sparing diuretics, ACE inhibitors, ciclosporin, etc.).
- Intercompartmental shift of K^+, e.g. acidosis (H^+ is taken into the cell, in exchange for K^+), rhabdomyolysis, trauma, MH, suxamethonium (especially with burns or denervation injuries), familial periodic paralysis.
- Pseudohyperkalaemia due to *in vitro* haemolysis.
- Commonest risk factors are AKI, cardiovascular disease, CKD, diabetes and drugs affecting CVS/renal–aldosterone–angiotensin system.

Clinical manifestations

- ECG changes, progressing through peaked T waves, widened QRS, prolonged P–R interval, loss of P wave, loss of R wave amplitude, ST depression, VF, asystole. ECG changes potentiated by low Ca^{2+}, low Na^+ and acidosis.
- Muscle weakness at K^+ >8.0mmol/L.
- Nausea, vomiting, diarrhoea.

Management

- Treatment should be initiated if K^+ >6.5mmol/L or ECG changes present.
- Unlike hypokalaemia, the incidence of serious cardiac compromise is high and therefore, intervention is important. Treat the cause, if possible.
- Ensure IV access and cardiac monitor.
- Insulin 10 units in 50mL of 50% glucose IV over 30–60min. This has the fastest onset of action and is very effective in reducing serum K^+ by shifting K^+ into the cells. Beware rebound occurs within 2h.
- β2-agonist, salbutamol 5–10mg nebulised, but beware tachycardia. Should see a response at 30min and has a longer duration of action than insulin.
- 5–10mL of 10% calcium gluconate or 3–5mL of 10% calcium chloride. Ca^{2+} stabilises the myocardium by increasing the threshold potential. Rapid onset, short-lived.
- If acidotic, give HCO_3^- 50mmol IV.
- Ion exchange resin: calcium polystyrene sulfonate (e.g. Resonium calcium) 5g PO or 30g per rectum (PR) 8-hourly. This binds K^+ in the gut.
- If initial management fails, consider dialysis or haemofiltration.

Anaesthetic considerations

- Do not consider elective surgery. If life-threatening surgery, treat hyperkalaemia first.
- Avoid Hartmann's solution and suxamethonium; rocuronium is the preferred choice, although suxamethonium has been used safely with K^+ of 5.5.[3] Monitor NMB, since effects may be accentuated.
- Avoid hypothermia and acidosis.
- Control ventilation to prevent respiratory acidosis.
- Monitor K^+ regularly.

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ALGRAWANY

Hyponatraemia

Defined as serum Na^+ <135mmol/L.

• Mild	125–134mmol/L
• Moderate	120–124mmol/L
• Severe	<120mmol/L

ECF volume is directly proportional to total body Na^+ content. Renal Na^+ excretion ultimately controls the ECF volume and total body Na^+ content. To identify the causes of abnormalities of Na^+, assess plasma and urinary Na^+ levels and the patient's state of hydration. Causes are summarised in Fig. 9.1. Presentation depends on speed of onset, rather than on absolute Na^+ level—it is rare to get clinical signs if Na^+ >125mmol/L.

Signs

Neuropsychiatric symptoms, nausea/vomiting, muscular weakness, headache, lethargy, seizures, coma and respiratory depression.

Treatment of acute symptomatic hyponatraemia (<48h)

- Aim to raise serum Na^+ by 2mmol/L/h until symptoms resolve. Complete correction is unnecessary. May need to infuse hypertonic (3%) sodium chloride at a rate of 1.2–2.4mL/kg/h through a large vein (can be ↑ to 4–6mL/kg/h if seizures/coma). Care needed—measure Na^+ levels hourly.
- In cases of fluid excess, give furosemide 20mg IV.

Treatment of chronic hyponatraemia

- Asymptomatic, fluid-restrict to 1L/d.
- Symptomatic (>48h): aim to correct serum Na^+ by 5–10mmol/d. Rapid correction (serum Na^+ rise of >0.5mmol/L/h) can lead to central pontine myelinolysis, subdural haemorrhage and cardiac failure.
- Hypovolaemic: correct with 0.9% sodium chloride (removes ADH response that accentuates Na^+/water imbalance).
- Hypervolaemic: fluid-restrict and give furosemide. For syndrome of inappropriate antidiuretic hormone (secretion) (SIADH), also give demeclocycline 300–600mg/d.
- Consult with an endocrinologist and treat the underlying cause.

Anaesthetic implications

- No elective surgery if Na^+ <120mmol/L or symptomatic hyponatraemia. If surgery urgent, consult endocrinologist and assess risk/benefit ratio.

Further reading

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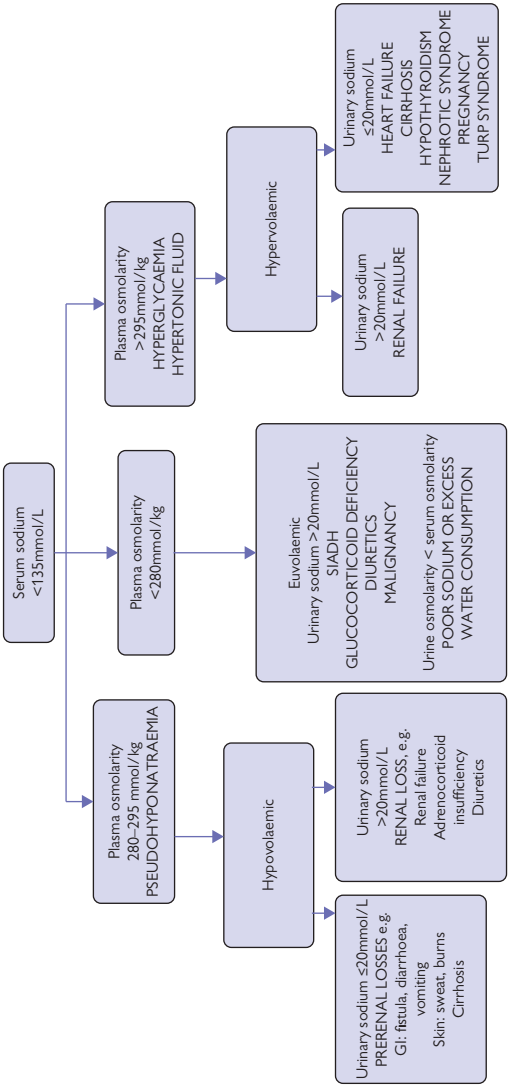

Fig. 9.1 Diagnosis of hyponatraemia.

ALGRAWANY

Hypernatraemia

Defined as serum Na^+ >145mmol/L.

• Mild	145–150mmol/L
• Moderate	151–160mmol/L
• Severe	>160mmol/L

Caused by excessive salt intake or, more frequently, inadequate water intake. Important to assess the volume status.

Causes

Hypovolaemic

Renal (loop/osmotic diuretics, intrinsic renal disease, post-obstruction) and extrarenal (diarrhoea/vomiting, burns, excessive sweating, fistulae).

Euvolaemic

Diabetes insipidus, insensible losses.

Hypervolaemic

Na^+ ingestion/administration of hypertonic sodium chloride, Conn's syndrome, Cushing's syndrome.

Presentation

- CNS symptoms likely if serum Na^+ >155mmol/L due to hyperosmolar state and cellular dehydration, e.g. thirst, confusion, seizures and coma.
- Features depend on the cause, e.g. water deficiency will present with hypotension, tachycardia and ↓ skin turgor.

Management

- Correct over at least 48h to prevent occurrence of cerebral oedema and convulsions.
- Treat the underlying cause. Give oral fluids (water), if possible.
- Hypovolaemic (Na^+ deficiency): 0.9% sodium chloride until hypovolaemia corrected, then consider 0.45% sodium chloride.
- Euvolaemia (water depletion): estimate the total body water (TBW) deficit; treat with 5% glucose.
- Hypervolaemic (Na^+ excess): diuretics, e.g. furosemide 20mg IV and 5% glucose; dialysis if required.
- Diabetes insipidus: replace urinary losses, and give desmopressin (1–4 micrograms daily SC/IM/IV).

Anaesthetic implications

- No elective surgery if Na^+ >155mmol/L or hypovolaemic.
- Urgent surgery: consider CVP monitoring and be aware of dangers of rapid normalisation of electrolytes.

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Chapter 10

Bone, joint and connective tissue disorders

David Howell

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ALGRAWANY

Rheumatoid arthritis

RA is a chronic, systemic inflammatory disorder, mainly involving synovial joints, but with extra-articular effects. Overall prevalence in the UK is 1.5 per 100 000 in ♂, and 3.6 per 100 000 in ♀, with a peak incidence in the 7th decade. It also affects children as Still's disease. There is a higher-than-average mortality due to both the disease itself and the presence of concurrent disorders.

Preoperative assessment

Articular

- **Temporomandibular**: assess for limited mouth opening.
- **Cricoarytenoid**: fixation of the cricoarytenoid joints may lead to voice changes or even rarely to stridor from glottic stenosis. The larynx can also be obstructed by amyloid or rheumatoid nodules. Minimal oedema may lead to airway obstruction postoperatively.
- **Atlantoaxial subluxation** occurs in ~25% of severe rheumatoid patients, but of these, only a quarter will have neurological signs or symptoms. Risk factors include disease duration >8y, glucocorticoid use, seropositivity and nodular and erosive peripheral joint disease. Enquire about tingling hands or neck pain, and assess the range of neck movement (Table 10.1).
- **Subaxial subluxation** (i.e. below C2): >2mm loss of alignment is significant. Look for this particularly if the patient has undergone previous fusion at a higher level.
- **Other joints**: assess joint deformities with a view to positioning and possible regional anaesthesia. Manual dexterity may be important if planning to use standard PCA apparatus after surgery.

Non-articular

- **CVS**: ↑ risk of CAD. Systemic vasculitis may lead to arterial occlusion in various organs and Raynaud's. They may have myocardial fibrosis and amyloid or nodular involvement. Pericarditis and pericardial effusions uncommon. Aortic incompetence and endocarditis rare. At risk of VTE.
- **Respiratory**: interstitial lung disease leading to restrictive defects may also be 2° to drugs (fibrosing alveolitis, pneumonitis). Pulmonary nodules. Pleural disease (pleuritis and effusions) and airway involvement (obliterative bronchiolitis). Costochondral disease may reduce chest wall compliance.
- **Haematological**: normocytic, normochromic anaemia of chronic disease. Drug-associated myelosuppression and NSAID-associated blood loss. Felty's syndrome is a combination of splenomegaly and neutropenia and may be associated with anaemia and thrombocytopenia.
- **Nervous system**: central (due to axial involvement) and peripheral compression neuropathies (e.g. carpal tunnel syndrome).
- Sjögren's syndrome and resultant dry eyes put patients at risk of corneal ulceration under anaesthesia.
- **Infections**: common from the disease itself and drug effects.
- **Renal and hepatic**: CKD commonly from drugs, less frequently from glomerulonephritis. ↓ albumin, ↑ fibrinogen and α-1 acid glycoprotein (acute phase protein).
- Fragile skin and difficult venous access.

Table 10.1 Atlantoaxial subluxation

Type	Incidence	Pathology	Imaging
Anterior	80%	Destruction of transverse ligament C1 forward on C2 Exacerbated by neck flexion	>3mm between odontoid and arch of atlas in lateral flexion
Posterior	5%	Destruction of odontoid peg C1 backward on C2 Exacerbated by neck extension	Loss of odontoid peg on lateral neck extension
Vertical	10–20%	Odontoid moves upward through foramen magnum to compress the cervicomedullary junction Destruction of lateral masses of C1	>4.5mm movement of odontoid above McGregor line (lateral view)
Lateral	Uncommon	Destruction of C1/C2 facet joints Spinal nerve + vertebral artery compression Lateral/rotation of C1 on C2	>2mm difference in lateral alignment on odontoid view

Investigations

- Rheumatologist or spinal surgeon involvement is recommended in patients with neurological symptoms or signs and in those with persistent neck pain. Preoperative cervical spine flexion/extension views are controversial. Stabilisation surgery may be necessary before elective surgery is undertaken. Specialist radiological advice should be sought.
- PFTs should be carried out for patients with suspected respiratory involvement.
- Nasendoscopy preoperatively if suggestion of cricoarytenoid arthritis.
- Echocardiography is needed if there is valvular or pericardial involvement and in symptomatic cardiac disease.

Drugs in the perioperative period

- Steroid use should be minimised prior to surgery. Steroid supplementation if indicated (see pp. 230–1).
- NSAIDs: continue to enable early mobilisation. Stop if postoperative bleeding is a potential problem, hypotension or deterioration in renal function.
- DMARDs: these drugs include gold, penicillamine and immunosuppressant drugs such as methotrexate, azathioprine, cyclophosphamide, ciclosporin, leflunomide and sulfasalazine. Continue as little evidence that omission reduces postoperative complications (wound infections). If leucopenic or for high-risk procedures, consult with the rheumatologist.

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- Biological DMARDs include tumour necrosis factor (TNF) inhibitors (etanercept, infliximab, adalimumab, certolizumab), IL (interleukin)-1 inhibitors (anakinra), IL-6 inhibitors (tocilizumab) and B-cell inhibitors (rituximab). The potential ↑ risk of postoperative infection needs to be balanced against the risk of perioperative flare. Consider ceasing at least one dosing interval prior to surgery, or longer for high-risk procedures. Specifically, rituximab and tocilizumab should ideally be stopped 3–6mo and 4w, respectively, prior to elective surgery. Recommence around 14d postoperatively if uncomplicated postoperative course.

Operative considerations

- Consider regional anaesthesia (may be technically difficult), if possible, to minimise the need for airway instrumentation.
- Unless it is certain that the cervical spine is stable, all rheumatoid patients should be treated as if they have an unstable spine, given that the majority of patients with atlantoaxial subluxation are asymptomatic. Restriction of cervical spine motion (ROCSM) is necessary during airway manipulation while the patient is unconscious. If intubation is necessary, consider videolaryngoscopy or fibreoptic intubation if difficulties are anticipated, and a smaller ETT in the setting of cricoarytenoid disease.
- Blood conservation strategies in anaemic patients.
- Ensure careful positioning and padding/protection of vulnerable areas. Note comfortable position before induction, then try to maintain this during surgery.
- Avoid hypothermia which may increase the risk of wound infection.

Postoperative

- Monitor for post-extubation oedema from cricoarytenitis.
- Adequate pain control and physiotherapy for early mobilisation.
- DVT prophylaxis (see ➲ pp. 59–61) until the patient is fully mobile.
- Maintain fluid intake, and monitor renal function.

Ankylosing spondylitis

Inflammatory arthritis of the sacroiliac joints and spine, leading to ankylosis and 'bamboo spine'. Associated with HLA-B27 in >90% of cases. More common in ♂, with peak age onset in the 3rd decade.

Articular

- Progressive kyphosis and fixation of the spine may hinder intubation. Conventional intubation and tracheostomy may be impossible. Atlantoaxial subluxation and myelopathy can occur rarely. There may be limited mouth opening from temporomandibular involvement. Use of intubating laryngeal mask airway (LMA) or videolaryngoscopy described, but fibreoptic intubation usually preferred.
- At risk of occult cervical fracture with minimal trauma, so ensure the head is supported and not left self-supporting.
- Cricoarytenoid arthritis may make cords susceptible to trauma and oedema.
- Spinal anaesthesia is often difficult; consider using a paramedian approach. Possible ↑ risk of epidural haematoma with epidural block.
- Restrictive pulmonary disease from diminished chest expansion and spinal mobility. Effective external cardiac massage may be impossible.
- Deformity leads to difficulty with positioning, particularly if a prone position is required.

Non-articular

- Apical fibrosis, which contributes to the restrictive lung defect.
- AR (1%). Mitral valve involvement and conduction defects are rare.
- Amyloid may cause renal involvement.
- Cauda equina syndrome may occur in long-standing cases.
- Side effects of NSAIDs and biological and non-biological DMARDs (see pp. 247–8 and pp. 1155–6).

ALGRAWANY

Systemic lupus erythematosus

This is a chronic multisystem autoimmune disease, commonest in young ♀, especially in pregnancy. It is characterised by the presence of numerous antibodies, including antinuclear antibody, and immune-mediated tissue damage.

Preoperative assessment

- Skin and joint involvement is common, as are oral and pharyngeal ulcerations.
- CVS: pericarditis in 15% of cases. Myocarditis, endocarditis and conduction abnormalities are less common. Raynaud's phenomenon 30%. CAD.
- Respiratory: infections and PEs common. Pleuritis and pleural effusion. Pulmonary fibrosis less common.
- Neurological: cranial and peripheral nerve lesions may occur 2° to arteritis and ischaemia. Transverse myelitis, leading to weakness or paraplegia, is rare. Depression, psychosis and seizures are possible.
- Renal: glomerulonephritis, which may lead to nephrotic syndrome and renal failure.
- Haematological: cytopenias, clotting disorders or hypercoagulable states can occur. Immune thrombocytopenia or circulating anticoagulants (e.g. antibodies to factor VIII) may be present. Up to a third of patients with SLE may demonstrate features of antiphospholipid syndrome (see ➲ p. 289).
- Higher risk of CVE with antiphospholipid antibodies.
- Steroids and other immunosuppressant drugs are used.

Anaesthesia

- There may be absolute or relative contraindications to neuraxial blocks in patients taking anticoagulants or in patients with coagulopathy (see ➲ p. 1110). The presence of a peripheral nerve lesion may be a relative contraindication to neuraxial/regional nerve blockade.
- Maintenance of normothermia may reduce the risk of infection, as well as lessening the impact of Raynaud's phenomenon, if present.
- Laryngeal erythema and oedema are common—try to minimise trauma to the airway.
- Consider hourly urine output and invasive monitoring.
- Steroid supplementation (see ➲ pp. 230–1).
- Strict asepsis with invasive procedures, as ↑ risk of infection.
- Perioperative VTE prophylaxis.

Systemic sclerosis (scleroderma)

A progressive multisystem disease characterised by widespread vascular dysfunction and fibrosis of the skin and viscera.

The limited cutaneous form, comprising calcinosis, Raynaud's, oesophageal dysfunction, sclerodactyly and telangiectasia (CREST), is more common (60% of cases) than the more aggressive diffuse cutaneous form, which has more widespread effects and a high mortality.

The following may be relevant to anaesthesia:

- CVS: Raynaud's, pericarditis or myocardial fibrosis. Conduction defects. Pulmonary hypertension is common.
- Pulmonary: fibrosing alveolitis in both forms (40% in diffuse form).
- Renal: may develop renal crisis associated with malignant hypertension.
- GI: oesophageal reflux invariable.
- Airway: limited mouth opening and tightened skin around the face/neck, leading to difficult mask ventilation and intubation. Careful airway instrumentation in setting of telangiectasia.
- Dermal thickening and contractures: difficulty with IV access and positioning.
- Immunosuppressant medication.

Scoliosis

Progressive lateral curvature of the spine with added rotation. Scoliosis may lead to an increasing restrictive ventilatory defect which, in turn, leads to hypoxia, hypercapnia and pulmonary hypertension. Scoliosis may be idiopathic (~75%) or 2° to other conditions with anaesthetic implications:

- Muscular dystrophies
- Poliomyelitis
- Cerebral palsy
- Friedreich's ataxia
- Marfan syndrome and Ehlers–Danlos syndrome.

Conduct of anaesthesia

- Formal PFTs. Cobb angle exceeding 65° is likely to be associated with compromised respiratory function.
- Check for pulmonary hypertension and right heart failure.
- Some muscular dystrophies may be associated with cardiac abnormalities. Consider echocardiography (see p. 318).
- May be difficult laryngoscopy.
- Neuraxial anaesthesia more difficult.
- Intraoperative spinal cord monitoring is recommended for all spinal deformity corrective surgery (see pp. 629–33).
- Invasive arterial monitoring and potential significant blood loss.
- Multimodal analgesia, including regional techniques (e.g. paravertebral blocks) where possible.
- Plan for high dependency or intensive care in complex cases.

ALGRAWANY

Achondroplasia

The commonest form of dwarfism is caused by premature ossification of bones, combined with normal periosteal bone formation, giving a characteristic appearance of short limbs and a relatively normal cranium. The following should be noted:

- Large head and tongue, mid-facial hypoplasia, obesity and small larynx may lead to difficulties managing the airway. Laryngoscopy may additionally be compromised by pectus carinatum.
- Foramen magnum stenosis is common. Avoid hyperextension during intubation.
- Central and peripheral venous access is often difficult.
- Use an appropriately sized BP cuff.
- OSA is common (see pp. 73–5).
- Restrictive ventilatory defects may occur and can lead to pulmonary hypertension.
- Regional techniques may be difficult.
- The back may be normal. The epidural space is often narrowed with spinal canal stenosis. The volume of LA needed for a subarachnoid and epidural block is reduced. Epidural over spinal anaesthesia may be preferred due to ability to titrate to desired height.
- The patient is of normal intelligence.

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Chapter 11

Haematology

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Anaemia

Anaemia is defined as Hb below normal for age and sex. Conventionally, this is <130g/dL in an adult ♂ and <120g/dL in an adult ♀. Common causes of anaemia in the surgical patient are:

- Blood loss: acute or chronic (usually resulting in iron deficiency)
- Bone marrow failure: infiltration by tumour or suppression by drugs
- Megaloblastic anaemias: folate or vitamin B_{12} deficiency
- Complex anaemias: effects on production and breakdown, e.g. renal failure, RA and hypothyroidism
- Haemolytic anaemias: either inherited (thalassaemia, sickle-cell disease (SCD), spherocytosis) or acquired (autoimmune, drugs, infections), or physical (mechanical heart valves, disseminated intravascular coagulation (DIC), prolonged marching known as 'march haemoglobinuria').
- Chronic disease or inflammation leads to ↑ hepcidin release from the liver which inhibits iron absorption from the gut despite normal to high ferritin. Unlikely to respond to oral iron therapy.

Clinical

- Associated with fatigue, dyspnoea, palpitations, headaches and angina. Severity often reflects the speed of onset more than the degree of anaemia, as there is less time for adaptation.
- Common causes should be elicited, including relevant family history; always enquire about NSAIDs and alcohol.
- Respiratory and cardiovascular pathology may be worsened by the anaemia or make its impact greater.

Investigations

- Measure Hb prior to surgery in appropriate patients (see ➲ p. 28), including all those at risk of anaemia undergoing major surgery and patients with significant comorbidities, especially heart or lung disease.
- Much can be deduced from Hb and mean corpuscular volume (MCV) alone but in many instances a blood film gives additional useful information.
- Confirmatory tests, such as ferritin, cobalamin (B_{12}) and folate levels, reticulocyte count, direct Coombs' test, erythrocyte sedimentation rate (ESR), liver and renal function and bone marrow cytology, should be requested, as appropriate.

Preparation

Patients scheduled for elective surgery should have FBC checked in the weeks approaching the operation so that abnormalities can be investigated and corrected in time (Fig. 11.1).

- Where surgery can be safely postponed, it is more appropriate and safer to treat the underlying cause and raise the Hb slowly with simple, effective measures, e.g. PO or IV iron and B_{12} injections. Transfusing a patient with pernicious anaemia may precipitate heart failure.

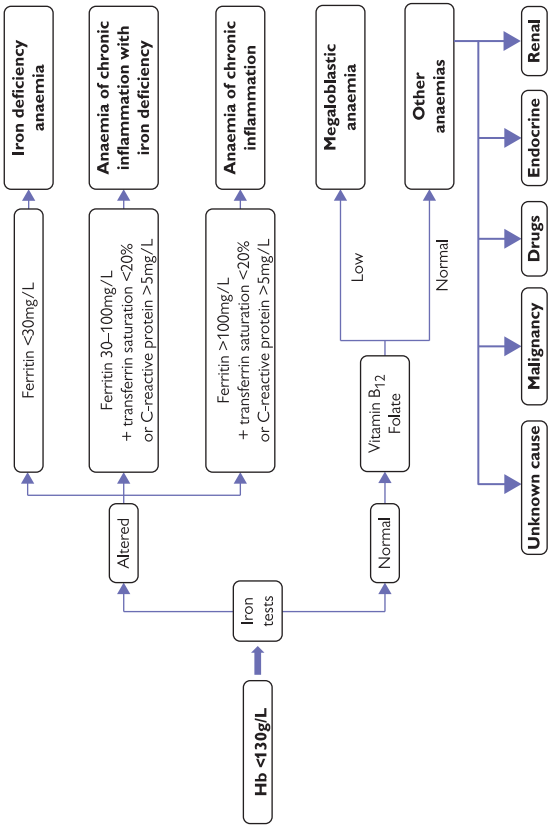

Fig. 11.1 Algorithm for classification of perioperative anaemia. Reproduced from Klein AA, *et al.* (2016). International consensus statement on the peri-operative management of anaemia and iron deficiency. *Anaesthesia*, **72**, 233–47, with permission from John Wiley & Sons Ltd. © 2016 The Authors.

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IV iron preparations are now available that can be given over a few minutes and will render a patient immediately iron-replete; typical Hb increase 10–20mg/L after 10d (see also ➲ pp. 54–6).

- Many IV preparations exist. A common preparation is iron isomaltoside (Monofer®); a single dose of 20mg/kg (typically 1000mg) will render a patient iron-replete with a 15min infusion and with minimal side effects. Check your local protocol or discuss with a haematologist.

Perioperative blood transfusion

(See also ➲ pp. 450–9.)

Recent RCTs have confirmed that transfusion is not required for mild anaemia, even in the presence of CVS disease. In some of these trials, the use of a lower 'transfusion trigger' has been associated with lower mortality. Coexisting disease, chronicity of anaemia and the expected perioperative blood loss all inform the decision of whether to transfuse or not. The following are accepted levels for transfusion:

- Red cell transfusion is indicated if the Hb level is <70g/L.
- Checking a HemoCue® gives comparable results to a Coulter counter and can help to avoid a transfusion if >80g/L.
- For patients with IHD:
  - Mild (angina rarely): accept Hb 70–80g/L
  - Moderate (angina regularly, but stable): accept Hb 80–90g/L
  - Severe (recent MI, unstable angina): accept Hb 100g/dL or higher.

Sickle-cell disease

Sickle-cell disease (SCD) is caused by inheriting sickling haemoglobinopathies, either in the homozygous (HbSS; sickle-cell anaemia) or heterozygous (HbAS; sickle-cell trait) state, or in combination with another Hb β chain abnormality such as Hb C (HbSC disease), Hb D (HbSD disease) or β-thalassaemia (HbS/β-thal). It is estimated that there are now over 15 000 patients with SCD in Britain. SCD is endemic in parts of Africa, the Mediterranean, the Middle East and India. The highest incidence is from equatorial Africa; all patients from areas with a high prevalence should have a sickle test preoperatively.

The pathology of SCD is primarily a result of vaso-occlusion by sickled red cells, leading to haemolysis and tissue infarction. This can be precipitated by hypoxia, hypothermia, pyrexia, acidosis, dehydration or infection. Other variant haemoglobins Hb C and Hb D-Punjab, in association with HbS, enhance the sickling process, whereas HbF (fetal Hb) impedes it.

- Susceptibility to sickling is proportional to the concentration of HbS. In the heterozygous state (sickle-cell trait), sickling is extremely uncommon as HbS concentration is <50%.
- These patients have a positive sickle solubility test (Sickledex®), but normal blood film and Hb level. This can be confirmed by Hb electrophoresis or, in an emergency, a normal blood film should suffice.
- These patients do not need special treatment, other than avoidance of extreme hypoxia, dehydration, infection, acidosis and hypothermia.

Clinical features

The manifestations of SCD do not become apparent before 3–4mo of age, when the main switch from fetal to adult Hb occurs.

- There is great variability, not only between patients, but also within individual patients at different periods of life. Many remain well most of the time.
- Vaso-occlusive crises are the commonest cause of morbidity and mortality. The presentation may be dramatic with an acute abdomen, 'acute chest syndrome' (acute pneumonia-like), CVE, priapism and painful dactylitis. By the time patients reach adulthood, most will have small, fibrotic spleens and are functionally asplenic, with the associated risk of overwhelming septicaemia. A less acute complication is proliferative retinopathy due to retinal vessel occlusion and neovascularisation (more common in HbSC disease).
- Aplastic crises are characterised by temporary shutdown of the marrow, manifested by a precipitous fall in Hb and an absence of reticulocytes. Infection with parvovirus B19 and/or folate deficiency are the two precipitating factors.
- Sequestration crises occur mainly in children. Sudden massive pooling of red cells in the spleen can cause hypotension and severe exacerbation of anaemia, with fatal consequences, unless transfusion is given in time.
- Haemolytic crises manifest by a fall in Hb and a rise in reticulocytes/bilirubin, and usually accompany vaso-occlusive crises. Chronic haemolysis leads to gallstones in virtually all patients with SCD, though many remain asymptomatic.

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Laboratory features

- Hb is usually 6–9g/dL (often lower than suggested by the clinical picture). Reticulocytes are almost always ↑, and the film shows sickled cells and target cells. Howell–Jolly bodies are present if the spleen is atrophic. Leucocytosis and thrombocytosis are common reactive features. In the sickle-cell trait, the Hb and film are normal.
- Screening tests for sickling which rely on deoxygenation of HbS are positive in both HbSS and HbAS.
- Hb electrophoresis distinguishes SS, AS and other haemoglobinopathies. Measurement of the HbS level is important in certain clinical situations where a level of <30% is aimed for. It is not necessary to wait for the results of electrophoresis before embarking on emergency surgery. Clinical history, Hb level, a positive sickle test and the blood picture usually allow distinction between SCD and the sickle-cell trait.

Management

As no effective routine treatment exists for SCD, care is directed towards prophylaxis, support and treatment of complications. Folic acid supplements, pneumococcal and *Haemophilus influenzae* type b (Hib) vaccinations and penicillin prophylaxis (to protect from the susceptibility to infection caused by ↓ splenic function) are recommended from an early age, preferably within a comprehensive care programme.

- For crises: rest, rehydration with PO/IV fluids, antibiotics if infection is suspected; maintain PaO_2; keep warm; prompt and effective analgesia (traditionally diamorphine/morphine is used over pethidine; regional anaesthesia very effective).
- Blood transfusions may be lifesaving, but the indications are limited.[1] Exchange transfusions have a role in some vaso-occlusive crises (acute chest syndrome, CVE). Always discuss with a haematologist. For patients with high perioperative risk, transfusing to achieve an HbS level of <30% may decrease complications but is controversial.

Preoperative preparation

Always seek expert advice from a haematologist well before surgery. A sample for group and antibody screening should be sent well in advance, as previously transfused sickle-cell patients often have red cell antibodies.

Perioperative and postoperative care

Special attention must be given to hypoxia, dehydration, infection, acidosis, hypothermia and pain. These considerations should be continued well into the postoperative period.

- Dehydration: allow oral fluids as late as possible, and pre- and postoperative IV fluids.
- Hypoxia: pulse SpO_2 and prophylactic O_2.
- Prophylactic antibiotic cover should always be considered because of ↑ susceptibility to infection.
- Positive pressure ventilation may be required to achieve normocapnia and avoid acidosis.
- Hypothermia should be avoided by warming the operating room, using a fluid warmer and active warming such as a Bair Hugger®. Core temperature should be monitored.
- Regional anaesthesia is not contraindicated, and tourniquets can be used if limbs are meticulously exsanguinated prior to inflation.

HbSC disease

- Results from compound heterozygosity for HbS and HbC.
- Affects 0.1% of African Americans.
- Causes SCD, but phenotype may be milder than homozygous HbSS.
- Patients develop anaemia, splenomegaly, jaundice, aseptic necrosis of the femoral head, hepatic disease, retinal disease and bone marrow and splenic infarcts.
- Myocardial necrosis has been described after GA.
- Management principles are as for SCD.
- Patients with HbSC disease are more prone to retinal disease and often maintain splenic function into adult life.

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Porphyria

The porphyrias are a group of diseases in which there is an enzyme defect in the synthesis of the haem moiety, leading to an accumulation of precursors that are oxidised into porphyrins. There are hepatic and erythropoietic varieties. There are eight different forms of porphyria. There are three autosomal dominant acute hepatic forms, with typically 50% reduction in usual enzyme activity, that affect the administration of anaesthesia.

Acute intermittent porphyria

The commonest and most severe form of acute porphyria. Common in Sweden. ↑ urinary porphobilinogen and d-aminolaevulinic acid.

Variegate porphyria

Common in Afrikaners. ↑ Copro- and protoporphyrin in the stool. Dermal photosensitivity.

Hereditary coproporphyria

Very rare. ↑ urinary porphyrins. Dermal photosensitivity.

Porphyric crises

- Attacks occur most frequently in women in the 3rd to 4th decades.
- Fifty per cent of normal enzyme activity is usually enough to maintain haemostasis. ↑ demand for haem increases flow through pathway and increases aminolaevulinic acid and porphyrin production, leading to symptoms. Factors increasing haem demand, including haemorrhage, induction of P450 enzymes, stress and dehydration, should be avoided.
- Acute porphyric crises may be precipitated by drugs, stress, infection, alcohol, menstruation, pregnancy, starvation and dehydration.
- Symptoms include acute abdominal pain, vomiting, motor and sensory peripheral neuropathy, autonomic dysfunction, cranial nerve palsies, mental disturbances, coma, convulsions and pyrexia.

General principles

- Patients may never have had an attack; therefore, a positive family history must be taken seriously.
- Individuals may have normal biochemical tests between attacks.
- Patients may present with unrelated pathology, e.g. appendicitis.
- Symptoms may mimic surgical pathologies, e.g. acute abdominal pain, acute neurology.
- Any patient giving a strong family history of porphyria must be treated as potentially at risk. Latent carriers may exhibit no signs and be potentially negative to biochemical screening, but still be at risk from acute attacks.

Anaesthetic management

Many commonly used drugs are thought to have the potential to trigger porphyric crises. However, it is difficult to be definitive, as crises can also be triggered by infection or stress, which often occur simultaneously. Table 11.1 lists the relative safety of commonly used anaesthetic drugs in patients with porphyria. The website www.drugs-porphyria.org contains more complete and up-to-date prescribing information.

Table 11.1 Anaesthetic drugs in porphyria

	Not porphyrinogenic	Probably safe	Probably or possibly porphyrinogenic	Porphyrinogenic
Anaesthetic agents	Propofol	Sevoflurane Isoflurane Desflurane N_2O	Ketamine Etomidate	Thiopental Halothane
Antiemetics and antacids	Cimetidine Omeprazole	Ondansetron Cyclizine Metoclopramide Ranitidine	Dexamethasone	
Neuromuscular-blocking drugs	Suxamethonium Mivacurium Rocuronium	Atracurium Vecuronium Pancuronium		
Reversal agents	Intralipid®	Neostigmine Sugammadex Naloxone Glycopyrronium bromide	Flumazenil	
Analgesics	Morphine Pethidine Clonidine Aspirin	Fentanyl Alfentanil Ibuprofen Paracetamol		
Local anaesthetics	Bupivacaine Prilocaine	Lidocaine Levobupivacaine		
Sedatives		Diazepam Midazolam Lorazepam	Dexmedetomidine	
Cardiovascular	Adrenaline Noradrenaline Adenosine GTN	Ephedrine Phenylephrine Atropine	Amiodarone	Methyldopa Hydralazine
Antibiotics	Benzylpenicillin Teicoplanin	Ceftriaxone Cefalexin Metronidazole	Clarithromycin	
Other	Dalteparin Enoxaparin Salbutamol	Furosemide Haloperidol Olanzapine Levetiracetam	Hydrocortisone Prednisone	

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- It is important to minimise stress, so consider premedication with midazolam.
- Minimise preoperative fasting. Use 5% glucose/0.9% sodium chloride IV.
- Regional anaesthesia is useful, but in acute porphyric crises, regional anaesthesia should be avoided as neuropathy may be rapid in onset and progressive. Detailed preoperative examination and documentation of any neuropathy are essential. Bupivacaine is considered safe for epidural anaesthesia.
- PONV prophylaxis is especially recommended, as prolonged starvation time can be contributory to porphyric crises.
- Measure BP invasively during an acute crisis, as hypovolaemia is common and autonomic neuropathy may cause labile BP.
- Once common causes (e.g. pain, light anaesthesia) are ruled out, hypertension and tachycardia should be treated promptly with β-blockers such as metoprolol.
- Treat seizures with diazepam, propofol or magnesium sulfate (avoid barbiturates and phenytoin).
- Manage the patient on ICU/HDU if a crisis is suspected.
- Remember that the onset of a porphyric crisis may be delayed for up to 5d.

Treatment of acute porphyric crises

- Withdraw drugs that may have precipitated the crisis.
- Give haem arginate 3mg/kg IV once daily for 4d (leads to negative feedback to aminolaevulinic acid synthetase—the initial enzyme responsible for haem production). Treat infection, dehydration and electrolyte imbalance, and give glucose (20g/h).
- Treat symptoms with 'safe' drugs.
- Monitor the patient appropriately.

Rare blood disorders

Hereditary spherocytosis

Autosomal dominant condition in which erythrocytes have a smaller surface to volume ratio and are abnormally permeable to Na^+.

- Inflexible red cells are phagocytosed in the spleen, resulting in microspherocytic anaemia with marked reticulocytosis. The blood film is usually diagnostic.
- Splenomegaly is common. Splenectomy leads to a 50–70% increase in red cell survival.
- Splenectomy should not be performed in children <6y of age and should ideally be preceded by pneumococcal, meningococcal and Hib vaccines and lifelong PO penicillin, to help avoid infection.
- There are no particular anaesthetic considerations.

Glucose-6-phosphate dehydrogenase deficiency

X-linked trait with variable penetrance in African Americans and people from the Mediterranean.

- The disease may afford some protection against malaria.
- The glucose-6-phosphate dehydrogenase (G6PD) enzyme is responsible for the production of NADPH, which is involved in the cell's defence against oxidative stresses such as oxidative drugs and infections (usually viral, but also septicaemia, malaria and pneumonia).
- Drugs producing methaemoglobinaemia, such as nitroprusside and prilocaine, are contraindicated, as patients are unable to reduce methaemoglobin, thereby diminishing the O_2-carrying capacity.
- Ingestion of broad (fava) beans results in haemolysis (favism).
- Usually haemolysis of red cells occurs 2–5d after exposure, causing anaemia, haemoglobinaemia, abdominal pain, haemoglobinuria and jaundice.
- Demonstration of Heinz bodies and red cell G6PD assay are diagnostic, but G6PD levels may be falsely raised or normal in acute haemolysis.
- Signs and symptoms include: headache, breathlessness, nausea, tachycardia, fatigue and cyanosis.
- Treatment includes discontinuation of the offending agent, and transfusion may be required.
- Anaesthetic management should focus on avoiding drugs that cause oxidative stress or can induce methaemoglobinaemia (Table 11.2).[2] There are circumstances where clinical necessity may outweigh the potential risk of drug administration, but this should be considered with expert advice and careful observation for the signs and symptoms of methaemoglobinaemia.
- Pain-induced stress can cause haemolysis, so ensure good pain relief.

Thalassaemias

Thalassaemias are due to absent or deficient synthesis of α- or β-globin chains of Hb. The severity of these disorders is related to the degree of impaired globin synthesis.

- The hallmark of β-thalassaemia is anaemia presenting in the 1st year of life.
- α-thalassaemia is more commonly associated with hydrops fetalis.

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- Diagnosis is confirmed by Hb electrophoresis and/or globin chain analysis.
- The disease is prevalent in people of Mediterranean (mainly β), African (β and non-deletional α), and Asian (deletional α) ethnicity.
- Regardless of the underlying chain involved, thalassaemia is classified as major, intermediate, or minor.
- Those with thalassaemia major are transfusion-dependent.
- Iron from transfused blood builds up in the reticuloendothelial system, until it is saturated, when iron is deposited in parenchymal tissues, principally the liver, pancreas, endocrine system and heart.
- Preoperative assessment of the degree of major organ impairment (heart, liver, pancreas, endocrine) due to iron overload.
- High-output heart failure with intravascular volume overload is common in severe anaemia. Treat preoperatively by cautious transfusion to correct anaemia and minimise further overload.
- Previous transfusion exposure may cause antibody production, and therefore X-matching may be delayed.

The hyperplastic bone marrow of the major thalassaemias may cause overgrowth and deformity of the facial bones and difficult intubation; this is less common with current management.

Table 11.2 Drug safety in patients with G6PD

Safe		Controversial/ uncertain	Unsafe
Bupivacaine	Paracetamol	Alfentanil	Aspirin (high dose)
Glycopyrronium bromide	Pethidine	Aspirin (low dose)	Diclofenac
Halothane	Phenytoin	Fentanyl	Diazepam
Heparin	Propofol	Isoflurane	Gentamicin
Ibuprofen	Rocuronium	Metronidazole	Lidocaine
Ketamine	Suxamethonium	Midazolam	Methylthioninium chloride (methylene blue)
Mannitol	Thiopental	Penicillin	Metoclopramide
N_2O	Tramadol	Prilocaine	EMLA® cream
Neostigmine		Remifentanil	Quinolones
		Sevoflurane	Sulfonamide antibiotics
		Vitamin K	

Source: data from: Foedinger A, Luger TJ. (2016) Glucose-6-phosphate dehydrogenase deficiency *Anästh Intensivmed* **57**:S115–38. https://www.ai-online.info/images/ai-ausgabe/2016/04-2016/orphan-supplement-6-2016/2016_4_S123-S136_Supplement%20Nr.%206%20-%202016%20-%20Glucose-6-phosphate%20dehydrogenase%20deficiency.pdf.

Disorders of coagulation

(For regional anaesthesia and coagulation abnormalities, see ➲ p. 1110.)

The classic description of the clotting cascade was initially proposed in 1964 by Macfarlane and was generally accepted for over 50y. This historical description involves two pathways, an extrinsic pathway and an intrinsic pathway, that both converge on a common pathway which leads to the formation of a fibrin clot. Both pathways are complex and involve sequential proteolytic activation of proenzymes by plasma proteases (clotting factors), resulting in the formation of thrombin, which then splits the fibrinogen molecule into fibrin monomers.

With further advances in the knowledge of *in vivo* blood coagulation, a cell-based model of coagulation was developed, proposing that coagulation takes place on different cell surfaces in four overlapping steps:

- Initiation
- Amplification
- Propagation
- Termination.

The cell-based model allows a more thorough understanding of how coagulation works *in vivo* and sheds light on the pathophysiological mechanism for certain coagulation disorders. Congenital disorders of clotting may not be present until challenged by trauma or surgery in adult life.

- Acquired disorders are due to ↓ synthesis of coagulation factors, ↑ consumption (e.g. DIC, massive blood loss) and ↑ production of substances that interfere with factor function.
- A family history may be elicited—haemophilia A and B (sex-linked recessive), von Willebrand's disease (autosomal dominant with variable penetrance)—but cannot be relied upon. Family history is absent in 30% of haemophilia cases.
- Response to previous haemostatic challenges (tonsillectomy, dental extractions) may indicate the severity of the coagulopathy; for example, in severe haemophilia A (factor VIII <2%), bleeding occurs spontaneously; in mild haemophilia A (factor VIII 5–30%), bleeding occurs only after trauma.
- Concurrent and past medical problems, such as liver disease, malabsorption (vitamin K deficiency), infection, malignancy (DIC) and autoimmune disease (SLE, RA), as well as medications (anticoagulants, aspirin and NSAIDs), may be relevant.
- Abnormalities due to liver disease and vitamin K deficiency; give daily vitamin K (phytomenadione) 10mg IV slowly. FFP (15mL/kg) may be needed, in addition, if the presenting symptom is bleeding. Coagulation tests may be misleading in the presence of liver disease, overemphasising the bleeding risk; consider using TEG® to evaluate the true underlying coagulopathy.[3]

For blood test results in common coagulation disorders, see Table 11.3.

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Table 11.3 Blood results in common coagulation disorders

Disorder	Platelet count	INR	APTT	TT	Fibrinogen	Other
Haemophilia A	Normal	Normal	↑	Normal	Normal	↓ VIII
Haemophilia B	Normal	Normal	↑	Normal	Normal	↓ IX
von Willebrand's disease	Normal (usually)	Normal	↑	Normal	Normal	↓ VIII, vWF, ↑ bleeding time
Liver disease	Normal or ↓	↑	↑	Normal	Normal or ↓	↓ V
Vitamin K deficiency	Normal	↑	↑	Normal	Normal	↓ II, VII, IX, X
DIC	Normal or ↓	↑	↑	↑	Normal or ↓	↑ FDPs, D-dimers ↓ II, V, VIII
Massive transfusion	↓	↑	↑	Normal or ↑	Normal or ↓	Normal FDPs
Heparin (un-fractionated)	Normal (rarely ↓)	Normal or ↑	↑	↑	Normal	↑ anti-Xa
Heparin (LMWH)	Normal (rarely ↓)	Normal	Normal	Normal	Normal	↑ anti-Xa
Warfarin	Normal	↑	↑	Normal	Normal	↓ II, VII, IX, X
Lupus anticoagulant	Normal	Normal or ↑	↑	Normal	Normal	DRVVT +ve, cardiolipin antibody

APTT, activated partial thromboplastin time; DIC, disseminated intravascular coagulation; DRVVT, dilute Russell's viper venom time; FDPs, fibrin degradation products; INR, international normalised ratio; LMWH, low-molecular weight heparin; TT, thrombin time; vWF, von Willebrand factor.

Haemophilia and related disorders

Inherited disorders of blood coagulation include:

- Haemophilia A (X-linked defect in factor VIII activity)
- Haemophilia B (X-linked defect in factor IX)
- von Willebrand's disease (types 1 and 2 are autosomal dominant; type 3 is autosomal recessive).

Haematological advice should always be sought.

- Untreated mild haemophilia requires strenuous effort to avoid blood products.
- Desmopressin infusion of 0.3 micrograms/kg in 50–100mL of 0.9% sodium chloride over 30min, with the use of tranexamic acid, can be used for mild disease or where there is low risk of bleeding.
- If possible, factor levels should be measured prior to surgery and should be 50–100% of normal and maintained for 2–7d post-procedure.
- If factors are necessary, the treatment of choice is now recombinant factor concentrate, in accordance with established guidelines.
- Cryoprecipitate (contains factor VIII) and FFP (contains factor IX) should be used to correct these clotting factors only in an emergency or if concentrate is unavailable.
- NSAIDs, other anticoagulants, antiplatelet drugs and IM injections should be avoided.
- von Willebrand's disease is divided into four subtypes: types 1, 2 and 3, and platelet type. Type 2 has four subtypes 2A, 2B, 2M and 2N. Desmopressin is given prior to surgical procedures in types 1 and 2A. It works by stimulating the release of von Willebrand factor (vWF) from endothelial cells. A rise in vWF is seen 30–60min after the infusion and maintained for about 6h.
- Desmopressin should not be given to type 2B because of thrombocytopenia and thrombotic complications. Desmopressin is not effective in types 2M and 2N.
- The treatment of choice for patients who are non-responders to desmopressin is virus-inactivated factor VIII concentrate of intermediate purity.
- Haematologists may now advise treatment with recombinant vWF marketed under the name Vonvendi®.

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Thrombocytopenia

Defined as a platelet count <150 × 10^9/L.

- Spontaneous bleeding is uncommon, until the count is < 20 × 10^9/L.

Causes of thrombocytopenia include:

- Failure of platelet production: selective (hereditary, drugs, alcohol, viral infection) or general marrow failure (aplasia, cytotoxic drugs, radiotherapy, infiltration, fibrosis, myelodysplasia, megaloblastic anaemia)
- ↑ platelet consumption: with an immune basis (idiopathic thrombocytopenic purpura (ITP), drugs, viral infections, SLE, lymphoproliferative disorders) or without an immune basis (DIC, TTP, cardiopulmonary bypass)
- Dilution, following massive transfusion of stored blood
- Splenic pooling (hypersplenism).

Preoperative preparation

Unexplained thrombocytopenia should be investigated before elective surgery, as the appropriate precautions will be determined by the underlying cause.

- Minor procedures may be performed without platelet support, provided adequate pressure is applied to the wound.
- For invasive procedures such as central line insertion, transbronchial biopsy, liver biopsy or laparotomy, platelets should be >50 × 10^9/L.
- For epidural or spinal anaesthesia, a platelet count of 80 × 10^9/L is adequate (see ➲ p. 841).
- For operations in critical sites, such as the brain or eyes, the platelet count should be raised to 100 × 10^9/L.
- In ITP, platelet transfusions should be reserved for major haemorrhage, since platelet survival is extremely short-lived. Preparation for surgery entails the use of steroids or high-dose immunoglobulins initially.
- In TTP, platelets are relatively contraindicated. Treatment should consist of plasma or plasma exchange, steroids and, where platelets >50 × 10^9/L, LMWH and aspirin.[4]

Postoperative management

If microvascular bleeding continues, despite a platelet count of >50 × 10^9/L, suspect DIC. If confirmed by coagulation tests, give FFP and cryoprecipitate, as appropriate.

- IM injections, aspirin and NSAIDs should be avoided.
- Desmopressin 0.3 micrograms/kg in 50–100mL of 0.9% sodium chloride over 30min may improve platelet function in renal failure.

Anticoagulants

Anticoagulants are used to reduce thromboembolic risk. However, in patients requiring surgery, they ↑ the risk of major bleeding. It is important to know the indication for anticoagulation and weigh the risk of VTE if it is stopped vs the risk of significant bleeding if it is continued.

The commonest indications for anticoagulation are AF, mechanical heart valves, previous VTE, thrombophilia, cancer or other prothrombotic state. Of these, AF is the commonest. Based on current evidence, patients are now treated with a broad range of anticoagulants based on their own individual risk factors. Direct oral anticoagulants are now generally favoured over warfarin.[5] A meta-analysis of 12 studies in 2015 showed an overall improvement in CVE prevention, a reduction in intracranial haemorrhage and a reduction in mortality when direct oral anticoagulants were used, compared to warfarin, for patients with AF and one other risk factor.[6] They also require no monitoring, unlike warfarin.

A word about terminology CHEST guidelines 2016[7] refer to non-vitamin K antagonist oral anticoagulants as NOACs, however there are other interpretations of this acronym. The International Society of Thrombosis and Haemostasis and the Association of Anaesthetists use the term direct oral anticoagulants (DOACs). These drugs are also referred to as TSOACs (target-specific oral anticoagulants).

CHA_2DS_2-VASc

AF increases a patient's risk of CVE and the CHA_2DS_2-VASc score is used to quantify this risk (Table 11.4). It is used to inform the discussion regarding the risks and benefits of long-term anticoagulation. Patients are scored 0–9. The score is also an indication of annual CVE risk (e.g. score of 1 = annual CVE risk 1.3%; score of 4 = annual CVE risk 4%; score of 9 = annual CVE risk of 15.2%). Anticoagulation should be considered in anyone with a score of ≥1 (except when the only risk factor is ♀ sex), although antiplatelets may suffice with scores ≤2. A balanced decision for treatment must also include assessment of the patient's annual risk of a major bleed 2° to the anticoagulation.

Perioperative risk

Weighing up the risks of stopping anticoagulation or not requires knowledge of the surgery and how likely major or significant blood loss is, as well as a calculation of the patient's thrombotic risk.

Surgical bleeding risk is divided into:

- Low risk (e.g. dental and minor skin procedures)
- Moderate risk (e.g. general surgery, joint replacement, genitourinary or maxillofacial surgery)
- High risk, both of bleeding or bleeding into an enclosed space (cardiac, complex general, vascular, posterior chamber ophthalmic, spinal and intracranial surgery).

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Table 11.4 CHA_2DS_2-VASc score

	Condition	Points
C	Congestive heart failure (or LV systolic dysfunction)	1
H	Hypertension: BP consistently above 140/90mmHg (or treated hypertension on medication)	1
A_2	Age ≥75y	2
D	Diabetes mellitus	1
S_2	Prior CVE or transient ischaemic attack or thromboembolism	2
V	Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)	1
A	Age 65–74	1
Sc	♀ sex	1

Reprinted from *Chest*, **137**, Lip G, Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the Euro Heart Survey on atrial fibrillation, 263–72, Copyright © 2010, with permission from The American College of Chest Physicians. Published by Elsevier Inc.

Evidence for perioperative bleeding risk in patients on antithrombotic therapy is limited. Individual assessment based on the surgery, surgical expertise and patient risk factors should be made.

- Thrombotic risk can be difficult to calculate due to the many indications for anticoagulation. Table 11.5 shows a risk stratification for perioperative thromboembolism.[6,8]

Table 11.5 Risk stratification for perioperative thromboembolism

Risk category	Mechanical heart valve	AF	VTE
High	Any mitral valve prosthesis or caged-ball or tilting disc aortic valve prosthesis	CHA_2DS_2-VASc score of ≥6 Rheumatic valvular disease	VTE within 3mo Severe thrombophilia*
Intermediate	Bileaflet aortic valve prosthesis + ≥1 of the following: AF, CVE, TIA, hypertension, DM, congestive heart failure, age >75y	CHA_2DS_2-VASc score of 4–5 CVE or TIA >3mo ago	VTE within 3–12mo Non-severe thrombophilia** Recurrent VTE Active cancer
Low	Bileaflet aortic valve	CHA_2DS_2-VASc score of ≤3	VTE >12mo ago with no other risk factors

* Severe thrombophilia is defined as deficiency in protein C, protein S or antithrombin; antibodies and those with multiple abnormalities.

** Non-severe thrombophilia is defined as heterozygous for factor V Leiden or prothrombin gene mutation.

Source: data from Hornor MA, *et al*. American College of Surgeons' Guidelines for the Perioperative Management of Antithrombotic Medication. *J Am Coll Surg*. 2018;**227**:521–36 and Douketis JD, *et al*. Perioperative management of antithrombotic therapy, Chest 2012;**141**: e326S–e350S.

As a general rule, the risk from a major perioperative bleeding event is likely to be less than that from thrombosis. Consider, for example, that the risk of major bleeding on antithrombotic agents is estimated at 6–10%, whereas the mortality associated with:[8]

- Embolic CVE is 37%
- Mechanical heart valve thrombosis is 17.5%
- VTE (DVT or PE) is 5–10%.

Fig. 11.2 illustrates perioperative management of a warfarinised patient relative to surgical bleeding risk and their risk of VTE.

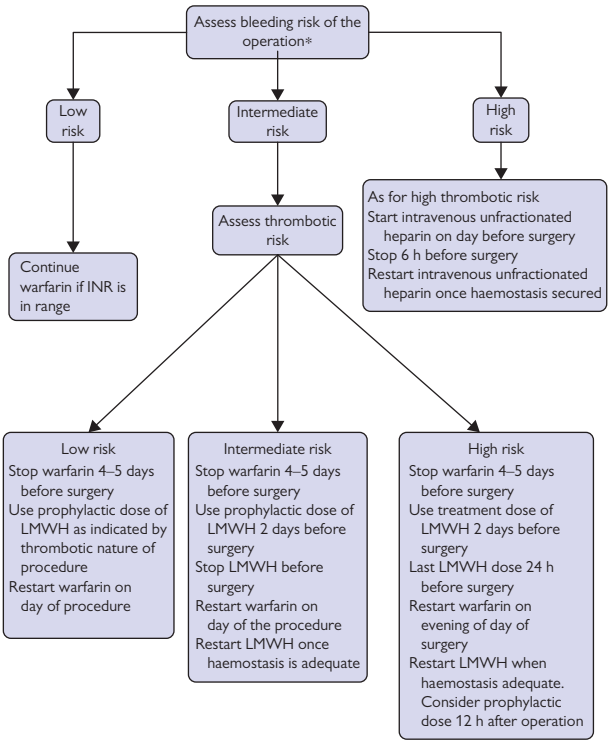

Fig. 11.2 Perioperative management of anticoagulation based on risk assessment. Reproduced with permission from Thachil J *et al*. Management of surgical patients receiving anticoagulation and antiplatelet agents. *British Journal of Surgery* 2008;**95**:1437–48. Copyright © 2008 British Journal of Surgery Society Ltd. Published by John Wiley & Sons, Ltd.

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- If using continuous IV infusion (IVI) of UFH for patients with a high risk of bleeding, start at 1000 units/h and adjust to keep the APTT between 1.5 and 2.5 (Fig. 11.2).
- Direct oral anticoagulants do not require bridging, but timing of the last preoperative dose is variable and covered later in the chapter.
- It is worth noting that the perioperative risk of arterial thromboembolism in patients who have AF and no anticoagulation is ~1%.
- Perioperatively, additional methods of VTE prophylaxis should be considered (e.g. compression stockings and pumps) in all patients at risk of VTE despite anticoagulation (see pp. 59–61).

Timing of surgery

Consider whether the surgery can be delayed in order to reduce the risk of VTE and associated morbidity and mortality.

- Prematurely stopping anticoagulation after VTE within the 1st month confers an estimated 30d risk of recurrent VTE of 40%. This falls to an annual risk of 15% after 3mo.
- Vena cava filters should be considered in patients who have had VTE <4w ago and require interruption of their anticoagulation or in whom postoperative anticoagulation may be delayed >12h.

Warfarin

Warfarin, a vitamin K antagonist, was the original anticoagulant used in patients with AF and remains the only treatment with established safety for patients with AF and rheumatic heart disease and/or mechanical heart valve prosthesis.

- Warfarin results in synthesis of non-functional vitamin K-dependent factors (II, VII, IX and X, proteins C and S), prolonging PT, but may take up to 48h to become effective.
- Warfarin is highly effective but has a narrow therapeutic window, requiring regular testing and dose adjustment.
- Warfarin is associated with haemorrhagic CVE, and intracranial and GI haemorrhage.
- The INR compares a patient's PT to a control, indicating the degree of anticoagulation.
- It reduces the risk of CVE by 67% and mortality by 25%, compared with control (aspirin or no therapy).
- Once the INR is <2, alternative pre- and postoperative prophylaxis should be considered.

Recommended INR targets

- INR 2–2.5 for prophylaxis of DVT
- INR 2.5 for treatment of DVT/PE, prophylaxis in AF and cardioversion
- INR 3.5 for recurrent DVT/PE or mechanical heart valves

The effects of warfarin can be reversed in a number of ways, dependent on the urgency.

- If no bleeding and INR <5, reducing or omitting a dose is usually sufficient, but if INR 5–9, 1–2mg vitamin K PO should also be given.
- If there is minor bleeding or a grossly raised INR >9, give 2–5mg vitamin K PO or IV.
- For emergency surgery or life-threatening bleeding, specialist haematological advice should be sought. Prothrombin complex concentrates (PCCs) have replaced FFP as 1st-line treatment. The dose varies, depending on the initial INR. PCC (e.g. Octaplex® or Beriplex®) is a pooled blood product and prothrombotic, and can be associated with DIC. Vitamin K 10mg should also be given as a slow IV injection. Where PCC is not available, then 10–15mL/kg FFP can be given, although it is not quite as effective.

The important anaesthetic interactions with warfarin include:
- Potentiation (by inhibition of metabolism): alcohol, amiodarone, cimetidine, ciprofloxacin, co-trimoxazole, erythromycin, indometacin, metronidazole, omeprazole, paracetamol
- Inhibition (by induction of metabolism): barbiturates, carbamazepine.

Drugs that affect platelet function can increase the risk of warfarin-associated bleeding, e.g. aspirin and NSAIDs.

Heparin (UFH and LMWH)

A parenterally active anticoagulant that potentiates antithrombin, which, in turn, inactivates thrombin and factor Xa, along with other proteases.
- Used for prophylaxis and treatment of thromboembolism.
- Heparin is a polymer of variable length. Available as UFH or LMWH which has undergone fractionation to make the effects more predictable.
- Common LMWHs include dalteparin, enoxaparin and tinzaparin. Doses can vary, depending on renal function and body weight.
- Suggested daily SC doses for surgical VTE prophylaxis in high-risk adults >50kg, eGFR >30mL/min/1.73m^2: tinzaparin 4500 units, dalteparin 5000 units, enoxaparin 40mg.
- Heparin is metabolised in the liver and renally excreted. Use caution with LMWH in patients with renal failure.
- Complications of heparin include heparin-induced thrombocytopenia (HIT), which can cause serious venous and arterial thrombosis. Patients on UFH for 5d or more should have their platelet counts checked. This is less of a problem with LMWH.
- UFH is given by IV bolus or IVI. It is monitored by prolongation of APTT (maintain at 1.5–2.5 times the normal laboratory value). It has a narrow therapeutic window with complex pharmacokinetics and great interpatient variation in dose requirements. Half-life is 1–2h.
- A validated regime is to give a bolus of 80 units/kg, followed by an infusion of 18 units/kg/h, checking the 1st APTT after 6h.
- Protamine is used to reverse both UFH and LMWH; however, the neutralising effect of protamine on the inhibition of factor Xa is 95% for UFH, but only 55% for LMWH. It should be given slowly to avoid hypotension.
- Reversal of UFH: if given within 15min of UFH, 1mg of protamine IV neutralises 100 units of UFH. After this, less protamine is required, as heparin is rapidly excreted.

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- Reversal of LMWH: 1mg of protamine is given per 1mg of enoxaparin or 100 units of dalteparin. Halve the dose if >8h since administration.
- LMWH has largely replaced UFH for both prophylaxis and treatment of thromboembolism and unstable CAD. Administered once daily by SC injection, it needs no monitoring (although antifactor Xa levels can be measured in renal failure).

Direct oral anticoagulants

Direct thrombin (factor IIa) inhibitors

Direct thrombin inhibitors were developed to overcome some of the limitations of the indirect thrombin inhibitors (UFH and LMWH).

- They offer a predictable response as protein binding is minimal.
- They also do not cause HIT.

They are divided into: univalent—binding only to the catalytic (active) site of thrombin; and bivalent—binding to both the catalytic site and a 2nd site, exocite-1.[9]

Univalent direct thrombin inhibitors: argatroban and dabigatran

- Argatroban binds reversibly to thrombin. Given IV, its plasma half-life is 39–51min and it undergoes hepatic metabolism to inactive metabolites. Approved for prophylaxis or treatment of thrombosis in patients with HIT, and as an anticoagulant in patients with or at risk of HIT requiring PCI. Monitored via APTT.
- Dabigatran is administered PO. It is licensed for extended VTE prophylaxis after hip and knee replacement surgery, and to reduce the risk of CVE and systemic embolism in patients with non-valvular AF.

Bivalent direct thrombin inhibitors: hirudin and bivalirudin

- Hirudin, originally isolated from saliva of the medicinal leech, binds irreversibly to thrombin. It has a plasma half-life of 60min if given IV (120min if given SC) and is renally excreted. APTT is used to monitor the effect, aiming for 1.5–2.5 times normal. It is approved for treatment of arterial and venous thromboembolism complicated by HIT and as an alternative to heparin for CPB surgery.
- Bivalirudin is a reversible thrombin inhibitor. Peak plasma levels are reached after 15–20min IV administration. Half-life is ~30min, with 80% eliminated by plasma enzymes and 20% by renal excretion. Monitored by activated clotting time (ACT).[9] Reversal in emergency is by recombinant factor VIIa, FFP and cryoprecipitate (or fibrinogen) or modified ultrafiltration. It is approved for use in coronary angioplasty.

Direct factor Xa inhibitors

- Anticoagulants acting directly on factor X, which can be given PO and have rapid onset and offset.
- They inactivate both circulating and clot-bound Xa.
- There is no need to monitor drug effect, although antifactor Xa assay can be used if needed.
- Examples include apixaban, rivaroxaban and edoxaban.

Indirect factor Xa inhibitor

- Fondaparinux is a synthetic inhibitor of factor Xa, which can be given PO or IV. Monitoring is not required, but since it is excreted largely unchanged, the dose should be altered in renal failure. APTT may be altered but is not a true reflection of action. It is licensed for prophylaxis of VTE in immobilised medical patients, after joint replacement or abdominal surgery and in the treatment of DVT and PE or acute coronary syndrome. Usual dose is 2.5mg SC for surgical VTE prophylaxis after major orthopaedic surgery.

Reversal of direct oral anticoagulants

If there is minor bleeding or no bleeding, just discontinue the agent. If there is major haemorrhage, specific reversal agents can be used (see Table 11.6), but also consider:

- Activated charcoal (only within 3h of ingestion)
- Dialysis for dabigatran
- 4-factor PCC which contains factors II, VII, IX and X
- Recombinant factor VIIa
- Tranexamic acid.

Idarucizumab is used for reversal of dabigatran; it is a humanised monoclonal antibody fragment that binds specifically to dabigatran and its metabolites, thereby reversing the anticoagulant effect. Full reversal dose is 5g (+ 5g if a 2nd dose is required). Dabigatran can be restarted after 24h.

Andexanet alfa is used for reversal of apixaban and rivaroxaban. Andexanet alfa is a recombinant form of human factor Xa protein which binds specifically to apixaban or rivaroxaban. At the time of writing, this product is not yet licensed in the UK.

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Table 11.6 Summary of direct oral anticoagulant characteristics

	Apixaban	Rivaroxaban	Edoxaban	Dabigatran
Dose (normal renal function)	5mg twice daily	20mg once daily	30–60mg once daily*	150mg once daily
Action	Direct factor Xa inhibitor	Direct factor Xa inhibitor	Direct factor Xa inhibitor	Direct thrombin inhibitor
CVE prevention in non-valvular AF	✔	✔	✔	✔
VTE prevention and treatment		✔	✔	✔
Prophylaxis after acute coronary syndrome or CAD		✔		
Half-life (h)	12	5–13	10–14	12–17
Reversal with 4-factor PCC	✔	✔	✔	✔
Specialist reversal available	Andexanet alfa	Andexanet alfa	No	Idarucizumab
Last dose before operation: low risk of bleeding (NICE guidance 2020**)	CC ≥30mL/min, stop for 24h CC 15–29mL/min, stop for 36h			CC ≥80mL/min, stop for 24h CC 50–79, stop for 48h CC 30–49, stop for 72h
Last dose before operation: high risk of bleeding (NICE guidance 2020**)	Stop for 48h			CC ≥80mL/min, stop for 48h CC 50–79, stop for 72h CC 30–49, stop for 96h

CC, creatinine clearance.

* Dose dependent on patient weight.

** NICE guidance on oral anticoagulation, updated January 2020. https://cks.nice.org.uk/anticoagulation-oral

Antiplatelet drugs

Antiplatelet drugs decrease platelet aggregation and inhibit thrombus formation in the arterial circulation where anticoagulants have little effect. Their indications include 1° or 2° prevention of CVE and CVS disease. There are now many antiplatelet medications available, some of which are summarised in Table 11.7.[10]

Aspirin

Aspirin irreversibly acetylates the active site of cyclo-oxygenase (COX) 1 (2 at higher doses), which blocks the production of thromboxane A2, a powerful promoter of platelet aggregation. Recovery of platelet function requires formation of new platelets. Low-dose aspirin is a mainstay for 2° prevention of thrombotic vascular events in vascular and cardiac disease. Also used in angina, post-coronary artery bypass surgery, intermittent claudication, AF and 1° prevention of IHD. Once stopped, it takes around 7–9d for platelet function to return to normal. This risk of bleeding must be balanced against the possibility of precipitating a thromboembolic event, particularly in patients with unstable angina.

Dipyridamole

A nucleoside transport and phosphodiesterase inhibitor, dipyridamole is an antiplatelet and a vasodilator. It is used in 2° prevention of CVE or with low-dose aspirin for post-coronary artery surgery and valve replacement. It has an elimination half-life of 10h. There is variability in the literature as to when, if at all, dipyridamole needs to be stopped prior to surgery. The AoA states that it does not need to be stopped at all prior to regional blocks, but other sources recommend it should be stopped anywhere from 24h to 7d. Check your local policy.

Adenosine diphosphate/$P2Y_{12}$ inhibitors

$P2Y_{12}$ is the chemoreceptor responsible for adenosine diphosphate (ADP) stimulation of the glycoprotein IIb/IIIa receptor. Stimulation of IIb/IIIa receptors leads to enhanced platelet degranulation, thromboxane release and prolonged platelet aggregation. Antagonism of the $P2Y_{12}$ receptor may be irreversible with clopidogrel or prasugrel, or reversible with ticagrelor or cangrelor. Often used with aspirin as DAPT for acute coronary syndrome, but also after PCI, CABG, AF in patients unable or unwilling to take anticoagulants, CVE (not prasugrel) and peripheral vascular disease. Clopidogrel remains the commonest agent, but guidelines are continually being revised. Recent evidence suggests that prasugrel and ticagrelor may be more effective than clopidogrel, but with a higher risk of bleeding. Cangrelor is given IV and may be an option for patients requiring PCI who have not been loaded with oral therapy.

Glycoprotein IIb/IIIa inhibitors

These medications compete with fibrinogen and vWF for IIb/IIIa receptors. They prevent both platelet crosslinking and platelet-derived thrombus formation. They are all given IV and are potent inhibitors of platelet activity. Abciximab is a large monoclonal antibody with a high affinity for binding to the glycoprotein IIb/IIIa receptor. It has the longest duration of action and has a UK licence as an adjunct to aspirin and UFH in PCI. While its biological half-life is 12–24h, due to slow clearance, its functional half-life is

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up to 7d, so it must be stopped 1w prior to elective surgery. Eptifibatide and tirofiban both have a rapid onset of action and a short half-life and are used to prevent early MI in unstable angina and non-ST-elevation myocardial infarction. Significant recovery of platelet aggregation is seen 4h after infusion is stopped and therefore, they only need to be stopped 4–8h before elective surgery.

Epoprostenol

Epoprostenol is prostacyclin or prostaglandin I_2 prepared in drug form. It is a potent peripheral and pulmonary vasodilator, as well as an antiplatelet medication. It is used in the treatment of pulmonary hypertension, and in haemodialysis where heparin is contraindicated. It is given by IVI and has a short half-life.

Perioperative management of antiplatelet drugs

Perioperative management of patients on antiplatelet therapy requires:

- Understanding the indication
- Timing of index event (i.e. CVE, MI, PCI)
- Risk of stent thrombosis
- Consequences of delaying surgery
- Risk of surgical bleeding.

Monotherapy

Continuation of aspirin monotherapy is widely accepted, unless surgery has a risk of major blood loss (e.g. CABG and transurethral prostatectomy) or risk of bleeding in confined space (posterior chamber ophthalmic, spinal and intracranial surgery). If in doubt, ask the surgeon. If aspirin needs to be stopped, the last dose should be 7d prior to surgery.

Dual antiplatelet therapy

With the increase in 1° PCI and coronary stenting, DAPT has become commonplace, often with aspirin and clopidogrel. After placement of a coronary stent, antiplatelet drugs are required to prevent late in-stent thrombosis. BMS only require DAPT for 6w, compared to the older-generation DES which require 12mo. The European Society of Cardiology now suggests that a patient's bleeding and thrombosis risk should be assessed on an individual basis and newer-generation DES may only require DAPT for as little as 6mo to >12mo.[11,12]

Wherever possible, it is recommended to complete the full course of DAPT prior to elective non-cardiac surgery. Early discontinuation of antiplatelet therapy is the most significant determinant of stent thrombosis, which can have a mortality of up to 50%. If surgery must occur while the patient is on DAPT, consider if it needs to be stopped. If there is a low risk of bleeding, it is often possible to continue DAPT. In surgical procedures where DAPT must be stopped, it may be possible to stop the $P2Y_{12}$ inhibitor, but continue aspirin monotherapy perioperatively. Patients with a high risk of bleeding perioperatively will need to stop DAPT. Stopping DAPT and starting parenteral glycoprotein IIb/IIIa inhibitors in the perioperative period has been proposed in patients with high thrombotic risk, and some centres may advocate bridging with heparin. If a patient must have surgery while on DAPT, then liaison with both the surgeon and physician responsible for starting the DAPT (usually the cardiologist) is paramount.

Management of intraoperative bleeding

TEG®, rotational thromboelastometry (ROTEM®) and Platelet Mapping Assays may be helpful in guiding management. Acute bleeding associated with antiplatelet medications can be partially treated with platelet transfusion, but free drug may continue to inhibit the transfused platelet function. Consider FFP, cryoprecipitate, recombinant factor VII and (with little evidence) desmopressin.

Restarting antiplatelet drugs

Antiplatelet drugs should be restarted as soon as possible, guided by the surgeon and their assessment of the risk of bleeding.

Table 11.7 Summary of antiplatelet medication

	Aspirin	Clopidogrel	Prasugrel	Ticagrelor	Abciximab	Epoprostenol
Mechanism of action	COX-1 inhibition	ADP receptor antagonist	ADP receptor antagonist	ADP receptor antagonist	Glycoprotein IIb/IIIa receptor inhibitor	Prostaglandin I_2 inhibits thromboxane A2
Half-life	15–30min	7–9h	7h	7–9h	10–15min	<6min
Typical loading dose*	300mg	300–600mg	60mg	180mg	0.25mg/kg	None
Typical daily maintenance dose*	75–300mg	75mg	5–10mg	90mg bd	0.125mg/kg/min	4ng/kg/min
Time to recover platelet function after stopping drug	30% at 48h	40% at 3d	2–3d	57% at 24h	12h	<30min
Route of administration	PO	PO	PO	PO	IV	IVI

* Dosing may vary, depending on age, weight and renal function.

Source: data from Oprea AD, *et al*. Perioperative management of antiplatelet therapy, *British Journal of Anaesthesia*, **111**(1) 2013, i3–17.

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Fibrinolytic drugs

These drugs act as thrombolytics by activating plasminogen to plasmin; this degrades fibrin and therefore dissolves thrombi.

- Newer drugs, such as reteplase and tenecteplase, are given by bolus injection, making them ideal for early community injection.
- Alteplase (recombinant tissue plasminogen activator (rt-PA)) and streptokinase are given by continuous infusions.
- Used for AMI where benefits outweigh risks, e.g. when PCI not available.
- Benefit greatest with early injection, ECG changes with ST-elevation or new bundle branch block and anterior infarction.
- Alteplase, reteplase and streptokinase need to be given within 12h of symptom onset, ideally within 1h; use after 12h requires specialist advice. Tenecteplase should be given as early as possible and usually within 6h of symptom onset.
- Should be used in combination with antithrombin (LMWH) and antiplatelet (aspirin) therapy to reduce early reinfarction.
- Alteplase, streptokinase and urokinase can be used for other thromboembolic disorders such as DVT and PE. Alteplase is also used for acute ischaemic CVE. Treatment must be started promptly.
- Contraindications include any risk of bleeding, especially trauma (including prolonged CPR), recent surgery and GI tract and intracerebral pathology.
- Streptokinase can cause allergic reactions and should be used only once. Antibodies can inactivate the drug.
- Serious bleeding calls for the discontinuation of therapy and may require coagulation factors. Cryoprecipitate (high levels of factor VIII and fibrinogen) and FFP (factors V and VIII), as well as platelets, may all be required. Antifibrinolytic drugs, such as aminocaproic acid or tranexamic acid, may also be useful.
- Bleeding times are prolonged for up to 24h after these drugs. In emergency surgery, reversal will be required.
- Urokinase is also licensed to restore the patency of occluded IV catheters and cannulae blocked with fibrin clots. Inject directly into the catheter or cannula 5000–25 000 units dissolved in a suitable volume of 0.9% sodium chloride to fill the catheter or cannula lumen; leave for 20–60min, then aspirate the lysate; repeat, if necessary.

Haemostatic drug therapy

Tranexamic acid (and aminocaproic acid)

Both these drugs are synthetic derivatives of the amino acid lysine and reversibly bind to plasminogen, preventing its conversion to plasmin and therefore its ability to cleave fibrin.

- Tranexamic acid is ten times more potent than aminocaproic acid.
- The CRASH 2 Trial[13] found tranexamic acid to decrease the risk of death in people who have significant bleeding due to trauma by 30% if given within 3h from the initial trauma; 1g IV over 10min, followed by 1g over the next 8h.
- The CRASH 3 Trial[14] supports the use of tranexamic acid in traumatic brain injury.
- Neither trial suggested an increase in vaso-occlusive events.
- Tranexamic acid is increasingly used to reduce surgical blood loss.
- Other uses include postoperative bleeding in prostatectomy and dental extractions (particularly in haemophilia), cardiac and craniofacial surgery and menstrual bleeding.
- Also useful in the reversal of thrombolytic drugs.
- Contraindicated in renal failure, epilepsy, benign gynaecological interventions (e.g. myomectomy), following an acute VTE and fibrinolysis due to DIC without any significant bleeding.
- Usual dose of tranexamic acid is 1g tds PO for relatively minor bleeding such as menorrhagia or epistaxis.
- For surgical bleeding (or anticipated bleeding as in orthopaedic surgery, e.g. total knee replacement), a typical dose would be 1g IV (or 10–20mg/kg) given prior to incision. A 2nd dose of 0.5–1g IV may be given postoperatively for continued bleeding.

Aprotinin

A serine protease inhibitor, it inhibits free plasmin (but not bound plasmin).

- The drug was temporarily withdrawn worldwide following the Blood Conservation Using Antifibrinolytics in a Randomized Trial (BART) trial[15] after studies suggested that its use ↑ the risk of complications or death in cardiac surgery.[15,16] In February 2012, the European Medicines Agency (EMA) reverted its previous standpoint regarding aprotinin and recommended that the suspension be lifted.
- The drug is still available—Nordic became the manufacturer in 2012.
- It is contraindicated for repeated use within a year due to anaphylaxis.
- It can cause renal failure and is contraindicated in renal insufficiency.
- The drug is useful during the anhepatic phase of liver transplantation where its use is guided by TEG®.

Desmopressin

An analogue of arginine vasopressin which induces the release of vWF from the vascular endothelium to increase both vWF and factor VIII.

- Can be used (0.3 micrograms/kg given in 50–100mL of 0.9% sodium chloride over 30min) for haemophilia A and von Willebrand's disease to double or quadruple the levels of vWF or factor VIII.
- Platelet function may also be improved in patients with renal failure and aspirin-induced platelet dysfunction.

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Factor VIIa

Recombinant factor VIIa (rFVIIa) acts at the 'tissue factor–factor VIIa' complex at the site of endothelial damage.

- This effect appears localised to the area where the vessel is damaged, leading to few systemic side effects.
- Numerous case reports have shown rFVIIa to have potent haemostatic effects, even when other treatments have failed, regardless of the cause of bleeding. Its use to stop bleeding during operations is off-licence, however. These potential benefits have to be weighed up against its thrombogenic risk; a Cochrane review (updated February 2011) concluded the data supporting the off-licence use of rFVIIa were weak, and the use of rFVIIa outside its current licensed use—haemophilia and inhibitory alloantibodies and for prophylaxis and treatment of patients with congenital factor VII deficiency—should be restricted to clinical trials.
- Dosing and mode of delivery (IV bolus or continuous infusion) have still not been established (20–40 micrograms/kg has been used).

Prothrombin complex concentrates

Dried prothrombin complex is prepared from human plasma and contains factor IX, together with variable amounts of factors II, VII and X. Often referred to as 4-factor PCC.

- Indications are treatment and prophylaxis of congenital or acquired deficiency of factors II, VII, IX and X (such as during warfarin treatment).
- Contraindications are angina, recent MI and history of HIT.
- Side effects include thrombosis and hypersensitivity, including anaphylaxis.

Disseminated intravascular coagulation

DIC occurs when a disease process disturbs normal haemostasis and leads to abnormal coagulation and fibrinolysis within the vasculature. It is often termed consumptive coagulopathy due to the extent of clot formation 'using up' clotting factors. Overactivation of coagulation leads to the production of thrombi consisting of fibrin and platelets. Thrombi in the microcirculation and larger vessels lead to further clot formation. Fibrinolysis follows, forming fibrin degradation products. End-organ damage can then occur from hypoperfusion, thrombosis and/or bleeding.

DIC may be acute or chronic. Chronic DIC occurs slowly, allowing balance between consumption and regeneration of clotting products, and is typically seen in malignancy (pancreatic, gastric, ovarian) and in patients with haemangiomas, aneurysms and carcinomatosis. Acute DIC is seen with:

- Sepsis, often Gram-negative bacteria
- Malignancy, e.g. acute promyelocytic leukaemia, mucinous tumours (pancreatic, gastric, ovarian) and brain tumours. Cancer procoagulant is a proteolytic enzyme capable of activating factor X
- Trauma, especially to CNS, or crush injuries
- Obstetric complications: pre-eclampsia, amniotic fluid embolism (AFE)
- Acute haemolytic transfusion reaction following ABO incompatibility. Tissue factor release from monocytes plus generation of cytokines (TNF, IL-1) and reduced nitric oxide (NO) lead to clot formation and vasoconstriction.
- Fat embolism.
- Heat stroke and amphetamine overdose.

Presentation and diagnosis

Presentation of DIC can be in the form of bleeding, VTE and arterial thrombosis with organ failure, with renal, hepatic, lung, CNS and adrenal being commonest.

Laboratory abnormalities are variable and non-specific, dependent on the severity of the DIC. The following would support a diagnosis of DIC:

- ↑ PT and APTT, thrombin time and D-dimer
- ↓ platelets, fibrinogen, clotting factors VII, X, V and II, coagulation inhibitors antithrombin III and proteins C and S
- Differential diagnosis typically presenting with thrombocytopenia, but with otherwise normal coagulation studies, include other microangiopathies (e.g. TTP and complement-mediated haemolytic uraemic syndrome (HUS)).

Treatment

Treatment aims to be supportive and should be discussed with a haematologist.

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Coagulation tests

Standard laboratory coagulation tests typically look at isolated areas of the clotting cascade and can be related to intrinsic and extrinsic pathways in the classical coagulation model. In the bleeding coagulopathic patient, pH, temperature and ionised Ca^{2+} can also be measured for correction.

- **PT**: the time in s for clot to form, following addition of thromboplastin and Ca^{2+} to citrated plasma. Detects deficiencies or inhibitors of factors II, V, VII and X, and fibrinogen. Normal range: 11–13.5s.
- **INR**: comparison of PT with laboratory reference sample to allow easy standardisation. Detects deficiencies or inhibitors of factors II, V, VII and X, and fibrinogen. Used for monitoring warfarin. Normal range: 0.8–1.1.
- **APTT**: time for clot to form after an intrinsic factor activator (i.e. kaolin) and Ca^{2+} added to citrated plasma. Detects deficiencies or inhibitors of factors XII, XI, X, IX, VIII, V and II, and fibrinogen. Normal range: 25–35s.
- **Fibrinogen**: usually calculated from a coagulation assay, e.g. Clauss assay (thrombin added to dilute plasma until clot forms). Normal range: 2–4g/L.

POCT provides rapid and accurate information typically in theatre or the ICU, allowing targeted, rather than empirical, treatment. There are three main types of point-of-care coagulation testing:

- Coagulation time analysers, e.g. PT/APTT/ACT
- Viscoelastic clot strength analysers, e.g. TEG® and ROTEM®
- Platelet function analysers.

Coagulation time analysers

- PT and APTT can be measured by a small portable analyser. A drop of whole blood is added to a cuvette. This can be useful for monitoring warfarin status or heparin therapy.
- ACT is used in patients treated with high concentrations of UFH. A drop of whole blood is added to a cuvette and the sample activated using kaolin or celite. Often used in CPB surgery. Normal range: 70–120s. For CPB, the target ACT is usually in the range of 400s.

Viscoelastic tests: thromboelastography/thromboelastometry

Two analysers TEG® and ROTEM® are available and measure the strength and elastic properties of clotting whole blood. TEG® uses thromboelastography, while ROTEM® uses rotational thromboelastometry. These viscoelastic tests give functional information on the time taken for clot formation to begin, the speed and strength of clot formation and clot lysis. Unfortunately, the standard tests are unable to test platelet function. The potent platelet activation by thrombin overwhelms the effects of the weaker platelet activators (ADP and arachidonic acid) on which antiplatelet medications work. Newer Platelet Mapping Assays can be used with some viscoelastic tests to allow assessment of antiplatelet drugs.

Techniques to assess the viscoelastic properties of clotting can involve a rotating cup into which a wire is inserted, a spinning wire in a cup and, more recently, subjecting the blood to vibration whereby the vertical movement of the blood meniscus is measured under LED illumination. A number of different reagents are used in both techniques, but no matter the technique, the same trace is produced which facilitates targeted correction of coagulopathy (Fig. 11.3). The nomenclature and normal values differ between the two techniques (Table 11.8).

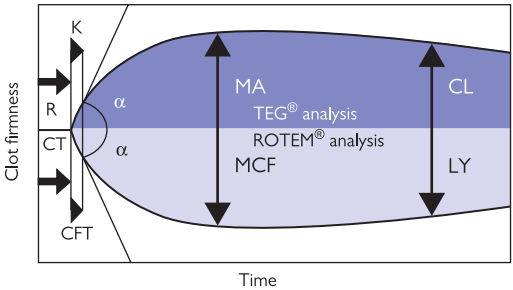

Fig. 11.3 Typical trace for both TEG® and ROTEM®.

TEG®

TEG® uses kaolin as the reagent and tests the intrinsic pathway.

- Rapid TEG® (rTEG®) takes just 15min, compared with over 30min for a standard trace, and uses kaolin and tissue factor to activate the extrinsic pathway, which speeds up the test. In this test, the R-value is replaced by the TEG-ACT value which is measured in s, rather than in minutes. The remainder of the TEG parameters do not differ from the standard test.
- Functional fibrinogen includes tissue factor and a platelet inhibitor (abciximab) which removes the platelet contribution to clot strength, so that the fibrinogen component can be seen independently.
- Platelet Mapping is a new addition to the TEG® repertoire and is explained below.

ROTEM®

ROTEM® has a number of different tests:

- INTEM uses phospholipid and ellagic acid to test the intrinsic pathway and is similar to APTT.
- EXTEM also uses tissue factor to test the extrinsic pathway and is similar to PT.
- FIBTEM uses a platelet inhibitor (cytochalasin D) to inhibit the platelet contribution and isolate the fibrinogen contribution to clot strength.
- HEPTEM is essentially the same as INTEM, but with the addition of heparinase which eliminates heparin from the sample, uncovering any other underlying coagulopathy.
- APTEM uses aprotinin to rule out excessive fibrinolysis which could be an indication for tranexamic acid.
- ECATEM is prolonged in patients on direct thrombin inhibitors. Using ecarin, a prothrombin activator, it is similar to the ecarin clotting time test.

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Table 11.8 Normal values for TEG® and the ROTEM®

	TEG®	ROTEM®
Clotting time (time to 2mm amplitude)	R* 4–8min	CT: INTEM** 137–246s EXTEM** 42–74s
Clot kinetics (2–20mm amplitude)	K* 1–4min	CFT: INTEM** 40–100s EXTEM** 46–148s
Clot strength (α angle)	α* 47–74°	INTEM** 71–82° EXTEM** 63–81°
Maximum strength	MA* 55–73mm	MCF: INTEM** 52–72mm EXTEM** 49–71mm
Clot lysis	CL30, CL60	LI30, ML

* Kaolin activated.

** Citrated.

CFT, clot formation time; CL30, clot lysis at 30min; CL60, clot lysis at 60min; CT, clotting time; K, kinetics; LI30, lysis index at 30min; MA, maximum amplitude; MCF, maximum clot firmness; ML, maximum lysis; R, reaction time.

Interpretation

Interpretation of a ROTEM® or TEG® is similar to learning how to interpret an ECG. A stepwise approach is used initially and later pattern recognition (Fig. 11.4).

Prolongation of clotting time/clot formation time or reaction/kinetics times

Has the patient had heparin? A HEPTEM assay will be able to rule this out. If heparin has been given and reversal is required, consider protamine. If no heparin, the results are due to clotting deficiencies, so consider FFP.

Reduced maximum amplitude or maximum clot firmness

Perform a FIBTEM or a functional fibrinogen test. Is maximum amplitude or maximum clot firmness reduced using these tests? If so, the result is due to fibrinogen deficiency, so consider using cryoprecipitate or fibrinogen concentrate. If not, the result is due to platelet deficiency, so correct with platelet transfusion.

Increased CL30/CL60 or LI30/LI60

There is excessive fibrinolysis, so consider an antifibrinolytic. An APTEM test will inhibit fibrinolysis, bringing LI30 and LI60 back to normal limits, confirming the result.

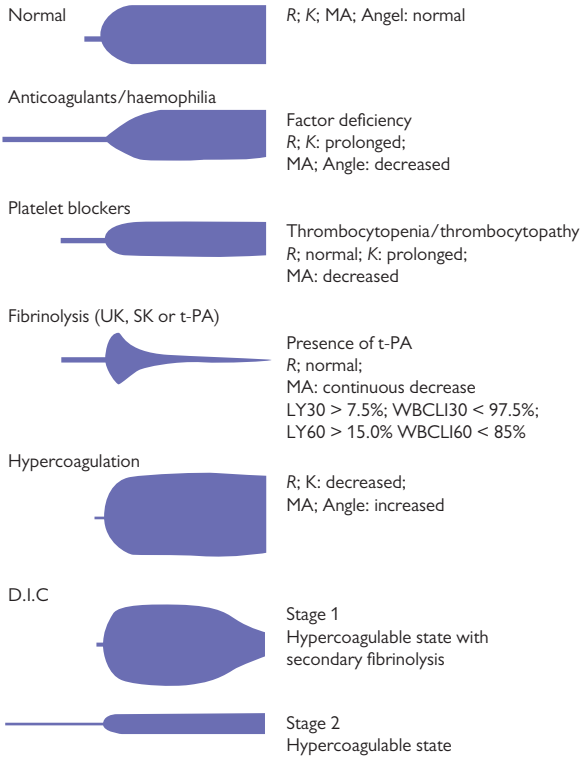

Fig. 11.4 Characteristic TEG® traces.

Platelet function analysers

Both quantitative and qualitative defects in platelet function can lead to major bleeding after surgery or trauma. This may be genetic, due to a disease process, consumptive or as a result of an antiplatelet agent. There are now many point-of-care platelet analysers available. They have specific activators to detect COX inhibitors (e.g. aspirin), $P2Y_{12}$ antagonists (e.g. clopidogrel) and glycoprotein IIb/IIIa antagonists (e.g. abciximab).

- Similar to viscoelastic tests for coagulation, a graph is produced and typically, inhibition of platelet function is reported as a percentage (e.g. platelet function is 90% inhibited by clopidogrel in this sample).
- Analysers include PFA-100®, Plateletworks®, TEG Platelet Mapping®, VerifyNow®, Multiplate® and ROTEM PM®. With the exception of PFA-100® which is only for aspirin, all these analysers measure all three types of antiplatelet agents.

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Hypercoagulability syndromes

Polycythaemia

A pattern of RBC changes that usually results in Hb >17.5g/dL in ♂ and >15.5g/dL in ♀. This is accompanied by a corresponding increase in the red cell count to 6.0 and 5.5 × 10^{12}/L and an Hct of 55% and 47%, respectively.

Causes

- 1°: polycythaemia vera (PV)
- 2°: due to compensatory erythropoietin (EPO) increase (high altitude, cardiorespiratory diseases—especially cyanotic, heavy smoking, methaemoglobinaemia) or inappropriate EPO increase (renal diseases: hydronephrosis, cysts, carcinoma; massive uterine fibromyomata; hepatocellular carcinoma; cerebellar haemangioblastoma)
- Relative: stress or spurious polycythaemia. Dehydration or vomiting
- Plasma loss: burns, enteropathy.

Polycythaemia vera

Presenting features include headaches, dyspnoea, chest pain, vertigo, pruritus, epigastric pain, hypertension, gout and thrombotic episodes (particularly retinal).

- Splenomegaly is typical.
- Thrombocythaemia occurs in 50% of cases.
- Differential diagnosis is with other causes of polycythaemia. These can be excluded by history, examination and blood tests, including bone marrow aspiration, ABGs and EPO levels.
- Genetic testing can reveal the *JAK2* mutation in 90–95% of patients with PV, and in 50% of patients with myelofibrosis.
- Therapy is aimed at maintaining a normal blood count by venesection and myelosuppression with drugs.
- Thrombosis is a potential cause of death, and 10% of cases develop myelofibrosis and rarely acute leukaemia.

Essential thrombocythaemia

Megakaryocyte proliferation and overproduction of platelets are the dominant features, with a sustained platelet count >450 × 10^9/L.

- Closely related to PV, with recurrent haemorrhage and thrombosis as the principal clinical features.
- Abnormal large platelets or megakaryocyte fragments may be seen on a blood film.
- Differential diagnosis is from other causes of a raised platelet count, e.g. haemorrhage, chronic infection, malignancy, PV, myelosclerosis and chronic granulocytic leukaemia.
- Platelet function tests are consistently abnormal.
- Hydroxycarbamide is the mainstay of therapy, but some treatments are more toxic.

Antiphospholipid syndrome

This is a rare, but increasingly recognised syndrome resulting in arterial or venous thrombosis or recurrent miscarriage, with a positive laboratory test for antiphospholipid antibody and/or lupus anticoagulant. It may present with another autoimmune disease such as SLE (2°) or as a 1° disease. The main feature of the disease is thrombosis, with a spectrum from subacute migraine and visual disturbances to accelerated cardiac failure and major CVE. Arterial thrombosis helps distinguish this from other hypercoagulable states. Paradoxically, the LA leads to a prolongation of coagulation tests, such as the APTT, but detailed testing is needed before the diagnosis can be confirmed. Patients may present for surgery because of complications (miscarriage, thrombosis) or for incidental procedures. Initially, patients are started on aspirin, but after a confirmed episode of thrombosis, they usually remain on lifelong warfarin. High risk of thrombosis in these patients means that if warfarin needs to be stopped for surgery, IV heparin should be commenced both pre- and postoperatively.

Anaesthesia and surgery in the hypercoagulable patient

There are no published guidelines, but it seems prudent that elective patients who are polycythaemic should be venesected to a normal blood count to decrease the risk of perioperative thrombosis.

- Antithrombotic stockings and intermittent compression devices should be used with SC heparin.
- Haematological advice may be required.

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Chapter 12

Neurological and muscular disorders

Adam Young, Selin Kabadayi, and Sarah Marsh

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Parkinson's disease

General considerations

Parkinson's disease (PD) is a multisystem neurological disorder characterised by a triad of muscle rigidity, bradykinesia and a resting tremor. Loss of dopaminergic neurones in the substantia nigra of the basal ganglia results in a dopamine deficiency and excessive thalamic inhibition. This leads to the classical symptoms seen. The disease affects over 100 000 people in the UK, with a prevalence of ~1% in those aged over 65, and is associated with an ↑ risk of perioperative morbidity and mortality. The disease is incurable and progress cannot be slowed; treatment is aimed at relieving symptoms with either medication or surgical intervention such as insertion of a deep brain stimulator.

Drug therapies

- The goal of drug therapy is to increase the amount of dopamine readily available within the CNS (Table 12.1) and to reduce cholinergic activity. This can be achieved either by administering a dopamine agonist or by inhibiting the enzymatic breakdown of dopamine in the CNS by monoamine oxidase B (MAO-B) or catechol-O-methyltransferase (COMT).
- Drug therapy is, however, limited by severe side effects (nausea and confusion), especially in the elderly. Up to 20% of patients will remain unresponsive to drug therapy.
- Dopamine is administered as a prodrug (such as levodopa) to allow it to cross the blood–brain barrier, and must be administered with a DOPA decarboxylase inhibitor (such as carbidopa) to prevent peripheral metabolism.
- Anticholinergic drugs are used when symptoms are mild and tremor predominates, and can be useful in drug-induced parkinsonism.

Table 12.1 Drug therapies in Parkinson's disease

Class of drug	Examples	Notes
Dopamine precursors in combination with DOPA decarboxylase inhibitor	Levodopa–carbidopa Levodopa–benserazide	Short half-life necessitates frequent administration
Dopamine agonists (enteral)	Pramipexole Ropinirole Bromocriptine	Adjunctive therapy; also used if levodopa therapies cause intolerable side effects
Dopamine agonists (parenteral)	Rotigotine Apomorphine	Transdermal patch SC infusion; highly emetogenic
MAO-B inhibitors	Selegiline Rasagiline	Avoid pethidine and selective serotonin reuptake inhibitor/TCA–risk of serotonin syndrome
COMT inhibitors	Entacapone Tolcapone	Caution with drugs metabolised by COMT (adrenaline, noradrenaline)
Anticholinergics	Procyclidine Benztropine	Limited efficacy; side effects (confusion, urinary retention) limit use

Preoperative assessment

Patients with PD have ↑ perioperative morbidity and mortality, as well as an ↑ length of stay.

In addition, the complexity of the timing and administration of drug therapies necessitates a multidisciplinary approach during the perioperative period, including from a PD specialist.

Important points:

- A history of dysphagia or excessive salivation (sialorrhoea) is suggestive of upper airway dysfunction and indicates an ↑ risk of failing to protect the airway during the perioperative period. This may manifest as aspiration, laryngospasm or postoperative pulmonary complications such as infection.
- Respiratory function may be further impaired by rigidity and bradykinesia, as well as sputum retention.
- Cardiovascular instability can result from arrhythmias or orthostatic hypotension 2° to the disease itself or as a side effect of medications.
- The management of medication administration must be clear and appropriate preparations made to be able to source and administer the drug; where enteral medications cannot be given, substitutive parenteral therapy must be instigated (see ➲ p. 292).
- The patient should be warned that, even with extensive planning, perioperative management of their parkinsonism may not be optimal and distressing symptoms may recur. Delirium is also common, with reported rates as high as 60%.

Drugs to avoid

- Several drugs commonly used in the perioperative period are contraindicated in PD as they can significantly worsen symptoms or cause harmful interactions.
- Antiemetics (prochlorperazine, droperidol, metoclopramide) antagonise dopamine and exacerbate symptoms.
- Pethidine interacts with selegiline which can lead to serotonin syndrome (see ➲ p. 332).
- Synthetic opioids cause presynaptic inhibition of dopamine and cause dose-related muscle rigidity.
- Clonidine can cause profound hypotension by enhancing the relative hypovolaemia.

Conduct of anaesthesia

- Medications for PD should continue uninterrupted until the start of anaesthesia. Distressing symptoms may develop as little as 3h after a missed dose and acute withdrawal of drugs may precipitate symptoms resembling neuroleptic malignant syndrome.
- Consider antisialagogue premedication in the presence of significant sialorrhoea such as glycopyrronium (200–400 micrograms IM).
- Patients with PD have a high incidence of dysphagia, gastroparesis and gastro-oesophageal reflux. Consideration therefore should be given to securing the airway with RSI and intubation.
- Fixed flexion deformity of the neck can occur.

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- Regional anaesthesia has advantages for those with a difficult airway or significant respiratory or cardiovascular complications. It also allows a rapid return to oral intake. However, muscle rigidity and tremor can interfere with positioning, surgery and monitoring and the sympathetic blockade can exacerbate autonomic dysfunction.
- Hypotension is common and can be difficult to treat.
- Domperidone, which does not cross the blood–brain barrier, may be used safely.
- If dysphagia will prevent administration of oral analgesia, an NGT may be required.
- Muscle rigidity may result in the patient being unable to operate patient-controlled analgesia.
- For prolonged procedures, administration of dopaminergic medications through an NGT may be necessary.

Postoperative care

- ⚠ The 1° consideration is restoration of dopaminergic therapy to prevent an exacerbation of symptoms or an acute withdrawal syndrome.

Withdrawal syndromes in Parkinson's disease

Parkinsonism–hyperpyrexia syndrome

This follows an abrupt withdrawal of levodopa and resembles neuroleptic malignant syndrome with rigidity, pyrexia, autonomic instability and impaired mental status. It is treated by reinstigating dopaminergic agents and has a high mortality if untreated.

Dopamine agonist withdrawal syndrome

This condition manifests with neuropsychiatric symptoms, including depression, fatigue, irritability, diaphoresis and orthostatic hypotension.

- Patients with advanced disease should be considered for admission to critical care due to the high risk of potential complications.
- If prolonged GI dysfunction is anticipated postoperatively (such as following emergency laparotomy), parenteral antiparkinson therapy must be instigated. This can be commenced relatively easily with the use of rotigotine patches or apomorphine infusions (more difficult to dose, however, and highly emetogenic).

Dosing of rotigotine

- Take advice from a specialist in the management of PD for conversion of enteral medications to transdermal rotigotine.
- In an emergency, or if specialist help is unavailable, dose equivalence calculators are available online at
  http://www.parkinsonscalculator.com

Special considerations

- Implantation of deep brain stimulators is increasingly being performed in patients with advanced disease. Should the patient then require further unrelated surgery, standard precautions for implantable devices should be used such as using bipolar diathermy. The device should be turned off prior to anaesthesia and checked postoperatively.
- Postoperative delirium is common in patients with PD and may be delayed. A thorough assessment should be made for potential triggers such as hypoxia or urinary retention. Non-pharmacological management, such as orientation measures, are preferable to pharmacological treatment. However, if required, low doses of benzodiazepines or quetiapine can be given. Dopamine antagonists such as haloperidol must be avoided.

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Cerebrovascular events

CVEs are a leading cause of death and disability worldwide. In the UK, 1.2 million people have a history of CVE. Around 85% of CVEs are ischaemic in nature, and 15% are haemorrhagic. The resulting clinical outcome can range from no deficit, a relatively mild focal neurological deficit to profound disability. Given the ongoing improvement in outcomes following CVEs, these patients are increasingly likely to present for surgery and, as such, require careful perioperative management.

⚠ The most important risk factor for a perioperative CVE is a previous CVE.

General considerations

Outcome following a perioperative CVE is poor and optimal 2° prevention measures are extremely important to maximise recovery. Perioperative hypotension can, for example, precipitate a watershed infarct at a vulnerable boundary between vascular territories.

When to operate following a CVE

- The risk of major adverse cardiovascular events is markedly ↑ following CVE. This risk decreases with time and plateaus after 9mo but remains elevated compared to a patient who has never suffered a CVE.[1]
- If possible, delay surgery for 9mo after a CVE, unless the benefits of earlier surgery outweigh the risks.
- TIAs require urgent investigation by a CVE physician and non-emergency surgery should be delayed until this is completed.
- Emergency surgery should not be delayed; surgery within 3d of a CVE may have a better outcome than surgery at d3–14 due to delayed impairment of cerebral autoregulation.[2]

Preoperative assessment

- Common associations with CVEs are older age, frailty, hypertension, diabetes, respiratory disease, and cardiac disease.
- Medication and lifestyle changes should all be encouraged, including: cessation of smoking, anticoagulation for valvular heart disease and AF, lipid-lowering medications, glycaemic control and healthy diet to normal BMI.
- For the symptomatic patient with known carotid artery stenosis of >60%, discussion with a vascular surgeon regarding consideration for endarterectomy may be appropriate.
- Physical disability may mean that impaired cardiorespiratory reserve is not immediately obvious.
- Neurological examination is necessary prior to anaesthesia so that any new perioperative neurological deficit can be identified early.
- Ischaemic CVEs are associated with AF. An ECG is required to determine the underlying cardiac rhythm. Rate control may be required.
- Assess for bulbar involvement. Tracheal intubation may be needed and the risk of postoperative pulmonary complications is raised.
- If patients are taking oral anticoagulation, INR, eGFR or CC will be needed to inform preoperative cessation of these drugs.
- Post-CVE epilepsy occurs in ~10% of patients. Antiepileptic drugs should be continued in the perioperative period.

Antiplatelet and anticoagulant therapy

- Aspirin and clopidogrel are the most commonly used antiplatelet drugs for 2° prevention of CVEs.
- Anticoagulant drugs, such as warfarin, or direct oral anticoagulants, such as apixaban, are used to reduce the risk of embolic CVEs, usually in the setting of AF.
- Withdrawal of antiplatelet and anticoagulant therapy is associated with an ↑ risk of perioperative CVE, thought to be due to rebound hypercoagulability aggravating the prothrombotic state that exists after surgery.[3] The ↑ risk of CVE must be balanced against the risks of ↑ surgical bleeding and the exclusion of central neuraxial techniques. This decision may require specialist physician, anaesthetic and surgical input and documented discussion with the patient.
- Ideally, antiplatelet and anticoagulant agents should be continued in procedures with a low risk of bleeding.

Conduct of anaesthesia

- Antihypertensive medication should be continued in the perioperative period, with the exception of ACE inhibitors and ARBs which should be withheld for 24h before and after surgery. These medications may cause profound, resistant hypotension, especially at induction.
- Contractures may make venous access and patient positioning challenging.
- Suxamethonium can cause hyperkalaemia in hemiplegic patients; this has been reported as early as 1w and as late as 6mo after CVE.
- Haemodynamic instability may occur due to a relatively fixed vascular system. Invasive arterial monitoring and careful use of vasoactive agents may be required.
- Anaesthetic technique does not appear to influence the risk of perioperative CVE, except in lower limb arthroplasty where neuraxial blockade may reduce the risk of death or further CVE.
- Intraoperative hypotension should be avoided in patients at risk of perioperative CVE. BP should be maintained at the patient's baseline. If a baseline BP is not available, the MAP should not be allowed to decrease below 80mmHg.[4]
- Hypotension and hypocapnia must be avoided to prevent the watershed area around the infarcted cerebral tissue from also becoming irreversibly damaged.
- Blood glucose should be maintained between 4 and 10mmol/L.
- Metoprolol should not be used to treat intraoperative hypertension—it is associated with an ↑ risk of perioperative CVE. Other β-blockers appear to be safe.
- New neurological signs postoperatively will require urgent imaging and discussion with the CVE team.

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Mechanical thrombectomy

- Mechanical thrombectomy or clot retrieval is an intervention that can remove an obstructing embolus from the affected vessel.
- A minimally invasive angiographic technique is used with a catheter passed via the groin or wrist to the site of the embolus.
- If performed promptly, the neurological deficit can be minimised or even reversed completely.
- The procedure can be carried out under GA or sedation. GA may be preferred if there is bulbar involvement, agitation or a reduced level of consciousness (e.g. basilar artery thrombus).
- Patient outcome correlates with BP management, not the anaesthetic technique. Hypotension is particularly poorly tolerated. Systolic pressure should be maintained at 140–180mmHg, with diastolic pressure <105mmHg.[5]

Postoperative care

- Reinstate anticoagulants as soon as the surgical team allows.
- Intraoperative BP targets should continue postoperatively.
- Consider critical care admission for comorbid patients or those with bulbar involvement.

Haemorrhagic CVE

- This may be due to aneurysmal subarachnoid haemorrhage, spontaneous intracerebral haemorrhage, rupture of an arteriovenous malformation, haemorrhage into a tumour or cerebral amyloid angiopathy.
- Hypertension is the commonest risk factor for spontaneous intracerebral haemorrhage.
- Other risk factors include DM, cigarette smoking and alcohol excess.
- Patients who survive the acute phase may be significantly impaired with weakness, contractures and/or cognitive deficits.
- BP following intracerebral haemorrhage should be maintained <140mmHg systolic.[6]
- When bleeding has occurred due to an aneurysmal rupture, systolic BP should be maintained between 110 and 160mmHg until the aneurysm is secured.[7]

(See also ➲ p. 576.)

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Epilepsy

Epilepsy is characterised by a predisposition of the brain to generate abnormal, synchonous, neuronal activity. The clinical manifestations vary widely and can be focal or generalised, with consciousness maintained or impaired. Epilepsy may be idiopathic or 2° to a structural, metabolic or traumatic cause. Antiepileptic drug therapy is used to reduce and/or control the rate of seizures. Surgery or implantation of neuromodulatory devices (such as a vagus nerve stimulator) may be required in refractory cases.

General considerations

- It is vital that antiepileptic drug therapy is maintained throughout the perioperative period, through either enteral or parenteral routes.

Preoperative assessment

- The nature, timing and frequency of seizures should be recorded, along with a full drug history and timing of antiepileptic drug therapy. This can help to determine strategies to minimise disruptions in treatment regimes.
- Any associated medical conditions must also be assessed.
- If there is suboptimal control of seizures or in those where return to therapy may be delayed after surgery, consult their specialist.
- Consider drug titres in patients as a baseline prior to major surgery or if control is suboptimal.

Conduct of anaesthesia

- Perioperatively antiepileptic drugs may need to be given, parenterally if possible
- Specific drug considerations are considered in Table 12.2.
- Check electrolyte and glucose levels as derangement reduces seizure threshold, as does hypocapnia.
- IV induction can be performed with propofol or thiopental as both have anticonvulsant properties at doses used for GA. Ketamine has proconvulsant properties when used at low doses, but anticonvulsant properties at GA doses.
- Regional anaesthesia may help to minimise disruption in antiepileptic drug regimes and can also negate the possibility of missed seizure activity occurring under GA. LAs can readily cross the blood–brain barrier and result in seizure activity if maximum doses are exceeded.
- Carefully assess any abnormal movements on induction or emergence as misdiagnosing dystonic movements or shivering as epilepsy can have profound implications for the patient (Box 12.1).

Box 12.1 Driving and epilepsy

A perioperative seizure in a patient holding a UK driver's licence can result in withdrawal of the licence for up to 1y. The impact of this on a patient's life cannot be overstated. Advice on fitness to drive is available from the Driver and Vehicle Licensing Agency (http://www.gov.uk/dvla).

Table 12.2 Drugs with special considerations in epilepsy

Drug	Notes
Pethidine and tramadol	Both lower the seizure threshold and should be avoided
Alfentanil	Enhances EEG activity and prolongs seizures in electroconvulsive therapy; avoid or use with caution
Ketamine	Proconvulsant at low doses, but anticonvulsant at anaesthetic doses (>1mg/kg IV)
Etomidate	Avoid as associated with postoperative seizures. Prolongs seizure duration in electroconvulsive therapy
Metoclopramide, prochlorperazine, droperidol	High incidence of dystonic reactions which may lead to confusion with epileptic activity
Enflurane	Associated with postoperative seizures (no longer available in the UK)
Aminosteroid neuromuscular blockers (e.g. vecuronium, rocuronium)	Hepatic enzyme induction by antiepileptic drugs may shorten duration of action

What if oral or nasogastric therapy is not possible?

Phenytoin, sodium valproate and levetiracetam can be given IV at equivalent doses to the oral formulation and carbamazepine can be given rectally. Hypoalbuminaemia, acid–base derangement and concomitant drug administration can markedly affect the antiepileptic drug levels. Advice from a pharmacist and measurement of drug levels in the critically ill is very helpful.

Postoperative care

- Ensure antiepileptic therapy is reinstated as soon as possible.
- See Table 12.3 for antiepileptic medication considerations.
- Abnormal movements in the postoperative period may not be due to epilepsy. Look for other causes of seizure/abnormal movement. The consequences of a postoperative seizure for some patients will be significant.

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Table 12.3 Antiepileptic medication considerations and interactions

Effect	Consequence
Enzyme inducers (e.g. carbamazepine, phenytoin, phenobarbital)	Reduce serum concentration of certain drugs, e.g. valproate, lamotrigine, steroids, oral contraceptives, amiodarone, digoxin, warfarin
Enzyme inhibitors (e.g. valproate and stiripentol)	Increase serum concentration of certain drugs, e.g. lamotrigine, phenobarbital, nimodipine, amitriptyline, warfarin
Metabolism inhibited by other drugs	Clarithromycin and erythromycin increase serum concentration of carbamazepine Amiodarone increases serum concentration of phenytoin May cause toxic levels of antiepileptic drugs
Metabolism induced by other drugs	Oral contraceptives markedly reduce serum lamotrigine concentrations, which may result in subtherapeutic levels
Pharmacokinetic effects on absorption, distribution or excretion	Continuous NG feed impairs gastric phenytoin absorption Highly protein-bound drugs (valproate, phenytoin) may displace other drugs from protein-binding sites—rarely of clinical importance
Effect on NMBAs	Antiepileptic drugs may potentiate the effect of non-depolarising neuromuscular blockers when administered acutely, yet cause resistance to their effects in chronic use. Caused by a combination of effects such as enzyme induction and upregulation of acetylcholine receptors
Impaired clearance of antiepileptic drugs	Renal/hepatic failure, hypoalbuminaemia

Further reading

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Anaesthesia in spinal cord lesions

In the UK, there are ~50 000 patients currently living with chronic spinal cord injuries. Around 1000 people sustain a new spinal cord injury in the UK each year, with trauma being the commonest aetiology. Non-traumatic causes include infection, vascular disruption and degenerative disease. The average age of patients suffering a spinal cord injury is increasing due to falls from standing height in the expanding elderly population. Complex physiological changes render anaesthetic management of these patients challenging in both the acute and chronic phase.

Phases of acute spinal cord injury

Injury to the spinal cord results in three physiologically distinct phases. (See also ➲ p. 1003.)

Hypertension

This starts almost immediately. There is massive catecholamine release due to direct stimulation of the spinal cord, which causes hypertension and tachycardia. This may result in myocardial ischaemia, cardiac failure and pulmonary oedema.

Neurogenic shock

This stage follows rapidly and lasts for several weeks; most commonly seen in lesions above T6. Disruption of autonomic pathways below the level of injury results in hypotension (due to loss of sympathetic tone) and bradycardia (if the cardiac accelerator fibres at T1–T4 are affected). Unopposed vagal tone may result in severe bradycardia or even asystole during procedures such as laryngoscopy and tracheal suctioning. The patient will be hypotensive, but peripherally warm and vasodilated; excessive fluid administration can result in pulmonary oedema and early use of vasopressors should be considered to maintain organ perfusion.

Spinal shock

This phase accompanies neurogenic shock and commences at the time of injury. It initially consists of flaccid areflexia with loss of sensation and motor paralysis. A four-phase course is described, with reflexes beginning to return 1d after injury and achieving a hyperreflexive, spastic state after ~1y. Autonomic dysreflexia is a life-threatening manifestation of this hyperreflexia (see below).

Primary and secondary injury

- 1° injury occurs at the time of the injury itself.
- 2° injury begins within minutes of the 1° injury and is due to spinal cord oedema and ischaemia. The extent of 2° injury can be modified by optimal management, e.g. stabilisation of affected vertebrae, avoidance of hypotension.
- Spinal cord injury without radiographic abnormality (SCIWORA) describes a neurological deficit without X-ray or CT evidence of injury to the spinal column. MRI will usually identify the lesion. Maintain a high index of suspicion if clinical features do not correlate with initial imaging.

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Anaesthetic management of acute spinal cord injury

Whether the patient is in the ED or theatre, a high index of suspicion should be maintained for coexisting life-threatening injuries, as these occur in approximately one-third of spinal cord injury patients. A patient who has suffered major trauma and is unconscious should be assumed to have a spinal cord injury until proven otherwise (see ➲ Spinal trauma, pp. 1002–3.

Airway

Manual in-line stabilisation is mandatory during laryngoscopy to prevent extension of the cervical spine from causing further damage. Videolaryngoscopy may result in less movement of the cervical spine than direct laryngoscopy. AFOI can also be considered. Coughing or gagging must be avoided.

Breathing

Ventilatory support may be required in cervical and high thoracic lesions. Table 12.4 describes the ventilatory impact of cervical injury depending on the level. In spinal cord injury where ventilation is dependent entirely on the diaphragm, the patient should remain supine or slightly head down. This increases VC as the abdominal contents push the diaphragm up into the chest, allowing greater excursion during inspiration. For neuroprotective reasons, normocapnia should be maintained and hypoxia avoided.

Circulation

In neurogenic shock, hypotension is due to sympathetic outflow disruption and subsequent vasodilation. Avoid excessive fluid resuscitation as this may precipitate pulmonary oedema; judicious use of vasopressors and vagolytic agents is recommended. Assuming no major haemorrhage is present, a neuroprotective MAP target of 85–90mmHg may help to optimise spinal cord perfusion and reduce 2° injury. Early urinary catheterisation is advised to prevent vagal stimulation caused by bladder distension and to allow accurate fluid balance calculations.

Disability

The extent of the spinal cord injury should be classified using the American Spinal Injury Association Impairment Scale. Neurological deficit often does not fit the patterns of spinal cord injury, as described in Table 12.5, in the acute setting. Note that spinal cord injury alone does not cause loss of consciousness and so intracranial pathology should be excluded. Maintenance of in-line immobilisation is mandatory until spinal cord injury can be excluded with clinical examination or radiological investigation.

Exposure

Ensure a full 2° survey is performed to identify any concurrent injuries. Be aware that there is a risk of hypothermia due to peripheral vasodilation.

Table 12.4 Respiratory effects by level of lesion

Above C3	Loss of phrenic nerve innervation results in diaphragmatic paralysis and complete dependence on mechanical ventilation
C3–C5	Variable diaphragmatic function remains; mechanical ventilation may be required, depending on extent of loss of function
C6–C8	Diaphragmatic function is intact and may be adequate to support inspiratory effort in combination with accessory neck muscles. Intermittent non-invasive ventilatory support may be required. Cough is markedly impaired, with consequent risk of sputum retention, atelectasis and pneumonia
Below C8	Minimal ventilatory compromise, but cough may be impaired with ↑ risk of sputum retention and pneumonia

Table 12.5 Patterns of spinal cord injury

Type	Injury pattern	Consequences
Complete	No intact neurones pass through the site of injury	Complete absence of motor and sensory function below the lesion
Incomplete		
Anterior cord syndrome	Usually due to disruption of the anterior spinal artery	Loss of motor function, pain and temperature sensation below the lesion Preservation of fine touch, proprioception and vibration
Brown-Séquard syndrome	Cord hemisection (e.g. trauma)	Ipsilateral loss of motor function, fine touch, proprioception and vibration below the lesion Contralateral loss of pain and temperature
Central cord syndrome	Injury to central grey matter of the cord, most commonly in the cervical spine	Greater motor impairment in upper limbs, compared to lower limbs
Posterior cord syndrome	Rare—can be due to infarction of posterior spinal arteries, trauma or disc compression	Loss of vibration, fine touch and proprioception below the lesion

Autonomic dysreflexia

Autonomic dysreflexia is a potentially life-threatening condition, characterised by an inappropriate autonomic response to a stimulus below the level of the spinal cord lesion. It is seen in up to 50–70% of patients with a spinal cord injury at or above T6. The incidence is greater with higher lesions and complete cord injury. It may occur as early as 3w following the initial injury.

Common precipitants include: pain, bladder distension, urinary tract infections, catheter insertion and faecal impaction.

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Activation of the sympathetic chain causes intense vasoconstriction and subsequent hypertension, compounded by loss of descending inhibitory control due to cord injury. Features include hypertension, headache, flushing, sweating and anxiety.
Life-threatening hypertension can cause cardiac emergencies, including myocardial ischaemia, arrhythmias and pulmonary oedema. Neurological complications are also possible, including seizures and intracranial haemorrhage.
Management involves determining the cause of the autonomic dysreflexia and managing the consequent hypertension:

- Exclude bladder distension and constipation, and search for trigger.
- Sublingual GTN or nifedipine 10mg, or slow IV hydralazine 20mg.
- If hypertension persists, IV GTN, phentolamine or magnesium may be used.

Chronic spinal cord injuries and anaesthesia

Chronic spinal cord injuries result in marked alterations of physiology.

- Reduced blood volume and anaemia
- Postural hypotension due to pooling of blood in the lower limbs resulting from lack of muscle pump and reduced sympathetic tone
- Spasticity and hyperreflexia causing contractures. Baclofen (γ-aminobutyric acid agonist) is the commonest medication used to treat contractures and can be administered intrathecally via an SC pump. This must be taken into account when applying a diathermy pad or performing neuraxial anaesthesia.

Suxamethonium

Following a spinal cord injury, upper motor neurone denervation results in nicotinic acetylcholine receptors propagating beyond the motor endplate of the neuromuscular junction. Suxamethonium administration can cause an elevated release of K^+ due to the ↑ number of receptors being depolarised. Life-threatening hyperkalaemia can occur. Suxamethonium is contraindicated from 72h to 6mo after injury.

Conduct of anaesthesia

- Patients are likely to have undergone multiple previous anaesthesia; review previous anaesthetic charts.
- If surgery is planned for an insensate region, autonomic dysreflexia may still be precipitated, even if pain is not. It may be possible to undergo the procedure without anaesthesia, but vigilance for the sequelae of autonomic dysreflexia must be maintained.
- Autonomic dysreflexia and IHD are common. Consider invasive arterial BP monitoring, especially if significant fluid shifts are anticipated.
- Laryngoscopy may be difficult if there has been cervical fixation.
- Spinals and epidurals are advantageous in reducing postoperative ventilation and autonomic dysreflexia. Less cardiovascular instability as sympathetic tone is already reduced. May be unreliable and can be difficult to determine the level of block achieved in complete spinal cord

lesions. A change in tone from spasticity to flaccid paralysis can indicate an effective block. Scoliosis and previous surgery can make spinal and epidural placement difficult.
- If upper limb surgery is being performed in a patient with impaired respiratory function, consider carefully the risks of a brachial plexus block. Phrenic nerve palsy may cause a dramatic deterioration in respiratory function.
- Consider RSI as gastric emptying is delayed, particularly with higher spinal cord injuries.
- Prolonged ventilation and a tracheostomy can result in respiratory complications such as altered lung dynamics and tracheal stenosis—ensure such issues are investigated prior to surgery.
- A proportion of patients with high cervical injuries require domiciliary ventilation via an uncuffed tracheostomy tube. Consider replacing this for a cuffed tube before ventilation.
- Any history of autonomic dysreflexia should be carefully explored.
- Reduced chest wall compliance due to intercostal muscle spasticity in cervical lesions results in hypoventilation and an abnormal response to hypercapnia. Sleep apnoea is more frequent.
- Anticholinergics may be required for any procedure that is likely to stimulate the vagus nerve (e.g. laryngoscopy, laparoscopy) if cardiac accelerator innervation is impaired (T1–4).
- Accelerated atherosclerotic cardiovascular disease is seen and may be asymptomatic due to the inability to stress the CVS. Have a high index of suspicion for cardiovascular complications.
- Impaired thermoregulation is common, therefore consider siting a temperature probe.
- Osteoporosis is common; take great care when moving patients. Ensure meticulous positioning; atrophic skin with poor cutaneous perfusion is at high risk for developing pressure areas.
- Over 50% of patients have chronic neuropathic pain and may be on long-term analgesics. A tailored perioperative analgesic regime is required involving acute and chronic pain teams.
- High risk of VTE; consider the need for mechanical and/or pharmacological thromboprophylaxis.

Postoperative care

- Patients with injuries at the cervical level should be recovered in a supine position to optimise respiratory function by maximising diaphragmatic excursion.
- Autonomic dysreflexia may occur due to pain from the surgical site or other common triggers.
- In cervical and upper thoracic lesions, respiratory function will be suboptimal and the risk of postoperative pulmonary complications is high. Consider elective postoperative respiratory support.

Obstetric anaesthesia

(See ➲ pp. 838–9.)

- Obstetric patients with chronic spinal cord injury have an ↑ risk of preterm delivery, thromboembolism, sepsis and CS. Assessment in an obstetric anaesthetic clinic is highly advisable, with a detailed plan made for management in both elective and emergency situations.

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- The effect of the gravid uterus on diaphragmatic excursion may greatly affect respiratory function in patients with high thoracic/cervical injuries. An increase in respiratory support may be required and regular measurement of VC should be performed.
- While a spinal can usually be sited, performing an epidural in an area of spinal fusion frequently can result in failure, a dural tap or an inadequate block. An alternative plan should be considered.
- Patients with a history of autonomic dysreflexia should be particularly closely monitored.
- Anaemia of chronic spinal cord injury may be compounded by pregnancy.
- Patients with lesions above T10 are unlikely to feel contractions. They can, however, be a trigger for autonomic dysreflexia. Epidural analgesia is the most effective way of preventing this and consideration should be made to siting this prior to induction of labour, especially as it may be challenging to insert and assess.
- Pre-eclampsia should always be considered as a differential diagnosis to suspected autonomic dysreflexia in these patients.
- Patients have exaggerated postural hypotension and tolerate aortocaval occlusion poorly. Great care must be taken with patient positioning.
- The contracted uterus may still act as a trigger for autonomic dysreflexia. If an epidural is present, consider leaving it *in situ* for up to 48h in case this occurs.
- Postpartum autotransfusion of blood from the contracted uterus may be sufficient to cause pulmonary oedema and respiratory failure in patients with cervical lesions.

Further reading

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Neuromuscular disorders

Neuromuscular disorders can be categorised into acquired or hereditary conditions, and the anatomical site affected (Table 12.6). Common conditions will be discussed in more detail.

Table 12.6 Categorisation of neuromuscular disorders

Acquired	Prejunctional	Motor neurone disease (amyotrophic lateral sclerosis, spinal muscular atrophy, spinal bulbar muscular atrophy), multiple sclerosis, Guillain–Barré syndrome, peripheral neuropathies
	Junctional	Myasthenia gravis, Lambert–Eaton myasthenic syndrome, congenital myasthenic syndrome
	Post-junctional	Inflammatory myopathies and critical illness polyneuropathy and myopathy
Hereditary	Prejunctional	Peripheral neuropathies, e.g. Charcot–Marie–Tooth, Friedreich's ataxia
	Post-junctional	Muscular dystrophies, e.g. Duchenne, Becker, limb-girdle, myotonic dystrophy, fascioscapulohumeral, Emery–Dreifuss Non-dystrophic myotonias: myotonia congenita, periodic paralysis, central core disease, myotonia fluctuans Metabolic and mitochondrial disorders, e.g. lactate dehydrogenase deficiency, mitochondrial myopathy

Neuromuscular conditions are a concern for the anaesthetist as many will have some of the features listed below:

- Respiratory: pharyngeal and respiratory muscle involvement results in respiratory insufficiency, aspiration risk and OSA. Difficult airways due to dysmorphic features that can be a feature of some diseases. Restrictive pulmonary picture due to scoliosis
- Cardiovascular: autonomic dysfunction, cardiomyopathy and conduction defects
- Metabolic: electrolye imbalance, rhabdomyolysis due to sustained muscle contraction, MH-like phenomena
- Pharmacological: sensitivity to muscle relaxants and life-threatening sequelae of suxamethonium administration.

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Motor neurone disease

Motor neurone diseases are a group of neurodegenerative disorders in which motor neurone degeneration leads to progressive muscle weakness. Most commonly sporadic, but they can also be hereditary and infection-related. The disease can affect purely lower motor neurones (progressive muscular atrophy), upper motor neurones (1° lateral sclerosis), mixed upper and lower neurones (amyotrophic lateral sclerosis) or variants restricted to upper/lower limbs or the bulbar region.

Amyotrophic lateral sclerosis

Amyotrophic lateral sclerosis is the commonest form of motor neurone disease. It initially presents with weakness, atrophy and fasciculation of peripheral muscles (usually those of the hands) and progresses to axial and bulbar weakness, including dysarthria and dysphagia. There is no sensory loss and cranial nerves are unaffected.

Death usually results from respiratory failure, with 50% of patients dying within 20mo of diagnosis. Management is focused on symptom relief and palliation, led by a multidisciplinary team. Complex end-of-life discussions are mandatory and interventions such as domiciliary non-invasive ventilation should be considered in totality. Around 50% of patients with motor neurone disease will experience changes in cognitive function, ranging from mild impairment to severe dementia, so forward planning is essential.

Conduct of anaesthesia

- Consider PFTs prior to surgery.
- Autonomic dysfunction can lead to significant hypotension during induction, positive pressure ventilation and postural changes.
- Denervation of muscle leads to atrophy and development of extrajunctional acetylcholine receptors. Suxamethonium should therefore be avoided as it may cause life-threatening hyperkalaemia.
- NMBAs should be used at ↓ doses due to ↑ sensitivity.
- Respiratory complications are common, including ↑ risk of postoperative ventilation, weaning difficulties, infection and atelectasis. Those with bulbar involvement are at particular risk and elective critical care admission should be considered.
- Consider regional anaesthesia techniques where possible, but minimise the dose of LA to prevent nerve toxicity.
- Baclofen is sometimes used for spasticity and should not be abruptly withdrawn due to precipitation of MH-like crisis.

Further reading

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Multiple sclerosis

Multiple sclerosis is an autoimmune disease of the CNS characterised by the development of inflammatory plaques, demyelination and axonal damage to the brain and spinal cord. Onset usually occurs in early adulthood; the disease may relapse and remit or follow a chronic progressive course. Treatment aims are to limit progression and manage symptoms.

General considerations

- Patients are frequently on multiple medications. Immunosuppressants to manage disease progression (e.g. steroids, interferon beta), analgesics for neuropathic pain and antispasmodics. Perioperative management and potential interactions must be considered.
- Symptoms range from isolated visual disturbances to severe weakness and typically recur and relapse. Profound spasticity, respiratory failure and bulbar palsy can occur in end-stage disease.
- Demyelinated axons are sensitive to heat; an increase in temperature may cause a marked deterioration in symptoms.

Preoperative assessment and investigation

- A documented, detailed preoperative neurological examination is essential, especially if a regional technique is planned.
- Assess the current state of the disease and consider delaying elective surgery if the patient is actively relapsing.
- Review any change in respiratory function. Bulbar palsy causes an ↑ risk of aspiration and reduced airway reflexes in the postoperative period. Critical care may be required.

Conduct of anaesthesia

- GA itself does not affect the course of multiple sclerosis.
- Demyelinated axons are more susceptible to LA toxicity. Use the lowest possible dose of LA, in combination with adjuncts such as low-dose opioids.
- Peripartum use of spinal or epidural techniques does not appear to affect the occurrence of a postpartum relapse. Prior full neurological examination and discussion with the patient are required to prevent the patient from incorrectly attributing a postpartum relapse to anaesthesia.
- Suxamethonium should be avoided when severe weakness and spasticity are present as it may result in life-threatening hyperkalaemia.
- Response to non-depolarising drugs is normal.
- Autonomic dysfunction can lead to haemodynamic instability.
- Pyrexia should be avoided, if possible, and treated aggressively with cooling measures.

Further reading

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ALGRAWANY

Guillain–Barré syndrome

Guillain–Barré syndrome is a rare disease (1:100 000) forming part of a group of neuropathic conditions characterised by progressive weakness and absent reflexes. The most common presentation is an acute inflammatory demyelinating polyneuropathy presenting with progressive motor weakness. Less common subtypes or variants are listed in Table 12.7. The anaesthetic implications related to NDMRs persist after recovery of symptoms and therefore they should be used with caution in patients with a history of Guillain–Barré syndrome.

Guillain–Barré syndrome often occurs as an autoimmune response following a viral or bacterial illness within the preceding month. Weakness is classically ascending and symmetrical, with associated areflexia. Sensory and autonomic dysfunction can also occur. Severity can range from mild to severe debilitation. Diagnosis is based on clinical features, CSF testing (high protein, low WCC) and nerve conduction studies.

In patients with Guillain–Barré syndrome:

- ~25% will require intubation and mechanical ventilation
- ~10% will die from associated complications
- ~10% will suffer long-term neurological complications and physical dependence.

Treatment is generally supportive, however, regardless of subtype. Specific interventions such as immunoglobulins or plasmapheresis may be used to reduce neural inflammation and expedite recovery. Steroids are not indicated.

Table 12.7 Guillain–Barré syndrome variants

Miller–Fisher syndrome	Rare. Ataxia, ophthalmoplegia, bulbar and facial weakness and absent reflexes. Specific antibodies often identified. Good prognosis
Acute motor axonal neuropathy (AMAN)	Strongly associated with *Campylobacter jejuni* and sometimes *Haemophilus influenzae* infections; more common in summer and in younger patients. Acute motor weakness with sensory preservation. Axonal degeneration with no demyelination. Specific antibodies can be identified
Acute motor-sensory axonal neuropathy (AMSAN)	Similar to AMAN, but patients present with rapid and severe motor and sensory dysfunction with muscle wasting. Recovery probably poorer than with AMAN

General considerations

The main systemic effects of Guillain–Barré syndrome and the subsequent anaesthetic implications are summarised in Table 12.8.

Table 12.8 Systemic effects of Guillain–Barré syndrome and anaesthetic implications

Cardiovascular	
Autonomic dysfunction	Over- or underactivity of both sympathetic and parasympathetic nervous systems: • Tachyarrhythmias, AV block, prolonged QT and asystole • Haemodynamic instability during induction, positive pressure ventilation or postural changes • Ileus, urinary retention and abnormal sweating Consider invasive cardiovascular monitoring and large-bore IV access
Respiratory	
Weakness of respiratory muscles	PFTs preoperatively Risk of aspiration—consider RSI Difficulty clearing secretions: risk of pulmonary infection Difficulty weaning from ventilatory support: postoperative ventilation ± tracheostomy
Neurological	
Bulbar and facial palsy	↑ risk of aspiration and upper airway collapse
Neuropathic pain	Difficult perioperative analgesic management—multimodal approach recommended
Neurological deficits	Document any pre-existing deficit if performing regional anaesthesia

Specific anaesthetic considerations

The 1° goals of anaesthesia in patients with Guillain–Barré syndrome are to:

- Minimise aspiration risk
- Maximise respiratory function
- Maintain haemodynamic stability
- Avoid depolarising NMBAs; there is upregulation of acetylcholine receptors at the neuromuscular junction, which can persist for a prolonged period after recovery. Administration of suxamethonium risks precipitating hyperkalaemia and subsequent life-threatening arrhythmias. These patients also have ↑ sensitivity to NDMRs.

Further reading

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Myasthenia gravis

Myasthenia gravis is an autoimmune condition characterised by muscle weakness and fatigability on repetitive exertion. It is caused by antibodies acting at the nicotinic acetylcholine receptor on the postsynaptic membrane of the neuromuscular junction. Fewer functioning receptors are then available for acetylcholine to bind to, resulting in impaired generation of action potentials and subsequent muscle weakness. Disease onset shows a bimodal distribution, primarily in younger women and older men. Thymus hyperplasia may have a role.

Symptoms range from mild ptosis to life-threatening bulbar and respiratory insufficiency. Weakness illustrates fatigability. Ptosis is often the 1st sign. Diagnosis is made with blood tests for acetylcholine receptor antibodies, edrophonium administration or the ice-pack test. The mainstay of chronic management is oral anticholinesterase medication. Some patients may require immunosuppressant therapy with steroids, plasma exchange or IV immunoglobulins. A myasthenic crisis is an exacerbation of the disease that necessitates mechanical ventilation due to respiratory or bulbar impairment.

General considerations

- Optimal management of the disease in the perioperative period is imperative. Involvement of a neurologist may help symptom control and to reduce the risk of a myasthenic crisis.
- Patients who have recently undergone plasmapheresis will have depleted plasma-esterase levels, prolonging the effect of suxamethonium, mivacurium, ester-linked LAs and remifentanil.

Preoperative assessment

- Take a full drug history and determine the effect of a missed dose of anticholinesterase (missing a dose may precipitate severe symptoms). Plan how anticholinesterase therapy will be administered in the perioperative period (Table 12.9).
- Assess weakness and the duration and progression of symptoms. Isolated, long-standing ocular symptoms are less likely to progress.
- Assess bulbar and respiratory function. Intubation and ventilatory support may be required.

Table 12.9 Adult treatment for myasthenia gravis

Drug	Dose	Notes
Pyridostigmine PO	30–120mg 4- to 6-hourly. Max 450mg/24h	Gradual onset and offset. Reduced severity of side effects. No parenteral preparation available 30mg of PO pyridostigmine = 1mg of IV neostigmine
Neostigmine SC/IM	1–2.5mg 2- to 4-hourly. Max 20mg/24h	Anticholinergic agents required to avoid marked bradycardia
Neostigmine PO	15–30mg 2-hourly. Max tolerable dose 180mg	↑ GI side effects and shorter duration of action than pyridostigmine

Conduct of anaesthesia

- Maintain anticholinesterase therapy up to the time of induction.
- Avoid premedication with agents that may worsen symptoms of myasthenia or cause sedation.
- Intubate if FVC <15mL/kg.
- Use short-acting anaesthetic agents to minimise respiratory depression on emergence.
- LA or regional anaesthesia should be used where possible.
- Patients are resistant to suxamethonium. It can be used, but an ↑ dose is required (1.5–2.0mg/kg). Do not administer any other neuromuscular blocker until no fade is present on quantitative nerve monitoring.
- Non-depolarising drugs: doses of 10–20% normal are often adequate. Monitor response with a quantitative nerve stimulator.
- Use short-acting non-depolarising relaxants that are metabolised spontaneously or reversed by sugammadex.
- Neostigmine use may trigger a cholinergic crisis, especially if the patient is taking other anticholinergic medications.
- Avoid ester-linked LAs, such as prilocaine, as anticholinesterase therapy may interfere with metabolism.
- Aminoglycosides, macrolides and fluoroquinolones all have neuromuscular-blocking activity.
- Magnesium reduces presynaptic acetylcholine release at the neuromuscular junction.
- IV lidocaine infusions may cause weakness.
- Steroids may worsen weakness in high doses.
- Ca^{2+} channel and β-blockers, gabapentinoids, phenothiazines and phenytoin can all affect neuromuscular function and care should be taken.
- Use multimodal analgesia to minimise the use of opioids.
- Extubate only if neuromuscular function is adequate.
- Consider admission to critical care for ongoing respiratory support.

Postoperative care

- Reinstitution of drug therapy is vital. An NGT may be required if bulbar symptoms prevent swallowing.
- In the event of GI failure, instigate parenteral therapy and seek expert opinion from a neurologist.
- Close monitoring of respiratory function is mandatory, ideally with serial VC measurements.
- Blood gases and SpO_2 may be normal up to the point of respiratory failure.

Predictors for postoperative ventilation

- Preoperative bulbar symptoms
- Previous myasthenic crisis
- Duration of disease >6y
- Coexisting chronic respiratory disease
- Pyridostigmine dose >750mg/d
- Preoperative VC <2.9L
- Intraoperative blood loss >1000mL
- Major body cavity surgery
- Serum acetylcholine receptor antibody >100nmol/L
- Pronounced decremental response on repetitive nerve stimulation

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Special considerations

Myasthenic crisis vs cholinergic crisis

- Myasthenic crisis occurs when a patient with myasthenia receives insufficient medication and a cholinergic crisis occurs when the patient receives too much anticholinesterase therapy. They have similar presentations; both may cause paralysis, bronchospasm, respiratory failure and diaphoresis.
- Can be distinguished with a small dose of edrophonium which improves a myasthenic crisis, but this can precipitate respiratory failure in cholinergic crises.
- In a cholinergic crisis (e.g. organophosphate poisoning), too much acetylcholine at the nicotinic receptor produces flaccid paralysis and parasympathetic effects from the muscarinic receptors such as salivation, lacrimation, and GI upset. Treatment is IV atropine or glycopyrronium bromide to counteract the muscarinic effects.
- Myasthenic crises can also be triggered by trauma, infection or metabolic disturbances and can result in bulbar and respiratory muscle weakness which improves with anticholinesterase treatment.

Thymectomy

- Current evidence favours thymectomy in myasthenic patients; clinical outcome is improved with a reduction in myasthenic crises and the need for immunosuppressive therapy.
- Trans-sternal, transcervical or thoracoscopic approaches can be used.
- Anaesthetic management for thymectomy follows the general principles outlined above.

Lambert–Eaton myasthenic syndrome

Lambert–Eaton myasthenic syndrome is a rare autoimmune disorder that causes proximal muscle weakness. It is associated with malignancy, particularly small cell lung cancer.

- Antibodies to voltage-gated Ca^{2+} channels impair acetylcholine release from the presynaptic junction into the synaptic cleft.
- Unlike in myasthenia, muscle weakness is improved by exercise and anticholinesterase medication is less effective.
- Amifampridine is a treatment that enhances acetylcholine release at the neuromuscular junction.
- Autonomic dysfunction and bulbar involvement may occur in advanced disease.
- Patients with Lambert–Eaton myasthenic syndrome are extremely sensitive to both depolarising and non-depolarising NMBAs. These should be used with caution and in reduced doses.
- Postoperative issues are similar to those in patients with myasthenia gravis.

Further reading

Wolfe GI, Kaminski HJ, Aban IB, *et al*. (2016). Randomized trial of thymectomy in myasthenia gravis. *N Engl J Med*, **375**, 511–22.

Muscular dystrophies

Muscular dystrophies are a collective group of progressive muscle disorders, most often caused by defective or absent glycoproteins in the muscle membrane such as dystrophin. There are >30 forms of muscular dystrophies. Inheritance follows one of three patterns:

- **X-linked recessive**: Duchenne muscular dystrophy, Becker muscular dystrophy, scapuloperoneal muscular dystrophy
- **Autosomal recessive**: most 'limb girdle' types, scapulohumeral muscular dystrophy, congenital muscular dystrophy, childhood muscular dystrophy
- **Autosomal dominant**: facioscapulohumeral muscular dystrophy, some oculopharyngeal/ocular muscle dystrophies.

Duchenne muscular dystrophy and myotonic dystrophy are discussed below. Table 12.10 summarises features of some of the other muscular dystrophies.

Duchenne muscular dystrophy

Duchenne muscular dystrophy is the most common muscular dystrophy (incidence of 1:5000 ♂ newborns) and is rapidly progressive. It is caused by an alteration in the gene for dystrophin, a cytoskeletal protein that contributes to the strength, stability and functionality of myofibrils. This leads to disruption of the integrity of the sarcolemma and, consequently, myofibril atrophy, necrosis and fibrosis. Although primarily an X-linked condition affecting ♂, some ♀ carriers are symptomatic, exhibiting a milder phenotype.

General considerations

- Muscle weakness typically manifests between the ages of 2 and 5.
- It is characterised by a waddling gait, asymmetrical lower limb strength, delayed motor milestones, calf hypertrophy and falls.
- Proximal muscles are involved before distal, and lower limb extremities are affected before upper limb.
- Affected ♂ are usually wheelchair-bound by the age of 12 and suffer from concomitant scoliosis and contractures. Death occurs from cardiac or respiratory failure typically in 20–30s.
- Key aspects of management are physiotherapy and treatment with glucocorticoids to slow progression of muscular weakness.
- MD patients may test positive for MH, but this can be a false positive. Treat as MH susceptible (see ➲ pp. 1096–8).

Physiological considerations

- **Cardiovascular**: dilated cardiomyopathy (up to 50% of those aged 15 or over will have dilated cardiomyopathy), myocardial degeneration, heart failure and arrhythmias (AF/atrial flutter, ventricular tachycardia (VT) or VF). Signs and symptoms of heart failure in non-ambulatory individuals are often subtle.
- **Respiratory**: progressive respiratory muscle weakness, mucus plugging, atelectasis, pneumonia and restrictive respiratory failure. Management includes physiotherapy with assisted coughing, assisted ventilation, subsequent daytime ventilation and tracheostomy.
- **Musculoskeletal**: severe kyphoscoliosis, muscular contractures and osteoporosis (worsened by glucocorticoid use).

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- **Haematological**: vascular smooth muscle and platelet dysfunction can result in ↑ blood loss.
- **GI**: oesophageal dysmotility, delayed gastric emptying causing reflux.

Preoperative assessment and investigations

- Multidisciplinary team involvement is paramount prior to any surgical intervention.
- Specific perioperative management is based on the recommendations of an international body of experts in the management of Duchenne muscular dystrophy (for Further reading, see ➲ p. 320).
- Respiratory: physician review, comprehensive spirometry, measurement of O_2 saturation and assessment of blood or end-tidal carbon dioxide ($ETCO_2$) level if saturations are <95% on air are recommended preoperatively. Preoperative training in the use of assisted cough techniques (if baseline peak cough flow <270L/min) and non-invasive ventilation are advised (necessary if baseline FVC <30% predicted, strongly recommended if FVC <50% predicted).
- Cardiovascular: ECG, echocardiography and cardiology review. Patients may be on antiarrhythmic medications or have implanted cardiac devices.
- CK is often elevated, but a baseline level is useful in case of perioperative complications such as rhabdomyolysis.

Conduct of anaesthesia

- Volatile agents and suxamethonium use in Duchenne muscular dystrophy have been implicated in 'anaesthesia-related rhabdomyolysis' (severe rhabdomyolysis with hyperkalaemic cardiac arrest). These drugs should therefore be avoided. The condition produces signs similar to, but distinct from, those of MH.
- If volatile anaesthesia is used for induction, it should be discontinued as soon as possible, with conversion to TIVA.
- Non-depolarising neuromuscular blockers are safe, although reduced doses are required. Suxamethonium should be avoided.
- There is a risk of haemodynamic instability due to cardiac disease, especially in major surgery. Consider invasive arterial monitoring.
- Regional analgesia can minimise opioid use and potential postoperative respiratory complications.
- Patients on glucocorticoids may require perioperative stress dosing.
- Consider extubating patients directly onto non-invasive ventilation if their FVC is <50% predicted (and especially if <30% predicted). Ideally, extubation should be delayed until the secretion load is minimal and O_2 saturations are at baseline on air.
- Assisted cough techniques should be used postoperatively in patients with baseline peak cough flow <270L/min to reduce the risk of retained secretions and pneumonia.

Myotonic dystrophy

Myotonic dystrophy is an autosomal dominant disorder that usually presents in the 2nd or 3rd decade of life. It exhibits symptoms and signs of both dystrophies and myotonias. Abnormal Na^+ or chloride channels in the musculature result in the discharge of repetitive action potentials, leading to sustained muscle contraction in response to stimulation. The disease is progressive and has multisystem effects. Life expectancy depends on the subtype of disease and may be normal or reduced.

General considerations

Clinical features are highly variable but include:

- Degradation of cardiac conduction system leading to dysrhythmias and AV block. Mitral valve prolapse and cardiomyopathy possible
- Respiratory insufficiency due to atrophy of respiratory muscles and diaphragm, impaired cough, OSA and central respiratory depression
- Muscular atrophy, particularly facial and peripheral muscles
- Dysphagia, dysmotility, reduced gastric emptying and bulbar palsy—↑ risk of aspiration
- Possible cognitive impairment after 2nd decade of life
- DM, hypothyroidism, adrenal insufficiency and gonadal atrophy
- Pregnancy may aggravate the disease, and an elective CS is often required due to uterine muscle dysfunction.

Preoperative assessment

- Assess respiratory function with spirometry; consider CXR and arterial blood gases if significant impairment suspected.
- ECG ± echocardiography to look for dysrhythmias and cardiac dysfunction. An implantable pacemaker may be *in situ*.
- Serum thyroid function tests, glucose and cortisol measurements.

Conduct of anaesthesia

⚠ **It is vitally important to avoid factors that may precipitate sustained muscular contraction.**

These include: hypothermia, drugs, shivering, mechanical or electrical muscle stimulation including diathermy and NMBA monitoring.

- Premedication with a prokinetic and/or antacid is helpful as patients are at risk of aspiration; RSI may be required.
- Patients are sensitive to sedatives, so use sparingly.
- Avoid suxamethonium as it may cause sustained muscle contractures, rhabdomyolysis and hyperkalaemia. Rocuronium allows use of sugammadex for reversal, allowing complete reversal of NMBA and avoiding the risk of drug-induced muscle contraction. Atracurium can also be used. Do not use nerve stimulator or anticholinesterase as these may provoke muscle contraction.
- Inhalational anaesthetic agents have been implicated in rhabdomyolysis and MH-like reactions. Consider TIVA. If using inhalational maintenance, avoid high concentrations which may affect cardiac contractility and conduction.
- Induction agents cause profound CVS depression.

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- Sustained muscular contraction may make intubation, ventilation and subsequent surgery difficult, and are not classically responsive to NMB. Quinine and phenytoin have been reported to attenuate problematic sustained contractions, as well as direct infiltration of the muscle with LA.
- Invasive arterial monitoring should be considered in patients with cardiovascular impairment. Facilities should be available for transcutaneous pacing in case of significant bradyarrhythmia.
- Consider regional anaesthesia or analgesia to minimise the use of sedatives and opioids. Note that this does not prevent muscle contraction in the area of block, however.
- Opioids may cause sustained muscle contraction.
- Avoid hypothermia; warm theatre and fluids, and use forced air warmers, even during short procedures.

Postoperative care

- Consider HDU/ICU for anything other than minor surgery.
- Chest physiotherapy is vital.
- Prolonged postoperative ileus can occur.

Further reading

Birnkrant DJ, Bushby K, Bann CM, *et al*.; the DMD Care Considerations Working Group (2018). Diagnosis and management of Duchenne muscular dystrophy, part 2: respiratory, cardiac, bone health, and orthopaedic management. *Lancet Neurol*, **17**, 347–61.

Schieren M, Defosse J, Böhmer A, *et al*. (2017). Anaesthetic management of patients with myopathies. *Eur J Anaesthesiol*, **34**, 641–9.

Birnkrant DJ, Panitch HB, Benditt JO, *et al*. (2007). American College of Chest Physicians Consensus Statement on the respiratory and related management of patients with Duchenne muscular dystrophy undergoing anaesthesia or sedation. *Chest*, **132**, 1977–86.

Table 12.10 Summary of features of some muscular dystrophies

	Usual age at diagnosis	Most affected muscles	Progression	Respiratory	Cardiac	Anaesthetic notes
Duchenne muscular dystrophy	2–5y ♂ only	Arms, legs and spine (loss of gait by 10y)	20–30y	Respiratory and laryngeal muscles involved	Conduction abnormalities, arrhythmias, dilated cardio myopathy and heart failure are possible with all muscular dystro phies. Look for PPM	As for myotonic dystrophy (see p. 319)
Becker muscular dystrophy (partial dystrophin deficit)	Late childhood to early adulthood	Arms, legs and pelvis	30–60y			
Myotonic dystrophy	10–40y	Contractures, particularly distal, spinal and respiratory	Slow—type 1 more severe	Respiratory and pha ryngeal weakness		
Limb-girdle muscular dystrophy	Late childhood to early adulthood	Proximal muscle weakness	Moderate, life expec tancy reduced to middle age			
Emery–Dreifuss dystrophy	Childhood	Joint contractures (Achilles, elbow, spine)	Moderate, life expec tancy reduced to middle age	Difficult intubation and regional techni ques		
Congenital muscular dystrophies	Birth	Muscle weakness, hypotony and contractures	Variable	Severe restrictive lung disease		

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Rare conditions

There are many rare neurological conditions that require a considered approach to anaesthesia. A brief discussion of several key conditions is outlined below.

Creutzfeldt–Jakob disease

This is a neurodegenerative, prion-related disease that may be sporadic, inherited or transmitted through contaminated surgical instruments, transfused human tissue or dietary exposure to the bovine form of the disease. There is a rapid cognitive and neurological decline over months, leading to death. Anaesthetic considerations are based on the severity of neurological impairment. While there are no particular isolation precautions required, airway equipment must be single-use only and surgical instruments should be disposed of following the procedure, as autoclaving does not destroy transmissible prions.

Charcot–Marie–Tooth

A condition characterised by chronic peripheral neuromuscular denervation. Most forms are inherited in an autosomal dominant pattern. Muscle atrophy results in spinal and limb deformities; restrictive lung disease, difficult airway management and postoperative respiratory failure may result. Avoid suxamethonium due to the risk of severe hyperkalaemia. NDMRs may have a prolonged action; consider using a reversible agent.

Critical illness polyneuromyopathy

Critical illness polyneuromyopathy describes an overlapping syndrome of diffuse, symmetrical, flaccid muscle weakness that occurs in critically ill patients and can contribute to failure to wean from mechanical ventilation. There is no specific treatment other than best supportive care, treating the underlying condition and minimising risk factors during critical illness. There are no specific concerns regarding the conduct of anaesthesia. Suxamethonium should be avoided.

Familial periodic paralysis

Hyperkalaemic periodic paralysis

An autosomal dominant condition in which episodes of flaccid paralysis are triggered by hyperkalaemia and stress states such as cold and hunger. Loop diuretics can be used for preoperative K^+ reduction. Avoid medications that increase serum K^+ such as suxamethonium. Volatile agents are safe to use. Physiological stress should be avoided by maintaining normothermia, minimising fasting and infusing glucose-containing fluids during surgery. Assess for paralysis on emergence from anaesthesia; patients may remain paralysed for a prolonged period postoperatively and require mechanical ventilation.

Hypokalaemic periodic paralysis

This is an autosomal dominant condition. Patients present with severe muscle weakness 2° to hypokalaemia. The focus of treatment is on maintaining a normal serum K^+ level and avoiding hypothermia. Paralysis should be assessed for on emergence from anaesthesia.

There is a theoretical link between hypokalaemic periodic paralysis and MH as both conditions are related to an abnormality in the gene for the dihydropyridine receptor. Patients have been reported to exhibit hypermetabolic states following administration of drugs that trigger MH. Consider therefore avoiding the use of volatile agents. Suxamethonium should be avoided in all cases.

Friedreich's ataxia

An autosomal recessive ataxia characterised by skeletal muscle weakness and progressive limb ataxia. Cardiac involvement usually leads to death from cardiac failure. Avoid suxamethonium due to the risk of marked hyperkalaemia. Diaphragmatic involvement may result in the need for postoperative ventilation. Haemodynamic instability can occur due to myocardial involvement; consider invasive arterial BP monitoring.

Further reading

Marsh S, Pittard A (2011). Neuromuscular disorders and anaesthesia. Part 2: specific neuromuscular disorders. *BJA Educ*, **11**, 119–23.

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Drug considerations in neurological disorders

(See Table 12.11.)

Table 12.11 Drug considerations in specific neurological disorders

Disorder	Suxamethonium	Non-depolarising muscle relaxant	Anticholinergics	Comments
Motor neurone disease	!	↓		Respiratory compromise
Multiple sclerosis		N		Avoid hyperthermia
Parkinson's disease	N	N	Caution	Avoid drugs that worsen parkinsonism
CVE (hemiplegia)	!	N		Avoid hypotension and hypocapnia
Spinal cord injury	!	N		Observe for autonomic dysreflexia
Epilepsy	N	N/↑		Avoid drugs that reduce seizure threshold. Hepatic enzyme induction may reduce duration of NDMRs
Guillain–Barré syndrome	!	↓		Autonomic instability
Myasthenia gravis	↑	↓	!	Avoid drugs interfering with neuromuscular transmission

Disorder	Suxamethonium	Non-depolarising muscle relaxant	Anticholinergics	Comments
Lambert–Eaton syndrome	↓	↓		Autonomic dysfunction
Myotonic dystrophy	❗	↓	❗	Avoid cold, shivering and mechanical and electrical stimulation
Muscular dystrophy	❗	↓		Respiratory and cardiac compromise
Friedreich's ataxia	↓	N		Avoid negative inotropes
Charcot–Marie–Tooth	❗	↓		↓ thiopental dose
Hyperkalaemic periodic paralysis	❗	↓		Avoid K^+, avoid prolonged fasting
Hypokalaemic periodic paralysis	❗	↓		Maintain normokalaemia and normothermia
Creutzfeldt–Jakob disease	N	N		Dispose of airway and surgical instruments following case
Critical illness poly neuromyopathy	❗	↓		Consider underlying critical illness

❗, must avoid; ↑, give higher dose; ↓, give reduced dose; N, normal dose.

Source: data from Marsh, S; Pittard, A; (2011). Neuromuscular disorders and anaesthesia. Part 2: specific neuromuscular disorders. *BJA Education* **11**(4): 119–123.

Chapter 13

Psychiatric disorders

Aidan O'Donnell

Psychiatric disorders *328*
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See also

➲ Long-term opioid use and the opioid-dependent patient pp. *1176–8*

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Psychiatric disorders

The anaesthetic implications of psychiatric illness include:

- Where capacity of the patient to give informed consent may be impaired (e.g. dementia, mania, psychosis)
- Where the psychiatric illness itself also causes physical illness (e.g. anorexia nervosa)
- Where the psychiatric medication may interact with anaesthetic drugs and techniques (e.g. antidepressants).

Some form of psychiatric illness is present in about 10% of the UK population at any time, but most patients are usually well-controlled. The commonest psychiatric disorder is depression. Many patients are on long-term drug therapy, which should be continued perioperatively where possible.

Major psychiatric illness affects about 1% of the population and carries a significant risk of self-harm or suicide. Misuse of alcohol and drugs is also common among the psychiatric population. The stress of hospitalisation for surgery may exacerbate coexisting psychiatric problems.

Consent

The ability to give consent is assumed unless proven otherwise, as laid out in the Mental Capacity Act (England and Wales, 2005) and the Adults with Incapacity Act (Scotland, 2000). Patients who are detained under the Mental Health Act (1983) may be compelled to accept psychiatric treatment (but not other types of treatment) under the terms of the Act. In the UK and other countries, someone with 'Lasting Power of Attorney', previously Enduring Power of Attorney, may legally give (or withhold) consent on behalf of a patient who has been deemed to lack capacity. Check for an advanced decision/directive ('living will') as this may supersede or limit the powers of the Lasting Power of Attorney.[1]

Anxiety

Preoperative anxiety is common and can be managed with explanation, reassurance, oral premedication, IV sedation (e.g. midazolam 1–2mg) or a combination of these. Anxiety disorder may be acute or chronic, or occur as part of other disorders (e.g. depression). Extreme agitation may make cannulation difficult. Patients may hyperventilate. Higher doses of induction agents may be required.

Attention-deficit hyperactivity disorder (ADHD)

This condition is present in 1.4–3% of children; more common in boys. Behavioural problems may make children uncooperative at induction, and disrupted behaviour can be worse for a few days afterward. Children with severe ADHD are often prescribed methylphenidate, an amphetamine-like drug, which could theoretically cause them to require ↑ doses of anaesthetics. In practice, anaesthesia in this group of children is manageable without special precautions, and their stability is helped by continuing their usual medications[2] (see also ➲ p. 914).

Dementia

Dementia refers to an irreversible global deterioration in higher mental functioning. Fifty per cent of cases are due to Alzheimer's disease.[3]

- Prevalence: 1% aged 65–74, rising to 10% aged >75 and 25% aged >85y. Slightly more common in women.
- Mean life expectancy is ~7y from diagnosis.
- Patients may be unable to give informed consent. In the absence of a Lasting Power of Attorney, clear documentation of the patient's incapacity and reasons to proceed (e.g. the patient's best interests) should be made.
- Patients can be confused and may be agitated (occasionally violent) or profoundly withdrawn.
- Patients with mild to moderate dementia are commonly treated with anticholinesterase drugs such as donepezil, rivastigmine or galantamine. These may prolong the action of suxamethonium and partially antagonise the effects of non-depolarising NMB drugs.
- Regional anaesthesia may still be desirable if significant comorbidity; ketamine (e.g. 5–20mg IV) may facilitate this and preserves airway reflexes and BP (titrate to effect). (Midazolam may cause disinhibition, which may paradoxically worsen agitation.)
- The association between postoperative neurocognitive disorder (POND) and dementia remains unclear, but dementia is considered a risk factor for the development of POND (see ➲ p. 90).

Anorexia nervosa

Anorexia nervosa is a chronic, severe multisystem disorder, which carries the highest morbidity and mortality rate of any psychiatric disorder (see ➲ pp. 86–7).

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Antidepressant drugs

The aetiology of depression is complex and multifactorial. The monoamine theory of depression postulates that depression is caused by functional deficiency of serotonin and noradrenaline in the CNS. Manipulation of CNS monoamines remains the most successful pharmacological approach to depression. Several families of drugs have this effect.[4,5]

Tricyclic antidepressants

TCAs, such as amitriptyline or imipramine, have largely been superseded by selective serotonin reuptake inhibitors (SSRIs) (fewer side effects and safer in overdose) for the treatment of depression. However, TCAs may be used in the treatment of other problems, e.g. chronic pain. They need to be given for 2–4w to become effective.

- TCAs block the reuptake of monoamines (e.g. serotonin, noradrenaline) from the synaptic cleft by competing for a transport protein.
- Most have atropine-like side effects: dry mouth, blurred vision, urinary retention and constipation. Other common side effects are sedation, postural hypotension and delayed gastric emptying.
- They are strongly bound to plasma proteins, and their effects may be enhanced by competing drugs (e.g. aspirin, warfarin, digoxin).
- In overdose, TCAs cause agitation, delirium, respiratory depression and coma. Cardiac arrhythmias with prolongation of the QT interval are frequent. There is no specific antidote, and treatment is supportive, although intensive care may be required. Alkalinisation of plasma reduces the amount of free drug.
- TCAs should not be withdrawn perioperatively.
- ↑ sensitivity to catecholamines may result in hypertension and arrhythmias, following the administration of sympathomimetic drugs (adrenaline, noradrenaline). Indirect sympathomimetics (e.g. ephedrine, metaraminol) should be used with caution.
- Anticholinergic drugs (e.g. atropine) which cross the blood–brain barrier may contribute to postoperative confusion.
- Co-administered tramadol increases the risk of serotonin syndrome.
- St John's wort (*Hypericum perforatum)* contains alkaloids which resemble TCAs in structure and is useful and safe in mild depression, but can also contribute to serotonin syndrome (see ➲ p. 332).

Selective serotonin reuptake inhibitors

SSRIs are the most commonly prescribed antidepressants worldwide and are also prescribed for other conditions, e.g. panic disorder, obsessive–compulsive disorder. They are highly specific inhibitors of presynaptic reuptake of serotonin from the synaptic cleft and are much less toxic in overdose than TCAs. Common examples include fluoxetine, sertraline and citalopram.

- Common side effects affect the GI tract (nausea, vomiting, diarrhoea, upper GI bleeding) and the CNS (insomnia, agitation, tremor, headache, sexual dysfunction). CVS side effects are rare (occasional reports of bradycardia).
- SSRIs may precipitate coronary vasoconstriction in those with IHD.
- Check Na^+, especially in the elderly, as SSRIs have been known to cause hyponatraemia 2° to SIADH (see ➲ p. 242).

- High doses may impair platelet aggregation and cause prolonged bleeding times. Check coagulation screen.
- SSRIs inhibit cytochrome P450 enzymes, which may prolong or enhance the activity of other drugs, notably warfarin, theophylline, phenytoin, carbamazepine, tolbutamide, benzodiazepines (diazepam, midazolam), type 1c antiarrhythmics (e.g. flecainide), TCAs and some NSAIDs.
- SSRIs may interact with other drugs with serotonergic properties to cause serotonin syndrome. This includes the opioids tramadol, pethidine, pentazocine and dextromethorphan.
- SSRI use is common, and anaesthesia usually uneventful.
- Abrupt withdrawal of SSRIs can precipitate a withdrawal syndrome.

Monoamine oxidase inhibitors

MAOIs are 3rd-line antidepressants with significant anaesthetic implications due to their many drug and food reactions. The enzyme MAO is present on mitochondrial membranes where it inactivates monoamine neurotransmitters in the cytoplasm. It has two isoenzymes MAO-A and MAO-B.

- MAO-A preferentially metabolises serotonin, noradrenaline and adrenaline, and predominates in the CNS. MAO-B preferentially metabolises non-polar trace amines such as the neurotransmitter phenethylamine. It predominates in the liver, lungs and non-neural cells.
- Dopamine and tyramine (found in cheese and other foods) are substrates for both MAO-A and MAO-B.
- The older MAOIs phenelzine, tranylcypromine and isocarboxazid bind covalently to MAO. Regeneration of new enzyme takes 2–3w. Ideally, they should be stopped at least 2w prior to surgery. This may provoke relapses in symptoms and should not be done without consultation with a senior psychiatrist. If stopped for <2w, patients should be considered as still on MAOI.
- Phenelzine decreases plasma cholinesterase levels and prolongs the action of suxamethonium.
- Newer drugs are reversible inhibitors of monoamine oxidase A (RIMAs). Moclobemide is the only RIMA available in the UK and can be omitted safely for 24h preoperatively.
- Linezolid (antibiotic) is a non-selective reversible MAOI.
- Selegiline is a MAO-B inhibitor used in PD. At doses of <10mg/d, it is not necessary to stop it preoperatively as there is no reaction with sympathomimetics. Pethidine, however, should still be avoided.
- Methylthioninium chloride (methylene blue) has MAOI properties.
- Indirect sympathomimetics (ephedrine, metaraminol, amphetamine, cocaine, tyramine) are metabolised by MAO and may have a greatly exaggerated effect. Displacing noradrenaline from neurotransmitter vesicles can cause a life-threatening hypertensive crisis.
- Direct sympathomimetics (e.g. noradrenaline, adrenaline, phenylephrine, methoxamine, dopamine, dobutamine, isoprenaline) may have an exaggerated effect and should be used with caution.
- Treat hypotension initially with IV fluid, then with cautious doses of phenylephrine (e.g. 10–20 micrograms).
- Opioid drugs: MAOIs can inhibit hepatic microsomal enzymes, prolonging the action of opioids and enhancing their effect.

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- Pethidine is contraindicated with all MAOIs because of the risk of serotonin syndrome. Avoid tramadol and dextromethorphan, which also have serotonergic properties.
- LA drugs (except cocaine) are safe (caution if contain adrenaline). Axial and regional blocks are ideal; however, hypotension should be treated cautiously. Felypressin is a satisfactory alternative to adrenaline if a vasoconstrictor is required.
- Anticholinergic drugs (atropine, glycopyrronium) are safe.

Serotonin syndrome

Serotonin syndrome is a toxic crisis resulting from ↑ synaptic levels of serotonin in the brainstem and spinal cord due to overdose of SSRIs or a combination of other drugs affecting serotonin (especially TCAs, MAOIs, pethidine and tramadol). It presents as a clinical triad:

- Alteration in behaviour (agitation, confusion)
- Abnormal motor activity (rigidity, myoclonus, hyperreflexia)
- Autonomic instability (pyrexia, tachycardia, diarrhoea, unstable BP).

It may progress to seizures, oculogyric crises, DIC, rhabdomyolysis, myoglobinuria, AKI, arrhythmia, coma and death. It may mimic the neuroleptic malignant syndrome (see ➲ p. 334) (Table 13.1)[6] and MH. The patient is likely to require intensive care. Treatment is mainly supportive, and the episode usually lasts <24h.[4,6]

Lithium

Lithium is an inorganic ion used as a mood stabiliser in the treatment of bipolar affective disorder. It has a low therapeutic ratio, with an optimal plasma concentration of 0.4–1.0mmol/L.[7]

- Chronic use causes weight gain, renal impairment and hypothyroidism.
- Lithium toxicity occurs at >1.5mmol/L and is exacerbated by hyponatraemia, diuretic therapy and renal disease. Features include lethargy, restlessness, nausea, vomiting, thirst, tremor, polyuria, renal failure, ataxia, convulsions, coma and death. Haemodialysis is effective.
- Lithium potentiates both depolarising and non-depolarising NMB; nerve stimulator monitoring should be used.
- Lithium may cause T-wave flattening or inversion, but clinically important CVS effects are rare.
- NSAIDs should be used with caution (risk of exacerbating renal impairment and causing toxicity).

Table 13.1 Comparison of serotonin syndrome and neuroleptic malignant syndrome

	Serotonin syndrome	Neuroleptic malignant syndrome
Cause	Serotonergic drugs (MAOIs, TCAs, SSRIs, stimulants, ondansetron, St John's wort)	Not clear, but likely to involve dopamine receptor blockade (typical and atypical antipsychotics, metoclopramide, promethazine, prochlorperazine)
Features	Tachycardia Tachypnoea Hypertension Hypersalivation Hyperthermia Coma Hyperreflexia + clonus Dilated pupils ↑ bowel sounds ↑ tone (especially in lower limbs)	Tachycardia Tachypnoea Hypertension Hypersalivation Hyperthermia Coma Hyporeflexia Normal pupils ↓ or normal bowel sounds Lead-pipe rigidity
Treatment	Discontinue causative drugs Supportive treatment Short-acting drugs (e.g. esmolol) to control BP If hypotensive, can give noradrenaline, adrenaline or phenylephrine Benzodiazepines for agitation Active cooling if needed Cyproheptadine 12mg PO if supportive care fails	Discontinue causative drugs Supportive treatment Control BP (e.g. clonidine, esmolol) Fluid to prevent renal failure associated with rhabdomyolysis Benzodiazepines for agitation Active cooling if needed Amantadine, bromocriptine and dantrolene (avoid if LFTs deranged) have all been used

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Antipsychotic drugs

Antipsychotic drugs

Antipsychotic drugs (formerly known as neuroleptics) include haloperidol, chlorpromazine, olanzapine, quetiapine and risperidone, and are used in the treatment of schizophrenia and similar disorders. Their main action is antagonism at CNS dopamine (D_2) receptors, but most antagonise other receptors, including histamine (H_1), serotonin (5-HT_2), acetylcholine (muscarinic) and α-adrenergic receptors. Many have antiemetic effects.[4,5]

- Common side effects include sedation, extrapyramidal motor disturbances and tardive dyskinesia with chronic use. Less common side effects include gynaecomastia, weight gain, postural hypotension, antimuscarinic effects, obstructive jaundice and agranulocytosis (rare, but severe).
- Many drugs prolong the QT interval, especially when combined with other drugs which may do the same (e.g. antidepressants).
- Clozapine is associated with a risk of agranulocytosis.
- Abrupt withdrawal of antipsychotic medication is dangerous.
- Antipsychotic drugs potentiate sedative and hypotensive effects of anaesthetic agents (including opioids).

Neuroleptic malignant syndrome

A rare idiosyncratic reaction to antipsychotic drugs which resembles MH and serotonin syndrome (see ➲ p. 332) (Table 13.1). Typical patients are young ♂ . Features include hyperthermia, tachycardia, extrapyramidal dysfunction (rigidity, dystonia) and autonomic dysfunction (sweating, labile BP, salivation, urinary incontinence). CK and WCC are raised. Patients should be treated in the ICU. Mortality approaches 20%.[6]

Agitated patients on the ward

Anaesthetists may be asked to help with sedation of agitated patients on general wards and are more likely to be familiar with the effects of sedation than junior medical or surgical staff.

- Patients are likely to be in an acute confusional state or delirium, exacerbated by pain, unfamiliar/threatening surroundings and strangers. They may be disoriented, agitated, disinhibited or violent, and may experience visual or auditory hallucinations.
- When presentation is acute in a previously lucid patient, the cause is usually organic. Establishing and treating the cause may remove the need for sedation.
- Exclude hypoglycaemia, hypoxia, pain, alcohol withdrawal and a full bladder.
- Differential diagnosis includes infection (chest, urine, lines), drugs (cocaine, LSD, sedatives, analgesics) and metabolic derangement (e.g. hyponatraemia, hypoglycaemia). Less frequently: head injury, CVE, acute psychiatric disorder (e.g. mania) and acute porphyria.

Approach to the patient

- Ensure the safety of yourself and other staff. A calm and reassuring approach will help the patient and any onlookers.
- If physical restraint is necessary, ensure plenty of help is available (hospital security, porters and even the police) and discuss with a psychiatrist.
- Establish venous access, if possible, and bandage the cannula.
- Aim to render the patient calm and cooperative, rather than unconscious.
- Do not leave a sedated patient unattended.

Drug therapy

- Haloperidol 5mg IV initially (reduce dose in the elderly, e.g. 1mg). Repeat after 5min if required. Titrate to effect. Maximum dose: 18mg/24h, according to *BNF*, but higher doses are occasionally warranted.
- Midazolam 1–2mg IV may also be useful (titrate to effect). May cause paradoxical disinhibition, especially in the elderly.
- Alcohol withdrawal: give diazepam 5–10mg IV (or chlordiazepoxide 50mg PO), and repeat as required.
- Ketamine is useful in emergencies if the patient is extremely violent or dangerous. Give 0.5–1mg/kg IV (or 5–10mg/kg IM).
- Do not use propofol (too short-acting), opioids (respiratory depression) or drugs with which you may be unfamiliar. Clonidine may provoke hypotension.
- In ED, or where the history is unknown, further investigation (e.g. CT scan) may be appropriate. In this circumstance, RSI of anaesthesia with full monitoring may be required.

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Substance abuse disorder

General considerations

In the UK, about 10% of adults have used an illegal drug in the past year. Misuse of street drugs occurs in all socioeconomic groups. A substance abuse disorder (SAD) exists when the extent and pattern of substance use interfere with the psychological and sociocultural integrity of the person. Patients with SAD may be abusing CNS depressant drugs (alcohol, benzodiazepines, opioids) or CNS stimulant drugs (cocaine, amphetamines, ecstasy, cannabinoids).

- Acute intoxication may impede informed consent. In addition, the effects of the drug may counteract (stimulants) or enhance (sedatives) the effects of anaesthetic drugs. Consider drug misuse in all patients with a reduced conscious level or requiring emergency surgery and anaesthesia.
- Chronic drug misuse is associated with poor nutrition, medical and psychiatric comorbidity and the presence of viral infections.
- IV drug users often have no accessible veins. IV drug misuse is associated with IV infective complications. HIV and viral hepatitis are the commonest. Bacterial endocarditis is rare but serious, and associated with pulmonary abscesses, embolic phenomena from vegetations and vasculitis.
- Drugs in common use fall into several groups (Table 13.2). Combinations of drugs are common, often with alcohol. Street drugs may be impure or deliberately adulterated with other substances.
- Anaesthetists may be at elevated risk of SAD (see p. 16).

Table 13.2 Street drugs in common use

Drug	Clinical signs
Cannabis	Tachycardia, abnormal affect (e.g. euphoria, anxiety, panic or psychosis), poor memory, fatigue
Stimulants: cocaine, amphetamines, ecstasy	Tachycardia, labile BP, excitement, delirium, hallucinations, hyperreflexia, tremors, convulsions, mydriasis, sweating, hyperthermia, exhaustion, coma
Hallucinogens: LSD, phencyclidine, ketamine	Sympathomimetic, weakly analgesic, altered judgement, hallucinations, toxic psychosis, dissociative anaesthesia
Opioids: morphine, heroin, oxycodone	Euphoria, respiratory depression, hypotension, bradycardia, constipation, pinpoint pupils, coma

Anaesthetic considerations in patients with SAD[8]

- Keep a high index of suspicion, especially in trauma.
- Difficult venous access: IV drug users may be able to direct you to a patent vein. Consider ultrasound scanning (USS) for vein location; rarely, the patient may need central venous cannulation. Consider inhalational induction.
- Take full precautions against infection risk.

- Plan postoperative analgesia with the patient preoperatively.
- Do not attempt drug withdrawal perioperatively.
- Effective management of acute pain in patients with SAD may be complicated by:
  - Psychological and behavioural characteristics
  - Presence of the drug of abuse and morbidities related to it
  - Physical dependence (physiological phenomenon characterised by a withdrawal reaction when the drug is withdrawn or an antagonist is administered) and the risk of withdrawal
  - Medications used to assist with drug withdrawal or rehabilitation
  - Ethical dilemmas arising as a result of the need to balance concerns of undermedication against anxieties about safety and possible abuse or diversion of the drugs.
- Management of pain in patients with SAD should focus on prevention of withdrawal, effective analgesia and symptomatic treatment of affective and behavioural problems.

Opioids

(See ➲ pp. 1157–60.)

Cannabis[8,9]

Cannabis is cheap and widely available, and consumption is common, usually by smoking. The active ingredients have a very long half-life, but toxicity seems to be low. Evidence for the medicinal benefits of cannabinoids (e.g. for analgesia) is sparse. In practice, ordinary cannabis use causes few problems for anaesthetists.

- Do not prescribe purified cannabinoids for analgesia.

Cocaine and crack cocaine[8,9]

Cocaine is usually 'sniffed' in powder form. Free-base ('crack') cocaine is heat-stable and can be smoked. Frequent cocaine use predisposes to dilated cardiomyopathy due to microinfarcts from small-vessel ischaemia, which may be asymptomatic in its early stages.

- Cocaine's effects are mediated by central and peripheral adrenergic stimulation. Most of the life-threatening side effects of cocaine (including MI and CVE) are due to vasospasm and can be treated using combinations of vasodilators, antiarrhythmic agents and α/β-blockers titrated against effect, using full invasive monitoring.
- Avoid vasoconstrictors (including those in LA preparations): hypertensive crisis may result. If vasopressors are required in theatre, use very small doses, and titrate against response.
- Accidental intra-arterial injection of cocaine can cause critical limb and organ ischaemia. Successful treatment has included aggressive anticoagulation, thrombolysis and vasodilator therapy.
- Use caution in instrumenting the airway or nasopharynx, as chronic nasal use can cause perforation of the nasal septum or soft palate.

Amphetamines[8,9]

- Includes MDMA ('ecstasy') as well as methamphetamine ('meth').
- Amphetamines mimic endogenous monoamines and cause excitation and euphoria. Toxicity is treated symptomatically. Labetalol is effective in controlling BP. Beware of hyperthermia and dehydration.

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Alcohol[10,11]

Alcohol use is common and causes problems as a result of both acute intoxication and the health effects of chronic consumption. Ask all adults about alcohol consumption. Surgery should be avoided, if possible, in the acutely intoxicated as consent is difficult.

- Ketoacidosis may present after binge drinking, in association with vomiting and fasting. Blood alcohol levels may already have normalised.
- Acute intoxication may cause vomiting, dehydration, hypoglycaemia and delayed gastric emptying. Careful rehydration, with attention to electrolytes and glucose, and RSI are advised.
- Chronic alcohol excess induces tolerance to GA and is associated with an increase in postoperative complications, including infection.
- Alcoholic cardiomyopathy is characterised by a dilated, hypokinetic LV and ↓ EF. Patients may present with CCF and oedema, exacerbated by low serum albumin. Consider echocardiography.
- Alcoholic liver disease: the earliest form is reversible fatty liver, progressing to alcoholic hepatitis (abdominal pain, weight loss, jaundice, fever) and later cirrhosis (jaundice, ascites, portal hypertension, hepatic failure). Correct clotting abnormalities preoperatively. X-match blood. Patients with liver failure may require intensive care if surgery is planned (see also ➲ pp. 205–10).
- Consider IV thiamine to reduce the risk of Wernicke's encephalopathy.
- Anticipate alcohol withdrawal symptoms. Most patients can tolerate 24h abstinence perioperatively—many hospitals have benzodiazepine regimens to prevent withdrawal syndrome.
- Seizures are most commonly seen 6–48h after cessation of drinking, typically tonic–clonic. Several fits over a period of a few days are common. Low K^+ and Mg^{2+} predispose. Seizures may be preceded by disorientation and agitation (*delirium tremens*). Treat with benzodiazepines, e.g. diazepam 10mg IV, repeated as required.

Electroconvulsive therapy

Procedure	Electrically induced seizure
Time	5–10min
Pain	±
Position	Supine
Blood loss	Nil
Practical technique	Short IV GA, face mask only, bite-block

General considerations

Electroconvulsive therapy (ECT) is safe and effective in the treatment of mental disorders, most commonly severe depression unresponsive to drugs, or where there is self-neglect or a high risk of suicide. ECT is commonly carried out in an isolated site—ensure skilled assistance, adequate monitoring and resuscitation facilities. Anaesthetic equipment may be older or unfamiliar.[12]

Physiological effects of electroconvulsive therapy

During the seizure, there is parasympathetic hyperactivity, bradycardia and hypotension, lasting about 15s, followed by a more prolonged (5min) sympathetic stimulation: tachycardia, hypertension, dysrhythmias and ↑ myocardial O_2 requirement. During this time, there is also ↑ ICP, cerebral blood flow and cerebral O_2 requirement. There may also be hypersalivation, ↑ intragastric pressure, ↑ intraocular pressure (IOP) and occasionally incontinence.[13]

Preoperative

A careful preoperative assessment (including investigations) should be undertaken, as for any GA. Consent is normally arranged by the psychiatry team. ECT is typically given twice weekly for several weeks, reducing in frequency as the patient improves. Read the notes for documentation of previous problems.

- Absolute contraindications: recent MI or CVE, phaeochromocytoma, intracranial mass lesion, intracranial or aortic aneurysm.
- Relative contraindications: uncontrolled angina, CCF, severe osteoporosis, major bone fracture, glaucoma, retinal detachment. ECT during pregnancy is acceptable.
- Avoid sedative premedication, which is anticonvulsant.
- Glycopyrronium (0.1–0.3mg IV) may be used to reduce secretions and to counteract bradycardia. Consider antacids if history of reflux.

Perioperative

Efficacy of ECT is dependent on seizure quality, as measured by EEG-derived variables, rather than just on duration. There is no further benefit beyond about 60s of seizure time. Good technique provides short GA, muscle relaxation to lessen the risk of trauma, attenuation of physiological effects and rapid recovery.[12,13,14]

- Thorough preoxygenation is recommended.
- All GAs shorten the seizure in a dose-related fashion; use light doses.

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- Any traditional induction agent may be used for induction. Propofol attenuates the sympathetic response but shortens the seizure more than the others. Etomidate shortens the seizure less, but the sympathetic response may be more pronounced. Inhalational sevoflurane is effective but takes longer.
- The combination of a small dose of propofol with remifentanil (1 microgram/kg) provides good conditions and attenuation of the sympathetic response. A combination of ketamine and propofol is sometimes used.
- Suxamethonium (0.5–1mg/kg) is given to 'modify' the seizure (reduces muscle power to prevent injury). Mivacurium (0.2mg/kg) may be used instead but will probably require reversal. The combination of rocuronium and sugammadex is also effective.
- Insert a bite-block to prevent damage to the mouth and teeth.
- Maintain the airway with a face mask and/or oral airway. Hand-ventilate the patient with O_2 until breathing resumes afterward.
- The psychiatrist may titrate the magnitude of the stimulus to the quality of the seizure; be prepared to maintain the anaesthetic with further boluses of induction agent if a 2nd stimulus is required.
- Sympathetic response may also be attenuated with alfentanil (10 micrograms/kg) or esmolol (e.g. 0.25mg/kg). Labetalol, sodium nitroprusside and hydralazine have also been used.
- Seizure augmentation: both caffeine and theophylline lower the seizure threshold and prolong the seizure. Moderate hyperventilation with bag and mask before the seizure is also somewhat effective.
- If the seizure lasts longer than 60s, it should be terminated, e.g. with propofol titrated to response.

Postoperative

- Post-ictal agitation, confusion or aggression may occur in some patients. They should be nursed in a calm environment and may occasionally require sedation (e.g. midazolam 1mg IV).
- Headache and muscle pains are commonly reported and usually respond to simple analgesics.
- Drowsiness and cognitive impairment are very common but typically resolve within a few hours.
- No evidence of memory loss is demonstrable at 6mo. However, patients sometimes complain of memory loss for specific life events.
- Other complications include nausea, exacerbation of IHD, fractures/dislocations, dental/oral injury and laryngospasm.
- ECT does not increase the risk of other types of seizure.
- Overall mortality is about 1 per 80 000 treatments.

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Chapter 14

Getting started

Mincho Marroquin- Harris

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Getting started

A thorough working knowledge of anaesthetic equipment is essential for safe and effective anaesthetic practice. Although many different types of equipment are in use throughout the world, a few basic principles underlie the functions of nearly all of them.

Anaesthetic gases

Piped medical gases and scavenging system (PMGSS)

The PMGSS is part of the hospital infrastructure and supplies clinical areas with compressed O_2 and vacuum. Operating theatres are also supplied with N_2O, medical air and gas scavenging.

- The PMGSS is a system of seamless copper alloy pipes. Pipes are cleaned, degreased and free of particulate matter. Colour-coding is used on the outside of pipes at set intervals. These pipes supply the terminal gas outlets in clinical areas.
- Terminal outlets are labelled and colour-coded, and have self-sealing Schrader valves to prevent gas leakage. Each gas has a specific outlet shape and diameter to prevent misconnection of equipment to the wrong outlet.
- Alarms are activated in case of gas failure with low pressure (<370kPa) and at high pressures (>500kPa).
- Isolation valves located in theatre complexes allow PMGSS to be turned off in case of leak, fire or other emergency.
- Most incidents occur just following installation rather than during long-term use. Consequently, after installation or modifications, an inspection process is performed to ensure the correct gases are being supplied to wall outlets.
- Safety standards and colour codes for medical gas supplies for both pipeline and cylinder storage conform to International Organization for Standardization (ISO) requirements. The US (and hence the airline industry) observes its own colour scheme, in which O_2 is colour-coded green.

Oxygen

O_2 is stored in bulk for hospital supply, either in a vacuum-insulated evaporator (VIE) or in a cylinder manifold.

Vacuum-insulated evaporator

The VIE is the typical main O_2 store for most hospitals and is a large tank of liquid O_2. One litre of liquid O_2 can provide >800L of gaseous O_2. Typically, the VIE is sized to hold 200–1000L. In order to maintain its liquid state, the VIE stores O_2 at –183°C at a pressure of 1050kPa. Prior to entering the PMGSS, O_2 flows through a pressure regulator to provide a supply pressure of 400kPa.

Cylinder manifold

A cylinder manifold consists of two banks of cylinders which are at least size G (Table 14.1). When one bank of cylinders is exhausted, a reduction in pressure will automatically open the 2nd bank, ensuring ongoing gas supply.

Cylinder supply

O_2 is available in individual portable cylinders of various sizes. Since O_2 is a gas in the cylinder, the gauge pressure is directly proportional to the quantity in the cylinder. Traditional steel cylinders are pressurised to 13 700kPa, but more modern materials allow higher pressures in the cylinder. A type E cylinder at 13 700kPa contains 680L of gaseous O_2.

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Nitrous oxide

N_2O for hospitals is often stored in a cylinder manifold, though smaller facilities may mount a N_2O cylinder on the back of the anaesthetic machine. The mains pressure of N_2O is 400kPa.

- N_2O is stored as a liquid in the cylinder at 4500kPa. The gauge pressure is not proportional to the quantity in the cylinder.
- High ambient temperatures could cause the N_2O cylinder to explode. Therefore, the cylinder is only partially filled with liquid to allow for some expansion.

Medical air

Medical air is atmospheric air which is dehydrated and ultrafiltered to remove particulates. It can be supplied by an air compressor, in cylinder form or as 'synthetic air' blended from gaseous O_2 and nitrogen. It is often supplied at two mains pressures: 700kPa to drive surgical tools, and 400kPa for medical and anaesthetic uses.

Medical gas cylinders

Under the ISO standard, all medical gas cylinders have a white body. The contents of the cylinder are denoted by the colour of the shoulder (Table 14.1).

Newer materials allow the cylinders to be light and sustain high internal pressures. A pin-index system prevents the incorrect gas from being connected to the yoke of the anaesthetic machine.

Table 14.1 Description of medical gas cylinders*

	Shoulder colour	Pressure when steel cylinder is full (kPa)
O_2	White	13 700
Air	White/black	13 700
N_2O	French blue	4500(maintains this pressure until almost empty)
Entonox®	Blue/white	13 700
Heliox® (28% O_2, 72% helium)	Brown/white	13 700
CO_2	Grey	5000

* Some suppliers may vary. Check product information.

Vacuum system

The vacuum system includes both medical suction and scavenging for anaesthetic gases. The suction for both systems is provided by electrical pumps, so it is susceptible to power outage.

Medical vacuum

Provides high vacuum to suction for sputum or blood. A pressure of –40kPa or below is provided with a sustained flow of at least 40L/min.

- Suctioned material is collected in a reservoir to prevent contamination of the PMGSS.
- Loss of suction pressure can occur from disconnection/leak in the apparatus, blockage of filter or excessive demand.

The anaesthetic gas scavenging system

Removes expelled anaesthetic gases to minimise exposure of staff in the operating room.

- Provides high flow (80–130L/min) just below atmospheric pressure.
- Gases released from the adjustable pressure-limiting (APL) valve or expiratory port of the ventilator are collected in a reservoir before passing to the disposal system and ultimately vented to the atmosphere.
- A flow indicator on the side of the anaesthetic machine shows that the scavenging system is working within safe flow rates.
- Tubing connectors for the vacuum systems are sized specifically so that they cannot be accidentally attached to airway equipment.

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Anaesthetic machine

The continuous-flow anaesthetic machine receives a supply of high-pressure gases, reduces the pressure to safe levels and provides accurate and controlled flow of gases and vapour to the patient via a breathing system (Fig. 14.1).

High-pressure system

Takes high-pressure gas (from cylinders or wall outlets) and steps it down to the working pressure of the machine, at the flow-control valves or flowmeters. In older machines, this is mechanical; newer machines feature electronic control of the process.

- Gas pressures from the PMGSS are measured and reduced by regulators. Valves ensure flow is unidirectional.
- Safety features include antihypoxia and low-pressure alarms. If the O_2 supply fails, the supply of N_2O is also stopped.

Low-pressure system

Extends from the flow-control valves/flowmeters to the common gas outlet. In modern anaesthetic machines, the flow and composition of gases are set digitally. Gases are regulated electronically based on feedback from electronic flow and gas-content sensors. Traditional anaesthetic machines use flowmeters. Flow is adjusted by a control knob and displayed by the height of a rotating bobbin in a fluted glass column. Flowmeter controls are labelled and colour-coded, and the O_2 control is distinguishable by touch.

Vaporisers

Provide precise concentrations of volatile agents. Traditional variable-bypass plenum vaporisers are calibrated mechanical devices. Newer vaporisers are electronically controlled. Some devices are not vaporisers in the strictest sense such as the dual-circuit gas-vapour blenders used for desflurane or the direct-injection devices which inject precise amounts of volatile into the gas flow under electronic control.

Common gas outlet

This is the final pathway of gases and volatile agents out of the machine. It is standardised with a 22mm external and 15mm internal connector. Traditionally, the breathing attachment was connected here, but in many modern machines, the connection is internal and the common gas outlet is absent.

Oxygen flush

- Rapidly provides 100% O_2 to the common gas outlet at high flow (30–75L/min). This O_2 bypasses the low-pressure system and contains no anaesthetic gases. The high flow and pressure have the potential for causing barotrauma.
- Many machines feature an auxiliary O_2 outlet with its own flowmeter. However, the O_2 has the same source as the common gas outlet and should not be used if there is a problem with this supply.

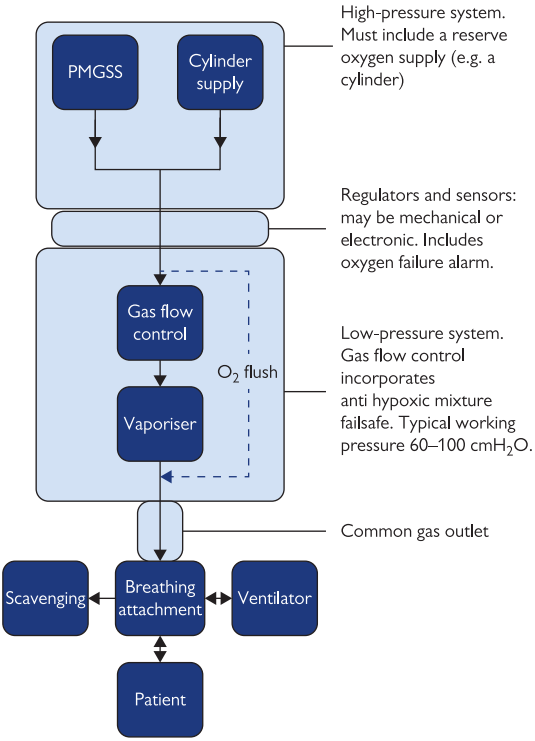

Fig. 14.1 Simplified schematic of a continuous-flow anaesthetic machine.

Further reading

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Breathing systems

For paediatric breathing systems, see ➲ pp. 910–12. In anaesthesia, many different breathing attachments are used to bridge the gap from the anaesthetic machine to the patient. One of the commonest examples in use is the circle breathing circuit, as it is versatile and efficient (Fig. 14.2). It comprises an inspiratory limb, an expiratory limb, a reservoir bag, a valve and a CO_2 absorber. Gas flows via the inspiratory limb to the patient via a Y-piece, then leaves via the expiratory limb. Exhaled gas is recycled through the CO_2 absorber, which allows very economic use of anaesthetic gases and agents. This, in turn, minimises atmospheric pollution. The recycling of expired gases allows heat and moisture to be conserved.

Features of the circle system

- Low-resistance corrugated tubing has low compliance and low resistance. Tubing connectors have standard 22mm (external) and 15mm (internal) dimensions.
- Unidirectional ('flutter') valves are present on both limbs and prevent backflow. Moisture may accumulate on the expiratory valve, making it sticky.
- The APL valve allows the anaesthetist to adjust the pressure in the system during manual ventilation.
- The reservoir bag allows hand ventilation, manual appreciation of airway compliance and visualisation of SV.
- The CO_2 absorber (containing pellets of predominantly calcium hydroxide) is located on the expiratory limb. It chemically removes CO_2 in an exothermic reaction which also produces water vapour.

Other adjuncts and considerations

- An O_2 analyser is always positioned on the inspiratory limb.
- The heat and moisture exchange (HME) filter was designed to prevent particulate material from being inadvertently inhaled. Modern filter technology provides an effective bacterial and viral barrier. The HME filter minimises loss of heat and moisture to dry fresh gas by providing up to 70% relative humidity. It functions poorly at high minute ventilation and low temperatures.
- A CO_2 and volatile sampling line often attaches to, or near, the HME filter .
- At steady state and in ideal conditions, the fresh gas flow (FGF) can be reduced to provide the patient's basal O_2 requirement (e.g. 250mL/min) plus some volatile. However, at low flows, most of the gas the patient receives is their own recycled expired gas. ⚠ This can result in the patient receiving lower FiO_2 and anaesthetic agents than those selected. Some machines offer electronic control of FiO_2 and end-tidal volatile concentrations. Higher FGF is recommended at induction of anaesthesia to ensure MAC is reached rapidly.
- Paediatric circle systems have a small (e.g. 500mL) reservoir bag and narrower tubing.

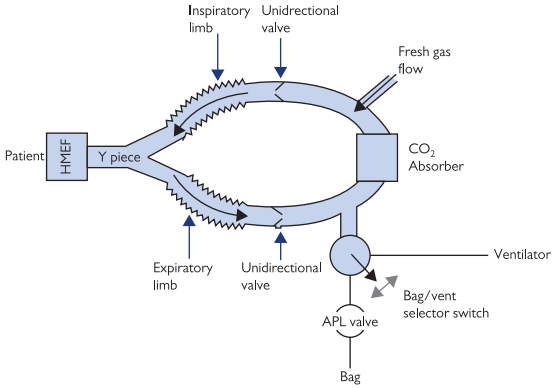

Fig. 14.2 Layout of a circle breathing attachment. Reproduced from Brockwell RC: Inhaled Anesthetic Delivery Systems. In Miller RD (ed): *Anesthesia*, 6th ed, p 295. Philadelphia, Churchill Livingstone, 2004, with permission from Elsevier.

Semi-closed breathing attachments

The efficiency and versatility of the circle system have led to it being used almost universally. However, there are two main drawbacks: complexity of design and resistance to breathing. There is still a role for simpler semi-closed breathing systems. These were classified by WW Mapleson in 1954, but all require FGF far higher than the circle system to prevent rebreathing of exhaled CO_2.

The Mapleson F attachment is a modified T-piece with an open-ended reservoir bag. It is still favoured among anaesthetists for infants, because of its extremely low resistance to gas flow and the ability to hand-ventilate with great precision and sensitivity.

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Ventilation

Self-inflating bag

The self-inflating resuscitation bag allows the anaesthetist to deliver positive pressure ventilation without an O_2 source, e.g. ventilation outside of the operating theatre, resuscitation of a collapsed casualty or in case of ventilator malfunction. A self-inflating bag should always be immediately available in theatre.

- There are several brands with different sized bags, typically adults 1.5–2L and paediatrics 250mL to 1L bags.
- A unidirectional valve controls the direction of flow and prevents rebreathing of CO_2. There is little apparatus dead space. The casing is clear, allowing the valve to be assessed during an equipment check.
- A PEEP valve may be applied to the expiratory port.
- The patient end of the self-inflating bag can be connected to anaesthetic face masks, ETTs or LMAs.
- When a face mask is attached, it is recommended to use 15L/min of O_2 which may splint the unidirectional valve slightly open, reducing work of breathing. Otherwise spontaneous breaths should be assisted due to ↑ work of breathing through the unidirectional valve.
- The bag reinflates with room air, but an O_2 line and a reservoir bag can be added. High FiO_2 is possible with a good seal.

Mechanical ventilation

The goal of mechanical ventilation in the anaesthetised patient is to support adequate gas exchange and minimise iatrogenic injury.

Modes of ventilation

When patient-centred outcomes are considered, no particular ventilation mode has been shown to be better than another. Modern ventilators offer a large variety of electronically controlled modes, the terminology of which varies between manufacturers. Mandatory modes tend to ignore any respiratory effort from the patient, but triggered (supportive) modes permit the patient to initiate a breath, which is then augmented by the ventilator.

Volume control ventilation

Volume control ventilation is a mandatory mode of ventilation where the anaesthetist sets a desired V_T and RR. This mode is tolerant of changes in respiratory compliance, though any ↓ in compliance may lead to ↑ airway pressures, and volume control ventilation usually delivers higher pressures for the same V_T, compared to pressure control ventilation. The mode is usually intolerant of leaks.

Pressure control ventilation

Pressure control ventilation is a mandatory mode where the anaesthetist sets the pressure to be delivered and the RR. This mode is tolerant of small leaks, but any ↓ in respiratory compliance will lead to ↓ in V_T.

Pressure control ventilation, volume guaranteed (PCV-VG)

This is a mandatory mode of ventilation which attempts to provide the advantages of both pressure control ventilation and volume control ventilation, delivering a set V_T at the lowest possible pressure. This allows for relatively constant V_T despite changes in respiratory compliance.

Pressure support ventilation

Pressure support ventilation is a triggered mode of ventilation. The ventilator senses when the patient initiates a breath and then delivers a set pulse of positive pressure to augment the respiratory effort. Supporting the patient's breathing in this way may reduce work of breathing and potentially improve oxygenation by increasing mean inspiratory pressure. The patient determines the RR with no backup rate, so prolonged apnoea may occur if the patient does not trigger the ventilator.

Synchronised intermittent mandatory ventilation

Synchronised intermittent mandatory ventilation provides a set number of ventilator-controlled breaths and provides support to any spontaneous breaths the patient takes in addition. This is to allow for better patient–ventilator synchronisation.

Oxygenation

The ideal FiO_2 is not known. The effects of GA (supine position, ↓ FRC, ↑ dead space) make oxygenation less efficient, and many patients will become desaturated below an FiO_2 of 30%.

- Keeping FiO_2 low while providing adequate SaO_2 may minimise the risk of O_2 toxicity, coronary/cerebral vasoconstriction and absorption atelectasis.
- High FiO_2 may be needed in severely anaemic patients or during massive haemorrhage or other emergencies (see ➲ pp. 1070–2).
- Oxygenation can be improved by ↑ FiO_2 or ↑ mean inspiratory pressure, or both. The mean inspiratory pressure may be ↑ by ↑ inspiratory time, ↑ inspiratory pressure or ↑ PEEP.

Clearance of carbon dioxide

CO_2 clearance is proportional to minute ventilation (V_T × RR). Most patients can tolerate a modest ↑ $ETCO_2$ without coming to harm, but in some circumstances (e.g. neurosurgery), tight control of CO_2 is needed.

Lung-protective ventilation

Considered the standard of care for mechanical ventilation as it has been demonstrated to reduce postoperative pulmonary complications.[1] Aims include:

- V_T 6–8mL/kg (IBW, not actual body weight)
- Individualised PEEP (usually ≥5cmH_2O), with or without recruitment manoeuvres
- Airway plateau pressures <30cmH_2O.

Positive end-expiratory pressure

PEEP is the airway pressure above atmospheric pressure at end-expiration. It is usually set between 5 and 20cmH_2O.

- PEEP is important as it provides pressure to splint open alveoli during expiration. This improves oxygenation, minimises alveolar collapse and minimises trauma from collapse and re-expansion of alveoli during mechanical ventilation.
- Methods to titrate PEEP include titrating up or down to find the best compliance or best gas exchange, or titrating PEEP to oesophageal pressure with oesophageal manometry.

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- Higher PEEP may be required if the patient is in a head-down position or obese or during laparoscopic surgery.
- Lower PEEP may be required in obstructive lung disease to minimise gas trapping and hyperinflation, or in life-threatening hypotension or states of low preload.

Inspiratory:expiratory (I:E) ratio and flow rates

By default, the I:E ratio is set to 1:2.

- Higher I:E ratios (e.g. 1:4 or higher), and thus shorter inspiratory time, provide more time for exhalation, so minimise gas trapping, at the cost of ↑ inspiratory flow rate and ↑ peak airway pressure.
- Lower I:E ratios (e.g. 1:1 or 1:1.5) provide better oxygenation and more time for alveoli to participate in gas exchange. Beware of gas trapping or breath-stacking due to inadequate exhalation.

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Airway equipment

For paediatric airway equipment, see ➲ pp. 908–10.

Endotracheal tube

The ETT remains the gold standard for securing and protecting the airway. It is usually made of polyvinyl chloride, which is clear. Silicone tubes are softer to reduce trauma, but opaque. The size of the tube refers to its internal diameter in mm.

- The proximal end has a standard 15mm connector. The tube has markings for length. The distal end has a bevelled tip assisting passage through the vocal cords, and often a Murphy eye to reduce the likelihood of obstruction against the wall of the airway. ETTs should have a radio-opaque insert for visualisation on X-ray.
- The cuff is normally the low-pressure, high-volume type. Cuff pressure can be measured via the pilot balloon and should be 20–30cmH_2O. Very small-diameter tubes (e.g. for neonatal use) usually have no cuff (see ➲ pp. 922–4). There are many variations on the basic ETT design.

Ring, Adair and Elwyn (RAE) tubes

These are preformed tubes, designed to pass downward over the chin (oral) or upward over the forehead (nasal), and hence obstruct the surgical field as little as possible. They can be difficult to pass over a bougie, and the position of the bend varies by manufacturer.

Nasal tubes tend to be made of silicone to reduce trauma.

Reinforced tubes

These have a metallic coil in the wall of the tube which reduces kinking.

Laser-proof tubes

These are resistant to laser and used for surgery involving laser in the oropharynx. They may have two cuffs in case one is punctured by the laser (see ➲ pp. 478–9).

Microlaryngeal tubes

These have a smaller outer diameter for the same inner diameter and are longer. Used for laryngeal surgery.

Neural integrity monitor

Enable monitoring of laryngeal nerve function via EMG and attached electrodes.

Double-lumen tubes (DLTs)

There are two tubes, within one outer casing, which can be ventilated together or independently. These tubes allow lung isolation for thoracic surgery. The sizing and shaping of DLTs are specific to this tube (see ➲ pp. 535–7).

Laryngectomy and tracheostomy tubes

Short, kink-resistant, J-shaped tubes for placement into a tracheostomy.

Supraglottic airways

Since the introduction of the classic LMA in the late 1980s, there has been considerable modification and refinement of the basic design, leading to a wide range of devices becoming available.

The SGA (also referred to as supraglottic airway device (SAD)) consists of a flexible tube ending in a soft (usually inflatable) cuff designed to form

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a seal around the larynx without entering it. SGAs are popular due to ease of placement and avoid many of the drawbacks of intubation, e.g. ↓ sympathetic stimulation, ↓ sore throat, ↓ trauma to teeth and airway structures, ↓ bronchospasm, ↓ coughing on emergence. With a reasonable seal, SGAs are suitable for mechanical ventilation. They do not provide protection from laryngospasm or aspiration. Newer designs incorporate a gastric channel to ↓ risk of aspiration.

First-generation SGA

One airway tube with the laryngeal mask cuff at the end (Fig. 14.3). Common variants of the classic LMA include:

- **Flexible LMA**: longer and narrower, wire-reinforced tube to prevent kinking, allowing movement away from surgical field
- **Intubating LMA** (Fastrach®): designed to allow blind intubation of the trachea by passing a Fastrach® ETT through the airway channel. This device is rigid and has a preformed 90° curve.

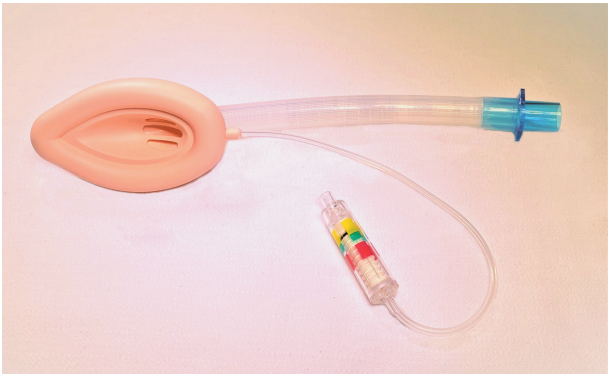

Fig. 14.3 A #4 Ultimate™ laryngeal mask, an example of a 1st-generation SGA. Courtesy Aidan O'Donnell.

Second-generation SGA

These have design features that are intended to reduce the risk of pulmonary aspiration of gastric contents, such as a gastric channel from the tip of the cuff up to the proximal end. Some also incorporate a rigid airway channel, or bite-block. They may permit blind or fibreoptic intubation via the airway channel. Common examples include ProSeal® LMA often used for IPPV or PSV, and i-gel® which has a gel-filled laryngeal cuff which requires no inflation. Often used in ED and prehospital care. (See Fig. 14.4.)

'Third-generation' SGA

This term has appeared in commercial literature for some products. It currently has no uniform definition in published literature.

Fig. 14.4 A #4 Auragain™, an example of a 2nd-generation SGA. Courtesy Aidan O'Donnell.

Other airway adjuncts

Oropharyngeal airways (OPAs) (e.g. Guedel) are tubes made of rigid plastic that follow the curvature of the palate and open the upper airway by holding the tongue away from the posterior pharyngeal wall. Flanges on the external end limit the depth of insertion and should rest on the teeth/lips. They are colour-coded to help rapidly identify the size and have a standardised sizing from 000 to 6. Inappropriately sized OPAs may worsen obstruction. Not tolerated in awake patients.

Nasopharyngeal airways are made of soft tubing (sized by internal diameter (ID) in mm) designed to prevent occlusion of the pharynx by the soft palate. Better tolerated in awake patients than OPAs. To reduce the risk of epistaxis, lubricate well and consider topical vasoconstrictor application prior to gentle insertion following the floor of the nasal cavity.

Laryngoscopes

Direct laryngoscopes

Designed to provide line of sight to the glottis for intubation. The Macintosh blade is the most popular design, curved longitudinally with a flange to sweep the tongue out of the way, and a blunt tip designed to engage the vallecula and lift the epiglottis forward, exposing the glottis. The handle contains batteries and a light source. Other direct laryngoscopes include:

- **Straight blades**. The tip of the straight blade is designed to lift the epiglottis, which is especially suitable for intubating children.
- **McCoy blade**. A variant of the Macintosh blade with a manual hinge which allows the tip of the blade to further elevate the epiglottis and improve the view.

Videolaryngoscopes (VLs)

Advantages include a better view, which can be seen by assistants, less extension of the neck and less trauma to the mouth. (See Fig. 14.5.) There are a variety of systems available from different manufacturers. They can be classified by the design of the blades.

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Fig. 14.5 A McGrath™ VL. Courtesy Aidan O'Donnell.

- **Macintosh blades**, e.g. C-MAC®, McGrath®, Glidescope®
- **Hyperangulated blades**—permit visualisation of the larynx without alignment of the oropharyngeal and laryngeal axes. Examples include the D-blade for C-MAC® and X-blade for McGrath®. A stylet or bougie is required to direct the ETT into the larynx
- **Blades with ETT-guiding channels**, e.g. Pentax Airway Scope® or King Vision®.

Endotracheal tube introducers

Intubating stylet

Made of malleable metal. It is inserted into an ETT to preform it to a desired shape or curvature prior to intubation.

Intubating bougie

A flexible, long plastic device with an angled tip used to facilitate intubation of an anterior larynx. The ETT is then railroaded over the bougie. Modern variants are hollow to permit a degree of oxygenation.

Airway exchange catheter

Designed to be passed down an ETT and left in the airway as the ETT is removed, and to act as a guide for a replacement ETT if required. It is hollow to permit oxygenation.

Long-term venous access

Long-term venous access is a term used to describe venous cannulae, both central and peripheral, that can remain *in situ* for protracted periods of time (Table 14.2). Patients can be discharged with some types *in situ*, facilitating early discharge and community care. As experts in vascular access, placement of these lines often falls to anaesthetists or radiologists. Common indications include cancer chemotherapy, long-term antibiotics, total parenteral nutrition or repeated transfusion/venesection. Long-term venous catheters avoid the risks and costs of repeated short-term access such as peripheral cannulation.

Choice of device

Requires consideration of indication, duration of therapy, risk of contamination of catheter, anatomical idiosyncracies and risk from sclerosant drugs. The Michigan Appropriateness Guide for Intravenous Catheters (MAGIC) provides an evidence-based algorithmic approach that can assist clinicians in choosing the best device for their patient.

Midline catheters

Midline catheters are 10–20cm soft catheters inserted via the antecubital fossa, with the tip of the device situated in the upper 3rd of the basilic or cephalic vein, and short of the great vessels. Although not long-term venous access, midline catheters may be useful for short to medium term access (e.g. 1–4w) for drugs that are safe to infuse into peripheral veins (e.g. antibiotics).

Peripherally inserted central catheter (PICC)

A PICC is typically inserted in the upper limb via the basilic or brachial vein, proximal to the cubital fossa (the cephalic vein is often avoided due to its tortuous course as it joins the axillary vein). The advantages of PICCs are their safer insertion and convenient position. Disadvantages include risk of thrombosis, infection, occlusion, malposition and rarely arrhythmia or damage to great vessels or pericardial tamponade. Some can be used for venous blood sampling; others cannot and will reduce the life of the catheter. If in doubt, check.

Table 14.2 Service life of various intravenous access devices

Device	Normal duration
Peripheral cannula	48–72h
Midline catheter	14–28d
Non-cuffed, non-tunnelled CVC	5–14d
Tunnelled, non-cuffed CVC	5–21d
PICC	Several months
Tunnelled, cuffed CVC (Hickman line)	Months/years
Subcutaneous port catheter	Months/years

CVC, central venous catheter; PICC, peripherally inserted central catheter.

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Tunnelled, cuffed CVCs

Tunnelled catheters (e.g. Hickman™, Broviac™, cuffed Groshong™) reduce the risk of the catheter becoming infected. Infection risk is reduced by the tunnel itself and by a subcutaneous Dacron cuff which anchors the catheter and induces surrounding fibrosis which acts as a barrier to bacteria. It takes 3w for fibrous adhesions to develop, and hence they should not be inserted for shorter-term use.

- Catheters are usually inserted into the subclavian or internal jugular vein, with the right side being favoured as it provides a shorter and simpler route to the vena cava.
- **Broviac™ catheters** have a smaller lumen than **Hickman™ catheters**, but both have open venous ends. **Groshong™ lines** have a valve on the venous end which prevents back bleeding and air embolism but prevents the catheter from being cut down in size prior to insertion. Syringes <10mL should not be used on tunnelled catheters, as high pressure may damage the catheter tubing.

Port catheters

Port catheters are used for prolonged periods of intermittent therapy, e.g. chemotherapy. The catheter has a silicone entry port that is located under the skin, usually on the chest, abdomen or arm. The SC location results in much lower infection rates than tunnelled catheters, and enables bathing/immersion.

Accessing the port is gained by a specific 'non-coring' needle pushed through the silicone membrane to the reservoir below. Initial access to the port causes discomfort, and topical LA cream can be applied 30min before. There is a distinct 'clunk' as the needle hits the back wall of the port after penetrating the membrane. The needle may be left *in situ* if repeated access is required.

Practical advice

Multidisciplinary discussion prior to insertion of long-term venous access is needed for patients with chronic kidney disease who may require future dialysis, as it is imperative to preserve peripheral and central veins.

The catheter tip should be positioned in the lower SVC or the cavoatrial junction (up to two vertebral spaces below the carina on CXR). Position is often confirmed or facilitated by fluoroscopy at the time of placement, but this can move over time. Traditionally, optimal tip position has been regarded as at the level of the carina on CXR (corresponding to the upper border of the pericardium, to minimise the risk of cardiac tamponade). The catheter should run in the long axis of the vein, i.e. not abutting the vein at an acute angle. This is important as catheter position may change with respiration, body position or upper limb movement.

Placement of the tip in large-diameter, high-flow vessels reduces the risk of thrombosis, migration with a risk of extravasation and sclerosis of veins.

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Chapter 15

Airway assessment and management

Jules Cranshaw, Emira Kursumovic and Tim Cook

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Airway terminology

There is an increasing focus on standardising airway nomenclature for safer and more effective communication. All who manage the airway should use, as far as possible, the same terminology. Some terms are archaic and can be simplified. Latin or ancient Greek should not be needed! The following terms are used in this chapter:

- Flexible optical bronchoscope (FOB) in preference to fibreoptic bronchoscope. The abbreviation is retained, but this better reflects modern equipment which does not contain fibres.
- Awake tracheal intubation (ATI) in preference to awake fibreoptic intubation (AFOI). Modern ATI may be performed with many instruments, including videolaryngoscopes. Few are now fibreoptic.
- Cannot intubate, cannot oxygenate (CICO) in preference to 'cannot intubate, cannot ventilate', 'total airway failure' or other similar descriptors. Safe communication requires all the words are spoken. 'CICO' may be used in writing, but in speech, different pronunciations (khy-kho, si-co, psycho, etc.) and incomprehension hinder a time-critical message.
- Cricoid force in preference to cricoid pressure. This is applied in units of force (N or kg) and is correctly described as such.
- Front of neck airway (FONA) and in an emergency, eFONA, in preference to front of neck access, emergency surgical airway, infraglottic access, etc.
- Supraglottic airway (SGA) in preference to supraglottic airway device (SAD).
- Tracheal tube (TT) in preference to endotracheal tube (ETT).

SGAs

For basic information on adult and paediatric airway equipment, see ➲ pp. 355-8; ➲ pp. 908-10.

The division between first- and second-generation SGAs is pragmatic with first-generation devices being 'simple airway tubes', including the 'classic LMA' and 'flexible LMA'. Second-generation devices are those with design features intended to reduce the risk of pulmonary aspiration of gastric contents. These include the 'LMA ProSeal™', 'LMA Supreme™' and 'i-gel®'.

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Airway assessment

The difficult airway is **the** most important cause of anaesthesia-related morbidity and mortality. Around 30% of deaths attributable to anaesthesia are associated with problematic airway management. Some catastrophes are due to suboptimal management in unforeseen circumstances. Others are due to poor planning in patients with known or predictable difficulties. History, examination and investigations must detect risks to airway maintenance, ventilation and oxygenation, not just difficult laryngoscopy. Assessment must inform an airway 'strategy' that anticipates and avoids predicted difficulties, or mitigates adverse effects.

- Beware multiple predictors of difficulty (an indication of high risk).
- Difficulty or failure with one airway management technique is associated with difficulty and failure with other techniques.
- Intubation is difficult in ~1:50 cases (~1:10 in ICU) and impossible in ~1:200–1500, depending on setting. Beware risk factors for rapid hypoxaemia (pregnancy, obesity, infants, existing hypoxaemia). These mandate great care planning intubation **and** ventilation strategies (e.g. preoxygenation, peroxygenation, high-pressure airway seal and PEEP).
- Be familiar with your rescue devices: SGAs, VLs, intubation aids and eFONAs.

Predictors of difficult airway management

History

Question the patient and check health care records, anaesthetic charts, alerts and databases for previous airway difficulties, including:

- Anaesthesia-associated dental damage or severe sore throat
- Associated syndromes (e.g. Down, Klippel–Feil, craniofacial)
- Acquired difficulties (e.g. pregnancy, obesity, diabetes, RA, ankylosing spondylitis, acromegaly, Still's disease, snoring, OSA)
- Iatrogenic problems (e.g. C-spine fusion, oral/pharyngeal radiotherapy, laryngeal/tracheal/temporomandibular joint (TMJ) surgery).

Examination

- Anatomical impediments (e.g. small mouth, receding chin, high arched palate, large tongue, bull neck, obesity, large breasts).
- Acquired problems (e.g. head/neck burns, goitre, tumour, haematoma, infection, abscess, restrictive scars). Reduced mouth opening and temporomandibular advancement (e.g. dental abscess, quinsy, TMJ surgery, post-radiotherapy).
- Poor C-spine movement, especially upper extension.
- Poor dentition (e.g. anterior gaps, sharp/loose/protruding/inward/awkward teeth).
- Orthopaedic/neurosurgical/orthodontic equipment (e.g. neck collar, halo traction, external fixator, stereotactic locator, dental wiring).
- If using the nasal route for FOB, check patency of nasal passages.
- Facial hair may hide adverse anatomical predictors.
- Some hairstyles cause difficult intubation.

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Radiology

- Check imaging (including CXR) for potentially difficult anatomy. A recent, focused CT/MRI will give more valuable information.
- Check C-spine X-rays. Occipito-atlanto-axial disease is more predictive of difficult direct laryngoscopy than disease below C2.
- Loss of cervical disc space predicts difficult intubation.
- Flexion/extension or dynamic C-spine studies can confirm instability. Plain X-rays are inadequate predictors of cervical stability.

Predictors of difficult mask ventilation

- Mask ventilation is difficult in ~1–3% of cases; impossible in ~1:600.
- Age >60yr, ♂ sex, full beard, BMI >35kg/m², previous difficult tracheal intubation, snoring, OSA, absence of teeth, thyromental distance <6cm, modified Mallampati classes 3 and 4 (Fig. 15.1), facial abnormalities, neck radiotherapy, DIFFMASK score ≥5.[1]
- The DIFFMASK score uses ten predictors of difficult mask ventilation and ranges 0–18. Scores 6–10 merit detailed further assessment; higher scores predict risk.

Predictors of difficult SGA insertion and ventilation

- First-time SGA insertion and ventilation fails in >5% of attempts.
- Difficult SGA placement is associated with: narrow gape (insertion may be impossible); intraoral/pharyngeal masses (e.g. lingual tonsils); obesity; and poor dentition.
- Second-generation SGAs are more likely to succeed at first insertion. Some can aid intubation. Higher laryngeal seal pressures (~26–30cmH_2O vs ~15–20cmH_2O) may enable ventilation of patients with low chest compliance and use of PEEP. With higher oesophageal seal pressures and a drain port, they may reduce aspiration risk.

Predictors of difficult direct laryngoscopy

- Direct laryngoscopy needs a line of sight from upper teeth to glottis. It entails mouth opening, extension of upper C-spine and displacing the mandibular arch tissues.
- Predictive tests check one or more of these capabilities, but direct laryngeal view grade (Fig. 15.2) correlates modestly with difficult intubation. Most failures occur with grade 2 or 3. Functional grades have therefore been proposed (Fig. 15.3).
- Problems with predictive tests include frequent false positives (a smaller number are actually difficult) and negatives (most difficulties are unpredicted). Combining tests exacerbates these problems.

Interincisor gap (II gap)

The distance between the incisors (or alveolar margins) with the mouth open maximally:

- <3cm predicts difficulty.
- <2.5cm—SGA insertion will also be difficult.

Mandibular protrusion

- Class A: can protrude lower incisors anterior to upper incisors.
- Class B: can protrude lower incisors to, but not beyond, upper incisors.
- Class C: cannot protrude lower incisors to upper incisors.

Classes B and C are associated with difficulty.

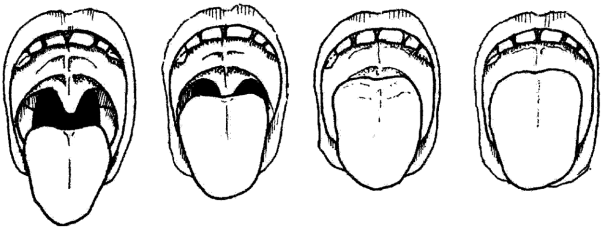

Fig. 15.1 Modified Mallampati classes 1, 2, 3 and 4.

Fig. 15.2 Cormack and Lehane's classification of laryngeal view.

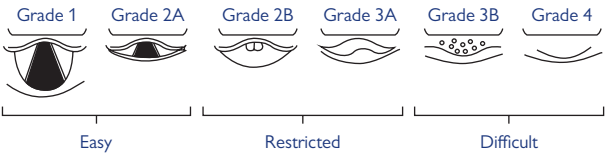

Fig. 15.3 Cook's modified classification of the laryngeal view. Grades 1–4 refer to Cormack and Lehane's classification. In Cook's classification, 'easy' views (glottis visible) require no adjuncts; 'restricted' views (posterior laryngeal structures visible or epiglottis liftable) require a bougie; 'difficult' views (epiglottis not liftable or not visible) require advanced techniques to intubate.

Upper lip bite test

The patient attempts to bite as far up their upper lip as they can:

- Class I: lower incisors bite upper lip, upper lip mucosa invisible
- Class II: upper lip mucosa partially visible
- Class III: the lower incisors fail to bite the upper lip.

Class III is a strong predictor of difficult laryngoscopy.[2]

Mallampati test (with Samsoon and Young's modification)

(See Fig. 15.1.)

Position yourself opposite the patient. Examine the oropharynx while the patient opens his/her mouth maximally, protruding the tongue without phonating.

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- Class 1: faucial pillars, soft palate and uvula visible
- Class 2: faucial pillars and soft palate visible—uvula tip masked by base of tongue
- Class 3: only soft palate visible.
- Class 4: soft palate not visible.

Class 3 and 4 views (no posterior pharyngeal wall view) are associated with difficult laryngoscopy. This test is prone to interobserver variation. Used alone, it correctly predicts about 50% of difficult laryngoscopies and has a false positive rate of >95%.

Extension of the upper cervical spine

When limited, the risk of difficult direct laryngoscopy is ↑. Movement may be assessed by:

- Flexing the head on the neck, immobilising the lower C-spine with one hand on the neck, then fully extending the head. Placing a pointer on the vertex or forehead allows estimation of the angle of movement.
- Placing one finger on the patient's chin and one finger on the occipital protuberance, and extending the head maximally.

With normal C-spine mobility, the finger on the chin is higher than the one on the occiput. Level fingers indicate moderate limitation. If the finger on the chin remains lower than the one on the occiput, there is severe limitation.

Thyromental distance (Patil test)

Tip of thyroid cartilage to tip of mandible, neck fully extended:

- Normal >7cm; <6cm predicts ~75% of difficult laryngoscopies.
- Combined Patil and Mallampati tests (<7 cm and classes 3–4) increase the specificity (97%) but decrease the sensitivity (81%).

Sternomental distance (Savva test)

Sternal notch to tip of mandible, neck fully extended, mouth closed: <12.5cm associated with difficulty (positive predictive value 82%).

Wilson score

- Five factors: weight, upper C-spine mobility, jaw movement, receding mandible and protruding upper teeth. Factors are scored, some objectively and some subjectively, from broadly normal to severely abnormal, and allocated 0, 1 or 2 points.
- A total score of ≥2 predicts 75% of difficult intubations; 12% false positives.

MACOCHA score[3]

- Identifies critically ill patients at risk of difficult intubation.
- The name is an aide memoire for its variables.
- Weighted variables: **M**allampati 3 or 4 (scores 5); **A**pnoea/OSA (scores 2); reduced **C**-spine mobility (this and all others score 1); Mouth **O**pening <3cm; **C**oma; severe **H**ypoxaemia; non-**A**naesthetist intubator.
- Range 0–12 (score >2 predicts difficult intubation).

Predictors of difficult videolaryngoscopy

- Narrow and rigid oro- and laryngopharynx (e.g. tumour, trauma, cervical pathology, radiotherapy change), fixed neck flexion.
- A narrow gape relative to a wide VL makes insertion difficult or impossible.

Predictors of difficult paediatric mask ventilation and intubation

- Difficult mask ventilation has a similar prevalence to that in adults but is ↑ in the very young. Insert a 'shoulder roll' for children <2y. **Beware laryngospasm as a cause of difficult mask ventilation.**
- Difficult intubation is associated with weight <10kg, short thyromental distance (micrognathia) and >2 direct laryngoscopy attempts before an indirect technique.

Predictors of difficult front of neck airway

- Obesity (↑ pretracheal tissue makes reaching the trachea more difficult), goitre and other anterior neck masses or vessels, infection/scarring, tracheal deviation, fixed neck flexion, previous neck radiotherapy, surgical collar/external fixator preventing access.
- Inspect a recent CT/MRI. Ultrasound may be useful.

Predictors of difficult awake tracheal intubation

- In experienced hands, the complication of unplanned removal of an FOB, VL or TT during ATI is 1–2%.[4]
- Predictors include: inadequate oxygenation, uncooperative patients, operator and/or assistant inexperience, suboptimal environments (e.g. ED or ICU), airway blood or unmanageable airway secretions, inadequate topical anaesthesia, oversedation, inability to pass a nasal tube, ill-fitting adjuncts (e.g. FOB conduits; see ➲ p. 395), very narrow airway ('cork-in-bottle') and larger TT.

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ALGRAWANY

Unanticipated difficult airway in adults

- Adverse events usually develop from both difficult intubation and difficult mask ventilation.
- When disasters occur, patients die from failed oxygenation, not failed intubation.
- Difficult ventilation makes oxygenation harder.
- Call for the 'difficult airway trolley' and experienced help early. Avert detrimental consequences: desaturation, multiple interventions causing airway trauma and CICO, aspiration and awareness.
- Use a 'strategy'; a pre-agreed sequence of plans that manages failure of previous attempts.
- Preoxygenate to delay desaturation. Give 100% O_2 whenever airway problems are anticipated or develop.
- Confirm tracheal intubation with capnography and examination.
- Attend regular training in creating FONAs.

Unanticipated difficult tracheal intubation

Consider this four-step strategy (Fig. 15.4) to maintain oxygenation.

Plan A: mask ventilation and tracheal intubation

- Optimise the 1st laryngoscopy. Use the 'sniffing position'. With obesity, use 'ramping' 20–25°. Preoxygenate. Target ETO_2 >85%. Ensure complete NMB.
- Use a laryngoscope and blade with which you have training and experience. Ready a 2nd choice that you are also trained to use. 'Shared-screen' VLs aid teamwork.
- A smaller TT provides a better view and easier passage than larger. Improve the view by optimal external laryngeal manipulation (OELM), or backward, upward, rightward pressure (BURP) with cricoid force.
- Use a gum elastic bougie (GEB), TT introducer or stylet carefully. If the view or TT passage is difficult, reduce any cricoid force. Have suction ready. Maintain oxygenation by mask ventilation with O_2 between attempts. Limit attempts (≤3). Allow a more experienced colleague one attempt. If Plan A fails, declare, 'Failed intubation'.

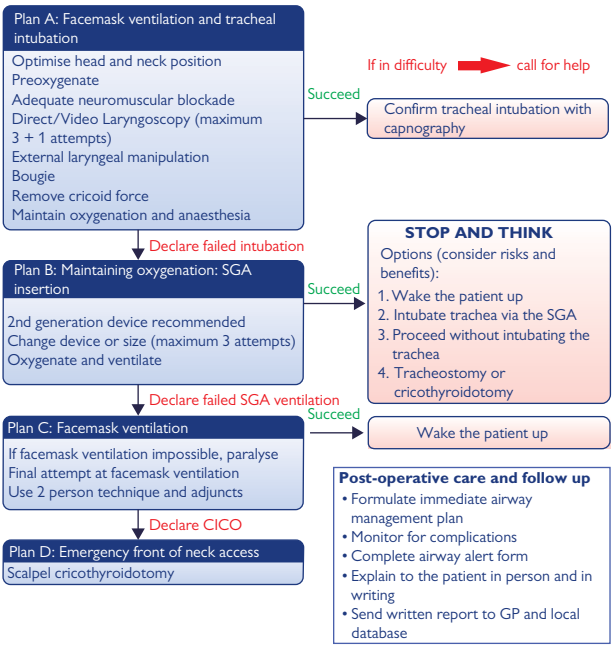
Plan B: ventilation with an SGA

SGAs rescue the airway in >90% of cases (see pp. 355–7; p. 379). Remove any cricoid force. Have suction ready. Limit attempts (≤3).

- In difficulty, try a different insertion technique, size or SGA.
- If oxygenation is successful, 'stop and think' about options: wake up; proceed with the SGA; intubate via the SGA with FOB assistance (one attempt only); create a tracheostomy or cricothyroidotomy in controlled circumstances.
- If oxygenation fails, declare, 'Failed SGA ventilation'.

Plan C: face mask ventilation—a final attempt

- Ensure complete NMB. If you successfully oxygenate, and waking will sustain this, reverse NMB and wake the patient.
- Beware. Residual anaesthesia, airway trauma and airway or pulmonary pathology may make waking unfeasible.
- If Plan C fails, declare clearly, 'I cannot intubate and cannot oxygenate. We need to perform an emergency front of neck airway!'
- Hypoxic brain injury and cardiac arrest are imminent.



This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

Fig. 15.4 Unanticipated difficult tracheal intubation. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6), 827–848. doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

Plan D: emergency front of neck airway

- Several techniques are described. Be decisive.
- Based on your training, choose scalpel or cannula cricothyroidotomy. Familiar kit must be immediately available.
- Continue oxygenation efforts with mask or SGA. Maintain complete NMB. Hyperextend the neck.
- Perform a laryngeal handshake: move the upper larynx from side to side between thumb and index finger; slide the fingers to the thyroid laminae; stabilize the larynx between middle finger and thumb; use the index finger to find the cricothyroid membrane. Stabilise the larynx with one hand.
- For the scalpel cricothyroidotomy technique, see ➔ p. 381 and Fig 15.7.
- For the cannula technique, see ➔ pp. 381–3.

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Unanticipated difficult face mask ventilation

- The commonest cause is lost upper airway patency. Rarer causes are listed below.
- An effective seal requires good mask fit (consider leaving well-fixed dentures), good technique and assistance. Ensure complete NMB.
- Reposition the patient to improve ventilation, not laryngoscopy.
- Insert a correctly sized oropharyngeal airway (OPA). Use a 'VE' grip, thumbs caudad on each side of the mask while simultaneously performing a chin lift and jaw thrust with your fingers. Ask an assistant to hold the O_2 'flush' button if needed and to attempt gentle ventilation (two-person technique). Another assistant may provide more effective jaw thrust.
- Reduce any cricoid force. Have suction ready.
- Ventilation via an SGA or intubation usually forms part of a strategy for unanticipated difficult mask ventilation.
- Consider a nasal airway, but beware bleeding.

Other causes of difficult face mask ventilation

- Anaesthetic circuit problems (e.g. switch, blockage, disconnection, leak). Check entire circuit, mask and catheter mount before induction. Change to a self-inflating bag and new mask if doubt remains.
- Laryngospasm (see below; ➲ p. 1073).
- Bronchospasm (see ➲ pp. 1078–1079). Beware in asthma, anaphylaxis and smokers.
- Foreign body (e.g. throat pack, object from anaesthetic circuit, teeth, denture, blood clot, mucus plug). Exclude by inspection and FOB.
- Laryngeal pathology (rare). eFONA, including tracheostomy, may be lifesaving.
- Lower airway pathology (rare, e.g. mediastinal masses). Tracheal intubation or rigid bronchoscopy may be lifesaving.

Laryngospasm in adults

Laryngospasm can be rapidly fatal. Consider in airway obstruction without obvious supraglottic cause. Sometimes preceded by stridor or a characteristic 'crowing' noise, it may be instant, complete and silent.

- Inspect the airway. Use suction to remove all contaminants.
- Obtain a tight mask seal and patent upper airway (as above). Close the expiratory valve. Apply CPAP with 100% O_2. Use an assistant to squeeze the bag and to attempt gentle manual ventilation.
- 'Forcible' jaw thrust or bilateral anterior pressure on the mandibular rami just in front of the mastoid process (Larson's point) may 'break' laryngospasm by a combination of stimulation and airway opening.
- Deepening anaesthesia with a small dose of propofol (20–50mg) may relax laryngospasm.
- If desaturation begins, consider a small dose of IV suxamethonium (0.1–0.5mg/kg actual body weight). If laryngospasm is severe, intubate using IV 1–1.5mg/kg (actual body weight, maximum 150mg).
- If there is no venous access, consider submental intralingual suxamethonium 3mg/kg (actual body weight, maximum 150mg) with massage; or IM in the thigh 4mg/kg (actual body weight, maximum 150mg) with massage.
- Before waking, consider a change in airway management (e.g. exchange a TT for an SGA) to try to reduce the risk of recurrence.

Diagnosing oesophageal intubation

- Always suspect oesophageal intubation after difficult intubation. Patients continue to die of this avoidable complication.
- Do not induce anaesthesia without a working waveform capnograph.
- Tracheal intubation and ventilation produce a sustained waveform capnograph trace.
- Even after cardiac arrest, a small waveform trace will be seen, with or without chest compressions.
- Difficult mask ventilation can push expired CO_2 into the oesophagus and stomach. After oesophageal intubation, this CO_2 may be detected but will rapidly diminish to zero with ventilation.
- Rarely, complete obstruction (circuit, tube, airway or severe bronchospasm) prevents ventilation, and no trace can be produced.
- Otherwise, a flat trace indicates no lung ventilation; oesophageal intubation should be assumed and excluded—'No trace equals wrong place'.
- Clinical signs may be used but are unreliable, as you tend to see and hear what you expect and want.
- If in doubt, remove the TT. Oxygenate.

Paediatric considerations

(For management of paediatric airway, see ➲ pp. 921–4.)

- Consider specific paediatric guidelines.
- Hypoxaemia is rapid in neonates, infants and small children if ventilation is inadequate. Maintain oxygenation and consider options.
- Each direct laryngoscopy, after the first, is associated with complications (e.g. desaturation <85% and cardiovascular instability).
- Minimise the number of direct laryngoscopic attempts. Consider VL or FOB-assisted intubation possibly via an SGA.
- SGA insertion failure is lower in older children than in adults, but failure is associated with more cardiorespiratory complications.
- Reflex responses to airway manipulation or suxamethonium, aggravated by hypoxaemia, may cause bradycardia and cardiac arrest.

Paediatric laryngospasm

(See ➲ p. 921 for additional information.)

- Laryngospasm is more common than in adults and hypoxaemia develops faster. Decision-making and action must be quicker.
- Consider deepening anaesthesia with propofol 0.5mg/kg; a small dose of IV suxamethonium (0.1–0.5mg/kg); or, if laryngospasm is severe, intubate with IV suxamethonium 1mg/kg (maximum 150mg) (2mg/kg in infants). Intubation without NMB may be attempted but is not always possible. If there is no IV access, submental intralingual suxamethonium 3mg/kg (maximum 150mg) with massage will relax the larynx in ~2min. Alternatively, give IM suxamethonium 4mg/kg in the thigh (maximum 150mg) with massage.
- Beware stomach inflation during ventilation attempts.
  - This can trigger vagal reflexes and regurgitation, and splint the diaphragm.
  - Consider early gastric drainage.

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Obstetric considerations

(For failed intubation in obstetric anaesthesia, see ➲ p. 864.)

- The incidence of failed intubation is high (~1:300) due to patient, surgical and environmental factors.
- Consider obstetric difficult airway guidelines. Most advocate ≤2 intubation attempts, with one more reserved for an experienced practitioner.
- High-flow nasal oxygen (HFNO) delays desaturation after induction of GA (see also ➲ p. 397).

Follow-up of patients with difficult airways

- Document difficulties and successful and unsuccessful approaches in health care records.
- Make sure these are easily accessible and prominent (e.g. in 'Alert' sections). Complete an 'airway alert' form for local and national (e.g. DAS) databases.
- Advise patients that they may register with independent medical alert providers. Warn about delayed symptoms of airway trauma and how to seek help.

Intubating critically ill patients

Critically ill patients often need emergency intubation in challenging circumstances and 'hostile' environments. Environmental stress undermines teamwork (human factors). When intubation is difficult, hypoxaemia increases, but waking the patient may not be lifesaving.

- In the ICU and ED, intubation fails first time in >10% of cases. Desaturation to <80% occurs in ~20%. Difficulties are more likely to be managed suboptimally. Airway catastrophes in the ICU are more likely to lead to brain damage and death.[5]
- Prepare for airway difficulty, aspiration, hypotension (systolic BP <90mmHg in ~30%) and CVS collapse (cardiac arrest in 2%).[5]
- Optimise oxygenation before, during and after intubation.
- Training and experience with kit are essential.

The 'Vortex Approach' vs an 'Airway Strategy'

The Difficult Airway Society (DAS) and other guidelines provide didactic strategies to manage unanticipated difficult intubation after inducing GA in the form of logically prioritised sequences of plans. The 'Vortex Approach' to airway crises is unconstrained by this context and removes potentially restricting and complex linearity. It uses a simple visual cognitive aid; a picture of a vortex with an outer zone where oxygenation is successful; and an inner zone where the patient is in danger, marked with three equally spaced icons for face mask ventilation, SGA insertion and tracheal intubation. In crisis, each technique may be tried in any order. With each device a maximum of three attempts or one optimal attempt is permitted and might move a patient out of the vortex or risk tipping them further in towards eFONA. Failure of all attempts or critical deterioration mandates eFONA. The 'Vortex Approach' is currently considered complementary to airway algorithms.

Preparation

- Consider an A, B/C, D airway management strategy (Fig. 15.5).
- Identify risks of difficult intubation (e.g. MACOCHA score; see ➔ p. 366) and difficulty with rescue techniques.
- If you suspect upper airway pathology and time allows, obtain a nasendoscopic view of the larynx.
- Identify aspiration risk. Leave an NGT. Stop feed. Aspirate the stomach. Consider RSI.
- After assessment: modify your plan accordingly.
- Brief your team. Allocate roles (e.g. CVS management). Use checklists and cognitive aids such as in Fig. 15.6. Know how to access expert airway help quickly. Be aware of time passing.
- Check your kit. Have familiar second-generation SGAs, FOB and eFONA kit immediately available. Apply routine monitoring (at least). Waveform capnography is mandatory.
- Perform a laryngeal handshake. Consider marking the trachea and midline. Use ultrasound if needed and trained. Mark the cricothyroid membrane in the neck position that cricothyroidotomy will be performed.
- Be prepared to manage acute adverse CVS changes during and after intubation (particularly with recruitment manoeuvres).

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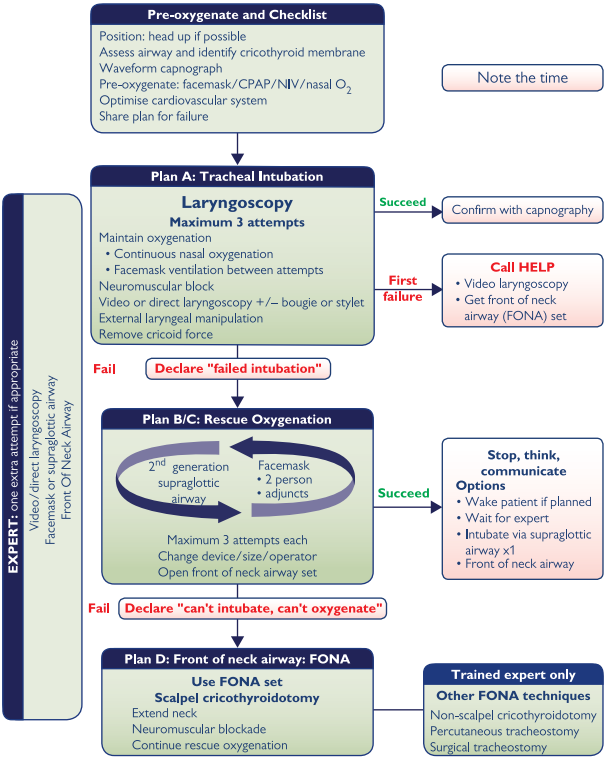

This flowchart forms part of the DAS, ICS, FICM, RCoA Guideline for tracheal intubation in critically ill adults and should be used in conjection with the text.

Fig. 15.5 Tracheal intubation in critically ill adults. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6), 827–848. doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

Prepare the patient

- ☐ **Reliable IV/IO access**
- ☐ **Optimise position**
  - ☐ Sit-up?
  - ☐ Mattress hard
- ☐ **Airway assessment**
  - ☐ Identify cricothyroid membrane
  - ☐ Awake intubation option?
- ☐ **Optimal preoxygenation**
  - ☐ 3 mins or ETO_2 > 85%
  - ☐ Consider CPAP/NIV
  - ☐ Nasal O_2
- ☐ **Optimise patient state**
  - ☐ Fluid/pressor/inotrope
  - ☐ Aspirate NG tube
    Delayed sequence induction
- ☐ **Allergies?**
- ☐ ↑ **Potassium risk?**
  - ☐ avoid suxamethonium

Prepare the equipment

- ☐ **Apply monitors**
  - ☐ SpO_2/waveform $ETCO_2$/ECG/BP
- ☐ **Check equipment**
  - ☐ Tracheal tubes x 2 - cuffs checked
  - ☐ Direct laryngoscopes x 2
  - ☐ Videolaryngoscope
  - ☐ Bougie/stylet
  - ☐ Working suction
  - ☐ Supraglottic airways
  - ☐ Guedel/nasal airways
  - ☐ Flexible scope/Aintree
  - ☐ FONA set
- ☐ **Check drugs**
  - ☐ Consider ketamine
  - ☐ Relaxant
  - ☐ Pressor/inotrope
  - ☐ Maintenance sedation

Prepare the team

- ☐ **Allocate roles**
  One person may have more than one role.
  - ☐ Team Leader
  - ☐ 1st Intubator
  - ☐ 2nd Intubator
  - ☐ Cricoid force
  - ☐ Intubator's assistant
  - ☐ Drugs
  - ☐ Monitoring patient
  - ☐ Runner
  - ☐ MILS (if indicated)
  - ☐ Who will perform FONA?
- ☐ **Who do we call for help?**
- ☐ **Who is noting the time?**

Prepare for difficulty

- ☐ **Can we wake the patient if intubation fails?**
- ☐ **Verbalise** "Airway Plan is:"
  - ☐ **Plan A:**
    Drugs & laryngoscopy
  - ☐ **Plan B/C:**
    Supraglottic airway
    Face-mask
    Fibreoptic intubation via supraglottic airway
  - ☐ **Plan D:**
    FONA
    Scalpel-bougie-tube
- ☐ **Does anyone have questions or concerns?**

Fig. 15.6 Intubating checklist for critically ill adults. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6), 827–48. doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

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Plan A

The goal is timely, atraumatic first-time intubation by optimising position, NMB, laryngoscopy, teamwork and equipment. This is supported by maximised preoxygenation and peroxygenation. Beware a low PaO_2 after preoxygenation predicts hypoxaemia after induction.

Preoxygenation

Consider using:

- Tight-fitting mask, O_2 at 15L/min with a system enabling some CPAP, e.g. Water's circuit (Mapleson C).
- HFNO at 30–70L/min.
- CPAP with 100% O_2.
- Non-invasive ventilation with 100% O_2.
- Nasal cannula O_2 at 5L/min while the patient is awake and 15L/min when the patient is induced.

Nasal cannulae can prevent mask seal. If a seal is made, simultaneous HFNO may cause high airway pressure, as HFNO systems have no pressure relief valve.

- HFNO contraindications include basal skull and facial fractures.
- Running HFNO, a breathing system, e.g. Water's circuit (Mapleson C), and a ventilator simultaneously requires three near-patient O_2 sources.
- Existing CPAP and non-invasive ventilation systems might be left *in situ* during induction, but plan when and how to remove masks quickly to allow laryngoscopy.

Peroxygenation

HFNO or nasal O_2 at 15L/min should be provided until intubation is successful.

Positioning

Raise the bed head up 20–30° to aid oxygenation. Tilting the whole bed may be safer in known or suspected spinal injury. Start with the 'sniffing position'. In obesity, 'ramp' the upper body so the external auditory meatus is level with the sternal notch and the face horizontal.

Induction

- Ketamine may have advantages over other induction agents. Small doses during preoxygenation may ease cooperation.
- Rapid-acting opioids, such as fentanyl or alfentanil, may aid CVS stability.
- For rapid NMB, rocuronium has advantages over suxamethonium; fewer contraindications and adverse effects (fasciculations consume O_2). Rocuronium 1.2mg/kg achieves intubating conditions as fast as suxamethonium 1mg/kg.
- If indicated, ask a trained assistant to apply cricoid force—10N (1kg) on induction, ↑ to 30N (3kg) after loss of consciousness. Cricoid force may reduce aspiration and stomach inflation but, especially if excessive, can impede face mask ventilation, laryngoscopy and passage of the TT. VL may aid appropriate cricoid force. Remove cricoid force if vomiting and when inserting an SGA.
- While waiting for effective NMB, sustain oxygenation by ventilation. Ventilation may also control $PaCO_2$. This might be important in raised ICP, pregnancy, pulmonary hypertension and acidaemia. Keep airway pressure low (preferably <20cmH_2O) to avoid stomach inflation. Reduce cricoid force if ventilation is difficult. Have suction ready.

- Use a laryngoscope and blade with which you have gained confidence. Ready your second choice. Limit intubation attempts to ≤3. Allow an expert one attempt. Maintain oxygenation between attempts.
- If direct laryngoscopy is your first approach, ready a GEB, introducer or stylet. Use carefully. Avoid 'blind' insertion.
- Consider VL as your first choice if trained. Switch to VL if you have difficulty. It is likely VL will improve your view, reduce trauma and increase success. If trained, use a hyperangulated blade with a stylet when a Macintosh-style blade fails. Use your assistants. VL with a screen shared by assistants can direct them to improve OELM, BURP and cricoid force. Blood, secretions and reflux may hamper VL (and direct laryngoscopy).
- Reduce or release cricoid force if the view or TT passage is problematic. Be ready with suction.
- Where there is difficulty, choose a TT type and size allowing the best laryngeal view, easy passage and least trauma. TTs with subglottic suction have larger external diameter. The size and type may not be optimal for long-term ICU ventilation. However, TT exchange after stabilisation may be safer than failing with a large TT.
- If Plan A fails, declare clearly, 'Failed intubation!'
- Call for expert help.
- Open and prepare your eFONA kit.
- If Plan A succeeds, planned and preset ventilation strategies may re-establish and improve oxygenation quickly.

Plan B/C for failed intubation

The goal is successful oxygenation (Plan B). The 'high' seal pressure provided by a second-generation SGA may enable you to ventilate a low-compliant chest with PEEP. Aspiration risk is reduced.

- Minimise insertion attempts (≤3).
- If an attempt fails, try a new SGA size, type, insertion technique or operator.
- One failed optimal attempt might enable you to rule out SGA insertion as a successful approach.
- Attempt mask ventilation (Plan C) between SGA insertion attempts. Insert a correctly sized OPA. Use a 'VE' grip, thumbs caudad on each side of the mask. Use your fingers to lift the chin. Ask an assistant to add 'jaw thrust'. Use a Water's circuit (Mapleson C) to give 100% O_2 with CPAP.
- Minimise mask ventilation attempts (≤3).
- One failed optimal attempt might lead you to abandon mask ventilation as a successful option.
- Consider a nasal airway, but beware bleeding.
- Allow an expert one attempt at SGA insertion and mask ventilation, BUT they may choose an immediate eFONA.
- Recognise failure of Plans B and C within 1min.
- Clinical deterioration and worsening oxygenation mandate rapid transition to eFONA.
- Declare clearly, 'I cannot intubate and cannot oxygenate. We need an emergency front of neck airway!' (Plan D).

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- If oxygenation via an SGA or mask ventilation is successful, stop and think. Consider your plan.
  - Wait for an expert if stability allows.
  - Waking may not be an option due to neurological impairment, drug effects, airway pathology, pulmonary pathology and other critical illness.
  - If oxygenation via an SGA is acceptable, consider FOB-assisted intubation via the SGA. If feasible, make one optimal attempt only.
  - If a TT is essential, consider eFONA during stability, especially if oxygenation is precarious, ventilation is difficult, aspiration is a risk and FOB-assisted intubation is unlikely or has failed.
  - Do not wait for life-threatening hypoxaemia to proceed to eFONA.

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Equipment and techniques for management of difficult intubation

For basic information on adult and paediatric airway equipment, see ➲ pp. 355–8; ➲ pp. 908–10.

GEBs and tracheal tube introducers

- There are numerous designs. Performance varies. Incorrect use risks airway trauma and perforation. Rigidity increases trauma risk.
- These devices ease intubation with 'restricted' glottic views (laryngoscopy grades 2b and 3a; see ➲ p. 365). 'Blind' passage is discouraged (e.g. in grade 3b or 4 view). It risks airway trauma.
- Keep the device anterior and midline to avoid the piriform fossae and oesophageal insertion. An assistant may 'feel' the device pass through the larynx. You may feel it 'bumping' down tracheal rings. Do not insert beyond the carina (~24–26cm). Do not deliberately elicit 'rotation' or 'hold-up' (~40cm) by entering the bronchi. This can cause trauma.
- 'Railroad' the TT over the device, carefully maintaining the pharyngeal space with the laryngoscope. Rotation 90° counterclockwise may avoid a bevel sticking on the larynx. A smaller TT or one with a bullet tip is easier to 'railroad'.

Intubation via an SGA

- An SGA can act as a 'dedicated airway', enabling oxygenation and ventilation during intubation via the airway channel.
- FOB increases success to near 100%.[6] 'Blind' intubation via an SGA is not recommended due to higher failure and complication rates.
- One technique uses an Aintree Intubation Catheter (AIC) (Cook Critical Care®) (ID 4.7mm, external diameter 6.3 mm) over an FOB. Place the AIC supracarinally, and then remove the SGA and FOB. A TT (ID 7.0mm) is railroaded over the AIC. This is suitable for a number of SGAs (e.g. i-gel® and LMA ProSeal™). It is less well suited to the LMA Supreme™ or Laryngeal Tubes™ because of their narrow airway lumens.
- The airway channel of some SGAs can accommodate a TT. These allow intubation and leaving the SGA *in situ*. Beware. Before attempting to railroad a TT through an SGA, you must know the TT will fit through and that its cuff will reach the trachea (e.g. consider LMA Fastrach™ (intubating LMA) or Parker Flex-Tip® reinforced TT).

Videolaryngoscopes

- There are various devices. They are not equal. All require training and experience for safe and effective use.
- VLs provide better laryngeal views than direct laryngoscopes. With skill, they increase intubation success in difficult airways. Use by unskilled practitioners in crises is discouraged.
- VLs may become less useful if their 'eye' is covered by secretions or blood but the impact is no greater on a VL than direct laryngoscope.
- VL uses less force than direct laryngoscopy, reducing airway trauma and facilitating ATI. Some prefer intubating with VL during C-spine immobilisation.

There are three main types.

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Bladed videolaryngoscopes

- Include straight and Macintosh style (enabling direct laryngoscopy) and hyperangulated blades (which do not). May be single use or reusable.
- Blade design may facilitate standard, midline or paramedian insertion and enable some airway manipulation, but a stylet or bougie may be essential to intubate.
- These include C-MAC®, GlideScope®, I-view™ and McGrath™.

Conduited videolaryngoscopes

- Have a channel to guide a TT into the camera view.
- TT size and type relative to the conduit may affect intubation success. Avoid relatively small TTs. They may 'cut the angle' created by the channel and point posteriorly.
- These include Airtraq™, Pentax AWS®, King Vision™ and Venner A.P. Advance™.

Optical stylets

- Are preformed rigid or malleable metal guides connecting an imaging system to an eyepiece or screen.
- A TT is preloaded, manipulated to the glottis and advanced into the trachea. Stylets require minimal mouth opening but have limited ability to displace tissues.
- They may be used for ATI or as a 'light wand'.
- These include Bonfils®, Levitan, Shikani and Trachway®.

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Emergency front of neck airway

Rescue techniques for 'cannot intubate, cannot oxygenate'

- eFONA can prevent hypoxic brain damage and death in CICO situations if used rapidly and effectively before critical desaturation.
- Anticipation and preparation are key.
- eFONA relies on palpable landmarks. Identifying these takes practice. When eFONA may be needed, if the situation allows, the landmarks should be identified and marked with the patient in the position in which eFONA will be undertaken. In obesity, ultrasound may help but is an acquired skill.
- During airway difficulty, eFONA equipment should be called for early. If difficulty progresses, it should be opened and prepared, well before it is needed. This reduces delays and is a cognitive aid.
- Continue oxygenation efforts from above during insertion.

See also ➲ p. 1095.

Scalpel cricothyroidotomy

(See Fig. 15.7.)

- Cricothyroidotomy 'kits' should be immediately available wherever GA is induced.
- Less fine motor skill is required than cannulation.
- With a cuffed TT, scalpel cricothyroidotomy allows effective conventional ventilation, capnography and oxygenation faster than cannula techniques, while preventing aspiration.
- Tracheal location can be confirmed by capnography or FOB.
- Risks include failure, bleeding (sufficient to obscure the scalpel, bougie or TT entering the trachea), creating a false passage, soft tissue trauma and tracheal ± oesophageal damage. An uncuffed TT risks hypoventilation and aspiration.

Cannula cricothyroidotomy

- Cannulating may be more familiar than using a scalpel.
- Insert a narrow (<4mm ID) cannula through the cricothyroid membrane.
- Tracheal position must be confirmed by aspirating air during and after insertion. It is important to confirm position before ventilating.
- Attach a high-pressure ventilation device. Cannulae may enable wire-guided dilation to insert a tracheostomy tube or catheter using dedicated kits.
- Risks include failure, cannula kinking, obstruction, misplacement and tracheal ± oesophageal damage. Some cannulae are <5cm long and may not reach the trachea effectively with obesity or oedema.
- Risks of high-pressure source cannula ventilation include subcutaneous emphysema (sufficient to obscure neck anatomy and reinsertion) and barotrauma. Inadequate exhalation via an obstructed upper airway risks breath-stacking, hyperinflation, CVS collapse and tension pneumothoraces.
- Risks increase in emergency circumstances.

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CALL FOR HELP

↓ Continue 100% O_2
Declare CICO

Plan D: Emergenecy front of neck airway

Continue to give oxygen via upper airway
Ensure neuromuscular blockade
Position patient to extend neck

Scalpel cricothyroidotomy

Equipment: 1. Scalpel (number 10 blade)
2. Bougie
3. Tube (cuffed 6.0mm ID)

Laryngeal handshake to identify cricothyroid membrane

Palpable cricothyroid membrane

Transverse stab incision through cricothyroid membrane
Tum blade through 90° (sharp edge caudally)
Slide coudé tip of bougie along blade into trachea
Railroad lubricated 6.0mm cuffed tracheal tube into trachea
Ventilate, inflate cuff and confirm position with capnography
Secure tube

Impalpable cricothyroid membrane

Make an 8–10cm vertical skin incision, caudad to cephalad
Use blunt dissection with fingers of both hands to separate tissues
Identify and stabilise the larynx
Proceed with technique for palpable cricothyroid membrane as above

Postoperative care and follow up

- Postpone surgery unless immediately life threatening
- Urgent surgical review of cricothyroidotomy site
- Document and follow up as in main flow chart

This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

Fig. 15.7 Cannot intubate, cannot oxygenate. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6), 827–48. doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

Ventilating with narrow (<4mm ID) cannulae

- Cannulae <4mm ID require a high-pressure O_2 source using commercial 'injectors' or an improvised apparatus from 'wall' 4 bar O_2. These may take time to collect and assemble. A breathing system or self-inflating bag is ineffective.
- Exhalation must occur via the upper airway as the cannula is too narrow. Maximise upper airway patency (e.g. airway manoeuvres), and leave any OPA or SGA in place from Plan B or C.

- If exhalation is impossible, reduce O_2 flow to <0.5L/min. This provides some O_2 and reduces risks created by hyperinflation.
- Hypercapnia is a risk. Make a rapid plan for conversion to a definitive airway. Ask for specialist surgical assistance if available.
- The Ventrain® is a single-use device capable of O_2 insufflation and generation of subatmospheric pressure (suction) to improve expiration through a 2mm ID catheter. Failed ventilation and hyperinflation risks are reduced.
- The Rapid-O_2® device for jet ventilation has a flow and pressure release enabling potentially safer oxygenation.

Ventilation with wide (≥4mm ID) cannulae

- Catheters ≥4mm ID are suitable for conventional ventilation but may only be adequate when cuffed or the upper airway is obstructed.
- Larger catheters can be placed with a Seldinger technique, e.g. Cook® Melker cricothyrotomy catheter (4.0 mm ID uncuffed / 5.0mm ID cuffed).

Paediatric considerations

(See ➲ pp. 923–4.)

- CICO is rare in infants.
- The cricothyroid membrane is cephalad and difficult to identify.
- Models suggest scalpel and cannula techniques have high risks of posterior tracheal wall and oesophageal damage.
- Cannula cricothyroidotomy may be preferable because it risks less damage to surrounding neck structures.
- Adult eFONA techniques may be appropriate in older children.

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Emergency management of the obstructed airway

This section concerns unexpected critical airway obstruction (e.g. presenting to ED or occurring on a ward or ICU). Elective or fast-track management of partial airway obstruction in ENT patients is discussed on ➔ pp. 762–4.

- These cases are always difficult and stressful for the patient and the airway team.
- Unfamiliar environments and personnel require heightened 'situation awareness'. Establish experience and skill of individuals at the scene. Summon multidisciplinary senior and expert help urgently.
- Approaches to relieving the obstruction differ according to:
  - Level(s) of obstruction.
  - Urgency. This limits time to investigate the site, extent and severity of the obstruction and the involvement of related structures.
  - The patient's physiological state. Hypoxic patients can be anxious, confused and uncooperative.
  - The patient's location. The airway team's preferred location is usually a dedicated equipped operating theatre, but the patient's condition may deteriorate during, or due to, transfer. An experienced judgement must be made whether to risk airway stabilisation *in situ*, in challenging circumstances with reduced resources, or transfer to a safer environment. Know what essential equipment can be brought quickly to the bedside that will function effectively.
- Always give O_2 (e.g. HFNO, nasal cannulae or mask) throughout.
- If inflammation is a component of the obstruction, nebulised adrenaline (transiently) and corticosteroids (slowly) may help.
- Experienced anaesthetists and operators must agree a 'strategy' to relieve the obstruction with backup plans. Poor planning and execution increase risks. Ensure your team knows what to do.
- All approaches risk life-threatening complications: bleeding, swelling and total obstruction.
- As lesions may progress quickly, recently successful approaches to known lesions may fail.
- ⚠ IV induction and laryngoscopy, with or without NMB, and no backup plan, is NOT a 'strategy'.

Assessment

- Take a careful history; ask about dyspnoea and cough, especially lying flat, night-time panic, swallowing and reflux.
- Examine the patient. Determine best breathing position and do not insist the patient moves from this for the purposes of examination. Severity is suggested by respiratory distress, accessory muscle use, voice change or inspiratory stridor (laryngeal signs), monophonic wheeze or expiratory stridor (intrathoracic signs), positional dyspnoea, hypoxaemia and silent chest. In chronic obstruction, signs may be reduced or absent.

- Review existing investigations. In extremis, it is unlikely that further investigation is possible, but if the situation allows, obtain:
  - Nasendoscopic laryngoscopy
  - CT/MRI with 3D reconstruction
  - CXR: although less informative, it is easy to obtain, non-invasive and avoids the supine position. It may suggest tracheal deviation or reduced tracheal diameter.
- Interpret all available information intelligently to ascertain:
  - The level(s) of obstruction: oral, supraglottic, laryngeal, subglottic (mid-tracheal and/or lower tracheal)
  - The nature of the lesion: mobility, friability, collapsibility and severity
  - Ease of eFONA. Examine the front of the neck.

Oral, supraglottic and laryngeal obstruction

Common causes: trauma, burns, tumour, infection, stenosis.
Challenge: a safe approach to, and passage of a TT or ventilating catheter beyond the laryngeal inlet.

- If not Plan A, eFONA is often the backup plan. Thus an experienced operator, engaged in the strategy, must be scrubbed, equipped, assisted and ready to perform eFONA in a crisis. If this occurs, consider an SGA to assist ventilation under GA and NMB.
- Consider prophylactically assisting oxygenation via a cricothyroid cannula. It may enable jet ventilation in a crisis.

Options

- Awake techniques are rarely easy in distressed, hypoxic patients, but consider ATI and awake FONA.
- SV under GA: slow incremental propofol TCI or inhalational induction, then laryngoscopy with VL, FOB or VL-assisted FOB. Direct laryngoscopy is not the preferred option. Pass an appropriate TT or ventilation catheter. Have a backup plan for apnoea, especially if mask ventilation will be difficult.
- In specialist hands:
  - TIVA, rapid NMB and rigid bronchoscopy. The bronchoscope establishes airway patency and acts as a dedicated airway while assessment, ventilation, oxygenation and surgery take place.
  - IV induction or TIVA, rapid NMB and insertion of an anterior commissure laryngoscope as a conduit for a bougie, then intubation.
  - With fixed laryngeal or tracheal stenosis: TIVA, rapid NMB and insertion of an SGA as a dedicated airway prior to definitive surgical management of the obstruction.

Subglottic: mid-tracheal

Common causes: airway or retrosternal tumour, e.g. goitre. Obstructing masses may expand suddenly due to haemorrhage.
Challenges: passing a TT or suitable ventilating catheter into a potentially displaced larynx and trachea and beyond the narrowing. The lesion may preclude eFONA. Attempts may risk bleeding and complete obstruction.

- Delineating the obstruction is vital, if at all possible.
- The end of the TT or ventilating catheter must lie safely between the end of the obstruction and the carina.
- Patient position (sitting up, lateral, prone) may be crucial for SV.

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Options

- ATI. Consider if mask ventilation will be difficult. However, ATI may be intolerable to the patient or cause crisis. Coughing, respiratory distress and passing an FOB, TT or ventilating catheter through a narrowing may critically increase obstruction ('cork-in-a-bottle').
- In specialist hands:
  - FOB-guided insertion of a long, narrow TT (e.g. microlaryngeal tube, endobronchial tube or AIC (6.3mm external diameter)) allows conventional ventilation.
  - Jet catheters (e.g. LazerJet®, Hunsaker) or other narrow catheters, such as Cook® airway exchange catheters, enable jet ventilation but risk barotrauma. A route of exhalation is essential. A Tritube® (4.4mm external diameter) used with a Ventrain® or Evone® ventilator enables 'jet' inspiration and assisted expiration, reducing the chance of barotrauma.
  - If rigid bronchoscopy is available: IV induction and rapid NMB or inhalational induction, and passage of a rigid bronchoscope. The bronchoscope also allows resection, laser or stenting.
  - Inhalational induction may be very slow and worsen obstruction, especially in collapsible lesions. Have a backup plan, especially if mask ventilation is likely to be difficult.

Lower tracheal lesions and bronchial obstruction

Common causes: tumours, trauma and large mediastinal masses. These are best managed in specialist centres with facilities for CPB but may present to ED in extremis. Depending on the lesion, laser resection or stenting may be required to maintain a patent airway.

Options

- Either IV induction with rapid NMB or inhalational induction followed by passage of a rigid bronchoscope or FOB-guided double-lumen or endobronchial tube.

Extubation

Have a management plan for extubation, which may need to be delayed. Prolonged instrumentation may cause upper airway oedema. Transfer to ICU or HDU is often necessary before extubation.

Further considerations

- A specific tissue diagnosis may enable preoperative shrinking of a lesion with antibiotics, steroids, chemotherapy or radiotherapy where time allows.
- Heliox (premixed helium/O_2 containing 21–40% O_2) improves gas flow through narrowed airways, but the FiO_2 is low. Increasing FiO_2 reduces the effect of the helium. Specific delivery systems and ventilators for Heliox exist. Heliox can be useful for any obstruction but is usually a temporary measure while organising definitive management.

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Rapid sequence induction

For general information on induction of anaesthesia, see ➔ p. 406.

- 'Classic' RSI comprises IV induction of anaesthesia with a predetermined dose of induction agent, immediately followed by rapid NMB combined with cricoid force, and tracheal intubation to reduce the risk of pulmonary aspiration of stomach contents.
- There is variation in practice.
- If airway assessment indicates intubation will be difficult, consider a local/regional anaesthetic technique or ATI.
- A checklist is recommended, particularly when performing an RSI in the non-theatre environment. Agree a strategy for failed intubation. Difficulty occurs in ~1 in 20 and failure in ~1 in 200 RSIs.

Checks

- Anaesthetic machine, vaporisers and anaesthetic infusion pumps, breathing system, ventilator, suction, intubation aids, TT cuff and rescue equipment.
- The operating table, trolley or bed should be able to lift head up and tip down.
- Two functioning laryngoscopes (preferably including VL).
- Reliable wide-bore IV access with fluid running.
- Drawn-up predefined dose of induction agent (propofol 1–2.5mg/kg; ketamine 1–2mg/kg; thiopental 2–5mg/kg). Ketamine is favoured in unstable patients. TCI of propofol ± remifentanil is possible.
- Drawn-up predefined dose of suxamethonium (1–1.5mg/kg) or rocuronium (1–1.2mg/kg).
- Emergency drugs (anticholinergics and vasopressors).

Procedure

- Leave an NGT *in situ*. Aspirate stomach contents.
- Position head up 20–25°. Use the 'ramped' position for obese patients.
- Position in the 'sniffing' ('flextension') position with lower neck flexed, upper neck extended on a firm pillow.
- Switch on suction. Place in easy reach.
- Apply routine monitoring, including waveform capnography.
- Preoxygenate. Options:
  - Tight-fitting mask with O_2 15L/min for 3–5min or until ETO_2 is >85%. (Do not remove mask until laryngoscopy)
  - HFNO
  - In extreme emergency, four VC breaths with O_2 flush.
- Consider peroxygenation with HFNO or nasal cannulae with O_2 15L/min.
- Ask a trained assistant to apply cricoid force 10N (1kg), beginning as induction starts.
- Give the induction agent, immediately followed by the NMBA.
- Ask your assistant to increase cricoid force to 30N (3kg) at full loss of consciousness.
- Gently mask ventilate to reduce risk of hypoxaemia.
- Intubate 30–45s after IV suxamethonium or fasciculations end, or 45–60s after rocuronium.
- Inflate the cuff, hand-ventilate and confirm correct TT placement by capnography and clinical examination.
- When correct TT position is confirmed, ask the assistant to remove cricoid force.

Risks

- RSI with cricoid force increases the risk of difficult intubation, partly due to pressure of time and incorrect cricoid force.
- Excessive induction agent may cause CVS collapse, especially in the presence of hypovolaemia or septic shock.
- Inadequate induction agent may cause tachycardia and hypertension. Thiopental, in particular, risks accidental awareness during GA (AAGA).

Cricoid force

- Applying cricoid force is a skill. It is often poorly taught and practised.
- Learn to identify the cricothyroid membrane. It is usually in the middle of the neck, below the 'Adam's apple' at the 2nd skin crease. The cricoid cartilage is immediately below.
- Hold the cricoid cartilage between the thumb and middle finger and push onto the neck with the index finger.
- Practise applying the correct force. Compress an air-filled, sealed syringe positioned vertically on the plunger. Compressing 20mL of air to 12mL, or 50mL to 32mL requires ~30N (3kg).
- Correct application improves direct laryngoscopy, reduces the risk of gastric inflation and does not occlude the airway.
- Excessive force >50N (>5kg) produces airway obstruction and makes intubation more difficult.
- If intubation is difficult, reduce and if necessary, remove cricoid force. This may improve the laryngeal view and enable intubation. Have suction ready. If a patient regurgitates, cricoid force may need to be reapplied.
- BURP may improve the laryngeal view but increases the risk of obstruction. If ventilation is difficult, remove BURP.
- Release cricoid force if a patient vomits early during induction. Vomiting does not occur after loss of consciousness.
- Bimanual force (other hand behind the neck) has not been shown to be of benefit in supine patients and uses up one of the assistant's hands. It is not recommended.
- Some patients may only tolerate cricoid force after induction.
- Applying consistent cricoid force is difficult for >5min.
- If intubation fails, cricoid force must be removed to enable SGA placement.

Controversies

- RSI with cricoid force has not been proven to reduce aspiration.
- Titrating induction agent to loss of consciousness is sometimes preferred.
- Rapid-acting opioids of short duration are often used to aid haemodynamic stability and improve intubation conditions, e.g. alfentanil (10–30 micrograms/kg) or remifentanil (1–2 micrograms/kg) 1min before induction. Lidocaine (1–1.5mg/kg) is less commonly used.
- Using sufficient opioid to avoid NMB is not commonly practised nor recommended.
- After RSI for CS, beware neonatal respiratory depression if opioids are given to the mother on induction.
- It is a fallacy that 'RSI is safe because the patient will wake if there are airway complications'. In the event of failed intubation, whatever drug combination is used, induction agents and NMBAs are very unlikely

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to wear off before the onset of life-threatening hypoxaemia (and awareness). Manage the airway, oxygenate and provide IV anaesthesia.
- Reversing rocuronium with sugammadex (16mg/kg) in response to airway difficulty is likely to be rapid only if the drug is predrawn up or in the room in adequate quantities. It does not guarantee restoration of breathing. It will not reverse pathological obstruction.

Paediatric considerations

(See ➲ p. 924.)
- Appropriate cricoid force has not been established for children.
- Young children are unlikely to cooperate with preoxygenation and application of cricoid force before induction.

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Inhalational induction

See also ➲ pp. 408–9.

Relative indications

- To avoid IV induction: children, needle phobia, difficult IV access.
- To maintain airway patency and SV during induction:
  - Anticipated difficult intubation ± difficult mask ventilation, e.g. acute epiglottitis, perilaryngeal tumours
  - Inhaled foreign body
  - Bronchopleural or tracheo-oesophageal fistula.

Preparation and practice

- Explain the process to the patient/parents/carers on the ward. Warn parents/carers about the 'excitation' stage.
- An antisialagogue is optional.
- Without IV access, have a skilled assistant present to obtain IV (or intraosseous (IO)) access at sufficient anaesthetic depth or in crisis.
- Apply routine monitoring as soon as possible.
- Use a tight-fitting mask to speed induction. In young children, some prefer a cupped hand to deliver the fresh gas supply initially.
- Use 100% O_2 with actual or anticipated airway obstruction.
- Use sevoflurane. It is the best tolerated agent in routine use. Although it can be started at 8%, tolerance may be improved by gradual introduction and a 50:50 mix of N_2O and O_2, which also speeds induction.
- When tolerated, use CPAP and gentle assisted ventilation to maintain the airway and speed induction.

Difficulties

- Beware. A belief that inhalational induction is always safe, because if the airway obstructs, anaesthesia will reverse and adequate SV will restart, is not supported by fact. Persistent obstruction, laryngospasm, hypoxia and arrhythmia can occur and lead to morbidity and mortality. Anticipate difficulty. Have a rescue plan.
- Mask leak, low alveolar ventilation (e.g. partial/intermittent obstruction, breath-holding) and high CO slow induction.
- The correct stage to cannulate veins, instrument the airway, apply cricoid force or intubate is a matter of experience and may be misjudged.
- During airway intervention, rapid offset with sevoflurane may cause lightening of anaesthesia.
- The 'excitation' stage may be long and associated with complications. Induction will only progress if the airway is patent.
- Additional cautious IV induction, while maintaining spontaneous breathing, may rarely be appropriate.
- Inhalational induction may be part of a 'strategy' for managing airway obstruction, but you must have backup plans.

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A spontaneous breathing technique with propofol TCI

Maintaining spontaneous breathing during slow, careful induction by incremental propofol TCI is an alternative to inhalational induction. It similarly demands early detection of airway problems. It also requires understanding and experience of the effects of pre-programmed pharmacokinetic models, particularly as they relate to weight, obesity and children. It has some advantages:

- Sedative levels of propofol provide anxiolysis, assisting the progress of anaesthesia.
- Increasing depth of anaesthesia is independent of ventilation.
- The rate of increase in depth is titrated by the anaesthetist (not dictated by the patient).
- In difficulty, stopping the infusion enables anaesthesia to lighten, without requiring a patent airway.
- Airway reflexes (coughing, bucking and laryngospasm) can be rapidly suppressed.
- Secretions are not increased.
- Assisted ventilation may be attempted at an earlier stage, even when still responsive to verbal stimulus.
- Airway adjuncts are also tolerated considerably earlier.
- Staff and the environment are not exposed to anaesthetic gases.

Paediatric considerations

(See ➔ pp. 919–20.)

- Explanation and support are essential.
- Optimal positioning depends on size. Young children may settle best on the carer's lap. The carer should be instructed to restrain the child gently during induction. For older children, sitting on a trolley may be more appropriate.

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Awake tracheal intubation

- ATI must be considered when airway management will, or is, predicted to be hazardous.
- ATI should be the first plan when ventilation by face mask or SGA will be difficult and front of neck anatomy impedes elective and emergency tracheal access.
- ATI should present low risks to patients, preserving airway tone, patency and SV.
- In experienced hands, ATI has 98% success with either FOB or VLs.[4] Other devices may be used, depending on experience and circumstance.
- Success depends on setup, skill, cooperation and safe, effective LA. Sedation is optional but has risks. Oxygenation remains the priority.
- Have backup plans for airway obstruction and inadequate ventilation, including postponement, high-risk GA and eFONA.

Indications

- Known or anticipated difficult airway: particularly if backup plans are at higher than normal risk of failure (e.g. morbid obesity, OSA)
- Known or suspected C-spine instability (e.g. trauma or RA)
- Head and neck pathology affecting the airway or its manipulation
- Progressive airway obstruction
- Previous failed intubation.

Relative contraindications

- Unreliable cooperation that jeopardises safety and success
- Airway contamination likely to obscure scope optics (e.g. bleeding)
- Coagulopathy. Beware causing bleeding, particularly by the nasal route
- Critical airway obstruction (e.g. periglottic masses and stenosis). Beware causing complete airway obstruction, laryngospasm and coughing.

Absolute contraindications

- Patient refusal
- LA allergy.

Procedure

- Explain the procedure to the patient and obtain consent.
- Consider antisialagogue premedication and sedation.
- Decide an airway topicalisation method.
- Optimise setting and 'ergonomics'. Ideally, use an operating theatre.
- Organise your team. Rescue strategies must be ready and understood. Include and ready the surgical team.
- Consider using a checklist or cognitive aid to improve teamwork.
- Consider the DAS ATI method (Fig. 15.8).
- Apply routine monitoring.
- Maintain oxygenation with supplemental O_2, HFNO if available.
- Clear a soiled airway carefully with suction.
- Limit the number of attempts (preferably ≤3 and one by an expert).
- After intubation, visualise the carina AND confirm intubation success with capnography by connecting the breathing system. The bag should move with respiration when the cuff is inflated.

ALGRAWANY

Fig. 15.8 Difficult Airway Society ATI technique. Reproduced from Ahmad I, *et al.* (2019). Difficult airway society guidelines for awake tracheal intubation (ATI) in adults, *Anaesthesia*, **75**(4), 509–28 with kind permission of the Difficult Airway Society.

Local anaesthesia

- Specific airway nerve blocks (sphenopalatine, ethmoid, glossopharyngeal, superior laryngeal, recurrent laryngeal) can be effective in skilled hands. Topical LA is an alternative.
- The DAS ATI LA technique is outlined in Fig. 15.8. Lidocaine is potentially safer than other LAs. Nebulised, it has variable success. Transtracheal LA injection is invasive, effective and ensures careful examination of the front of the neck. The total dose of lidocaine should not exceed 9mg/kg, but this is rarely needed.
- Treatments for toxicity must be immediately available.

Postoperative care

- Plan extubation and potential reintubation carefully. If the airway remains at risk, delay extubation. The TT may need to be exchanged for another type or size to facilitate intensive care.
- Postoperatively, if you can, check if direct laryngoscopy is possible.
- Keep the patient starved until airway sensation and reflexes have returned. After lidocaine, this usually takes >2h.
- Reusable FOB, VLs and other instruments must be decontaminated and disinfected.

Dealing with difficulty

Sedation

- Sedation can help anxiety, discomfort and intolerance *but* can cause airway obstruction, cardiorespiratory depression and hypoxia. Sedation is *not* a substitute for effective LA.
- A cautious remifentanil or dexmedetomidine infusion may be safer than propofol.[4] Another anaesthetist monitoring sedation improves safety. Adding another drug (e.g. midazolam) adds risk. Antagonists must be immediately available if using opioids and benzodiazepines.

Oral route

- Specialised FOB conduits, e.g. the Berman Airway, Ovasappian Airway and Williams Airway Intubator, protect the FOB and can improve laryngoscopy. However, they may not fit well and move from the midline, making laryngoscopy and intubation difficult.
- Awake insertion of an SGA as a conduit has been described.

Nasal route

- This may be the only option with a narrow gape.
- Assess the patency of nasal passages and any history of epistaxis.
- Topical vasoconstrictors (e.g. phenylephrine) reduce bleeding risk.
- Guiding a TT through the nose with an FOB before advancing to the larynx avoids realising the TT will not pass through the nose later.
- Nasal dilators (e.g. lubricated nasopharyngeal airway or Hegar cervical dilators) may make TT passage easier but risk epistaxis.

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Type of tracheal tube

- With an FOB, use a TT with a rounded tip (optimised), e.g. LMA Fastrach™ or Parker Flex-Tip™. A small size that fits snugly on the FOB reduces laryngeal impingement during passage and airway trauma.
- Optimised TT, sizes and introducers, including simultaneous FOB, might also increase VL ATI success.
- Effective TT lubrication is essential.

Technique

- Using an FOB, 'black' is the airway, and 'pink' the airway wall. Aim for 'black'.
- 'Red out' suggests the FOB tip is against the mucosa. Withdraw until recognisable structures are seen, then advance more slowly, avoiding the mucosa.
- If negotiating the oropharynx is difficult: ask the patient to protrude their tongue and jaw; ask an assistant to pull the tongue forwards with gauze and provide gentle jaw thrust; or insert a laryngoscope.
- Ask the patient to breathe deeply to inhale LA into the trachea and widen the aperture as the TT approaches the glottis.
- Railroading problems may be overcome by gentle anticlockwise TT rotation.
- Warn the patient that breathing may be difficult as you railroad the TT.

Complications

- Refusal, coughing, airway bleeding, laryngospasm, vomiting, regurgitation, aspiration, airway obstruction, hypoxaemia.
- Complications may occur in up to 18% of cases. Call for help early.

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Apnoeic oxygenation

- Preceded by effective preoxygenation, administering O_2 via a patent airway prolongs time to desaturation during apnoea.[7]
- Alveolar O_2 diffusing into blood draws O_2 from the airways by convection. Optimising lung volume and blood flow matching, e.g. by positioning and CPAP, may enhance this process. Air entrainment must be minimised.
- Insufflation sources for airway surgery include nasal cannulae, a RAE TT wrapped round the cheek, nasopharyngeal airways and catheters, dedicated ports on surgical laryngoscopes and bronchoscopes, and airway and cricothyroid catheters.
- Note that insufflation devices rarely incorporate pressure-limiting valves. Inadequate O_2 egress from the airway can cause barotrauma.

High-flow nasal oxygenation

HFNO systems deliver heated, humidified O_2 via nasal cannulae at up to 50–70L/min. Perioperative systems typically provide 100% O_2 only. With SV, HFNO can provide CPAP (~5cmH_2O) when the mouth is closed. Anatomical dead space and upper airway resistance are reduced.

Uses of HFNO

To decrease the risk of hypoxia during intubation in

- Anticipated and known difficult airway management
- RSI
- Obesity (BMI >40kg/m^2)
- OSA
- Obstetric patients
- Critically ill patients.

With a patent upper airway, HFNO can enable prolonged apnoeic oxygenation during airway management. Beware. An airway rescue strategy is essential.

Tubeless anaesthesia

- Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE), i.e. continuous HFNO during apnoea, is a method of increasing apnoea time in patients with difficult airways undergoing 'tubeless' upper airway surgery.
- Sustained oxygenation may exceed 30min, sometimes up to ~1h, in some low-risk patients.
- Patient selection is crucial. Hypoxaemia onset is more rapid with obesity.
- Although gas mixing by turbulence and cardiogenic airway oscillation enables some CO_2 clearance, some hypercarbia is the norm.
- $ETCO_2$ measurement can be obtained by intermittent ventilation but values underestimate $PaCO_2$ which rises unpredictably. $PaCO_2$ may be >10kPa after 20min. Blood gas or transcutaneous CO_2 monitoring has been recommended but is not routine.

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Contraindications to HFNO

- Maxillofacial trauma (e.g. base of skull, mid-facial fractures)
- Epistaxis
- Nasal obstruction
- Airway laser or diathermy (fire risk)
- Lack of patient cooperation
- Contraindications to CPAP.

A plan for preoxygenation and THRIVE with HFNO

- Set up and warm the system before the patient arrives.
- Apply HFNO at ~20L/min immediately on arrival.
- Ask the patient to breathe through their nose, mouth closed.
- Position 20–45° head up.
- Increase O_2 flow to 50–70L/min if tolerated.
- After induction and loss of consciousness, maintain oropharyngeal patency with two-handed jaw thrust.
- Increase O_2 flow to 70L/min.
- Place nasal cannulae on the forehead.
- Confirm you can mask ventilate.
- Return the nasal cannulae to the nares.

Special considerations

- To reduce barotrauma risk, make sure the APL valve of any breathing system is fully open if you apply simultaneous mask O_2 and HFNO.
- Even though HFNO may provide prolonged oxygenation, when an aspiration risk is present or the airway may be lost, secure the airway by intubation as quickly as feasible.

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Extubation after difficult intubation

- Develop a 'strategy' for extubation that manages airway obstruction. Extubation should not be an emergency.
- Check any required equipment. Assemble and brief skilled personnel. Assess risks of laryngeal dysfunction on extubation (e.g. recurrent laryngeal nerve injury, laryngospasm), difficult reintubation (e.g. bleeding and swelling) and new airway impediments (e.g. cervical fusion, external fixators, dental wiring).
- Consider surgical airway optimisation (e.g. haemostasis, haematoma evacuation, stitches to bring the tongue forward).
- If your strategy in difficulty is eFONA, consider an elective FONA.
- If deferring extubation might reduce risks, consider postponing extubation and reassessing.
- Consider a course of corticosteroids to reduce inflammatory oedema.

Preparation before extubating awake

- Clear the entire upper airway carefully.
- Suction the trachea.
- Empty the stomach, if necessary.
- Perform a 'leak' test. Deflate the cuff (if present). Ventilate at low pressure. Check for leak around the TT. If there is no leak, consider deferring extubation. (The sensitivity and specificity of this test are imperfect and depend critically on TT size.)
- Place the patient in their most advantageous position for breathing and airway patency. This is often sitting up.
- Preoxygenate to an ETO_2 ~0.9.
- Insert a 'bite block' to prevent TT or SGA occlusion.
- Fully reverse NMB.
- Wake.
- Extubate when obeying commands and breathing normally.
- Anaesthetics with rapid offset may be advantageous.
- Provide high-flow O_2, HFNO or CPAP as indicated.
- Monitor in the recovery period for as long as necessary.

Difficult Airway Society extubation guidelines (2015)

(See Figs. 15.9 and 15.10.)

- 'Low-risk' patients are at low risk of aspiration, exacerbations of life-threatening disease and airway compromise.
- 'At-risk' patients are at risk of aspiration, acute exacerbation of life-threatening conditions (e.g. bronchospasm, cardiac failure, raised ICP), airway obstruction (e.g. due to airway trauma, surgery, oedema, OSA, obesity) and difficult rescue oxygenation.
- Awake extubation is the default for both groups. All other techniques are considered 'advanced' and require practice, teamwork and attention to detail.
- In 'low-risk' patients, extubating during 'deep' anaesthesia may reduce risks of exacerbating life-threatening conditions and adverse surgical outcomes (e.g. bleeding and dehiscence). However, these benefits must be balanced with risks of airway obstruction after extubation, and coughing and laryngospasm during emergence.

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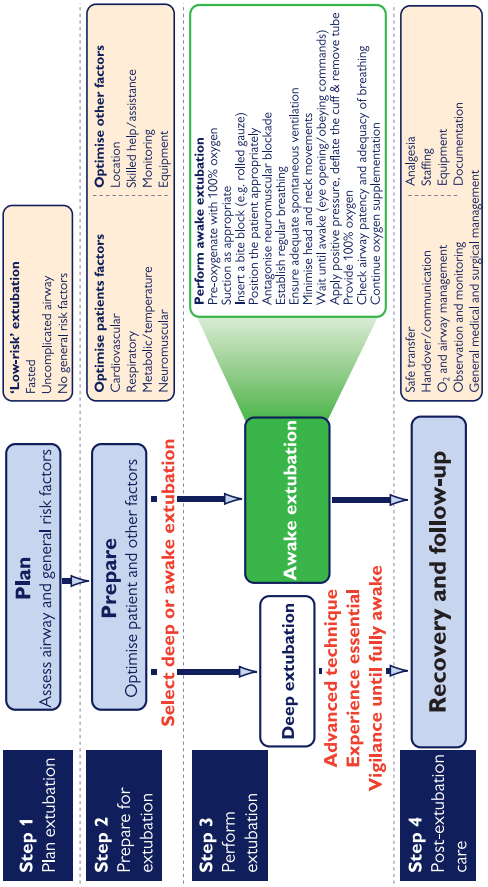

Fig. 15.9 Difficult Airway Society Extubation Guidelines: low-risk algorithm. Reproduced from Popat M, *et al.* Difficult Airway Society Guidelines for the management of tracheal extubation. *Anaesthesia* 2012; **67**:318–40, with permission from the Association of Anaesthetists of Great Britain & Ireland/Blackwell Publishing Ltd.

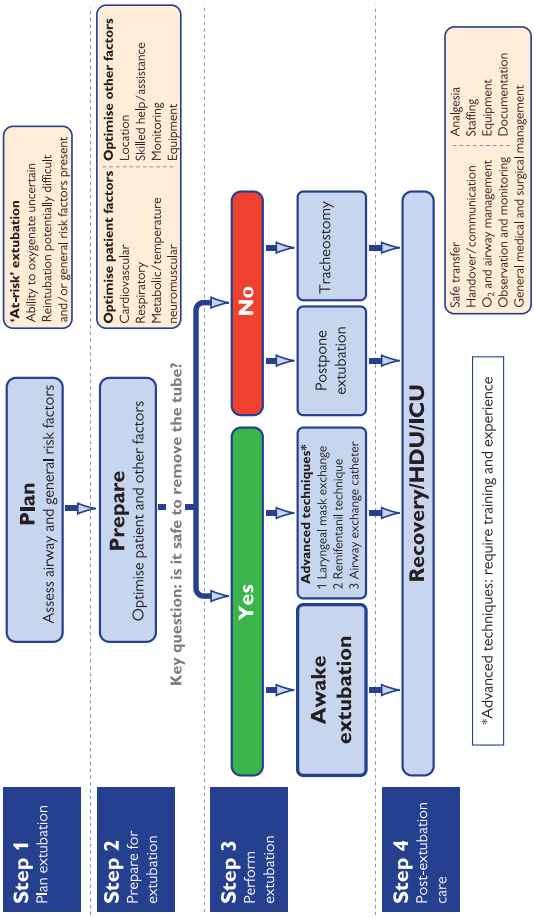

Fig. 15.10 Difficult Airway Society Extubation Guidelines: at-risk algorithm. Reproduced from Popat M, *et al.* Difficult Airway Society Guidelines for the management of tracheal extubation. *Anaesthesia* 2012; 67:318–40, with permission from the Association of Anaesthetists of Great Britain & Ireland/Blackwell Publishing Ltd.

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- For some 'at-risk' patients, two 'advanced' techniques may reduce risks of exacerbating life-threatening conditions and adverse surgical outcomes, but avoid these techniques if reintubation will be difficult.
  - Remifentanil infusion-assisted extubation. Emergence occurs during remifentanil suppression of airway reflexes. The TT is tolerated during SV or breathing to command. Extubation occurs awake with patient cooperation. Beware. Perfect titration can be difficult.
  - TT exchange for an SGA. Emergence with an SGA *in situ* maintains the airway, is usually well tolerated and may reduce risks of laryngeal soiling. Several methods exist. Deep anaesthesia or NMB are needed to avoid airway reflexes during exchange. Airway pathology or surgery can preclude SGA insertion.
- One 'advanced' technique, proposed when reintubation will be difficult, involves inserting an airway exchange catheter as a conduit for reintubation.
  - All staff caring for the patient require training in airway exchange catheter use.
  - The airway exchange catheter is passed through the TT into the trachea before extubation. Thin airway exchange catheters (11 Fr) can be tolerated awake. LA applied to the airway exchange catheter improves tolerance.
  - The airway exchange catheter must be secured above the carina (depth marker ≤25cm from the mouth in adults) and position checked with CXR.
  - Distal migration can cause perforation.
  - Reintubation is complex and requires great care for success. If reintubation is required, anaesthesia, NMB, a narrow specialised TT if possible (e.g. LMA Fastrach™ or Parker FlexTip®) and VL aid reintubation.
  - O_2 should NOT be given via an airway exchange catheter. If required, O_2 should be delivered by conventional means.

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Airborne respiratory viruses

- The emergence of severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) in 2019 led to a global pandemic of this coronavirus disease (COVID-19).
- Previous coronavirus outbreaks included SARS-CoV (causing severe acute respiratory syndrome) and MERS-CoV (causing Middle East respiratory syndrome). Similar outbreaks should be anticipated.
- These viruses are spread by inhalation of droplets and aerosols at short distances. Contact and distant aerosol spread are controversial.
- Some airway procedures have been designated as aerosol-generating procedures (AGPs) requiring enhanced precautions against aerosol transmission. The science supporting this is evolving.
- Appropriate steps should be taken during airway management to reduce cross-infection risk.
- Fundamental infection control includes: separating patients with and without infection; isolation; exemplary cleaning, decontamination and disinfection processes; minimising 'footfall' and the duration of direct patient care; and training in donning and doffing personal protective equipment (PPE).

Tracheal intubation

This should be Safe, Accurate and Swift (SAS).

Safe: for both staff and patient

- Ensure adequate preparation by using a checklist.
- Wear PPE for AGPs (e.g. FFP3/N95 filter mask, eye protection, fluid-resistant long-sleeved gown, gloves).
- When you can, use a well-ventilated room (e.g. >12 air changes/h), ideally with negative pressure.
- Minimise staff present: an intubator, an assistant and a 'runner' outside the room.
- Place a combined HME/viral filter between the breathing system and catheter mount.
- Insert a closed tracheal suction system.
- Avert accidental disconnection. Push and twist circuit connections.
- Check and appropriately preset a ventilator.
- Minimise or avoid mask ventilation or airway pressure before intubation. If ventilation is necessary, use low O_2 flow. Optimise mask seal with a 'VE' grip (thumbs caudad on each side of the mask with your fingers lifting the jaw; ask an assistant to squeeze the bag).
- Inflate the TT cuff before ventilating. Avoiding leakage, manage cuff pressure appropriately when possible.
- Minimise any breathing system disconnection during the procedure.

Accurate (familiar and reliable technique)

- Maximise first attempt success: use experienced intubator; familiar kit, including VL; rapid, complete NMB.
- Use rescue techniques in which you are trained and that are proven and reliable. Avoid untried and untested techniques.
- Use a second-generation SGA for airway rescue (better seal).

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Swift (timely, without rush or delay)

- Preoxygenate for 3–5min with routine monitoring.
- Use an RSI technique for emergency intubation. Consider ketamine 1–2mg/kg, rocuronium 1.2mg/kg or suxamethonium 1.5mg/kg (maximum 150mg). Prepare for CVS instability.
- Rapidly confirm successful tracheal intubation with capnography. Confirm mechanical ventilation and O_2 presettings are appropriate.

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Chapter 16

Conduct of anaesthesia

John Newland and Heng-Yi (Henry) Wu

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See also
➲ Conduct of anaesthesia pp. *913–25*

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Induction of general anaesthesia

Induction of GA establishes a state of reversible unconsciousness smoothly, safely and pleasantly, while maintaining haemodynamic stability and ventilation. Risk assessment, planning and vigilance are needed during this time of complex physiological change. For patients and relatives, this is a time of unfamiliarity, anxiety and stress. Establishing rapport and putting the patient at ease during induction is an important skill to master, with each anaesthetist developing their own techniques and style to achieve this.

Preparation for induction

Induction plan

This should be based on your preoperative assessment of the patient (see ➲ p. 23).

- The detail of this assessment will depend on the urgency of the operation and the complexity of the patient and surgery.
- Particular focus should be paid to airway assessment, aspiration risk, allergies, stability of comorbidities and cardiopulmonary reserve.
- Consider backup plans and communicate these to your team.
- Seek advice if the assessment identifies significant issues.

Theatre preparation

Ensure anaesthetic equipment is checked and emergency equipment is available. Prepare drugs, with emergency drugs rapidly accessible.

- Ensure WHO Surgical Safety Checklists are followed (see ➲ p. 5).

Preparing for induction

Positioning

The patient's head should be placed in the sniffing position (neck flexed and head extended), with the head end of the bed elevated 20–30°. Morbidly obese patients should be in the ramped position.

Preoxygenation

Aim for an ETO_2 >80% to allow for a longer safe apnoea time. An airtight seal with the mask is important to increase ETO_2. Application of PEEP (by closing the APL valve slightly), CPAP (by using the ventilator) or nasal cannulae (for apnoeic oxygenation) can be helpful in patients who may desaturate quickly.

Environment

A quiet atmosphere prior to induction is important to help reduce patient anxiety and allow the focus to remain on the patient.

Difficulties with induction

Induction can be a busy and complex time. Unexpected haemodynamic instability or a difficult airway can quickly task-overload anaesthetists and potentially deteriorate into an emergency.

- Do not hesitate to ask for a 2nd pair of experienced hands to be present during induction if difficulties are anticipated.

Induction technique

Induction is accomplished using either IV or inhalational agents or a combination of both. Careful preassessment will determine the induction technique and doses of induction agents.

Intravenous induction

IV induction is faster and more reliable than inhalational induction and is generally preferred in adults.

- Multiple drugs are often co-administered to reduce dose-dependent side effects of each individual drug.

Induction agents

- Propofol (1–2.5mg/kg) is the commonest agent used. It provides rapid onset and emergence, making it suitable for day case surgery. It has antiemetic properties and can be used for maintenance of anaesthesia. Propofol obtunds airway reflexes and as such, it is the ideal drug to use with an SGA. Moderate hepatic or renal impairment do not alter its pharmacokinetics significantly.
- Ketamine (1–2mg/kg IV or 5–10mg/kg IM) has multiple administration routes available, making it very versatile. Despite having a direct myocardial depressant effect, sympathetic stimulation results in a largely cardiovascularly stable induction. Airway reflexes and respiratory drive are preserved, but ↑ salivation can lead to upper airway obstruction. It is frequently used in the ED and the prehospital environment.
- Thiopental (2–5mg/kg) produces a smooth induction of anaesthesia with a well-defined endpoint, usually within 30s. Airway reflexes are largely preserved, making it less suitable for use with an SGA. Intra-arterial injection can cause tissue necrosis.
- Etomidate (0.3mg/kg) is useful for induction of haemodynamically unstable patients as it causes the least cardiovascular depression of all the IV induction agents. Controversy exists around its use as it causes reversible adrenocortical suppression for 12–24h. A single induction dose is safe, but continuous infusions may lead to an increase in mortality in critically ill patients.

Titrating propofol

Propofol causes the greatest fall in BP of all IV induction agents, primarily due to vasodilation. It therefore requires careful titration of a 'sleep dose', with consideration of factors such as patient weight, age, CO and arm–brain circulation time.

- In low CO states (shocked patients or the elderly), the body compensates by diverting a higher proportion of the CO to the brain, reducing flow to other areas of the body. This results in a smaller initial volume of distribution and a greater proportion of the drug diverted towards the cerebral circulation. Furthermore, the time taken for the drug to reach the brain is prolonged. Thus, a slow titration of a reduced dose is the safer way to induce these patients.

Opioids

Opioids work synergistically with propofol to allow a dose reduction, as well as obtund the autonomic response to airway instrumentation.

- Timing of administration should take into consideration time to peak effect. Fentanyl (1–2 micrograms/kg) should be administered 3–5min prior to the induction agent, whereas alfentanil (10–30 micrograms/kg) or remifentanil can be administered concurrently due to their faster onset of action.

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Adjuncts

- Midazolam (0.5–5mg) is commonly used as a co-induction agent to reduce the propofol dose required, therefore minimising the cardiovascular effects of propofol. It can also reduce the incidence of PONV.
- Lidocaine (0.5–1.5mg/kg) can reduce the induction dose, as well as blunt stimulus from airway instrumentation. A small amount (20–40mg) can be added to 200mg of propofol to reduce pain on injection.

Neuromuscular-blocking agents

NMBAs (see ➲ pp. 420–2) are generally used to facilitate intubation, but care must be taken to avoid awareness. As such, NMBAs should only be given after loss of consciousness.

Rapid sequence induction

An RSI (see ➲ pp. 388–90) is performed in those with a high aspiration risk. This technique involves delivering a predetermined dose, rather than a sleep dose, of IV induction agent, avoiding titration. This is immediately followed by an NMBA and early intubation.

- Opioids, particularly alfentanil, can be considered for use in RSI.

Other considerations

- Adequate depth of anaesthesia needs to be achieved prior to insertion of an SGA to avoid biting, hiccupping, breath-holding or laryngospasm. Reduce opioid dose to facilitate SV.
- Spontaneous breathing induction of anaesthesia with slow incremental titration of a TCI of propofol is a useful technique, particularly in airway surgery.
- In those with actual, or potential for, haemodynamic instability, preloading with IV fluid and co-administration of a vasopressor infusion may be necessary.
- High-dose opioid, including remifentanil, may be used to blunt the sympathetic response to laryngoscopy.

Inhalational induction

(See also ➲ p. 919.) Indications for inhalational induction (see ➲ pp. 391–2) include:

- Avoiding awake IV access, e.g. in children, needle-phobic patients and patients with difficult IV access
- To maintain SV during induction (TIVA is becoming more popular for this indication).

Inhalational induction, whether of a child or an adult, requires cooperation from the patient. Age-appropriate explanation and interaction form part of the 'art of anaesthesia'.

- Sevoflurane is the commonest agent used and the best tolerated. A 50:50 mix of N_2O:O_2 improves tolerance and speeds onset.
- If inhalational induction is used for difficult IV access, have a skilled assistant present to secure cannulation early.

Paediatric considerations

Parental understanding and support are essential, as their assistance is often very useful. Warn parents about the excitation phase, with noisy breathing and movement to be expected.

- Optimal positioning will depend on child size. Between ages 2 and 5, the child can be sat on the parent's lap, with the parent cuddling/gently restraining the child during induction. For older and younger children, the operating table or bed may be more appropriate.
- A close-fitting face mask speeds up induction; however, in those children not wanting to accept a mask, a cupped hand to deliver the fresh gas supply can be used initially before moving to a mask.
- Slowly uptitrating sevoflurane is better tolerated in children.

Difficulties with inhalational induction

- In adults, it can take a long time to achieve adequate depth of anaesthesia. It can be further slowed by a leak around the mask, airway obstruction, breath-holding and high CO.
- The excitement stage may be long and associated with complications such as airway obstruction. Induction will only progress past this phase if the airway remains patent. Application of PEEP/CPAP/gentle assisted ventilation can be useful if airway obstruction occurs.
- ⚠ The traditional view that inhalational induction is safe because if the airway obstructs, anaesthesia will lighten is false. The NAP4 report[1] highlighted that in practice, when total airway obstruction occurs, patients do not exhale the volatile and hypoxia rapidly ensues. If this technique is used for a difficult airway, have backup airway plans prepared.

Further reading

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Maintenance and TIVA

Following induction of anaesthesia, maintenance may be achieved by either inhalational or IV anaesthesia, or a combination of both. The choice of which technique to employ comes down to patient and surgical factors, as well as the anaesthetist's preference. No matter what technique is employed, the endpoints for maintenance anaesthesia remain the same: unconsciousness, amnesia, analgesia, akinesia and physiological homeostasis. The maintenance period between induction and emergence requires the application of a wide range of knowledge and skills, as well as planning and vigilance.

Phases of anaesthesia care

Induction: mind the gap

The shift from induction to maintenance phases frequently requires transitioning between anaesthetic agents, commonly IV to volatile. This requires careful attention, as the offset of IV induction agents can occur before the volatile has reached adequate levels, resulting in a period where the overall concentration of anaesthetic agents may be lower than desirable. This time also coincides with transferring and positioning of patients, during which ventilation, and thus volatile delivery, can be interrupted. Consequently, this phase of anaesthesia is associated with a higher incidence of accidental awareness. To reduce this risk, an age-adjusted MAC target of at least 0.7 should be achieved as soon as possible following induction.

- Complicating this period is a high incidence of post-induction hypotension. Causes are multifactorial, but it is associated with older age, emergency surgery and higher ASA scores. Even short periods of pre-incisional hypotension have been linked to unfavourable patient outcomes.
- The transition phase is a balancing act between providing adequate anaesthesia to prevent awareness and avoiding the vasodilatory effect of the anaesthetic agents.
- Fluid boluses and vasopressors should be used to treat hypotension, instead of reducing the depth of anaesthesia.

Maintenance

Both depth of anaesthesia and analgesia are not static and may require titration, especially during periods of intense stimulation such as surgical incision, dilation of sphincters, tunnelling of ventriculoperitoneal shunts, etc. Experienced anaesthetists are often able to predict stages in the surgery where anaesthetic levels will need to be titrated, minimising haemodynamic changes.

Emergence

Optimal timing for discontinuation of anaesthetic agents will depend on the specific agents, doses used and the duration of their administration, as well as patient-specific factors. A conservative approach is advised as the outcomes of early emergence tend to be worse than delayed emergence.

Paralysis

The depth of anaesthesia for maintenance requires consideration of whether NMBAs are used. Without NMBAs, maintenance anaesthesia needs to be dosed higher to inhibit spinal reflexes and prevent movement. When using NMBAs, a lower dose is required to maintain unconsciousness and prevent awareness only. However, NMBAs are a known risk factor for awareness as movement, a marker of light depth of anaesthesia, is inhibited.

Combined technique

All maintenance anaesthetic agents have dose-dependent cardiovascular effects and variable offset times. Combined techniques, using inhalational and IV techniques, can be used to minimise the total dose of any one agent, thus minimising side effects and potentially hastening emergence, particularly in long procedures. However, this does increase complexity and the risk of error, possibly increasing the likelihood of awareness from underdosing or cardiovascular compromise from a 'double anaesthetic'. Depth of anaesthesia monitoring (see ➲ pp. 426–9) should be considered if this technique is employed, particularly when using NMBAs.

Inhalational anaesthesia

Volatile agents remain key components of modern GA. They are proven to be safe, easy to use and effective. There are, however, disadvantages, many being dose-dependent (Table 16.1).

Table 16.1 Advantages and disadvantages of inhalational agents

Advantages	Disadvantages
Ease of administration	Myocardial depression and vasodilation
IV not required for induction	↑ risk of PONV
End-tidal concentration gives estimate of effect site concentration	Trigger for MH
Anaesthetic preconditioning	Potent greenhouse gases
Bronchodilation	Tocolytic
	Airway irritation (isoflurane, desflurane)
	Emergence delirium in children

The dose of volatile agents is dependent on their potency, which is expressed as the minimum alveolar concentration of a volatile, at one atmospheric pressure, producing immobility in 50% of adult subjects exposed to a standard noxious stimulus (MAC). MAC values differ for each agent and are inversely related to lipid solubility. Desflurane is the least lipid-soluble; thus, 6.6% is required to achieve 1 MAC, whereas sevoflurane is more lipid-soluble and only requires 1.8% for 1 MAC.

- Memory formation is inhibited at around 0.3 MAC (MAC aware) and loss of movement to command is 0.5–0.7 (MAC awake). Therefore, we expect an inadequately anaesthetised patient to move before experiencing recall. Opioids dramatically reduce the MAC required to suppress movement due to effects on the spinal cord, but have a lesser effect on the dose required to suppress awareness.

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- MAC requirement is also dependent on a number of patient characteristics. Concurrent use of CNS depressants, patients' haemodynamic state and their age are the most important covariates to consider, with most anaesthetic machines now providing age-adjusted MAC values.

Titration of volatiles

Speed of volatile onset and offset is determined by: FGF, inspired concentration of volatile, FRC, minute volume, CO and blood gas solubility.

- There is no significant difference in onset between commonly used volatiles, but this is not the case for offset.
- With longer duration of anaesthesia, volatile solubility and body composition become increasingly important factors determining offset. This is due to accumulation of volatile in tissues. Modern anaesthetic agents such as sevoflurane and desflurane have relatively low solubility and thus have a distinct advantage over older volatiles such as isoflurane. The more soluble the agent, the sooner washout must commence as the operation ends, to allow timely emergence.

Sevoflurane

Fast uptake and elimination due to low blood and tissue solubility, allowing rapid titration and recovery. Non-pungent, thus suitable for inhalational induction.

Desflurane

Has the lowest blood and tissue solubility of the potent volatile inhalation agents, resulting in very rapid uptake and elimination, with little accumulation in tissues. It is beneficial in prolonged operations, the morbidly obese and those for whom an accelerated emergence is desired. It is pungent and thus unsuitable for inhalational inductions. It can also cause airway irritation, tachycardia, bronchospasm or laryngospasm, particularly in asthmatics or when rapidly uptitrated. The benefits of desflurane need to be weighed against its higher cost and negative environmental impact, as it is a potent greenhouse gas.

Isoflurane

Now less commonly used due to the favourable properties of newer agents. Slower offset due to higher blood and tissue solubility, compared with sevoflurane and desflurane.

Nitrous oxide

Commonly used as an adjuvant to volatile anaesthetics during induction; less commonly used for maintenance. It is inexpensive and widely available. Co-administration allows for a reduction in MAC of other volatile agents, but it cannot be used as the sole agent due to its low potency. It is rapidly titratable due to its low solubility and is useful for its analgesic and anxiolytic properties. It increases the incidence of PONV, but this can be overcome by antiemetic prophylaxis. It must be avoided in patients with gas-trapped spaces (e.g. bowel obstruction, middle ear conditions, pneumothorax, pneumocephalus). N_2O is also a greenhouse gas and contributes to ozone depletion.

TIVA

TIVA utilises IV agents alone, avoiding the use of inhalational agents. Although theoretically any combination of induction agent and opioid can be used, in practice, the commonest agent used is propofol alongside an opioid. Pharmacokinetic models for TCI have resulted in ↑ use of TIVA in various clinical settings (Table 16.2).

Table 16.2 Advantages and disadvantages of TIVA

Indications and advantages	Disadvantages
Reduced PONV	Plasma or effect site concentrations are not easily measured
Improved quality of recovery	Potentially ↑ risk of awareness
Does not require an anaesthetic machine—useful for remote-location anaesthesia and transporting intubated patients	Dependent on the continuity of a functioning IV line
Safe in MH	TCI models may be inaccurate in some patients (e.g. obese)
Can be used for surgery requiring spinal cord monitoring (scoliosis surgery)	Syringes need to be replaced intraoperatively, presenting opportunity for error
Ideal for tubeless airway surgery	Pain on injection
Potentially more environmentally friendly than gases and vapours	May require additional monitoring equipment (processed EEG)
Reduced emergence delirium in children	May be more expensive than volatiles

Choice of agent

Ideal TIVA agents have fast onset and offset to allow rapid titration and recovery. Generally, the offset of IV agents slows as the duration of the infusion increases, which is reflected in the context-sensitive half-time (CSHT). Propofol and remifentanil exhibit short CSHTs and thus are the closest to ideal TIVA agents available. Opioids serve two purposes during TIVA. They provide analgesia and allow a reduction in the required dose of propofol due to their synergistic effect. If remifentanil is used, a longer-acting opioid should be given 30–40min prior to stopping the infusion to cover postoperative pain.

Administering TIVA

There are several effective ways to perform TIVA. The use of TCI is recommended over other methods, if available.

Anaesthetist-administered boluses

Can be useful for very short procedures but often results in excessive drug at the time of bolus or inadequate effect prior to the next bolus. This oscillation can lead to cardiovascular instability and/or awareness.

Manually controlled infusion regimes

These regimes allow anaesthetists to utilise a 'multicompartmental model' without the use of pharmacokinetic infusion pumps. They are not appropriate for all patients. Boluses and infusion rates may need to be altered to achieve adequate depth of anaesthesia (Table 16.3).

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Table 16.3 Suggested manual infusion regimes for propofol and remifentanil

Propofol		
Known as the 'Bristol' regime Achieves a plasma propofol concentration of ~3 micrograms/mL	50mL of 1% propofol (500mg)	1mg/kg induction bolus 10mg/kg/h for 10min 8mg/kg/h for 10min 6mg/kg/h for maintenance
Remifentanil		
Achieves a remifentanil plasma concentration of ~6 nanograms/mL	Remifentanil 50 micrograms/mL	0.5 micrograms/kg/min for 3min 0.25 micrograms/kg/min for maintenance

Target-controlled infusions

TCIs allow accurate maintenance of concentrations of anaesthetic agents. The technique requires an infusion pump programmed with a pharmacokinetic model. Instead of selecting a set rate, a target concentration is set, with a target of either plasma (Cpt) or effect site (Cet). The pump will automatically adjust its infusion rate to achieve a predicted target site concentration.

Basic pharmacokinetics

A multicompartment model is often used to describe the redistribution and elimination of drugs such as propofol. The drug is delivered into a central compartment (V_1), with the initial induction bolus calculated according to the estimated volume of V_1. The drug is then distributed to compartments V_2 and V_3. Movement of the drug between compartments is governed by rate constants (K) (Fig. 16.1).

- Although the various compartments are often equated to biological compartments within the human body, such as blood vessel rich/poor, this is technically incorrect. Compartments and rate constants are purely a mathematical construct used to predict target site concentrations, with the models validated in small populations of patients.
- Depending on which model is used, compartmental size and rate constants can be either fixed or variable. Variable parameters are determined by patient data entered into the pump.

Plasma vs effect site targeting

Cpt achieves and maintains a predetermined target plasma concentration of drug as rapidly as possible without overshooting. Plasma concentration will not approximate effect site (CNS) concentration until a steady state is reached, which can take a number of minutes. Thus, onset of anaesthesia using Cpt is slow. This can be overcome by targeting a higher Cpt transiently during induction, then reducing it to the desired maintenance level. Cet achieves a faster effect site concentration by overshooting the plasma concentration and, as such, is more useful for rapidly achieving the desired depth of anaesthesia.

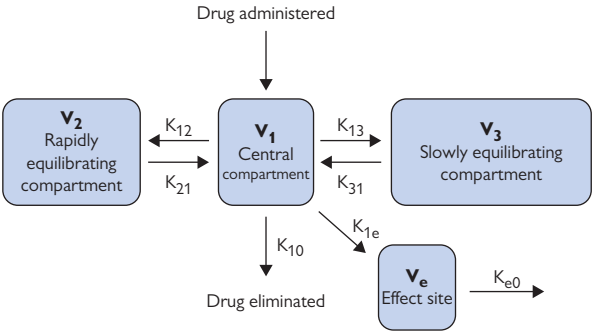

Fig. 16.1 The multicompartment pharmacokinetic model. K refers to the rate constant of transfer between compartments.

Starting infusions

- Use a TIVA checklist for safety (Table 16.4).
- Timing of when to start each infusion should be guided by time to peak effect. If using Cet, both remifentanil and propofol can be started simultaneously. If using Cpt, remifentanil will equilibrate at the effect site long before propofol, potentially leading to an apnoeic (but aware) patient. In this instance, propofol should be started first.
- Some anaesthetists employ a stepwise increase of target concentrations, starting at 0.5–1.0 micrograms/mL of propofol and increasing by 0.5–1.0 micrograms/mL increments once the target site has equilibrated. This method avoids a large initial bolus and the associated CVS instability. It also allows identification of the Cet at which the patient becomes unrousable. The target concentration is ↑ above this level for a layer of safety, but it does guide the minimal level that will be required to keep the patient anaesthetised.

Maintenance

When used correctly, TCI accurately predicts measured plasma concentrations of drugs in most patients. However, due to pharmacodynamic variation, target concentrations must still be titrated to achieve the required effect in any individual. Targets need to be adjusted based on age, ASA status, adjuvant agents used, degree of surgical stimulation and processed EEG (Table 16.5).

When to stop the infusions

This is a balance between maintaining adequate anaesthesia and facilitating rapid emergence. TCI targets can be gradually ↓ near the end of surgery, but caution must be exercised as wound closure can sometimes be a significant noxious stimulus.

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Table 16.4 TIVA checklist

Infusion pumps checked	Prior to using pumps, ensure they are serviced, plugged into the mains power and charged. Ensure low- and high-pressure alarms are set—this can warn of disconnection or a blocked cannula, respectively.
A well-functioning IV line	Ideally insert the line yourself and avoid sites where a tissued line is hard to detect, e.g. antecubital fossa. Secure the line firmly to the patient's skin. Keep the line visible throughout the case to allow early identification of disconnection, leakage or a tissued cannula.
TIVA administering tubing	Use a dedicated TIVA administering set which incorporates: • Antisiphon valves on the drug lines • A non-return valve on the IV fluid line • Minimal dead space between the patient and where the drugs and IV fluid mix • Luer lock rather than Luer slip connections.
Drug preparation	Have drugs already drawn up in preparation for syringe exchanges.
Monitoring	Consider processed EEG, particularly if NMBAs are used.
Practical aspects	Ensure the infusion continues after altering pump settings or a syringe exchange.
Specific to running TCI	Be aware that most TCI pumps will forget their programming with complete failure of both mains and battery power. Have a backup plan in the event of pump failure. Ensure the right model is used for the right drug. Ensure drug dilutions and syringe size and type are correctly entered into the pump. Double-check patient data are entered correctly. Ensure targets are suitable for the patient's age and ASA status.

Table 16.5 Suggested minimum TCI target concentrations

Age	Spontaneous breathing		IPPV	
	Propofol (micrograms/mL)	Remifentanil (nanograms/mL)	Propofol (micrograms/mL)	Remifentanil (nanograms/mL)
<50	4–6	1–3	3–4	5–8
>50	2–4	1–2	2–3	3–6

- Avoid reducing targets inappropriately to facilitate a more rapid emergence, particularly in short cases. However, after a prolonged TIVA-based anaesthetic, often emergence is slow and reducing targets before the end of surgery may be appropriate. This can be guided by processed EEG monitoring if used or with knowledge of the Cet at which the patient became unrousable.

- Most patients wake at a propofol Cet of 1–2 micrograms/mL. Remifentanil can be continued at an effect site of 1–2 nanograms/mL to smooth extubation and then stopped. ⚠ Remain vigilant for post-extubation apnoea.

Schnider vs Marsh

Each pharmacokinetic model uses different fixed and variable parameters, which helps to explain some of the important differences.

- Early Marsh models only provided Cpt, though the newer Modified Marsh now provides Cet. Age and weight are entered into the pump; however, only weight is used to calculate compartment sizes. The age input is only there to prevent the model's use in those <16y.
- Schnider's model provides both Cpt and Cet. Age, sex, weight and height parameters are entered into the pump.
- The most important clinical difference between Schnider and Marsh is the V_1 compartment size. Marsh is 19.4L vs Schnider 4.27L for an 85kg person. As a result, Marsh will give a larger bolus when uptitrated, compared to Schnider (when the same target site is used). This is important because:
  - Schnider may be more useful in frail/unstable adults as haemodynamic side effects are lessened.
  - Using Cpt with Schnider should really be avoided as the fixed, small V_1 leads to inconsistent results with inappropriately small boluses, independent of patient size.

Depth of anaesthesia monitoring for TIVA

⚠ As TIVA has no 'end-tidal' equivalent, it is recommended that processed EEG monitoring be used as an additional tool to determine depth of anaesthesia, particularly in paralysed patients (see ➲ pp. 426–9).

Morbid obesity

TIVA in the morbidly obese is practised, but caution must be exercised as TCI models are not validated in these patients and as such, target concentrations become unreliable. Currently, there is no consensus on whether to input total, lean, ideal or adjusted bodyweight for these patients. If TCI is used, target concentrations should be titrated to clinical effect, with processed EEG strongly recommended. The recent development of models specifically designed for the morbidly obese will likely resolve many of these issues.

Children

The use of TIVA in children is becoming increasingly popular due to reduction in PONV and emergence delirium, as well as use in conditions such as muscular dystrophy and central core myopathy. Paediatric TCI models include:

- Kataria: 3–16y and >15kg
- Paedfusor: 1–16y and 5–61kg.

Although this age group seems predisposed to propofol-related infusion syndrome in critical care settings, the risk of this developing during anaesthesia seems exceptionally low.

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Environmental impact of anaesthesia

As there is minimal metabolism of inhalational agents, nearly all end up in the environment unchanged. Although globally they are a small contributor to total greenhouse gas emissions, health organisations and anaesthetists have an important role to play in mitigating climate change and environmental degradation by reducing our carbon footprint. Per bottle of volatile, sevoflurane (250mL) produces 49kg of CO_2 equivalents, whereas desflurane (240mL) produces 886kg.[2]

- The climate crisis has repercussions for population health.[3] Reducing carbon emissions is vital to mitigate this effect. Anaesthetists are well placed to make these reductions within health care.
- Anaesthetic gases have a damaging effect on the environment.[2] The NHS Long Term Plan 2019 identifies reducing anaesthetic gas emissions as part of achieving net zero emissions by 2050.[4] In particular, desflurane and N_2O should be avoided as much as possible.
- Peripheral nerve blocks, neuraxial blocks and TIVA are significantly less carbon-intensive than volatiles,[5,6] and should be considered whenever clinically appropriate.
- Currently, anaesthetic gas capture technology is not widely established. As such, reliance on the future potential of technology is insufficient to meet the requirements of the NHS Long Term Plan.
- Other important concerns include embodied carbon within equipment and pharmaceuticals, as well as the significant contribution of the supply chain.[7] For example, over their lifetime, reusable equipment has been consistently shown to cost less and have a lower carbon footprint than disposable items.[8]
- Medical waste is tangible evidence of anaesthesia's environmental impact, but strategies to manage waste alone only addresses a small fraction of the carbon footprint of health care.[7]
- By choosing low-carbon, sustainable, cost-effective treatments and technologies, it is possible to maintain or improve patient outcomes while reducing the environmental, social and economic impacts of anaesthetic practice.

Courtesy of Dr Chris Allen, Environmental Sustainability (Anaesthetic) Fellow, Newcastle, UK.

Further reading

Al-Rifai Z, Mulvey D (2016). Principles of total intravenous anaesthesia: practical aspects of using total intravenous anaesthesia. *BJA Educ*, **16**, 276–80. doi: 10.1093/bjaed/mkv074

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Campbell M, Pierce JMT (2015). Atmospheric science, anaesthesia, and the environment. *BJA Educ*, **15**, 173–9. doi: 10.1093/bjaceaccp/mku033

Sedation

See ➲ Premedicants, p. 66; for premedication in children, see ➲ pp. 917–18.
Sedation refers to the use of drugs to produce anxiolysis, analgesia and a degree of amnesia, to allow patients to tolerate uncomfortable treatments or investigations without resorting to GA. Anaesthetists are well placed to provide effective and safe sedation. When offered by non-anaesthetists, the level of training, techniques used and degree of observation of the patient are highly variable.

- There is a perception that sedation is less stressful or safer than GA. Sedation may be requested in isolated areas such as radiology or the ED. Standards of equipment, assistance and monitoring may not be the same as in theatre. Nonetheless, sedation has the potential to pose significant risks.
- Sedation is a continuum from alertness to GA. Therefore, definitions of levels of sedation are somewhat arbitrary.

Minimal sedation

Minimal sedation is where a mild anxiolytic is provided. The patient remains conscious and has normal response to verbal stimulation.

Conscious sedation

Conscious sedation refers to a state where the patient may have their eyes closed but will still obey commands or respond to verbal interaction. Respiration is usually preserved.

Deep sedation

The patient responds only to repeated or painful stimuli, by purposeful movement (not simply withdrawal). Manoeuvres may be required to keep the airway open. This state is close to GA and may inadvertently turn into it.

Good practice

It is good practice to perform a full anaesthetic assessment on someone requiring sedation. Insert a cannula and apply routine monitoring. An anaesthetic assistant is likely to be helpful.

- Titrate drugs slowly to effect. Short-acting benzodiazepines are effective for anxiolysis and amnesia, but have no analgesic effect. Opioids have synergistic effects if used with them. TCI propofol is a good choice, as it is highly titrable if the patient becomes too deep.
- Often the most stimulating part is near the beginning, e.g. injection of LA into a sensitive area.
- Sedation is often carried out on elderly patients with significant comorbidities. Sedation in the elderly may produce paradoxical disinhibition and agitation.
- Communication is reassuring to the patient and allows monitoring of their conscious level.

Further reading

Blayney MR (2012). Procedural sedation for adult patients: an overview. *Contin Educ Anaesth Crit Care Pain*, **12**, 176–81. doi:10.1093/bjaceaccp/mks016

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Neuromuscular blockade

Depolarising agents

Suxamethonium is the only depolarising NMBA in clinical use. Given IV (dose 1–2mg/kg), it has a fast onset of action (<1min), making it the ideal agent when performing an RSI or for use in an airway emergency. Its short duration of action (5–10min) also means that it is well suited for procedures that require a brief period of muscle relaxation (e.g. ECT). Suxamethonium can also be given IM (dose 2–4mg/kg) in the absence of IV access.

Metabolism is by plasma cholinesterase, a hepatically synthesised circulating enzyme. Acquired factors that reduce the level of circulating plasma cholinesterase (e.g. hepatic diseases, malnutrition) or reduce plasma cholinesterase activity (e.g. presence of anticholinesterase drugs, pregnancy) can therefore lead to an increase in duration of action.

Congenital factors, such as variants in the genes that encode the plasma cholinesterase enzyme, can reduce its activity and prolong the duration of action of suxamethonium (Table 16.6).[9]

Adverse effects of suxamethonium may be predictable or unpredictable.

Table 16.6 Inheritance of plasma cholinesterase

Type	Genotype	Dibucaine number	Apnoea duration	Incidence
Normal	EuEu	80	1–5min	94%
Atypical	EuEa	60	10min	1:25
Atypical	EaEa	20	2h	1:3000
Silent	EuEs	80	10min	1:25
Silent	EsEs	Minimal activity	2h	1:100 000
Fluoride-resistant	EuEf	75	10min	1:300 000
Fluoride-resistant	EfEf	65	2h	1:150 000

Predictable adverse effects

Related to muscle fasciculation:

- Postoperative myalgia, especially in young, muscular adults.
- Increase in ICP and IOP. This is of minimal clinical significance in healthy patients but makes suxamethonium relatively contraindicated in those with an already raised ICP (e.g. traumatic brain injury) or in situations where any rise in IOP is undesirable (e.g. penetrating eye injury).
- Increase in plasma K^+ concentration (by 0.5mmol/L after 1mg/kg dose). This is inconsequential in most patients but may have deleterious effects in those who are already hyperkalaemic (see pp. 240–1).
- Parasympathomimetic actions, especially after repeated doses in children. These include bradycardia and ↑ salivary and bronchial secretions.

Unpredictable adverse effects

- ⚠ Anaphylaxis; suxamethonium has the highest rate of anaphylaxis, compared to other NMBAs (11 per 100 000 exposures in the NAP6 study).[10]
- ⚠ MH; suxamethonium is a known triggering agent in predisposed individuals.
- ⚠ Exaggerated hyperkalaemic response. This is due to the proliferation of extrajunctional, fetal-type nicotinic acetylcholine receptors in certain neuromuscular conditions (spinal cord injury, muscular dystrophies, prolonged immobilisation) and after severe burns. Stimulation of these abnormal receptors by suxamethonium results in greater than expected intracellular to extracellular K^+ leakage. Suxamethonium is safe within the first 24h of spinal cord injury or burns, with risk increasing after 2–3d.

Non-depolarising agents

Non-depolarising NMBAs can be classified by their structures into aminosteroids (rocuronium, vecuronium) and benzylisoquinoliniums (mivacurium, atracurium).

Dosing is based on the dose at which 50% of the patients would achieve a 95% twitch height depression (ED_{95}). After a standard intubation dose (ED_{95} × 2), onset of action is within 2–3min and duration of action ranges from 15min (mivacurium) to 30–40min for other agents. Onset of action for rocuronium can be reduced to within 30s when given at a higher dose (ED_{95} × 4), making it a useful alternative to suxamethonium when performing an RSI (Table 16.7).

Table 16.7 Dosing of non-depolarising NMBAs

Non-depolarising NMA	ED_{95}	Dose for standard intubation
Rocuronium	0.3mg/kg	0.6mg/kg (1–1.2mg/kg for RSI)
Vecuronium	0.05mg/kg	0.1mg/kg
Mivacurium	0.07mg/kg	0.15mg/kg
Atracurium	0.25mg/kg	0.5mg/kg
Cisatracurium	0.05mg/kg	0.1mg/kg

Elimination of aminosteroids is by a combination of hepatic metabolism and biliary and renal excretion. Mivacurium is metabolised by plasma cholinesterase and its duration of action can be prolonged by the same factors as outlined above for suxamethonium. The metabolism of atracurium is unique in that the majority of the drug undergoes non-organ-dependent Hofmann elimination (spontaneous degradation at body temperature and physiological pH), making it the ideal agent for use in patients with hepatic or renal impairment.

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Adverse effects of non-depolarising NMBAs

- Rocuronium: pain on injection.
- Atracurium: direct histamine release and production of a potentially epileptogenic metabolite (laudanosine). Cisatracurium is an enantiomerically pure preparation of atracurium that is more potent with less or no histamine release and significantly lower laudanosine concentrations.
- Anaphylaxis: rates are said to be similar among the non-depolarising NMBAs (3–6 per 100 000 exposures in the NAP6 study),[10] although some data suggest a higher rate for rocuronium.[11] ⚠ Risk of cross-reactivity is high between suxamethonium and aminosteroid NMBAs; in these patients, consider using a central neuraxial or regional anaesthesia technique. If this is not possible, consider a relaxant-free GA technique. If an NMBA is necessary, consider using cisatracurium or atracurium due to their low intrinsic rates of anaphylaxis and low risk of cross-reactivity in the setting of proven NMBA hypersensitivity.[11]

Neuromuscular function monitoring

⚠ Monitoring of neuromuscular function is mandatory whenever an NMBA is used. It can be used at induction to optimise timing for tracheal intubation, during maintenance to guide repeat NMBA dosing and at emergence to aid reversal of NMBA.

- A peripheral nerve stimulator (PNS) is applied to the skin over a peripheral nerve such as the ulnar nerve. It delivers a current of 40–70mA, a supramaximal stimulus sufficient to depolarise all axons. The different patterns of stimulation and their uses in different clinical situations is summarised in Table 16.8.[12]
- Assessment of muscle contractions may be qualitative or quantitative.
- Qualitative assessment relies on the visual and/or tactile confirmation of muscular contractions and is therefore limited by its subjective nature and its inability to discern minor degrees of block. For example, fade on the train-of-four (TOF) cannot be detected subjectively at a TOF ratio (TOFR) >0.4; for double-burst stimulation (DBS), this occurs at TOFR >0.6.
- Quantitative assessment is the gold standard. This employs a mechano-myographic, accelero-myographic or kine-myographic device coupled to the patient's thumb to objectively measure the number and strength of adductor pollicis contractions.
- If the hands are not accessible, stimulation of the facial nerve and qualitative assessment of orbicularis oculi and corrugator supercilii muscle contractions is a reasonable alternative.
- Clinical tests such as sustained head lift or hand grip for 5s have traditionally been used as indicators of adequate reversal of NMB. They have been shown to be inaccurate and unreliable and should not inform clinical practice.

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Table 16.8 Patterns of stimulation

Pattern of stimulation	Muscle response to depolarising NMBA	Muscle response to non-depolarising NMBA	Uses
Single twitch (ST): one stimulus at 1s or 10s intervals (1 or 0.1Hz)	Stimulus Response during onset of block	Stimulus Response during onset of block	Determines onset of action. Requires a baseline measurement before NMBA administration
Train of four (TOF): four stimuli at 0.5s intervals (2Hz)	Stimulus Response during partial blockade (no fade)	Stimulus Response during partial blockade (fade)	Assesses degree of non-depolarising NMBA block and recovery without needing a baseline measurement. Allows for quantitative assessment using TOFR
Post-tetanic count (PTC): tetanic stimulation (50Hz for 5s), 3s pause, then ST stimulation at 1Hz	Stimulus Response not useful clinically	Stimulus Response	Evaluation of the degree of block even when there is no response to TOF
Double-burst stimulation (DBS): two bursts of two minitetanic 50Hz stimuli separated by 0.75s	Stimulus Response during partial blockade (no fade)	Stimulus Response during partial blockade (fade)	Slight qualitative improvement in the evaluation of fade, compared with TOF-induced fade

Reversal of neuromuscular blockade

Why reverse neuromuscular blockade?

Postoperative residual curarisation (PORC) is defined as having a TOFR of <0.9 after anaesthesia. It is common (estimated incidence of 40% in recovery)[13] and often under-recognised.

- PORC is associated with ↑ rates of postoperative pulmonary complications such as respiratory failure, microaspirations, hypoxaemia and the need for reintubation.

How to reverse neuromuscular blockade

Suxamethonium- and mivacurium-induced NMB does not require pharmacological reversal. Giving an anticholinesterase drug may paradoxically increase the duration of block due to inhibition of plasma cholinesterase.

For other NMBAs, anticholinesterase drugs and sugammadex are the two main classes of reversal agents available. The choice of agent and dose required depend on the type of NMBA used and the depth of paralysis at the time of reversal. This is summarised in Table 16.9.[13]

- Neostigmine is an example of an anticholinesterase. When given at a dose of 0.05–0.07mg/kg, it has an onset time within 1–2min, reaching its peak effect around 10min, and duration of action of around 30min. It is co-administered with an antimuscarinic drug such as glycopyrronium (dose 0.01–0.015mg/kg) to mitigate anticholinesterase action at muscarinic acetylcholine receptors.
- Sugammadex is a cyclodextrin compound designed specifically to encapsulate aminosteroid NMBAs. Onset is rapid (within 2–3min), even in the setting of profound aminosteroid-induced NMB, when given in appropriate doses. Sugammadex cannot be used to reverse non-aminosteroid-induced NMB.

Table 16.9 Time course of recovery from rocuronium-induced neuromuscular blockade, associated monitoring and appropriate reversal

Depth of block	Time after 0.6mg/kg dose of rocuronium (median and range in min)	Neuromuscular monitoring modalities			Reversal drug and dosage (mg/kg)	
		TOFC	PTC	TOFR	SGX	Neo
Profound	5(3–15)	0	0	0	16	None
Deep	12(5–22)	0	≥1	0	4–8	None
Moderate	26(17–38)	1–3	NA	0	2	0.05–0.07
Shallow	30(20–45)	4(fade)	NA	0.1–0.4	2	0.05
Minimal	43(30–60)	4(no fade)	NA	>0.4 to <0.9	2	0.02
Full recovery	50(35–85)	4(no fade)	NA	≥0.9	None	None

Neo, neostigmine; PTC, post-tetanic count; SGX, sugammadex; TOFC, train-of-four count; TOFR, train-of-four ratio.

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Depth of anaesthesia monitoring

AAGA with explicit recall of intraoperative events is relatively common; prospective studies using the modified Brice interview have consistently reported an incidence of around 1–2 per 1000 cases.[14] Retrospective studies, such as NAP5, reveal a much lower incidence (1 per 20 000 cases overall).[15] Regardless, its occurrence can be devastating to the patient, with long-term psychological harm such as post-traumatic stress disorder a possibility.

AAGA occurs when too little anaesthetic is delivered to the brain. This may be due to:

- Failure of delivery, e.g. IV cannula failure during TIVA
- Inadequate anaesthesia to maintain unconsciousness. This may be unintentional (e.g. not accounting for age-related changes in MAC requirement) or intentional (e.g. purposefully reducing anaesthetic dose during periods of cardiovascular instability).

The risks of AAGA can be reduced by monitoring the effects anaesthetic drugs have on the brain or by monitoring anaesthetic drug concentrations.

Monitoring anaesthetic drug effects on the brain

Patient response to stimulation

A fully conscious patient is able to respond purposefully to verbal command or light tactile stimulation while maintaining a patent airway and SV. Increasing anaesthetic depth leads to progressive unresponsiveness to external stimuli, beginning with loss of verbal contact.

- The patient is judged to be in a deep plane of anaesthesia sufficient for surgery to commence when there is no response to sustained painful stimuli. However, in the presence of NMB, it becomes impossible to use this to distinguish an awake, paralysed patient from one who is adequately anaesthetised. Autonomic signs of arousal such as tachycardia, hypertension and lacrimation are unreliable indicators of consciousness.
- Therefore, use of NMBAs is considered the most frequently implicated risk factor for AGAA. Avoidance of pharmacological paralysis might be the most effective method of prevention, but this may not be indicated in all patients or possible for all surgery.

Processed EEG (pEEG) monitoring

The anaesthetised brain undergoes characteristic EEG changes, shifting from a high-frequency, low-amplitude asynchronous signal to one that is lower frequency, higher amplitude and more regular. This is followed by burst suppression where episodes of electrical quiescence (suppression) are interspersed with high-frequency, high-amplitude electrical activity (bursts), and then finally a completely isoelectric EEG (Fig. 16.2).[16]

Unfortunately, raw data from multichannel EEG are too complex for the anaesthetist to process in real time to guide anaesthesia titration, limiting their applicability. In contrast, commercial pEEG monitors, such as the BIS®, Narcotrend® and E-Entropy® monitors, use EEG data obtained from forehead electrodes to generate a simplified, user-friendly output (a calculated dimensionless variable between 0 and 100), giving an indication of the degree of cortical suppression (Table 16.10). This is done by using algorithms derived from analysing EEG data of healthy volunteers with known consciousness levels and exposed to known concentrations of anaesthetic drugs.

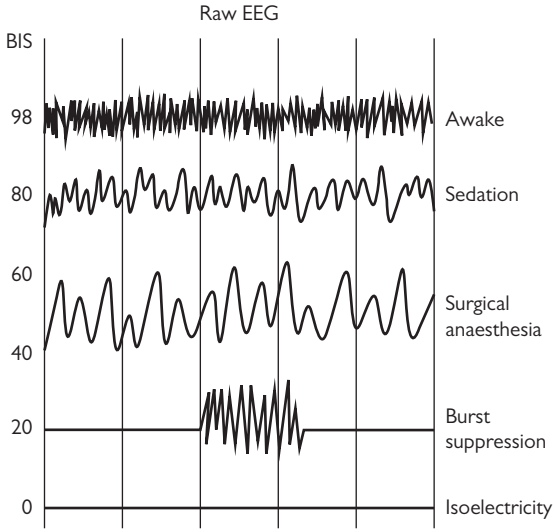

Fig. 16.2 EEG changes and corresponding BIS values with increasing depth of anaesthesia.

Table 16.10 Commonly used commercial pEEG monitors

Device	Main output and range	Target range
BIS®	BIS: 0 (almost flat EEG activity) to 100 (awake)	40–60
Narcotrend®	Narcotrend stage: stage A (awake) to F (deep anaesthesia) Narcotrend index: 0 (no detected electrical activity) to 100 (awake)	Stage E
E-Entropy®	Response entropy: 0 (suppressed EEG) to 100 (awake) State entropy: 0 (suppressed EEG) to 90 (awake)	40–60
SedLine®	Patient State index (PSi): 0 (suppressed EEG) to 100 (awake)	25–50

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Limitations of pEEG monitors

- Electromechanical interference can falsely elevate the displayed value. This may be due to artefacts from electrical equipment used in the vicinity of the pEEG electrodes or signal contamination from facial muscle activity. Studies have demonstrated a fall in BIS with onset of muscle paralysis and a rise after reversal of blockade.
- Ketamine and N_2O do not produce the same pattern of EEG changes as described above, making pEEG monitors less useful when they are used to supplement propofol or volatile anaesthesia.
- A 15–30s time lag in the processing of raw EEG data means that the monitors are only able to provide information about the conscious state after it has arisen. Therefore, there is a risk of AAGA if a pEEG monitor is used reactively. Instead, anaesthesia should be deepened in anticipation of an impending surgical stimulus before a rise in pEEG values has occurred.

Monitoring anaesthetic drug concentrations

End-tidal anaesthetic gas (ETAG) monitoring

ETAG monitoring provides a real-time display of volatile anaesthetic concentration in the brain as long as a state of equilibrium is reached between the lungs (where the measurements take place), plasma and the brain (the target end-organ). When the measured concentration is compared to the MAC value of each agent, it gives an indication as to whether the dose delivered is adequate in achieving a certain clinical endpoint on a population level. Incidence of AAGA is low when the measured concentration is above 0.7 age-adjusted MAC.

Limitations of ETAG monitoring

- Population-derived quantal dose–response relationships such as MAC may not apply to individuals; thus, a 0.7 age-adjusted MAC may be an underdose or an overdose for certain patient groups.
- Limited utility around the time of induction as equilibrium has yet been achieved; therefore, measured ETAG concentrations do not accurately represent concentrations in the brain.

Estimated plasma and effect site propofol concentrations

Point-of-care blood propofol measurement is not currently widely available. Instead, a TCI pump estimates plasma and effect site propofol concentrations based on its algorithm and patient variables.

Limitations of estimating propofol concentrations

- The pump assumes successful IV delivery of propofol and would display an adequate estimated brain propofol concentration, even when an IV cannula has failed.
- Population-derived assumptions do not apply well to individuals at the fringes of the pharmacokinetic models, e.g. the obese or elderly.
- Current pharmacokinetic models are not validated in pregnancy.

Evidence-based approach to the use of depth of anaesthesia monitors

ETAG monitoring is simple, reliable and readily available. Its alarm should be turned on and set to an age-adjusted MAC of >0.7 whenever a volatile agent is used as the primary anaesthetic. Use of an additional pEEG monitor is unnecessary, even in high-risk patients, as prospective RCTs (B-Unaware, BAG-RECALL, MACS) have failed to demonstrate the superiority of a BIS-guided protocol in reducing AAGA, when compared to ETAG monitoring.[17,18,19]

Processed EEG monitors should be used for patients receiving an NMBA with TIVA or for those having TIVA who are at high risk of AAGA. The B-Aware trial demonstrated that a BIS-guided protocol (aiming for a BIS of between 40 and 60) reduces the risk of AAGA, compared to using clinical signs alone.[20]

Processed EEG monitors may also have a role in:

- Titrating anaesthetic dose in those who are at risk of an underdose (e.g. chronic substance misusers) or an overdose (e.g. frail patients) of volatile anaesthesia when targeting an age-adjusted MAC of >0.7
- Titrating TIVA in patients outside of the demographic norms of the pharmacokinetic models employed in TCI systems.

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Cardiac output monitoring

Accurate quantification of CO is an integral part of advanced haemodynamic monitoring, which is a cornerstone in the management of high-risk surgical and critically ill patients. Monitoring CO helps guide IV fluid therapy and vasopressor and inotropic support, with the aim of improving CO and O_2 delivery to tissues. When incorporated into protocols with target thresholds, they form the basis of GDFT.

Goal-directed fluid therapy

The fundamental principle of GDFT is to minimise complications associated with fluid imbalance perioperatively through optimisation of CO and tissue perfusion. Parameters from CO monitors are used to help differentiate fluid-responsive from non-fluid-responsive patients. Fluid-responsive patients are on the upward slope of the Frank–Starling curve (Fig. 16.3), with a fluid challenge resulting in an ↑ stroke volume and CO. Repeated fluid boluses may continue to improve the stroke volume. Once a patient reaches the plateau on the Frank–Starling curve, there is no further improvement in stroke volume. Ongoing fluid administration will be ineffective or even potentially harmful. In this setting, vasopressors may be employed to support BP, or if CO remains low, this may be a situation suitable for inotropic support.

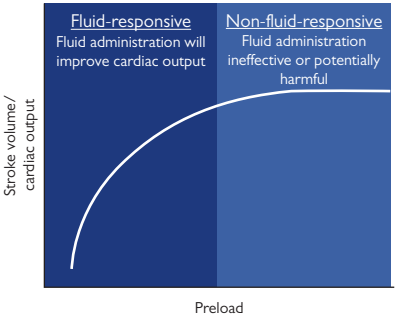

Fig. 16.3 Simplified explanation of the Frank–Starling law.

- The evidence supporting GDFT is conflicting. Early studies showed dramatic improvements in morbidity and mortality which led to rapid uptake into clinical practice and incorporation into ERAS protocols.[21] However, more recent large RCTs have failed to replicate many of these findings. This is likely the result of widespread uptake of ERAS and restrictive fluid strategies as a standard of care. Although GDFT appears to be superior to traditional liberal or fixed volume approaches,

it seems to offer minimal additional benefit when compared to a restrictive fluid strategy, especially if this is combined with ERAS.
- This is not to say GDFT is redundant. Literature has consistently demonstrated a reduction in morbidity and hospital length of stay when GDFT is applied to high-risk surgical patients.[22] Further, following the publication of the RELIEF trial,[23] overly restrictive fluid regimes may also be harmful in higher-risk patients undergoing major abdominal surgery. GDFT may therefore have greater benefit in high-risk patients and thus should be reserved for this patient group.

Cardiac output monitors

There are numerous CO monitors available utilising various technologies, which can lead to confusion when trying to differentiate among them.[24] They can be invasive, minimally invasive or non-invasive. They can also be calibrated or non-calibrated, with parameters being directly measured or derived.

Invasive

Pulmonary artery flotation catheter

Regarded as the gold standard for measuring CO.
- Cold or room temperature 0.9% sodium chloride is injected into the right atrium through the proximal port, with temperature change measured in the PA using a thermistor close to the tip of the catheter. CO and other parameters are then derived using the thermodilution curve and the Stewart–Hamilton equation.
- Newer devices can provide continuous CO by instead mildly heating the blood and measuring the temperature change in the PA.
- Reliability of measurements can be affected by tricuspid incompetence, shunts and misplacement of the catheter tip.
- Use has declined due to PA catheters being associated with ↑ mortality in ICU patients and the availability of less invasive monitors.

Minimally invasive

Arterial waveform analysis

These devices analyse the shape of the arterial waveform to calculate stroke volume, CO and many other derived parameters. They are able to provide continuous haemodynamic information, allowing for real-time monitoring of fluid responsiveness.
- Accuracy is dependent on the consistency of the waveform and can be affected by arrhythmias, rapid changes in haemodynamic stability, valve insufficiency, aortic aneurysms and the use of an intra-aortic balloon pump (IABP).
- Non-calibrated devices use a sensor attached to a standard arterial cannula. They cannot measure a true CO but derive this from the arterial trace. As such, they provide less accurate absolute numbers, compared to calibrated devices. However, they are still able to provide useful numerical trend information which can guide therapy.
- Non-calibrated devices:
  - ProAQT™—pulse contour analysis
  - LiDCOrapid™—pulse power analysis
  - Flotrac™ with EV1000™ or Vigileo™ monitors—pulse contour analysis.

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- Calibrated devices utilise a 2nd technique intermittently to accurately measure CO and then correlate this with the arterial trace to provide a continuous CO. As such, they are resistant to changes in haemodynamic stability and tend to provide more accurate absolute numbers. They often require insertion of a central venous line and are therefore slightly more invasive than non-calibrated devices.
- Calibrated devices:
  - PiCCOplus™—pulse contour analysis calibrated with transpulmonary thermodilution. Requires a thermistor-tipped arterial catheter and a central venous line.
  - LiDCOplus™—pulse power analysis calibrated with transpulmonary lithium dilution. A lithium sensor attaches to a standard arterial cannula. Lithium chloride is injected into either a central venous line or peripheral cannula. Measurements are affected by concurrent lithium treatment and high peak doses of NMBA. Contraindicated in <40kg and 1st trimester of pregnancy.
  - Volumeview™ with EV1000™ monitor—pulse contour analysis calibrated with transpulmonary thermodilution. Requires a thermistor-tipped catheter in the femoral artery and a central venous line.

Doppler technology

- Oesophageal Doppler measures blood flow velocity in the descending aorta and multiplies this by the estimated cross-sectional aortic area to calculate stroke volume. Aortic diameter is estimated from a nomogram or directly measured, depending on the device.
- Oesophageal Doppler-guided fluid management has been associated with reduced hospital stay.
- Placement in patients at risk of oesophageal trauma or bleeding needs special consideration.
- Inaccuracy of absolute values can be caused by epidural anaesthesia, thoracic aortic aneurysms or morbid obesity, although trends and dynamic parameters can still be used to guide fluid therapy.

Non-invasive

Vascular unloading technique

Also known as the finger volume clamp method. An infrared light source and a sensor continuously measure blood volume in a finger. A finger pressure cuff inflates and deflates to maintain a constant blood volume. This fluctuation in cuff pressure produces a continuous BP waveform from which haemodynamic variables can be derived. These are new to the market, with limited validation studies.

Applied Fick principle

A rebreathing loop incorporating CO_2 and airflow sensors is attached to the ventilator circuit, allowing partial rebreathing of CO_2. Assuming CO_2 production, alveolar ventilation and CO remain unchanged over the rebreathing cycle, CO can be calculated. Requires an intubated, sedated and mechanically ventilated patient. It is unable to provide many of the useful parameters other devices provide.

Bioimpedance/bioreactance

Cyclical changes in blood flow within the thorax induce changes in impedance to alternating electrical current, which can be measured using electrodes. Newer devices use bioreactance, which is less susceptible to interference and shows good correlation with pulmonary artery flotation catheters (PAFCs).

Parameters of volume responsiveness

Most CO monitors display a multitude of measured and derived haemodynamic parameters. These can be either static or dynamic.

Static parameters

- These parameters include stroke volume, CO, CVP, pulmonary artery occlusion pressure (PAOP), mixed venous O_2 saturations, SVR, etc. Unfortunately, the absolute values for these parameters fail to predict volume responsiveness in the majority of patients. However, they can be reliably used to observe trends and the effect of interventions such as fluid administration.

Dynamic parameters

- These include stroke volume variation (SVV) and pulse pressure variation (PPV). Changes in intrathoracic pressure due to IPPV induce changes in the stroke volume and pulse pressure as a result of a reduction in preload. The greater the change in stroke volume or pressure, the greater the likelihood that the patient is intravascularly deplete.
- Dynamic parameters are superior to static parameters when determining volume responsiveness; however, the reliability of these variables can be adversely affected by arrhythmias, right heart failure, spontaneous breathing and V_T <8mL/kg.

Fluid administration

The interpretation of data from CO monitoring should be made in light of clinical examination and investigations such as serum lactate and base deficit trends.

- When trying to optimise stroke volume, fluid is usually administered in boluses over 5min, in volumes of around 3mL/kg (200–250mL) (Table 16.11).
- The response to a passive leg raise (or head-down position) is also a reversible way to demonstrate whether the patient is likely to be volume-responsive.

Table 16.11 Recommended thresholds for GDFT

Parameter	Thresholds	Intervention recommended
Stroke volume (SV)	SV increase >10% following 200–250mL fluid challenge over 5–10min	Give further volume challenge of 250mL
	SV increase <10% following 200–250mL fluid challenge over 5–10min	No further fluid. Continue to monitor SV
SVV, PPV	>12–13%	Give volume challenge of 250mL
Corrected flow time (FTc) (oesophageal Doppler)	<0.35s	Give volume challenge of 250mL

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Temperature control

Core body temperature in an awake patient is tightly controlled between 36.7°C and 37.1°C. Deviation above or below these temperatures causes the hypothalamus to initiate behavioural and autonomic mechanisms to restore normothermia. This tight regulation is lost under GA and neuraxial anaesthesia due to:

- Drug-induced vasodilation
- Abolition of behavioural responses to thermal discomfort and
- Resetting of the threshold temperatures in which autonomic responses are activated, resulting in a widened range.

The anaesthetised patient is prone to hypothermia (defined as core body temperature <36°C). This typically occurs in a triphasic pattern.

Phase 1

Rapid loss of ~1°C within the first 30–45min of anaesthesia due to heat redistribution from the core compartment to the peripheries.

Phase 2

Loss of ~1°C over 2–3h from radiation (40%), convection (30%), evaporation (25%) and conduction (5%).

Phase 3

A plateau occurs when heat loss is finally matched by heat production as maximal vasoconstriction is activated.

Hypothermia in the perioperative period is not benign. Adverse consequences include:

- Coagulopathy and an ↑ need for transfusion
- ↑ risk of surgical site infection
- ↑ rate of MACE
- Reduced rate of drug metabolism (e.g. prolonged NMB and delayed emergence from anaesthesia).

Measures to reduce the risk of perioperative hypothermia include:

- Identifing high-risk patients (high ASA grade, low BMI, undergoing major surgery and using a combined general/neuraxial technique) and treating hypothermia before commencing anaesthesia
- Monitoring core body temperature (distal oesophagus, nasopharynx, tympanic membrane or bladder) at 30min intervals
- Maintaining ambient temperature in preop areas and operating theatres, prewarming fluids to 37°C for IV administration or internal irrigation and humidifying respiratory gases using an HME filter
- Active cutaneous warming, most commonly achieved using a forced air device, which should be used for all procedures with anaesthesia time >30min
- Active internal warming techniques, such as CPB and peritoneal lavage, are invasive and therefore are not indicated for most routine operations.

Patient positioning for surgery

Positioning patients for surgery is a compromise between optimising surgical access and limiting patient harm from its deleterious effects.

Supine position

The supine position provides good operative access for many procedures. One or both arms may be abducted out on padded arm boards or tucked in next to the patient's body using lift sheets (Fig. 16.4).

Physiological changes

- Reduced FRC due to cranial displacement of the diaphragm when lying supine, compounded by the loss of diaphragmatic tone after induction of anaesthesia. This can be mitigated by the use of PEEP or a slight head-up tilt.
- Aortocaval compression. This can occur in advanced stages of pregnancy leading to hypotension. It may be alleviated by a slight leftward bed tilt or by using a wedge under the right hip.

Potential complications associated with supine positioning

- The arms should be abducted <90° to avoid stretch injury to the brachial plexus; the forearm should be neutral or in supination to minimise pressure on the ulnar nerve. When adducted, the arm should be in a neutral position, with the palm facing the patient.
- Back strain—this can be reduced by placing a pillow under the knees to restore the normal lumbar lordotic curve.
- Pressure injuries to the occiput, sacrum and heels can occur after prolonged contact with the operating table. Intermittent repositioning of the head and the use of gel heel pads and gel mattress help reduce this risk by pressure redistribution.

Fig. 16.4 Supine position. Image courtesy of Medical photography, Waikato District Health Board.

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Trendelenburg (head-down) position

Trendelenburg positioning requires the supine patient to be tilted head down, improving access to pelvic organs. The patient is secured using a variety of devices, including slip-resistant table padding, straps and vacuum bean bag supports. The arms may be abducted and secured onto arm boards or, more commonly, adducted and tucked in by the side.

Complications associated with Trendelenburg position

- Increase in venous return and central blood volume (effect increases with degree of tilt). May compromise patients with cardiac disease.
- Reduced FRC and lung compliance, leading to impaired ventilation and atelectasis.
- Venous congestion leading to raised ICP and IOP and the potential for airway oedema after a long case.
- Patients may slide on the table, resulting in pressure areas, inadvertent endobronchial intubation, traction on lines and tubes or limbs falling from the table.
- Passive regurgitation of gastric contents is more likely.

Reverse Trendelenburg position

With reverse Trendelenburg positioning, the supine patient is tilted head up to improve access for open or laparoscopic upper abdominal surgery. The patient is secured onto the operating table, as described previously, with the addition of a padded foot board if necessary.

Venous pooling in the lower extremities is the most significant adverse physiological change. This leads to a reduction in venous return, CO and BP. Increasing the angle of tilt and a diminished cardiac reserve exaggerate the magnitude of these changes. Implementing a gradual, incremental head elevation, giving IV fluids together with a vasopressor and applying compression stockings may mitigate these effects.

Potential complications are similar to those described above, except that the risk for passive regurgitation is not elevated.

Lithotomy and Lloyd-Davies positions

With lithotomy positioning, the supine patient's legs are separated, with the hips and knees flexed to a variable degree and supported using stirrups or slings (Fig. 16.5). This is often used in combination with a degree of Trendelenburg to provide access for urological, gynaecological and rectal procedures. In hip fracture surgery, a hemilithotomy position may be used where the operative leg is placed in traction and the non-operative leg in a lithotomy stirrup.

The Lloyd-Davies position is similar to lithotomy, with the hips and knees flexed to a much lesser extent (Fig. 16.6).

Adverse physiological effects occur as a result of a rise in abdominal pressure, which can in turn obstruct venous return (leading to hypotension), reduce lung compliance and impair ventilation.

Potential complications associated with lithotomy positioning

- Crush injury to the hand of the adducted arm is possible when the stirrup is being adjusted or when the foot section of the operating table is reattached. It is thus imperative to confirm that the hands are in a safe position before these actions are carried out.

Fig. 16.5 Lithotomy position. Image courtesy of Medical photography, Waikato District Health Board.

Fig. 16.6 Lloyd-Davies position. Image courtesy of Medical photography, Waikato District Health Board.

- The peroneal nerve is at risk of compression injury against the fibular head in the stirrup, and the sciatic nerve is at risk of stretch injury from prolonged, extreme hip flexion.
- Compartment syndrome is possible for surgery lasting longer than 2–3h in the lithotomy position.[25] Legs should therefore be elevated to the least possible extent for the shortest amount of time. Concurrent Trendelenburg positioning, intraoperative hypotension and the use of a sequential compression device on the calves have also been implicated and should either be minimised or avoided.

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Lateral decubitus position

The lateral decubitus position allows access to the non-dependent side for operations such as hip arthroplasty. Flexion is often added just beneath the iliac crest to increase exposure for operations such as nephrectomy or thoracotomy.

The torso is kept in place using a vacuum bean bag or pelvic and lumbar supports. The head should be kept neutral using pillows (check lower ear is not folded and eyes not pressed upon). The dependent arm rests on a padded arm board, with a roll placed between the chest wall and the operating table to alleviate pressure on axillary structures. The non-dependent arm is secured on an arm support. Legs are placed in slight flexion, with a pillow between the knees (Fig. 16.7).

Fig. 16.7 Lateral decubitus position. Image courtesy of Medical photography, Waikato District Health Board.

Physiological changes include

- Venous pooling in the dependent leg leads to reduced venous return, CO and MAP. This is exacerbated by concurrent lateral flexion at the hips, especially if the IVC becomes obstructed.
- Increase in V/Q mismatch in mechanically ventilated patients due to preferential perfusion in the dependent lung and preferential ventilation in the non-dependent lung. This can result in hypoxaemia.

Potential complications associated with lateral decubitus positioning

- The brachial plexus in the dependent arm is at risk of compression injury in the axilla; the chest should therefore be supported with a roll caudal to this point or by using a bean bag with its upper edge aligned below the level of the axilla.
- The brachial plexus in the non-dependent arm is at risk of stretch injury if patient's neck is flexed laterally downwards or if the arm is placed in

forward flexion >90°, as is often required in thoracic surgery. The head should therefore be supported in a neutral position throughout the operation and the extent and duration of forward arm flexion should be discussed with the surgeon.

- The peroneal nerve in the dependent leg is at risk of compression against the fibular head and the operating table; adequate padding is essential.

Prone position

The prone position is used for the posterior approach to the spine, superficial procedures on the back, surgery on posterior extremities and rectal and buttock procedures.

Patients are usually anaesthetised and intubated in the supine position before being turned prone onto the operating table. The head is rested on a foam or gel head rest or held in skull pins using a Mayfield clamp system. The torso is supported using firm rolls or bolsters under the chest and pelvis, taking care not to compress the abdomen. More commonly for spinal surgery, a dedicated torso support system such as the Wilson frame or a specialised Jackson table is used. Arms are placed on arm boards in the 'surrender' position—the shoulders in slight forward flexion and abduction, the elbows flexed and the hands up. Arms are tucked in by the side if there is limited range of motion in the upper extremities. The hips and knees are flexed, with the feet resting on a pillow (Fig. 16.8).

Fig. 16.8 Prone position. Image courtesy of Medical photography, Waikato District Health Board.

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Complications of abdominal compression

- Caval compression leads to a reduction in venous return, CO and MAP. It also increases the risk of epidural venous plexus bleeding during spine surgery.
- Cranial displacement of the diaphragm leading to reduced compliance and impaired ventilation.

Complications associated with prone positioning

- Injuries can occur while turning an anaesthetised patient prone. This has to be done in a coordinated fashion by staff familiar with the process. Tubes, lines and catheters need to be secured to prevent dislodgement. Monitoring cables should be minimised or removed completely to avoid entanglement.
- Neurovascular compromise can occur in the upper extremities due to axillary compression or stretch injury from overabduction and overextension of the shoulders.
- Prone spinal surgery is associated with ischaemic optic neuropathy, resulting in permanent vision loss. Risk factors include prolonged multilevel surgery and large volume blood loss. The use of the Wilson frame has also been implicated, potentially because it positions the head below the level of the heart.[26] Measures to reduce this risk include: staging complex spinal surgery; avoiding hypotension; large-volume crystalloid fluid administration; and keeping the head neutral at, or above, the level of the heart.

Sitting position

Surgery on structures in the posterior fossa of the cranium or on the shoulder often requires the patient to be in the sitting position. Measures to ensure safety in this position include:[27]

- Fixing the head in skull pins for neurosurgery or supporting it between a headrest and a padded face mask for shoulder surgery.
- Ensuring the buttocks are firmly against the operating table and aligned with the break of the bed. A safety strap across the lap and pillows under the legs prevent the patient from sliding caudally.
- Placing adducted arms on padded armrests adds lateral stability. For shoulder surgery, the operative arm can be secured to a commercial arm-holding device or rested freely on a padded Mayo stand (Fig. 16.9).

Physiological changes associated with the sitting position are similar to those already outlined for the reverse Trendelenburg position.

Complications associated with sitting position

- CVE. The sitting position is associated with a drop in cerebral perfusion pressure, potentially resulting in ischaemia. Maintaining O_2 delivery is essential. This can be achieved by having:
  - Adequate pressure. Set a MAP target around the patient's baseline (or at least 70mmHg for an otherwise healthy patient) and maintain this with vasopressors and IV fluids. The table should be raised in a gradual, incremental manner to allow time for treatment to be instituted. Ensure measurements reflect pressure in the cerebral circulation by zeroing the arterial line transducer at the level of the external auditory meatus. If NIBP monitoring is used, the hydrostatic pressure difference between the level of the cuff and the brain should be accounted for and the MAP target adjusted accordingly (1mmHg for every 1.25cm difference in height).

Fig. 16.9 Sitting position. Image courtesy of Medical photography, Waikato District Health Board.

- Adequate flow. The position of the head and neck should be anatomical to ensure that neck vessels are not occluded. Avoid hypocapnia which induces cerebral vasoconstriction.
- Adequate O_2 content. Avoid anaemia and hypoxaemia.
- Use of IV sedation and interscalene regional anaesthesia rather than GA with IPPV has been shown to be associated with less hypotensive episodes for shoulder surgery in the sitting position and may be the preferred technique in higher-risk patients.

- Venous air embolism (VAE). This is possible during posterior fossa craniotomy because the operative site is above the level of the heart and injury to dural venous sinuses simultaneously creates a pressure gradient and a portal of entry for air entrainment.
- Pressure injuries at the elbow and over the ischial tuberosity, resulting in ulnar and sciatic nerve compression respectively.

Further reading

Welch MB (2019). Patient positioning for surgery and anesthesia in adults. [In UpToDate database on the Internet] https://www.uptodate.com/contents/patient-positioning-for-surgery-and-anesthesia-in-adults

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Postoperative nausea and vomiting

(See also ➔ p. 925.) Nausea is the unpleasant sensation of the need to vomit. Vomiting is the forced expulsion of upper GI contents via the mouth.

- PONV refers to nausea or vomiting occurring during the first 24–48h after surgery. The incidence is around 30% in adults and 42% in children, rising to 80% in unmedicated high-risk patients.
- Post-discharge nausea and vomiting refers to PONV experienced after discharge from recovery. The incidence is about 37%.

Consequences of PONV

- Patient morbidity: wound dehiscence, bleeding, pulmonary aspiration, oesophageal rupture, fluid and electrolyte disturbances, ↓ patient satisfaction.
- ↑ health care costs: prolonged stay in post-anaesthesia care unit, delayed hospital discharge, unanticipated hospital admission, readmission to hospital post-discharge.

Neural pathways for nausea and vomiting

Afferent inputs arise via the chemoreceptor trigger zone (CTZ; also known as the area postrema), the GI tract via the vagus nerve, the vestibular system and the cerebral cortex.

- The CTZ is a circumventricular organ located on the dorsal surface of the medulla, on the floor of the 4th ventricle. It lacks the characteristic endothelial tight junctions of the blood–brain barrier, allowing communication between the CSF and blood.
- The vomiting centre/'central pattern generator for vomiting' is the integration of these inputs into a final common pathway and is located in the medulla.

The nucleus tractus solitarius triggers vomiting by stimulating the rostral nucleus, nucleus ambiguus, ventral respiratory group and dorsal motor nucleus of the vagus.

- Vomiting is preceded by characteristic autonomic changes (salivation, sweating, hypertension, tachycardia, cutaneous vasoconstriction) and subsequent coordinated contraction of the diaphragm and abdominal muscles to expel gastric contents.

The following receptors are involved: histaminergic (H_1), dopaminergic (D_2), serotonergic (5-HT_3), muscarinic and neurokinin (NK-1).

Risk factors for PONV

Patient factors

- ♀ gender (3× risk)
- Previous PONV or motion sickness (2–3× risk)
- Non-smokers (2× risk)
- Age <50y (2× risk).

Note: ASA status, BMI, menstrual cycle phase, perioperative fasting status and preoperative anxiety have no proven association with PONV.

Anaesthetic factors

- Use of volatile anaesthesia (2× risk). Note this risk increases in a dose-dependent manner and risk peaks in the first 2–6h following surgery
- Duration of anaesthesia (1.5× risk if exceeding 90min)
- Postoperative opioid use (1.5× risk). Note that this is independent of the opioid administered
- Use of N_2O (1.5× risk).

Note: NGT placement, muscle relaxant reversal and use of supplemental O_2 have no proven association with PONV.

Surgical factors

- Laparoscopic surgery, gynaecological surgery and cholecystectomy.

Note: head and neck, breast, abdominal, posterior cranial fossa and ophthalmic surgery are all associated with ↑ PONV but have not been proven to be independent risk factors.

Simplified risk scores for predicting PONV

- The **Apfel PONV risk score** assesses four domains: ♀ gender, history of PONV and/or motion sickness, non-smoking status and predicted postoperative use of opioids. When zero, one, two, three or four factors are present, the risk of PONV is 10%, 20%, 40%, 60% and 80%, respectively.
- The **Koivuranta PONV risk score** assesses five domains: ♀ gender, non-smoking status, history of PONV, history of motion sickness and duration of surgery >60min. If zero, one, two, three, four or five risk factors are present, the incidence of PONV is 17%, 18%, 43%, 54%, 74% and 87%, respectively.
- The **POVOC PONV risk score** for paediatric patients assesses five domains: duration of surgery ≥30min, age ≥3y, strabismus surgery and history of PONV in the child or of PONV in his/her relatives. When zero, one, two, three or four risk factors are present, the incidence is 9%, 10%, 30%, 55% and 70%, respectively.
- The **Apfel PDNV score** assesses five domains: ♀ gender, age <50y, history of PONV, opioid use in the post-anaesthesia care unit and nausea in the post-anaesthesia care unit. When zero, one, two, three, four or five factors are present, the risk of PONV is 10%, 20%, 30%, 50%, 60% and 80%, respectively.

Prevention and prophylaxis

Risk assessment

- If low risk (0–1 risk factors), consider no prophylaxis.
- If moderate risk (2–3 risk factors), consider 1–2 interventions.
- If high risk (4+ risk factors), consider >2 interventions, including regional anaesthesia.

Methods to reduce PONV

- Prophylactic antiemetic administration (Table 16.12).
- Avoid GA and opioids by using LA or regional anaesthesia (9× reduced risk).
- Avoid volatile and N_2O by using propofol for induction and maintenance (number needed to treat (NNT) = 5).
- Use multimodal analgesia to reduce opioid requirement.

Non-pharmacological methods to reduce PONV include adequate hydration, and acupuncture at P6 point, on the volar wrist (NNT = 5).

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Table 16.12 Drugs available for prophylaxis and treatment of PONV

Intervention	Mechanism	Dose	NNT	Notes
First line				
Dexamethasone	Steroid	4–8mg IV at induction Single dose only	4	4mg equal in effect to 1.25mg of droperidol and 4mg of ondansetron. Reduces postoperative opioid use. Does not increase risk of wound infection, may cause lability in blood glucose readings in diabetic patients
Droperidol	Butyrophenone D_2 antagonist	0.625–1.25mg IV at induction Q8H	5	Superior to metoclopramide. Similar effect on corrected QT (QTc) interval to ondansetron; however, when used together, does not significantly further increase QTc. Haloperidol at low doses (<2mg) has similar antiemetic efficacy. May cause sedation, anxiety, extrapyramidal signs (EPS) and abnormal LFTs
Ondansetron	$5HT_3$ antagonist	4–8mg IV at end of surgery Q6H	6	Less effective than aprepitant and palonosetron. Similar activity to granisetron, tropisetron and dolasetron. Constipation and headache are common. May cause elevated LFTs
Second line				
Aprepitant	NK-1 antagonist	Single dose of 40–80mg PO preinduction	4	40h half-life. More effective than ondansetron at 24 and 48h postoperatively
Cyclizine	H_1 antagonist Antimuscarinic	25–50mg IV Q8H	10	Small increase in HR via anticholinergic activity. May cause sedation, dry mouth and blurred vision. Painful on injection
IV fluid	Suppression of ADH release	30mL/kg IV	5in high-risk patients	Superior to 10mL/kg IV. No difference between crystalloid or colloid. ADH release associated with nausea and vomiting

Intervention	Mechanism	Dose	NNT	Notes
Metoclopramide	Benzamide D_2 antagonist	10–20mg IV Q8H	30 for 10mg 16 for 25mg	EPS, abdominal cramping, orthostatic hypotension, QTc prolongation, sedation, hyperprolactinaemia
Midazolam	Benzodiazepine	2mg IV 30min prior to end of surgery	6	More effective 30min prior to end of surgery than as a premed
Prochlorperazine	Phenothiazine D_2 antagonist	5–20mg IV 12.5mg IM Q6H		EPS, orthostatic hypotension, QTc prolongation, sedation, hyperprolactinaemia
Propofol	Unknown	TIVA 20mg IV boluses PRN for treatment	5	Only administer in a monitored environment. Antiemetic effect of small boluses likely brief
Scopolamine (hyoscine hydrobromide)	Anticholinergic	1.5mg transdermal patch from the evening prior up to 2h before induction	6	2–4h for onset of effect. May cause visual disturbance, dry mouth and dizziness. CNS effects in elderly patients

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Treatment principles

Using combination therapy with drugs targeting different receptor classes is the most effective strategy due to their additive effect.

- Ondansetron, dexamethasone and droperidol are the most studied and are widely considered to be 1st-line antiemetics in both the prophylaxis and treatment of nausea and vomiting.
- In the context of failed prophylaxis, repeating doses of prophylactic antiemetics already administered is of no benefit. Choose a drug from another class.

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Chapter 17

Blood products and fluid therapy

Nicholas Eaddy and Alexandra Cardinal

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Blood products

The goal of transfusion is to efficiently correct tissue hypoperfusion, coagulopathy or anaemic hypoxaemia, while minimising the risk of transfusion-related adverse outcomes for patients. Blood products are largely obtained through voluntary donation prior to undergoing expensive and labour-intensive processing, storage and dispensing. Consideration of both the intrinsic and financial values of blood products is appropriate. In this spirit, we aim to give the right blood products to the right patients at the right time in the right amount.

Donation and testing

Whole blood donation

Each donor provides 500mL of blood. A total of 470mL goes on to be processed while 30mL is utilised for routine testing. Donors can typically give blood four times annually.[1]

Apheresis donation

A cell separator is used to collect plasma, platelets, white cells or haematopoietic progenitor cells. Donor red cells are returned to the donor. Donors may give these fractions every 2w.[1]

Testing

Donated blood is tested for ABO/rhesus D (RhD) blood groups and screened for infectious diseases (HIV, hepatitis B, hepatitis C, human T cell lymphotropic virus (HTLV)-1/2 and syphilis). Donations at risk of malaria or Chagas disease are tested for *Plasmodium* and *Trypanosoma cruzi*. Cytomegalovirus (CMV) antibody-negative components derived from CMV antibody-negative donors are available for immunosuppressed patients and neonates.

Blood processing

Whole blood is a heterogeneous suspension of cellular and protein elements in plasma. Donated blood is usually processed into a range of blood components and fractionated products (Table 17.1).[2] In low- and middle-income countries, and in military surgery, whole blood is often used.

Prestorage leucodepletion

Filtration reduces the risk of bloodborne infections such as HTLV-1/2 and CMV. Leucodepletion may also prevent alloimmunisation, febrile reactions and transfusion-related acute lung injury (TRALI).

Centrifugation and cryoprecipitation

Separates red cells, platelets, plasma and protein components.

Irradiation

Gamma or X-ray irradiation eliminates lymphocytes for patients at risk of transfusion-associated graft-versus-host disease (TA-GvHD).

Viral inactivation

Plasma-derived products (e.g. FFP and cryoprecipitate) to be used in those born after 1996 in the UK are sourced from outside the UK and have undergone viral inactivation with either solvent detergent or methylene blue.[2]

Table 17.1 Comparison of blood components

	Packed red blood cells (PRBCs)	Platelets	FFP	Cryoprecipitate
Derivation	Centrifuged from single-donor whole blood	Pooled buffy coat from centrifugation from 4–6 whole blood donations **or** by apheresis from a single donor	Apheresis donation from a ♂ donor. Rapidly frozen within 8h of donation	Controlled thawing of single-donor FFP. Pooled from 4–6 donors
Composition	Hct 50–70% suspended in SAGM*	>240 × 10^9 platelets	0.5g fibrinogen, factor VII >0.7IU/mL, labile factors	2g fibrinogen, vWF, factor VIII, factor XIII, fibronectin, factor VII >1.5IU/mL
Volume	300mL	300mL	300mL	Pooled 100–200mL
Storage	2–6°C	20–24°C on an agitator	–30°C	–30°C
Shelf-life	35d	7d	2y (frozen) 4h (thawed 2–6°C)	2y (frozen) 4h (thawed room temp)
Dose	4mL/kg will increase Hb by 10g/L	1 unit will increase platelet count by 20–40 × 10^9/L	10–15mL/kg	1 unit per 30kg body weight to increase plasma fibrinogen by 1.0g/L

* Sodium chloride 0.9%, adenine, glucose and mannitol.

Other derived products

- **Fibrinogen concentrate** is a freeze-dried powder containing 1g of purified human fibrinogen which has undergone pasteurisation and sterile filtration. Stored at room temperature for up to 30mo, it is reconstituted and raises a patient's fibrinogen by ~0.25g/L. No thawing or blood type matching is required. Indicated for use in hypofibrinogenaemia, cardiac surgery, obstetric bleeding and major trauma.[1,3]
- **Recombinant factor VIIa** is a freeze-dried powder used in patients with congenital factor VII deficiency, in bleeding episodes in patients with haemophilia A or B, with inhibitors to factors VIII or IX, and occasionally in those with severe uncontrolled bleeding as part of a massive transfusion. Dosing should be discussed with a haematologist (see ➲ p. 282).[1]

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- **Prothrombin complex concentrate (e.g. Prothrombinex®)** is a sterile, freeze-dried powder for reconstitution containing 500IU of purified human factors II, IX and X. It also contains small amounts of factors V and VII, 25IU of antithrombin III and 200IU of heparin. Used to replace congenital factor deficiencies when purified single-factor concentrates are unavailable, or for the reversal of vitamin K antagonist anticoagulants (e.g. warfarin). It does not need to be X-matched or thawed. Dosing is dependent on coagulation studies, desired clotting profile, patient weight and the factor deficiency being reversed.[1]
- **Individual factor concentrates** are available for congenital or acquired factor deficiencies.[1]
- **Immunoglobulin products**. Immunoglobulin concentrate contains 160mg/mL of human plasma proteins and is 98% immunoglobulin (mainly immunoglobulin G). Specific immunoglobulins are obtained from individuals with a high titre of the antibody required and include tetanus, anti-D and hepatitis B immunoglobulin. Mostly administered via a slow IM injection, except for IV immunoglobulin.[1]
- **Albumin** suspension concentrations vary internationally but are typically available in 4–5% (40–50g/L) and 20% (200g/L). They are pasteurised to reduce the transmission of viral diseases (see ➲ p. 467).

Safe transfusion

- **Informed consent**. Competent patients should be provided with information on risks, benefits and alternative therapies.
- Confirm patient identity, verbally with the patient if possible and by identification band.
- **Check unit to be transfused** against prescription. Check unit is within expiry date and that unit numbers match between the laboratory-generated label attached to the pack and the pack itself.
- **Inspect the bag**, ensuring integrity of the plastic casing. Look for discoloration or evidence of clumping.
- **Infuse** through a blood administration set with a 170–200 micrometres integral screen filter. Typically each filter can be used for 4 units of packed red cells during normal transfusion or 8–10 units during a massive transfusion, provided flow rates are adequate without evidence of clogging of the filter. Platelet concentrates should not be infused through giving sets that have been used for red cells due to risk of clumping. Infuse each unit within recommended time frames (Table 17.2).[1,2]
- **Monitor** for adverse events.
- **Single unit transfusion**. In stable, non-bleeding, normovolaemic patients, transfusion should be conducted 1 unit at a time.[2]
- **Documentation**. A 100% traceability of transfused blood is a legal requirement in the UK[2] and other countries.

Compatibility testing

Group and screen

A patient's blood sample is tested to determine ABO and RhD antigen type and to detect red cell antibodies. The sample validity period during which X-matched blood may be provided depends on the patient's transfusion and obstetric history and varies from 72h to 21d. Once transfusion has commenced, samples are valid for 72h.

Table 17.2 Time limits for infusions

Component	Start time	Completion time
Packed red cells	<30min	<4h
Platelets	Immediately	<1h
FFP	<30min	<4h
Cryoprecipitate	Immediately	<4h

Compatibility testing

This involves either a serological crossmatch to ensure compatibility or an electronic crossmatch on the basis of the antibody screen.

Transfusion compatibility

ABO compatibility

Normal individuals produce immunoglobulin M (IgM) antibodies against the A or B antigens which are not expressed on their cells. ABO compatibility prevents acute haemolytic transfusion reactions caused by recipient IgM antibodies binding A or B antigens on donor red cells.[1]

- **Red cells** express ABO antigens and recipients must be transfused with ABO-compatible units to prevent serious harm or death.[1]
- **Platelets** weakly express ABO antigens. Therefore, recipients should be transfused with ABO-compatible units. Non-ABO-compatible units of platelets can be used in the event of life-threatening haemorrhage, but these platelets will have a reduced lifespan *in vivo*.[1]
- **Cryoprecipitate and FFP** contain anti-B or anti-A IgM antibodies, depending on the donor blood group. For donated plasma to be compatible, it must not contain IgM antibodies against antigens expressed on recipient cells.[1]

(See Table 17.3.)

Table 17.3 ABO compatibility of components and recipients

Recipient	Compatible donor		
	Red cells	Platelets	FFP/cryoprecipitate
O	O	O or A	O, A, B or AB
A	A or O	A or O	A or AB
B	B or O	B or O	B or AB
AB	AB, A, B or O	AB, A, B or O	AB
Unknown	O	O	AB

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RhD compatibility

Immunoglobulin G antibodies against the RhD antigen (also known as anti-D) only form as a result of allogeneic exposure (e.g. transfusion or pregnancy). Compatibility testing prevents exposing RhD-negative recipients to the RhD antigen in transfused units.[1]

- **Red cells** express RhD antigens. This is of particular importance for all ♀ of reproductive potential to prevent future haemolytic disease of the newborn (due to maternal anti-D harming RhD-positive fetuses). RhD-positive blood may be provided for RhD-negative ♂ or ♀ beyond reproductive age if blood bank reserves of RhD-negative blood are depleted.[1]
- **Platelets** do not express RhD antigens. However, units should be RhD-compatible with the recipient as the RhD antigen is highly immunogenic and residual red cells in the units may sensitise RhD-negative patients.[1]
- **Cryoprecipitate and FFP** may contain red cell fragments; however, these are far less immunogenic than whole red cells. Therefore, cryoprecipitate and FFP of any RhD type can be safely given.[1]

Positive antibody screens

Patients with positive antibody screens may require specific antigen-negative blood components. Not all antibodies detected are of clinical significance, and discussions with blood bank and a transfusion specialist are warranted.

Uncrossmatched blood

Immediate dispensing of emergency O RhD-negative blood is reserved for haemorrhagic emergencies in the absence of a group and screen.[2] Obtaining blood samples for transfusion X-matching is a priority early in haemorrhage management. Group-specific blood (ABO/RhD) can be issued significantly quicker than fully grouped and screened blood (15min vs 45min). Although it has undergone limited testing, group-specific blood is an alternative to using O RhD-negative blood when transfusion is required urgently but not immediately.

Transfusion indications and triggers

Clinical judgement, lab results, POCT and best available evidence determine the blood components to be prescribed, as well as the timing, dose and rate of administration. Consideration should also be given to risks, benefits and available alternatives to transfusion.

Globally, red cell transfusion policies have become increasingly restrictive in response to emerging evidence of harm associated with unnecessary transfusion. FFP should not be given for prolonged PT or INR in the absence of bleeding. For transfusion triggers, see Table 17.4 (red cell),[2,4] Table 17.5 (cryoprecipitate),[2] Table 17.6 (platelet)[2,4] and Table 17.7 (FFP).[2]

Table 17.4 Haemoglobin transfusion triggers

Hb level	Red cell transfusion indication
<70g/L	Transfusion threshold for clinically stable ICU patients[5]
<80g/L	Patients with acute coronary syndrome, hip fractures or CVS disease[2,5]
70–100g/L	Intraoperative major blood loss or evidence of hypoxaemia
>90g/L	Transfusion usually inappropriate. Exceptions include impaired tissue O_2 delivery such as cerebral ischaemia or sepsis

Table 17.5 Cryoprecipitate transfusion triggers

	Cryoprecipitate transfusion indication
Massive transfusion	Given empirically; aim for fibrinogen >1.5g/L[2]
Obstetric haemorrhage	Given empirically; aim for fibrinogen >1.5g/L[2]
DIC	If clinical bleeding and consumptive coagulopathy. Aim for fibrinogen level >1.0g/L[2]

Functional fibrinogen assays may guide cryoprecipitate transfusion more accurately than absolute levels in the presence of dysfibrinogenaemia (see ➲ pp. 284–6).

Table 17.6 Platelet transfusion triggers

Platelet count	Platelet transfusion indication
$<10 \times 10^9$/L	Transfusion indicated as risk of spontaneous haemorrhage[2]
$<20 \times 10^9$/L	Transfusion indicated in the presence of risk factors for haemorrhage (minor bleeding, sepsis) or in critically ill patients[2]
$<50 \times 10^9$/L	Transfusion is indicated for most invasive surgery.[2] AoA now recommends $>50 \times 10^9$/L for performance of regional blockade[2]
$<75 \times 10^9$/L	Transfusion is indicated in massive haemorrhage[2] in order to consistently maintain platelet count at $>50 \times 10^9$/L
$<100 \times 10^9$/L	Transfusion is indicated for high-risk closed-compartment surgeries (ocular surgery, neurosurgery) [2]
Other	Regardless of platelet count, transfusion may be appropriate in the presence of pharmacological platelet inhibition or inherited disorders of platelet function (see ➲ pp. 277–9)
Contraindications/ cautions	TTP, HUS, HIT, immune thrombocytopenic purpura

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Table 17.7 FFP transfusion triggers

Platelet count	FFP transfusion indication
Coagulation factor/ protein deficiencies	Only if specific factors are unavailable.[2] Treatment of pseudocholinesterase deficiency
Massive transfusion	Empirically as 1:1 ratio with RBCs, or 15mL/kg per blood volume. Maintain INR <1.5[2]
Warfarin reversal	FFP used when PCC inappropriate or unavailable
TTP	For plasma exchange therapy[2]
DIC	Indicated if DIC is associated with bleeding.[2] Initial dose is 15mL/kg to maintain INR <1.5

Transfusion risks

Transfusion safety requires robust and reliable systems from the point of blood donation to the point of administration. Timely reporting of adverse events to blood banks is an essential step to aid systems improvement for the future. Adverse events include:[1]

Common

- **Febrile non-haemolytic transfusion reaction** (1–3:100) occurs within 30min of transfusion and is mediated by either cytokines or alloimmune reactions to contaminant leucocytes. Simple cooling and paracetamol are sufficient for mild reactions (<1.5°C rise in temperature). Mitigated by slowing infusions.
- **Minor allergy** (1–5:500) commonly presents with mucocutaneous manifestations, flushing, angio-oedema or urticaria due to recipient antibodies against leucocyte antigens or plasma proteins.
- **Hypotension** (1–2:1000). Idiosyncratic reaction, possibly related to bradykinin in transfused blood to recipients on ACE inhibitors. Defined as a 30mmHg drop in systolic BP.
- **Transfusion-associated circulatory overload** (1–10:1000). Incidence is determined by both the volume transfused and patient comorbidity.
- **Hypothermia**. Prevalent in rapid infusions of large volumes of blood products. Can worsen all physiological processes, including cardiovascular function and coagulation.
- **Immunosuppression**. Transfusion may influence the recurrence or spread of malignancies, as well as the incidence of postoperative bacterial infections through immunomodulation.

Rare

- **Acute haemolytic transfusion reaction** (1–8:100 000). Severe, life-threatening reaction occurring within 24h of red cell transfusion due to ABO incompatibility. Rarely may result from Kell, Duffy or Kidd antigen incompatibility. Recipient antibodies bind to and haemolyse transfused erythrocytes, causing complement activation, inflammation, DIC and shock. Signs and symptoms may be indistinguishable from bacterial sepsis or anaphylaxis.

- **Delayed haemolytic transfusion reaction** (1:5000). Usually occurs within 7d but may occur up to 28d after transfusion due to previous recipient allosensitisation to erythrocyte antigens during pregnancy or a previous transfusion. Results in jaundice, anaemia and rarely splenomegaly and renal injury due to haemoglobinaemia.
- **Bacterial sepsis** (<1:10 000). Rare, but the leading cause of transfusion-related mortality. Contamination may occur at any point from donor phlebotomy to recipient infusion. The highest risk is seen in platelet transfusions, which are stored at room temperature and are most prone to bacterial growth. Sepsis due to contaminated red cell transfusion has a 60% mortality. Inspection of blood components to detect clumping or discoloration may detect contamination.
- **Anaphylaxis** (<1:20 000). Recipient immune reactions can occur against any component (see ➔ pp. 1081–3 for anaphylaxis management).
- **Viral infection**. Screening of donors and donated blood for viral infections mitigates most risk. Residual risk results from individuals donating blood during 'window periods' of active viral infection. Overall the risk of contracting bloodborne viral infection from transfusions is 1:9.2 million for HIV, 1:6.9 million for hepatitis C and 1:0.8 million for hepatitis B.
- **Post-transfusion purpura** (<1:100 000). Most common in ♀ who have been previously pregnant, due to recipient antibodies against human platelet-specific antigen. Rare and potentially lethal due to thrombocytopenic haemorrhage.
- **TRALI** (<1:5000). Non-cardiogenic pulmonary oedema which occurs within 6h of transfusion of plasma or plasma-containing components. Clinically indistinguishable from ARDS. Severe microvascular injury results from recipient antibody reactions against transfused leucocyte or neutrophil antigens. Risk is reduced through use of ♂-only plasma components as multiparous women have higher titres of antihuman neutrophil antigen and antihuman leucocyte antigen (HLA), implicated in TRALI .[2]
- **TA-GvHD**. A usually fatal complication occurring 1–6w following transfusion. Transfused viable lymphocytes engraft within an immunocompromised host. These engrafted lymphocytes proliferate and precipitate multiorgan failure and death through autoimmunity.

Blood warmers

Warming systems are not required for routine transfusion. Indications include massive transfusion, neonatal exchange transfusions, rewarming during CPB and transfusions for patients with clinically significant cold reactive antibodies (cold haemagglutinin disease). Blood components must not be warmed >41°C and warmers should be fitted with visible thermometers and audible alarms to avoid haemolysis.[1]

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Patient blood management

Perioperative anaemia is an independent predictor of morbidity and mortality, and blood transfusion is a further independent predictor of poor clinical outcome.[6] Patient blood management is an evidence-based, multidisciplinary approach to reducing requirements for allogeneic blood transfusions and improving patient outcomes.[6,7] A patient's own blood is now recognised as a resource which should be conserved and managed to reduce transfusion rates. Patient blood management consists of three pillars: improving red cell mass, reducing blood loss and optimising physiological tolerance to anaemia.[6] (See also ➲ p. 53.)

Improving red cell mass

Anaemia is defined by the WHO as an insufficient circulating red cell mass with Hb <120g/L in ♀ and <130g/L in ♂.[8] Interventions to improve red cell mass increase the margin before transfusion thresholds are reached. Red cell mass is ↑ by:

- **Investigating anaemia** (see ➲ p. 54)
- **Iron replacement** (see ➲ p. 56)
- **B_{12} and folate replacement** in the presence of deficiencies
- **Haematopoietic growth factors**. Human recombinant EPO stimulates erythropoiesis from bone marrow progenitors[6] and is most effective in patients with renal failure who have reduced levels of endogenous EPO.

Reducing blood loss

- **History**: identify history of prior haemorrhage, menorrhagia, dental bleeding, easy bruising and inherited bleeding tendencies.[6]
- **Anticoagulation stewardship**: withhold anticoagulant/antiplatelet agents as appropriate (see ➲ pp. 269–75; ➲ pp. 278–9).
- **Surgical technique**: minimally invasive/percutaneous surgery, local vasoconstriction, topical haemostatic agents (e.g. fibrin glues), tourniquets and surgical devices (ultrasonic or laser scalpels).
- **Anaesthetic technique**: venous pressure can be reduced by avoiding high intrathoracic pressure, careful patient positioning and avoiding hypercapnia. Coagulation is optimised by maintaining normothermia and avoiding acidaemia and hypocalcaemia. Procedure-specific techniques: venodilation in liver surgery, TIVA in ENT surgeries and avoiding jugular venous obstruction in neurosurgery.
- **Tranexamic acid**: widely used antifibrinolytic with surgical applications in trauma, obstetrics, cardiac surgery and orthopaedics.[9,10] Administered IV and topically[10] (see ➲ p. 281; ➲ pp. 880–1; ➲ pp. 976–8).
- **Desmopressin**: synthetic analogue of ADH used in von Willebrand disease and to improve platelet function in uraemia, cirrhosis and other platelet function disorders. At high doses (>0.3 micrograms/kg), desmopressin induces the release of vWF and factor VIII from endothelial cells[1] (see ➲ p. 281). Caution must be exercised in decompensated cardiac failure and hyponatraemia due to antidiuresis.
- **Red cell salvage**: cell salvage should be considered for all patients where >500mL, or >10% of total blood volume, loss is predicted.[7] Autologous whole blood is collected via suction tubing into a reservoir during surgery using a filter. The blood is anticoagulated with citrate or heparin, centrifuged to isolate red cells and washed with 1–2L of 0.9% sodium chloride before being resuspended and reinfused.[11] Cell

salvage produces packed red cells suspended in 0.9% sodium chloride, with an Hct of 50–60%, without coagulation factors or platelets. Cell salvage devices can provide the equivalent of 10 units of bank blood per hour. Absolute contraindications to cell salvage include aspiration of substances that must not be given IV (e.g. chlorhexidine wash or fibrin glues).[11] Relative contraindications requiring risk–benefit assessment and consent from the patient include collection of blood containing septic material, malignant cells, and amniotic fluid.[11] Leucodepletion filters reduce the risk associated with salvaged blood in cancer surgery or from an infected surgical field. In cancer surgery, allogeneic blood transfusion may worsen outcomes, possibly by immunomodulation, so some centres advocate cell salvage to avoid this. In CS, the SALVO trial did not record any AFE following the routine use of cell salvage, but also did not demonstrate any reduction in allogeneic transfusion.[12] This has prompted guidelines to recommend cell salvage during CS only where there is high risk of obstetric haemorrhage.[11]

- **Autologous transfusion**: recipients donate and store their own blood for future requirements to eliminate the need for donor blood transfusion. Preoperative autologous donation involves blood donation once a week for 4w leading up to surgery. Acute normovolaemic haemodilution involves whole blood collection immediately preoperatively; normovolaemia is maintained with crystalloid volume expansion. Units are anticoagulated, stored and reinfused once surgical bleeding is encountered.[6]
- **Phlebotomy**: avoid unnecessary blood sampling, and sample minimum blood volumes for tests that are required.

Optimising physiological tolerance to anaemia

Anaemia reduces O_2 delivery to tissues by reducing oxyhaemoglobin concentrations. This can be partially compensated by optimising cardiorespiratory function and minimising the metabolic demands of tissues to match O_2 supply to demand.

- **Oxygenation**: ↑ FiO_2, hyperventilation.[6]
- **Tissue perfusion**: increase both CO and tissue perfusion with fluid therapy, vasopressors and inotropes.[6]
- **O_2 consumption**: reduced by treating pain and infection.[6] In severe life-threatening anaemia, hypothermia, hyperbaric oxygenation, sedation, muscle paralysis and mechanical ventilation can also reduce metabolic work.

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Massive transfusion

Introduction

More than 5 units of PRBCs in <4h or >10 units of PRBCs administered to a patient within 24h is considered a massive transfusion.

- Goals of therapy are to expand circulating volume rapidly, economically and without adverse effects.
- The current standard of care in the resuscitation of severe haemorrhage is administration of blood components (platelets, plasma and PRBCs) in a 1:1:1 ratio.[13]

Physiology of massive haemorrhage and transfusion[14]

- Rapid, large-volume haemorrhage results in systemic hypoperfusion and reduced O_2 delivery to tissues, causing acidosis.
- Acidosis, hypothermia and coagulopathy are known as the 'triad of death' in massive haemorrhage. Each individually causes worsening of the other two components of the triad.
- Hypoperfusion also activates the protein C pathway, deactivating factors Va and VIIIa and initiating fibrinolysis and coagulopathy.
- Consumption of clotting factors may lead to DIC.
- Administration of cold IV fluids and exposure of the patient for vascular access and resuscitation may lead to hypothermia.
- Hypothermia and acidosis reduce myocardial contractility, precipitate bradycardia and dysrhythmias and cause vasodilation and hypotension. They also reduce activity of clotting factors and platelets, with clotting factor function ↓ by 10% for every 0.1 reduction in pH.
- Transfused red cells are not as effective at O_2 delivery as endogenous red cells.

Resuscitation priorities during massive haemorrhage and transfusion[1]

(See also ➲ p. 971.)

Adequate staffing and assistance

- Call for additional senior surgical and anaesthetic help.
- Nominate a crisis leader to manage and assign roles to staff present.
- Notify blood bank and declare need for massive transfusion; this will result in more blood bank staff dedicated to X-matching and thawing blood products in readiness for use.
- A dedicated 'runner' to obtain blood products from the blood bank.
- Additional staff to check/administer blood products as they arrive.
- Additional theatre staff to set up key equipment.

Maintain circulating volume

- Obtain rapid source control of haemorrhage (e.g. surgical, endoscopic, interventional radiology).
- Immediately request 3 units of PRBCs; use O-negative emergency blood if X-matched blood not immediately available.
- Obtain large-bore IV access; give crystalloid until blood arrives.
- Increase FiO_2 to 100% to improve tissue O_2 delivery. Consider reducing dose of anaesthetic agent administered.

Request additional equipment

- Rapid-infusing device (e.g. Belmont™ or Level-1™)
- Deployment of cell salvage.

Prevent hypothermia

- Forced air warming devices (e.g. Bair Hugger™)
- Increasing the ambient room temperature
- Infusing via fluid warmers.

Prevent and treat coagulopathy

- Balanced transfusion of blood products in 1:1:1 ratio.
- POCT to identify factor deficiencies and detect DIC.
- ABG, formal FBC, coagulation studies and electrolytes sent to the lab and marked as urgent—should be performed every 30min.
- Consider dose of antifibrinolytic (tranexamic acid, 1g IV over 10min) if within 3h of traumatic event.
- Consider recombinant factor VIIa (NovoSeven®; 90 micrograms/kg IV) if surgical bleeding controlled and pH >7.2. Use with caution in patients at risk of thrombosis and discuss with haematologist.
- Discuss with haematologist in non-surgical uncontrolled bleeding (e.g. variceal bleeding) unresponsive to PRBCs/FFP/platelets, anticoagulated patients or patients with inherited bleeding disorders.

Treat sequelae of massive transfusion

Treat ↓ Ca^{2+} and ↑ K^{+} with slow IV injection of 10mL of calcium chloride 10% (see ➔ pp. 240–1 for further treatment of ↑ K^{+}).

Treatment goals[1]

Mean arterial pressure

- Do not aim to normalise until there is surgical control.
- >50mmHg (>70mmHg if head injury).

Haemoglobin

- >70g/L.

Fibrinogen

- >1.5g/L (>2g/L in obstetric patients).

Platelets

- >75 × 10^{9}/L.

Ionised calcium

- >1mmol/L on blood gas sample.

pH

- 7.35–7.45.

PT/APTT

- <1.5 times upper limit of normal/baseline.

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Jehovah's Witnesses

There are over 8 million members of the Jehovah's Witness movement worldwide.[15,16,17] Most Jehovah's Witnesses refuse receipt of 'primary blood components' (whole blood, including autologous preoperative donation, PRBCs, platelets, white cells and FFP). The acceptance of 'blood fractions' is variable and up to each individual's interpretation of the church's teachings. Blood fractions, or derivatives, include cryoprecipitate, clotting factor concentrates, albumin and immunoglobulin. So long as lines are primed with non-blood fluids, intraoperative cell salvage is often acceptable, as are apheresis, haemodialysis, CPB and normovolaemic haemodilution. IV iron and tranexamic acid are pharmacological agents that present no quandary and may reduce postoperative anaemia. Determining and documenting precisely which products, components and techniques a Jehovah's Witness patient is willing to accept are therefore of critical importance when planning for surgery.[16,17]

Ethical and legal considerations

Competent patients

Competent patients have the right to autonomously decline transfusion. For this to be informed consent, an anaesthetist is obliged to inform patients of the risks, benefits and alternatives to their decision, including the probable outcomes of doing nothing. Clinicians must also be satisfied that a patient's decision is free from coercion,[16] which may require consenting a patient individually, away from family or associates. Administration of blood products to a competent patient who has refused blood transfusion is unlawful and may lead to professional and criminal proceedings against the doctor.[17]

Advance directives

Advance directives state a patient's acceptance or otherwise of specific medical interventions for situations in which they would be unable to provide consent. They must contain clear and medically interpretable instructions about the treatments which are acceptable or unacceptable. They must also be clear that these directives are to apply even if a patient's life is at risk.[17] Advance directives may be issued by a church; however, these often lack sufficient medical detail.[16] Many hospitals will therefore have checklist-styled forms to clearly document a patient's consent.

Emergency surgery

Conscious and competent patients

Conscious and competent patients must be provided the opportunity for informed consent and have their wishes regarding blood transfusion acceptance—or refusal—respected.[16,17]

Unconscious or incompetent patients

Unconscious or incompetent Jehovah's Witness patients present uncertainty. In emergency situations where a patient's wishes are unknown, doctors must act in the best interests of the incompetent patient, which may include providing blood transfusion.[17] Opinions of relatives or associates that the patient would not accept a blood transfusion are insufficient, and advance directives should be sought and verified. If the proposed treatment

is included in a verified advance directive, the patient's wishes must be respected unless there is evidence that the directive no longer represents the patient's wishes. [17] Inclusion of senior departmental and legal support staff is advisable.

Children

Children of Jehovah's Witnesses may present particular difficulty. If the parents refuse permission for blood transfusion, it may be necessary to apply for a legal guardianship in order to administer a blood transfusion. In an emergency situation, when the child of Jehovah's Witnesses is likely to die without blood transfusion, blood should be given.[17] Courts are likely to uphold this medical decision.[16]

Elective surgery

- **Early assessment**: meet the patient as early as possible to maximise available time for communication, optimisation and planning.
- **Patient blood management** (see ➔ pp. 458–9): optimise red cell mass preoperatively; minimise bleeding and optimise physiological tolerance to anaemia. Patients should be assessed >6w before major surgery to ensure Hb >130g/L.[17]
- **Multidisciplinary input** to produce a comprehensive management strategy.
- **Clearly communicate** with the patient at each stage of planning. Support people (e.g. family members, church members or the Jehovah's Witness hospital liaison) may assist communication and the understanding of both parties.[17] Be mindful that patient consent must be free from coercion.[16]
- **Document** all meetings, discussions, correspondence, consents and advance directives.
- **Surgical planning**: decide when, where, how and by whom the patient will be operated upon. Consider booking surgery early in the week and 1st on the list to allow for expedient management of complications if they occur. Plan for a senior surgical team, minimally invasive surgery and staged procedures if appropriate. The role of interventional radiologists for pre-embolisation or placement of arterial balloon catheters should also be considered.
- **Autologous transfusion**: strongly consider red cell salvage—the majority of Jehovah's Witnesses will accept this. Acute normovolaemic haemodilution may also be acceptable.[17]
- **Intraoperative**: issues need to be highlighted at team briefing and during the surgical safety checklist.[17]
- **Postoperative**: anticipate requirement for critical care, monitoring and resources required for expedient intervention for haemorrhagic complications. Ensure thorough handover, including the wishes of the patient.[17]

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Fluid therapy

Routine fasting for minor surgery is usually well tolerated by healthy patients with minimal disturbance to fluid status or electrolyte levels.[18] The magnitude of disturbance to fluid and electrolyte homeostasis is dependent on fasting times, the extent of surgery, age, patient comorbidity and the nature and severity of acute pathology present. Disease, anaesthesia, trauma and surgery precipitate a combined metabolic, neuroendocrine, immunological and inflammatory stress response which readily disrupts fluid homeostasis (Table 17.8). Concepts such as liberal, restrictive and goal-directed fluid therapy have all gained popularity within the last two decades at various times. Clarity around the optimal strategy for a given patient, with a given pathology and a given collection of comorbidities, undergoing a given surgery remains elusive, however.

Normal fluid and electrolyte balance

Under normal homeostatic conditions, net input and output of fluid and electrolytes are equal. Mean 24h water intake, around 2500mL, roughly constitutes 1500mL of fluid, 750mL of food and 250mL of metabolic water. Output is balanced with 1500mL as urine, 100mL in faeces and 900mL as insensible loss. Daily requirements of electrolytes are listed in Table 17.8.

Table 17.8 Daily requirements of common electrolytes

	Plasma concentration (mmol/L)	Daily requirement (mmol/kg/d)
Na^+	135–145	1–1.5
K^+	3.5–5.0	1–1.5
Mg^{2+}	0.75–1.05	0.1–0.2
Ca^{2+}	2.12–2.65 (total), 1.0 (ionised)	0.1–0.2
Cl^-	95–105	0.07–0.22
Phosphate	0.8–1.45	20–40

Fluid compartments

Water constitutes 60% total body weight (600mL/kg). TBW comprises:

- Intracellular fluid: 66% total body weight (400mL/kg)
- ECF: 33% total body weight (200mL/kg)
  - Interstitial fluid: 75% ECF (150mL/kg)
  - Plasma: 25% ECF (50mL/kg).

The glycocalyx

- **Structure**: membrane-bound, carbohydrate-rich proteoglycan and glycoprotein layer on the luminal surface of vascular endothelium.
- **Function**: vascular permeability, coagulation, inflammation, antioxidation and regulating transluminal oncotic pressures.[19] A non-circulating volume of 700–1000mL of plasma volume is fixed within the endothelial glycocalyx. Theoretically, isotonic crystalloid will distribute through this, while colloid fluids will not.

- **Pathology**: the glycocalyx is readily disturbed by trauma, metabolic/osmotic insults, inflammation, hypovolaemia, hypervolaemia and artificial colloids. Collectively, this is called shock-induced endotheliopathy and results in fluid movement from the intravascular to the interstitial compartments.[19]

Assessment of fluid status

Accurate assessment of fluid status is difficult, but history, examination and bedside tests are usually sufficient to assess potential fluid responsiveness. This clinical assessment is then supported by available investigations.

- **History**: fasting times, self-reported oral intake/GI losses, blood loss, timing since last dialysis, etc.
- **Medications**: diuretics, antihypertensives.
- **Fluid balance chart**: assess trends between enteral and parenteral fluid intake, balanced against GI losses, urine output and surgical drains, etc.
- **Vital signs**: trends in BP, HR, RR, GCS and urine output.
- **Clinical examination** (Table 17.9): possibly the least predictive tool when assessing fluid responsiveness.[20]
- **Passive leg raise**: increases preload in the volume-deplete patient to augment stroke volume and BP. This is a simple, highly predictive means of assessing the utility of fluid therapy in a hypotensive patient. If negative, a patient is highly unlikely to be fluid-responsive.[20]
- **SVV/PPV**: cyclical changes in intrathoracic pressure from positive pressure ventilation induce changes in LV preload. This variation in preload, as seen in SVV and PPV, is predictive of fluid responsiveness.
- **CVP**: low CVP <8mmHg is associated with fluid responsiveness.[20] CVP monitoring is unlikely to be useful in haemodynamically stable patients.[21]
- **Haemodynamic monitoring**: PPV, oesophageal Doppler, PICCO™, LiDCO™ rapid, PA catheters and Cheetah NICOM®, especially with dynamic fluid challenges, are probably the most reliable method of measuring and replacing lost fluid.
- **CXR**: provides evidence of cardiomegaly, pulmonary congestion and pulmonary oedema due to fluid overload.
- **Laboratory investigations**: tissue hypoperfusion will manifest in acidosis, acidaemia, lactataemia and reduced base excess. Elevated creatinine/urea/Hct, reduced CC and low urine acidity/Na^+ concentration indicate dehydration.
- **Echocardiography**: quantifies ventricular function, as well as preload, via vena caval and ventricular filling. Respiratory variation in vena cava diameter >15% is associated with fluid responsiveness.[20]

Table 17.9 Signs and symptoms of dehydration

Body weight loss (%)	Signs and symptoms
5	Thirst, dry mouth
5–10	↓ peripheral perfusion, ↓ skin turgor, postural dizziness, oliguria, ↓ CVP, lassitude, tachycardia
10–15	↑ RR, hypotension, anuria, delirium, coma
>15	Life-threatening

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Dynamic fluid challenges

This is a real-world approach to fluid management, commonly employed in the theatre environment.

- Use boluses of 100–200mL of crystalloid.[22] Assess clinical response to boluses. Haemodynamic endpoints may include improvements in urine output, HR, stroke volume, BP, CVP, PPV, SVV, lactate, pH, etc.
- Remain vigilant for alternative causes for haemodynamic compromise. Hypoperfusion may be due to hypovolaemic or cardiogenic causes, or distributive or obstructive processes.
- Assess cumulative blood loss and requirement for transfusion.
- Repeat boluses, with frequent reassessment.

Crystalloids

(See Table 17.10.)

Balanced salt solutions

Examples: Hartmann's solution, Plasma-Lyte 148®:

- First-line replacement therapy in the perioperative period.
- Isotonic balanced salt solutions are the 1° maintenance fluid for all major surgery at 1–3mL/kg/h.
- Physiological composition, used to replace ECF.
- Organic anions offset chloride concentrations to maintain electroneutrality. May reduce iatrogenic hyperchloraemic metabolic acidosis and associated renal dysfunction, associated with infusions of solutions with higher chloride concentrations.[23,24,25,26,27]
- The addition of K^+ and Ca^{2+} may limit usefulness in ↑ K^+ or with citrated blood transfusions.

Sodium chloride 0.9%

- Commonly used for electrolyte replacement and useful for replacing electrolyte-rich GI losses.
- Infusion of 0.9% sodium chloride solutions is associated with hyperchloraemic acidosis which may induce renal hypoperfusion and inflammation.[26] This may contribute to AKI in both critically unwell and non-critically unwell patients.[23,24,25,26,27]

Glucose solutions

Examples: Glucose 5%, glucose 4%–sodium chloride 0.18%:

- Glucose 5% is a convenient way of giving free water, used to restore dehydration associated with water loss.
- ↓ Na^+ may occur with excessive use; therefore, these have little role in routine daily fluid management in adults.
- **No role** as plasma expanders due to deleterious osmotic effects.
- Glucose solutions are not an effective energy source for metabolism, but glucose is available in 10%, 20% and 50% solutions to promote normoglycaemia.

Colloids

(See Table 17.10.)

Colloids are homogeneous, non-crystalline substances, consisting of large molecules which persist in the vascular compartment to expand the functional plasma volume (lasting several hours to several days). Duration of action is determined by physicochemical properties, integrity of the glycocalyx/capillary membrane and pharmacokinetics of metabolism and clearance.

Human albumin solution

- Molecular weight (MW) 69 000.
- Available as a 4–5% solution for the treatment of hypovolaemia, and as a salt-poor 20% solution for the treatment of hypoalbuminaemia.
- Like other blood products, HAS is manufactured from fractionation of whole blood. Concern of theoretical transmission of variant Creutzfeldt–Jakob disease (vCJD) in the UK has resulted in this product currently being imported from the US, and it is thus expensive.
- Albumin has not been shown to improve survival in either all-comer admissions to intensive care[28] or in the treatment of severe sepsis.[29]
- Used in the treatment of hypovolaemic shock, although post hoc analysis of the SAFE trial demonstrated an association with ↑ mortality when albumin was used, rather than 0.9% sodium chloride, in the resuscitation of critically ill patients with traumatic brain injuries.[30]
- Also used as a priming fluid in CPB and as a replacement solution in plasma exchange.

Table 17.10 Composition of common intravenous fluids

	Na^+ (mmol/L)	K^+ (mmol/L)	Ca^{2+} (mmol/L)	Chloride (mmol/L)	Other	pH	mOsmol/L	Cost (£/L)
Sodium chloride 0.9%	154			154		5	308	1
Glucose 4%–sodium chloride 0.18%	30			30	Glucose 40g	4	263	1
Glucose 5%					Glucose 50g	4	278	1
Hartmann's solution	131	5	2	111	Lactate 29mmol/L	6.5	278	2
Gelofusine®	154	<0.4	<0.4	120	Gelatin 40g	7.4	274	10
Albumin 4–5%	<160	<2		136	Albumin 40–50g	7.4		95

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Gelatins

Examples: Gelofusine® 4%, Physiogel®, Gelafundin® 4%:

- Succinylated gelatins (MW 30 000), presented in sodium chloride solution.
- Manufactured from bovine collagen from bovine spongiform encephalopathy-free herds. There have been no reports of vCJD.
- Initially these have a powerful osmotic effect. Administration may rarely lead to histamine release, causing bronchospasm, urticarial rash, hypotension and tachycardia. May trigger anaphylaxis.
- Therapeutic maximal dose is limited by haemodilution.

Hydroxyethyl starches

- Starch solutions have been associated with considerable side effects, including accumulation, pruritus, renal impairment,[31] coagulopathy, haemorrhage, anaphylaxis and death.
- Hydroxyethyl starch products were withdrawn in the UK in 2013 because of safety concerns over their use as resuscitation fluids in critically ill patients and patients with pre-existing renal dysfunction.

Liberal vs restrictive fluids

Over the last two decades, styles of administering fluids have varied between liberal strategies (designed to replace '3rd space' fluid shifts and 'insensible' losses from surgery) and restrictive strategies, aiming to achieve 'zero balance', or zero weight gain due to postoperative fluid retention.[32] Many ERAS pathways incorporate restrictive strategies. However, both liberal and restrictive strategies have been associated with problems.

- From this point of uncertainty, the RELIEF trial by Myles *et al*. in 2018 has offered some guidance to clinicians.[33] This multicentre trial of 3000 patients undergoing major abdominal surgery had a restrictive arm and a liberal arm during and up to 24h after surgery. AKI and renal replacement therapy rates were lower in the liberal arm, although disability-free survival was the same in both groups.
- There is now support for 'moderately liberal' fluid regimens for major abdominal surgery requiring 10–12mL/kg/h of IV fluid administered intraoperatively and 1.5mL/kg/h in the following 24h post-surgery. Other major surgeries which do not result in major fluid shifts are unlikely to require as much fluid.[33,34]

Goal-directed fluid therapy

GDFT uses a combination of fluids and inotropic agents administered in a predetermined algorithm to optimise blood flow to organs.

IV fluids and inotropes are sequentially administered to achieve specific haemodynamic endpoints (CVP, MAP, stroke volume, CO, cardiac index, etc.). Although appealing, GDFT has not resulted in significant benefits for patients. Clinical benefits seen in small trials have not been corroborated in later literature. Nonetheless, GDFT remains in many ERAS protocols for abdominal surgery worldwide.

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Chapter 18

Specific circumstances

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Oncological impact of anaesthesia

The perioperative period

The perioperative period is a crucial phase in the surgical treatment of cancer, and events occurring during this time may affect patients' oncological outcomes.

Surgical tissue trauma and manipulation have been shown to produce both local and haematogenous spread of tumour cells from the 1° tumour. This and occasions where resection has been incomplete result in the persistence of viable malignant cells throughout the perioperative period—so-termed 'minimal residual disease'.

The surgical stress response creates a protumorigenic microenvironment within the body via a variety of mechanisms, including:

- **Release of growth and angiogenic factors**: prostaglandin E2, platelet-derived growth factor (PDGF), insulin growth factor (IGF), hypoxia-inducible factor 1α, vascular endothelial growth factor (VEGF)
- **Release of factors that promote tumour cell invasiveness**: such as matrix metalloproteinases
- **Induction of intracellular mitogenic signalling pathways**, promoting cellular proliferation and reducing apoptosis
- **Immunosuppression**, especially of cell-mediated immunity with a reduction in both the volume and activity of natural killer (NK), CD4 and CD8 cells. This part of the immune system is thought to be particularly vital in the detection and destruction of micrometastases.

The perioperative period therefore may comprise a 'perfect storm' of circumstances that favour the early recurrence/metastasis of cancer. There is evidence from both animal and human studies for this effect.

Rationale for the role of anaesthesia in influencing oncological outcome

There is a large body of evidence demonstrating how various anaesthetic techniques/perioperative interventions may modify different elements of the surgical stress response.

It is postulated that by altering both the nature and magnitude of the stress response, different anaesthetic techniques may produce different postoperative oncological outcomes.

Current controversies and the corresponding evidence

Volatile anaesthesia vs TIVA

Volatile anaesthetic agents have been shown to induce the expression of tumorigenic growth factors (including hypoxia-inducible factor 1α and IGF) and produce immunosuppression, principally via inhibition of NK cells and cytotoxic T-lymphocytes. Conversely, propofol has been shown to reduce the expression of hypoxia-inducible factor 1α and have much less immunosuppressive effects. This has led to a body of opinion that propofol-based TIVA may be preferential to volatiles in oncological surgery and may even be associated with better oncological outcomes. While there are a number of retrospective cohort studies that support this hypothesis, there are no positive prospective studies to date. A recent prospective RCT found no benefit of propofol TIVA with regional anaesthesia vs volatile anaesthesia

and opioids in breast cancer patients. The definitive case for the benefit of propofol over volatile anaesthesia in cancer surgery is yet to be made; however, it should be noted that no evidence exists showing propofol is worse.

Opioid analgesia

Opioid analgesics mediate their effect principally via the mu-opioid receptor. This receptor is known to be overexpressed on many cancer cell types and is postulated to play a role in tumour growth and development. High levels of mu-opioid receptor expression have been correlated with worse oncological outcomes in clinical studies. It has therefore been postulated that perioperative opioids may be tumorigenic via their action on, and induction of, the mu-opioid receptor. The clinical evidence to date does not support this and it remains a theoretical risk.

Regional anaesthesia

Following on from the theoretical risk of opioid analgesics, it has been postulated that, by minimising surgical stress and reducing opioid usage, regional anaesthesia may be beneficial in oncological surgery. Current evidence is mixed and the use of regional anaesthesia for this purpose alone cannot be recommended.

NSAIDs

Via inhibition of COX, NSAIDs reduce the expression of a number of growth factors, including prostaglandin E2, which has been implicated in a variety of tumorigenic processes. It is postulated that perioperative NSAID use may therefore reduce the risk of tumour recurrence/metastasis. Clinical evidence of a significant effect following perioperative NSAID administration is lacking.

Dexamethasone

Steroids are known to be immunosuppressive and therefore, it has been questioned whether the use of dexamethasone perioperatively is appropriate in cancer surgery. There is no clinical evidence to support this hypothesis at the time of writing.

Recommendations for practice

While there is biological plausibility for some of the interventions above being recommended/avoided in cancer surgery, definitive evidence—and therefore evidence-based guidance for practice—is still some way off.

The best approach has to be a pragmatic one, taking into account the patient's needs and preferences and the options available. The overall guiding principle must be to adopt a strategy that minimises the physiological insult and stress of surgery and produces a rapid, high-quality recovery and return to premorbid function, allowing the patient to move on to the next phase of their cancer treatment in the optimum time frame. Initiatives such as ERAS, or other fast-track recovery programmes, utilising many of the interventions described above, produce good short-term postoperative recovery and may also be beneficial for oncological outcomes.

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Oncological considerations

General considerations

Cardiac injury

May be induced by drugs (anthracyclines, fluorouracil, trastuzumab) or the stress of chemotherapy on a compromised heart. Anthracycline-induced cardiac failure may be irreversible and has a mortality of above 30%.

Bleomycin Exposure as part of chemotherapy conveys the potential risk of rapidly progressive pulmonary toxicity. Pulmonary toxicity occurs in 10% of patients exposed to bleomycin, with acute, followed by chronic, fibrosing alveolitis. Avoidance of high inspired O_2 concentrations and careful fluid management are recommended. Bleomycin is notably administered in germ cell tumours and Hodgkin lymphoma.

Cytokine release syndrome An acute systemic inflammatory response to immunotherapies, including chimeric antigen receptor T-cell therapy, heralded by fever. Associated features may range from mild flu-like symptoms to severe life-threatening circulatory collapse.

Hepatic veno-occlusive disease A progressive obliteration of venous channels in the liver.

Tumour lysis syndrome Can follow initial chemotherapy (typically for lymphoma and high-count leukaemias). Mass cell death leads to acute renal impairment, with hyperkalaemia, hyperuricaemia, hyperphosphataemia and hypocalcaemia.

Mediastinal masses (Particularly in leukaemia or lymphoma patients) can cause complete airway collapse under anaesthesia, even in the asymptomatic. Warning signs include stridor, wheeze, orthopnoea and SVC obstruction.

SVC obstruction Can arise from compression by a tumour or lymph nodes (usually bronchogenic carcinoma) or direct vessel invasion. Pleural effusions and ascites are common in ovarian cancer, metastatic disease and mesothelioma.

Paraneoplastic syndromes

Occur in 10% of cancer patients[1] (especially lung, lymphoma, breast, prostate, ovarian and pancreatic tumours). Anaesthetic considerations:

- Lambert-Eaton myasthenic syndrome is common in small-cell lung cancer and breast, thymus and GI tract tumours (see ➲ p. 316).
- Cushing's syndrome may occur in tumours of the lung, pancreas, thymus and ovary (see ➲ p. 232).
- Hypercalcaemia may be caused by bony metastases or PTH-like compounds.
- Hyponatraemia and SIADH-like syndromes may be caused by small-cell lung cancer and also lymphoma, leukaemia and pancreatic/carcinoid tumours (see ➲ p. 242).
- Cachexia can be caused by vomiting, loss of appetite or other GI disturbances. Hypoalbuminaemia (<35g/L) is a risk factor for poor outcomes.[2]

Radiotherapy May cause fever and nausea/vomiting, and patients may be dehydrated. Previous radiotherapy causes ongoing localised fibrosis, which may impede laryngoscopy and airway management.

Chemotherapy Commonly causes immunosuppression and myelosuppression.

VTE Affects at least 15% of cancer patients.

Do Not Attempt Resuscitation (DNAR) orders May be present in cancer patients. Where these conflict with safe anaesthetic principles, it is reasonable to modify or suspend the order perioperatively (see ➲ p. 42).

Anaesthesia for oncological procedures

Venous access port insertion (e.g. Portacath)

Commonly targets the left subclavian vein as the 1st-choice access vessel, and makes use of a totally implantable venous access device. Line placement can be under GA or LA ± sedation, dependent on local practices and patient choice. Following insertion, a chest radiograph is required to confirm placement and exclude complications, including pneumothorax and haemothorax. PICC lines are frequently used in oncology patients. (See also ➲ p. 360.)

Isolated limb perfusion Used in some centres for unresectable cases of cutaneous melanoma and sarcoma. It involves obtaining access to an extremity artery and vein, an extracorporeal circuit and a pneumatic tourniquet to isolate the target limb. It enables significantly higher concentrations of cytotoxic agents (e.g. melphalan and TNF-α) to be administered while limiting systemic side effects. Consequently, careful monitoring for evidence of systemic leakage is required, and appropriate invasive monitoring and central venous access is advised.

Brachytherapy Places a radioactive source close to the tumour via an applicator. It is often used in patients unfit for surgery and may involve single or multiple treatments. Procedures usually last 1.5–3h but may be longer and may include a transfer to CT. Blood loss is usually minimal, but postoperative pain may be an issue. Postoperative radioactivity can require patients to be recovered in an isolated environment. Anaesthetic options are:

- Light GA
- Sedation (although this may not produce reliable immobility)
- Epidural or spinal anaesthesia (some procedures may outlast spinal block, requiring the insertion of catheters). (See ➲ p. 729.)

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General principles of laser surgery

Laser is an acronym for Light Amplification by Stimulated Emission of Radiation. The laser is a high-energy light beam capable of delivering a large amount of energy to very small areas. ⚠ Lasers have numerous medical and surgical applications, but also create unique hazards to patients and staff.

- Light is a form of radiant energy that spans the mid-range of the electromagnetic spectrum. Laser light is generated by applying energy to a 'lasing medium' to cause photon release. Light is generated when electrons move from higher- to lower-energy shells surrounding the nucleus of an atom.
- Because the emitting atoms in a lasing medium are all identical, the photons released when the electrons change shells are also identical—the photons are all of the same wavelength (colour); they are in-phase (the peaks and troughs of the wave are synchronised, amplifying, rather than cancelling out, each other), and they have the same polarisation (wave orientation). This explains why laser light is of much higher intensity and energy than normal light.
- Mirrors direct escaping photons back into the lasing medium to generate the release of yet more photons. The laser beam escapes through a narrow aperture in the mirror.
- Fibreoptic bundles are used to transmit visible and near-infrared wavelength lasers. Lasers of longer wavelength, e.g. the CO_2 laser, can only be directed by a series of mirrors.
- The type of atoms in the lasing medium determines the laser wavelength, which in turn determines the tissue penetration and clinical applications. Each substance has a particular absorption spectrum, which is determined by its chemical structure. Laser light at, or close to, these frequencies will be the most effective (Table 18.1).
- Laser light striking a tissue surface may be:
  - **Reflected**: reflection off shiny surfaces may damage the eyes of staff in the vicinity.
  - **Transmitted** to deeper layers: lasers pass through tissues to a variable depth, which is partially determined by the wavelength.
  - **Scattered**: shorter wavelengths induce greater scattering.
  - **Absorbed**: this produces the clinical effect when the absorbed light is converted to heat. Organic tissue contains various substances capable of absorbing light. These are termed chromophores and include Hb, collagen and melanin. Each substance has a particular absorption spectrum, which is determined by its chemical structure.

Table 18.1 Types of surgical laser in common use

Laser type	Wavelength (nm)	Colour
Dye laser	360–670	Blue to red
Argon	488–515	Blue/green
Helium–neon	633	Red
Ruby	694	Red
Nd–YAG	1064	Near-infrared
CO_2	10 600	Far-infrared

Examples of medical lasers

Pulsed dye laser

This uses light at a wavelength that targets RBCs within blood vessels. The energy is dissipated within the dermis and causes only minimal epidermal scarring. This is used mainly for treating port wine skin lesions. Children requiring laser therapy to these lesions will often be subjected to multiple treatments, usually under GA. Postoperative pain may be a problem, particularly if large areas are treated. Combinations of paracetamol and NSAIDs may be effective, but occasionally opioid analgesics are required.

Carbon dioxide laser

These lasers have a long wavelength (10 600nm, outside the visible spectrum) and are preferentially absorbed by water. Target cells are heated to the point of vaporisation by the beam. They penetrate to only a very shallow depth, so tissue damage can be directly observed. They are used in aesthetic facial surgery to reduce the wrinkling associated with ageing, and in ENT practice to vaporise vocal cord and airway lesions. Care must be taken to avoid eye and airway injury (see ➲ pp. 478–9).

Nd–YAG laser

This laser is also outside the visible range and, unlike the CO_2 laser, is transmitted through clear fluids and absorbed by dark matter. It can penetrate to a depth of 1cm. It has multiple applications, including airway neoplasms, vascular malformations and ophthalmic surgery.

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Safety in laser surgery

The main hazards from laser surgery are inadvertent tissue damage to either the patient or to staff, risk of fire and inhalation of laser plume particles.

- ⚠ A designated laser safety officer should be present at all times when a laser is in use. In the UK, anyone in theatre where laser is being used should have had the appropriate training. Theatre doors should be locked from the inside, and signs displayed outside. A checklist should be completed prior to turning the laser on.
- Medical instruments used with lasers should have matt (rather than shiny) surfaces to reduce reflection of laser beams around the theatre.
- ⚠ Eyes are very susceptible to injury. Retinal and corneal damage can occur, depending on the frequency of the beam. All operating room personnel must wear safety glasses appropriate for the laser in use. These should have side shields to protect the lateral aspect of the eye. If an anaesthetised patient is receiving laser radiation near the eyes, protective matt metallic eye covers should be applied.
- Some lasers may damage exposed skin, so anaesthetised patients must have all exposed skin covered with moist swabs. In all cases, the eyes should be taped closed and covered with moist swabs. Many types of surgical drapes and anaesthetic tapes are combustible.
- Some skin preparation fluids are flammable and should not be used during laser surgery.
- ⚠ Laser light can ignite plastic and rubber materials. Carefully consider the optimum method of airway maintenance if lasers are employed within the airway. The simplest method is to use a laser-safe ETT. HFNO or jet ventilation may also be employed (Fig. 18.1). A low FiO_2, e.g. 30%, reduces the risk of fire but also decreases apnoea time.
- IV anaesthesia is usually employed to ensure an adequate depth of anaesthesia. It is also important to prevent the patient from moving or coughing, so a suitable muscle relaxant should be administered, and NMB monitored with a nerve stimulator.
- If the use of an ETT is required, unmodified conventional plastic tubes cannot be used, because they support combustion and can potentially cause airway fires. Various laser-safe ETTs are available. The cuffs of these tubes are vulnerable and can be protected by damp pledgets. The cuff should be filled with 0.9% sodium chloride, which can be mixed with methylthioninium chloride (methylene blue) so that a cuff puncture is obvious.
- Both N_2O and O_2 readily support combustion. If using a circuit, 30% O_2 with air is a sensible choice.
- If a fire occurs in an airway during laser surgery, switch off the laser immediately, remove the tube and flood the airway with 0.9% sodium chloride. Once the fire is extinguished, mask ventilation with 100% O_2 should be initiated. Reintubation and bronchoscopy with lavage may be required. Severe damage may require a tracheostomy.
- ⚠ A laser plume is created at the site of contact with human tissue. This contains fine particulates which are potentially hazardous to health workers. Smoke evacuation systems must be used to remove laser plume contaminants. Aerosolisation of viruses can occur during laser surgery for papillomata—special masks are worn to reduce the risk.

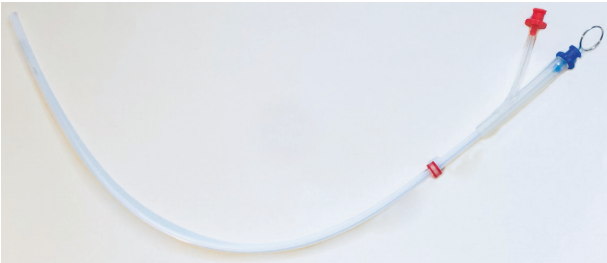

Fig. 18.1 A LaserJet ETT, with a side port for CO_2 sampling. Courtesy Aidan O'Donnell.

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Robotic surgery

The number of procedures undertaken robotically around the world continues to grow, with an increasing variety of surgery now possible with robotic systems (Table 18.2).

- Advantages for the surgeon include ↑ comfort while operating, 3D video, filtering out of any tremor and scaling of movement to allow for precision work.
- Patient-based advantages include a better cosmetic result from minimally invasive approaches, reduced pain from port sites and reduced length of stay.
- The effect of robotic surgery on outcomes is controversial and procedure-specific. While some papers suggest better oncological outcomes from robotic procedures, there have been warnings about poorer survival from other robotic procedures for cancer.
- The ↑ cost and length of time it takes to complete robotic surgery are an important consideration for institutions, particularly at the establishment of any robotic programme.
- The considerations for the anaesthetist are largely due to positioning, length of procedure and the significantly reduced access to the patient caused by most robotic systems. The docking and undocking of a robot often takes significant time and rapid removal is rarely possible.

Table 18.2 Examples of robotic surgery procedures

Specialty	Procedure	Considerations
Breast	Mastectomy	Supine, axillary approach
Cardiac	Mitral valve replacement	Bleeding from remote port sites
Colorectal	Large bowel resections	Steep head-down, bowel preparation
ENT	Thyroidectomy Base of tongue tumours	Axillary or transoral approach
Urological	Prostatectomy	Steep head-down
Orthopaedic	Arthroplasty	

Preoperative

It is important to clarify with the surgeon how they will need the patient positioned as it is likely to be much more extreme or different from a non-robotic procedure. A high-quality team brief is critical to the smooth running of a robotic theatre.

- The docking of the robot is one of the rate-limiting steps of robotic surgery and so it is useful to ensure a consistent theatre team to reduce the delay this incurs to a minimum.
- The preassessment of the patient should be as normal. However, for procedures where prolonged steep head-down is anticipated, a note should be made of significant glaucoma as this may lead to an ↑ incidence of ophthalmic complications, including visual loss.

- In robotic laparoscopic surgery, consideration should be made whether the patient will tolerate a prolonged pneumoperitoneum for robot-assisted surgery.
- Patients due for intra-abdominal surgery, for which an intracorporeal anastomosis is planned, may have received bowel prep and this should be enquired about in order to plan fluid therapy.

Perioperative

The overwhelming number of robotic procedures will be done under GA in order to ensure a fixed reference point for the robot and due to the length of most procedures.

- Due to limited access to the patient during surgery, the default airway choice is an ETT. This should be taped (rather than tied) to facilitate venous drainage of the head.
- ⚠ Care must be taken with eyes, particularly in robotic laparoscopic surgery when the combination of pneumoperitoneum and steep positioning increases the risk of regurgitation and chemical burns. Eyes should be securely taped, and pads placed.
- ⚠ Robot manipulators can extend significantly beyond where a human operator would hold an instrument. Robotic arms can cause significant injury to the patient before their clash detection is activated. This should be mitigated by a secure horizontal metal bar to protect the face or other vulnerable areas of the patient. If the patient is repositioned during the procedure, it is important to be vigilant for any new potential clashes between robot and patient.
- IV access should be well secured with extensions that allow remote injection of medication. Infusions should be prepared and connected before docking.
- Positioning of the patient is the most important aspect of robotic surgery. Meticulous attention should be paid to the proper padding of vulnerable areas, wires, IV lines and the use of any patient supports.
- If shoulder supports are used to prevent the patient from slipping while in the head-down position, care should be taken that the head is not hanging free, as this can cause a traction injury of the brachial plexus.
- ⚠ Where feasible, breaks of extreme positioning should be taken every 90–120min to mitigate the risk of compartment syndromes.
- Some robotic systems allow for synchronisation between the table and robot movements and it is important to check if this is the case before adjusting the table position while the robot is docked.
- Patient movement during the procedure can cause significant problems and so an infusion of either muscle relaxant or remifentanil is useful.

Postoperative

- Prior to extubation, a leak test should be performed, as there can be significant airway oedema.
- ⚠ Cerebral oedema 2° to prolonged head-down may lead to agitation and delirium in the immediate recovery period.
- Typically, the analgesic plan should be the same as for the non-robotic case but can be titrated down as familiarity with the procedure increases.

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- Spinal anaesthesia is an excellent option for robotic laparoscopic surgery, but given the length of some procedures, it may need to be supplemented at the end with abdominal wall blocks if an open incision is made to deliver the specimen or create an anastomosis.

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Day surgery

Organisation

A surgical 'day case' is a patient who is admitted, operated upon, and discharged on the same calendar day. In the UK (unlike the US), a 24h stay is not classed as day surgery, as this requires overnight admission and a hospital bed.

The surgery must have been planned as day surgery. Patients who are booked as inpatients but are successfully discharged on the day of surgery will not appear in hospital day case statistics. Booking potential day case patients via an inpatient pathway can result in patient cancellation due to lack of availability of a postoperative bed which would not have been required if a day surgery pathway was planned from the outset.

Organisation is the key to efficient, good-quality day surgery and requires close cooperation between all agencies involved, including surgeons, anaesthetists, day unit staff, GPs, patients and their carers.

Facilities An efficient organisation requires 'ring-fenced' theatres and ward space. Day surgery can be managed successfully in a variety of hospital configurations; however, day cases on inpatient wards and theatres have a higher admission rate and will suffer cancellation when there are bed shortages. Self-contained units with their own facilities, but within an acute hospital, offer the best option.

Escalation It is essential that day surgery facilities will not be used to accommodate overnight patients at times of hospital escalation. At times of escalation, it is even more important that the day surgery unit continues to function efficiently and hence enable the majority of elective surgery to proceed uninterrupted.

Staff Adequately trained and experienced staff should perform day case anaesthesia and surgery to minimise unplanned admissions. The day surgery environment provides many opportunities for training of junior staff, but supervision and guidance from senior clinicians are essential. The unit should be staffed with nurses and operating department practitioners who are in themselves expert day surgery practitioners, invested in the success of the day surgery team.

Day surgery procedures

The British Association of Day Surgery has produced a directory which details over 200 procedures now deemed appropriate to be undertaken on a day case basis. Day surgery is no longer confined to minor procedures but embraces the majority of elective surgery and a large percentage of urgent surgery. There is no time limit for surgical duration; however, consideration must be given when scheduling an operating theatre list to potential postoperative recovery times for more complex surgery, such that longer procedures will usually be undertaken in the morning or early afternoon.

For a procedure to be appropriate for day surgery, the following criteria must be met:

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- It should be possible to control pain with simple oral analgesia supplemented, if appropriate, by regional anaesthetic techniques.
- The patient must be able to eat and drink postoperatively.
- The patient must be able to mobilise (with aids if required) postoperatively.

Example day surgery procedures are shown in Table 18.3.

Table 18.3 Example day surgery procedures

Gynaecology	Laparoscopic hysterectomy, vaginal repair, vaginal hysterectomy
General surgery	Laparoscopic fundoplication
Urology	Laparoscopic nephrectomy, laparoscopic prostatectomy, laser resection of prostate
Breast surgery	Mastectomy, all non-reconstructive breast cancer surgery
Vascular surgery	Endovascular aneurysm repair
Orthopaedics	Total hip/knee or shoulder replacement
Emergency surgery	Appendicectomy, testicular torsion, 'hot' laparoscopic cholecystectomy, laser of ureteric stones

Emergency day surgery

Moving emergency surgical procedures to an ambulatory pathway confers many benefits to hospitals and patients. Where possible, patients should be seen and assessed in a surgical assessment unit and discharged home to wait in their own homes rather than a hospital bed for their procedure. Traditionally, many patients awaiting 'urgent' surgical procedures, such as appendicectomy, laparoscopic cholecystectomy, evacuation of retained products of conception (ERPC) or surgical abscess drainage, are not prioritised for the emergency list and end up being fasted for a number of days, occupying surgical beds until a slot becomes available.

There are a variety of opportunities for urgent patients to be scheduled:

- Dedicated 1st slot on emergency list: the patient attends the day unit at 7.30 a.m. and is prioritised as the 1st patient on the emergency list. Surgery can be undertaken while the team is coordinating the day's activity.
- Planned slots on elective day surgery lists: high-volume urgent procedures can be accommodated on elective day surgery lists by setting aside a number of dedicated slots, e.g. ERPC on a gynaecology list.
- Running dedicated emergency day surgery lists: this enables the patient to be discharged and brought back to an urgent day surgery list within the next 48h, rather than spend 2d in hospital awaiting a theatre slot, e.g. 'hot' laparoscopic cholecystectomy, management of urgent ureteric stones.
- Proactively filling any vacant slots on elective day surgery lists or vacant lists with emergency day surgery patients: available theatre time, due to elective cancellations or unanticipated early finish of day surgery lists, can be utilised by early communication with emergency teams, e.g. appendicectomy, management of ectopic pregnancy, testicular torsion.

Patient preparation and optimisation

Patients should follow a sequential pathway and undergo preoperative assessment by experienced day surgery nursing staff, according to set day case criteria. Nurses should have the support of a consultant anaesthetist whom they can approach for advice.

- Ideal practice is where patients attend assessment at the hospital on the day of their surgical outpatient appointment. This is convenient for patients, avoiding an additional hospital visit. It also ensures time to complete any investigations and to review difficult patients.
- Some patient groups can undergo telephone assessment, in particular those who are young, fit, on no medication and undergoing a procedure that does not require specific tests. However, most patients benefit from a face-to-face assessment.
- Successful day surgery requires anaesthetic departments to consider patients who fall outside traditional guidelines and judge if they can be made fit for day surgery.
- Protocols can be used for preoperative investigations, but these should be kept to those absolutely essential for the surgery, as very few investigations in a day surgery pathway alter management.
- Clear instructions must be given to the patient regarding what will happen on the day of surgery, what they need to bring on the day and the organisation of someone to take them home and care for them postoperatively. These instructions should be reinforced with clear written information.
- Most cancellations on the day of surgery can be avoided by careful preoperative assessment by experienced staff. Those due to acute illness, however, may still occur.

Day case patient selection criteria

Criteria need to be agreed with the anaesthetic department and will vary according to the day surgery unit setting, e.g. more challenging patients and procedures can be undertaken where the unit is integrated into a hospital with all support services. Stand-alone units on isolated sites will need more conservative criteria. However, patients deemed inappropriate for surgery in these off-site units may still undergo a day surgery pathway via the main hospital facility.

Health status (ASA) Day surgery is no longer reserved purely for the fit and healthy. Most patients with stable medical conditions are appropriate for day surgery, and many complex patients will do better in a day case environment if they are managed appropriately, e.g. insulin-dependent diabetics, severe COPD patients, patients on dialysis or those who suffer from epilepsy. If a patient with an unstable medical disease is presenting for surgery, they should have their condition optimised before proceeding with surgery and can then often be managed via a day case pathway. Always try to remember these two questions:

- What can we do to enable this patient to be managed as a day case?
- Would anything be done differently if the patient were to be treated as an inpatient? If the answer to the question is no, then day surgery is probably the optimal pathway.

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Age There is no upper age limit. Physiological fitness should be considered, rather than the chronological age, remembering that the elderly are usually better managed in their home environment. Babies as young as 6w can be managed on a day case basis (excluding premature infants <60w post-gestational age due to the risk of sudden infant death syndrome (SIDS)).

Obesity With appropriately skilled staff and equipment, even the morbidly obese can be successfully managed as day cases. It can be the ideal option, as early mobilisation reduces the risk of complications. There is no longer any BMI limit for day surgery. Assessment should include questions about OSA as caution should be used with regard to postoperative opioid medication if OSA is suspected (see ➲ pp. 73–5).

Transport and social support All patients must be escorted home by a responsible, informed adult and, in most cases, be adequately supervised during their recovery at home for a minimum of 24h. Some units now employ carers for patients without home support to enable them to be treated as day cases. There is increasing acceptance that patients undergoing more minor surgery (this excludes airway or laparoscopic surgery) who are judged appropriate at preoperative assessment and wish to go home alone are permitted to do so and this is preferable to an overnight hospital admission. Patients must have suitable home conditions with adequate toilet facilities, and a telephone should be readily available for advice in an emergency. Patients must agree/understand that they should not drive, cycle, operate machinery or consume alcohol for 24h after their anaesthetic. This advice must be contained in preoperative verbal and written instructions given to the patient, and reinforced prior to discharge.

Geography Though it is procedure-dependent, as a rule, the patient should live within 1h travelling distance from a hospital which could deal with complications arising from the surgery that has been undertaken. This may not always be the hospital where the surgery was performed.

Conduct of day case anaesthesia

The principles of day surgery anaesthesia are well established:

- Premedication with oral analgesia
- Short-acting anaesthetic agents
- Avoidance of emetogenic medication
- Multimodal analgesia
- Short-acting opioids for rescue analgesia if required
- Good postoperative analgesia.

Preoperative

- Avoid sedative premedication, if at all possible. If necessary (usually only for patients with behavioural issues), use PO midazolam, up to 0.5mg/kg, in a little undiluted sweet fruit cordial (as it tastes awful).
- Routine use of antacid drugs is unnecessary; however, in those with a history of regurgitation, ranitidine 300mg PO, or omeprazole 40mg PO, is appropriate.
- Oral analgesics. Paracetamol 1g and NSAIDs reach peak effect after 1–2h and are a useful adjunct to anaesthesia, with very few side effects. Controlled-release NSAIDs (e.g. ibuprofen 1600mg) preoperatively are very effective and provide long-acting postoperative pain relief.
- Patients should be encouraged to remain warm and hydrated preoperatively. Delaying changing from street clothes into theatre gowns until absolutely necessary minimises hypothermia, and encouraging liberal consumption of water preoperatively minimises rates of PONV. Increasing evidence supports free access to water preoperatively rather than arbitrary 'cut-off' times after which a patient must not drink, which are associated with excessive periods of fluid deprivation.

Perioperative

- Short-acting agents should be used. TIVA is ideally suited to day surgery; however, units have also had success with sevoflurane. Avoid agents likely to contribute to PONV such as N_2O or long-acting opioids (e.g. morphine).
- For larger procedures, use incremental fentanyl, up to 2–4 micrograms/kg in divided doses. IV morphine should be avoided.
- Consider NSAIDs, if not already given, and LA for every suitable patient/operation.
- Patients should be kept warm and hydrated with 1L of crystalloids (15mL/kg for paediatric patients). This reduces the incidence of dizziness and PONV and aids recovery.
- Whenever possible, use an LMA, avoiding intubation, muscle relaxants and reversal agents. LMAs for gynaecological laparoscopy and reinforced LMAs for wisdom tooth extraction can be used safely in many circumstances.
- Antiemetics are not indicated routinely, especially if emetogenic triggers (such as N_2O, volatile anaesthesia or long-acting opioids) are avoided, but should be reserved for treatment of any PONV or for prophylaxis in those with a significant history of PONV or surgical procedures with a high incidence of PONV (e.g. ovarian/tubal surgery, squints, scrotal surgery) (see ➲ pp. 442–3).

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Postoperative

- Balanced analgesia with paracetamol, NSAIDs, LA and short-acting opioids is usually adequate. If more analgesia is needed, it is imperative to use it early; consider IV fentanyl 50–100 micrograms.
- Give PO morphine or other agents such as tramadol if stronger analgesia is required. Remember that morphine in doses above 0.1mg/kg increases the admission rate.
- Antispasmodic agents, such as hyoscine, and physical therapies, such as hot water bottles, may help, particularly for cramping lower abdominal pain following gynaecological surgery.

Postoperative nausea and vomiting

(See also ➲ pp. 442–8.)

- A multifactorial approach to the prevention of PONV should be used. The Apfel scoring system may significantly overestimate the risk of PONV if the anaesthetic techniques described above are employed. Patients experiencing PONV must be actively managed before discharge home.
- For high-risk patients, LA techniques or GA using TIVA, avoidance of opioids, multimodal analgesic therapy, good hydration (IV fluids) and minimal fluid fast are appropriate. Dexamethasone 8mg, in combination with cyclizine 50mg, or a 5-HT_3 antagonist are effective prophylactic agents. This is an approach that works well and leaves only a small number of patients requiring rescue treatment postoperatively.

Regional anaesthesia

(See also ➲ Chapter 40.)

The ↑ use of ultrasound enables many procedures to be undertaken under purely regional anaesthesia with huge efficiency gains, particularly if entire lists are undertaken this way. It is reasonable to discharge patients with working plexus blocks, thus allowing the benefit of prolonged postoperative analgesia. Remember that patients need special instructions on the care of the anaesthetised part so as to avoid inadvertent damage. This would include a sling for patients with brachial plexus blocks.

Local anaesthesia

Surgeons should be encouraged to use liberal infiltration of LA for any incision 'if you cut it, block it'. For many procedures such as inguinal hernia repair or breast cancer surgery, surgical infiltration is as effective as regional anaesthesia. It can also be more efficient and associated with fewer complications.

Specific blocks

Field block performed by the surgeon Excellent for LA hernia repair, as provides postoperative analgesia and obviates the need for GA. LA for inguinal surgery is best placed by the surgeon under direct vision. Attempt at ilioinguinal nerve block by anaesthetists has a high incidence of blockade of the femoral nerve, with subsequent unplanned admission due to difficulty mobilising.

Spinals Shorter-acting spinal agents such as 1% chloroprocaine (40–60min duration) and 2% hyperbaric prilocaine (90–120min duration) have revolutionised the ability to provide a day surgery spinal anaesthetic service with rapid postoperative mobilisation. This has been pivotal in the introduction of day case arthroplasty services to many units (see Fig. 18.2 for an example flow chart). If shorter-acting agents are unavailable, consider reduced-dose spinals (5–7.5mg of bupivacaine). This gives a similar onset of anaesthesia with less motor block and a shorter discharge time, compared with standard doses (4h vs 6h). Bupivacaine 0.25% (3–3.5mL) has become widely used for day case joint replacement surgery; however, it is important to use the racemic mixture, rather than levobupivacaine which is associated with a more patchy, unreliable block when used at lower concentrations.

Femoral blocks These are usually inappropriate due to difficulty mobilising postoperatively; however, subsartorial blocks which block the sensory, but not the motor, branch of the femoral nerve are ideal for procedures such as anterior cruciate ligament repair, or unicondylar or total knee replacement.

Discharge drugs

All patients should have a supply of suitable oral postoperative analgesics at home or be given them on discharge. There should be procedure-specific analgesic protocols to ensure consistent prescribing between clinicians (Table 18.4). Audit of postoperative pain scores should be undertaken to ensure the protocols are appropriate.

Discharge criteria

- Stable vital signs.
- Fully awake and orientated.
- Tolerating oral fluids.
- Passing urine is only essential after specific procedures (e.g. urology, gynaecology) or spinal/caudal anaesthesia.
- Ambulant.
- Pain and nausea well controlled.
- Minimal bleeding or wound drainage.

Specific discharge criteria for regional techniques

Spinal anaesthesia

- Full recovery of motor power and proprioception
- The patient has passed urine.

Regional blocks

- Understanding of protection of partially blocked limb
- Instructions regarding when the block should have regressed and whom to contact if it has not
- Adequate mobility on crutches, if required.

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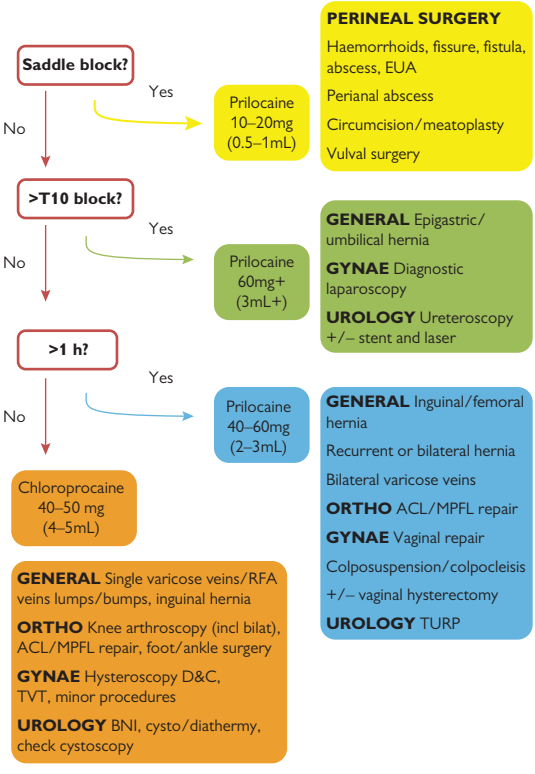

Fig. 18.2. Protocol for use of short-acting spinal anaesthetic agents in day surgery. Courtesy Dr Robbie Erskine, Dr Gillian Turner and Eleanor Erskine.

Discharge organisation

Written and verbal discharge information must be provided. It is essential that patients are given the contact number of someone in the hospital, should they require advice overnight. This may be the day surgery unit within opening hours and either a surgical ward or senior surgical nurse overnight. It is not appropriate for patients to be directed to out-of-hours GP services or emergency departments.

Table 18.4 Example protocol for day surgery take-home analgesia

Expected pain	Example procedures	Analgesia protocol
None	Cystoscopy	None
Mild	Prostatic biopsy	Paracetamol 1g qds
Moderate	Dental extractions Mastectomy Arthroscopy	Paracetamol 1g qds Ibuprofen 400mg qds
Severe	Laparoscopic cholecystectomy Laparoscopic hysterectomy Hernia repair	Co-codamol 30/500 2 tabs qds Ibuprofen 400mg qds Macrogol one sachet bd

Postoperative admission

The most common reasons for overnight admission are:

- The patient not fulfilling discharge criteria before the unit closes
- Observation after surgical or anaesthetic complications
- Unexpectedly more extensive surgery
- Uncontrolled pain or PONV.

Overall, unanticipated admission rates will depend upon the surgical case mix undertaken. With increasingly complex procedures entering the day surgery arena, timely discharge is more challenging to achieve, and consideration to the development of a procedure-specific anaesthetic guideline is required. Examples of procedures where this has been found useful include joint replacements, hysterectomy, laparoscopic cholecystectomy and tonsillectomy.

Follow-up, audit and benchmarking

Patient follow-up is an essential part of the day surgery pathway to ensure patients are comfortable and satisfied postoperatively. The most effective method of undertaking this is via a postoperative telephone service. A structured questionnaire should be undertaken covering the following:

- Postoperative pain score
- PONV score
- Patient satisfaction
- Satisfaction with the day surgery pathway
- Requirement for any postoperative advice or support.

This service provides detailed audit information, analysis of which drives continuous service improvement. It also provides the opportunity for patient support, should they have any queries or concerns following their procedure.

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Society for Ambulatory Anesthesia. http://www.sambahq.org

Neurological determination of death

Dying is a process. Death can be diagnosed when that process has progressed to the irreversible loss of the capacity for consciousness, combined with the irreversible loss of the capacity to breathe.

The standard used for neurological determination of death (NDD) in many countries is that of **brain death**. This requires the loss of all brain function (cerebral and brainstem). In the UK, **brainstem death** is used for NDD. In contrast to brain death, brainstem death does not require that all brain functions have ceased, only that any functions that might persist do not indicate any form of consciousness. The clinical determination of whole brain death and brainstem death are the same and stressing the differences between them results in unnecessary confusion and controversy.

- The 1° reason to confirm brainstem death is that it provides a legal and ethical basis for the withdrawal of invasive ventilation. Secondly, confirmation of brainstem death enables the donation of organs for transplantation if that is consistent with the patient's and their families' wishes (deemed consent applies; see ➲ pp. 497–502).
- The most common causes of brainstem death are traumatic brain injury, intracranial haemorrhage and hypoxic brain injury. To diagnose brainstem death, the patient needs to fulfil certain preconditions and have absent brainstem reflexes.
- Two doctors are required to determine death by neurological criteria. The qualifications required of the doctors vary between jurisdictions, but they must be familiar with, and competent at, brainstem testing. At least one should be a consultant.

Preconditions for neurological determination of death

- Patient comatose, apnoeic and dependent on mechanical ventilation.
- Coma caused by irreversible brain damage of a known aetiology.
- The patient must be normotensive (MAP ≥60mmHg in an adult and an age-appropriate BP in children).
- The patient must be normothermic (temperature >34°C).
- Reversible causes for brainstem depression excluded: sedatives, muscle relaxants, alcohol and metabolic or endocrine disturbances (severe untreated hypothyroidism or hypoadrenalism) (Table 18.5).
- Absence of acute liver failure or decompensated chronic liver disease.
- If there is any doubt about the persisting effects of opioids or benzodiazepines, an appropriate antagonist should be administered at the time of examination or drug levels measured (thiopental <5mg/L, midazolam <10 micrograms/L).
- Unless it is known for certain that neuromuscular-blocking medications have not been administered, a PNS or other recognised method (e.g. EMG) should always be used to confirm that neuromuscular conduction is normal.
- It must be possible to examine all brainstem reflexes, with at least one ear and one eye available for examination.
- It must be possible to perform an apnoea test. Severe hypoxic respiratory failure or high cervical spine injury may preclude this.

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- A period of observation is required from when the patient meets the preconditions and has a GCS 3, with fixed pupils and absent cough, and is apnoeic.
- Clinical testing should be delayed for 24h when the cause of coma is hypoxic–ischaemic encephalopathy or if the patient has been hypothermic.

Table 18.5 Acceptable metabolic levels to fulfil preconditions of clinical testing*

Factor	Acceptable range (mmol/L)
Na^+	115–160
K^+	>2
Mg^{2+}	0.5–3
Phosphate	0.5–3
Glucose	3–20
Urea	<40

* These are not prescriptive and should not replace clinical judgement, especially if there has been rapid correction.

Testing for absence of brainstem responses

Tests of brainstem reflexes should be performed only when the preconditions are fulfilled.

- The pupils are fixed and there is no response to sharp changes in the intensity of incident light. The pupils are usually dilated, but this is not essential for the diagnosis.
- The corneal reflex is absent.
- There is no motor response within the trigeminal nerve distribution to painful stimuli applied centrally or peripherally. Spontaneous and reflex movements (spinal reflexes) may persist in brainstem-dead patients.
- The oculovestibular reflex is absent. There is no eye movement in response to the injection of 50mL of ice-cold water into the external auditory meatus, with the head at 30° to the bed. Direct access to the tympanic membrane should be verified using an otoscope. The eyes should be observed for at least 1min after each injection.
- There is no gag in response to posterior pharyngeal stimulation.
- There is no cough reflex in response to a suction catheter passed down the ETT.

Confirmation of apnoea

This test is done last to avoid unnecessary hypercapnia or hypoxaemia, should any of the other reflexes be present.

- The patient should be preoxygenated by ventilating with 100% O_2 for at least 5min and the minute ventilation reduced to achieve an end-tidal $PaCO_2$ of 6kPa (45mmHg). $ETCO_2$ can be used to guide the start of the apnoea test and when to measure an arterial $PaCO_2$ but does not replace the pre- and post-arterial $PaCO_2$.

- Check an ABG to confirm that $PaCO_2$ is at least 6kPa and the pH is <7.4. In patients with chronic CO_2 retention, the CO_2 should be allowed to rise such that the pH is <7.4 before commencing the apnoea test.
- The patient is then disconnected from the ventilator and the ETT is attached to a self-inflating bag with at least 5cmH_2O of PEEP and 15L/min of O_2 flow. This provides apnoeic oxygenation and minimises atelectasis.
- Observe the patient continuously for a minimum of 5min for any respiratory movement. The $PaCO_2$ should be measured and should be high enough to ensure an adequate stimulus to ventilation.
- The apnoea test is positive (absent breathing) if no spontaneous respiration is observed and the $PaCO_2$ has risen by >0.5kPa.
- The ventilator is then reconnected with settings to allow gradual return of the ABGs to the pretest level.

Paediatrics

The criteria for NDD in children over 2mo of age are the same as in adults.

- NDD by clinical examination in neonates <37w of post-conceptual age is unreliable due to the immaturity of brainstem reflexes.
- Between 37w of post-conceptual age and 2mo of age, it is possible to confirm death by neurological criteria, but caution is required and specialist advice recommended.
- At least one of the doctors examining the child should be a paediatrician.

ECMO

NDD testing may be performed on patients who are on ECMO.

- A guideline for performing this test is available from the Faculty of Intensive Care Medicine and the Intensive Care Society of the UK.

Ancillary and confirmatory investigations

Ancillary tests or confirmatory tests are not required in the UK. However, if preconditions are not met and where a full neurological examination is not possible (e.g. extensive facial trauma, inability to perform apnoea test due to high cervical spine injury or hypoxia), it is essential to perform further investigations.

- Investigations should be deferred until responsiveness, examinable brainstem reflexes and breathing effort are all absent.
- Investigations may include neurophysiological demonstration of loss of bioelectrical activity in the brain (e.g. EEG) and radiological demonstration of absent cerebral blood flow or brain tissue perfusion (e.g. CT angiography, 4-vessel angiography, transcranial Doppler).
- The two testing doctors must determine the utility of additional investigations and it is recommended they should seek further professional opinion from other specialties and other expert centres, where appropriate.

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Other considerations

- The tests must be performed twice, each by a different doctor.
- The diagnosis should not normally be considered until at least 6h after the onset of an apnoeic coma or 24h after the restoration of circulation if the cause was a cardiac arrest.
- If prolonged hypothermia (<34°C for >6h) has occurred, the tests should be performed at least 24h after the restoration of normothermia.
- Death is confirmed after the 2nd set of tests, but in the UK, the time of death is recorded as the completion of the 1st set.
- The coroner (Procurator Fiscal in Scotland) should be informed in the usual referral manner. If organ donation is contemplated, this should be discussed.
- Care of the relatives is essential at this time, irrespective of whether the patient is to be an organ donor or not.
- Observations that are incompatible with the diagnosis of brainstem death include seizures, decorticate and decerebrate posturing and limb movements elicited with stimulation in the cranial nerve distribution
- Observations that are compatible with brainstem death include spinal reflexes, blushing and the absence of diabetes insipidus.
- Spinal reflexes can be spontaneous or in response to stimulus. They do not occur in the motor distribution of the cranial nerves or in response to stimulation within the cranial nerve distribution.

Circulatory determination of death

For organ donation following circulatory determination of death, the following observations are required:

- The person responsible for confirming death must observe apnoea, unconsciousness and the absence of pulsatility on an arterial line trace or of electrical activity on ECG for 5min.
- After 5min of continued cardiorespiratory arrest, death is confirmed if there is no pupillary response to light, no corneal response and no motor response to supraorbital pressure.

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Organ donation surgery after brain death

Procedure	Procurement of donor organs via midline laparotomy and median sternotomy
Time	Up to 6h, depending on which organs are retrieved
Pain	N/A
Position	Supine
Blood loss	Large fluid losses likely, X-match 4 units
Practical techniques	IPPV, central venous line and arterial line

Transplantation of donated organs to selected patients with end-stage organ failure is a successful and cost-effective therapy, improving both quality and duration of life. However, demand for donor organs continues to exceed supply. Organ donation discussions should be considered as a usual part of 'end-of-life care'.

There are few absolute contraindications to donation (e.g. transmissible spongiform encephalopathies, disseminated metastatic malignancy), but many relative contraindications, and so it is recommended that all potential donors are identified and discussed early with the organ donation agency before being ruled out and prior to any family discussions about donation.

Legislative changes in the UK mean that *deemed consent* now applies for all potential donors over 18y old who have lived in the UK for >12mo. Families must be consulted and their objections to donation must be respected.

The characteristics of deceased donors continue to change, with relative stability in the number of donors after brain death (DBD) and ongoing increase in donors after circulatory death (DCD) (see ➲ pp. 501–2).

There are tending to be fewer donors after traumatic brain injury and more from CVE, hypoxic–ischaemic encephalopathy and other non-traumatic conditions.

The number of donors with comorbidities or potentially reversible organ dysfunction is increasing. This is a result of changes in admission criteria to the ICU and end-of-life practices, and more liberal acceptance by transplant services of these donors.

Brainstem death is associated with a series of predictable physiological changes that, if inadequately managed, can lead to significant deterioration in organ function.

Pathophysiology of brainstem death

- Early, short-lived, massive sympathetic outflow occurs during brainstem herniation, causing hypertension, tachycardia, myocardial dysfunction, impaired organ perfusion and tissue ischaemia.
- Autonomic collapse results in a reduction in CO, loss of vasomotor tone, hypotension and atropine-resistant bradycardia. If untreated, hypoperfusion of all organs will occur. Tissue ischaemia can trigger a systemic inflammatory response that contributes to cardiovascular instability.

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- Deterioration in lung function may occur due to neurogenic pulmonary oedema and acute lung injury (ALI).
- Endocrine changes are variable in timing and severity. Posterior pituitary function is commonly lost. Reduced ADH secretion leads to neurogenic diabetes insipidus, with hypovolaemia and electrolyte disorders (↑ Na^+, ↓ K^+, ↓ phosphate)
- Some anterior pituitary function may remain, but a fall in T_3 and T_4 levels may cause myocardial dysfunction and a global shift to anaerobic metabolism.
- Hyperglycaemia is due to both reduced circulating insulin and insulin resistance.
- Release of tissue fibrinolytic agents and plasminogen activators from the necrotic brain causes a coagulopathy.
- Hypothermia occurs due to reduced whole body metabolism, hypothalamic dysfunction and loss of large volumes of dilute urine in untreated diabetes insipidus, and from the inability to shiver and vasoconstrict.

Prior to donation

Maintaining physiological support to a patient until brainstem testing is undertaken preserves the opportunity for a discussion with the family about the potential for organ donation.

- Typically, once a potential donor is identified, there will be several conversations between the hospital staff, the organ donation agency and the family.
- In anticipation of cardiovascular instability as brainstem death occurs, a multilumen central line (internal jugular or subclavian is preferable), one large peripheral IV line for rapid infusion (14G or 16G) and an arterial line (upper limb preferable) should be sited.
- Check that brainstem death has been confirmed and that consent for organ donation has been obtained from the relatives and the coroner.
- Emphasis in management changes from cerebral resuscitation to optimal organ perfusion and oxygenation. When sympathetic hyperactivity occurs, use a short-acting β-blocker (e.g. esmolol) to treat severe hypertension (MAP >130mmHg) and tachycardia. Do not use long-acting agents or aim for full β-blockade.
- At the time of brainstem death, sympathetic tone is lost and hypotension occurs. Give a volume challenge of resuscitation fluid (500mL to 1L) and be prepared to start vasopressin (0.01–0.04 units/min). Some countries use noradrenaline as their 1st choice of vasopressor.
- Large fluid volumes can be lost through untreated diabetes insipidus and unrecognised blood loss. Ensure adequate intravascular volume resuscitation using appropriate clinical assessments and investigations.
- It is not necessary to raise the CVP if BP and other perfusion goals are met. Avoid fluid overload in potential lung donors as this reduces the number of donor lungs that can be retrieved.
- If hypotension persists despite adequate fluid resuscitation and treatment of vasoplegic shock, an inotrope may be required. There is little evidence to justify one specific agent over another.

- If inotropic requirements remain high, search for blood loss, sepsis or cardiac dysfunction. If available and ICU staff are familiar with their use, serial echocardiography, continuous TOE or PAFC may be helpful in guiding therapy.
- Patients on vasopressin may still require desmopressin (DDAVP®) to control diabetes insipidus as this has greater antidiuretic effect. Start with a dose of 4 micrograms and repeat if urine output remains >3mL/kg/h.
- Lung function is commonly abnormal due to direct lung injury, pulmonary oedema, atelectasis and pneumonia. Lung donation is usually possible if PaO_2 >40kPa on 100% O_2 and 5cmH_2O of PEEP.
- Minimise ventilator-induced lung injury by ventilating with V_T of 6–8mL/kg. Use at least 5cmH_2O PEEP, the lowest FiO_2 to maintain O_2 saturations of >92%, and ventilate according to the targets in Table 18.6.
- Continue regular chest physiotherapy, 2-hourly suctioning and turns.
- Thromboprophylaxis, infection control measures and oral hygiene should be continued.
- Enteric feeding may reduce inflammation and have beneficial effects in the transplanted organs, but if gastric aspirates are high, the risk of aspiration is ↑.
- Prevent hypothermia by using warmed IV fluids and warming blankets.
- The use of hormone therapy with T_3 replacement, methylprednisolone and vasopressin varies among transplant centres. Their use should be guided by the local retrieval team or in-house protocols.
- Correct ↑ Na^+ with 5% glucose in water. ↑ Na^+ (Na^+ >155mmol/L) in the donor has been associated with worse outcomes for liver transplant recipients. Aim for serum Na^+ of 135–150mmol/L.
- Use 1mL/kg/h maintenance fluid as 5% glucose in water. Resuscitation fluids (e.g. 0.9% sodium chloride, PlasmaLyte® 148) are not maintenance fluids and their continued use may lead to oedema, hyperosmolarity and organ dysfunction.
- ↓ K^+ can occur during brainstem death and increase the risk of cardiac dysrhythmias. Replace K^+ IV and aim for serum K^+ of 4.0–4.9mmol/L.
- Maintain glucose between 4 and 12mmol/L, with insulin as required to prevent glycosuria.
- Clotting abnormalities should be corrected with clotting factors and platelets.
- FBC, coagulation screen and LFTs should be measured at least daily.
- Blood gases (with Hb, Na^+, K^+, glucose and lactate), as well as serum urea and creatinine, should be measured 6-hourly (more frequently if clinically indicated).
- Order CXR, ECG and echocardiography for potential heart/lung donors. Transplant teams may request other investigations, e.g. coronary angiogram or CT thorax.
- Target physiological parameters are outlined in Table 18.6.

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Table 18.6 Target parameters for organ retrieval

Target parameters	
MAP	60–80mmHg
Pulse	60–120/min
Cardiac index	>2.1L/min/m²
Core temperature	36–37.5°C
Hb	>70g/L for CV stable donor >90g/L for unstable donor
SpO_2	>95% with lowest FiO_2 to maintain PaO_2 >10kPa
V_T	6–8mL/kg
$PaCO_2$	NHS guidelines: 5–6.5kPa as long as pH>7.25 ANZICS and US guidelines: 4.7–6.0kPa (35–45mmHg)
Plateau pressure	<30cmH_2O
Urine output	0.5–2mL/kg/h

Perioperative

- The WHO Surgical Safety Checklist (or similar) should be applied.
- During the organ retrieval operation, the anaesthetist should aim to maintain physiological stability in the donor to ensure the donated organs are in optimal condition for transplantation.
- Standard monitoring is required, plus an arterial line, core temperature and urine output.
- Patients who are brain-dead cannot experience pain and lack consciousness and so a true 'anaesthetic' is not required. However, spinal cord function remains intact and administration of NMBAs, volatile agents and, at times, an opioid is required to prevent **spinal** reflex motor responses and reduce **spinal** sympathetic responses (tachycardia and hypertension) that can occur spontaneously or during surgical stimulation.
- Opioids are sometimes given to suppress catecholamine-mediated sympathetic activity, although they may not be sufficient. GTN or a β-blocker may also be needed to treat hypertension.
- Spinal reflexes in the limbs can be distressing for observers and could potentially lead to contamination of the operative field. Excessive sympathetic responses can result in myocardial injury or excessive bleeding, with subsequent haemodynamic instability and detrimental effects on graft function.
- Large and frequent haemodynamic fluctuations occur due to compression of the IVC, manipulation of the adrenal glands and blood/fluid loss. Hypotension is treated with crystalloids, blood products, vasopressin infusion and inotropes as indicated. There is no role for synthetic colloids as these may impair graft function. Have 4 units of RBCs available in theatre.
- Broad-spectrum antibiotics are given as per local transplant protocol.
- Cardiothoracic and abdominal surgeons work concurrently to mobilise the organs.

- IV heparinisation (300 units/kg) should be administered on request of the surgeons prior to aortic cross-clamping. This is to prevent clot formation in retrieved organs.
- Following aortic cross-clamp and the start of perfusion fluids, mechanical ventilation ceases and the anaesthetist is no longer required.
- Organs are retrieved in the order of the heart, lungs and then liver.
- The abdominal surgical team continues to operate in circulatory arrest, retrieving the kidneys, pancreas and blood vessels.

Special considerations

Ongoing care of the patient includes maintenance of their dignity and showing empathy and sensitivity in dealing with the donor's family.

- Good communication within an atmosphere of mutual respect and trust is paramount throughout the management of the potential organ donor.
- The quality of care afforded to the multiorgan donor could affect the outcome of >6 recipients.
- In the event of a cardiac arrest in theatre, CPR should be commenced as procurement of the liver and kidneys can still proceed rapidly with cross-clamping of the aorta at the diaphragm and infusion of cold preservation solution into the distal aorta and portal vein.

Donation after circulatory death

Retrieval of organs for the purpose of transplantation can occur under specific circumstances when death is confirmed using cardiorespiratory criteria (see ➲ p. 496).

- DCD was previously referred to as donation after cardiac death or non-heart-beating organ donation.
- Relative contributions of DBD and DCD vary around the world. DCD has ↑ significantly in the UK in the last decade and now accounts for almost 40% of deceased organ donors.
- The liver, kidneys, heart, pancreas and lungs are all potentially donatable after DCD.
- DCD may be considered only once a decision has been made that it is in the best interests of the patient to withdraw life-sustaining therapies. This is followed by discussions with the organ donation agency and with the patient's family.
- Managing a potential DCD donor through the donation pathway is a complex process.
- When life-sustaining therapies are withdrawn, standard end-of-life care continues, including administration of medications to prevent suffering. The location where this occurs will be determined by local practice.
- If lung donation is to occur, it is permissible to reintubate the donor to prevent aspiration. A single recruitment manoeuvre may be required, but repeat insufflation of the lungs is not appropriate.
- If death does not occur within the time frame to allow organ donation, there should be an established plan for ongoing end-of-life cares. The option of tissue donation should remain.

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Major anaesthetic mishaps: what to do in the aftermath

Over the course of a career, most anaesthetists will experience at least one catastrophic anaesthetic mishap which will result in either death or major injury for the patient.

The psychological consequences of this are variable. Whether a death is 'anticipated' or 'unexpected' is not relevant, as any death, serious injury or 'near miss' in theatre can have a profound and potentially lasting psychological impact on the anaesthetist.

Immediate actions in the operating theatre

Contact a senior colleague

A consultant anaesthetist should be involved as soon as possible. These are rare events which carry heavy emotional loads and the actions required in the aftermath are unfamiliar to most. You will need help and this must be provided.

Clinical notes Detailed, truthful and ideally contemporaneous notes of the event should be recorded. During a resuscitation, this task may be allocated to another member of the team, but it is essential the anaesthetist records a full account as soon as possible with a time and date. This record should include: details of preoperative discussions, the patient's premorbid condition, how the emergency was recognised and treated and the anaesthetic record annotated as necessary.

If possible, keep an encrypted copy of your clinical notes with additional information (your thought processes and opinions), as medicolegal events take time and your memory may fade.

Caring for the patient If the patient has died, this needs to be certified. All intraoperative deaths must be reported to the coroner (England and Wales) or the Procurator Fiscal (Scotland). Although lines, tubes and other equipment should typically remain in place, the patient can be cleaned, and wounds sutured closed and dressed, in preparation for viewing by the family.

If the patient has not died, the anaesthetist should follow up on the progress of the patient. The patient will now be under the 1° care of another team (likely ICU) and the anaesthetist's role is to show appropriate interest and empathy for their wellbeing, but they should not get involved in treatment decisions, prognostication or independent discussions with the family.

Isolate equipment and drugs If there is concern that equipment or medications were the cause of the catastrophe or if the cause is unknown, then all procedural equipment, medications and syringes must be isolated and stored for subsequent investigations. If there is concern the theatre or anaesthetic machine have contributed to the event, the theatre should be closed pending investigation.

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Caring for the family A formal meeting with the family needs to be arranged immediately. Bad news must not be broken over the phone, although they should be informed there has been a complication and that they need to come to the hospital immediately. It is advisable they bring a support person. If English is not the family's first language, a translator must be used.

Breaking bad news can be challenging, especially for those who do not do it very often. This involves a team approach and should include senior members of the anaesthetic, nursing and surgical teams.

Prior to meeting with the family, a brief premeeting should occur with the attendees to identify who will be the 1° spokesperson; if the patient has survived, this role may be most appropriately performed by the intensivist now caring for the patient. At the premeeting, a summary of the events and facts to be discussed should be agreed upon. Information that is unknown should be acknowledged.

The meeting should take place in a quiet room where interruptions will not occur. All members of the team should be introduced, as well as all family members. It is important the bad news is delivered succinctly at the beginning, in easy-to-understand, non-medical language. Apologising to the family that this event has occurred is not an admission of guilt, but a sign of both empathy and caring and may provide comfort to a grieving family. A bad news meeting can be emotionally draining for all concerned and only a small proportion of the information presented may be retained. If the family asks questions to which you do not know the answer, reply honestly and tell them if and how you will find out this information. A record of the meeting must be documented in the clinical notes and should include who attended, what was covered and what follow-up is planned.

Caring for the team Ideally, after a major event, the theatre should be immediately closed and subsequent cases cancelled or performed by another team. A 'hot' debriefing session should be facilitated as soon as possible. This allows staff to talk about what has happened and aims to defuse emotions. It is also used to let staff know what other resources are available to them, e.g. counselling or further more formal debriefing sessions.

Caring for the anaesthetist It is essential that the anaesthetic department provides support for the anaesthetist.

Surveys reveal >70% of anaesthetists who experience a catastrophic perioperative event will have a high degree of emotional impact, experiencing guilt, anxiety, sleeplessness, fear of judgement by colleagues and of litigation, anger and reliving the event.[3] They may also suffer from physical effects such as tiredness, muscle tension and nausea. These are a natural consequence of such an event but should lessen within a week or so. Depending on the event and the individual, time off work to emotionally recover may be helpful and on return to work, additional support may be needed.

Support from a mentor should be sought or arranged by the clinical lead as soon as possible. Discussing the events in an open, non-judgemental manner can assist the anaesthetist in coming to terms with what has

happened. Professional psychological support may also be required and the anaesthetist should be assisted in accessing this. Occupational health may need to be notified. A 2nd 'cold' debrief with all team members may be useful.

Notifying other staff members It is important to remember the other health professionals who have been caring for the patient, e.g. ward nurses and house officers, who should be told directly rather than hearing it through the 'hospital grapevine'. The patient's GP should also be advised.

Formal investigations and medicolegal considerations Following a major anaesthetic catastrophe, one or more investigations may take place. Any doctor who is involved should assist with these investigations in order to identify the cause of the event and whether lessons can be learnt to prevent further events occurring. The hospital will provide legal advice to the individual doctor, but it is recommended that the anaesthetist notifies their own medical defence organisation as soon as possible.

Mortality and morbidity meeting This should be chaired by a senior anaesthetist and should be undertaken in a manner so as to learn lessons from the incident, as opposed to apportioning blame. It may be most useful to do this once other investigations have been completed and the cause is known.

Internal enquiry/root cause analysis With unexpected serious incidents, it is extremely likely an internal enquiry will be undertaken. This will typically involve the anaesthetist being interviewed and giving a statement. The aim of this sort of enquiry is to identify any processes, system failures or other weaknesses in order to prevent the event occurring again. However, disciplinary actions can potentially follow, and so notifying your medical defence organisation is recommended. They will provide you with advice and representation as needed. It is also wise to take a mentor or trusted colleague along to provide support, as these investigations can add considerable stress.

Coronial inquest All deaths under anaesthesia or related to an operation require referral to the coroner (or the Procurator Fiscal in Scotland). Inquests may take place months after the death and so good record-keeping is vital.

Medical Council investigation If there are concerns about a doctor's performance or competence during a critical incident, the General Medical Council (GMC) should be notified. The GMC will inform the individual if a complaint has been received and ask them to respond. Following an initial investigation, the GMC may determine no further action is required, issue a formal warning, agree undertakings with the individual to address a problem in their practice or refer the case to the Medical Practioners Tribunal Service.

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Criminal prosecution Police investigations are unusual. They occur if there are suspicious circumstances, if it is felt that a clinician has been grossly negligent or as a recommendation following a coronial inquest. Any communications with the police should be done in conjunction with your medical defence organisation. These investigations are invariably extremely slow and can take months or even years to complete. Any doctor involved in this must be offered support as this process is incredibly stressful.

Civil litigation Following a major anaesthetic mishap, a civil case for negligence can potentially be brought against the doctor or the hospital. This is an exceedingly slow process and can drag on for years.

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Chapter 19

Cardiac anaesthesia

Kelly Byrne, Kate Goldstone and **Peter Simmons**

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See also

➲ Anaesthesia for transcatheter aortic valve implantation p. *834*

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Preoperative considerations

As with all anaesthetic assessment, it is crucial in cardiac surgery to understand the indications for the surgery and the expected benefits, so an effective risk–benefit analysis can be undertaken. Most successful cardiothoracic units will use a multidisciplinary team approach where the best option for the patient is considered among cardiologists, surgeons, anaesthetists and critical care specialists. Decisions regarding the choice between a percutaneous intervention and surgery are not as clear-cut as they were previously, and may depend on institutional expertise or protocols. Increasingly, there are two potential intervention pathways for patients to consider. There are well-described guidelines for the indications for CABG and the timing of valve replacement or repair. According to current guidelines, CABG has a survival benefit over PCI in patients with left main stem disease, severe (>70% stenosis) three-vessel disease, reduced EF, DM and patients with a SYNTAX score (used to score the complexity of CAD) >22.[1]

Valve replacement should be considered in symptomatic patients, patients where the valve disease is severe and the patient is considered low risk for surgery and those where there has been myocardial change as a result of the valve lesion, even in the absence of symptoms, e.g. ventricular dilation in AR or MR.[2]

In addition to the routine preoperative assessment, careful attention should be paid to:

- Recent MI and stability of ischaemic symptoms
- History of heart failure
- Bleeding history
- Current rhythm and history of arrhythmia
- Other organ dysfunction, and reserve including cerebral reserve
- Previous cardiac surgery or interventions
- Previous radiotherapy to the chest
- Assessment of LV function (usually via echocardiography, occasionally LV ventriculogram during angiogram)
- Other important echocardiographic findings (significant valvular heart disease, presence of ↑ pulmonary pressures, RV dysfunction)
- Configuration of CAD if present
- Exercise tolerance—gives an indication of cardiorespiratory reserve
- Routine investigations, including the following:
  - Bloods: FBC, U&E/eGFR, LFTs, coagulation, group and screen (G&S)
  - ECG, CXR, echocardiography, angiogram (or CT angiogram)
- Additional tests (e.g. iron studies, cardiac biomarkers, HbA1c, PFTs, carotid artery doppler)
- A history of dysphagia, hiatus hernia or surgery on the oesophagus, relevant and important for any patient requiring TOE insertion.

Medications

- ACE inhibitors and ARBs are stopped perioperatively in many centres.
- Check that antiplatelet and anticoagulant drugs (including novel oral anticoagulants) have been stopped appropriately. Platelet assays can be undertaken to determine whether any residual activity remains.
- With cardiac preadmission clinics and day of surgery admissions, many centres do not premedicate patients the night prior to surgery.
- Patients often receive midazolam after IV cannula placed in theatre.
- A cautious approach should be taken if administering premedication in patients with pulmonary hypertension/RV dysfunction, or severe AS.

Risk stratification

The EuroSCORE II and Society of Thoracic Surgeons (STS) scoring systems are frequently used cardiac risk models (Table 19.1). They predict an individual's mortality after cardiac surgery. These risk calculators are available online at:

- http://riskcalc.sts.org/stswebriskcalc/calculate
- http://www.euroscore.org/calc.html

Some preoperative interventions aim to improve an individual's risk profile, such as:

- IABP for those with critical CAD
- Placement of defibrillation pads in those with a history of VF arrest
- Preoperative pulmonary vasodilators in those with ↑ PAP
- Awareness that patients with critical mitral stenosis and AS tolerate AF poorly.

Table 19.1 Comparison of STS and EuroSCORE II risk scores

	STS scoring system	EuroSCORE II
Patient factors	Demographics, weight, height, blood tests, comorbidities, endocarditis, smoking and drinking status, family history, mediastinal radiation, home O_2, cardiac interventions	Demographics, renal impairment, extracardiac arteriopathy, mobility, chronic lung disease, cardiac surgery, preoperative state, DM, endocarditis
Cardiac factors	NYHA class, angina, MI, arrhythmias, LV function, pulmonary hypertension, number of diseased vessels, antiplatelets, valvular disease, resuscitation	NYHA class, angina, MI, LV function, pulmonary hypertension
Operative factors	Urgency, type of operation, IABP or ECMO preoperatively	Urgency, type of operation, surgery on thoracic aorta

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Intraoperative transoesophageal echocardiography

TOE has become standard practice during cardiac surgery in many centres, although this is not completely without controversy. While generally very safe, there is a 0.2% chance of complications during intraoperative TOE, ranging from minor abrasions to oesophageal rupture (0–0.3%).[3] Oesophageal rupture can be fatal and is ↑ in elderly ♀.

Class I indications

Valve repair or replacement, unexplained haemodynamic instability.

Absolute contraindications

- Perforated viscus, oesophageal stricture, oesophageal tumour, oesophageal perforation, laceration, oesophageal diverticulum, active upper GI bleed.

A comprehensive perioperative TOE includes 28 standard 2D TOE views.[3] (See Fig. 19.1.)

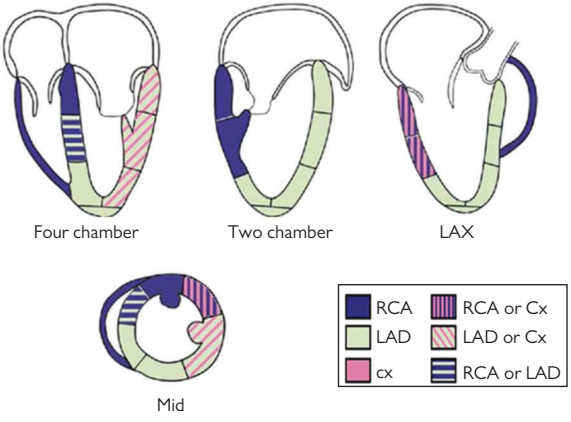

Fig. 19.1 This image demonstrates the typical distributions of the right coronary artery (RCA), left anterior descending (LAD) and circumflex (Cx) artery supply to the left ventricle, as seen on 2D TOE. Modified with permission from Lang RM *et al*. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiograpy, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005; **18**:1440–63, with permission from Elsevier.

Cardiopulmonary bypass

- CPB replaces the function of the heart and lungs while the heart is arrested, allowing for a bloodless, motionless surgical field.
- The pump (roller or centrifugal) requires full anticoagulation of the patient with systemic heparin.
- The pump is primed with crystalloid, heparin, mannitol, HAS and HCO_3^-. The pump delivers non-pulsatile flow of around 2.4L/min/m^3 (correlating with a normal cardiac index), maintaining a MAP of between 50 and 70mmHg.
- Volume can be added to the pump via the reservoir or removed by ultrafiltration to maintain an Hct of 20–30%.
- CPB causes platelet dysfunction, haemolysis and consumption of coagulation factors. This is minimal for the first 2h but increases with prolonged duration.
- Membranous oxygenators are incorporated into the CPB circuit, providing oxygenation via diffusion down a concentration gradient over a large surface area; increasing gas flow removes more CO_2, and increasing the O_2 concentration increases alveolar partial pressure of oxygen (PAO_2).

(See Fig. 19.2.)

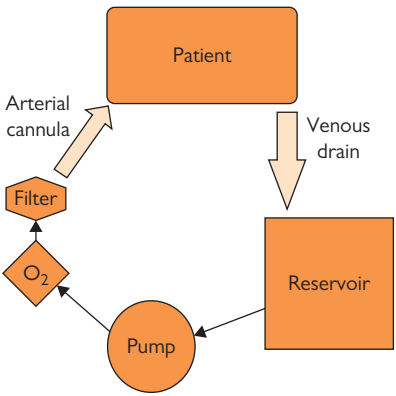

Fig. 19.2 Schematic of CPB circuit. Venous blood drains into a reservoir bucket; a pump controls onward flow through the oxygenator and a bubble filter ensures no gas embolises into the systemic circulation via the arterial cannula.

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Myocardial protection during CPB

The myocardium is protected by achieving diastolic electrochemical arrest following the delivery of a cardioplegic solution. A cross-clamp is placed on the aorta proximal to the cannula to isolate the coronary circulation and heart from the patient's circulation.

- There are a number of different cardioplegia solutions that can be used to arrest the heart. The basic mechanism of action is to alter the resting membrane potential of the heart so that it arrests in diastole. Institutional beliefs tend to drive the use of particular cardioplegia solutions.
- The most widely used cardioplegic solution consists of crystalloid mixed with blood and can be administered warm or cold. The crystalloid contains high concentrations of K^+ which precipitates the arrest of the heart in diastole. Glutamate and aspartate may be added to promote oxidative phosphorylation.
- Delivered via an antegrade approach either via injection into the aortic root following cross-clamping or, in the instance of AR, following aortotomy and direct cannulation of the coronary ostia (this requires fibrillation of the heart to prevent bleeding).
- Alternatively, it is administered via a retrograde approach by cannulating the coronary sinus (infusion pressure is monitored by running a pressure line from the retrograde cannula to the anaesthetic CVP or PAP transducer). This approach is not as effective for right heart protection but can be followed by anterograde administration.
- CPB allows for systemic cooling to 18–32°C to reduce the metabolic rate and protect vital organs. Greater depth of cooling is associated with increasing risk of coagulopathy and platelet dysfunction.

Cardiac anaesthesia: routine elective surgery

Procedure	CABG/valve surgery
Pain	+++/++++
Position	Supine ± crucifix
Blood loss	X-match 4 units
Practical techniques	ETT, IPPV, arterial line, large-bore peripheral IV access and/or vascular sheath, CVC with pressure monitoring, urinary catheter, temperature monitoring, CPB, TOE

The following approach to anaesthesia can be applied to any patient having cardiac surgery. There are additional considerations for valve, or complex cardiac, surgeries and these points of difference are outlined subsequently in this chapter.

Perioperative

- Insert large-bore peripheral venous access and arterial lines pre-induction (check whether the surgeon is harvesting radial grafts). Preoxygenate, administer 5–10 micrograms/kg fentanyl and gently induce with volatile or IV anaesthetic agents. Paralyse with an NMBA. Intubate and maintain anaesthesia with volatile anaesthesia or TIVA (no significant difference in mortality at 2y with either group),[4] and ventilate with a mixture of O_2-enriched air.
- A CVC (3–4 lumens) is then inserted and will provide sufficient access in the presence of a large-bore peripheral line. A vascular sheath can be added as a volume line or a conduit for a PAFC.
- PAFC may be indicated for LVEF <30%, mitral valve surgery, severe pulmonary hypertension and pulmonary hypertension with severe RV dysfunction—essentially, any situation where cardiac output or PAPs are likely to change rapidly or unpredictably.
- Many centres routinely use TOE for intraoperative monitoring.
- Five-lead ECG monitoring (lead II for rhythm, lead V_5 for ischaemia). Consider external defibrillator pads for significant left main stem disease as these patients have an ↑ risk of arrhythmia.
- Nasopharyngeal and urinary catheter temperature probes.
- Depth of anaesthesia monitoring (CPB is associated with ↑ risk of awareness).[5]
- Prophylactic antibiotics are given prior to skin incision.
- Periods of apnoea are often requested prior to CPB (sternotomy and internal mammary artery harvest).
- Aim to maintain cardiovascular stability; anticipate hypertensive surges, and treat appropriately. A repeated dose of fentanyl is often administered prior to sternotomy.
- A vasopressor infusion is usually connected to the CVC and titrated to maintain a MAP >65mmHg.

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- Blood conservation strategies are employed: cell salvage, minimal IV fluid prior to CPB to avoid haemodilution and infusion of an antifibrinolytic agent (usually tranexamic acid) to reduce blood loss.[6]
- Baseline bloods: ABG, ACT ± point-of-care TEG® prior to, or shortly after, commencing surgery.

Establishing cardiopulmonary bypass

- The patient is anticoagulated with 300–400 units/kg heparin (administered centrally to minimise risk of delivery failure), aiming for an ACT of >480s.
- Aim for systolic BP <100mmHg for aortic cannulation to minimise the risk of dissection.
- The venous cannula is placed directly into the IVC or right atrium and the patient is connected to the CPB pump with extreme care to avoid any air bubbles in the lines.
- Careful closed-loop communication is used to initiate CPB by removing the venous clamp at the pump and allowing drainage into the reservoir. The pre-primed pump returns blood to the circulation via the aortic cannula. Once the pump is at 'full flow', the ventilator is turned off. Amnesia during bypass is provided by volatile anaesthetics used by the perfusionists or an infusion of propofol may be commenced.
- Drugs may be redosed at this time, including antibiotics (to account for ↑ volume of distribution) and NMBA.
- Blood gases and ACT are checked every 30min.

Surgery is carried out with readministration of cardioplegia as required. Typical procedures include CABG, mitral valve repair/replacement and aortic valve replacement. Procedures may be combined, depending on the severity of concurrent disease (e.g. CAD with moderate aortic valve disease) in order to prevent a redo-sternotomy in the future and potentially to avoid complications in the perioperative period where concurrent valvular disease may become clinically significant.

Separation from cardiopulmonary bypass

- Once the procedure has been completed, the team prepares for separation from CPB.
- After the aortic cross-clamp has been removed, the perfusionist progressively clamps the venous line, reducing the flow through the CPB pump and allowing the heart to do some work and contribute to cardiac output.
- It is essential to ensure the following:
  - Core temperature is >36°C.
  - ABG parameters are normalised (correct ↑ K^+ and ↓ Ca^{2+}).
  - The heart has a rhythm and rate compatible with life (may require defibrillation or pacing)—aim for HR of 70–100bpm.
  - Ventilation is recommenced (turn on the volatile agent if using for maintenance) and machine alarms are activated.
  - Vasopressors are running and volume status is assessed.
- Cardiac function can be assessed using TOE: filling, contractility and valvular function. If a valve has been repaired or replaced, it is interrogated after separating from CPB but before protamine, with the option of reinstating CPB if required.

- Active recruitment of collapsed lungs is helpful before reinstating ventilation.
- It is usual practice to come off bypass relatively underfilled, with the perfusionist transfusing 100mL boluses as guided to ensure ventricular filling. If cardiac output is impaired, inotropy can be added.
- Once the patient is stable following separation from bypass and there are no concerns regarding replaced or repaired valves, protamine is administered to reverse the anticoagulant effect of heparin (1mg for each 100 units of heparin). ⚠ Ensure the team has been informed beforehand and the perfusionist turns off the pump suckers.
- Protamine can cause systemic hypotension and pulmonary hypertension and should be administered slowly to minimise these effects.
- Once the volume from the pump has been returned to the patient's systemic circulation, the aortic cannula can be removed (take care to keep the systolic BP <100mmHg).
- ACT, TEG® and ABG are repeated to guide transfusion and cell-salvaged blood is transfused. Desmopressin 0.3 micrograms/kg may be given to support platelet function.
- Once haemostasis is satisfactory, haemodynamics are stable and the chest is closed, the patient is transferred, with ETT *in situ*, to ICU.

Postoperative

- The patient is transferred to the cardiac ICU after surgery. Once the patient is warm and stable, and any bleeding from the chest drains is within unit protocols, the sedation is weaned to facilitate extubation. The following criteria should be met:
  - CVS: MAP >65mmHg, with stable haemodynamics; pacing reliable or patient's own native sinus rhythm established
  - Respiratory: O_2 weaned to ≤28%; adequate minute ventilation on a spontaneous breathing ventilator mode
  - Metabolic: acid–base status normal; a very mild increase in lactate is common in the hours immediately post-surgery, but a significant increase could indicate poor cardiac output
  - Coagulation: check FBC + coagulation test ± TEG®/ACT; blood loss should be <100mL/h. Patient normothermic
  - Neurological: able to follow commands.
- PCA fentanyl/morphine is administered for analgesia.
- Some centres use regional anaesthetic techniques in a multimodal analgesic approach to minimise systemic opioids.

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Surgery-specific considerations

For the following specific procedures, the same considerations apply as for routine cardiac surgery, with the addition of the following points.

Off-pump coronary artery bypass grafting

Advantages of off-pump coronary artery bypass grafting (OPCABG) are perceived to be avoiding manipulation of the great vessels, therefore reducing the risk of atheroma embolisation, and avoiding the physiological insult of CPB.

Perioperatively

- Line placement is the same as on-pump CABG.
- Active warming device(s) are required to maintain normothermia.
- The surgeon performs bypass grafts on a beating heart aided by stabilising and suction devices (Octopus® and Starfish® devices) to position the heart and isolate the target vessel. Intracoronary shunts help minimise intraoperative ischaemia.
- Significant haemodynamic instability can occur with lifting of the heart, particularly to access the posterior heart for grafting the left circumflex or crux of the right coronary artery.
- Techniques to counter BP changes: vasopressors (phenylephrine may allow more rapid titration than noradrenaline); crystalloid to augment preload; positioning of the bed head down/up to increase/reduce venous return; atropine to prevent vagally mediated bradycardia; low-dose inotrope to support severe LV impairment; IABP for left main stem disease.
- Heparin 150 units/kg is given, aiming for an ACT of ~300. Reversal of heparin at the end varies by surgeon preference.
- TOE useful for pre- and post-graft assessments. Images often poor intraoperatively due to anatomical distortion. Refractory hypotension may be due to severe MR when heart lifted.
- Be prepared to transition rapidly to bypass if patient is unstable. Rate of conversion from off- to on-pump CABG varies in publications (1–15%).[7] Emergency conversion (after distal anastomoses have been started) is associated with ↑ morbidity and mortality.

Postoperatively

- OPCABG may result in a ↓ blood transfusion rate, compared with CABG, as well as ↓ length of ICU stay. OPCABG is associated with a lower rate of successful revascularisation, although this is not associated with an ↑ risk of postoperative MACE.
- No reduction in CVE has been demonstrated in trials comparing OPCABG to on-pump CABG.[7,8]
- Debate remains regarding long-term mortality outcomes for OPCABG vs on-pump CABG, with some analyses reporting no difference and others showing a small, significant trend towards a mortality benefit at >5y for on-pump CABG.[7]

Emergency coronary artery bypass grafting

This patient group may include unsuccessful PCI with ongoing ischaemia, persistent acute coronary syndrome not amenable to PCI and coronary artery dissection as a complication of coronary angiography or PCI.

Perioperatively

- Patients will have a recent history of DAPT administration and are at high risk of bleeding.
- Patients may have intravascular access from the catheter lab (arterial line, vascular sheath) or the presence of an IABP.
- Patients often present in cardiogenic shock.
- Central venous access may be placed prior to induction to facilitate administration of inotropes.
- Induction strategies include: midazolam 5–10mg, etomidate (reduced dose 0.05–0.2mg/kg) or ketamine 0.5–1mg/kg, supplemented with cautiously titrated fentanyl (these patients are highly dependent on sympathetic drive).
- Progression to CPB will depend on patient stability after induction.
- Left internal mammary artery is often preferred as a conduit for severe left anterior descending artery disease and needs to be harvested prior to CPB, but if the patient is unstable, then grafting with vein conduit only may be appropriate. Vein can be harvested while on bypass.

Postoperatively

- Platelet mapping (see ➲ p. 287) should be carried out in addition to standard POCT to assess platelet function, and there should be a low threshold for platelet transfusion.
- ↑ risk of AKI due to contrast load and the patient's critical status.
- Patients have a higher likelihood of requiring inotropic or mechanical support.

Redo surgery

Redo cardiac surgery via a sternotomy is technically more challenging than 1° surgery, with ↑ morbidity and mortality. A multidisciplinary team approach to these specific cases is important as percutaneous interventional cardiology strategies, such as stenting coronary bypass grafts and valve-in-valve procedures, may be an alternative to redo sternotomy.

Preoperatively

- ↑ risk associated with:
  - Patient factors: age >70; DM; COPD; history of CVE; renal failure; NYHA III–IV; severe LV systolic impairment (LVEF <20%)
  - Surgical factors: prosthetic valve endocarditis as indication for redo surgery; urgent surgery; >2 previous sternotomies; mediastinal radiation.
- Identify and treat iron deficiency anaemia preoperatively.
- Review the preoperative planning CT scan for important information about the patency of grafts, aortic pathology and the proximity of cardiovascular structures to the posterior sternum, as well as the extent of adhesions.

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Perioperatively

- Ensure large-bore IV access in case of massive haemorrhage.
- Place external defibrillation pads prior to induction because of ↑ risk of ventricular arrhythmias and difficulty accessing the chest for internal defibrillation.
- Ensure availability of immediate access to PRBCs.
- Femoral or axillary cannulation may precede sternotomy if surgeon considers patient very high risk for re-entry (institution of peripheral bypass allows greater control of haemorrhage/ischaemia but requires sternal re-entry to be performed with systemic heparinisation).
- Sternal re-entry may be complicated by adhesions between the sternum and key cardiovascular structures such as the RV, grafts or great vessels. Injury can cause haemorrhage, ischaemia, arrhythmias or cardiac arrest.
- Longer surgical and CPB time resulting in ↑ risk of transfusion and coagulopathy. POCT is to guide therapy.
- Myocardial protection in redo surgery is more difficult in the presence of a functioning internal mammary artery graft. Supplementing antegrade cardioplegia with retrograde cardioplegia with temporary occlusion of the internal mammary artery graft may be used to overcome this.

Valve surgery

Table 19.2 highlights aspects of patient presentation and management specific to each valve surgery which tailor the standard approach to cardiac surgery for grafting. (See ➲ pp. 120–7 for haemodynamic goals of specific valve lesions.)

Table 19.2 Valve surgery

Operation	Preop	Pre-CPB	Post-CPB
Aortic valve replacement—stenosis	Timing of AVR should balance risk of surgery/living with a prosthetic valve/anticoagulation/need for redo surgery with the development of irreversible cardiac deterioration	Maintain MAP/diastolic pressure at induction to ensure coronary perfusion LVH and diastolic dysfunction common Tachycardia/arrhythmias poorly tolerated due to reliance on the contribution of atrial systole for LV filling Avoid vasodilators which reduce preload Care with central venous access wires causing arrhythmias If single-procedure AVR, progress onto CPB is rapid	Common to require vasopressor after AVR, but less so for inotropes Patients with LVH require adequate filling In severe LVH, there is a risk of SAM post-AVR Sequential A-V pacing is beneficial in LVH to optimise LV filling using atrial kick

(Continued)

Table 19.2 *(Contd.)*

Operation	Preop	Pre-CPB	Post-CPB
Aortic valve replacement—regurgitation	May coexist with aortic pathology (dilation/aneurysm/dissection) Acute severe AR poorly tolerated. Can present with severe dyspnoea, respiratory failure and low CO. Urgent surgery may be required In chronic AR, severe LV dilation (LVESD indexed >2cm/m²) less likely to remodel following surgery, with worse prognosis	LV dilation compensates for severe AR for years before LV systolic dysfunction occurs. LV dysfunction is a poor prognostic sign Anaesthetic agents reduce LV afterload, which is beneficial Faster HR helps promote higher aortic diastolic pressure and forward flow Careful use of vasopressors which increase SVR, worsening regurgitant fraction In moderate or greater AR, an additional cardioplegia strategy is required due to backward flow of antegrade cardioplegia and LV dilation	Sequential A-V pacing and a cautious approach to filling is important for a dilated LV Inotrope support may be needed due to increase in afterload post-AVR
Mitral valve replacement—stenosis	Major cause of mitral stenosis is rheumatic heart disease May coexist with other valve abnormalities Patients may be young at presentation High incidence of AF, severe pulmonary hypertension (raised PAP may be irreversible) and RV dysfunction	Take great care with sedation to avoid reduction in preload, as well as hypercapnia/hypoxia which will worsen pulmonary hypertension Goals at induction similar to AS, but with additional meticulous attention to minimising increases in PAP and protecting the RV Careful use of fluids because further increases in LAP can cause acute pulmonary oedema PA catheterisation useful for monitoring PAP/CO	Ongoing management of pulmonary hypertension required Inotropes commonly required

(Continued)

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Table 19.2 *(Contd.)*

Operation	Preop	Pre-CPB	Post-CPB
Mitral valve repair or replacement—regurgitation	TTE assessment of LVEF will overestimate true LV systolic function in severe MR Acute MR can occur due to sternal trauma or IHD. Acute MR often precipitates acute pulmonary oedema Pulmonary hypertension common	MR tolerated well at induction, with the effect of anaesthesia in reducing SVR improving forward flow Careful use of vasopressors which can increase regurgitant fraction PAFC useful if significant pulmonary hypertension	TOE assessment essential for assessing success of mitral valve repair MVR corrects the low-resistance retrograde ejection route. It is common to need an inotrope post-MVR to support a dysfunctional LV

AF, atrial fibrillation; AR, aortic regurgitation; AS, aortic stenosis; AVR, aortic valve replacement; CO, cardiac output; CPB, cardiopulmonary bypass; HR, heart rate; IHD, ischaemic heart disease; LAP, left atrial pressure LV, left ventricle/ventricular; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVH, left ventricular hypertrophy; MAP, mean arterial pressure; MR, mitral regurgitation; MVR, mitral valve repair or replacement; PAFC, pulmonary artery flotation catheter; PAP, pulmonary artery pressure; RV, right ventricle/ventricular; SAM, systolic anterior motion (SAM) of the anterior leaflet of the mitral valve; SVR, systemic vascular resistance; TOE, transoesophageal echocardiograph; TTE, transthoracic echocardiography.

Deep hypothermic circulatory arrest

- Deep hypothermic circulatory arrest (DHCA) is used in cases where complete cessation of the circulation is required to enable a bloodless field for the surgeon to complete the operation. The basis of this technique predates the invention of CPB and was the 1° technique for undertaking cardiac surgery in parts of the USSR for several decades.[9] It is used primarily for aortic arch surgery and pulmonary endarterectomy.
- The metabolic requirements of the tissues, and most crucially the brain, are significantly reduced by hypothermia. Traditionally, the patient was cooled to 18°C to afford maximal cerebral protection during the cessation of circulation. While this is a widely accepted practice, it may be more accurate to cool the patient until an isoelectric EEG is obtained on modified EEG monitoring. There is unlikely to be any further reduction in cerebral O_2 requirements once an isoelectric EEG is obtained. It is unpredictable in any given patient at what temperature an isoelectric EEG is obtained.
- The safe period for cessation of the circulation is again unpredictable between patients but is generally thought to be up to 40min in the presence of an isoelectric EEG.[10] Certainly, circulatory arrest times of over 40min have been associated with ↑ CVE rates.[11] The advent of widely available cerebral oximetry monitoring may provide extra information as to the safe duration of circulatory arrest.
- In cases expected to require >40min before the circulation can be recommenced or in cases where the cerebral oximetry values fall by >20% relative to baseline, it is possible to instigate selective antegrade perfusion of the head and neck vessels, which allows perfusion of the brain without affecting the surgical field. This may be superior to DHCA alone in protecting the brain, although high-quality RCT evidence is currently lacking.
- DHCA has multiple other effects, apart from reducing the O_2 consumption of the organs. It has profound effects on the coagulation system, and transfusion is frequently required following circulatory arrest. Also, crucial to ensuring a good outcome for the patient is careful rewarming. A slower rewarming rate may be associated with improved cognitive performance at 6w.[12]

Ascending aorta and aortic arch surgery

- Can be elective surgery to treat aneurysmal disease of the ascending aorta, or acute surgery to treat a dissection of the ascending aorta.
- Arterial hypertension and connective tissue disease are the most prominent risk factors for aortic syndromes.
- Complicated surgery that is generally performed in specialist centres, although, dependent on geographical restrictions, most units may need the ability to deal with acute dissections.
- While DHCA is not mandatory for all aortic arch surgery, it is probably the most commonly used technique (see ➔ pp. 599–600).
- Branch-first aortic arch surgery has been described,[13] which allows replacement of the aortic arch without DHCA.
- The following principles apply to both acute and elective thoracic aortic surgery, with acute surgery representing the higher risk type of surgery.

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Perioperatively

- Acute aortic (Stanford type A) dissection is a cardiac surgical emergency and the patient can present in extremis.
- Depending on the anatomical location of aortic pathology, tamponade, cardiogenic shock, myocardial ischaemia, visceral ischaemia or arch vessel occlusion may be present.
- Without surgery, mortality can be as high as 2% per hour, so expedient care is imperative.[14]
- For acute cases, aggressive preoperative BP control is important, including the use of antihypertensive infusions (GTN/labetalol/esmolol), to prevent progression of dissection.
- The following monitoring is required:
  - Nasopharyngeal temperature (representing brain temperature)
  - Core temperature measurement (often urinary catheter)
  - Modified EEG monitoring (BIS™/Entropy™)
  - Cerebral oximetry—should be available
  - Potentially >1 arterial line, dependent on planned CPB cannulation sites—aiming to monitor proximal (right radial) and distal aortic arch flow (left radial/femoral).
- Avoidance of hypertension during laryngoscopy and intubation is essential to prevent aortic rupture.
- Peripheral bypass cannulae may be placed prior to sternotomy, depending on aortic pathology. TOE can be used to confirm wire placement within the true lumen of the aorta during femoral arterial cannulation.
- When DHCA is instituted, drug infusions are stopped.
- On rewarming, infusions are recommenced.
- Consider running a heparinase TEG® once core temperature reaches 36°C to guide transfusion (cryoprecipitate and FFP take time to thaw).
- Inotropic support is often required to allow separation from CPB.
- These patients are at high risk of bleeding.

Postoperatively

- These patients are at high risk of coagulopathy and neurological disturbance or injury. Prolonged intubation after these procedures is common.

Intra-aortic balloon pump

- An IABP is inserted percutaneously in awake or asleep patients.
- A balloon catheter is positioned with its tip 1–2cm distal to the left subclavian artery in the thoracic aorta.
- The balloon is inflated with helium in early diastole following closure of the aortic valve, thereby augmenting aortic diastolic pressure and coronary blood flow. It then rapidly deflates at the onset of systole during isovolumetric contraction, reducing the impedance to LV ejection when the aortic valve opens.
- Via these mechanisms, it improves the myocardial O_2 supply–demand ratio, increases CO and supports the aortic Windkessel mechanism.
- The balloon may be triggered by ECG or the arterial pressure wave form.[15]
- Indications:
  - Complications of an AMI, including cardiogenic shock
  - Acute post-infarct MR
  - Ventricular septal rupture
  - Ongoing severe unstable angina
  - Refractory ventricular arrhythmias
  - Failure to wean from CPB
  - Low CO syndrome following cardiac surgery.
- Contraindications:
  - Moderate or greater AR
  - Aortic dissection or significant aortic aneurysm
  - Severe peripheral arterial disease
  - Severe coagulopathy
  - Uncontrolled sepsis.
- The position of the balloon may be checked on CXR, with fluoroscopy or with TOE.
- Patients need to be monitored for vascular complications (e.g. leg ischaemia, kidney injury, direct femoral vessel injury). It should not be left in standby mode for >20min due to risk of thrombus formation.
- Literature is inconclusive (available studies criticised for being underpowered), but potential survival benefit with preoperative IABP placement in high-risk patients undergoing CABG (EF <30% plus one or more additional risk factors: reoperation, unstable angina, preoperative use of IV GTN, left main stem stenosis, AMI within the previous 7d, non-elective operation, NYHA III–IV symptoms).[16,17]

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Cardiac surgery, pulmonary hypertension and the right ventricle

(See ➔ p. 139.)

- Left heart disease is the commonest cause of pulmonary hypertension. Raised left atrial pressure is transmitted retrogradely to the pulmonary veins, capillaries (isolated post-capillary pulmonary hypertension) and finally pulmonary arteries (combined pre- and post-capillary pulmonary hypertension).[18]
- Cardiac surgical patients with mitral valve disease have a high incidence of severe pulmonary hypertension. However, pulmonary hypertension can develop 2° to long-standing LV systolic or diastolic dysfunction due to any cause. Pulmonary hypertension is less common but also possible with aortic valve disease.
- Patients with moderate or severe pulmonary hypertension undergoing cardiac surgery are at ↑ risk of morbidity and mortality.[19]
- The clinical significance is that the RV has limited capacity to compensate for raised PAP or acute increases in afterload. When the RV fails, it dilates, and via interventricular dependence, the intraventricular septum shifts towards the LV. ↑ RV systolic pressure reduces the gradient for RV coronary perfusion, risking RV subendocardial ischaemia and further dilation. LV preload is then compromised with reduced CO and a patient's haemodynamics can rapidly become unstable.[20]
- RV systolic function is often transiently reduced following cardiac surgery. Acute RV failure can occur in the perioperative period. Patients with moderate to severe pulmonary hypertension are at higher risk, especially if they have preoperative RV dysfunction. Pulmonary hypertension may be under-represented in moderate to severe RV dysfunction where the RV cannot generate a high PAP. In these cases, measuring the PVR may reveal the extent of the problem.
- Pulmonary artery systolic pressure (PASP) is estimated non-invasively using transthoracic echocardiography by measuring RV systolic pressure from the tricuspid valve regurgitation jet and then adding right atrial pressure (RAP): ($PASP = RAP + 4(Vtr)^2$).
- The gold standard for measuring PAP and PVR is a right heart catheter study.
- The goals for managing pulmonary hypertension and RV dysfunction are:
  - Maintenance of RV coronary perfusion pressure by maintaining SVR/MAP and thereby optimising aortic diastolic pressure
  - Minimising increases in RV afterload
  - Keeping $PaCO_2$ low normal and avoiding hypoxia
  - Using a ventilator strategy that limits plateau and driving pressure (avoid high V_T and PEEP)
  - Pulmonary vasodilators can be used to reduce PAP/RV afterload.

- Inhaled pulmonary vasodilators[19] avoid systemic hypotension and worsening V/Q mismatch. NO 5–40ppm requires a proprietary delivery system, but the following agents can be given to an awake patient via a Hudson mask nebuliser or to intubated patients via the ETT using an in-line ultrasonic nebuliser device:
  - Iloprost (prostacyclin analogue) 25–50 micrograms every 2–4h
  - Milrinone 2–5mg every 1–2h
  - GTN 5mg (very short-acting).
- Improve RV inotropy with low-dose adrenaline or an inodilator such as dobutamine or milrinone.
- Maintain sinus rhythm/AV synchrony.
- TOE used to assess RV volume status.
- Correct acidosis.
- A PA catheter can be helpful to monitor the impact of treatment on PAP and CO.

Table 19.3 Grading systems for pulmonary hypertension

	Mild (mmHg)	Moderate (mmHg)	Severe (mmHg)
Estimating PH using TTE[21]	PASP 35–45	PASP 46–59	PASP >60
Grading PH on right heart catheter study[22]	mPAP 25–40	mPAP 41–55	mPAP >55

PVR >3WU indicates raised PVR and more severe PH.

mPAP, mean pulmonary artery pressure; PASP, pulmonary artery systolic pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; TTE, transthoracic echocardiography; WU, Wood units.

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Controversies in cardiac surgery

Aprotinin

Aprotinin is a serine protease inhibitor that has been proven to reduce blood loss in cardiac surgery when compared to placebo. Concerns regarding an ↑ incidence of renal dysfunction and mortality[23] in patients who had received aprotinin led to its withdrawal from the market in 2007. In 2012, the EMA reversed this decision and aprotinin was relicensed for low-risk patients.

There is much debate about the current role of aprotinin in clinical practice. Strong evidence for the benefit of tranexamic acid in reducing blood loss during cardiac surgery[24] has probably led this to be the 1st-line antifibrinolytic in most cardiac units, at the expense of an ↑ postoperative seizure rate.

However, contrary to the EMA's decision to license for only low-risk patients, there may be a place for aprotinin use in the patient who is at very high risk of bleeding, and in the situation where the risk of postoperative seizure is high and therefore use of tranexamic acid carries with it particular risk.

Transfusion threshold

Transfusion threshold in cardiac surgery has been a much-debated topic in the literature for several years. The basic conflict is that the myocardium has a high O_2 extraction ratio. Therefore, maintaining a higher Hb level is attractive in promoting O_2 supply in a situation where it may be constricted by coronary artery stenosis. However, this theoretical benefit is offset by the theoretical disadvantage of suppression of the immune system with autologous blood transfusion and therefore increasing the chance of local or systemic infection.

After a number of flawed retrospective studies, two large prospective studies have attempted to resolve this question. TiTRE2[25] enrolled patients postoperatively to transfuse or not transfuse if the patient hit a certain Hb target. The TRICS 3[26] trial enrolled and randomised patients preoperatively to either a restrictive (transfusion if Hb reaches 70) or a liberal transfusion strategy (transfusion if Hb falls to 90) during the entire intraoperative and postoperative period.

While the subtleties of these different approaches and the slightly divergent results of these trials (in TiTRE2, the higher Hb patients tended to do better, and in TRICS, the lower Hb patients tended to do better) can be debated, the take-home message is probably the same. Having a transfusion threshold of 70g/L of Hb is mostly likely to be non-inferior to having a higher transfusion threshold.

Steroids

There have been two notable large studies into the use of steroids in cardiac surgery, both of which have shown no statistically significant difference in mortality between high-dose steroids (1mg/kg dexamethasone[27] and 500mg methylprednisone)[28] and placebo. However, despite this, many units continue to routinely use steroids in their clinical practice. The SiRS trial came close to showing a statistically significant difference in terms of mortality (5% vs 4%; $p = 0.19$),[28] and the DeCS trial showed a reduction in infective complications in patients receiving steroids.[27]

There may still be some benefit to some patient-centred outcomes such as quality of recovery with the use of steroids in cardiac surgery. However, it is unlikely that there is a benefit with regard to major complications such as mortality, CVE or MI following cardiac surgery.

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Chapter 20

Thoracic surgery

Charlotte Earnshaw and **Kajan Kamalanathan**

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General principles

Successful thoracic anaesthesia requires the ability to control ventilation of the patient's two lungs independently, management of the shared lung and airway and a clear understanding of planned surgery.

Long-term smoking, bronchial carcinoma, pleural effusion, cardiac disease, oesophageal obstruction and cachexia are all common and can significantly reduce the cardiorespiratory physiological reserve.

General considerations

- Discuss planned procedure and potential problems with the surgeon.
- Smoking cessation, optimising respiratory function and cardiovascular fitness in preparation for surgery reduce perioperative morbidity.
- Place patients on an ERAS pathway at time of referral (see pp. 44).
- The lateral decubitus position, with the operating table 'broken' to separate the ribs, is used for the majority of procedures.
- Increasingly, cases are done by video-assisted thoracoscopic surgery (VATS).
- Postoperative mechanical ventilation stresses pulmonary suture lines and increases air leaks and risk of chest infection; avoid, if possible.
- Minimise respiratory dysfunction by providing good analgesia and physiotherapy. Patients go to HDU/specialist ward postoperatively.
- Postoperative O_2 is routine to compensate for ↑ V/Q mismatch. Warmed humidified 40% O_2 via a face mask is recommended after pulmonary surgery.

General preoperative assessment considerations

- Pay attention to functional status and cardiorespiratory reserve.
- Patients with significant cardiac disease form a high-risk group.
- Discuss results of CXR and CT scans with the surgeon, focusing on airway problems making DLT placement problematic, tumours impinging on the chest wall and crossing fissures or vessels.

Preoperative assessment for lung resection

Risk assessment is based upon history, examination and assessment of functional status, in combination with spirometry, tests of diffusion capacity (e.g. DLCO) and calculation of Thoracoscore.

- The Thoracoscore gives an estimate of mortality risk based on a number of variables (e.g. demographics, degree of dyspnoea and comorbidities, performance status and type of surgery). It is especially useful in the consent process. A multidisciplinary team approach is essential and should involve the patient, anaesthetist, surgeon, clinical nurse specialists, respiratory physician and radiologist.
- Patients may be classified as:[1,2]
  - Clinically fit—good exercise tolerance, normal spirometry: accept for surgery
  - Major medical problems, minimal exercise capacity, grossly impaired PFTs: consider alternative treatment
  - Reduced exercise capacity (shortness of breath climbing two flights of stairs) and abnormal spirometry, with or without moderate coexisting disease: further assessment and careful evaluation of risks/benefits of surgery.

- PFTs (see ➲ p. 164) are often used to determine suitability for lung resection surgery by estimating the postoperative lung function. Put the results in the context of the patient's general health and proposed operation.
- Spirometry should be performed in addition to tests of diffusion capacity (e.g. DLCO). Patients with diffuse alveolar lung disease can have severely impaired gas transfer with relatively normal spirometry.
- Predicted postoperative (ppo) value of the PFT results is calculated by the following formula: ppo = preoperative value × (19 – number of segments resected)/19.
- If preoperative DLCO or ppoFEV_1 is <40% predicted normal, the patient should undergo CPET prior to surgery.
- CPET assesses VO_2max which is used to inform the risk of perioperative morbidity and mortality. Patients with a VO_2max 10–15mL/kg/min are higher risk and should have postoperative HDU admission. VO_2max <10mL/kg/min are very high risk and surgery may not be appropriate—further discussion is needed with the multidisciplinary team and with the patient.
- All patients undergoing pneumonectomy or bilobectomy should have CPET, echocardiography and a postoperative HDU admission. Ventilation scans may be used to assess for non-functional lung (e.g. atelectasis beyond an obstructing tumour). All patients with an active cardiac condition should undergo cardiology review.

Analgesia

- Chronic pain syndrome after thoracic surgery occurs in 25–60% of patients and the risk is ↑ by high-intensity postoperative pain, so optimal analgesia is essential.
- Inadequate analgesia increases the neurohumoral stress response, impairing mobilisation and respiration and increasing complications.
- Combining opioid-sparing agents such as paracetamol, NSAIDs, clonidine, magnesium, ketamine, glucocorticoids and a regional block is recommended.[3]
- Regional anaesthesia typically involves surgically performed internal intercostal nerve blocks alongside a paravertebral catheter for both open and video-assisted thoracotomy (equivalent analgesic efficacy to epidurals, but associated with fewer adverse events and may be better suited to patients taking anticoagulants or in renal failure).
- Continuous postoperative infusion of levobupivacaine 0.375% for 48–72h at 0.1mL/kg/h via the paravertebral catheter is advised.
- Limiting the use of PCAs aids enhanced recovery and VATS procedures are generally less painful.
- Thoracic epidural may be necessary for bilateral procedures (match the level of block to that of the incision—usually T5/6 or T6/7).[4]
- Perineural catheters and regional analgesia into the serratus anterior or erector spinae plane can be considered for single-port VATS and also provide good analgesia for rib fractures (see ➲ pp. 995–6).

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Enhanced recovery after surgery

An ERAS pathway is initiated at referral and follows the patient to discharge. It encompasses multiple elements that, when delivered in combination, improve patient outcome. Attenuation of the stress response and organ dysfunction reduces length of stay and complications.

Preoperative recommendations

- Patient engagement, education and psychological preparation are paramount to improving motivation and compliance with pulmonary rehabilitation programmes and reducing postoperative pain.[5]
- Assessment of nutritional status and weight loss. Prescribe oral nutritional supplements for malnourished or at-risk patients.
- Smoking cessation and reducing alcohol consumption 4w preoperatively reduces pulmonary complications and risk of death.
- Identify anaemic patients and correct iron deficiency. Blood transfusion has been shown to reduce lung cancer survival, but if unavoidable, preoperative transfusion is preferable to intraoperative.
- Prehabilitation programmes for those with borderline lung function or poor exercise capacity improve physiological reserve.
- Avoid starvation and consider carbohydrate loading.
- Minimise postoperative respiratory dysfunction with good analgesia and physiotherapy. Preoperative sedative agents should be avoided.

Perioperative recommendations

- Appropriate antibiotic prophylaxis, skin preparation and temperature monitoring are mandatory. Avoid hypothermia.
- A combination of regional and general anaesthetic techniques permits early extubation. Most volatiles weakly inhibit hypoxic pulmonary vasoconstriction, but with little impact at ≤1 MAC. Lung-protective ventilation should be employed (see p. 170).
- PONV should be avoided (see pp. 442–7). Higher-risk patients should be given multimodal antiemetic prophylaxis.
- Aim for euvolaemia (dry lung) with balanced crystalloid; avoid being over-restrictive (2–3mL/kg/h not been shown to be detrimental).
- Minimally invasive surgery is preferred; avoid external suction on chest tube, if possible.
- Warmed, humidified 40% O_2 to compensate for ↑ V/Q mismatch after pulmonary surgery. O_2 3L/min via nasal cannulae is better tolerated and satisfactory for most other patients.
- Early mobilisation, physiotherapy, incentive spirometry and mechanical and pharmacological VTE prophylaxis are all recommended, if possible.
- New-onset AF or atrial flutter is common postoperatively, with an incidence of 12%. β-blockers should be continued; replace magnesium and consider preoperative diltiazem or postoperative amiodarone. Digoxin should not be used.

Miscellaneous thoracic procedures

(See Table 20.1.)

Table 20.1 Miscellaneous thoracic procedures

Operation	Description	Time (pain)	Position/ approach	Notes
Fibreoptic broncho-scopy	Visual inspection of tracheo-bronchial tree ± biopsy and bronchial brushings/ lavage	5–10min (+)	Supine	GA rarely used. Single-lumen tube (SLT) (8–9mm) with bronchoscopy diaphragm on angle piece. IPPV with relaxant appropriate to duration. Expect high airway pressures while scope in ETT. Suction can empty breathing system. Surgeon may perform via DLT at start of a major case
Lung biopsy	Diagnostic sampling of lung tissue for localised or diffuse abnormality	30–60min (+++/ ++++)	VATS (open procedure becoming rare)	DLT and one-lung ventilation (OLV) facilitate VATS procedures. Patients with diffuse disease can have very poor lung function. Minor blood loss, G&S: X-match if anaemic
Oesopha-goscopy and dilation (O&D)	Visual inspection of oesophagus via rigid or fibreoptic scope ± dilation of stricture	5–20min (–/+)	Supine	Regurgitation risk, so RSI advised. SLT on left side of mouth—watch for airway obstruction and ETT displacement during procedure. Flexible oesophagoscopy often done under IV sedation
Oesophageal stent insertion	Endoscopic placement of tubular stent through oesophageal stricture	10–30min (+/++)	Supine	Often emaciated, may be anaemic. Preoperative IV fluids to correct dehydration. RSI, SLT and awake extubation in lateral position. Small risk of oesophageal rupture

(Contd.)

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Table 20.1 *(Contd.)*

Operation	Description	Time (pain)	Position/ approach	Notes
Fundo-plication/ hiatus hernia repair	'Antireflux' procedure: fundus of stomach wrapped around lower oesophagus, may need gastroplasty to lengthen oesophagus	2–3h (+++/ +++++)	Supine/ laparotomy. Lateral/ left thora-cotomy. Majority done laparo-scopically	Fundoplication patients often obese—check respiratory function. RSI or AFOI mandatory. DLT helpful for thoracic approach. Multimodal and regional analgesia advised. Moderate blood loss, G&S: if Hb <120, X-match 2 units
Pectus excavatum/ carinatum repair	Correction of 'funnel chest'/ 'pigeon chest' deformity of sternum	3–5h (+++/ ++++)	Supine, arms to sides, midline sternal incision	Primarily cosmetic unless deformity severe. Usually young, fit adults. GA, IPPV via SLT. Risk of pneumothoraces. Minimally invasive technique for pectus excavatum repair is becoming increasingly popular. Moderate to severe blood loss, X-match 2 units
Thymectomy	Excision of residual thymic tissue and/ or thymoma from superior and anterior mediastinum	2–3h (++/ +++)	VATS, semi-lateral position	Usually for myasthenia gravis. Check for airway compression, other autoimmune diseases, thyroid function and steroid, immunosuppressive and anticholinesterase therapy (see p. 316). DLT if VATS, IV anaesthesia, small doses of muscle relaxant or use rocuronium/ sugammadex. Likely to need HDU bed postop. Moderate blood loss, X-match 2 units

Isolation of the lungs

Achieving independent ventilation of the lungs is not always straightforward. OLV is associated with complications and should be used only when the benefits outweigh the risks.

Indications for OLV

Absolute indications

Avoid contamination of a lung in cases of infection, massive pulmonary haemorrhage or bronchopulmonary lavage; control the distribution of ventilation in massive air leaks or severe unilateral lung disease (e.g. giant bullae and lung cysts) and for VATS procedures.

Relative indications

Improving access for surgery. If isolation proves difficult, communicate with the surgeon as lung retraction may be an alternative.

Techniques

Double-lumen endobronchial tubes (DLTs)

- DLTs are the most common and most versatile approach.
- DLTs are described as 'right' or 'left', according to the main bronchus they are designed to intubate.
- Right-sided tubes have a hole in the wall of the endobronchial section (Murphy's eye) to facilitate ventilation of the right upper lobe.
- Sizes of plastic DLTs are given in Charrière (Ch) gauge (equivalent to French gauge), which is the external circumference of the tube in mm. Thus, a 39Ch tube has an external diameter of about 13mm. Note that the diameter of the bronchial segment of the tubes varies between manufacturers (for the same tube gauge).
- The lumens of DLTs are small, compared with standard SLTs. The IDs of the lumens of the 39 and 35Ch Broncho-Cath® DLTs are only 6.0 and 4.5mm, respectively.
- Bronchoscopic placement and checking require a narrow scope (<4mm in diameter).

Types of double-lumen endobronchial tube

- Carlens' (left-sided): has a carinal 'hook' to aid correct placement.
- White's (right-sided): has a carinal hook and slit in the tube wall.
- Robertshaw (right- and left-sided): D-shaped lumens; traditionally a red rubber, now available as single-use in small, medium and large.
- Single-use PVC (right- and left-sided): high-volume, low-pressure cuffs; bronchial cuff and blue-coloured pilot tube; radio-opaque marker stripe running to the tip of the bronchial lumen; available in sizes 28–41Ch, e.g. Mallinckrodt®, Broncho-Cath® and Sheridan®.

Right or left?

- Right-sided DLTs are harder to place due to the fact that the Murphy's eye needs to be placed over the right upper lobe bronchus. This means there is less margin for error. The right-sided DLTs absolutely need a fibreoptic bronchoscope check for positioning. Right-sided tubes are required for surgery on the left main bronchus, obstruction of the left main bronchus or a short left main bronchus.

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- Left-sided DLTs are easier to place and more commonly used. They can be used for most cases, although for VATS major lung resections, the opposite side tube to the side of surgery is preferred. This avoids trauma from the end of the bronchial portion of the tube.

Placement of double-lumen endobronchial tube

- Assess the risks/benefits of using a DLT.[6]
- Check the DLT prior to use, including checking both cuffs and that all connections fit together appropriately, including the Y-connector.[7]
- Most plastic DLTs are supplied with a malleable stylet which can be used to adjust the curve of the tube to facilitate intubation.
- Commence intubation with the concavity of the endobronchial section of the DLT facing anteriorly. Once the tip is past the glottis, withdraw the stylet and rotate the tube 90–180° to bring the oropharyngeal curve into the sagittal plane. Gently advance while bringing the DLT back into a neutral position.
- Advance the tube to around 29cm, which is the average depth for patients who are 170–180cm tall. There is a 1cm change in depth for every 10cm variation in the patient's height from this position.[8]
- Do not simply advance until resistance as this could result in trauma.
- At this stage, treat the DLT as an ordinary ETT. Inflate the tracheal cuff, check for $ETCO_2$ and confirm placement in both lungs.
- The diameter of a DLT makes intubation more difficult than with a standard tube, even with a good view of the larynx. Despite a grade 1 view of the larynx, the DLT can end up in the oesophagus, so vigilance should be maintained throughout.
- Bougies can be used, but check that they are compatible with a DLT. Other useful equipment for a difficult DLT are a VL and an airway exchange catheter (AEC).

Confirmation of double-lumen endobronchial tube position (left)

- Clamp the tracheal lumen on the Y-connector and open the port to feel for a leak. Look at the movement of the chest and check if there is appropriate unilateral expansion.
- Inflate the bronchial cuff until the leak disappears. Check with the DLT manufacturer the maximum amount of air that can be placed into the cuff. Continue to observe movement of the chest and that it is unilateral and appropriate for the side that has been clamped.
- Another method to check that the DLT is in the correct position is to auscultate over the side you wish to isolate. Listen while placing the clamp on and inflating the bronchial cuff; if correct, the chest sounds should disappear.
- Next, confirm it is possible to isolate and achieve OLV of the opposite (operative) lung via the tracheal lumen by clamping the opposite side on the Y-connector.
- ETTs often move when the patient is placed in the lateral position. Recheck isolation and OLV once in position and before surgery.

Fibreoptic bronchoscope

- Ideally, the position of every DLT should be checked bronchoscopically. At the very least, a suitable bronchoscope must be immediately available to assess DLT placement if there are clinical problems with the tube or with OLV.
- The position of a right-sided tube should always be checked with a bronchoscope to ensure correct position of the Murphy's eye.
- It may be necessary to use the bronchoscope to help intubate the correct bronchus and then railroad the tube over the bronchoscope. Several bronchoscopic studies have shown that up to 80% of DLTs are malpositioned to some extent, even when clinical signs are satisfactory. The upper surface of the bronchial cuff (blue) should lie just below the carina when visualised via the tracheal lumen.

Bronchial blocker technique

- Bronchial blockers (Univent™ tube or Arndt endobronchial blocker) are useful in patients who are difficult to intubate, have distorted tracheobronchial anatomy/tracheostomy and occasions when isolation of a lobar bronchus is required (localised bronchiectasis or haemorrhage, lung abscess, bronchopleural fistula, previous lung resection and poor tolerance of OLV).[9]
- A balloon-tipped catheter ('blocker') is manipulated through an SLT into the appropriate main (or lobar) bronchus with the aid of a narrow fibreoptic bronchoscope.
- Good lubrication of both the bronchoscope and blocker is essential.
- The position of the blocker should be rechecked after the patient has been positioned for surgery. They move out of position easily.
- Placement is usually straightforward in the supine position but can be awkward in the lateral position.
- The lung or lobe is isolated from ventilation by inflating the balloon within the bronchus. The lung slowly collapses, as the trapped gas is absorbed or escapes via the blocker's narrow central lumen.
- Collapse can be accelerated by ventilating with 100% O_2 for a few minutes and then inflating the blocker at end-expiration when lung volume is at its minimum.
- Reinflation of the collapsed lung requires deflation of the blocker and consequent loss of isolation of the lungs. A DLT will maintain separation of the airways to each lung until extubation.
- During pneumonectomy or sleeve resection (bronchial reanastomosis), the blocker has to be withdrawn to allow surgical access to the bronchus.
- There are two modern forms of bronchial blocker: Univent™ tube (SLT with an internal channel containing an adjustable blocker bearing a high-volume, low-pressure cuff) and Arndt wire-guided endobronchial blocker (Cook™). This is a stiff catheter with a cylindrical cuff and an adjustable 'wire' loop at its tip to guide the blocker along the fibreoptic bronchoscope into the required bronchus (special adapter allowing deployment through a conventional single-lumen or cuffed tracheostomy tube).

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Management of one-lung ventilation

A V/Q mismatch is created when a patient is put onto OLV leading to a shunt. Patients tend to be turned lateral, allowing the weight of the mediastinum and abdominal contents to reduce the FRC. The Bohr effect (O_2 being released from Hb) can occur due to the physiological changes due to a raised CO_2 and resulting respiratory acidosis.[10,11]

Initiating one-lung ventilation

- Start with typical ventilator settings you would use during two-lung ventilation.
- Increase FiO_2 to 0.5–1.0 before initiating OLV. Note the airway pressure (P_{aw}) generated by this V_T.
- Clamp the Y-connection to the operative (non-dependent) lung, and open the sealing cap on that lumen of the DLT to allow the gas to escape.
- If correctly isolated, the V_T should drop to around two-thirds of what it was when not isolated.
- If P_{aw} is excessive (>35cmH_2O) or rises abruptly with each inspiration, exclude mechanical causes (e.g. kinked connector, clamp incorrectly placed) and DLT malposition or obstruction (e.g. ventilating the lobe, rather than the lung, sputum plugs, opening of the tracheal lumen against the wall of the trachea).
- Adjust V_T and ventilator profile to limit the P_{aw} to ≤35cmH_2O, and ideally to ≤30cmH_2O. Incidence of ALI is reduced by employing a 'protective ventilation strategy': lower P_{aw}, PEEP 5–10cmH_2O.
- Some monitoring systems allow you to compare the spirometry loop before and during OLV.
- Observe SpO_2 and $ETCO_2$ closely. If necessary, increase the ventilatory rate to maintain an acceptable minute volume and CO_2 clearance.
- Check with the surgeon that the lung is collapsing (may take a few minutes in patients with obstructive airways disease) and that the mediastinum has not 'sunk' into the dependent hemithorax. It is at these points where good communication with the surgeon is essential.
- If the lung fails to come down, gentle suction via a DLT suction catheter may help.

Hypoxia on one-lung ventilation

- Hypoxia is a frequent complication of OLV.[12]
- It usually occurs after a few minutes of OLV (as O_2 in the non-ventilated lung is absorbed).
- SpO_2 dips but then often rises again, due to hypoxic pulmonary vasoconstriction as blood flow through the non-ventilated lung decreases.

Actions: the ventilated lung

- Increase the FiO_2 and hand-ventilate with 100% O_2; this is to ensure the circuit is intact and delivering O_2 which will exclude circuit disconnection and O_2 failure. It will also help with checking compliance of the ventilated lung and if there are any secretions.
- Increase the minute ventilation.

- Check the tube position with a fibreoptic scope and make sure that the tube has not moved to obstruct the upper lobe.
- Suction out any secretions.
- Maintain perfusion with fluid and vasopressors.
- Apply PEEP to the ventilated lung, but be aware this can worsen the shunt.

Actions: the non-ventilated lung

- Insufflate O_2 via a suction catheter.
- Apply CPAP via a separate circuit to the non-ventilated lung.
- Requires discussion with surgeon as will make operative view more difficult, which is particularly a problem during VATS.
- Clamp the PA, but this is only really an option near completion of a pneumonectomy.

Returning to two-lung ventilation

- Gently suction the non-ventilated lung to clear any blood or pus—use the long suction catheters supplied with the DLT.
- Close the sealing cap on the lumen to the non-ventilated lung and remove the clamp on the Y-connector.
- Switch to manual ventilation and reinflate the collapsed lung under direct vision. Long, sustained ventilation breaths are effective, and inflation pressures of up to 30cmH_2O are often required to fully re-expand all areas of the lung.
- The surgeon will commonly observe for air leaks at this point and may ask for a specific P_{aw} to be generated.
- Return the patient to mechanical ventilation and, unless significant volumes of the lung have been resected, return to the original two-lung ventilator settings and FiO_2.
- Adjust the RR to maintain normocapnia.
- Always be prepared to return to OLV immediately, should problems occur, e.g. large air leak from the operated lung. It is prudent to keep your fibreoptic scope nearby until you are completely satisfied with the lungs.
- Many anaesthetists advocate deflating the bronchial cuff as soon as possible to prevent bronchial wall necrosis.

ALGRAWANY

Rigid bronchoscopy and stent insertion

Procedure	Endoscopic inspection of tracheobronchial tree ± biopsy, stents, removal of foreign body
Time	10–20min
Pain	+
Position	Supine with head and neck extended
Blood loss	Usually minimal
Practical techniques	TCI propofol, alfentanil/remifentanil, rocuronium, sugammadex for high-risk cases IPPV through bronchoscope with O_2 via Venturi needle and Sanders injector Apnoeic oxygenation via HFNO

Preoperative

- Check for airway obstruction: stridor, tracheal tumour on CT scan or history of foreign body inhalation.
- Suitable as a day case procedure in appropriate patients.
- Warn about postoperative coughing, haemoptysis and sore throat.
- Combined with mediastinoscopy to assess suitability for resection.
- The airway will be unprotected, so patients at risk of regurgitation may need premedication with omeprazole or ranitidine.

Perioperative

- Give full preoxygenation and check the jet ventilator is working before anaesthetising the patient.
- It is safer to anaesthetise these patients in theatre with the surgical team ready to go as soon as the patient is anaesthetised.
- Coordinate low-frequency jet ventilation with surgical activity—the surgeons will say when not to ventilate.
- Dexamethasone can be given to reduce airway swelling.

Postoperative

- Sit fully upright as soon as awake.
- A blood clot can cause severe lower airway obstruction, requiring immediate intubation, suction and repeat bronchoscopy.

Special considerations

- A stimulating procedure that can generate a marked hypertensive response.
- Extreme CVS responses need to be obtunded, and profound relaxation provided, but with prompt return of laryngeal reflexes and spontaneous respiration.
- Rarely, a biopsy can precipitate a life-threatening airway bleed.
- Stent insertion can be technically difficult and may involve periodic loss of airway control.

Superior/cervical mediastinoscopy and endobronchial ultrasound

Procedure	Inspection and biopsy of tumours and lymph nodes in superior and anterior mediastinum via small suprasternal or anterior intercostal incision
Time	20–30min
Pain	+
Position	Supine or slightly head up, arms by sides, and head ring with bolster under shoulders
Blood loss	Usually minimal, but potential for massive haemorrhage; G&S
Practical techniques	IPPV via SLT LA and/or propofol TCI for endobronchial ultrasound

Preoperative

- Suitable as day case procedure in appropriate patients.
- Check for SVC obstruction and tracheal deviation or compression due to large mediastinal masses.
- Sometimes preceded by rigid bronchoscopy ('Bronch & Med').

Perioperative

- Ensure the airway is secure as the head will be obscured by drapes.
- Give boluses of IV fentanyl during surgery.
- Insert a 16-gauge (G) cannula in a lower leg vein after induction (see Special considerations, p. 541).
- Beware surgical compression of the trachea (monitor V_T and Paw).

Postoperative

- Paracetamol and NSAID. Usual day case precautions.

Special considerations

- There is the potential for massive haemorrhage from the great vessels. The risk is ↑ in patients with SVC obstruction (hence cannula in the leg). May require immediate sternotomy.
- Mediastinotomy can cause a pneumothorax, recurrent laryngeal nerve injury and VAE.

Endobronchial ultrasound

- An alternative method for mediastinal staging of lung cancer. Can be done with LA ± sedation or GA (TIVA).
- Effectively, this is a flexible bronchoscopy through either a laryngeal mask or a single-lumen ETT, so be prepared for problems with ventilation and you may need to hand-ventilate on relatively high flows.

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Lung surgery: wedge resection, lobectomy and pneumonectomy

Procedure	Excision of pulmonary tissue either selectively (wedge resection or lobectomy) or a whole lung (pneumonectomy)
Time	2–4h
Pain	+++++
Position	Lateral decubitus with table 'broken', elbows flexed to bring forearms parallel to face
Blood loss	100–500mL; occasionally significantly more; G&S
Practical techniques	IPPV via DLT using OLV. Paravertebral regional anaesthesia with catheter for postoperative analgesia, arterial line

Preoperative

- Cancer is the commonest indication for lung resection; others include benign tumours, bronchiectasis and TB.
- Patients require extensive preoperative assessment to assess cardiovascular and functional fitness.
- Large majority of wedge resections and lobectomies now performed by VATS. This can be done with multiple ports or a single port, depending on the case and surgical preference.
- Assess the airway with respect to placement of the DLT.
- Ensure patient education, commence ERAS protocol and plan the postoperative analgesia regime.

Perioperative

- Select the appropriate DLT and check lung isolation carefully after intubation.
- The choice of side of tube to use will be dependent on the case and surgical technique. For VATS for major lung resections, use opposite side tube to side of operation to avoid any trauma to bronchus during surgery.
- Place arterial line for most cases involving OLV.
- OLV facilitates surgery and prevents soiling of the dependent lung.
- Continuous display of the P_{aw}/volume loop is a valuable adjunct to monitoring and managing OLV.
- Surgical manipulation often causes cardiac and venous compression, which reduces the CO/BP and may cause arrhythmias.
- Suction the airway to the collapsed lung prior to reinflation.
- The bronchial suture line is 'leak-tested' under 0.9% sodium chloride by manual inflation to 20–30cmH_2O.
- Use multimodal analgesia as described previously.

Postoperative

- Aim to extubate the patient awake and sitting at the end of the procedure.
- Prescribe continuous humidified supplementary O_2.
- Ensure good analgesia is achieved. Many centres are now using paravertebral analgesia, rather than epidurals, especially for VATS cases. Intercostal blocks can also be placed under direct vision by the surgeons.
- Good regional analgesia may avoid excessive amounts of opioids and PCA may not be required.
- A CXR is usually required postoperatively.

Special considerations

- Occasionally, patients with bronchial carcinoma may have 'non-metastatic' manifestations (Lambert-Eaton myasthenic syndrome or ectopic hormone production) (see ➲ p. 316; ➲ p. 232; ➲ pp. 235–7).
- Perioperative mortality from pneumonectomy is 5–13%. ALI occurs in 4% of resections and the mortality rate is 30–50%.[13]
- Additional risk factors include the inflammatory response to surgery, chronic alcohol abuse, genetic predisposition, intraoperative plateau pressures >15cmH_2O and >4000mL of IV fluid in first 24h.
- Incidence may be reduced by the intraoperative use of lung-protective strategies (as established in acute respiratory distress syndrome (ARDS) management) and GDFT.
- Heparinisation may be required during sleeve lobectomy if vessel resection is necessary.

ALGRAWANY

Lung volume reduction surgery, bullectomy and endobronchial valves

Procedure	Non-anatomical resection, or occlusion, of regions of hyperinflated and poorly functioning pulmonary tissue
Time	2–5h
Pain	+++/+++++
Position	Median sternotomy (bilateral surgery): supine with arms to sides. Thoracotomy or VATS: lateral decubitus (as for lung resection)
Blood loss	100–500mL; G&S
Practical techniques	Careful IPPV with OLV Paravertebral catheter for analgesia

Lung volume reduction surgery is a surgical treatment for selected patients with severe respiratory failure 2° to emphysema.[14] The aim is to reduce the total lung volume to more physiological levels by resecting the most diseased areas, thereby improving the respiratory function. Most of these patients belong to a group in which GA would normally be avoided at any cost. The procedure is also considered for those with bullous disease and recurrent pneumothoraces.

Preoperative

- Patients require intensive assessment, careful selection and optimisation prior to surgery.
- These are extremely high-risk cases which are being performed less frequently due to the relatively high mortality risk (around 8%).
- A clear understanding of the pathophysiology and adequate thoracic experience are essential to safe anaesthetic management.[15]

Perioperative

- Surgery may be performed via sternotomy or thoracotomy or by VATS.
- Arterial line required for invasive monitoring.
- Thorough preoxygenation is necessary as desaturation can be rapid.
- There is a serious risk of rupturing emphysematous bullae with IPPV, causing leaks and tension pneumothoraces.
- N_2O is contraindicated.
- Limit the risk of 'gas trapping' and dynamic pulmonary hyperinflation by deliberate hypoventilation and permissive hypercapnia (pH >7.2). Recommend V_T 5–6mL/kg, RR 10–12 breaths/min, I:E ratio 1:4 and P_{aw} <30cmH_2O.
- Air trapping can prolong collapse of the operative lung. External pressure by the surgeon and endobronchial suctioning can help.
- Bronchospasm and sputum retention with mucus plugging can also be a problem.
- Cautious fluid use is advised unless the patient requires fluid/blood due to haemorrhage.

Postoperative

- Aim to extubate at the end of the procedure. HDU or ICU care will be required in most cases.
- Watch closely for air leaks and alert surgeons to any concerns.
- Requires excellent pain relief, skilled physiotherapy and a pulmonary rehabilitation programme.

Special considerations

- Commonest complication is prolonged air leak: >7d in 50% of patients.
- Mortality from recent series is 5–10%.
- The National Emphysema Treatment Trial demonstrated that lung volume reduction surgery benefits patients with predominantly upper lobe disease and a low baseline exercise capacity.
- Patients with an isolated congenital bulla or 'lung cyst' require the same careful intraoperative anaesthetic management but are usually much fitter and do not normally require invasive cardiological assessment.

Endobronchial valves

- These are one-way valves that are placed into specific lung segment to prevent air moving in during inspiration but allow air and mucus to leave during expiration. The idea is that this will lead to atelectasis of emphysematous areas of the lung.[16]
- This procedure is done bronchoscopically with either sedation or GA.
- There is much less morbidity associated with this technique, compared to complete lung reduction surgery.
- Interlobar collateral ventilation which occurs in up to two-thirds of patients with severe emphysema will prevent the use of endobronchial valves. In this scenario, endobronchial coils may be considered.[17]

ALGRAWANY

Drainage of empyema and decortication

Procedure	Surgical removal of pus (empyema) and organised thick, fibrinous pleural membrane (decortication)
Time	Drainage 20–40min; decortication 2–3h
Pain	+++/+++++
Position	Lateral decubitus for VATS
Blood loss	Simple drainage: minimal Decortication: 500–2000mL; X-match 2 units
Practical techniques	GA, ETT and IPPV with DLT advised for decortication (risk of air leaks); arterial line, CVP if septic and will require vasopressors

Thoracic empyema describes pus-filled pockets in the pleural space. Often 2° to pneumonia, but also caused by penetrating chest trauma, bronchogenic carcinoma or post-surgery.[19]

Preoperative

- Patients may be overtly septic and respiratory function often already compromised by pneumonia or prior lung resection.
- Check for bronchopleural fistula created by erosion into the lung.

Perioperative

- Usually drained by rib resection and large-bore intercostal drain.
- VATS approach if patient can tolerate OLV.
- If a thoracotomy is required, an epidural should be considered.
- Decortication frequently causes significant haemorrhage.
- Arterial line ± CVP, depending on how septic the patient is.
- Be prepared for conversion to thoracotomy.

Postoperative

- Multimodal analgesia with serratus anterior or erector spinae block (for VATS procedures), with intercostal blocks at the start. Paravertebral catheters are not possible due to loss of the pleura.
- HDU is recommended for decortication in debilitated patients.

Special considerations

- Surgical goal is to remove infected tissue, including pleural 'peel', fully re-expand the lung, and obliterate the infected pleural space.
- Air leaks are common following decortication of the visceral pleura, and lobectomy is occasionally required for a massive air leak.
- Decortication is a major procedure which requires careful evaluation of risks and benefits in elderly, frail and sick patients.

Repair of bronchopleural fistula

Procedure	Closure of communication between pleural cavity and trachea or bronchi
Time	2–3h (for thoracotomy approach)
Pain	++++/+++++
Position	Keep sitting upright, with affected side tilted down until good lung isolated, then lateral decubitus for thoracotomy
Blood loss	300–800mL; G&S. X-match if anaemic
Practical techniques	RSI induction and fibreoptic-guided endobronchial intubation with DLT

The severity of symptoms is proportional to the size of the fistula—big fistulae with large air leaks cause severe dyspnoea and may necessitate urgent respiratory support; critical care input is imperative. Features are productive cough, haemoptysis, fever, dyspnoea, SC emphysema, persistent air leak and falling fluid level in the post-pneumonectomy space on the CXR.

Preoperative

- Patients are often debilitated, with the respiratory function compromised by infection and prior lung resection.
- Check previous anaesthetic charts for ease of intubation and the type of DLT used.
- Check the anatomy of the lower airway carefully on CXR—it is often distorted by previous surgery.
- Patients require resuscitation and a functioning chest drain prior to induction.

Perioperative

- Key principles are to protect the 'good' lung from contamination and to control the distribution of ventilation. Failure to adequately isolate the lungs after induction will put the patient at grave risk.
- Commence invasive arterial pressure monitoring before induction.
- Lung isolation must be confirmed prior to positive pressure ventilation or repositioning of the patient.
- Many thoracic anaesthetists use a modified RSI and advance the DLT under direct vision with a fibreoptic bronchoscope to ensure correct placement in the bronchus contralateral to the fistula, before ventilation is commenced.
- Ideally, three anaesthetists are required, one for laryngoscopy and intubation, one to man the fibreoptic scope and one to watch the anaesthetic.
- Once the tube has been confirmed in the correct bronchus by fibreoptic bronchoscopy, inflate the bronchial cuff first and ventilate directly on the bronchial port of the tube.
- The potential exists to enlarge the fistula by inappropriate placement of the DLT.
- IPPV increases gas leakage, causing loss of V_T and the risk of tension pneumothorax. Minimise P_{aw}.
- Plan HDU/ICU care for all but the most straightforward cases.
- Extubate as soon as possible.

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Special considerations

- Most fistulae are postoperative complications of pneumonectomy or lobectomy, but some are 2° to pneumonia, lung abscesses and empyema.
- Anaesthesia for repair of a bronchopleural fistula is challenging and not recommended for an 'occasional' thoracic anaesthetist!

Pleurectomy/pleurodesis

Procedure	Stripping of parietal pleura from inside of chest wall (pleurectomy). Production of adhesions between parietal and visceral pleura either chemically (talc, tetracycline) or by physical abrasion (pleurodesis)
Time	Pleurectomy 1–2h; pleurodesis 20–40min
Pain	+++/++++
Position	Lateral decubitus for VATS or open thoracotomy
Blood loss	Can bleed from the stripped pleura; G&S
Practical techniques	IPPV and OLV advised for open/VATS procedures

Preoperative

- Patients fall into two groups: the relatively young and fit with recurrent pneumothoraces (check for asthma) and older patients compromised by COPD or recurrent pleural effusions (check respiratory reserve).
- Check a recent CXR for pneumothorax and/or effusion.
- Ensure any infection is treated.
- A preoperative intercostal drain is advised if pneumothorax present.
- Check the planned surgical approach.
- Discuss postoperative analgesia and the regional technique.

Perioperative

- Keep P_{aw} as low as possible in patients with a history of pneumothorax.
- Be alert for pneumothoraces, as they can tension rapidly on IPPV, even with a drain *in situ*, and can be on the 'healthy' side.
- Avoid N_2O.
- Aim for full expansion of the lung at the end of the procedure to oppose the parietal and visceral pleurae.

Postoperative

- Extubate and sit the patient upright before transfer to the recovery room.
- A CXR is needed to check full lung expansion. Pleural inflammation usually causes severe pain, particularly when abrasion of the pleura is performed.
- Multimodal analgesia is again imperative but tend to avoid NSAIDs which may make pleurodesis less effective due to anti-inflammatory effects.
- Paravertebral blocks are usually unsuitable due to damage to the pleura. Intercostal blocks can be sited at the start of the procedure.

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Special considerations

- Pleurectomy is usually performed for recurrent pneumothorax, combined with stapling of the lung tissue responsible for recurrent air leaks (usually apical 'blebs' or small bullae).
- Pleurodesis is often used to manage malignant pleural effusions (mesothelioma, metastatic carcinoma)—there may be large volumes of fluid causing significant respiratory compromise.
- Patients with massive pleural effusions (more than two-thirds of the hemithorax on CXR or >2000mL) should have these 'tapped' and partially drained at least 12h before surgery, because rapid intraoperative reinflation of the collapsed lung can precipitate unilateral postoperative 're-expansion' pulmonary oedema.
- Patients with extensive effusions are also at risk of circulatory collapse when turned 'effusion side up' for surgery. The mechanism is probably a combination of mediastinal shift and high intrathoracic pressure from IPPV reducing the venous return and CO. If this occurs, return the patient to the supine position and drain the effusion before proceeding.

Oesophagectomy

Procedure	Total or partial excision of oesophagus with mobilisation of stomach (occasionally colon) into chest
Time	3–6h
Pain	+++++
Position	Supine with arms by sides and/or lateral decubitus for thoracotomy
Blood loss	500–1500mL; X-match 2 units
Practical techniques	IPPV, OLV via DLT or bronchial blocker, arterial/CVP lines, urinary catheter, thoracic epidural or paravertebral catheter for thoracoabdominal incision

Types of procedure

Minimally invasive

Thoracoscopic oesophageal mobilisation, laparoscopic gastric mobilisation and cervical anastomosis.

Ivor–Lewis

Laparotomy and right thoracotomy, often laparoscopic-assisted.

Transhiatal

Laparotomy and cervical anastomosis.

Thoracoabdominal

Left thoracotomy crossing the costal margin and diaphragm.

McKeown 3-stage

Laparotomy, right thoracotomy and cervical anastomosis.

Preoperative

- Establish the indication for surgery—usually oesophageal cancer, but occasionally for non-malignant disease (benign stricture, achalasia).
- Preoperative malnutrition or cachexia is common and associated with a higher risk of postoperative morbidity and mortality.
- Preoperative adjuvant chemotherapy may leave residual immunosuppression but can dramatically improve dysphagia.
- Some centres utilise enhanced recovery protocols.
- Will need HDU or ICU, depending on local protocols.

Perioperative

- Consider all patients with oesophageal disease to be at risk of regurgitation; some patients may require a modified RSI.
- If thoracotomy is planned, use a DLT or bronchial blocker.
- Prepare for long surgery, sometimes involving repositioning.
- Multimodal analgesia is necessary, including regional block. NSAIDs should be avoided, but give intraoperative paracetamol and magnesium. Consider clonidine, and for shoulder tip pain, tramadol.
- Plan regional anaesthesia according to the surgical approach. Paravertebral catheter infusion with morphine PCA for the thoracoabdominal approach. For laparotomy/thoracotomy, a mid-thoracic epidural is advised for intra- and postoperative use.

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- An NGT will be required initially. It is removed for resection and reinserted under surgical guidance following anastomosis.
- Monitor the core temperature, and be obsessional about keeping the patient warm.
- A fluid-restrictive regime is advised, typically 2mL/kg/h as an infusion, with additional fluid boluses as guided by haemodynamic parameters for hypovolaemia. Vasopressors should be used where required.
- Check Hb and blood gases regularly.
- Arrhythmias and reduced CO causing hypotension may occur during intrathoracic oesophageal mobilisation.
- Change the DLT to an SLT to improve surgical access prior to cervical anastomosis (if performed).

Postoperative

- Early extubation is ideal. If cold (<35.5°C) or haemodynamically unstable, ventilate until the condition improves.
- Patients require intensive and experienced postoperative nursing care in a specialist ward, HDU or ICU.
- Use a jejunostomy or nasoduodenal tube for early enteral feeding. A bridle can be useful in these patients who are prone to postoperative delirium.

Special considerations

- Oesophagectomy has one of the highest perioperative mortality rates of all elective procedures (up to 5%, even in specialist centres).
- Sixty-six per cent of deaths are from systemic sepsis 2° to respiratory complications or anastomotic breakdown.
- Over 30% of patients suffer a major complication.
- The majority of centres perform minimally invasive (endoscopic) oesophagectomy. Beware 'tension capnothorax' if the pleura is breached during laparoscopic hiatal dissection.
- Occasional practice in anaesthesia (or surgery) for oesophagectomy is not recommended.

Chest injury

The emergency diagnosis and initial treatment of major thoracic trauma are described on ➲ pp. 995–8. This section deals with the anaesthetic management of rib fractures and the definitive repair of ruptures of the diaphragm, oesophagus and tracheobronchial tree.

Rib fractures

- These are common and associated with a high risk of respiratory complications. Trauma injuries are increasingly being seen in those aged >65y who have ↑ comorbid burden and polypharmacy, including anticoagulation.
- Prompt multimodal analgesia including paracetamol, NSAID where appropriate, oral or PCA opioids and regional block are essential to improve respiratory mechanics and reduce complications.
- Regional analgesia can be afforded by paravertebral catheter insertion or fascial plane catheters. An increasing number of ultrasound-guided erector spinae or serratus anterior plane blocks are being performed, which are both relatively simple to do, reduce the risk of pneumothorax and can be performed in coagulopathic patients.
- Plane block catheters can be bolused with 30–40mL of 0.25% levobupivacaine and then run as 10mL/h of 0.125% levobupivacaine.
- Paravertebral catheter insertion is an alternative and provides a reliable unilateral block with reduced hypotension, motor blockade and urinary retention seen with thoracic epidurals, which are no longer considered the gold standard in traumatic rib fractures.
- Operative fixation may be indicated, especially in those with flail segments and uncontrollable pain.
- Humidified O_2, nebulised 0.9% sodium chloride and respiratory physiotherapy also help reduce respiratory complications. Non-invasive ventilation or high-flow O_2 reduce atelectasis and improve the paradoxical movement of a flail segment. IPPV should be avoided, if possible.

Repair of ruptured diaphragm

- Clinical features and diagnosis are described on ➲ p. 998.
- May present as a chronic condition or as intestinal obstruction of a herniated bowel, so check preoperative fluid and electrolyte status.
- The defect should be closed promptly, but rarely emergently.
- The surgical approach is via a standard lateral thoracotomy or a thoracoabdominal incision. DLT facilitates surgical access.
- Management is as for a fundoplication (see ➲ p. 534).
- Avoid N_2O, as it distends the bowel and may make reduction of the hernia more difficult.
- An NGT should be used to decompress the stomach.

Repair of ruptured oesophagus

- Clinical features and diagnosis are described on ➲ p. 997. Surgical emphysema and empyema are frequently present.
- Other causes of oesophageal rupture include excessive abdominal straining and uncoordinated vomiting (Boerhaave's syndrome).

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- Oesophageal perforation can be caused by foreign bodies but is often iatrogenic (during endoscopic procedures).
- Mediastinitis is followed rapidly by sepsis and a systemic inflammatory response syndrome (SIRS), with associated problems of circulatory shock, renal failure and ARDS.
- The principles of surgical management are initially drainage and prevention of further contamination.
- Endoscopy will determine the extent of oesophageal disruption.
- Small tears in unfit patients may be managed conservatively with chest drainage and NG suction, but often urgent surgery is required.
- Patients should be stabilised preoperatively (chest drainage, IV fluid replacement, analgesia, invasive monitoring and inotropic support).
- Intraoperative management is as for oesophagectomy.
- Upper and lower oesophageal injuries require right and left thoracotomy, respectively.
- 1° closure may be possible if the oesophagus is healthy; if not, oesophagectomy will be required.
- Arrhythmias are common, particularly AF, due to mediastinitis.
- Change the DLT for an SLT before transfer to ICU.
- All patients are high risk of major complications for several days.
- Early postoperative feeding via feeding jejunostomy or parenterally.
- There is a significant incidence of dehiscence, resulting in an oesophagopleurocutaneous fistula with high mortality.

Repair of tracheobronchial injury

- Most patients with significant tracheal/bronchial disruption do not reach hospital alive.
- Clinical features of laryngeal and tracheobronchial injuries are described on ➲ p. 998.
- 100% O_2 and relief of tension pneumothorax (see ➲ p. 974).
- If ventilation and oxygenation are acceptable, call for thoracic surgical assistance and try to assess and identify the site of airway injury by fibreoptic bronchoscopy before intubation.
- Airway management and anaesthetic principles apply as for a large bronchopleural fistula (see ➲ pp. 547–8).
- Adequate positive pressure ventilation may be impossible with an SLT.
- A torn bronchus can be isolated by fibreoptic-guided intubation of the contralateral intact main bronchus with an appropriate DLT.
- An uncut SLT can be guided past an upper tracheal tear with a bronchoscope, so its cuff lies distal to the injury.
- Once the airway is secure and ventilation is stabilised, proceed to an urgent thoracotomy for repair.
- Carinal disruption may require CPB to maintain oxygenation during repair.
- Inappropriate management can lead to long-term airway problems.

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Chapter 21

Neurosurgery

Gemma Nickols and **Amit Goswami**

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See also

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General principles

Intracranial pressure

Normal ICP is 5–15mmHg. Changes in ICP reflect changes in the volume of intracranial contents held within the confines of the skull (brain substance 1200–1600mL, blood 100–150mL, CSF 100–150mL, ECF <75mL). Compensatory mechanisms initially reduce the effect of an intracranial space-occupying lesion on ICP by displacing the CSF into the spinal subarachnoid space, increasing the absorption of CSF and reducing intracranial blood volume. Eventually, these mechanisms are overwhelmed, and further small increases in intracranial volume result in a steep rise in ICP (Fig. 21.1). If a lesion develops slowly, it may reach a relatively large volume before causing a significant rise in ICP. Conversely, a small lesion may have developed quickly, allowing little time for compensation.

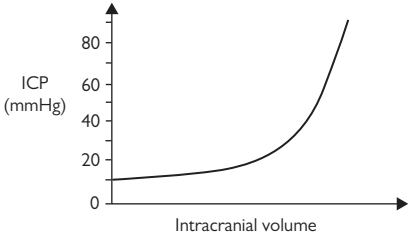

Fig. 21.1 Intracranial pressure and volume.

Causes of raised intracranial pressure

- ↑ brain volume: generalised swelling, cerebral oedema
- Mass effect: space-occupying lesion, haematoma, tumour, abscess
- ↑ CSF volume: ↑ production or ↓ resorption, e.g. hydrocephalus, meningeal inflammation
- ↑ blood volume:
  - ↑ CBF: hypoxia, hypercapnia, volatile anaesthetic agent
  - ↑ cerebral venous volume: ↑ thoracic pressure, venous stasis/thrombosis, venous obstruction in the neck, head-down tilt, coughing
- Other: benign or idiopathic intracranial hypertension.

Cerebral perfusion pressure

Cerebral perfusion pressure (CPP) is the effective pressure that results in blood flow to the brain. Venous pressure (VP) at the jugular bulb is usually zero or less, so CPP is related to ICP and MAP alone.

$$CPP = MAP - (ICP + VP)$$

The CPP varies with the patient's MAP, but CBF is maintained constant by autoregulation.

Cerebral blood flow

Autoregulation maintains CBF (50mL/100g brain tissue/min) between a MAP of 60 and 150mmHg. Outside this, CBF varies passively with perfusion pressure. In patients with chronic hypertension, the lower and upper limits of autoregulation are higher than normal, so a MAP that may be adequate in a normal patient may lead to cerebral ischaemia in the hypertensive patient. Autoregulation is also impaired or abolished acutely in the presence of brain tissue acidosis, i.e. with hypoxia, hypercapnia, acute intracranial disease and following head injury.

CBF varies with:

- Metabolism: CBF is primarily determined by the metabolic demands of the brain; ↑ during epileptic seizures and with pain/anxiety. ↓ in coma, hypothermia and with anaesthetic agents.
- CO_2 tension: hypocapnia → cerebral vasoconstriction and ↓ CBF. The greatest effect is at normal $PaCO_2$ where a change of 1kPa (7.5mmHg) results in a 30% change in blood flow. MAP modifies the response of CBF to hyperventilation. High perfusion pressures increase the responsiveness to hyperventilation, whereas hypotension of 50mmHg abolishes the effect of $PaCO_2$ on CBF.
- O_2 tension: PaO_2 is not an important determinant of CBF, cerebral vasodilation occurring only <7kPa (53mmHg).
- Temperature: cerebral metabolism ↓ ~5% per °C, thereby ↓ CBF.
- Viscosity: there is no effect on CBF when the Hct is between 30% and 50%. CBF will increase with reduced viscosity.
- Anaesthetic agents.

Measuring intracranial pressure

- Ventricular: a catheter inserted into a lateral ventricle via a burr hole is the gold standard, also allowing drainage of CSF. Risks include haemorrhage at insertion and ventriculitis with prolonged use. Insertion may be difficult in cerebral oedema and small ventricles.
- Intraparenchymal: microminiature silicone strain gauge monitors inserted into the brain parenchyma. Current commonest technique.

Anaesthesia in presence of raised intracranial pressure

- Symptoms and signs to identify patients with ↑ ICP preoperatively:
  - Early: headache, vomiting, seizures, focal neurology, papilloedema.
  - Late: hypertension and bradycardia. Agitation, drowsiness, coma, Cheyne–Stokes breathing, apnoea. Ipsilateral, then bilateral, pupillary dilatation; decorticate, then decerebrate posturing.
- Investigations: evaluate CT/MRI scans for presence of generalised oedema, midline shift, acute hydrocephalus and site/size of any lesion.

Management aims

Do not increase ICP further.

- Avoid increasing CBF by avoiding hypercapnia, hypoxia, hypertension and hyperthermia. Use IPPV to control $PaCO_2$, and ensure good oxygenation, adequate analgesia and anaesthetic depth.
- Avoid increasing VP. Avoid coughing and straining, the head-down position and obstructing the neck veins with ETT ties.

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- Prevent further cerebral oedema. While patients are generally fluid-restricted, it is important to maintain intravascular volume and CPP. Do not use hypotonic solutions; fluid flux across the blood–brain barrier is determined mainly by plasma osmolality, not oncotic pressure. Maintenance of a high normal plasma osmolality is essential.
- Maintain CPP: hypotension will decrease CPP in the presence of a raised ICP. Control BP using fluids and vasopressors, as necessary. Aim for CPP 60–70mmHg.
- Avoid anaesthetic agents that increase ICP (see below).

Specific measures to decrease intracranial pressure

- Reduce cerebral oedema using osmotic or loop diuretics, or both. Give mannitol 0.25–1g/kg over 15min or 5% sodium chloride (100mL) and furosemide 0.25–1mg/kg. Insert a urinary catheter in patients receiving diuretics.
- Modest hyperventilation to $PaCO_2$ of 4.0–4.5kPa (30–34mmHg) has a transient effect in reducing ICP for 24h, but should be used only as a temporising measure. Excessive hyperventilation results in cerebral ischaemia and loss of autoregulation.
- Corticosteroids reduce oedema surrounding tumours and abscesses but have no role in head injury. They take several hours to work. Dexamethasone 4mg 6-hourly often given electively preoperatively.
- CSF may be drained via a ventricular or lumbar drain.
- Position the patient with a head-up tilt of 30° to reduce CVP. Ensure that MAP is not significantly reduced, as the overall result could be a reduction in CPP.

Anaesthetic agents and intracranial pressure

- Volatile agents uncouple metabolism and flow, reducing cerebral metabolism, while increasing CBF and ICP. They abolish autoregulation in sufficient doses. Halothane causes the greatest increase in ICP, and isoflurane the least. ICP is unaffected by concentrations of <1 MAC of isoflurane, sevoflurane and desflurane. N_2O is a weak cerebral vasodilator increasing CBF, and therefore ICP. It has also been shown to increase cerebral metabolic rate.
- IV anaesthetic agents all decrease cerebral metabolism, CBF and ICP, with the exception of ketamine. Ketamine has some neuroprotective properties, with its use controversial in neurosurgery. CO_2 reactivity and autoregulation of the cerebral circulation are well maintained during propofol/thiopental anaesthesia.
- Other drugs:
  - Suxamethonium causes a rise in ICP through muscle fasciculation, increasing VP. This effect is of little clinical relevance. Suxamethonium can still be used when rapid intubation is required in the unstarved patient, although its use has largely been superseded by rocuronium.
  - Opioid analgesics have little effect on CBF and ICP if hypercapnia is avoided. CO_2 reactivity is maintained.

Craniotomy

Procedure	Excision or debulking of tumour, brain biopsy, drainage of cerebral abscess
Time	1–12h
Pain	+/+++
Position	Supine, head-up tilt or lateral decubitus
Blood loss	0–2000mL, G&S ± X-match
Practical techniques	ETT, IPPV, arterial line

Preoperative

- Assess the patient's current neurological state, including symptoms and signs of raised ICP, documenting deficits. Assess the gag reflex.
- Intracranial tumours may be metastatic; 1° sites include lung, breast, thyroid and bowel.
- Check CT/MRI scans: the duration and complexity of the procedure are determined by the size, site and vascularity of lesions.
- Patients vomiting or receiving diuretics may have disordered electrolytes. Patients receiving dexamethasone may be hyperglycaemic.
- Restrict IV fluids if cerebral oedema present. Avoid glucose-containing solutions, which can cause hyperglycaemia, associated with a worse outcome after brain injury. They also reduce osmolality, resulting in ↑ cerebral oedema.
- Mechanical methods of DVT prophylaxis should be utilised.
- Prophylactic or therapeutic anticonvulsants may be required. Levetiracetam (loading dose 500–1000mg) is now much more frequently used than phenytoin (loading dose 15mg/kg), with evidence for better efficacy.

Perioperative

- Patients undergoing burr hole biopsy require standard monitoring. Those scheduled for craniotomy also need an arterial line, and a urinary catheter for long procedures and patients on diuretics. Neuromuscular and core temperature monitoring are desirable. Depth of anaesthesia monitoring should be considered, especially with TIVA.
- Induce with propofol bolus or TCI infusion (or thiopental 3–5mg/kg if available), combined with remifentanil infusion or fentanyl. Give slowly to avoid reducing BP and CPP. A non-depolarising relaxant is used to facilitate intubation. Remifentanil usually attenuates the hypertensive response to intubation. Additional agents such as lidocaine 1.5mg/kg or a β-blocker (labetalol 5mg increments) can be used. Use an armoured ETT to prevent kinking, and tape in place, as ties may cause venous obstruction.
- Ensure adequate eye protection to avoid injury.
- Avoid N_2O. Maintain anaesthesia using either a volatile agent (sevoflurane/isoflurane <1 MAC) or TCI propofol. Remifentanil infusion (micrograms/kg/min) or TCI (nanograms/mL) or intermittent fentanyl boluses can be titrated to response. Top-up doses of muscle relaxants are rarely required when remifentanil is used.

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- Patients may be placed in the supine or lateral/park bench position. Avoid extreme neck flexion or rotation. If the head is turned for surgery, support the shoulder to reduce the effect on neck veins and maintain venous return. Protect all pressure points. Maintain a head-up tilt.
- Application of the Mayfield 3-point fixator to secure the head can cause a marked hypertensive response. Pin sites can be infiltrated with LA, and, if necessary, give a small bolus dose of remifentanil or propofol.
- Aim for normotension during most procedures. Modest hypotension may infrequently be required to improve the surgical field. Aim for normocapnia $PaCO_2$ of 4.5–5kPa (33–38mmHg).
- Avoid hypotonic solutions for fluid maintenance. Replace blood loss with colloid or blood.
- Maintain normothermia. Hypothermia is rarely indicated.
- Use intermittent pneumatic compression device to the calves or feet.
- Closure of the dura, bone flap and scalp takes at least half an hour. Administer IV morphine or oxycodone at this stage to provide analgesia when remifentanil is stopped. Sudden hypertension on awakening may be treated with small boluses of labetalol. Avoid coughing.

Postoperative

- Further IV opioid may be required in the immediate postoperative recovery period.
- Craniotomies can be managed postoperatively in a high-care area on the neurosurgical ward, or on the HDU. Continued monitoring of the patient's conscious level and neurological state is essential. Consider postoperative sedation and ventilation if there is continuing cerebral oedema or if the patient was severely obtunded preoperatively.
- On return to the ward, the majority of patients will experience pain in the mild to moderate range. At this stage, codeine phosphate (30–60mg), combined with regular paracetamol, is usually sufficient in >90% of patients, with oral morphine for breakthrough.

Special considerations

- The use of NSAIDs for postoperative analgesia needs careful consideration. While reducing opioid requirements, they also increase bleeding time. A postoperative intracranial haematoma is a serious complication; thus, they are not often routinely prescribed, and if needed, a delayed start may be sensible.
- Scalp blocks can be considered as a systemic analgesia sparing adjunct, performed at either the beginning or end of surgery.
- A central line is indicated for complex craniotomies or poor peripheral access. It allows measurement of CVP, infusion of vasoactive drugs and aspiration of air in the case of VAE.

Ventriculoperitoneal shunt

Procedure	CSF drainage for hydrocephalus
Time	45–120min
Pain	++
Position	Supine, head-up tilt
Blood loss	Minimal
Practical techniques	ETT, IPPV

Shunts are inserted for hydrocephalus, with CSF diverted from the cerebral ventricles to other body cavities, from where it is absorbed. Most commonly, a ventriculoperitoneal shunt is created, more rarely a ventriculoatrial or ventriculopleural shunt. An occipital burr hole enables a tube to be placed into the lateral ventricle. This is then tunnelled SC down the neck and trunk, and inserted into the peritoneal cavity through an abdominal incision. A flushing device can be placed in the burr hole to keep the system clear, and a valve system is incorporated to prevent CSF draining too rapidly with changes in posture.

Preoperative

- As for craniotomy (see ➲ pp. 561–2). Assume ↑ ICP in all patients.
- Patients requiring shunts are often children; therefore, usual paediatric considerations apply.
- Emergency cases may have a full stomach, requiring RSI.

Perioperative

- Shunt procedures are shorter and simpler than craniotomies. Use routine monitoring. Arterial lines are not usually required, unless significant comorbidity.
- IV antibiotic treatment or prophylaxis is required and intrathecal antibiotics are usually administered by the surgeon. Strict antisepsis protocols are normally followed to reduce the incidence of infection.
- Advancing the trocar to allow tunnelling of the shunt is particularly stimulating. Additional analgesia and/or muscle relaxation is often required at this stage.
- Establish warming with forced air warming blanket or underbody heated mat, as large area of patient often exposed.

Postoperative

- Any deterioration in conscious level is an indication for a CT scan to exclude shunt malfunction or a subdural haematoma.

Special considerations

- Risk of intracranial haemorrhage if CSF drained too rapidly.
- Shunts often block or become infected, requiring revision.
- Watch for signs of a pneumothorax, as the trocar is placed SC.

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Evacuation of traumatic intracranial haematoma

Procedure	Evacuation of extradural, subdural or intracerebral haematoma
Time	1–6h
Pain	+/+++
Position	Supine, head-up
Blood loss	200mL+, G&S ± X-match
Practical techniques	ETT, IPPV, arterial line, CVC

Intracranial haematoma may be extradural, subdural or intracerebral.

- Extradural: usually the result of a tear in the middle meningeal artery. Frequently associated with a skull fracture, except in children. Urgent evacuation is required, certainly within an hour of pupillary dilation.
- Subdural haematoma: bleeding from the bridging veins between the cortex and dura. Early evacuation of an acute subdural haematoma likely improves outcome. Chronic subdural haematomas may occur in the elderly, often after trivial injury. They present insidiously with headaches and confusion, and can be evacuated via a burr hole under GA or LA.
- Intracerebral haematoma: there may be some benefit for early surgery in reduced GCS. Commonly spontaneous in hypertensive individuals, as a complication of treatment with anticoagulants or bleeding from an intracranial aneurysm.

Preoperative

- As for head injury (see pp. 989–94).
- Most patients will have a reduced or deteriorating GCS.
- ICP is usually raised.
- Patients may have associated injuries, requiring resuscitation and treatment in their own right (see Chapter 37). Protect the spine, if indicated.
- Patients may have a full stomach, requiring RSI. Insert an OGT after intubation.
- Check the blood clotting profile and the availability of blood products, prior to surgery. Reversal of anticoagulants or antiplatelets may be required, with management of novel anticoagulants more problematic, but reversal agents increasingly available.
- Consider administration of tranexamic acid (loading 1g), ideally within 3h of injury (CRASH 3 trial).

Perioperative

- As for craniotomy (see pp. 561–2).
- Patients require standard monitoring, including invasive BP monitoring. A CVC may be required if poor peripheral access.
- Ensure smooth induction and normotension. Maintain CPP using fluids and vasopressors if necessary. Assume the ICP is 20mmHg and attempt to maintain a CPP of 60–70 mmHg (MAP 80–90mmHg), but certainly an SBP ≥100–110mmHg, dependent on age.

- Ensure a head-up tilt; avoid N_2O; ventilate to an $ETCO_2$ of 4.5kPa.
- Give mannitol (0.25–1g/kg) or 5% sodium chloride (2–6mL/kg) and furosemide (0.25–1mg/kg) as required.
- Once decompression has occurred, there may be a decrease in systemic BP, which can usually be treated with volume replacement or vasopressors.

Postoperative

- Most patients should be transferred to ICU. Further management should be guided by a protocol to maintain CPP and prevent 2° brain injury (see below).

Special considerations

- It is essential for the subspecialty teams to communicate and set priorities in the management of patients with multiple injuries. Priorities will vary from patient to patient.
- Hypotension in a head-injured patient is a medical emergency and must be treated promptly and aggressively.

Postoperative and intensive care unit management of the head-injured patient

- Management of head-injured patients is similar for postoperative patients and those not requiring surgery. Patients are best managed using a protocol designed primarily to maintain adequate CPP/cerebral oxygenation and control ICP. It involves preventing, identifying and treating causes of 2° brain insults (Fig. 21.2).
- Causes of 2° insults are:
  - Intracranial: haematoma, oedema, convulsions, hydrocephalus, abscess, hyperaemia
  - Systemic: hypotension, hypoxia, hyponatraemia, pyrexia, anaemia, sepsis, hypercapnia, hyperglycaemia.
- Steroids should not be administered to patients following severe head injury.
- Consider the use of anticonvulsants for prophylaxis of post-traumatic seizures.

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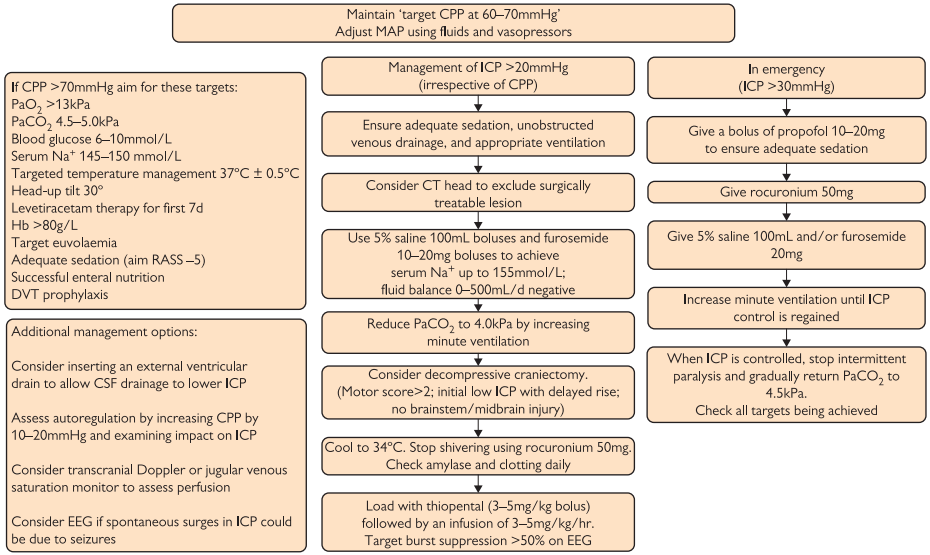

Fig. 21.2 Guidelines for managing adults with severe head injuries in the ICU.

Pituitary surgery

Procedure	Trans-sphenoidal hypophysectomy
Time	90–300min
Pain	++
Position	Supine, head-up tilt
Blood loss	Nil usually, but large if venous sinus disrupted, G&S
Practical techniques	ETT, IPPV, arterial line

Pituitary tumours account for 10–15% of all intracranial tumours. They present with:

- Hormone hypersecretion (acromegaly, Cushing's disease, prolactinoma, thyrotoxicosis)
- Hormone hyposecretion (adrenal insufficiency, hypothyroidism, diabetes insipidus)
- Mass effects (headaches, visual field defects, hydrocephalus).

Hypophysectomy is undertaken urgently if the patient's sight is deteriorating rapidly.

Preoperative

Special considerations for acromegalic patients (see also ➲ p. 222):

- Possible airway compromise due to macroglossia, prognathism and hypertrophy of the epiglottis/vocal cords. Advanced airway techniques may be required (videolaryngoscopy, AFOI).
- Hypertension, LVH, IHD, cardiomyopathy.
- OSA in up to 70%.
- DM and other endocrine pathology.

Special considerations for Cushing's patients (see also ➲ p. 232):

- Hypertension, truncal obesity, OSA
- Electrolyte abnormalities (hypokalaemia)
- Glucose intolerance/DM
- Higher risk of perioperative thromboembolic disease
- Steroid cover necessary pre- and postoperatively.

Perioperative

- As for craniotomy (see ➲ pp. 561–2).
- Invasive arterial monitoring generally advised.
- A throat pack should be inserted following intubation to reduce laryngeal soiling.
- A topical vasoconstrictor, e.g. Moffett's solution (see ➲ p. 758) may be instilled into each nostril to improve surgical conditions.
- Surgical access is via the sphenoidal air sinuses.
- If there is suprasellar extension, a lumbar drain can be inserted into the CSF. The anaesthetist may be required to instil a volume of sterile 0.9% sodium chloride to advance the tumour into the operative field.
- Major haemorrhage may occur if there is disruption of the cavernous sinus/carotid arteries, which are lateral to the pituitary gland.

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Postoperative

- Diabetes insipidus may occur within 12–24h of surgery, in up to 50% of patients. It is managed initially with IV desmopressin (0.25–1 micrograms). Can be given PO subsequently if required.
- Cerebrospinal rhinorrhoea may occur. It is usually self-limiting, but if persistent, intermittent CSF drainage via a lumbar drain may be required. Occasionally, surgery to repair the CSF leak is required.

Special considerations

Patients with preoperative panhypopituitarism or who develop post-operative endocrine disturbances should be referred to an endocrinologist for advice on hormone replacement.

If a craniotomy is planned, rather than a trans-sphenoidal approach, refer to pp. 561–2.

Posterior fossa surgery

Procedure	Excision or debulking of tumour, vascular procedures, foramen magnum decompression
Time	3–14h
Pain	+/+++
Position	See below
Blood loss	100–2000mL, G&S
Practical techniques	ETT, IPPV, arterial line, consider CVP/monitoring for VAE

- The posterior fossa lies below the tentorium cerebelli and contains the pons, medulla and cerebellum. Within the brainstem lie the main motor and sensory pathways, the lower cranial nerve nuclei and the centres that control respiration and CVS function.
- An increase in pressure in this area results in ↓ consciousness, hypertension, bradycardia, respiratory depression and loss of protective airway reflexes.
- The exit pathways for CSF from the ventricular system are also located here and obstruction results in hydrocephalus.
- Space-occupying lesions and surgical disturbance in this area can therefore have a profound physiological impact.
- Common pathologies requiring surgery include tumours, vascular malformations, cysts, cranial nerve lesions and craniocervical abnormalities.[1]

Preoperative

- Patients with posterior fossa lesions may have a reduced level of consciousness and impaired airway reflexes. Bulbar palsy may lead to silent aspiration. Pulmonary function must be assessed.
- Patients with pre-existing impaired airway reflexes may require postoperative ventilation or tracheostomy.
- Assess ICP: may be raised. If hydrocephalus is present, ventricular drainage may be required before the definitive procedure.
- Assess the fluid status: may be dehydrated if vomiting. A reduced intravascular volume will result in hypotension on induction or if placed in the sitting position.
- Check electrolytes and glucose, particularly if taking diuretics or steroids.
- Assess CVS function, particularly the presence of untreated hypertension, postural hypotension and septal defects.

Perioperative

- As for craniotomy (see ➲ pp. 561–2).
- Insert an NGT if risk of postoperative bulbar dysfunction.
- Further specialised monitoring is required for posterior fossa surgery, including monitoring for VAE (see ➲ pp. 584–5) and nerve tract injury. The appropriate neurophysiological monitor used to detect a nerve tract injury depends upon the neural pathway at risk during the procedure. Spontaneous or evoked EMG activity, somatosensory

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evoked potentials or brainstem auditory evoked potentials are frequently monitored. Lumbar CSF drainage is occasionally required to improve surgical conditions and to reduce the incidence of postoperative CSF leaks.

- Surgical interference with vital centres may result in sudden and dramatic CVS changes. Inform the surgeon—gentler retraction or dissection usually resolves the problem. Use drugs, such as atropine and β-blockers, only if absolutely necessary, as they make the interpretation of further changes difficult.
- The need to assess conscious state and the presence of a cough reflex must be balanced against a smooth extubation, avoiding excessive coughing and an associated rise in ICP. This can be difficult to achieve.
- If concerns exist about a poor or absent cough postoperatively, the patient may require a period of postoperative ventilation ± tracheostomy placement.

Patient positioning

- Surgical access to the posterior fossa requires the patient to be sitting, prone or lateral. Careful attention is required in positioning the patient, as procedures are often prolonged.
- Sitting position: use of this position is uncommon. It provides optimum access to midline lesions, improves cerebral venous drainage and lowers ICP. However, complications include haemodynamic instability, VAE and the possibility of paradoxical air embolism, pneumocephalus and quadriplegia. Absolute contraindications include cerebral ischaemia when upright and awake, the presence of a patent ventriculoatrial shunt or a right-to-left cardiac shunt (should be screened for preoperatively). Relative contraindications are patent foramen ovale, uncontrolled hypertension and extremes of age. To achieve this position, the head and shoulders are gradually elevated, with the neck partially flexed and the forehead resting on a horseshoe ring mounted on a frame. Avoid excessive head flexion, since this can cause jugular compression, swelling of the tongue and face and cervical cord ischaemia.[1]
- Prone position: allows good surgical access without the risks associated with the sitting position. Abdominal compression should be avoided, as it results in ↑ cerebral VP. This is achieved by adequately supporting the chest and pelvis.
- Lateral position: the lateral or 'park bench' position is particularly suitable for lateral lesions such as acoustic neuroma and operations on a cerebellar hemisphere. The neck is flexed and the head rotated towards the floor, ensuring that the jugular veins are not obstructed. Pressure points over the shoulder, greater trochanter and peroneal nerves should be protected.

Postoperative

- Most patients can be safely extubated and managed on a neurosurgical HDU postoperatively.
- Airway obstruction can occur after posterior fossa surgery due to macroglossia, partial damage to the vagus nerve and excessive flexion of the cervical spine.
- Surgery on the medulla or high cervical lesions carries a significant risk of postoperative impairment of the respiratory drive.
- The patient should be admitted to ICU for ventilation if the preoperative state was poor, the surgical resection was extensive, there is significant cerebral oedema, there is evidence of an inadequate cough or there are intraoperative complications.

Special considerations

- Acoustic neuroma: the facial nerve is particularly vulnerable and is monitored using evoked EMG needles placed over the face. This allows the surgeon to identify when the nerve is at risk. NMBA should be used only at induction to allow intubation. Often the 8th nerve function is also monitored to preserve any residual hearing. This requires a constant level of anaesthesia, so that neurophysiological changes can be attributed to surgery, rather than to variations in anaesthetic depth. These requirements can be easily met using TIVA (propofol and remifentanil).
- VAE (see pp. 584–5).
- Postoperative analgesia is managed as for craniotomy.

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Awake craniotomy

Procedure	Excision of tumours in eloquent cortex, epilepsy surgery, implantation of deep brain stimulator electrodes
Time	>1.5h
Pain	+/+++
Position	See below
Blood loss	100–2000mL, G&S
Practical techniques	LMA, arterial line

- Awake craniotomy allows intraoperative assessment of the patient's neurological status. It is commonly used in the excision of tumours from eloquent areas of the cortex (sensory, motor, speech areas).
- Proposed benefits of awake craniotomy include ↑ tumour resection (with associated improved survival benefit), a reduced requirement for high dependency care, reduced hospital length of stay and a reduction in postoperative complications such as nausea and vomiting.[2]
- A variety of anaesthetic techniques exist, including LA ± conscious sedation or GA. In cases of conscious sedation or GA, the patient is woken up for intraoperative assessment, or 'testing', before being resedated or reanaesthetised.
- During intraoperative testing, direct electrical stimulation may be applied to areas of the cortex to see if this produces transient functional deficit. This procedure, referred to as cortical mapping, can result in seizures.

Preoperative

- As for craniotomy (see ➔ pp. 561–2).
- Both the neurosurgeon and neuroanaesthetist must be experienced in awake craniotomy.
- Appropriate patient selection, based on patient comorbidity and psychological and surgical factors, is of paramount importance.
- Absolute contraindications include patient refusal, an inability to lie still for any length of time and confusion.[2]
- Relative contraindications include patient anxiety, cough, OSA and barriers to communication.[2]
- Preoperative preparation of the patient is vital. Meeting before the day of surgery allows the procedure to be discussed in detail and can help allay patient anxiety.[3]
- Consider premedication with antireflux medication. Sedative premedication should be avoided.
- Anticonvulsant prophylaxis should be prescribed routinely for all patients, with dexamethasone for those undergoing tumour surgery.

Perioperative

The surgery can be simply divided into three phases:

1. From start of surgery to bone flap removal and dural opening
2. Resection of the lesion with functional testing
3. From completion of resection to closure.

- Aims of anaesthesia are to ensure adequate sedation, analgesia, cardiorespiratory stability, avoidance of hypercapnia, nausea and vomiting, as well as ensuring an awake and cooperative patient when required for intraoperative testing.
- Routine monitoring as for craniotomy should be used, including urinary catheterisation if the procedure is expected to be prolonged.
- The location of the lesion will determine patient position—commonly the supine or lateral position is used. The patient will need to lie in this position for a prolonged period of time. Positioning the patient awake, if possible, can allow the most comfortable position to be achieved.[2]
- Effective LA is key to successful awake surgery. This can be achieved through a scalp block or local infiltration by the surgeon.[3]
- There is no clear consensus as to the best anaesthetic technique. This decision should be made on a case-by-case basis, dependent upon patient and surgical factors.[2,3] Two common techniques are discussed below.
- Additional complications of awake surgery include seizures, failure to complete intraoperative testing and unplanned conversion to GA.[3]

Asleep–Awake–Asleep

Using an Asleep–Awake–Asleep technique, the patient is given a GA during the initial phase of the craniotomy. The patient is then woken for intraoperative testing before being reanaesthetised for closure.

- Either TIVA or a volatile can be used. A commonly used recipe is TIVA (propofol and remifentanil) using an SGA with IPPV.[3]
- Benefits include avoidance of complications of the sedation technique (hypoxia, hypoventilation, airway obstruction) and the ability to control CO_2.[2]

Conscious sedation technique

Using a conscious sedation technique, the patient is sedated without airway intervention during the initial phase and then woken up for testing. The patient can be resedated for closure.

- Commonly used drug infusions include propofol, remifentanil and dexmedetomidine.[3]
- Benefits include improved compliance with testing, reduced opioid and vasoactive requirements and reduced hospital length of stay.[3]
- Achieving the right balance of sedation can be difficult—too little sedation can result in pain and discomfort, while too much can result in hypoventilation ± airway obstruction. Hypercapnia can lead to a raised ICP.[3]

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Postoperative

- A long-acting opioid (morphine, oxycodone) should be administered at the end of the procedure.
- Other aspects of postoperative care are as for craniotomy (see pp. 561–2).

Special considerations

- Discuss the plan for the management of seizures and emergency airway management in the team brief.
- Ensure that a calm and quiet atmosphere is maintained in theatre. The patient should be draped in a fashion that allows constant access to the patient's airway and minimises the feeling of claustrophobia. Transparent drapes can be used to achieve this.

Acknowledgement

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Intracranial aneurysms

- Aneurysms occur at vessel junctions. Cerebral arteries have a weaker, less elastic muscle layer than systemic vessels.
- The commonest sites are the junction between the anterior cerebral artery and the anterior communicating artery (40%), the bifurcation of the middle cerebral artery (34%) and the junction between the distal internal carotid artery and the posterior communicating artery (20%).[4]
- They are more common in ♀ and 40–60y olds. In 20% of cases, they are multiple. In the UK, the incidence is 6–12/100 000 per year. Autopsy studies show unruptured aneurysms are present in ~6% of the population.[4,5,6]
- Risk factors include hypertension, smoking, positive family history, polycystic kidney disease, cocaine use and connective tissue disorders such as Ehlers–Danlos and Marfan syndromes.[6]
- Aneurysms do not usually rupture until they are >7mm in diameter. They then present as a subarachnoid or an intracerebral haemorrhage.
- Classic symptoms include sudden onset of severe headache with loss of consciousness, which may be transient in mild cases. Occasionally, a patient presents with a focal neurological deficit due to the pressure of an enlarging aneurysm on surrounding structures.
- The World Federation of Neurosurgeons (WFNS) grade of SAH is related to morbidity and mortality (Table 21.1).
- Following aneurysm rupture, definitive management is either by endovascular treatment or surgical clipping. Endovascular treatment, most commonly coiling, is used for the majority of cases.[4]
- Current guidance recommends treatment should be performed to secure the aneurysm within 48h of ictus for patients with WFNS grades 1–3. If presentation is delayed, treatment should occur within 48h of diagnosis. The timing for treatment in poorer-grade patients is less clear.[5,7]

Table 21.1 World Federation of Neurosurgeons grading of SAH

Grade	GCS*	Motor deficit
1	15	–
2	13–14	–
3	13–14	+
4	7–12	±
>5	3–6	±

* See Table 37.4.

Source: data from Drake CG, *et al*. Report of World Federation of Neurological Surgeons Committee on a universal subarachnoid hemorrhage grading scale. *Journal of Neurosurgery*. 1988;**68**:985–6.

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Complications of aneurysmal subarachnoid haemorrhage

Neurological complications

Rebleeding

- Rebleeding is an independent prognostic factor for poor outcome. The risk of rebleeding is 5–10% within the first 72h, with the highest risk period within the first 24h. The overall aim of management is to prevent rebleeding by securing the aneurysm.[5]

Delayed cerebral ischaemia

- Vasospasm is defined as arterial narrowing, demonstrated radiologically, with a corresponding clinical picture. It can be seen in up to 70% of patients on angiography.[4,9]
- Delayed cerebral ischaemia is defined as neurological deterioration caused by ischaemia lasting over an hour that is not attributable to another cause. While delayed cerebral ischaemia can be caused by vasospasm, it can also occur independently of vasospasm.[4]
- Despite treatment, delayed cerebral ischaemia can result in cerebral infarction and death. It is seen in ~22% of patients and is an independent prognostic factor for poor outcome.[5,8]
- Peak incidence occurs at 4–10d after the initial bleed.[4]

Treatment

- Calcium channel blockers: nimodipine is a relatively selective calcium channel antagonist with effective penetration of the blood–brain barrier. It is started at the time of diagnosis and continued for 3w (60mg NG/PO 4-hourly). Alternatively, it can be administered IV (0.5–1mg/h, increasing to 2mg/h) centrally. Nimodipine may cause systemic hypotension, which should be managed aggressively with fluids and, if necessary, vasopressors.
- Classical treatment of delayed cerebral ischaemia involves 'triple H therapy': Hypertension, Hypervolaemia and Haemodilution. Concerns exist with elements of this treatment, including the risk of pulmonary oedema as a result of hypervolaemia and reduced O_2 carriage resulting from haemodilution.
- Current recommendations from the American Heart Association/American Stroke Association are for induced hypertension and maintenance of euvolaemia in the treatment of delayed cerebral ischaemia.[9]
- For severe cases, refractory to medical management, endovascular therapy can be considered. Evidence exists for the use of both intra-arterial vasodilators, such as nimodipine and verapamil, and transluminal balloon angioplasty.[7]

Hydrocephalus

- Blood in the subarachnoid space may obstruct drainage of CSF and result in hydrocephalus and raised ICP. About 30% of patients will develop hydrocephalus requiring CSF diversion.[5,8]
- Hydrocephalus must be ruled out by a CT scan before attributing neurological deterioration to delayed cerebral ischaemia.

Other neurological complications
These include seizures and cerebral oedema.

Medical complications

Life-threatening medical problems occur in a significant proportion of patients. Many of the cardiorespiratory complications following SAH are related to the massive sympathetic surge and catecholamine release that follow SAH.

- Cardiorespiratory complications include:[4]
  - ECG changes ranging from non-specific changes (common) to clinically significant arrhythmias (4–8% of patients)
  - Myocardial injury
  - Ventricular dysfunction
  - Pulmonary oedema.
- Hyponatraemia may result from cerebral salt wasting syndrome or SIADH.[4]
- Other complications include DVT, pneumonia and hepatic, renal and GI dysfunction.

Outcome following subarachnoid haemorrhage

- SAH is associated with significant mortality and morbidity.
- Fifty per cent of patients will die within 1mo of the initial haemorrhage. Only 25% will return to a relatively normal life, the remainder requiring assistance with activities of daily life.[5]
- Unfavourable outcomes are associated with ↑ age, a poor WFNS grade at presentation, rebleeding prior to aneurysm treatment, delayed cerebral ischaemia and the presence of hydrocephalus requiring CSF diversion.[8]

ALGRAWANY

Intracranial aneurysm clipping

Procedure	Clipping of intracranial aneurysm
Time	>3h
Pain	++/+++
Position	Supine, head-up, lateral or prone
Blood loss	200–2000mL, X-match 2 units
Practical techniques	ETT, IPPV, arterial line ± CVP

Clipping an aneurysm involves the use of microsurgery to apply a spring clip across the neck of the aneurysm. Aneurysms arising from branches of the vertebral or basilar arteries require a posterior fossa craniotomy, whereas others may be reached from a frontal or frontoparietal approach.

Preoperative

- Although the procedure can be performed electively to treat unruptured aneurysms, the majority of cases follow an aneurysm rupture. Assess carefully for complications associated with SAH (see pp. 576–7).[10]
- Document preoperative BP, GCS, pupillary response and the presence of any focal neurology.
- Rupture of the aneurysm before it is secured can lead to significant blood loss. Ensure X-matched blood is available.
- Discuss with the surgeon whether temporary flow arrest or temporary clip application is planned. Discuss the plan for management in the event of intraoperative aneurysm rupture.

Perioperative

- As for craniotomy (see pp. 561–2), but note the following.
- The key anaesthetic aim is to maintain cerebral perfusion while avoiding rises in BP that can result in aneurysm rupture. Induced hypotension is no longer performed routinely because of risk of cerebral ischaemia.[7]
- Optimal BP targets remain unclear. One guideline suggests a systolic BP of 110–160mmHg following spontaneous SAH. The authors of this document acknowledge there is limited evidence for this suggestion.[11,12]
- Institute standard monitoring, including invasive BP monitoring, prior to induction.
- A CVP line is not routinely required. Consider insertion in patients with cardiovascular comorbidity or those at high risk of vasospasm postoperatively.[7]
- Ensure adequate venous access with large-bore cannulae.
- Maintain core temperature at 36.5–37.5°C.[13]
- Prior to application of the permanent clip, the surgeon may ask for temporary flow arrest or apply a temporary clip.
- Temporary flow arrest can be achieved using adenosine. If use of adenosine is planned, apply transcutaneous pads to allow pacing if required. Ensure there are no contraindications to adenosine use.[13]

- Alternatively, a temporary clip can be applied to a proximal vessel to control the aneurysm prior to clipping. Induced hypothermia, administration of hypnotics and increasing BP have all been described as ways of minimising cerebral ischaemia following temporary clip application. There is no evidence these techniques improve outcome.[10,13]
- Once the permanent clip is in place, the aneurysm is secure.
- Aim for smooth emergence, avoiding coughing and an associated rise in ICP. This is especially important if the aneurysm has not been definitively secured.[10,13]

Postoperative

- Brain swelling may result from cerebral ischaemia, associated with temporary clip application, or peri-procedural aneurysm rupture. Patients should be transferred ventilated to ICU for ongoing care.[10]
- Patients with an uncomplicated procedure can be extubated and nursed postoperatively on HDU.
- A decrease in the GCS postoperatively may indicate vasospasm, intracranial haematoma or hydrocephalus. Perform a CT scan.

Complications

- Aneurysm rupture occurs most commonly during aneurysm dissection and clipping. Creation of a bloodless field will assist the surgeon in securing the aneurysm:[13]
  - Induce hypotension, targeting a MAP of 50–60mmHg. This can be achieved with a bolus of propofol. This will also lower the cerebral metabolic rate. Alternatively, short-acting agents such as esmolol can be used.[13]
  - The surgeon may ask for temporary flow arrest using adenosine.
  - If depth of anaesthesia monitoring is used, titrate propofol boluses to achieve burst suppression until the aneurysm is secured.[13]
  - Once secured, volume resuscitate with crystalloid and/or blood.
- If rupture occurs prior to aneurysm exposure, signs include a sudden rise in ICP and/or haemodynamic change. Management options include proceeding with surgery or abandoning surgery in favour of imaging ± endovascular treatment. Institute treatment for raised ICP[13] (see pp. 559–60).

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Endovascular treatment of intracranial aneurysms

Endovascular treatment, most commonly coiling, is performed by an interventional neuroradiologist within a radiology suite. Detachable platinum coils are released into the aneurysmal lumen via microcatheters inserted in the femoral artery until occlusion of the aneurysm is achieved. Newer techniques include the placement of stents, as well as coils, allowing more technically challenging aneurysms to be treated endovascularly.[14]

Preoperative

- Treatment may be performed for unruptured and ruptured aneurysms. Patients treated after aneurysm rupture may present with complications of SAH (see pp. 576–7).
- Document preoperative BP, GCS, pupillary response and the presence of any focal neurology.
- If an external ventricular drain (EVD) is *in situ*, note the height of the drain and confirm it is not obstructed. The drain should be closed during patient transfer but set to the correct height and left open during the procedure. If the radiology table moves up or down, the height of the drain will need to be adjusted.

Perioperative

- There is no consensus to the best anaesthetic technique. Some centres perform endovascular coiling in carefully selected patients (compliant patient with good WFNS grade) using conscious sedation. Concerns exist about patient movement, making the procedure both technically more difficult and potentially increasing the risk of peri-procedural aneurysm rupture.[11]
- By minimising patient movement, GA provides better operating conditions for the radiologist. Intubation provides a definitive airway, useful where access to the head is restricted, and allows control of CO_2.
- The key anaesthetic aim is to maintain cerebral perfusion while avoiding rises in BP that can result in aneurysm rupture.
- As with intracranial clipping, optimal BP targets remain unclear.
- Arterial line insertion preinduction aids careful BP control. The femoral artery introducer sheath inserted by the radiologist can be transduced; however, this is placed after induction.
- Induce anaesthesia with propofol, opioid and a muscle relaxant of choice. Fentanyl or remifentanil are used to obtund the pressor response to laryngoscopy.
- Set the ventilator to aim for normocapnia ($PaCO_2$ 4.5–5kPa).[12]
- Obtain large-bore IV access. Insert a temperature probe and use patient-warming devices. As large volumes of contrast and intra-arterial fluid can be given during the procedure, insert a urinary catheter.
- Either TIVA or a volatile can be used for maintenance. Avoid N_2O.
- Remifentanil infusion reduces the risk of coughing. Alternatively, intermittent boluses, or an infusion, of muscle relaxant can be used.
- The procedure is minimally stimulating. Metaraminol boluses or infusion may be required to maintain target BP.

- Once the aneurysm has been successfully treated, the femoral sheath is removed. An artificial collagen plug (e.g. AngioSeal™) can be used to seal the hole in the artery. Alternatively, haemostasis can be achieved by applying manual pressure for 15–20min. The patient should remain anaesthetised until this is achieved.
- Aim for smooth emergence, avoiding coughing and an associated rise in ICP. This is especially important if the aneurysm has not been definitively secured.[14]

Postoperative

- Patients with a reduced postoperative GCS should have a CT scan to exclude hydrocephalus or vascular complications.
- Analgesic requirements are minimal postoperatively.

Complications

- Peri-procedural aneurysm rupture, and the resultant intracranial haemorrhage, can be life-threatening. Intracranial hypertension can cause hypertension, with or without bradycardia. Contrast extravasation may be seen by the radiologist on imaging.
  - Aim to reverse the heparin and reduce MAP to the level before the bleed.
  - Institute treatment of raised ICP (see pp. 559–60).
  - If the extravasated blood load is significant, an EVD may be required, if not already in place. Craniotomy, evacuation of intracranial haematoma and aneurysm clipping may also be required.[14]
- Other complications include intraoperative vasospasm and vascular occlusion due to arterial thromboemboli and misplaced coils.[14]

Special considerations

- Unfamiliar environment, remote site, radiation, radiology equipment, contrast media, heparin, antiplatelet drugs and thrombolysis.

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Endovascular thrombectomy

Procedure	Interventional neuroradiological procedure where occlusive thrombus is aspirated in acute CVE
Time	1–2h
Pain	+
Position	Supine
Blood loss	Usually minimal
Practical techniques	LA, conscious sedation, GA (ETT, arterial line)

Introduction

- Acute ischaemic CVE accounts for ~85% of all cases of CVE.[15]
- A proportion of patients with acute ischaemic CVE will be eligible for endovascular thrombectomy. This is an interventional procedure where the thrombus is aspirated, resulting in cerebral reperfusion.[15]
- The decision to proceed with endovascular thrombectomy is based on the time since onset of symptoms, severity of CVE, location of the thrombus within the cerebral circulation and pre-existing functional status of the patient.[15]

Preoperative

- This is a true time-critical emergency—the greater the duration of time before vessel recanalisation, the greater the degree of cerebral infarction.
- Challenges can include:
  - Acute effects such as dysphasia and hemiparesis
  - Chronic comorbidities such as hypertension, AF, DM
  - Fasting status
  - Remote location of the interventional radiology suite.
- Options for anaesthesia include LA, with or without conscious sedation, and GA.
- Three single-centre RCTs comparing GA with conscious sedation for endovascular thrombectomy have produced equivocal results. Meta-analyses of these trials suggest GA is associated with better functional outcome.[15,16,17]

Perioperative

- For cases under GA:
  - Intubation and ventilation allow airway protection and control of CO_2.
  - Co-induction with opioids allows dose-sparing of propofol and obtunds the pressor response to laryngoscopy.
  - Invasive BP monitoring is recommended prior to induction. However, this should not delay the start of the procedure.
  - Either TIVA or a volatile can be used for maintenance.

- For cases under sedation:
  - Options for sedation include midazolam, dexmedetomidine, remifentanil or propofol TCI.[15]
  - Titrate sedation to patient response, aiming for a patient who is comfortable and easily rousable.
  - Administer supplemental O_2 and attach capnography to monitor respiratory effort.
- Irrespective of the anaesthetic technique, it is essential to maintain haemodynamic stability. Excessive hypotension will reduce collateral blood flow to the ischaemic penumbra and worsen the infarct; excessive hypertension can cause haemorrhage.
- Limited data exists concerning perioperative blood pressure management. Guidance from 2014 suggested systolic BP targets are 140–180mmHg if the patient has been thrombolysed, and 140–220mmG if not. More recently, a retrospective analysis has suggested a target MAP of 70–90mmHg is associated with better outcomes.[18,19]
- For all patients, maintain SpO_2 between 94% and 98% and maintain normocapnia.[18]

Postoperative

- Provided the procedure is uncomplicated, most patients can be cared for postoperatively on a hyperacute stroke unit after recovery.[15]
- Patients who had a reduced GCS preoperatively, or who have poor gas exchange precluding extubation, may require a period of postoperative ventilation.
- At the end of the procedure, a target postoperative BP should be specified. Patients may require vasopressor support to achieve this target. This may also necessitate ongoing care in a higher dependency area.

Special considerations

- As per endovascular treatment of aneurysms (see ➔ pp. 580–1)
- Basilar artery occlusions, which can interrupt the blood supply to the brainstem, are more commonly associated with a reduced level of consciousness and/or signs of brainstem dysfunction.

Acknowledgement

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Venous air embolism

- VAE is the entrainment of air into the venous circulation, leading to systemic effects.
- The volume of air entrained to cause significant injury is unknown but estimated to be 3–5mL/kg in adults. The greater the rate of entrainment, the smaller the lethal volume.
- VAE can occur whenever the operative site is higher than the right atrium. Its incidence is particularly high during craniotomy in the sitting position and when the surgeon is dissecting tissues that do not allow veins to collapse despite a negative pressure within them (e.g. the emissary veins in the posterior fossa).
- There are three main consequences of VAE:
  - Pulmonary microvascular occlusion, resulting in an ↑ physiological dead space, hypoxia and pulmonary oedema
  - Air bolus (larger volume) causing obstruction to RV ejection, leading to pulmonary arterial hypertension and RV failure
  - Paradoxical air embolism (see below).
- Signs of VAE depend on the rate and volume of air entrained and include: decrease in $ETCO_2$, bronchoconstriction, hypoxia, arrhythmias, myocardial ischaemia, hypotension and cardiac arrest.
- N_2O does not increase the risk of VAE but may worsen its outcome.

Detection of venous air embolism

- $ETCO_2$ is generally the most useful monitor, as it is widely available and sensitive. Air embolism results in a sudden reduction in $ETCO_2$. Hyperventilation, low CO and other types of embolism will also result in $ETCO_2$ reduction.
- Doppler ultrasound uses ultra high-frequency sound waves to detect changes in blood flow velocity and density. It is the most sensitive non-invasive monitor; however, it is not quantitative and does not differentiate between a massive or a physiologically insignificant air embolism.
- TOE is the most sensitive and specific monitor, but is invasive and difficult to place, and needs expertise to interpret. However, it can detect as little as 0.02mL/kg of air.
- PA catheters are invasive, but sensitive monitors for VAE. However, an increase in PAP is not specific for air and they are very rarely sited.
- The least sensitive monitor is a precordial or an oesophageal stethoscope to detect a 'millwheel' murmur. This is apparent only after massive VAE, which is usually clinically obvious.

Prevention

- Avoid the sitting position, unless essential.
- Elevate the head only as much as necessary.
- Ensure adequate blood volume to maintain a positive CVP.
- Small amounts of PEEP (5–10cmH_2O) may reduce the risk of air entrainment, although controversial.
- Medical antishock trousers may be used to increase VP and reduce hypotensive episodes in patients in the sitting position.
- Identify susceptible patients, if risk factors cannot be mitigated.

Treatment

- Treatment is supportive.
- Inform the surgeon, who should flood the operative field with fluid. This stops further entrainment of air and allows the identification of open veins that can be cauterised or waxed if within bone.
- If possible, position the operative site below the level of the heart to increase VP.
- Stop N_2O immediately if used, and increase the FiO_2 to 1.0.
- Consider increasing PEEP (not with patent foramen ovale).
- Support the BP with fluid and vasopressors.
- Attempt to aspirate air from the CVP line. The tip should be placed close to the junction of the SVC and the right atrium, and ideally confirmed radiologically prior to surgery.
- If a large volume of air has been entrained and surgical conditions permit, turn the patient into the left lateral decubitus position to attempt to keep the air in the right atrium. Or alternatively the Trendelenburg position. There is little evidence for this.
- Commence CPR, if necessary. This may break down a larger air bubble.

Paradoxical air embolism

- Air emboli may enter the systemic circulation through the Thebesian veins in the heart, the bronchial vessels or a patent foramen ovale. Such defects may be small.
- Small volumes of air in the systemic circulation can have disastrous consequences, resulting in temporary or permanent symptoms of CVE or ischaemia of other organs.
- Intracardiac septal defects are an absolute contraindication to surgery in the sitting position. This should be screened preoperatively in high-risk cases.

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Resuscitation in neurosurgery

- Cardiac arrest during anaesthesia for non-cardiac surgery occurs with an incidence of 0.01–0.34%.
- Factors such as type of neurosurgical procedure and patient positioning can have a significant effect on the occurrence of cardiac arrest.
- Appropriate management may need immediate attention to the underlying cause.[20]

Specific factors influencing CPR

The surgical procedure

- Procedures on the anterior hypothalamus, brainstem, cerebello-pontine angle, pituitary and trigeminal nerve can cause arrhythmias.
- Generally, severe bradycardia with hypotension and potentially asystole can occur, caused by the trigeminocardiac reflex.
- Caused by surgical traction/instrumentation—temporarily ceasing this often leads to resolution.
- If bradycardia persists, atropine (500–600 micrograms up to 3mg) can be administered, or glycopyrronium bromide (200–400 micrograms). Adrenaline may be required if no response, and CPR if asystole.
- VAE can lead to cardiovascular collapse and cardiac arrest (see pp. 584–5).

The position of the patient

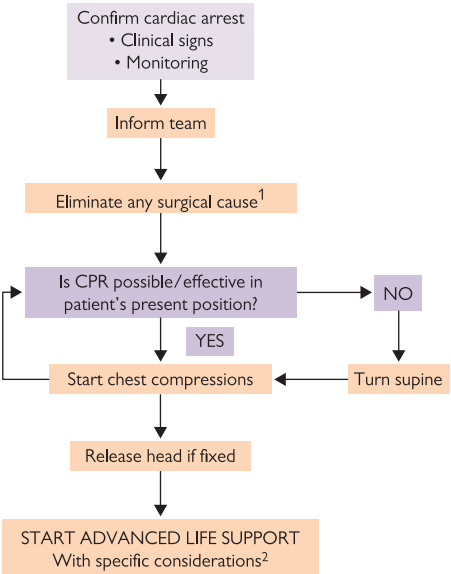
- Neurosurgical positioning can present significant challenges during cardiovascular resuscitation.
- Common positions include supine, lateral/park bench, prone and occasionally sitting.
- The Mayfield 3-point head fixator is frequently used and the clamp needs to be released before chest compressions commence, to avoid damage to the cervical spine and skull (fixed head and mobile torso).
- Patient positioning will need to be adjusted to safely allow CPR and defibrillation to occur.
- CPR can be commenced in the prone position initially. Adequacy can be determined from $ETCO_2$ and arterial line monitoring (often *in situ*). Turning supine will take time and personnel.
- Defibrillation pad positions: standard pad position if patient supine/sitting; anteroposterior position if lateral; and posterolateral or biaxillary position if prone.

Wound management

- The open wound needs to be expediently managed by surgeon.

Special management considerations

- Diagnosis and management of cardiac arrest should be managed as per advanced life support (ALS) and specific Cardiac Arrest in Neurosurgery algorithm (Fig. 21.3).
- Variation from the ALS algorithm is an initial dose of adrenaline. Give IV aliquots of 50–100 micrograms up to 1mg initially. This will prevent rebound hypertension and potential haemorrhage if resuscitation is successful. Further doses are given at 1mg.



SURGICAL CONSIDERATIONS

- Eliminate any surgical cause[1]
  - Asystole: retraction, instrumentation
  - Venous air embolism: flood field with saline, put patient head down if possible
- Attend to wound:
  - Control haemorrhage
  - Protect against trauma
  - Minimise contamination
- Stop surgery as soon as possible

SPECIFIC CONSIDERATIONS FOR ALS[2]

- Ensure correct tracheal tube position and ventilation
- Ventilate with 100% oxygen
- Get senior help
- Follow major haemorrhage protocol
- Give initial adrenaline doses as 50-100μg boluses IV
- Echocardiography to check for VAE

Fig. 21.3 Cardiac Arrest in Neurosurgery algorithm. Reproduced with the kind permission of Resuscitation Council UK

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Further reading

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Chapter 22

Vascular surgery

Mark Stoneham

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See also

➲ Ascending aorta and aortic arch surgery pp. *521–2*

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General principles

Vascular surgery usually involves operating on arteries diseased by atherosclerosis, causing poor peripheral blood flow (ischaemia) or emboli. Mortality is high; elective abdominal aortic aneurysm (AAA) surgery has a mortality of 2.4%,[1] while that of ruptured AAA is 30–40%. Operations may be long and involve blood transfusion, marked fluid shifts and significant impairment of lung function. In the UK, all major vascular operations now take place in designated 'vascular centres'.

- Vascular patients are usually elderly arteriopaths with significant associated disease. Hypertension (66%), IHD (angina, MI), heart failure, DM and COPD (many are current or ex-smokers) are common. Many patients are taking aspirin, β-blockers, diuretics, heart failure medication, insulin and/or oral hypoglycaemics.
- Some patients are anticoagulated; others will receive anticoagulants perioperatively—consider the pros and cons of regional techniques carefully (see ➲ p. 591). However, regional techniques can reduce morbidity and mortality (see ➲ p. 1109).
- Vascular patients tend to have serial operations, so there may be several previous anaesthetic records to review. A significant minority of vascular operations occur out of hours and these are often high-risk procedures in high-risk patients.
- Measure NIBP in both arms as these may differ due to arteriopathy (use the higher BP side clinically; put your arterial line in this side).
- All patients require prophylactic antibiotic cover.
- Develop a working relationship with your vascular surgeon; you will have a better chance of being warned of untoward events (e.g. aortic clamping/unclamping, sudden massive blood loss, etc.). Vascular surgery is the only anaesthetic subspecialty where the presence of a specialist consultant vascular anaesthetist has been shown to reduce long-term mortality of major vascular surgery. The most likely cause for this is better teamwork.[2]

Preoperative assessment

- Quantify the extent of any cardiorespiratory disease, both in terms of the planned surgical procedure and the postoperative period. Carefully consider (and document) if regional anaesthesia is appropriate.
- Include direct questions about exercise tolerance (walking distance on the flat, ability to climb stairs) and the ability to lie supine. Look for signs of cardiac failure.
- Investigations: FBC, U&E, coagulation, LFTs, ECG and CXR.

Premedication

Continue β-blockers and statins perioperatively. Anxiolytic premedication may be useful for major surgery.

Regional anaesthesia and analgesia in vascular surgical patients

Regional anaesthesia may be used alone for distal vascular surgery and is commonly used for carotid surgery. Epidural analgesia has been recommended by NICE to supplement GA for AAA. The advantages of regional techniques include:

- Improved patient monitoring (CEA)
- Reduced hospital stay and cost (CEA)
- Improved blood flow, reduced DVT, reduced reoperation (peripheral revascularisation)
- Postoperative pain relief (AAA, distal revascularisation, amputation)
- Reduced pulmonary complications (AAA)
- Pre-emptive analgesia for amputations—possible reduction in phantom limb pain
- Treatment of proximal hypertension during aortic cross-clamp.

Epidural catheters and anticoagulation

(See ➲ p. 1109.)

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Abdominal aortic aneurysm repair

Procedure	Excision of aortic aneurysmal sac and replacement with synthetic graft (tube/trouser graft)
Time	2–4h
Pain	++++
Position	Supine, arms out (crucifix)
Blood loss	500–2000+ mL, X-match 6 units. Suitable for cell salvage
Practical techniques	ETT + IPPV, arterial + CVP lines. CO monitoring. Epidural/spinal morphine for postoperative pain relief

Preoperative

- The elderly often have multiple coexisting diseases.
- The 30d mortality for elective open surgery is 2.4% (predominantly MI and multiorgan failure).
- A dynamic assessment of the cardiac function is required for all elective aortic surgery and for any patients with symptomatic/new cardiac disease. CPET is the 'gold standard' for all patients undergoing AAA repair (see pp. 33–5). Refer patients with critical IHD to cardiology for angiography and possible coronary revascularisation before aortic surgery. Emergent vascular patients may have to undergo surgery before such dynamic investigations.
- PFTs (including ABG analysis while breathing air) should be performed in patients with significant respiratory disease.
- Careful preoperative assessment is essential. Scrutinise the ECG for signs of ischaemia, and check for any renal impairment. Check access sites for CVC and arterial line.
- HDU/ICU for postoperative care. Alert the patient to this plan, especially if a period of postoperative IPPV is planned. Preoptimisation is performed in a few units but is not widespread—patients are admitted to the HDU/ICU preoperatively to have lines, etc. inserted and to have the haemodynamic status 'optimised'.
- Continue the usual cardiac medications perioperatively.

Perioperative

- Have available vasoconstrictors (ephedrine and metaraminol), vasodilators (GTN) and β-blockers (labetalol).
- Two 14G or greater IV access.
- Monitor intraoperative temperature and be obsessive about temperature control from the start. Avoid heat loss, as it is easier to keep a patient's temperature constant than to try to increase it. Use a fluid warmer (ideally fast-flow) and a forced air warmer (avoiding the lower limbs while the aortic cross-clamp is in place, as this may worsen limb ischaemia).
- There is no good evidence supporting the use of isovolaemic haemodilution. Cell salvage should be mandatory—there is good evidence that it reduces usage of allogeneic blood in aortic surgery.

- Arterial line and thoracic epidural (T6–T11) preinduction. Take a baseline blood gas some time before cross-clamping.
- Have syringe drivers available for inotropes and vasodilators.
- Use 5-lead ECG (leads II and V_5) to increase the sensitivity of detection of myocardial ischaemia.
- Multiple-lumen CVC after induction. Consider inserting a PAFC introducer to the right internal jugular or left subclavian vein in complex cases, as this will allow rapid fluid administration and facilitate PA catheter insertion if necessary.
- Continuous CO monitoring is useful during the cross-clamp period for all patients, particularly those with impaired cardiac function. Possibilities include: PA catheter, LiDCO™, PiCCO™ and oesophageal Doppler; however, the latter is not accurate during aortic cross-clamping.
- Careful induction with intra-arterial BP monitoring. Use moderate/high-dose opioid, e.g. remifentanil (0.1–0.2 micrograms/kg/min) or high-dose fentanyl (5–10 micrograms/kg). Treat hypotension with fluids at first and then with cautious vasoconstriction (metaraminol 0.25–0.5mg). There is no difference in cardiac outcome between sevoflurane-based anaesthesia and TIVA.[3]
- Insert a urinary catheter for hourly measurements of urine output.
- Heparin 3000–5000 units is usually given just before cross-clamp. This may be reversed after unclamping with protamine 0.5–1mg per 100 units heparin IV slowly—hypotension results if given too quickly.
- Proximal hypertension may follow aortic cross-clamping and is due to a sudden increase in SVR, ↑ SVC flow and sympatho-adrenal response. Treat by deepening the anaesthesia and/or with a bolus of β-blocker (labetalol 5–10mg), GTN infusion or epidural LA.
- While the aorta is clamped, metabolic acidosis will develop due to ischaemic lower limbs. Maintaining the minute ventilation will cause respiratory alkalosis to develop, which will minimise the effects of this metabolic acidosis when the aorta is unclamped. Check ABGs to assess Hct, metabolic acidosis, respiratory compensation and ionised Ca^{2+}.
- Cross-clamp time is usually 60+min. During this time, start giving fluid, aiming for a moderately ↑ CVP (5cmH_2O greater than the baseline) by the time unclamping occurs. This helps CVS stability, reduces sudden hypotension and may help preserve the renal function. Release of the cross-clamp one limb at a time also helps haemodynamic stability.
- Hypotension following aortic unclamping is caused by ↓ SVR, relative hypovolaemia and myocardial 'stunning' due to the return of cold metabolic waste products from the legs. Treat with IV fluids and/or lighten the anaesthetic depth and/or with small doses of inotropes, e.g. adrenaline 10-microgram aliquots (1mL of 1:100 000) and/or a bolus of calcium gluconate (up to 10mL of 10%). Inotropes may be needed postoperatively.
- For fluid replacement, give isotonic crystalloid or colloid to replace insensible, 3rd space and initial blood loss. Give blood products when a deficiency is identified, e.g. Hct <25%, platelets <100 × 10^9/L. Check the ACT (normal <140s) if you suspect coagulopathy. TEG® will give you the whole coagulation picture.
- TOE may provide useful data during the cross-clamp period.[4]

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Postoperative

- ICU/HDU is essential postoperatively. HDU may be appropriate for otherwise fit patients who are warm and haemodynamically stable, and with a working epidural. Otherwise transfer to ICU intubated.
- Opioid infusion and/or PCA if no epidural. Routine observations of arterial and CVP monitoring, distal pulses and urine output should be continued postoperatively to assess haemodynamic stability. There is potential for large fluid shifts which need replacement.

Special considerations

- Management of epidural: a bolus of epidural diamorphine 2–3mg at induction will last for 12–24h. Use epidural LA sparingly until the aorta is closed. It is easier to treat the hypotension of aortic unclamping with a functioning sympathetic nervous system.
- Renal failure occurs in 1–2% of cases, is multifactorial in origin and is associated with a mortality of 50% following AAA repair. It is more likely if the cross-clamp is suprarenal. There is no evidence that dopamine prevents renal failure, merely acting as an inotrope. Mannitol is used routinely by some (0.5g/kg during cross-clamp) as a free radical scavenger and an osmotic diuretic. Avoid hypovolaemia, and monitor the urine output hourly.

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Emergency open repair of abdominal aortic aneurysm

A true anaesthetic and surgical emergency. It may be:

- Acute: presents with CVS collapse. Death is likely, unless the rupture is contained in the retroperitoneal space
- Dissecting: dissects along the arterial intima, presenting with back/abdominal pain.

Prehospital mortality for ruptured AAA approaches 50%, and half of those reaching hospital also do not survive. Overall mortality is 40%.

The IMPROVE trial compared open and endovascular repair for ruptured AAA and found little difference in outcome, so many centres are now preferring endovascular aneurysm repair (EVAR) for ruptured AAA.[5]

Management of open ruptured AAA is as for elective AAA, with the following additional considerations:

- Where doubt exists and the patient is haemodynamically stable, the diagnosis is confirmed by ultrasound or CT scan.
- If hypovolaemic shock is present, resuscitate to a systolic pressure of 90mmHg. Avoid hypertension, coughing and straining, as this may precipitate a further bleed. Titrate IV morphine against pain.
- Preinduction, insert two 14G peripheral cannulae and (ideally) an arterial line. Use of the brachial artery may be necessary, and sometimes an arterial 'cut-down' is indicated. Central venous access can wait until after the cross-clamp is applied. If peripheral IV access is difficult, insert a PAFC introducer into the right internal jugular vein.
- Epidural analgesia is usually inappropriate.
- A urinary catheter can be placed before or after induction.
- Induction must be in theatre, with the surgeons scrubbed, surgical preparation completed, drapes on and blood available in theatre and checked. RSI is usually required. Suitable induction agents include midazolam/remifentanil, etomidate (also give hydrocortisone 50–100mg) and ketamine. As soon as endotracheal intubation is confirmed, the surgeons can begin. Treat hypotension with IV fluids and small doses of vasopressors/inotropic agents.
- Hot air warming and at least one warmed IVI are essential (a fast-flow blood warmer is invaluable).
- Use a colloid or crystalloid, depending on preference. Use a balanced crystalloid such as Hartmann's solution, rather than 0.9% sodium chloride (helps prevent metabolic acidosis).
- Have both IV lines running maximally at induction. One assistant should be dedicated to managing IV fluid and ensuring an uninterrupted supply. Once the cross-clamp is applied, some haemodynamic stability may be restored.
- Cell salvage, if available, is mandatory.
- Hypothermia, renal impairment, blood loss and coagulopathy are common perioperative problems. Hypothermia is a particular hazard, as bleeding postoperatively is likely (platelet function is markedly reduced below 35°C). While there is no place for routine administration of platelets and FFP, consider early use when needed.

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- Do not attempt to extubate at the conclusion of surgery—a postoperative period of ventilation on the ICU is essential to allow the correction of biochemical/haematological abnormalities.
- Use near-patient testing (Hb and TEG®), if available, to guide blood product administration. If the patient is exsanguinating and X-matched blood is not available, use type-specific.

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Endovascular stenting of elective or emergency abdominal aortic aneurysm

Procedure	Placement and deployment of bifurcated stent by interventional radiologists into aortic aneurysmal sac via femoral arteries
Time	1–4h
Pain	+
Position	Supine
Blood loss	0–2000+ mL, X-match 6 units
Practical techniques	Spinal + sedation, arterial line

This technique has become widely adopted in the last 15y. It is certainly associated with lower operative morbidity and mortality than standard open AAA repair,[6] but it is still unproven whether it lowers the risk of aneurysm rupture; thus, postoperatively, patients must be kept under CT surveillance for the rest of their lives. Significant complications, such as migration of the stent and endoleak, can develop, as well as rupture of the original aneurysm sac.

- The procedure is performed in the angiography suite with the interventional radiologists. In many hospitals, vascular surgeons gain access to the aorta via the femoral arteries, and the stent is inserted by an interventional radiologist, although all the procedure may be done by one or the other. Most straightforward EVAR procedures are done percutaneously; thus, LA may be suitable.
- During elective EVAR, if the aneurysm ruptures (incidence is around 2%), mortality rises to >50%.
- Preassessment, monitoring and X-matching are all exactly as for an open repair. However, since the patient will not undergo aortic cross-clamping, patients who have been refused open surgery because of significant LV impairment may tolerate EVAR. ICU is usually not needed postoperatively. Guidelines for who should be offered EVAR or open repair are still under development.
- GA, regional anaesthesia or LA is appropriate, depending on preference, although regional anaesthesia/LA may shorten the procedure and hospital stay.[7,8]
- Postoperatively, the patient may go to the HDU or the vascular ward for overnight monitoring. Hospital stay is usually only 24–48h.
- Complex EVAR procedures in which custom-made, fenestrated grafts allow reconnection of visceral aortic branches may take longer than standard EVAR and are usually therefore best performed under GA.
- EVAR is also now an increasingly popular technique for patients presenting with ruptured AAA. A balloon is inserted into the femoral artery percutaneously and inflated above the rupture which confers some haemodynamic stability. The IMPROVE Trial showed similar 30d mortality between open (37.4%) and endovascular (35.4%) techniques, but with women faring better than men with the EVAR technique. Hospital costs were marginally lower for the EVAR group.[5]

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- EVAR for ruptured AAA may be performed under GA or LA. A commonly used combination technique involves the inflation balloon being inserted under LA to prevent haemodynamic collapse, followed by induction of GA once haemodynamic stability improves.

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Thoracoabdominal aortic aneurysm repair

Procedure	Excision of aortic aneurysmal sac extending above the origin of the renal arteries and replacement with a synthetic graft. May involve thoracotomy and the need for OLV
Time	3–6h
Pain	++++
Position	Supine, arms out (crucifix), may be right lateral if thoracotomy
Blood loss	1000mL – +++, X-match 8 units, plus platelets and FFP
Practical techniques	DLT + IPPV, arterial + CVP lines. Thoracic epidural

Thoracic aneurysms of the ascending aorta require median sternotomy and CPB. Transverse aortic arch repair often requires hypothermic circulatory arrest as well.

In many vascular centres, most patients with thoracic AAA undergo endovascular repair which has many similarities to EVAR.

However, precautions to protect the spinal cord, such as spinal fluid drainage, are commonly performed for thoracic EVAR.

Thoracic EVAR operations are usually carried out with the patient under GA.

Special considerations

As for infrarenal aortic aneurysm repair, with the following considerations:

- The aneurysm may compress the trachea and distort the anatomy of the upper vasculature.
- Intensive care is essential for postoperative ventilation and stabilisation, until acidosis and hypothermia are corrected and the lungs fully re-expanded.
- The aortic cross-clamp will be much higher than for a simple AAA. This means that the kidneys, liver and splanchnic circulation will be ischaemic for the duration of the cross-clamp.
- Access to the thoracic aorta may require OLV, so a left-sided DLT may be required (see pp. 535–7). A Univent® tube is a possible alternative (see p. 537).
- Proximal hypertension following aortic cross-clamping is more pronounced. Use aggressive vasodilation with GTN (infusion of 50mg/50mL run at 10mL/h until it starts to work) or esmolol (2.5g/50mL at 3–15mL/h).
- Hypotension following aortic unclamping is often severe, requiring inotropic support postoperatively—use adrenaline (5mg/50mL), starting at 5mL/h.
- Acidosis is a particular problem—metabolic acidosis develops during cross-clamping and is potentially exacerbated by respiratory acidosis due to prolonged OLV. Use balanced crystalloids; consider using HCO_3^- and ventilate postoperatively until it is resolved.

ALGRAWANY

- Renal failure occurs in up to 25% of cases; principally related to the duration of cross-clamping. Monitor urine output; give mannitol 25g before cross-clamping, and maintain the circulating volume.
- Spinal cord ischaemia, leading to paralysis, may develop. This is related to the duration of cross-clamping and occurs because a branch of the thoracic aorta (artery of Adamkiewicz) reinforces the blood supply of the cord. Techniques used for prevention (none is infallible) include: CSF pressure measurement and drainage through a spinal drain, spinal cord cooling through an epidural catheter, intrathecal Mg^{2+}, distal perfusion techniques, CPB and DHCA. Surgeons performing this surgery have their own preferred techniques.
- Fluid balance is as for infrarenal AAA, although blood loss will be more extreme; blood transfusion will almost certainly be required, and platelets and FFP are more commonly used. Cell salvage is mandatory.

Carotid endarterectomy

Procedure	Removal of atheromatous plaque from the internal carotid artery. The internal carotid artery is clamped and opened, the plaque removed and the artery closed directly or with a patch
Time	1–3h
Pain	++
Position	Supine, head-up. Contralateral arm board
Blood loss	Minimal, G&S
Practical techniques	Cervical plexus block + sedation, or ETT + IPPV, arterial line

An operation to reduce the incidence of CVE in symptomatic (TIA or cerebrovascular accident) patients with >70% internal carotid artery stenosis. Combined perioperative mortality and cerebrovascular accident incidence of 1–5%. Patients usually elderly arteriopaths, but preoperative dynamic cardiac assessment is not required.

- Monitoring cerebral perfusion during carotid cross-clamping is important. Advocates of regional anaesthesia cite the advantages of having a conscious patient in whom neurological deficits are immediately detectable.
- Under GA, other techniques are used for monitoring cerebral perfusion such as EEG processing, somatosensory evoked potential monitoring, transcranial Doppler of the middle cerebral artery and near-infrared spectroscopy. Each unit will have its own protocols.
- Considerable controversy exists as to whether to use GA or regional anaesthesia. The largest RCT ever carried out comparing the two anaesthetic techniques failed to show any difference in outcome.[9] Teamwork is probably the most important factor in reducing surgical risk, rather than any particular technique.

Preoperative

- Elderly patients, often with severe CVS disease. Most are hypertensive. BP control during CEA can be difficult.[10]
- Determine the normal range of BP from ward charts. Measure BP in both arms. Use the highest of the two, and aim for 160/90mmHg.
- Document pre-existing neurological deficits, so that new deficits may be more easily detected.
- Have available vasoconstrictors (ephedrine and metaraminol) and vasodilators (GTN, labetalol).
- Premedicate with a sedative or anxiolytic, particularly if using GA.

Perioperative

- IV access (20G and 14G) plus an arterial line in the contralateral arm (out on an arm board).
- Monitoring: 5-lead ECG, arterial line, NIBP, SpO_2, $ETCO_2$.
- Maintain BP within 20% of baseline. During cross-clamping, maintain BP at or above baseline. If necessary, use vasoconstrictors, e.g. metaraminol (10mg diluted up to 20mL; give 0.5mL at a time).

ALGRAWANY

General anaesthesia for carotid endarterectomy

- Careful IV induction. BP may be labile during induction and intubation. Give generous doses of short-acting opioids, and consider spraying the cords with lidocaine.
- Most anaesthetists use an ETT; the LMA cuff has been shown to reduce carotid blood flow, but this is of unknown significance. Secure the tube, and check connections very carefully (the head will be inaccessible during surgery).
- Remifentanil infusion, combined with superficial cervical plexus block, gives ideal conditions, with rapid awakening. Otherwise isoflurane/opioid technique. Maintain normocapnia. Avoid N_2O.
- Extubate before excessive coughing develops. Close neurological monitoring in recovery until fully awake.

The 'awake carotid'

- Patient preparation and communication are vital.
- Cervical dermatomes C2–C4 may be blocked by deep, intermediate or superficial cervical plexus block performed using ultrasound or by anatomical methods[11] (see ➔ p. 1118).
- Avoid deep block in patients with respiratory impairment, as they may not tolerate unilateral diaphragmatic paralysis. The learning curve for the superficial block is steeper than the deep, but more LA is typically used and all blocks have complications described. Infiltration along the jawline helps to reduce pain from the submandibular retractor.
- Ensure the patient's bladder is emptied preoperatively. Give IV fluids only to replace blood loss; a full bladder developing, while the carotid is cross-clamped, can be tricky to manage.
- Sedation (e.g. remifentanil 0.05–0.1 micrograms/kg/min) may be carefully employed during block placement and dissection. Once dissection is complete, patient discomfort is reduced, so sedation can be stopped. Avoidance of sedation during carotid cross-clamping will allow better neurological assessment. Give O_2 throughout.
- An L-bar angled over the patient's neck allows good access.
- Despite an apparently perfect regional block, ~50% of patients will require LA supplementation by the surgeon, particularly around the carotid sheath. This is reduced using remifentanil sedation and can also be treated by direct lidocaine spray once dissection is complete.
- Monitor the patient's speech, cerebration and contralateral motor power while the carotid is cross-clamped. Neurological symptoms and signs are usually similar to those with which the patient presented.
- Neurological deficit presents in three ways:
  - Profound unconsciousness on cross-clamping
  - Subtle, but immediate, deficit following cross-clamping, e.g. confusion, dysphasia, delay in answering questions
  - Delayed deficit, usually related to relative hypotension.
- Attentive monitoring of the patient is vital, particularly during cross-clamping. If a neurological deficit develops, try augmenting the BP which may improve ipsilateral cerebration by increasing the pressure gradient across the circle of Willis. Increase the inspired O_2 concentration. Tell the surgeon immediately who will get a carotid shunt ready. Recovery should be rapid, once the shunt is in place. If not, convert to GA (use of an LMA probably easiest at this stage).

- For patients who do not tolerate being awake, GA is the best option, although techniques have been described in which the patient is 'woken up' intraoperatively to check neurological function.

Postoperative

- Careful observation in a recovery room for 2–4h is mandatory. HDU is preferable for patients who develop a neurological deficit.
- Airway oedema is common in both GA and regional cases, due to dissection around the airway. Cervical haematoma occurs in 5–10% of cases—remove skin sutures in recovery to allow drainage. Immediate re-exploration is required for developing airway obstruction. Although the block may still be working, plan for emergent control of the airway.
- Haemodynamic instability is common postoperatively. Hyperperfusion syndrome, typically presenting as headaches which may lead ultimately to haemorrhagic CVE, is caused by areas of the brain previously 'protected' by a tight carotid stenosis being suddenly exposed to hypertensive BP. Thus, BP must be tightly controlled. Careful written instructions should be given to staff about haemodynamic management. An example is:
  - If systolic BP >160mmHg, give labetalol 5–10mg boluses IV or a hydralazine infusion
  - If systolic BP <100mmHg, give colloid 250mL stat.
- New neurological symptoms and signs require immediate surgical consultation and investigation.
- Carotid stenting is a developing procedure for symptomatic carotid patients performed in the radiology suite, in which a stent is placed under LA into the stenotic carotid artery. Anaesthetic supervision may be required because of the complications, which include perioperative CVE and haemodynamic disturbances. CVE rates of carotid stenting remain higher than of operative intervention.

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10 Stoneham MD, Thompson JP (2009). Arterial pressure management and carotid endarterectomy. *Br J Anaesth*, **102**, 442–52.
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ALGRAWANY

Peripheral revascularisation

Procedure	Bypass operations for occlusive arterial disease. A vein or a synthetic graft is used to bypass occluded arteries
Time	1–6h
Pain	+++
Position	Supine
Blood loss	Usually 500–1000mL, X-match 2 units
Practical techniques	Combined spinal/epidural with sedation, arterial line. ETT/IPPV, consider LMA

- Femoro-popliteal bypass = femoral to above-knee popliteal artery.
- Femoro-distal bypass = femoral to anterior or posterior tibial artery.
- Femoro-femoral crossover = from one femoral artery to another.

Preoperative

- Constitute a large proportion of elective vascular surgery.
- Duration of surgery is unpredictable and overruns are not uncommon.
- Assess the CVS. Usually better tolerated than aortic surgery. A dynamic assessment of the cardiac function is not usually necessary, unless there have been new developments, e.g. unstable angina.
- The choice between GA and regional anaesthesia is up to the individual. There is a suggestion that regional anaesthesia is associated with lower reoperation rates. Long operations (>3h) mean regional techniques need careful thought.

Perioperative

- IV access: ensure at least one large (14 or 16G) IV cannula.
- Use arterial pressure monitoring unless it is a very short procedure. Otherwise standard monitoring with 5-lead ECG. CVC not required.
- GA techniques include ETT plus IPPV or LMA plus SV. The surgeon should be able to perform femoral nerve block perioperatively.
- Regional anaesthesia offers good operating conditions and postoperative analgesia. Single-shot spinal anaesthesia with plain bupivacaine, intrathecal clonidine (15–30 micrograms) and diamorphine (250–500 micrograms) can give good surgical anaesthesia for up to 6h. If using epidural anaesthesia, epidural diamorphine (2–3mg) and an infusion of 0.25% bupivacaine at 5–10mL/h will provide similar. Put the spinal in, with the patient sitting, so they can hang their leg off the bed, which will reduce their pain until the block is working. Give supplemental O_2. If the patient requests sedation, propofol TCI is ideal.
- Heparin (3000–5000 units) should be given before clamping—reverse with protamine 0.5–1mg/100 units of heparin slowly after unclamping.

Postoperative

- O_2 overnight.

Axillobifemoral bypass

Procedure	Extraperitoneal bypass (trouser graft) from axillary artery to femoral arteries
Time	2–4h
Pain	++++
Position	Supine
Blood loss	<1000mL, X-match 2 units
Practical techniques	GA—ETT, IPPV, arterial line, consider CVC

This operation is performed less commonly due to the rapid advance of stenting techniques; however, it is still occasionally performed on patients with completely occluded aortoiliac vessels. It is a last-chance operation for patients with completely occluded aortic or iliac arteries. Some will already have had aortic surgery and have infected grafts. It is an extraperitoneal operation, so patients with severe cardiorespiratory disease who might be excluded from aortic surgery may tolerate it better. However, do not be misled—it is still a long operation which can involve significant blood loss, morbidity and even mortality.

Preoperative

- Usual preoperative assessment of vascular patients (see ➲ p. 590). Try to obtain recent information about the cardiac function. An echocardiograph can easily be done at the bedside.
- Some of these patients will be very sick, either from pre-existing cardiorespiratory disease or from infected aortic grafts. Surgery may be their only hope of life, although it carries very high risk. Provided the patient understands this, the operation may be appropriate. These are not cases for inexperienced trainees to undertake alone.

Perioperative

- GA with ETT and IPPV is appropriate. An arterial line and large-gauge cannula are mandatory; CVP monitoring is optional.
- Heparin/protamine will be required at clamping/unclamping.

Postoperative

- Extubation at the end of surgery is usually possible, but a period of time on the HDU is recommended, if possible.
- PCA for postoperative analgesia.

ALGRAWANY

Amputations

(Below/through/above knee, Syme's, digits, etc.)

Procedure	Removal of necrotic or infected tissue due to vascular ischaemia
Time	30–120min
Pain	++++
Position	Supine
Blood loss	Usually 200–500mL, G&S
Practical techniques	Spinal or epidural with sedation. Sciatic/femoral blocks ± GA

Preoperative

- Commonly sick, bed-bound diabetic patients with significant CVS disease who have had repeated revascularisation attempts previously.
- Many will be in considerable discomfort preoperatively (less so the diabetics) and may be on large doses of enteral or parenteral opioids. Regional analgesia may give more predictable postoperative relief.

Perioperative

- Spinal anaesthesia ± sedation offers excellent anaesthesia which can be directed unilaterally. The duration of block (and postoperative pain relief) can be extended with intrathecal diamorphine (250–500 micrograms) or intrathecal clonidine (15–30 micrograms).[12]
- Diabetic patients presenting for amputations commonly have peripheral neuropathy. As a result, ankle blocks are very successful for minor amputations of the foot.
- Epidural analgesia offers better postoperative analgesia and can be sited preoperatively, if required (pre-emptive analgesia).
- GA is an option, but additional regional blockade is advisable (combined sciatic/femoral blocks will ensure analgesia for up to 24h). A wound catheter may be placed next to the sciatic nerve by the surgeon for postoperative infusion of LA (e.g. bupivacaine 0.25% at 10mL/h).
- Occasionally, these patients are septic due to the necrotic tissue. The only way they will improve is to have the affected part amputated, so cancellation may not be an option.

Postoperative

- Regional analgesia is the best option; otherwise PCA.
- Phantom limb pain is a problem for 60–70% of amputees at some time. It must be distinguished from surgical pain. If possible, get pain team input.
- Pre-emptive analgesia (preoperative siting of epidural) is believed by some to reduce the incidence and severity of chronic pain.
- Combined sciatic/femoral nerve blocks are an alternative to an epidural, particularly when the patient is receiving anticoagulation.
- Even with perfect regional analgesia, you may need to continue enteral opioids postoperatively.

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12 Ypsilantis E, Tang TY (2010). Pre-emptive analgesia for chronic limb pain after amputation for peripheral vascular disease: a systematic review. *Ann Vasc Surg*, **24**, 1139–46.

Thoracoscopic sympathectomy

Procedure	For patients with sweaty palms/axillae. The sympathetic trunk is divided via a thoracoscope inserted through a small axillary incision
Time	30–60min
Pain	++
Position	Supine, affected arm on arm board
Blood loss	Minimal
Practical techniques	IPPV via DLT, SV via LMA

- Patients are usually young and fit with hyperhidrosis (sweaty palms and axillae).
- Surgical technique involves cutting the thoracic sympathetic trunk at T2 or T3 thoracoscopically.
- Traditionally, this is done using one-lung anaesthesia (DLT), with the patient in the reverse Trendelenburg position.
- A simpler technique involves the patient breathing spontaneously through an LMA. When the surgeon insufflates CO_2 into the pleural cavity, the lung is pushed away passively, allowing surgery to take place. The degree of shunt produced is less dramatic than with OLV. Assisted ventilation must be avoided, except to reinflate the lung manually at the end. The CO_2 insufflator machine regulates intrapleural pressures.
- With either technique, at the conclusion of the procedure, the lung must be re-expanded (under the surgeon's direct vision) to prevent a pneumothorax.
- LA can be deposited by the surgeon directly onto the sympathetic trunk and into the pleural cavity.
- A postoperative chest radiograph is required to confirm lung reinflation.
- Synchronous bilateral sympathectomy is a much more challenging operation. This can lead to profound hypoxia when the 2nd lung is collapsed, due to persistent atelectasis in the 1st lung. It is certainly inappropriate for all but the very fittest patients.

ALGRAWANY

First rib resection

Procedure	Resection of the 1st/cervical rib in patients with thoracic outlet syndrome
Time	1–2h
Pain	++
Position	Supine, affected arm on arm board
Blood loss	Minimal
Practical techniques	IPPV via ETT, avoid muscle relaxants

- Patients are usually young and fit.
- The position is similar to that for thoracoscopic sympathectomy.
- Muscle relaxants should be avoided, as the surgeon needs to be able to identify the brachial plexus perioperatively. Intubate under opioid/induction agent alone, or use mivacurium/opioid, and then hyperventilate with isoflurane/opioid or similar.
- At the conclusion of surgery, the wound is filled with 0.9% sodium chloride, and manual ventilation performed with sustained inflation pressures >40cmH_2O. This is to check for a lung leak and exclude a pleural injury.
- A superficial cervical plexus block provides good postoperative analgesia (see ➔ p. 1118; ➔ pp. 602–3).
- A postoperative CXR is required in recovery.

Arteriovenous fistula formation

Procedure	Creation of an A–V fistula usually in the upper limb to facilitate haemodialysis
Time	1–2h
Pain	+
Position	Supine, operating arm out on side table
Blood loss	Minimal
Practical techniques	Brachial plexus block, IPPV via ETT

- These patients, by definition, have renal failure, so they may have hypertension, DM, fluid imbalance, previous transplants, failed fistulae, uraemia, coagulopathy, hyper-/hypokalaemia, anaemia or chronic acidosis.
- The patient may have been, or may still be, receiving a heparin infusion to try to keep the old fistula patent.
- There may be a venovenous dialysis line *in situ*. Avoid using this for IV access unless there are no alternatives.
- Know when the patient was last dialysed. Check K^+ and Hb. Think about volume overload or hypovolaemia. Be cautious giving IV fluids, which should not contain K^+.
- Discuss carefully with the surgeon exactly where the fistula will be formed and avoid cannulae/arterial line/BP cuff on this side.
- Avoid cannulating large veins in the forearm or antecubital fossa as these may be required for A–V fistulae in the future. Use veins on the dorsum of the hand, if possible. Only site an arterial line if vital.
- Local infiltration is often possible for 1° AV fistula creation. Otherwise, regional anaesthesia is the best option unless the patient is anticoagulated.[13] Ultrasound-guided brachial plexus block is appropriate. For proximal surgery, use the supraclavicular or interscalene approach, whereas the axillary approach may be better for more distal surgery.
- GA probably requires airway protection due to autonomic neuropathy. Difficult intubation is possible (prayer sign). Use appropriate muscle relaxants, e.g. cisatracurium, atracurium.
- Consider the effects of renal impairment on drug metabolism when prescribing postoperative analgesia.

References

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ALGRAWANY

Varicose vein surgery

Procedure	Removal of tortuous veins of the lower extremities: High tie and strip—long saphenous vein removal (sometimes bilateral) Short saphenous vein surgery—tied off in popliteal fossa
Time	30min to 3h
Pain	++
Position	Supine or prone for short saphenous surgery
Blood loss	Up to 1000mL
Practical techniques	LMA/SV for most; ETT/IPPV for prone

- The number of patients undergoing varicose vein surgery has dramatically fallen due to the introduction and popularity of techniques such as endovenous laser ablation or sclerotherapy. Despite this, there is still a core of patients who require surgery.
- Patients are usually young and fit.
- The main operation is usually combined with multiple avulsions to remove varicosities. These are minute scars, which can, however, bleed profusely.
- Blood loss can be minimised by elevating the legs intraoperatively.
- Patients may need combined long and short saphenous surgery (i.e. two operative incisions on the same leg) and may require turning during the operation. In selected slim patients without aspiration risk, this can be done with the patient breathing spontaneously through an LMA.
- A combination of NSAIDs and LA into the groin wound gives good postoperative analgesia. Caudal anaesthesia is possible for prolonged re-explorations.
- Bilateral surgery is common and takes 30–60min per incision.
- Redo surgery is also common and can be more prolonged.

Further reading

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Chapter 23

Orthopaedic surgery

Richard Griffiths and **David Brooks**

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See also

- Regional anaesthesia pp. *1099–1146*
- The major trauma patient pp. *967–1032*
- Pelvic injuries p. *1001*
- Spinal trauma pp. *1002–3*
- Anaesthesia in spinal cord lesions pp. *303–8*
- Amputations p. *606*
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- Femoral osteotomy p. *946*

ALGRAWANY

General principles

- About 220 000 major joint replacements are performed annually in England and Wales.[1]
- The population is ageing and these patients are now likely to be obese with multiple comorbidities.
- Many operations are amenable to regional anaesthesia and ultrasound-guided LA blocks (see ➲ p. 613).
- Patient education before surgery, using joint schools, has promoted patient choice and highlighted areas, such as the anaesthetic technique, well before the day of surgery.

Preoperative

- Liaison with the surgeon is essential, particularly if undertaking regional techniques.
- Arthritis often makes assessment of cardiorespiratory fitness difficult.
- If planning a regional technique (particularly CNB), it is important to consider factors affecting clotting (timing of the last dose of anticoagulant) and discuss specific risks and benefits with the patient[2,3] (see ➲ pp. 1100–1101).
- A high risk of VTE occurs with certain operations requiring antithromboembolic measures, e.g. LMWH, stockings, foot pumps (see ➲ pp. 59–61).

Perioperative

- Give IV antibiotic prophylaxis as per local protocols, but be aware of the potential for anaphylaxis[4] (see ➲ pp. 62–3).
- Utmost care with positioning is essential to avoid soft tissue or nerve injuries. This is a shared responsibility between the anaesthetist and the surgeon.
- Maintenance of normothermia with blood warmers and forced air warming blankets can reduce both morbidity and mortality.
- Hypotension should be avoided.[5]
- Consider invasive monitoring for those patients with CVS disease and those having revision lower limb surgery, especially if from a periprosthetic fracture.
- Blood loss may be significant.
- Monitor blood loss accurately. Consider cell salvage, including drain salvage.
- A urinary catheter should be inserted for long procedures or when epidurals/spinal opioids are used.
- Local infiltration analgesia (LIA), using low-dose, high-volume LA, is an effective technique for pain reduction in elective hip and knee arthroplasty surgery.[6]

Postoperative

- Good analgesia will have a positive effect on recovery, mobility and discharge.
- Liaise with the surgeon if prescribing NSAIDs, but use with care in those over 75 (see ➲ p. 91; ➲ pp. 1155–6).

Regional anaesthesia

- Regional anaesthesia may be used for most joint replacements (alone or with carefully administered sedation). Benzodiazepines should be avoided in elderly patients. CNB and major nerve blocks are commonly performed (see pp. 1099–146).
- In major orthopaedic surgery, blocks may provide postoperative pain relief and may reduce PONV.
- There is some evidence that regional anaesthesia, either alone or in combination with GA, may improve outcome in hip and knee arthroplasty, although these data are based on large observational studies.[6,7,8]
- Good fixation of cement and joint prosthesis requires a dry, bloodless surgical field. Regional anaesthesia (particularly spinal/epidural) reduces bleeding at the surgical site, without the need for other pharmacological hypotensive anaesthetic techniques.
- Surgeons often prefer the operating conditions produced by regional techniques.[8]

ALGRAWANY

Fat embolism syndrome

- Fat embolism syndrome (FES)[9] is associated with trauma or surgery and has an extremely variable presentation; diagnosis is often made by exclusion.
- Although embolisation of fat occurs frequently, the syndrome is comparatively rare (1%).
- Early surgery and avoidance of intramedullary fixation have both reduced the incidence.
- Current treatment is supportive (early mortality 1–20%), but serious long-term complications are uncommon.
- FES is classically seen in patients with long bone fractures who develop sudden tachypnoea and hypoxia.
- Although sometimes a petechial rash is seen (check conjunctivae), a firm diagnosis is frequently difficult.[9]

Features (as defined by Gurd)[10]

Major

- Respiratory symptoms: tachypnoea, dyspnoea, bilateral crepitations, haemoptysis, diffuse shadowing on CXR
- Neurological signs: confusion, drowsiness
- Petechial rash.

Minor

- Tachycardia
- Retinal change: fat or petechiae
- Jaundice
- Renal: oliguria or anuria.

Laboratory

- Thrombocytopenia
- Sudden decrease in Hb by 20%
- Raised ESR
- Fat macroglobulinaemia.

Treatment

- Early resuscitation and stabilisation are vital.
- Early O_2 therapy may prevent onset of syndrome.
- May require mechanical ventilation (10–40% of patients).
- Steroid use is controversial.[9]
- FES usually resolves within 7d.

Bone cement implantation syndrome

- Methyl methacrylate bone cement is an acrylic polymer that has been used extensively in orthopaedic surgery for 40y.
- Its use is associated with the potential for hypoxia, hypotension and CVS collapse.
- Fatal cardiac arrest is a reported complication.
- There are many suggested aetiologies, of which fat embolisation appears to be the most likely.
- Air embolisation (Doppler evidence in 30% of patients) and direct effects of the cement are also possible.
- There is now a proposed classification of bone cement implantation syndrome (BCIS), ranging from grade 1, with mild hypotension and hypoxia, to grade 3, with CVS collapse.[11]
- The incidence of grade 3 BCIS was 0.5% in the Anaesthesia Sprint Audit of Practice, conducted by the National Hip Fracture Database in 2014.[12]
- Following this report, a patient safety consensus document was produced which was a collaboration between the AoA, the British Orthopaedic Association and the British Geriatric Society.[13]

Prevention and treatment

- Certain patient groups, such as those with significant cardiorespiratory disease and those on diuretics, are at ↑ risk.[13]
- A 'cement curfew' should be part of the WHO checklist and each member of the theatre team should have specific roles and should not leave theatre around the time of prosthesis insertion.
- The conduct of anaesthesia and surgery are summarised in Table 23.1 from the guideline *Reducing the risk from cemented hemiarthroplasty for hip fracture*.[13]

Table 23.1 Summary of recommendations for the conduct of surgery and anaesthesia during cemented hemiarthroplasty

Conduct of surgery	Anaesthetist to confirm communication that preparation of femoral canal for cement is commencing Femoral canal prepared Pressurised lavage system recommended to clean endosteal bone of fat and marrow Distal suction catheter on top of intramedullary plug. Cement inserted in retrograde fashion No excessive pressurisation in high-risk patients
Conduct of anaesthesia	Ensure adequate hydration pre- and intraoperatively Maintain vigilance after femoral head removed Closed loop communication when surgeon communicates preparation of the femoral canal Maintain the systolic BP within 20% of preinduction values throughout. Use vasopressors and/or fluids. Invasive BP monitoring in high-risk patients Be ready for CVS collapse (metaraminol/adrenaline)

Source: data from Griffiths R *et al*. (2015). Safety guideline: Reducing the risk from cemented hemiarthroplasty for hip fracture 2015. *Anaesthesia*, **70**, 623–6.

ALGRAWANY

Tourniquets

Tourniquets are commonly used to produce a bloodless field.[14]

- Only pneumatic tourniquets should be used. Mechanical tourniquets can cause areas of unpredictably ↑ pressure in underlying tissues.
- Small tourniquets on fingers and toes are dangerous, because they are easily forgotten. Use a rubber strip with artery forceps.
- Expressive exsanguination using an Esmarch bandage is contraindicated in cases of tumour or severe infection because of the risks of dissemination. It is also contraindicated if DVT is suspected; fatal PE has been reported.[15] It also represents a potential risk of LV failure from fluid overload if compression of both legs is carried out simultaneously (adds 15% to the circulating volume); therefore, limit to one leg only in patients at risk. Effective exsanguination can be achieved by arm or leg elevation for 5min at 90°, without mechanical compression.
- Peripheral arterial disease is a relative contraindication to use.
- Avoid in severe crush injuries.
- Sickle-cell disease: use of tourniquets is controversial. Sickling of RBCs under anoxic conditions causes thrombosis, but some surgeons use limb tourniquets after full exsanguination. If employed, use for as short a time as possible (see also ➲ pp. 257–9).

Site of application

- The upper arm and thigh have sufficient muscle bulk to distribute the cuff pressure evenly and are the recommended sites.
- For short operations (<1h) in fit patients, a calf tourniquet is preferred by some surgeons.

Cuff width

- The American Heart Association concluded that if a sphygmomanometer cuff has a width of 20% greater than the diameter of the upper arm or 40% of the circumference of the thigh (to a maximum of 20cm), then the pressure in the underlying central artery will be equal to that in the cuff. This avoids the need for excessively high cuff pressures.
- Modern silicone cuffs tend to be smaller than this, measuring 90mm in width (bladder 70mm) for the arm and 105mm (bladder 75mm) for the leg.
- Cuff length should exceed the circumference of the extremity by 7–15cm. The cuff should be positioned at the point of maximum circumference of the limb.
- The tissues immediately underlying the cuff should be protected with cotton wool. This is not necessary with a correctly applied modern silicone cuff.

Pressure

- Base on patient's BP measured on the ward preoperatively.
- Upper limb: systolic BP + 50mmHg. Lower limb: twice systolic BP. This higher pressure is needed because there is often not enough room above the operating site for a full-sized cuff.
- The use of lower inflation pressures may minimise complications following the use of tourniquets and speed up postoperative recovery. In a normotensive patient, a pressure of 200mmHg should be ideal for the upper limb and 250mmHg for the lower limb.

Tourniquet time

- The minimum time possible should be the aim.
- Notify the surgeon at 1h, and remove as soon as possible after that.
- If the operation is difficult, the time can be extended to 1.5h. Two hours should be regarded as a maximum, but this will not be safe for all patients.
- PEs can occur following tourniquet release. When monitored using TOE, the rate was higher with ↑ tourniquet time.[16]

Tourniquet pain

- After 30–60min of cuff inflation, a patient may develop an increase in HR and diastolic BP. This response results from 'tourniquet pain'. This also occurs under anaesthesia, although the response is usually abolished by spinal or epidural techniques.
- In volunteers, when a tourniquet is inflated, a dull pain, associated with an increase in BP, occurs after 30min.
- Often the physiological changes are resistant to analgesic drugs and ↑ depth of anaesthesia. β-blockers, in particular labetalol, may be useful.
- Small doses of ketamine given IV (0.25mg/kg) before tourniquet inflation has been reported to attenuate these BP rises.[17,18]

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Total hip replacement

Procedure	Prosthetic replacement of femoral head and acetabulum
Time	60–90min
Pain	+++
Position	Lateral or supine
Blood loss	300–500mL
Practical techniques	Spinal with/without sedation or GA ± LIA or nerve block

Total hip replacement is one of the most frequently performed orthopaedic operations. The 17th annual report from the National Joint Registry (NJR)[1] shows that there were around 100 000 1° hip operations each year between 2016 and 2019. The average age of patients for 1° hip arthroplasty was almost 69y. Cemented arthroplasties account for 26% of all operations, with uncemented procedures totalling another 35%. Between 2006 and 2019, the number of cemented prostheses has stayed the same, but as a proportion of the total, it has halved.[1] Regional anaesthesia offers several advantages[7] and can be supplemented with sedation or GA. Prevention of thromboembolic complications is of the utmost importance.

NICE have produced a guideline on the perioperative management of hip replacements.[6]

Preoperative

- Careful preoperative evaluation of the patient is essential.
- Patients contemplating hip replacement should receive advice on preoperative rehabilitation.[6]
- No patient should be operated on electively who is anaemic (aim for Hb >130g/L in men and women) and blood transfusion is rare in 1° hip arthroplasty[19] (see pp. 254–6).
- Antithrombotic measures should commence on admission (see pp. 59–61).

Perioperative

- Place an 18G or larger cannula in the upper arm (if a lateral position is anticipated).
- Ensure adequate hydration prior to performing spinal anaesthesia and during cement insertion.
- For single-shot spinal anaesthesia: 2–3mL of hyperbaric bupivacaine 0.5%, depending on patient size. Diamorphine (0.25–0.5mg) may be added for more prolonged analgesia but can cause unpleasant PONV.
- When using spinal anaesthesia in the lateral position, TCI propofol is a useful sedation technique, with face mask supplemental O_2.
- Avoid benzodiazepines in the elderly, as their use may lead to acute delirium.
- Epidural analgesia is rarely indicated for 1° hip arthroplasty.
- Peripheral nerve blocks offer some advantages over systemic analgesia.[20]
- Aim to maintain BP at an adequate level, based on preoperative readings; hypotension is not indicated.[5]

- Intraoperative antibiotic prophylaxis as per local protocol will be required (see ➔ pp. 62–3).
- Actively warming the patient reduces intraoperative blood loss significantly and reduces morbidity and mortality.
- Consider IV tranexamic acid.[6]

Postoperative

- Surgeons usually prefer the patient to be placed on their bed in the supine position, with the legs abducted using a pillow, to prevent dislocation of the prosthesis.
- Antithromboembolic prophylaxis is important—at least 1% of patients develop DVT, even with measures in place (see ➔ pp. 59–61).
- O_2 therapy for up to 24h is advisable in most patients.
- Hb should be checked 24h postoperatively.
- Patients are mobilised as soon as possible after surgery; ideally, rehabilitation should be offered on the day of surgery, if possible.[6] Simple oral opioids with regular paracetamol or NSAIDs are usually sufficient for postoperative analgesia. Caution with NSAIDs in the elderly (see ➔ p. 91; ➔ pp. 1155–6).

Bilateral total hip replacement

This procedure is now rarely performed. Around 5000 have been recorded by the NJR since 2006. In the latest NJR report,[1] there were 386 bilateral procedures performed out of around 101 000 cases..

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Revision of total hip replacement

Procedure	Revision of previous total hip replacement Revision may include one or both components
Time	1–4h, depending on complexity
Pain	++++
Position	Lateral or supine
Blood loss	1L, occasionally considerably more, X-match 2 units
Practical techniques	Regional or GA ± nerve block

Aseptic loosening was the commonest indication in 2019 NJR data, and over 16% of the procedures were for periprosthetic fractures.[1]

Preoperative

General principles as for total hip replacement, except:

- The operation may take longer, but timings are variable. Discuss with your surgeon beforehand.
- Blood loss can be significant, with 1L or more commonly lost perioperatively.
- Postoperative pain can be a significant problem.

Perioperative

- As for 1° hip replacement, including a urinary catheter.
- If significant blood loss is anticipated or the patient's CVS status indicates it, insert an arterial line and make use of near-patient monitoring to assess fluid status and Hb concentration.
- Technique should be planned, according to the length of surgery, the operative position and patient factors.
  - If CNB is contraindicated, consider supplementing GA with nerve blocks (femoral, 3-in-1 or psoas compartment lumbar plexus).
- Use blood recovery and autologous transfusion wherever possible.
- Perioperative blood transfusion is frequently required, and blood loss may be substantial. Two units of X-matched blood should be available in theatre, with the ability to obtain more within 30min.
- Consider IV tranexamic acid.[6]

Postoperative

- Mobilisation varies with the complexity of the revision and the strength of reconstruction.
- PCA is a suitable alternative.
- Supplemental O_2 is required for 24h or longer, particularly if significant blood loss or an underlying cardiorespiratory disease.
- Remember thromboembolic prophylaxis (see pp. 59–61).

Total knee replacement

Procedure	Prosthetic replacement of the knee joint
Time	45–90min
Pain	++++/+++++
Position	Supine
Blood loss	Minimal **with** tourniquet; 250–500mL **without**. G&S
Practical techniques	Spinal analgesia plus LIA GA plus LIA Ultrasound-guided adductor canal block[17]

Similar patient population to hip surgery. Often a shorter operation with less blood loss and less chance of cement hypotension. A tourniquet is commonly used, so be aware of tourniquet pain. Postoperatively, pain can be extreme and must be anticipated.

There has been a move towards nerve blocks that spare the motor component, so that mobilisation is not impaired. An example of this is the adductor canal block.[21]

NICE have produced a guideline on the perioperative management of knee replacements.[6]

Preoperative

As for hip surgery.

Perioperative

- The patient is always supine, and therefore, airway control under sedation can be a problem.
- Spinal anaesthesia, with or without intrathecal opioids, is the preferred technique.[22] GA is often used.
- A tourniquet is commonly used; therefore, perioperative blood loss is not problematic, although expect to lose up to 500mL (and frequently more) from the drains in the 1st hour postoperatively. There is a trend to reduce the use of the tourniquet.
- Consider IV tranexamic acid.[6]
- If a tourniquet is used, one may see 'breakthrough' of tourniquet pain after about 1h, causing CVS stimulation and hypertension. This is more common with leg blocks and is treated by deepening anaesthesia or adding IV opioid. Ketamine (0.25mg/kg) is effective at preventing the associated rise in BP.[17] Ensure the patient is well preloaded before the tourniquet is released. A short-lived reperfusion event is common (fall in BP and SaO_2, rise in $ETCO_2$) and is usually best prevented by fluid loading before and during tourniquet release.

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Postoperative

- Postoperative pain is usually the most significant problem, and this is the main determinant of the anaesthetic technique. Many patients are now already on opioids, and this makes the management of postoperative pain more difficult.
- When blood loss into the drains continues to be brisk after the first 500mL, the surgeon will often clamp the drains for a period of time.
- Ideally, rehabilitation should be offered on the day of surgery if possible.[6]

Bilateral total knee replacement

- Bilateral knee replacements should only be considered in young, fit, motivated patients. Elderly patients and those with significant CVS disease are high risk.
- The advantage is that two admissions/operations are avoided.
- The disadvantage is that bilateral total knee replacement is a major CVS stress and is associated with unpredictable blood loss and fluid requirements.
- This can be achieved under regional anaesthesia and bilateral adductor canal blocks, but frequently GA may be required for over 2h of surgery.

Revision of total knee replacement

- Same as 1° knee replacement, except it takes longer (≥2h).
- The technique is as for 1° knee replacement.
- If done without a tourniquet, then 2 units of blood should be X-matched.

Arthroscopic lower limb procedures

Procedure	Arthroscopy, examination under anaesthesia (EUA) and washout ± excision of torn cartilage, meniscal surgery/loose body removal, synovectomy, ligament reconstruction
Time	20–60min
Pain	++
Position	Supine, with leg over side of table
Blood loss	Nil
Practical techniques	GA/LMA or ambulatory spinal anaesthesia

General principles

- The patient population is generally younger than those having joint replacements.
- Smaller procedures are done as day cases and therefore require a technique that allows early ambulation and discharge home.
- Virtually all are done on the knee, though arthroscopy is also performed on the ankle.
- Arthroscopy for knees with osteoarthritis is not supported by evidence of effectiveness.[23]

Technique

- Premedication with paracetamol and NSAID.
- GA/LMA is a 'standard' day case anaesthetic with IV opioids such as fentanyl 1 microgram/kg.
- Ambulatory spinal anaesthesia can be achieved with prilocaine due to its rapid onset, predictable regression and low incidence of adverse effects (see p. 490).[24]
- A tourniquet is often used.
- Prescribe NSAIDs and strong oral analgesics to take home.
- Many surgeons instil 10–20mL of 0.5% bupivacaine ± morphine (10mg) into the joint cavity for postoperative pain relief.
- Ketamine in low dosage (IV) has been suggested to enhance analgesia (0.15mg/kg).[25]
- Ideally, IV morphine should be avoided in day case arthroscopic procedures due to the high incidence of PONV.
- EUA ± washout can be performed under intra-articular and infiltration LA alone. Nerve blocks have been used but are limited by the long duration of action of anaesthesia and the failure to block the site of the arterial tourniquet.

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Cruciate ligament repair

Procedure	Arthroscopic reconstruction of anterior cruciate ligament using patellar tendon ± hamstrings
Time	1.5–2h
Pain	+++/++++
Position	Supine
Blood loss	Nil
Practical techniques	LMA + GA ± adductor canal block Spinal is an alternative

Technique

- These operations are of two main types: using the patellar tendon only for the repair, and using both the patellar tendon and hamstring ligaments.
- Usually 12h of analgesia are required prior to mobilisation.
- Nerve blocks have been used but hinder postoperative mobilisation.
- Oral opioids, combined with paracetamol and NSAIDs, are the mainstay of analgesia.
- If the hamstrings are used, the operation takes longer and there is more postoperative pain.

Ankle surgery

General principles

- Four main types of procedure: tendon transfers, open reduction and internal fixation (ORIF) of fractures, joint arthrodesis and prosthetic joint replacement (Table 23.2).
- Ankle arthrodesis takes 1–2h. Tendon transfer is generally quicker than this, and joint replacement may take longer.
- These operations are amenable to regional anaesthetic techniques, either alone or combined with GA.
- Tourniquets are often used, and tourniquet pain has to be considered (see ➔ pp. 616–17).
- Patients may be supine, prone or occasionally on their side.
- In the case of ORIF following trauma, surgery may need to be undertaken urgently if distal circulation is compromised. Beware of the risk of aspiration from a full stomach, and also take time to ensure that any other significant injury has been properly managed.
- If regional block is considered for ORIF, check that there is no concern about the development of compartment syndrome postoperatively, as the symptoms may be masked by the block (see ➔ p. 1005).

Technique

- Local, regional, general or a combination of techniques can be used for all procedures on the ankle.
- Nerve blocks under ultrasound guidance are popular and, for ankle surgery, require sciatic (or popliteal) and femoral (or saphenous) nerve blockade. The saphenous nerve (terminal branch of the femoral nerve) supplies the skin down to the medial malleolus of the ankle (see ➔ p. 1140).
- Nerve blocks following a spinal anaesthetic improve analgesia well into the 1st postoperative day. GA can also be combined with nerve blocks.
- Care must be taken in trauma cases with fractured ankles, as nerve blocks may mask compartment syndrome. Always discuss your proposed technique with the surgeon. The general rule is that nerve blocks are best avoided in ankle trauma cases. Local infiltration is useful.
- Tendon transfer surgery takes up to 1h and is not particularly painful postoperatively.
- ORIF may be an emergency if the vascular supply is compromised, and an RSI is the best anaesthetic option in this situation. A good alternative for ORIF is spinal anaesthesia. The addition of intrathecal opioid (e.g. diamorphine 0.25–0.5mg) prolongs the period of analgesia.
- Ankle joint replacement is a procedure that is increasing in popularity. Usually the procedure is accomplished within 2h.

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Table 23.2 Summary of ankle procedures

Procedure	Time (h)	Pain (+ to +++++)	Position	Blood loss	Practical technique
Tendon transfer/repair	~1	++	Supine (ruptured tendo-Achilles—prone)	Nil with tourniquet	GA + LMA with infiltration of LA by surgeon Spinal if supine. IPPV if prone
ORIF of ankle fracture	Variable 1.5–2	++/+++	Supine, occasionally on side or prone	Nil with tourniquet	GA (if in doubt, RSI) or spinal Generally avoid nerve blocks
Arthrodesis of ankle joint	1.5–2	+++	Supine	Nil with tourniquet	GA or spinal with nerve blocks PCA Spinal + nerve blocks
Prosthetic replacement of ankle joint	2+	++/+++	Supine	Nil with tourniquet	GA + nerve blocks Spinal + nerve blocks

Foot surgery

General principles

- Most operations are on the forefoot and toes, e.g. 1st metatarsal osteotomy, Keller's, excision of ingrowing toenails and terminalisation of toes. Other operations in the midfoot include tendon transfers and some osteotomies (Table 23.3).
- The patient population varies, and many are elderly. Those for terminalisation of toes may well have concomitant problems such as diabetes and/or CVS disease.
- Osteotomies tend to be painful postoperatively.
- Surgical time is 30min to 1h.
- Many are done as day cases and require early ambulation and discharge with adequate pain relief.
- Nerve blocks make a valuable contribution to postoperative analgesia, particularly in osteotomies and nail bed excision, and promote early ambulation. However, onset time is relatively long and they need to be performed a full 40min prior to surgery, if planned without GA. With experience, this can work well, but for the less experienced, it is best to undertake them primarily for postoperative pain relief in combination with LMA and GA.
- Patients may find an ankle block painful, so sedation may be required.
- Adrenaline must not be used for 'ring' or 'web-space' blocks and is best avoided in ankle blocks if the peripheral circulation is poor.
- Breakthrough pain from the tourniquet can be a problem, especially if surgery is longer than 45min. Place the tourniquet as distally as possible to reduce this effect.

Technique

- Regional blocks useful for foot surgery include ring/web-space or ankle blocks for toe surgery, ankle block for forefoot surgery and sciatic (or popliteal) nerve block for operations on the midfoot. Most commonly, these blocks are performed for postoperative pain relief and are combined with GA (see ➔ pp. 1142–4).
- An alternative in all cases is spinal anaesthesia.

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Table 23.3 Summary of forefoot procedures

Site	Procedure	Time (min)	Pain (+ to +++++)	Position	Blood loss/ X-match	Technique
Toes	Excision of nail bed, terminalisation	30	+++	Supine	Nil	Ring or toe web block with sedation or GA/LMA + ankle block
Forefoot	Tendon transfers	30–60	+/++	Supine	Nil	GA/LMA + local infiltration Ankle block with sedation or GA/LMA
Forefoot	1st metatarsal osteotomy, Keller's	30–60	+++	Supine	Nil	GA/LMA with ankle block or infiltration
Midfoot	Tendon transfers	30–60	+/++	Supine	Nil	GA/LMA + local infiltration
Midfoot	Osteotomy	30–60	+++	Supine	Nil	GA/LMA ± sciatic nerve block at knee

Spinal surgery

Definition

Surgery on the spinal column between the atlanto-occipital junction and the coccyx.[26,27]

Can be loosely divided into four categories (Table 23.4):[26,27,28]

- Decompression of the spinal cord and nerves
- Stabilisation and correction of spinal deformity
- Excision of spinal tumours
- Trauma.

General principles

Children present for scoliosis surgery,[28] young and middle-aged adults for decompressive surgery and older patients for stabilisation.

- Most procedures are in the prone position,[29] although anterior and lateral approaches are used. Some procedures will involve turning the patient during the operation (see pp. 435–41).
- Airway access will be limited during surgery and must be secure.
- Prevent excessive abdominal or thoracic pressure due to incorrect patient positioning, which may compromise ventilation and circulation.
- Surgical blood loss can be considerable. Ensure good vascular access and accurate measurement of blood loss. Consider tranexamic acid and cell salvage.
- Long procedures necessitate active prevention of heat loss.
- Assessment of spinal function may be required during the procedure and monitoring can be affected by the choice of anaesthetic technique.[30]
- Fastidious attention to detail is necessary to evade the many inherent complications in spinal surgery.

Anaesthesia

- Thorough preoperative assessment is essential. Any existing neurological deficit must be documented prior to the induction of anaesthesia.[26,27]
- Plans for the recovery period should be made in advance and will be dictated by local experience. Long cases, those involving excessive blood loss and major paediatric cases will need postoperative care in the HDU. Few patients require postoperative ventilatory support.
- Secure venous access is critical. It may be difficult to access the cannula, so an extension with a three-way tap is recommended. If multiple infusions are planned, additional cannulae may be prudent.
- Not all cases require invasive arterial pressure monitoring. Indications include planned postoperative critical care admission, predicted intraoperative instability, high predicted blood loss and a requirement for intraoperative blood investigations.
- Central venous access is not required for most cases.
- Choice of anaesthetic will be dictated by personal experience, but most will choose an IV induction with muscle relaxation and opioid supplementation. Both volatile anaesthesia and TIVA are frequently used. Remifentanil is useful perioperatively. The need for intraoperative neuromonitoring may preclude the use of volatile anaesthetic gases.

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Table 23.4 Summary of spinal surgery procedures

Operation	Description	Time (h)	Position	Blood loss/ X-match	Pain (+ to +++++)	Notes
Discectomy or microdiscectomy	Excision of herniated intervertebral disc	1–2	Prone	Not significant	+/++	Microdiscectomy can be done as day case
Cervical discectomy	Excision of herniated cervical intervertebral disc	2	Prone/head on horseshoe or halo traction pins	Not significant	++/ +++	May be an emergency with neurological deficit
Spinal fusion ± decompression	Correction of spondylolisthesis or spinal stenosis for pain or instability—often several levels	1–2 (then 1 per level)	Prone	500–2000mL, X-match 4 units	+++/ ++++	May take bone graft from pelvis Metal implantation
Cervical fusion ± decompression	Fusion of unstable neck (e.g. arthritis, trauma)	2–3	Supine or prone. Cervical traction in place or applied at start	300–1000mL, G&S	++/ +++	Neck can be very unstable and need AFOI. Application of traction pins very stimulating Retraction of the carotid sheath in anterior cervical discectomy and fusion may cause haemodynamic instability Postoperative airway compromise is a possibility

Operation	Description	Time (h)	Position	Blood loss/ X-match	Pain (+ to +++++)	Notes
Excision of spinal tumour (e.g. vertebrectomy)	Tumours may be 1° or 2° from any part of the spine	2–6+	Supine, prone or lateral tilt	Potentially massive, X-match 6 units + clotting factors available	+++/ ++++	Often difficult surgery with potential for major blood loss and neurological damage
Kyphoscoliosis surgery	Correction of major spinal deformities in patients who may have severe physical disability	3–6+	Supine and/or prone	Potentially massive, X-match 6 units + clotting factors available	+++/ +++++	Often in children with severe restrictive respiratory disease and coexisting abnormalities. May involve surgery in abdominal and thoracic cavities. Spinal nerve monitoring used in some centres. May need postop ICU for IPPV
Repair of vertebral fracture	Repair for neurological deficit or instability	2–6	Supine and/or prone	500–2000mL, X-match 4 units	++/ ++++	Often associated with other major injury (especially rib fracture). May be in ICU/IPPV. Neurological deficit often not reversible. Note: suxamethonium may be contraindicated

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- If spinal cord integrity is at risk during surgery, it may be necessary to use spinal cord monitoring.[30] This is a specialist service provided by a neurophysiologist but may require that muscle relaxation is allowed to wear off. It may be necessary to deepen the anaesthesia during this phase, but in reality, this is rarely a problem.
- In patients with paraplegia or other large areas of muscle denervation (2d to 8mo), suxamethonium should be avoided (see ➲ p. 306; ➲ p. 421).
- Prophylactic antibiotics should be given as per local protocol (see ➲ pp. 62–3).
- Airway access is likely to be limited once the procedure has started, so securing oral endotracheal intubation with a non-kinking tube is usual. It is critical to have a plan for how to manage inadvertent tube displacement. Patients with unstable necks due to trauma or RA can be intubated using an awake tracheal intubation technique (see ➲ pp. 246–8; ➲ pp. 393–6). Some patients will require MILS, depending on the degree of instability and the anticipated difficulty of intubation (see ➲ p. 973). The tube should be moulded around the face, with no bulky joints adjacent to the skin. A throat pack may be used to decrease the flow of secretions onto the pillow, and the tube then secured with adhesive tape or film. If a throat pack is used, this must be recorded and part of the pack should be left outside of the mouth so that it is visible.
- Attention to detail and the use of padding are vital to protect pressure areas.
- Most patients will be paralysed and ventilated for these procedures, with positional considerations noted above.
- Check the position of the ETT when the patient has been turned. Check that ventilation is adequate, without excessive inflation pressures, before surgery starts, as the only recourse may be to return the patient to the supine position if problems develop.
- Blood loss may be significant, with venous oozing proving hard to control. Tranexamic acid is commonly used. The use of cell salvage techniques (see ➲ pp. 458–9) is advisable for long procedures involving instrumentation of multiple levels. All patients should have samples grouped and saved, and more major procedures should have blood X-matched, even if cell salvage is employed (see below).
- Near-patient testing techniques such as TEG® or ROTEM® can help to guide transfusion requirements (see ➲ pp. 284–6).
- Hypotensive anaesthesia may reduce blood loss during major spinal surgery. The MAP should be maintained at a safe level—for normotensive patients, >60mmHg. Invasive blood pressure monitoring is mandatory when the BP is being manipulated.
- The type of analgesia required will vary, depending on the magnitude of surgery. Minor procedures (e.g. microdiscectomy) may manage with NSAIDs alone, in association with infiltration of the operative site with LA. Most procedures will necessitate opioids. PCA morphine is effective after adequate IV loading. The use of regional analgesia is encouraged where there is no need to assess neurological function, and the use of epidural and paravertebral analgesia is growing in popularity for major procedures such as correction of scoliosis. The catheter is usually placed by the surgeon at the end of the procedure, and infusions of LA or opioids continued for several days postoperatively.

- Effective analgesia is particularly crucial for surgery to the thoracic spine where postoperative respiratory function will be compromised if analgesia is inadequate. Consider also using incentive spirometry and chest physiotherapy.

The prone position

(See ➲ pp. 439–40 for full discussion of prone positioning.)

- Turning the patient from supine to prone requires log rolling by a trained team, compromising at least six members of staff, to avoid applying twisting forces in the axial plane.[27,29] This is especially important for the poorly supported cervical spine, which may be unstable due to fractures or degenerative disease.
- During long cases, it may be necessary to move the patient's limbs and head every hour to avoid stagnation of peripheral blood and the development of pressure necrosis. Pay particular attention to the nose, eyes, chin, elbows, knees and ankles.
- Ophthalmic complications are well documented and can include corneal abrasions through to visual loss.
- Pressure on the abdomen can be transmitted to the valveless epidural veins and result in ↑ bleeding.

Intraoperative neuromonitoring

- Neurological damage can result during operations on the spine. It is possible to use neuromonitoring techniques to assess neural pathways during surgery and thus increase safety.[27,30]
- Intraoperative monitoring has superseded the 'wake-up test' when patients were woken in the middle of surgery and asked to perform simple motor functions before being reanaesthetised.
- Modalities include: somatosensory evoked potentials, motor evoked potentials, EMG and EEG.
- Volatile anaesthetic agents decrease the amplitude of motor evoked potentials in a dose-related manner.
- NMBAs abolish motor evoked potential signals.
- It is essential for the neurophysiologist to know what drugs have been given, as this will affect the interpretation of signals.
- TIVA with propofol and opioids is a popular technique.

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Shoulder surgery

General considerations

- Shoulder surgery is often excruciatingly painful, more so than many other day case procedures.[31,32]
- Pain is not predictable and may last for several days, although it is undoubtedly worst within the first 48h.
- A multimodal approach to analgesia is crucial.
- Heterogeneous patient population: from young athletic patients with trauma through to elderly patients with numerous comorbidities such as RA.[31,32]
- Arthroscopic procedures are generally less painful than open procedures. Patients get effective postoperative analgesia if the surgeon injects LA within the joint space at the end of surgery.
- Bankart's and capsular shift operations for recurrent dislocations are more painful for larger, muscular patients, but not generally as painful as cuff repairs and open acromioplasties.
- Massive cuff repairs are often extremely painful, and an interscalene block is useful. Consider a PCA, and a loading dose of morphine during surgery. Consider an interscalene catheter with infusion of LA.[33]

Arthroscopic shoulder procedures

- Used diagnostically and for surgical procedures.
- Examples: acromioplasty, stabilisation, adhesiolysis and rotator cuff repair.
- Two to three ports (e.g. posterior port for viewing and anterior or lateral ports for instrumentation).
- Posterior port may require additional LA infiltration if surgery is performed under interscalene block alone.[32]

Open shoulder procedures

- Examples: arthroplasty of the glenohumeral joint, open stabilisation, open rotator cuff repair, subacromial decompression and procedures for trauma.[32]

Preoperative considerations

- Discuss the planned anaesthetic technique with the patient, with particular emphasis on proposed regional anaesthetic techniques and their complications.
- Choice of anaesthetic technique will depend on both patient and surgical factors.
- The RCoA produces patient information leaflets such as *Nerve blocks for surgery on the shoulder, arm or hand* which may be provided to the patient prior to surgery.[34]
- If surgery is to be performed with the patient conscious or sedated while employing a regional technique, the patient must understand that they are not receiving a GA.[35]
- Patients may not tolerate awake surgery, even with a fully working block, as instruments and drapes may be close to the face.[32]
- Patients should be aware of the likely postoperative course and the intended postoperative pain management regime.

- Many patients are elderly; severe rheumatoid disease is common and patients may have atlantoaxial instability and extra-articular involvement (see ➔ pp. 246–8).
- Ask about respiratory function/reserve if planning an interscalene block (some diaphragmatic function will be lost for several hours).
- Check the airway (particularly in RA) and range of neck movement. Some patients may require awake tracheal intubation.

Ultrasound-guided regional anaesthetic techniques

- Refer to ➔ Chapter 40 for details of how to perform specific blocks (see ➔ pp. 1119–27).
- Ultrasound-guided regional anaesthesia has numerous advantages in shoulder surgery: reduced postoperative pain despite reduced concomitant perioperative opioid usage, reduced PONV and subsequently improved patient satisfaction.[31]
- Regional techniques can be used on their own, or in combination with sedation or in conjunction with a GA.
- IV access should be inserted into the opposite arm or the feet prior to commencing the block.
- The appropriate block will depend upon the specific operative requirements, but common examples are interscalene and supraclavicular blocks.
- Continuous catheter-based interscalene block may provide superior analgesia, compared with single-injection interscalene block, in major shoulder surgery without increasing side effects or complications.
- Patients should be made aware of the adverse effects of the intended nerve block.

Intraoperative considerations

- The efficacy of any regional anaesthetic technique should be confirmed prior to commencing surgery.
- Use full monitoring, whether or not GA/sedation is to be used.
- Ensure the patient's bladder is empty prior to starting surgery, or insert a urinary catheter for longer procedures.
- Antibiotic prophylaxis should be given as per local protocols (see ➔ pp. 62–3).
- Avoid intraoperative hypothermia.
- Patients are usually positioned with their head distal to the anaesthetist, which presents several challenges:[31,32]
  - Pay particular attention to the security of the airway which may be covered by drapes and be out of sight.
  - Airway options include a supraglottic device, a south-facing RAE tube or a flexible ETT, depending upon the operation.
  - Long ventilator and gas sampling tubing is required.

  Long IV lines are required.
- Positioning: supine, head-up tilt, lateral, beach-chair position (see ➔ pp. 440–1).
- Be aware of complications when the patient is steeply head up in the beach-chair position:[32]
  - Cardiovascular instability or cerebral ischaemia; may benefit from a vasopressor infusion or cerebral oximetry monitoring.
  - Consider invasive arterial BP monitoring in selected patients.
  - Air embolus.

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  - Ensure the head and neck are properly secured.
  - Ensure eye padding.
  - Heels and arms at risk of pressure sores, so ensure they are padded properly.
  - Avoid overstretching the brachial plexus.
  - The ulnar nerve at the elbow is vulnerable.
  - Flex the legs at the knees (e.g. with a pillow) to avoid stretching the hamstrings.
- If the patient will be conscious during the operation, the surgical drapes must not cover their face.
- Have a strategy for managing intraoperative pain if the patient is awake (e.g. alfentanil bolus).
- Make use of multimodal analgesic techniques intraoperatively.
- The surgeon can infiltrate LA intraoperatively.
- Consider the use of tranexamic acid if there is significant blood loss.[36]

Postoperative considerations

- Most procedures can be performed as a day case, so the patient will be able to go home postoperatively.
- Patients must be given strict guidance to protect the limb while the nerve block is still working. A suitable sling can help to support the limb.
- The nerve block will eventually wear off (often when the patient is at home) and the patient can experience extreme discomfort.
- The patient must be prescribed (and encouraged to take) a multimodal analgesic regime, including paracetamol, NSAIDs and opioids.
- The patient should have appropriate postoperative physiotherapy.[6]

Total shoulder replacement

Procedure	Prosthetic shoulder replacement
Time	2–3h
Pain	+++/++++
Position	Supine, head-up or deck-chair
Blood loss	250–500mL
Practical techniques	GA with ETT (south-facing RAE) + IPPV, interscalene block ± interscalene catheter[33]

NICE has produced a guideline on the perioperative management of shoulder replacements.[6]

Indications

- Acute trauma, osteoarthritis, rotator cuff tear, trauma sequelae, other inflammatory arthropathies and avascular necrosis.[1,37]
- Osteoarthritis of the shoulder is the commonest indication for elective shoulder replacement.
  - Increasingly common as the population ages. The 2012–2019 NJR data showed the median age of patients was 73y.[1]
  - Presentation: pain, ↓ shoulder movement and function.
  - Initial treatment is non-operative: activity modification, oral analgesia, physiotherapy and steroid injections.
  - If non-operative strategies fail, the patient may require a shoulder replacement.

Types of shoulder replacement

- 1° total shoulder replacement: best indicated for patients with intact and functional rotator cuff muscles.
- Reverse total shoulder replacement: does not rely on an intact rotator cuff, often used in older patients.
- Hemiarthroplasty: younger patients with 1° osteoarthritis and in proximal humeral fractures.

Procedure-specific considerations

(For general principles, see ➲ pp. 634–6.)[31,32]

- If interscalene block has been performed, anaesthesia is usually unremarkable. Sometimes breakthrough stimulation occurs during the glenoid phase (may receive fibres from T2 which are not always covered by the block). If no interscalene block, load the patient with morphine and ask the surgeon to infiltrate with LA.
- Pain is worst in the first 24h postoperatively. PCA/intermittent morphine is usually satisfactory.
- Consider tranexamic acid if there is significant blood loss.[36]

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Elbow replacement surgery

Procedure	Prosthetic elbow replacement
Time	Variable
Pain	+/++
Position	Supine, arm out on table
Blood loss	Minimal
Practical techniques	GA, LMA/ETT, ultrasound-guided regional technique, tourniquet, multimodal analgesia

In 4373 operations between 2012 and 2019, NJR data show the median age of patients for 1° elbow replacement was 68y.[1]

Indications for elbow replacement surgery

- Acute trauma
- Osteoarthritis
- Other inflammatory conditions
- Trauma sequelae
- Essex-Lopresti fracture
- Avascular necrosis.

Types of elbow replacement surgery

- Total prosthetic replacement
- Radial head replacement
- Lateral resurfacing
- Distal humeral hemiarthroplasty.

Anaesthetic technique

- Assess the patient for manifestations of rheumatoid disease (see pp. 246–8).
- Insert IV access into the side opposite to surgery.
- Ultrasound-guided regional anaesthetic techniques will provide intraoperative and postoperative analgesia[38] (e.g. supraclavicular (see pp. 1120–1) or infraclavicular block (see pp. 1121–2)).
- Prophylactic antibiotics as per local protocol (see pp. 62–3).
- A tourniquet is often used; ketamine may help with tourniquet pain.
- Ensure careful positioning to prevent tissue injury and to reduce postoperative pain from other arthritic areas.
- Postoperative ulnar nerve compression is common and may necessitate further surgery.

Anaesthesia for hand surgery

Procedure	Various
Time	Variable
Pain	+/+++
Position	Supine, arm out on table
Blood loss	Minimal
Practical techniques	Regional analgesia ± GA, multimodal analgesia, tourniquet

The majority of hand surgery procedures (Table 23.5) are suitable for LA or regional anaesthesia as a day case. This can be combined with GA or additional sedation, if required. Some procedures, such as carpal tunnel release or trigger finger release, can be done under local infiltration alone. IV regional anaesthesia (IVRA), e.g. Bier's block, is suitable for procedures below the elbow of 30min or less (see ➲ p. 1127).

An upper arm tourniquet is almost always used for any type of hand surgery. Positioning and duration of use will be an important determinant of whether the patient is able to tolerate regional anaesthesia or LA alone. Patients with a good brachial plexus block will usually tolerate 60–90min of arm ischaemia.[38]

An axillary brachial plexus block can provide excellent anaesthesia to the hand, arm and forearm, although tourniquet pain may be a problem. Other approaches include infra- and supraclavicular approaches (see ➲ pp. 1120–1; ➲ pp. 1121–2).[38]

Preoperative

- Full assessment as for GA. The patient may request a GA, and regional anaesthesia may fail.
- Check that patients can lie flat for the proposed duration of operation if planned to be awake.
- Assess movement of the operative arm. Can the patient achieve the necessary position for regional block or the surgery planned?
- Discuss the planned anaesthetic technique with the patient, with particular emphasis on proposed regional anaesthetic techniques and their complications.
- The RCoA produces patient information leaflets such as *Nerve blocks for surgery on the shoulder, arm or hand* which may be provided to the patient prior to surgery.[34]
- If surgery is to be performed with the patient conscious or sedated while employing a regional technique, the patient must understand that they are not receiving a GA.[35]

Perioperative

- Make sure the patient's bladder is empty.
- Use full monitoring, whether or not GA/sedation is to be used.
- Prophylactic antibiotics as per local protocol.
- Perform a local block, with the patient awake or lightly sedated.
- Choose an appropriate and familiar block for the planned site of surgery ± tourniquet.

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Table 23.5 Surgical procedures on the hand

Operation	Description	Time (min)	Pain (+ to +++++)	Notes
Trigger finger release and carpal tunnel release	Tendon or nerve release	5–15	+	These procedures can usually be carried out under LIA
Dupuytren's contractures (simple)	Usually confined to ulnar and median distribution. Usually <30min tourniquet time	<60	+	GA with wrist block or infiltration. Brachial plexus block with upper arm tourniquet ± GA. Quick procedure: wrist block with wrist tourniquet
Dupuytren's contracture (complex)	Severe disease or redo procedure may need skin grafting	60–120	+	Prolonged tourniquet time means that a brachial plexus block or a GA with local block is often required
Metacarpophalangeal (MCP) joint replacement (e.g. Swanson)	MCP joint replacement usually for rheumatoid	30 per joint	++/+++	Generally frailer patients with systemic disease
Tenolysis, capsulotomies, tendon grafts	These procedures may need patient participation to assess the adequacy of the procedure	15–60	+/++	If hand movement is required, then any block must be distal. A wrist block with sedation is usually adequate
Digit reimplantation	Microvascular surgery	Hours	++	GA is usually required because of the prolonged procedure. Regional anaesthesia for the sympathectomy is helpful
Ulnar head excision or trapeziectomy	Surgery for wrist pain in rheumatoid disease	30–60	++/+++	As pain is severe, a single-shot brachial block or catheter technique is ideal, with or without GA

- Augment plexus anaesthesia with elbow or wrist blocks, as necessary, to improve success rates (see ➲ pp. 1124–6; ➲ pp. 1126–7).
- Provide sedation or GA, depending on safety and the patient's wishes. Have equipment and drugs ready to convert to sedation or GA, if necessary, during the operation.

Postoperative

- Surgery involving soft tissues and the skin is generally less painful than surgery to the bones and joints.
- Simple analgesic combinations are usually adequate for the less painful procedures.
- Opioids or regional catheter techniques may be required for the more painful operations.
- Some patients dislike the postoperative 'dead arm' following brachial plexus block.

Special considerations

- Tourniquet pain can be reduced by blocking the intercostobrachial nerve SC on the medial aspect of the upper arm above the level of the tourniquet (see ➲ p. 1124).
- Adrenaline-containing solutions should be avoided near digits.

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Anaesthesia for femoral neck fracture

Procedure	Cannulated screws, dynamic hip screw (DHS), cemented/uncemented hemiarthroplasty, total hip replacement
Time	10–90min
Pain	+/+++
Position	Supine (? on hip table), occasionally lateral
Blood loss	250–750mL
Practical techniques	SV LMA and regional block Spinal ± sedation ETT + IPPV

Hip fractures are common: ~67 000 per annum in the 2020 National Hip Fracture Database report[39] (80% ♀). Every hospital that does hip fracture surgery should have a National Hip Fracture Database Anaesthesia Lead. Average age is 84y, and 80% occur in those >75y. In Western society, the lifetime risk is 18% (women) and 6% (men). The 3mo mortality is ~12%, increasing to 21% at 1y.[40]

Preoperative

- Physiological reserve is reduced, and comorbidity is common. Ideally, resuscitation should start as soon as the patient is admitted to hospital. Thorough preoperative assessment must take place, and surgery should be scheduled for the earliest possible daytime session.
- Patients should be risk-assessed prior to surgery, e.g. with the Nottingham Hip Fracture Score, a frailty score and the 4AT delirium score.[2]
- Surgical treatment can be either fracture fixation or femoral head replacement, depending on the nature of the fracture, surgical preference, previous mobility and life expectancy.
- Determine which procedure is to be performed (Table 23.6). Cannulated hip screws are quick, largely non-invasive procedures with a small incision and little blood loss. Cemented/uncemented hemiarthroplasty is a longer procedure, similar to the femoral part of a 1° hip replacement. DHS/Richards screw and plate are intermediate procedures.
- Any decision to delay surgery should be based on a realistic attempt to improve the patient's medical condition, rather than a fruitless pursuit of 'normal' values. A mild chest infection is unlikely to improve in a bed-bound elderly patient, whereas frank pneumonia with sepsis and dyspnoea may respond to rehydration, antibiotics and chest physiotherapy.
- Surgery should not be delayed pending echocardiography for suspected valvular disease.[2]
- Hip fracture surgery should take place within 36h of sustaining a fracture.[2]
- Many patients will be taking anticoagulant or antiplatelet therapy; see the AoA guideline for advice on specific management[2] (see p. 1109).
- Good communication between the surgeons, orthogeriatricians and anaesthetists is important. There should be a daily multidisciplinary team discussion of each patient scheduled for surgery.

Table 23.6 Surgical procedures for fractured neck of femur

Operation	Description	Time (min)	Pain (+ to +++++)	Position	Blood loss/ X-match	Notes
Cannulated screws	Screws across femoral neck (previously 'Garden screws')	20	+++	Supine, hip table	Nil	Minimally invasive, small thigh incision. Can be done with local/nerve block and sedation, if necessary. X-ray-guided
Richards screw and plate (RSP)	Plate along femur, with compression screw into femoral head	30–45	++	Supine, hip table	<400mL	Somewhat larger thigh incision/blood loss. X-ray-guided
Dynamic hip screw (DHS)	As RSP	30–45	++	Supine, hip table	<400mL	As RSP
Dynamic compression screw (DCS)	As RSP	30–45	++	Supine, hip table	<400mL	As RSP
Total hip replacement	As for total hip replacement	60–90	Offered to those who were able to walk independently and were not cognitively impaired prefracture. For displaced intracapsular fractures	Lateral		A recent study shows the incidence of 2° procedures after 2y does not differ significantly between patients who receive total hip arthroplasty and those who receive hemiarthroplasty[42]
Intramedullary nail	Used for extracapsular fractures	45–60min	For extracapsular fractures	Supine		

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- Dehydration is common, as oral intake is often much reduced. IV fluids must be commenced as soon as the patient is admitted to hospital.
- Analgesia should be commenced, as often the patient is in considerable pain. A fascia iliaca block instituted in an ED setting can provide 'dynamic' analgesia and reduces the requirement for opioids.[41]

Perioperative

- For fracture fixation, the patient is usually positioned supine on a 'hip table'. This involves placement of a groin prop, with the table supporting the upper body only. Feet are tied into shoe supports, and the table is then elevated to allow radiographic screening. For hemiarthroplasty, the patient is lateral or supine on an ordinary operating table.
- Blood loss is variable. Much of the measured loss is old haematoma, but significant haemorrhage may occur and necessitate transfusion.
- Transfusion trigger is controversial but should be 90g/L.[43]
- Choice of anaesthetic technique. Regional anaesthesia and GA are both advocated, but the recent emphasis is on the maintenance of physiological stability, particularly maintenance of BP.[2,44,45]
- Peripheral nerve blocks should be used routinely to supplement GA or spinal anaesthesia (see ➲ pp. 1135–44).[2]
  - Sedation may be necessary, but any sedative can produce unpredictable effects in the elderly and should only be used when necessary.
- Check pressure points after placement on the 'hip table', as these patients are prone to pressure damage.
- Use some form of passive or active warming device to prevent hypothermia. Insulate the head, and secure a warming blanket/polythene sheet around the chest and lower abdomen.
- BCIS is a problem with hip fracture (see ➲ p. 615).[11,12,13]
- Take care to avoid intraoperative hypotension.

Postoperative

- Pain is often only due to the incision, which is small for cannulated screws and DHS, but larger for hemiarthroplasty, although DHS procedures may cause a considerable amount of postoperative pain.[46]
- Fracture pain will be reduced but is still present on rolling and turning in bed.
- Postoperative analgesia can be provided by regular IV paracetamol; opioids should be used sparingly.[47] Most patients will require some postoperative analgesia, although some do not. Take care with NSAIDs because of the ↑ risk of GI and renal complications (see ➲ p. 91; ➲ pp. 1155–6).
- Some patients may require a period of monitoring in the post-anaesthesia care unit, HDU or ICU postoperatively.[2]

Special considerations

- In high-risk patients, procedures can be undertaken with LA alone.
- Morbidity and mortality risks should be understood by the patient and relatives, and in some patients, the resuscitation status should be reviewed.
- A useful resource for all anaesthetists involved in the management of hip fracture patients is the NHS Hip Fracture Perioperative Network.[48]

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Chapter 24

Plastic and burns surgery

Brian Chen, Simon Davis and **Fynn Maguire**

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See also

➲ Burns: early management pp. *1013–18*

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General principles

The complexity of anaesthesia for plastic and burns surgery ranges from the routine to the challenging. Some extensive procedures (e.g. free flap repairs, craniofacial reconstruction) may involve invasive monitoring, extensive blood loss and postoperative intensive care support. See Table 24.1 for a range of plastic surgical procedures.

Regional techniques and sedation

Minor body surface procedures may be performed under LA infiltration alone. Significant body surface procedures in those unfit for GA, such as excision and grafting of skin tumours, can be accomplished using extensive infiltration of LA and IV sedation. Upper and lower limb surgery is especially suitable for regional anaesthesia. Consider the surgical site, placement of tourniquets and the location of split skin grafts (SSGs), if required, when deciding on the best regional block. Long procedures or those which would benefit from ongoing regional sympatholysis, such as digit reimplantation, would benefit from a regional catheter. Sedation to supplement a regional technique may be required in anxious patients or for longer or extensive procedures. Use any sedative technique which is familiar and appropriate for the patient's physiology, ideally with an agent which is easily titratable. Propofol (0.5–1.0 micrograms/mL TCI or 10–15mL/h of 1% solution), with or without supplemental midazolam (1–2mg), is effective. Alternatively, remifentanil (0.5–1.0 nanograms/mL TCI or 5mL/h of 1mg in 20mL solution) offers both sedation and analgesia, and respiratory depression can be avoided if the infusion rate is slowly titrated upward.

The difficult airway

(See also ➔ pp. 363–4.) Patients with head and neck pathology causing airway difficulty are often encountered. Airway difficulty may arise from anatomical deformity due to tumour, trauma, infection, previous operations, radiotherapy or scarring. Competence in difficult airway techniques (e.g. awake intubation) is required. The 'shared airway' is regularly a feature of head and neck surgery. Discuss with the surgeon which tube you propose to use and by which route to achieve the best surgical access (oral, nasal, conversion to tracheostomy) and how the tube will be secured (tied, taped, stitched).

Poor access to the patient

The operating site may be extensive (e.g. burns debridement) or multiple (e.g. free flap procedures). This may produce added difficulty with:

- Heat conservation: it may be difficult to achieve enough access to the patient's body surface area to maintain temperature. Heated underblankets are useful.
- Monitoring: ECG leads, the pulse oximeter probe and the BP cuff may all be difficult to position adequately.
- Vascular access: position cannulae away from the operative field. Use femoral vessels or the foot, if necessary. Long extension sets may be required.

Long operations

Patients undergoing complicated reconstructive procedures may be in theatre for many hours. Give careful consideration to:

Vascular access Check that line placement will not interfere with the site of surgery. Invasive arterial monitoring is desirable. A central venous line can assist with estimations of the intravascular volume and provide dependable venous access in the postoperative period. Site at least one large-bore peripheral (14–16G) cannula for fluid administration and a small cannula (20–22G) for other infusions such as TCI and PCA.

Blood loss Ensure blood has been X-matched. The initial dissection is usually the period of most blood loss, and a moderate hypotensive technique may help to limit this. Thereafter, losses may be insidious and ongoing. Aim to keep track by swab weighing, visual estimation and regular Hb or Hct measurement. Non-invasive CO monitoring can help optimise the fluid status. Assessment of stroke volume, flow time or SVV, using devices such as LiDCO™, may give valuable information to the anaesthetist.

Fluid balance Urinary catheterisation is essential. Ensure careful monitoring of fluid balance, especially in children and patients with poor cardiorespiratory function.

Body temperature Monitor the core temperature (e.g. rectal, nasopharyngeal, oesophageal). Maintain the temperature by using low FGFs, an HME filter, warmed IV fluids, a warm ambient theatre temperature (e.g. 24°C), a heated mattress or a forced air warming blanket. Take care not to overheat the patient.

Positioning Ensure that structures, such as the cervical spine and brachial plexus, are not in positions of stress. Take care with pressure areas. Pad bony prominences and raise the heels off the table.

VTE DVT prophylaxis is often initiated during surgery. All patients should have thromboembolism compression stockings and intermittent calf compression while in theatre, and be assessed for daily LMWH.

NGT Consider emptying the stomach. Children are especially prone to gastric distension during prolonged procedures.

Eye care Lightly tape and pad the eyes for protection. Avoid excessive padding, since this may negate the natural protection afforded by the bony orbit. Prophylactic antibiotic ointment is unnecessary. Do not allow corneal abrasion to develop from surface drying. A simple eye ointment is helpful if the eyes are left uncovered.

ETT cuff pressure Cuff pressure will gradually increase if N_2O is used. Where possible, recheck the cuff pressure at intervals during the case.

Postoperative care Discuss the preferred site of postoperative care with the nursing staff and surgical team. Surgeons often prefer patients to return to the plastic surgery ward where wound care and nursing observation may be more attuned to the specifics of the operation. Closer patient observation, invasive monitoring and regular blood gas estimation may be more achievable in an ICU/HDU. The site for immediate postoperative care is principally dictated by the general condition of the patient.

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Table 24.1 Miscellaneous plastic surgical procedures

Operation	Description	Time (min)	Pain (+ to +++++)	Position	Blood loss/ X-match	Notes
Abdomino-plasty	Excision of redundant lower abdominal skin	120	++ to +++	Supine	G&S	LMA or ETT, IPPV
Carpal tunnel release	Release of flexor sheath at the wrist to relieve median nerve entrapment	30	+	Supine, arm board	Nil (tourni-quet)	LA infiltration, brachial plexus block or day case GA
Dupuytren's contracture	Excision of contracted palmar fascia	60–90	+	Supine, arm board	Nil (tourni-quet)	Brachial plexus block or day case GA
External angular dermoid	Excision of congenital dermoid cyst, usually from lateral supraorbital ridge	30	+	Supine, head ring	Nil	LMA and SV
Flexor/ extensor tendon repair	Repair of hand tendons following trauma. Often multiple. May be extensive. May involve nerve/vessel repairs	30–120	+ to ++	Supine, arm board	Nil (tourni-quet)	Brachial plexus block ± GA, LMA and SV, IPPV for extensive repairs
Gynaeco-mastia	Excision or liposuction of excess ♂ breast tissue	45	+ to ++	Supine	Nil	LMA and SV
Hypo-spadias repair	Correction of congenital abnormality of ♂ urethra. Usually infant	90	++	Supine	Nil	LMA and SV. Caudal or penile block
Insertion of tissue expander	SC insertion of 0.9% sodium chloride-filled silicone bags, often scalp	45	+ to ++	Supine, head ring	Nil	LMA and SV

Operation	Description	Time (min)	Pain (+ to +++++)	Position	Blood loss/ X-match	Notes
Neck, axilla and groin dissection	Block dissection of regional lymph nodes to excise 2° malignant disease	90–120	++	Supine, head ring	2 units	LMA or ETT, IPPV
Otoplasty	Uni-/bilateral correction of prominent ears	60	+	Supine, 30° head up	Nil	Paediatric patients. Flexible LMA and SV. Day case
Preauricular sinus	Excision of congenital sinus tract, often bilateral	45	+	Supine, head ring	Nil	LMA and SV
Pretibial laceration	Excision of pretibial wound and SSG	45	+ to ++	Supine	Nil	Spinal or GA
Rhytid-ectomy	Cosmetic facelift, occasionally combined with septo-rhinoplasty	180–240	+ to ++	Supine, 30° head up	Nil	LMA or ETT, IPPV. Permissive hypotension
Syndactyly	Release of congenital fusion of two or more digits. May be bilateral. May require full-thickness skin graft (FTSG)	60–180	++	Supine	Nil (tourni-quet)	LMA and SV. ETT + IPPV for extensive repairs

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Smooth emergence

Avoid the patient coughing and straining at the end of the procedure. This will put tension on delicate suture lines and increase bleeding and haematoma formation, especially for facial procedures. Deep extubation, deep conversion of ETT to LMA (Bailey manoeuvre), extubation with a running remifentanil infusion or IV lidocaine 1.0mg/kg bolus 2min prior to extubation can smooth emergence.

Analgesia

- Most procedures are performed on the body surface. These tend to be less painful than procedures involving the body cavities and are usually amenable to LA infiltration or regional block. Continuous catheter techniques may be useful in limb procedures.
- Patients recovering from head and neck procedures are often surprisingly comfortable despite extensive surgery.
- Major body cavities and abdominal musculature are usually not involved. The pain experienced after an abdominoplasty is significantly less than pain following a laparotomy.
- Plastic surgery procedures seldom involve new fractures of long bones.
- The GI tract is usually unaffected. The oral route for drugs is frequently available, which may make dosing and administration of analgesics simpler.

Attention to detail

Successful anaesthesia for plastic surgery requires careful attention to detail. Patients for aesthetic surgery will have high expectations and will be well informed.

Breast surgery

General considerations

Breast cancer is now the commonest cancer in the UK, and the incidence has ↑ by >20% over the last 25y. Mortality from breast cancer, however, has fallen steadily since 1990, probably because of earlier detection and improved treatment. Over this time, there have been significant advances in more extensive combined procedures of breast resection and reconstruction. Radical mastectomy is now rarely indicated, as better efficacy is shown for breast-conserving treatment, such as wide local excision of tumour, in addition to chemotherapy or radiotherapy, compared to full mastectomy alone. The minimally invasive technique of sentinel lymph node biopsy has now predominantly replaced axillary lymph node dissection for breast cancer staging.

Preoperative

Anxiety is often high. It is important to gain the patient's confidence at the preoperative visit, discuss analgesia and prescribe anxiolysis if necessary.

- Adverse effects from any neoadjuvant therapies should be considered and optimised. Patients who have recently undergone chemotherapy may be immunocompromised. Check FBC for evidence of bone marrow suppression and anticipate potentially difficult venous access.
- Reconstructive procedures, mastectomy following radiotherapy, mastectomy where breasts are large and breast reduction surgery increase the risk of blood loss. Check Hb and ensure blood is grouped and screened.

Perioperative

LMA and SV are often appropriate for short- to medium-length procedures. Use intubation and mechanical ventilation for prolonged procedures, the obese and patients at risk of aspiration.

- Avoid placing venous access on the side of surgery.
- Standard monitoring is appropriate for most procedures. Longer procedures will require active warming and temperature measurement.
- Additional invasive monitoring may be required for prolonged reconstructive procedures, including free flap surgery.
- Give multimodal analgesia, including NSAID, systemic opioid and regional techniques, if possible.
- Breast surgery patients are at high risk of PONV. Avoid causative agents, and administer prophylactic antiemetics. TIVA may be useful.
- Beware of the blue dyes used in sentinel lymph node biopsy as they have been associated with anaphylaxis.

Regional analgesia

Regional techniques may offer advantages in some cases; however, the risks in healthy women undergoing minor procedures may outweigh the benefits.

- Consider regional techniques for more radical procedures, e.g. radical mastectomy/axillary clearance and breast reconstruction.
- Regional techniques include paravertebral block, thoracic epidural, interfascial plane (pectoral nerve and serratus anterior plane) blocks, intercostal blocks and intrapleural block. Beware of the complications of each technique. An ultrasound-guided approach is advocated.
- Recent evidence suggested that breast surgery supplemented by a paravertebral block does not reduce the risk of cancer recurrence.[1]

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Postoperative

- Minor procedures, including wide local excision with sentinel lymph node biopsy, could be suitable for day case surgery.
- HDU may be required after extensive procedures.
- If a paravertebral catheter or thoracic epidural is sited, continue infusion postoperatively.

Special considerations

Patients may present with previous breast surgery and axillary clearance. Cannulation should be avoided in the arm on the affected side due to the risk of infection and potential development of lymphoedema. There is limited evidence of the risk of short-term cannulation on the affected side, and if venous access is limited, it may be appropriate to use the affected side and remove at the end of the case.

- Chronic pain, typically presenting in the affected anterior chest wall, ipsilateral axilla or upper arm, may occur following breast surgery. Incidence is as high as 20–50% after mastectomy. Intensity of the pain following extensive surgery, postoperative radiotherapy and chemotherapy are risk factors.
- Breast reconstruction following mastectomy is common. The two main methods include implant-based or autologous tissue flap reconstruction. The tissue flap can be transferred as a free flap or a pedicled flap. Options for flap reconstructions are listed in Table 24.2.

References

1 Sessler DI, Pei L, Huang Y, *et al*. (2019). Recurrence of breast cancer after regional or general anaesthesia: a randomised controlled trial. *Lancet*, **394**, 1807–15.

Table 24.2 Types of tissue flap reconstruction

Type	Donor site	Free or pedicled flap	Harvest position
Abdomen			
TRAM	Transverse rectus abdominis muscle	Free or pedicled	Supine
DIEP	Deep inferior epigastric perforator	Free	Supine
SIEA	Superficial inferior epigastric artery	Free	Supine
Back			
Latissimus dorsi	Latissimus dorsi muscle	Pedicled	Lateral decubitus
LAP	Lumbar artery perforator	Free	Prone
Hip/buttock			
Gluteal	Superior gluteal artery perforator, inferior gluteal artery perforator	Free	Prone or lateral decubitus
DCIA	Deep circumflex iliac artery	Free	Supine
Thigh			
PAP	Profunda femoris artery perforator	Free	Supine or prone
TUG	Transverse upper gracilis muscle	Free	Supine
TFL	Tensor fasciae latae	Free	Supine
LTTF	Lateral transverse thigh	Free	Supine
ALT	Anterolateral thigh	Free	Supine

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Breast reduction

Procedure	Reduction of breast size by glandular resection. Usually bilateral
Time	3h
Pain	++
Position	Supine, 30° head-up, periodic deck-chair. Arms may be positioned on boards, or with elbows flexed and hands placed behind the upper part of the buttocks
Blood loss	500mL, G&S
Practical techniques	IPPV via ETT or LMA

Preoperative

Bilateral breast reduction is not primarily an aesthetic procedure. These patients may suffer from severe neck and back pain. Participating in exercise and sport is not possible. There may be symptoms of emotional disturbance.

- Patients are usually fit; aged 20–40y. Many surgeons exclude patients with a BMI >30 due to a higher incidence of wound breakdown, infection and haematoma formation.
- A mastopexy is a surgical procedure for correcting breast ptosis when breast volume is adequate. Anaesthetic implications are similar. Blood loss is less.
- Check FBC and G&S. X-matching is generally unnecessary, except for larger reductions.
- Timing in relation to the menstrual cycle is unimportant.
- All patients should receive DVT prophylaxis (compression stockings, daily LMWH).

Perioperative

- IPPV may be preferable, since the surgeon often puts pressure on the chest wall during surgery. IPPV will maintain satisfactory chest expansion with good aeration and control of $PaCO_2$, and help minimise blood loss. An LMA may be satisfactory for IPPV.
- Place ECG electrodes on the patient's back. Lie the patient on incontinence pads to absorb blood loss.
- Take care to position the patient carefully on the operating table. The anaesthetic machine is usually at the head end. Ensure that the chest and arms are symmetrical. Confirm that vascular cannulae are secured well and padded if the hands are to be positioned behind the buttocks. Local pressure damage to the skin may otherwise ensue.
- The patient may be placed in a deck-chair position briefly multiple times throughout the operation to allow the surgeon to check breast symmetry. The position changes mandate care with securing airway, vascular access, monitoring and maintenance of CPP.
- Blood loss depends on the surgical technique. Use of cutting diathermy causes less bleeding than a scalpel. Infiltration with dilute adrenaline-containing LA helps reduce blood loss. All surgeons have their own recipe. Check the dosage being used; in practice, this is seldom a concern (see ➔ p. 1102).

- Fewer than 5% of patients require transfusion. Mild falls in Hb are well tolerated in this young patient group.
- Moderate reductions may involve removal of 500g of tissue per breast.

Postoperative

- Bilateral breast reduction does not cause significant postoperative pain. Following a dose of morphine towards the end of surgery, regular simple analgesics and NSAIDs are usually adequate. IV PCA is generally unnecessary.
- Haematoma formation is an early complication. Occasionally, nipple perfusion may be compromised and requires decompression of the pedicle. Return to theatre may be indicated. Later complications include wound infection, dehiscence and fat necrosis.

Special considerations

Occasionally, patients for massive breast reduction are encountered (>1kg of tissue removal per breast). Two to 4 units of blood should be X-matched. The complication rate is higher.

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Breast augmentation

Procedure	Bilateral or unilateral augmentation of breast size
Time	90min
Pain	++/+++
Position	Supine, 30° head-up. Arms may be out on boards, or with elbows flexed and hands placed behind the upper part of the buttocks
Blood loss	Minimal
Practical techniques	SV or IPPV via LMA

Preoperative

Breast augmentation may be performed for:
- Reconstruction following mastectomy
- Correction of breast asymmetry
- Aesthetic bilateral augmentation.

Patients are usually fit and well.

Perioperative

Position on the operating table as for breast reduction.
- The four 1° augmentation techniques include inframammary, periareolar, transaxillary and transumbilical.

Postoperative

Postoperative discomfort may be related to the size of the implants. Large implants cause more tissue stretching and postoperative pain.
- In general, breast augmentation appears to cause more discomfort than breast reduction. Give regular simple analgesics. Opioid analgesia may be needed, but PCA techniques are seldom required.
- Haematoma formation may require early return to theatre. Later complications include infection, capsule formation and rupture.

Special considerations

An association between silicone breast implants and the development of systemic symptoms of connective tissue diseases has been suggested. This association has not been proven, following data from large studies.
- Soybean oil-filled implants have been withdrawn from use in the UK. There are insufficient data concerning the long-term consequences of soybean oil breakdown. Sodium chloride 0.9% implants are not perceived as sufficiently realistic and are unpopular with many women.

Free flap surgery

Procedure	The transfer of tissue from a donor site and microvascular anastomosis to a distant recipient site
Time	Variable, depending on procedure, often 6–8h or longer
Pain	++/+++
Position	Variable.Usually supine. May require position change during surgery
Blood loss	Variable, G&S to X-match 4–6 units
Practical techniques	ETT + IPPV, arterial line, CO monitoring, urinary catheter, patient warming

Preoperative

A free flap is a transfer of tissue with its blood supply from one part of the body to another when the vessels are disconnected during the transfer and microvascularly reanastomosed to a new artery and vein at or near the recipient site. The transferred tissue volume may contain skin, muscle, nerve, fascia and bone. Free flaps are commonly used to provide tissue cover for large defects following trauma or resection for malignancy, or when complex tissue is required to regain normal aesthetics or function.

- Surgical success depends on establishing and maintaining adequate flap perfusion. Hypercoagulable states, such as sickle-cell anaemia and polycythaemia, are contraindications to surgery. Smokers should be advised to stop smoking for at least 4w before surgery due to nicotine-induced vasoconstriction, carbon monoxide (CO)-related tissue hypoxia and blood hypercoagulability caused by ↑ platelet aggregation. In oncology patients, consider chemotherapy-related organ system dysfunction and anticipate difficulty gaining IV access.
- Elderly patients with head and neck cancer are often heavy smokers and drinkers and may have significant cardiac and respiratory comorbidity, as well as poor nutritional state. Their airway anatomy may be distorted, especially with previous head and neck surgery or radiotherapy, increasing the risk of a difficult intubation.

Perioperative

The physiological aim of anaesthesia is to optimise circulatory flow through the grafted flap. Anaesthetic management is largely guided by 1st principles and application of the Hagen–Poiseuille equation:

$$Q = \frac{\pi P r^4}{8 \eta l}$$

where Q = flow rate, ΔP = pressure gradient, r = vessel radius, l = vessel length, and η = blood viscosity.

The variables which can be controlled by the anaesthetist are pressure gradient, viscosity and vessel radius.

- Free flap surgery is a long procedure on multiple body sites and all patients should have a GA. Pain often originates from the flap donor site or from SSGs taken to cover deficits in either the donor or the recipient site and a regional block of these areas adds to effective analgesia. The flap itself is denervated and insensate and the recipient site is often relatively pain-free.

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- Either propofol or volatile maintenance can be justified. Propofol lowers SVR, is rapidly metabolised and is an antiemetic, may avoid postoperative shivering and may be more favourable to microvascular circulation by avoiding the effect of volatiles on red cell membrane stiffness. Isoflurane maintains microcirculatory flow. Sevoflurane may attenuate ischaemic–reperfusion injury and desflurane has a faster offset. Due to the duration of surgery, N_2O should be avoided. The addition of a remifentanil infusion offers intraoperative analgesia and marked vasodilation controlling arterial pressure, and negates the need for an NMBA.
- Vascular access can be limited if the upper limbs are involved as flap donor or recipient or the patient has previously undergone chemotherapy. An arterial line is useful for measuring the Hct throughout surgery and as an additional tool in estimating fluid requirements.
- Viscosity is closely related to Hct and rises steeply when Hct exceeds 40%. Aim for 30–35% which gives the best balance between blood viscosity, arterial O_2 content and tissue O_2 delivery.
- IV fluid is administered to ensure adequate flap perfusion, with the aim of normovolaemic haemodilution in a patient with lowered SVR. The disrupted lymphatic system of a flap increases the risk of interstitial oedema. Excessive plasma expansion can disrupt the endothelial glycocalyx layer and is associated with oedema and flap failure. Comparative studies between crystalloid and colloid regimens during microvascular surgery are limited. Crystalloids have a poor volume-expanding ability and are known for their predisposing effect on oedema, although colloid use is becoming increasingly uncommon amid concerns of anaphylaxis and renal injury. Regardless of fluid selection, current practice is fluid-restrictive, not exceeding 6mL/kg/h. GDFT is becoming more common as minimally invasive CO monitors using pulse contour analysis are now widely available.
- The use of vasoconstrictors is a contentious issue. While controlled hypotension during the initial dissection phase of surgery can reduce surgical blood loss, a return to normotension during anastomosis of the flap is targeted to ensure an adequate perfusion pressure through the tissue. There are fears that systemic vasoconstriction leads to reduced flap perfusion despite very few good-quality studies to support this. General advice is judicious use of both fluid and vasoconstrictors, such as metaraminol, to achieve normotension in a euvolaemic patient.
- Hypothermia leads to vasoconstriction, in addition to the ↑ risk of myocardial ischaemia, perioperative blood loss and surgical site infection. Intraoperative hypothermia develops in a characteristic pattern whereby the greatest fall in core temperature is within the first 30–45min of induction. Warming measures should ideally start before induction and care should be taken to maintain patient temperature throughout surgery and into the recovery phase. If surgery requires a large body surface area of the patient to be exposed, consider an underbody heating device or a forced air warmer. This is in addition to the usual interventions of an overbody forced air warmer, an HME filter and IV fluid warming.
- Ensure adequate ventilation to normalise arterial PO_2 and PCO_2. Both hypoxia and hypocapnia will cause vasoconstriction, and hypercapnia can cause sympathetic nervous system stimulation.

Postoperative

- Aim for a smooth emergence, avoiding any strain on suture lines.
- Vasoconstriction from cold, pain, low circulating volume, hypotension and hypocapnia will threaten the flap and should be addressed. If postoperative shivering occurs, exclude hypothermia and consider treating with a forced air warming device, 0.5–3mg/kg IV tramadol or 20–50mg IV pethidine.
- In addition to regional anaesthesia, oral opioids are usually sufficient. Consider IV PCA when swallowing is impaired such as with head and neck flaps. NSAIDs have been associated with flap haematoma, but selective COX-2 inhibitors, such as celecoxib, have been shown to be safe.
- Flap observation is a specialised nursing skill and care is often best provided on the plastic surgery ward. The need to escalate to critical care will be determined by patient factors. Postoperative flap observation will evaluate the Doppler signal of the arterial supply, flap colour, capillary refill time, skin turgor, skin temperature and bleeding on pinprick. Compromised flaps can be salvaged if early detection occurs. A revision operation on a compromised flap should employ the same anaesthetic principles to promote optimum flap perfusion.

Special considerations

- The reimplantation of severed digits or limbs should be managed as for a free flap. A brachial plexus catheter is helpful in managing reimplanted digits as it provides a continuous vasodilated limb.
- A pedicle flap is constructed when A–V connections remain intact, but the raised flap is rotated to fill a neighbouring defect. This procedure requires the same anaesthetic principles to optimise flap circulation.
- Overall free flap survival is >95%; patients in a poor general condition with coexisting disease have the highest risk of flap failure.

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Liposuction

Procedure	Vacuum aspiration of SC fat via a small skin incision and a specialised blunt-ended cannula
Time	Variable 30–90min
Pain	+
Position	Variable, depending on site. Usually supine
Blood loss	1% of the volume of fat aspirated with fluid infiltration technique
Practical techniques	Local infiltration with IV sedation/LMA and SV

Preoperative

Liposuction is a procedure to manage lipoma, gynaecomastia or reducing the bulk of transplanted flaps. Alternatively, a cosmetic procedure where adipose tissue is either removed or used for autologous fat grafting.

- In the obese patient, be aware of altered drug pharmacokinetics and pharmacodynamic effects and consider associated comorbidities such as metabolic syndrome, hypertension, DM, IHD and OSA.
- Use caution with patients vulnerable to large doses of adrenaline used (IHD, MAOI use) or large volume of fluid infiltrated (cardiac or renal failure).

Perioperative

- The total amount of fat aspirated depends on patient requirement and surgical judgement.
- Fat is infiltrated with dilute LA with adrenaline. Back-and-forth movement of the cannula disrupts fatty tissue, which is then aspirated by either a suction apparatus or a syringe.
- Injection of fluid helps fat breakdown and aids aspiration. A typical recipe for an SC infiltration solution would be 1000mL of warmed 0.9% sodium chloride or Hartmann's solution containing 50–100mL of 1% lidocaine (0.05–0.1% in final solution) and 1mL of 1:1000 adrenaline. The 'Superwet' technique of 1mL of infiltrate per 1mL of anticipated aspirate is commonly used.
- The 'Tumescent' technique refers to a large volume of LA/adrenaline infiltrate to produce tissue turgor. Developed as an outpatient technique and performed without additional anaesthesia or sedation. A total of 3mL of infiltrate per 1mL of aspirate is often used. There is little evidence that this technique is superior to the Superwet technique, and it may produce more complications. It may provide unsatisfactory anaesthesia when used alone, and sedation or GA may be required.
- Blood loss depends on the volume of LA/adrenaline infiltrate used and the extent of liposuction required. Loss is ~1% of the volume of the aspirate for the Superwet and Tumescent techniques. This may increase to 40% without SC infiltration.
- Extensive liposuction physiologically resembles a burn injury, and large fluid shifts result. Replace aspirate 1:1 with IV crystalloid, although take caution with the Tumescent technique where there is a net fluid gain. Patients with heart failure are at risk of pulmonary oedema.

- While procedural mortality is rare, the leading cause is VTE and appropriate mechanical and/or pharmacological prophylaxis should be utilised.
- Limit total adrenaline dose to 6mg to avoid toxicity. Due to delayed absorption, peak plasma levels are reached after 3–5h. Patients with IHD should have the dose limited and be observed over this period.
- Dose safety limits for LA infiltration are controversial.
- Doses significantly higher than the conventional lidocaine/adrenaline toxic dose (7mg/kg) are often used. This may be possible due to adrenaline-related vasoconstriction slowing drug absorption, poor vascularity of fat, sequestration of the lipophilic LA into fat and aspiration of much of the infused solution before the LA has been absorbed. Most studies have shown safety with lidocaine doses of up to 35mg/kg, but keep in mind peak plasma levels occur 8–12h after infiltration. All facilities performing liposuction should have 20% lipid emulsion available for managing LA toxicity.

Postoperative

- Pressure dressings are usually applied.
- Encourage oral fluids, and monitor urine output.
- Check Hct following extensive liposuction (>2500mL of aspirate).
- Bruising can be considerable.
- Use NSAIDs and simple analgesics for pain relief.

Special considerations

Complications are associated with excessive liposuction. In the UK, aspiration is restricted to ~2L of fat. Considerably higher-volume procedures have been reported, especially in the US (in excess of 10L). Morbidity is related to high aspiration volume and high lidocaine dosage.

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Skin grafting and burns reconstructive surgery

Procedure	Free skin grafts applied to surgically created raw surfaces following debridement, or to granulating wounds
Time	Variable 30min to 2h
Pain	++/+++ (especially the donor site)
Position	Variable. Depends on the area to be grafted. Usually supine
Blood loss	Nil for simple grafts. Extensive debridement and grafting of burns may require 6–8 units
Practical techniques	GA/LMA and SV (with lateral cutaneous nerve of the thigh or fascia iliaca block if thigh donor site). Spinal for lower limb surgery

Preoperative

Patients for simple excision and grafting of isolated lesions may be otherwise well.

- Elderly patients for excision/grafting of skin lesions or pretibial lacerations may be in poor general health. A local or regional technique may be preferable to GA. Sedation may be necessary for long procedures or in those unable to lie still.
- Patients with extensive burns for debridement and grafting require careful assessment.

Perioperative

Full-thickness skin graft

Consists of the epidermis and dermis. Used in small areas where the thickness, appearance and texture of the skin are important. Usually harvested with a scalpel. FTSG can be harvested using SC LA infiltration with a 27G needle. The donor site needs to be closed directly. Common sites include:

- Post-auricular skin for grafts to the face
- Groin or antecubital fossa to the hand for management of flexion contractures.

Split skin graft

Consists of the epidermis and a variable portion of the dermis. Much wider usage than FTSG. Usually harvested with a skin graft knife or a power-driven dermatome. Donor sites will heal spontaneously within 2w. Donor sites are chosen according to the amount of skin required, the colour and texture match and local convenience. Meshing is used to expand the extent of the area that the graft is required to cover. Common donor sites are the thigh, the flexor aspect of the forearm, the upper arm and the abdomen. SSG can be harvested using LA cream. It should be applied at least 2h in advance and covered with an occlusive dressing. Anaesthesia does not extend into the deeper dermis, so the technique is unsuitable for FTSG. The lateral cutaneous nerve of the thigh (LCNT) or fascia iliaca block provides useful analgesia of a thigh donor site. If regional block is not possible, then surgical infiltration of LA will be useful. Excess harvested skin can be stored at 4°C for 2–3w.

Postoperative

The SSG donor site is a painful wound. Supplement with LA (LCNT or femoral block) where possible. The type of dressing is important for donor site healing and comfort. Common dressings used are alginate dressings or an adhesive retention tape (such as Mefix®). Dressings are removed once the donor site has healed in 2–3w. NSAIDs and simple analgesics are usually required for 3–4d. Itching follows when the acute pain settles and healing is under way.

Acute burns surgery

(See also ➲ pp. 1013–18.) Extensive debridement and grafting of burns are major procedures. Current management is to aim to debride burnt tissue and cover at the earliest opportunity (often within 48h). Debrided areas may be covered with autograft (SSG or FTSG taken from healthy skin on the patient), allograft (skin from cadaveric donors) or a range of complex burns dressings. This converts the burn to a healthy surgical wound. Potential sources of sepsis are eradicated; fluid shifts are less, and intensive care management tends to be more stable. Wounds not covered with autograft will require further surgery in the following weeks to cover the wound fully with the patient's own skin. This process may take many weeks of repeated procedures.

- Two anaesthetists may be required. Two surgical teams will considerably speed up the procedure and help minimise complications.
- Ensure 6–8 units of red cells are X-matched. Debrided tissue bleeds freely. Losses can be rapid and difficult to estimate, particularly in small children. Regularly check Hct, and maintain at ~30%. Correct coagulopathy. Massive transfusion may be required.
- Perform a careful airway assessment: intubation is required for all but the smallest of burn debridements. Face mask ventilation may be made difficult by facial burns and the presence of feeding tubes. Laryngoscopy may be compromised by upper airway oedema due to burns and resuscitation. Securing the airway by tape or ties may not be possible with facial burns; suturing or even wiring the tube may be necessary.
- Use lung-protective ventilation: ventilation requirements will be high due to the high metabolic rate. Hypercapnia may have to be tolerated.
- Significant improvements in blood loss and patient comfort are achieved with surgical infiltration of tumescent solution containing adrenaline and LA (e.g. bupivacaine) into the tissues being debrided and used as donor sites. Care should be taken to not exceed toxic LA doses, although large doses of adrenaline can be infiltrated. Reduction in vasopressor requirements and lactic acidosis may be seen.
- Temperature control: a large exposed body surface area will lose heat rapidly by radiation and evaporation. Measure core temperature and use all methods available for heat conservation. This should include active warming of fluids, use of an HME filter, underpatient warmer, forced air warmer and attention to minimising patient exposure. Ongoing discussion with the operating team can ensure that only areas currently being operated on remain exposed; other areas can be covered with sterile insulation (e.g. Gamgee tissue). Little body surface area may be available for warming blankets; however, these should be utilised. The ambient temperature in the operating theatre will often

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need to be kept at a temperature that is uncomfortable for staff (above 30°C is common). If patient core temperature is being maintained, reducing operating room temperature first will be well received.

- Monitoring: pragmatism towards patient monitoring is required; not all monitoring usually essential for large procedures may be possible. Placement of non-invasive monitoring devices may be difficult. An arterial line facilitates measurement of BP and blood sampling. A central venous line is valuable to provide reliable venous access for this and future procedures, and helps in the management of intravascular volume. A urinary catheter is essential.
- Access: maintain strict asepsis during line insertion. Cannulae may need to be stitched. Try to place through intact skin. This may not always be possible; discuss with the surgical team for other options. Good sites for vascular access may be in short supply and these should only be used when absolutely necessary. Lines may be easily dislodged. Attentiveness by the anaesthetist may avert unplanned removal of lines by the surgical team.
- Suxamethonium is contraindicated, except in the first 48h following burn. Massive K^+ release may cause cardiac arrest. NDMR dosing and frequency requirements are ↑, due to hypermetabolism, ↑ protein binding to α-1 acid glycoprotein and alterations in acetylcholine receptors at the neuromuscular junction.
- Postoperative care: return to the burns unit. Large body surface area burns (e.g. >40%) or those with additional injury (e.g. smoke inhalation) may need continued ventilation on ICU until warm and stable.
- Analgesia is best provided by IV opioids. Requirements can be substantial. Suggest early intervention of the acute pain team. Good pain management is important to enable early mobilisation, which is critical to minimise contracture formation and maintain function.
- Dressing changes can often be facilitated humanely on the ward with experienced nursing staff. Anaesthetic support may be required to provide sedation and analgesia. Entonox® or ketamine/midazolam sedation are useful, but occasionally further agents are required. Consideration should be given to the limitations of providing care in a remote environment.
- Antibiotics and early nutrition are important to increase survival. NGT or nasojejunal tube are often required to provide or supplement nutrition.

Chronic burns surgery

Patients often require ongoing surgery for many years after initial recovery from an acute burn. This is usually for scar revision and contracture release. These patients can present with unique challenges, depending on the severity and location of their initial injury and how they have healed:

- Airway: scarring following face and neck burns can result in significant limitations in mouth opening and neck movement, which will not improve after GA and paralysis. Laryngoscopy may be impossible. AFOI must be considered. An LMA may be a useful rescue device, even in an anatomically challenging airway. Previous airway management should be reviewed, but progression of scarring may render this obsolete.

- Access: scarring over conventional venous and arterial access sites makes cannulation difficult. If it is away from the surgical field, these are often still viable with ultrasound.
- Positioning: contractures may prevent conventional positioning on the operating table. Careful attention to padding pressure areas and supporting the limbs and head will reduce risk of further injury.
- Psychological: patients have often had a prolonged and traumatic stay in hospital following their initial injury, with multiple trips to the operating theatre. Post-traumatic stress disorder is common. Ask for their input on what has worked well previously, and offer a premed.
- Pain: can be significant and poorly tolerated in this patient group. Consider regional anaesthesia to supplement GA if a block will cover the surgical site. Have a low threshold for involving the acute pain team.
- Early mobilisation and engagement with the multidisciplinary team remain critical for scar management, and effective analgesia will allow this.

Further reading

Bittner EA, Shank E, Woodson L, Martyn JAJ (2015). Acute and perioperative care of the burn-injured patient. *Anaesthesiology*, **122**, 448–64.

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Chapter 25

Gastrointestinal surgery

Matt Rucklidge and **Peter Garnett**

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Major GI surgery

General considerations

Major abdominal surgery generates a neuroendocrine, metabolic and inflammatory response which may result in adverse physiological changes, including pulmonary dysfunction, ↑ cardiac demand, pain, nausea and vomiting. This may result in delayed mobilisation, prolonged hospital stays and ↑ morbidity and mortality. Principles of prehabilitation and ERAS should be applied (see ➲ pp. 44–6). Major GI surgery may be performed by open and minimally invasive (laparoscopic and robotic) techniques.

Preoperative preparation

- History, examination, ECG if indicated, FBC, U&E. Other blood tests, as indicated by patient comorbidities.
- Assessment of exercise function (e.g. CPET; see ➲ pp. 33–5). Optimise cardiac and respiratory function.
- Optimise nutrition. Patients with inflammatory bowel disease may be significantly malnourished and immunosuppressed.
- Discuss multimodal analgesia. Key components for GI surgery include: abdominal wall blocks and catheters (see ➲ p. 1168), wound catheters, IV lidocaine infusions (see ➲ p. 1171) and neuraxial blocks (see ➲ pp. 1114–17).
- Withhold ACE inhibitors/ARBs 24h before surgery. The VISION study found reduced risk of mortality, CVE and myocardial injury if withheld.[1]
- Consider premedication with H_2 antagonist or PPI if at risk of regurgitation, and discuss RSI if indicated.
- Determine whether postoperative HDU/ICU care is indicated and ensure a bed is booked before surgery.
- Balance the health of the patient with the complexity and duration of the surgical procedure, and consider the additional information invasive monitoring will provide against the risks involved in placement and interpretation (Table 25.1).
- Minimise the period of fasting. Continue clear fluids until 2h prior to surgery and administer oral carbohydrate preload (caution in diabetic patients).

Perioperative

- Large-bore IV access, with long extension if access to arms restricted. Arms are commonly placed by the sides in laparoscopic colorectal surgery, limiting access intraoperatively.
- Perform neuraxial block for postoperative analgesia if indicated, e.g. spinal with long-acting opioid (e.g. diamorphine or morphine) or low thoracic epidural.
- Institute invasive monitoring as indicated.
- Consider appropriate line placement for postoperative parenteral feeding. This may be required in patients undergoing procedures for inflammatory bowel disease.
- RSI if evidence of bowel obstruction or risk of regurgitation.
- Prophylactic antibiotics before skin incision.
- Consider the need for perioperative steroid supplementation. Steroid therapy is common in patients with inflammatory bowel disease.

- Avoid prolonged exposure during preparation for surgery, and establish active patient warming as soon as possible (fluid warmer, hot-air blanket, warming mattress). Monitor central temperature and aim for normothermia.
- Avoid hypotension. There are strong associations between intraoperative hypotension and adverse outcomes, including myocardial injury and death. Maintaining systolic BP >100mmHg may reduce risk.[2]
- PONV is common after GI surgery. Reduce risk by adequate hydration, multimodal analgesia to avoid or limit opioids and administration of different classes of antiemetic.
- Procedures may be prolonged; pay special attention to protecting pressure areas.
- Be prepared for lithotomy or the Lloyd-Davies position (see ➔ pp. 436–7) and a need for steep Trendelenburg (head-down) that may impair CVS and respiratory function.
- Use a moderately liberal (1–2L positive) or goal-directed approach to fluid management. A restrictive fluid regimen is not associated with improved disability-free survival, compared to a liberal regimen, but is associated with a higher rate of AKI.[3]

Postoperative

- Aim for the patient to drink, eat and mobilise in the immediate postoperative period.
- Prescribe overnight O_2, and continue as required, to maintain SpO_2 >95%. Supplemental O_2 should be prescribed if using an opioid-based analgesic technique.
- Treat nausea and vomiting aggressively.
- Monitor fluid balance closely. Consider ongoing losses from abdominal drains, ileostomy and NG aspirate. Measure urine output hourly for at least 24h following major surgery.
- Arrange a CXR if CVP line sited.
- Continue epidural or abdominal wall catheter LA administration if sited.
- Prescribe regular simple analgesia, e.g. paracetamol and NSAIDs if not contraindicated. Be aware that enteral absorption may be impaired in some patients after abdominal surgery. Other agents, including clonidine, gabapentinoids and ketamine, may be beneficial and reduce postoperative opioid use and opioid side effects, including gut dysfunction.
- Refer to acute pain team for postoperative review.
- Worsening postoperative pain may indicate a complication of surgery.
- Consider daily FBC/U&E until return of normal bowel function.

Table 25.1 Suggested indications for additional monitoring

Minimally invasive CO monitoring	Major abdominal surgery with potential fluid shifts, CVS compromise, likely requirement for perioperative inotropes
Arterial line	CVS, respiratory compromise, major blood loss, need for blood gas sampling
CVP line	Need for vasopressors and/or inotropes. Requirement for postoperative parenteral nutrition

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Open GI surgery

Despite the increasing range and complexity of abdominal procedures performed laparoscopically, open techniques remain common and are listed on ➲ p. 672. Open GI surgery is associated with ↑ pain and tissue trauma, and may impair postoperative respiratory and GI function, compared with minimally invasive surgical techniques. Some open abdominal procedures may require the patient to be turned prone intraoperatively (e.g. abdominoperineal resection).

Summary of open GI procedures

(See Table 25.2.)

Table 25.2 General surgical open procedures

Operation	Time (h)	Position	Blood loss (L)	Notes and analgesia
Gastrectomy: Resection of the stomach	2–3	Supine	0.5–1	Consider neuraxial analgesia (epidural or spinal); consider CVP and arterial line
Pancreatectomy: Resection of the pancreas Commonly performed as part of a Whipple procedure (pancreaticoduodenectomy)	3–6	Supine	0.5–2	Often long and complex surgery Consider neuraxial analgesia (epidural or spinal); consider CVP and arterial line
Cholecystectomy: Resection of the gall bladder	1	Supine	0.5	Right upper quadrant incision. Transversus abdominis plane (TAP) block or wound catheter ± PCA
Appendicectomy: Resection of the appendix	1	Supine	Min	Right lower quadrant incision; consider TAP block ± PCA
Right hemicolectomy: Resection of the right colon	1–3	Supine	0.5–1	Consider neuraxial analgesia (epidural or spinal) or rectus sheath catheters ± PCA
Left hemicolectomy: Resection of the left colon	1–3	Lloyd-Davies	0.5–1	Consider neuraxial analgesia (epidural or spinal) or rectus sheath catheters ± PCA

Operation	Time (h)	Position	Blood loss (L)	Notes and analgesia
Anterior resection: Resection of the rectum	2–3	Lloyd-Davies	0.5–1.5	Consider neuraxial analgesia (epidural or spinal) or rectus sheath catheters ± PCA
Abdominoperineal resection: Resection of the rectum and anus	2–4	Lloyd Davies. Possible transition to prone intraoperatively	0.5–2	Consider neuraxial analgesia (epidural or spinal) or rectus sheath catheters ± PCA
Hartmann's procedure: Resection of the sigmoid colon with colostomy	1–3	Lloyd-Davies	0.5–1	Consider neuraxial analgesia (epidural or spinal) or rectus sheath catheters ± PCA
Reversal of Hartmann's: Laparotomy with bowel ends reanastomosed	1–2	Lloyd-Davies	0.5–1.5	Consider neuraxial analgesia (epidural or spinal) or rectus sheath catheters ± PCA
Closure of loop colostomy or loop ileostomy: Reversal of ileostomy via stoma site	0.5–1	Supine	Min	Minor procedure but generally requires muscle relaxation. Rarely may convert to laparotomy. May need abdominal wall block ± PCA

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Laparoscopic GI surgery

Laparoscopic surgery is well established for an increasing range and complexity of elective and emergency procedures. The principles of laparoscopic surgery can also be applied to robotically assisted GI procedures. Laparoscopic and robotic techniques can both be considered minimally invasive surgery. Minimally invasive surgery may have significant intraoperative effects on cardiopulmonary physiology because of the effects of the pneumoperitoneum and patient positioning (see Table 25.3). However, benefits of laparoscopy over laparotomy include:

- Reduced tissue trauma, wound size and postoperative pain
- Improved postoperative respiratory function
- Reduced postoperative ileus
- Earlier mobilisation, shorter hospital stays
- Improved cosmetic results.

Surgical requirements

- Creation of a pneumoperitoneum by insufflation of gas (usually CO_2) into the peritoneal cavity.
- CO_2 is non-combustible, colourless, non-toxic and highly soluble, and is continuously insufflated into the abdomen to maintain a pressure of 10–20mmHg.
- Devices that warm and humidify CO_2 for laparoscopic abdominal procedures are in use, but their effect on clinical outcomes is unclear.
- Adequate NMB is required in laparoscopy to optimise operating conditions and limit intra-abdominal pressure.
- Muscle relaxation is essential while docked in robotic surgery to avoid unexpected patient movement and injury.

Patient positioning

- Patient position will be determined by the type of surgery (see p. 678).
- Patients placed head-down are at greater risk of pulmonary impairment, including reduction in FRC, V/Q mismatch and atelectasis. There is an ↑ risk of endobronchial intubation due to cephalad movement of the lungs exacerbated by the pneumoperitoneum.
- Long periods positioned steeply head-down can result in facial, airway and cerebral oedema. This must be considered when planning extubation.
- Regurgitation of gastric contents in the head-down position can result in conjunctival chemical injury. Ensure eyes are protected.
- Patients placed head-up are at ↑ risk of reduced BP and CO due to ↓ venous return. Those most at risk include the hypovolaemic patient, the elderly and patients with pre-existing CVS disease.
- Patients are at risk of injury from slipping in both head-up and head-down positions. Consider use of specialised poisoning devices, e.g. non-slip padding, shoulder supports.

Effects of gas insufflation

- The physiological effects of pneumoperitoneum are summarised in Table 25.3.
- Stretching of the peritoneum may cause vagal stimulation, resulting in bradycardia and occasionally asystole. Treat with vagolytics, e.g. atropine, glycopyrronium, and ensure the surgeon ceases insufflation and releases the pneumoperitoneum.
- Gas insufflation may result in sympathetic response, leading to hypertension and tachycardia.
- CO_2 is readily absorbed from the peritoneum and may cause hypercapnia and acidosis.
- Extraperitoneal gas insufflation may occur through a misplaced trocar or insufflation needle, via an anatomical defect (e.g. between the pleura and the peritoneum) or when gas under pressure within the abdomen dissects through tissue planes. This may result in SC emphysema, pneumomediastinum, pneumopericardium or pneumothorax.
- Venous gas embolism may rarely occur when gas is inadvertently insufflated directly into a blood vessel. Physiological effects are less with CO_2 than air due to its greater plasma solubility; however, a significant embolism may be fatal.

Table 25.3 Physiological effects of pneumoperitoneum

Respiratory		CVS	
Airway pressure	↑	Venous return	↓
FRC	↓	SVR	↑
Pulmonary compliance	↓	CO	↔ ↓
V/Q mismatch	↑	Risk of arrhythmias	↑
Neurological		**GI**	
ICP	↔ ↑	Risk of regurgitation	↑
CPP	↔ ↑		

Trauma associated with abdominal access

- Accessing the abdominal cavity by trocar or insufflation (Veress needle) may cause damage to organs (e.g. spleen, bladder, liver, bowel, stomach). Organ damage may not always be apparent at the time of injury.
- Damage to blood vessels may result in massive haemorrhage, necessitating rapid conversion to an open procedure.

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Preoperative

- Contraindications to laparoscopic surgery are relative; risks are ↑ with IHD, valvular heart disease, ↑ ICP and hypovolaemia.
- All patients must be considered at risk of conversion to an open procedure, and a plan for analgesia considered.
- Laparoscopic procedures are increasingly performed in obese patients due to improved postoperative recovery when compared to an open procedure. However, the deleterious effects of the pneumoperitoneum and positioning may be exaggerated in this patient group.

Perioperative

- GA with endotracheal intubation, muscle relaxation and controlled ventilation is considered the safest technique, as it protects against pulmonary aspiration and enables control of $PaCO_2$.
- Avoid gastric distension during bag–mask ventilation which may increase the risk of gastric injury during trocar insertion. Consider inserting a gastric tube if laparoscopic entry via the left upper abdomen (Palmer's point) is planned.
- A urinary catheter may be required in lower abdominal procedures to decompress the bladder and reduce the risk of injury.
- Systemic absorption of CO_2 and raised intra-abdominal pressure will require ↑ minute volume and result in higher intrathoracic pressure.
- Aim for normocapnia, but beware of adverse effects of high intrathoracic pressure. Controlling $ETCO_2$ during prolonged procedures, especially in the obese and head-down position, can be difficult and may occasionally necessitate intermittent release of intraperitoneal gas or tolerance of a degree of hypercapnia.
- If high inspiratory pressures are encountered, exclude endobronchial intubation and inadequate NMB. Consider a change to pressure-controlled ventilation and a reduced I:E ratio (e.g. 1:1).
- Analgesia requirement is dictated by the procedure (see ➔ p. 678). Pain may be intense intraoperatively, but postoperative pain is generally much less than for open procedures. Perioperative IV lidocaine infusion may reduce postoperative pain and bowel dysfunction following laparoscopic surgery.
- Avoid hypovolaemia as this exaggerates the deleterious CVS effects of laparoscopy.
- Laparoscopic surgery is associated with a high incidence of nausea and vomiting. Administer prophylactic antiemetics and prescribe postoperatively.
- Invasive arterial BP and CVP monitoring may be required for extensive procedures or for patients with CVS or respiratory compromise.

Causes of hypoxia

- Hypoventilation: inadequate ventilation due to pneumoperitoneum and head-down positioning.
- Reduced CO: IVC compression, arrhythmias, haemorrhage, myocardial depression, venous gas embolism, extraperitoneal gas.
- V/Q mismatch: reduced FRC, atelectasis, endobronchial intubation, venous gas embolism, pulmonary aspiration and rarely pneumothorax.

Postoperative

- At the end of the procedure, encourage the surgeon to expel as much intraperitoneal gas as possible to reduce postoperative pain.
- LA infiltration of port sites and intraperitoneal administration of LA may reduce postoperative analgesia requirements.
- Pain varies and is often worst in the first few hours. Shoulder tip pain due to diaphragmatic irritation may be troublesome but is usually short-lived. Significant pain extending beyond the 1st day raises the possibility of intra-abdominal complications.

Special considerations

- An SGA may be used for some laparoscopic procedures. This should be avoided if the patient has a history of reflux or obesity and the procedure is anticipated to be difficult or prolonged, especially in the head-down position.
- Regional anaesthesia is not generally used as the sole anaesthetic technique because of the high level of block required to cover the pneumoperitoneum.

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Summary of laparoscopic GI procedures

(See Table 25.4.)

Table 25.4 General surgical laparoscopic procedures

Operation	Time (h)	Position	Blood loss (L)	Notes and analgesia
Gastrectomy: Resection of the stomach	2–3	Lloyd-Davies with head up	0.5	Consider CVP and arterial line
Pancreatectomy: Resection of the pancreas Commonly performed as part of a Whipple procedure (pancreatico-duodenectomy)	4–8	Lloyd-Davies with head up	0.5–1	Often long and complex surgery Consider CVP and arterial line Consider neuraxial analgesia Up to 20% risk of open conversion
Cholecystectomy: Resection of the gall bladder	1	Supine with head up and left tilt	Min	Consider gastric tube to deflate stomach for port insertion
Appendicectomy: Resection of the appendix	1	Supine with head down and left tilt	Min	Consider TAP block
Right hemicolectomy: Resection of the right colon	1–3	Supine with variable head up/down and left/right tilt during procedure	0.5–1	Consider neuraxial analgesia or PCA
Left hemicolectomy: Resection of the left colon	1–3	Lloyd-Davies with variable head up/down and left/right tilt during procedure	0.5–1	Consider neuraxial analgesia or PCA
Anterior resection: Resection of the rectum	2–4	Lloyd-Davies with variable head up/down and left/right tilt during procedure	0.5–1	Consider neuraxial analgesia. Be aware of time spent in extreme positioning

Operation	Time (h)	Position	Blood loss (L)	Notes and analgesia
AP resection: Resection of the rectum and anus	3–5	Lloyd-Davies with variable head up/down and left/right tilt during procedure Possible transition to prone intra-operatively	0.5–1	Consider neuraxial analgesia. Be aware of time spent in extreme positioning
Hartmann's procedure: Resection of the sigmoid colon with colostomy	1–3	Lloyd-Davies with variable head up/down and left/right tilt during procedure	0.5–1	Consider neuraxial analgesia or PCA. Be aware of time spent in extreme positioning
Reversal of Hartmann's: Removal of end-colostomy with bowel ends reanastomosed	1–2	Lloyd-Davies with variable head up/down and left/right tilt during procedure	0.5–1	Consider neuraxial analgesia or PCA. Significant chance of conversion to laparotomy

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Laparoscopic cholecystectomy

Procedure	Laparoscopic removal of gall bladder
Time	40–80min
Pain	++
Position	Supine, 15–20° head-up, slight left tilt
Blood loss	Not significant
Practical techniques	GA, ETT, IPPV

Preoperative

- Patients are classically, though not always, '♀, forty, fair, fat and fertile'.
- Gallstone-associated pancreatitis may make surgery more difficult and increase the risk of conversion to an open procedure.
- With appropriate patient selection and perioperative techniques, the procedure can be performed as a day case.

Perioperative

- Avoid gastric distension during bag–mask ventilation which may increase the risk of gastric injury during trocar insertion. Consider inserting a gastric tube prior to abdominal access.
- Ensure adequate IV access; haemodynamic changes may be profound and there is potential for sudden blood loss.
- The combination of pneumoperitoneum and obesity may make ventilation difficult.
- High risk for PONV. Administer prophylactic antiemetics.
- Short-acting opioids (alfentanil or fentanyl) may counter the haemodynamic fluctuations and limit postoperative opioid-related side effects.
- Ask the surgeon to infiltrate the port sites with LA.
- About 5% of cases require conversion to an open procedure.

Postoperative

- Prescribe regular simple analgesics, opioid as required (PRN), antiemetic and IV fluids until tolerating oral fluids.

Special considerations

- This can be a very stimulating procedure, particularly during diathermy around the liver.
- LA applied to the gall bladder bed may reduce postoperative analgesic requirements (e.g. 20mL of 0.25% bupivacaine).
- If conversion to open cholecystectomy, pain can be significant and a PCA may be required. LA should be infiltrated by surgeons, and a wound catheter or a subcostal TAP block may be beneficial.

Laparoscopic appendicectomy

Procedure	Resection of appendix
Time	20–40min
Pain	++
Position	Supine
Blood loss	Not significant
Practical techniques	RSI, ETT, IPPV, TAP block

Preoperative

- Patients are often young and fit unless appendix ruptured, in which case they may be septic and unwell.
- In the unwell patient, resuscitate preoperatively and administer timely antibiotics.
- Conversion to open appendicectomy is more likely if the appendix is ruptured.
- Occasionally presents in the elderly. May be the presenting condition of caecal adenocarcinoma requiring subsequent right hemicolectomy.

Perioperative

- Prophylactic antibiotics.
- RSI. Patients may be unfasted or at risk of aspiration due to delayed stomach emptying 2° to intra-abdominal pathology.
- Consider NSAID and paracetamol IV.
- Encourage the surgeon to infiltrate LA or perform a right-sided TAP block.
- Extubate awake (consider left lateral position).

Postoperative

- Prescribe regular simple analgesics and opioid PRN, antiemetics and IV fluids until tolerating oral fluids.

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Inguinal hernia repair

Procedure	Repair of inguinal muscular canal defect through which bowel may protrude
Time	30–60min
Pain	++
Position	Supine
Blood loss	Not significant
Practical techniques	GA, SV, SGA, inguinal field block. Spinal. Local infiltration and/or sedation

Preoperative

- Patients are usually older adult ♂ or young children.
- Can usually be performed as a day case procedure.

Perioperative

- Inguinal LA field block may be used as a sole technique for surgery or to complement GA.
- The iliohypogastric and ilioinguinal nerves are easily blocked, 2cm caudal and medial to the anterior superior iliac spine (ASIS).
- The genitofemoral nerve is located 1–2cm above the midpoint of the inguinal ligament, deep to the aponeurosis of the external oblique. This may be left to the surgeon to block, reducing the risk of vascular or peritoneal puncture.

Special considerations

- Inguinal hernia repair may be performed laparoscopically.
- Patients who are unfit and/or elderly may benefit from hernia repair under LA. However, some hernias may prove difficult to repair under LA and a spinal anaesthetic or GA may be preferable to avoid intraoperative GA conversion. Confirm with the surgeon the suitability for LA repair.
- A low-dose propofol infusion may be a useful adjunct in cases performed under LA.
- Other hernia repairs are commonly performed. It is important to establish the type of hernia repair and anaesthetic requirements. An incisional hernia repair may vary from a minor repair of a laparoscopic port site hernia to a complex repair of a large anterior abdominal wall defect.

Anal and perianal procedures

Procedure	Haemorrhoidectomy, fissure, sphincter repair, perianal abscess, pilonidal sinus
Time	20–30min
Pain	+++
Position	Supine, lithotomy, head-down, occasionally prone
Blood loss	Not significant
Practical techniques	GA, SV, SGA. Spinal ('saddle block')

Preoperative

- Assess suitability for SGA/lithotomy/head-down position.
- Consider ETT if the patient is obese or at risk of aspiration.

Perioperative

- Often intensely stimulating procedures, and laryngospasm is common. Maintain deep anaesthesia and consider short-acting opioids (fentanyl or alfentanil) and/or N_2O.
- Potential for bradycardia or asystole due to increase in vagal tone. Anticipate and have vagolytic to hand.
- LA infiltration by the surgeon during the procedure often provides effective pain relief.

Postoperative

- Avoid PR route of drug administration.
- Prescribe regular simple analgesics and opioid PRN.
- Consider aperients for discharge, particularly if opioids used.

Special considerations

- Surgery for pilonidal sinus may require prone positioning. Confirm with surgeon preoperatively and prepare patient accordingly.
- Avoid spinal anaesthesia followed by immediate head-down tilt.
- A sacral-only spinal block ('saddle block') using heavy bupivacaine is a useful alternative, with little effect on CVS dynamics.

References

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Chapter 26

Bariatric surgery

Nicholas Kennedy and **Katherine Reeve**

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Introduction

Bariatric surgery describes a variety of procedures that are performed on people with obesity. Weight loss is achieved by reducing the size of the stomach with gastric banding, removal of part of the stomach or rerouting part of the small intestine.

When anaesthetising patients for bariatric surgery, perioperative factors discussed in ➔ Chapter 2 should be considered.

Bariatric surgery should occur in a multidisciplinary team setting with rigorous preoperative assessment, intraoperative pathways and consideration for postoperative location and management.

Indications for surgery

NICE guidelines 2014

Bariatric surgery is a treatment option for people with obesity if all of the following criteria are fulfilled:[1]

- They have a BMI of ≥40kg/m² or between 35kg/m² and 40kg/m² and other significant disease (e.g. type 2 diabetes or high BP) that could be improved if they lose weight.
- All appropriate non-surgical measures have been tried, but the person has not achieved or maintained adequate, clinically beneficial weight loss.
- The person has been receiving or will receive intensive management in a tier 3 service.
- The person is generally fit for anaesthesia and surgery.
- The person commits to the need for long-term follow-up.

In addition, in 2014, NICE brought out specific points relating to bariatric surgery for people with recent-onset type 2 diabetes:

- Offer an expedited assessment for bariatric surgery to people with a BMI of ≥35kg/m² who have recent-onset type 2 diabetes, as long as they are also receiving or will receive assessment in a tier 3 service (or equivalent).
- Assessment for bariatric surgery should be considered in people with a BMI of 30–34.9kg/m² and/or people of Asian family origin at a lower BMI who have recent-onset type 2 diabetes.

References

1 National Institute for Health and Care Excellence (2014). *Obesity: identification, assessment and management*. Clinical guideline [CG189]. http://www.nice.org.uk/guidance/cg189

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Risk scoring in bariatric surgery

The Obesity Surgery Mortality Risk Score (OS-MRS) is a validated scoring system for risk stratification in bariatric surgery, and it aims to aid informed consent discussions, guide surgical decision-making and allow standardisation of outcome comparisons between treatment centres (Table 26.1).[2]

Table 26.1 Obesity Surgery Mortality Risk Score

Risk factor	Points
Age >45y	1
Hypertension	1
♂ sex	1
Risk factors for PE*	1
BMI ≥50kg/m²	1
Total:	

Risk group (score)	Postoperative mortality risk (death/total number of patients)
Class A (0 or 1 points)	0.2%
Class B (2 or 3 points)	1.1%
Class C (4 or 5 points)	2.4%

* Previous VTE, pulmonary hypertension, preoperative vena cava filter or hypoventilation due to obesity.

Reprinted from *Surgery for Obesity and Related Diseases*, **3**(2), DeMaria EJ *et al*. Obesity surgery mortality risk score: proposal for a clinically useful score to predict mortality risk in patients undergoing gastric bypass, 134–40, Copyright (2007), with permission from American Society for Bariatric Surgery. Published by Elsevier Inc.

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Intragastric balloon insertion/removal

Procedure	Placement of 700mL silicone balloon into stomach via gastroscope which is inflated with 700mL of 0.9% sodium chloride dyed with methylthioninium chloride (methylene blue). Balloons are removed after 6–12mo via gastroscopy
Time	15–30min
Pain	None
Position	Left lateral or sitting up
Blood loss	None
Practical techniques	IV sedation, topical anaesthesia or GA, ETT

Preoperative

Intragastric balloons are typically inserted in:

- Patients with BMI 25–35kg/m² as a weight loss adjunct in those who do not qualify for bariatric surgery. These patients are usually very low risk.
- Very high-BMI patients often >60–70kg/m², usually with many significant comorbidities, in whom invasive surgical procedures are deemed too risky.

Perioperative

- Topical anaesthesia is possible in cooperative patients.
- Low-risk patients are usually suitable for IV sedation, and often an anaesthetist is not required. Left lateral position is usual for insertion of the balloon. Balloon removal can be done in a similar fashion.
- IV sedation may be poorly tolerated and risky in high-risk patients due to hypoventilation, hypoxia and airway obstruction. A GA with intubation and ventilation may be indicated.
- Very large patients tolerate lying on their side very poorly and are better dealt with sitting up.

Special considerations

- Intragastric balloon insertion is associated with considerable nausea immediately postoperatively. Antiemetics should be given perioperatively and prescribed for the patient to take home. There is no nausea associated with balloon removal.
- Balloon removal can sometimes be surgically tricky and may take longer.
- Both insertion and removal of balloons can usually be done as day case procedures, even in high-risk patients.

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Gastric banding

Procedure	Placement of silicone adjustable band around the top of the stomach to create a small pouch above it. A small injection port is placed SC and connected to the band with tubing to allow the band to be inflated with 0.9% sodium chloride to control passage of food past it
Time	45–90min
Pain	+
Position	Supine, head-up
Blood loss	Minimal
Practical techniques	GA, ETT, IPPV (with PEEP)

Preoperative

- Gastric banding is a relatively straightforward laparoscopic procedure with a very low mortality rate.
- Gastric banding is commonly used for the lower-BMI and lower-risk bariatric surgical patients. However, some centres use gastric banding for most of their cases and some patients choose banding, so some very high-BMI and high-risk patients present.
- Preoperative analgesia with paracetamol is recommended.

Perioperative

- Ensure equipment (e.g. operating table, hover mattress) appropriate for weight and adequate staff numbers are available.
- Insert two IV cannulae.
- Take extreme care in positioning the patient to avoid damage due to pressure or overhanging tissue.
- Take precautions to ensure the patient does not slide down the table when head-up.
- Standard perioperative monitoring. Use forearm BP cuff if upper arm too large or wrong shape to place a cuff.
- Preoxygenate fully in head-up position.
- Intubation and ventilation are mandatory. Use V_T appropriate for IBW or lean body mass.
- Face mask ventilation can be difficult. Expect rapid desaturation during apnoea, and have a plan for airway management. RSI is not mandatory.
- Use short-acting anaesthetic agents such as sevoflurane. TIVA with propofol is a good technique, but correct dosing may be difficult.
- Good NMB is important.
- Antiemetics are important immediately postoperatively to prevent strain on the band sutures. Give two drugs perioperatively.
- Opioid analgesia is usually required postoperatively; usually fentanyl or morphine. Limit intraoperative opioid and titrate dosage upwards in recovery.
- Patients should be woken up and extubated sitting up. Plan for an electric bed.
- Ensure the surgeon infiltrates all port sites with LA.

Postoperative

- Ensure patients are nursed sitting up in recovery.
- Titrate opioids in recovery.
- Most patients can be safely managed without HDU, but this should be considered for patients with significant OSA.
- Encourage early mobilisation.
- Thromboprophylaxis as per local protocol.

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Gastric bypass

Procedure	Roux-en-Y gastric bypass. Almost always laparoscopic
Time	90–200min
Pain	++
Position	Supine, head-up
Blood loss	>500mL, occasionally more due to ooze from splenic injury or stomach. G&S required
Practical techniques	GA, ETT, IPPV (with PEEP)

Preoperative

- Gastric bypass involves a small bowel anastomosis, formation of a Roux limb, creation of a gastric pouch and a gastrojejunal anastomosis. Surgical techniques differ, and it is important to establish in what order the surgeon will do the procedure.
- Many surgeons ask for a large (typically 34Fr) bougie (or large NGT or dilator) to be passed orogastrically by the anaesthetist during pouch formation. This identifies the pouch and prevents stapling of the oesophagus. The bougie is then pushed distally into the Roux limb during gastrojejunal anastomosis to allow suturing around it. There are other techniques involving circular staplers, so ensure you understand what is used, how it works and when it is needed. Discuss with the surgeon preoperatively.

Perioperative

- Antiemetics immediately postoperatively to prevent strain on the anastomosis. Two agents are recommended perioperatively.

Postoperative

- Postoperative CPAP is quite safe. No evidence of damage to the gastric anastomosis.

Special considerations

- Sometimes surgeons ask for an NGT to be inserted to decompress the stomach prior to pouch formation. If so, insert it orogastrically, and **remove as soon as the stomach is decompressed**. Leaving an NGT *in situ* runs the risk of stapling it into the pouch, an avoidable disaster.
- Many surgeons test the gastrojejunal anastomosis for leaks by asking for an OGT to be passed into the pouch after the anastomosis is complete. Leak testing is achieved either by injecting air down the OGT and observing for bubbles in the fluid that has been added via the laparoscope, or by injecting ~60mL of dilute methylthioninium chloride (methylene blue) into the pouch. Leaks are usually obvious to see. Beware of the dyed fluid refluxing back into the mouth. Inserting a sucker into the mouth during this procedure helps prevent the dye from either being aspirated or refluxing out and onto the patient's face and hair! Consider cricoid pressure during this procedure.

- Postoperative complications to watch for:
  - Anastomotic leak: tachycardia (postoperative tachycardia is a leak until proven otherwise), excessive pain, pain on drinking.
  - In the event of a suspected leak, the best investigation is usually to relaparoscope the patient as soon as possible.
  - Bleeding: signs of severe bleeding similar to any other procedure. Staple line bleeding can present as melaena or haematemesis in the first 24h. Unless the patient is shocked, conservative management is warranted.

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Sleeve gastrectomy

Procedure	Stomach divided by stapling to reduce it to about 25% of its original size. A large portion of the stomach along the greater curvature is removed through a small incision. The result is a sleeve or tube-like structure. Almost always laparoscopic
Time	90–150min
Pain	++
Position	Supine, head-up
Blood loss	>500mL, occasionally more due to ooze from stomach staple line. G&S required
Practical techniques	GA, ETT, IPPV (with PEEP)

Preoperative

- Sleeve gastrectomy is becoming increasingly common as the weight loss procedure of choice.
- Often performed in high-risk patients, instead of a gastric bypass, as it is an easier and quicker procedure.
- Some surgeons ask for a large (typically 34Fr) bougie (or large NGT or dilator) to be passed orogastrically by the anaesthetist during the procedure. This allows the surgeon to staple alongside the bougie, identify the anatomy and prevent stapling of the oesophagus. Ensure you understand what is used, how it works and when it is needed. Discuss with the surgeon preoperatively.

Perioperative

- Antiemetics immediately postoperatively to prevent strain on the anastomosis. Two agents are recommended perioperatively.
- Avoid hypotension after stomach stapling. This helps identify staple line bleeding, reducing the risk of postoperative bleeding.

Postoperative

- Postoperative CPAP is safe.

Special considerations

- As per gastric bypass.

Chapter 27

Liver procedures

Alwyn Kotze and **Nilmini Manawaduge**

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ALGRAWANY

Anaesthetic management of acute oesophageal bleeding

Acute variceal haemorrhage is a medical emergency. Overall mortality in upper GI bleeding is 2–10%.[1] Anaesthetists will usually be involved if patients at risk of dying.

Treatment

Effective resuscitation and definitive therapy should be instituted simultaneously. Subsequent management targets normalisation of physiological parameters, alongside strategies to prevent rebleeding.[2]

Resuscitation

- Rapid assessment of Airway, Breathing and Circulation status.
- Supplementary O_2 PRN.
- Early airway control if indicated. Balance risk of airway loss against haemodynamic consequences of RSI.
- Insert large-bore IV cannulae × 2; consider CVP and arterial line.
- Use crystalloids for initial fluid resuscitation. No RCTs of fluid management in this situation, but balance essential between avoiding hypotension and avoiding exacerbation of bleeding.
- IV tranexamic acid reduces mortality and should be used early.[3]
- Urgent FBC, coagulation, point-of-care coagulation testing, U&E, LFTs, ECG.
- Urinary catheter if unstable.
- Keep nil by mouth.

Transfusion management

- Manage exsanguinating bleeding using massive haemorrhage protocol.[4]
- PRBCs: follow a restrictive strategy—transfuse if bleeding and haemodynamically unstable or if Hb <70–80g/L.
- FFP and/or cryoprecipitate indicated in patients with haemodynamic instability and coagulopathy (INR >1.5 times normal or fibrinogen level <1.5g/L, respectively).
- Platelets are indicated in patients who are actively bleeding and haemodynamically unstable, and have platelet count $<50 \times 10^9$/L.
- In patients on warfarin with active bleeding, reverse with PCC.

Stopping the bleeding

Medical management, along with endoscopic interventions:

- Stop NSAIDs.
- Consider stopping antiplatelet agents—discuss with appropriate specialty.
- Pharmacological therapy (terlipressin, somatostatin or somatostatin analogues), e.g. terlipressin 1–2mg IV 6-hourly (reduce if high risk for CAD).
- Prophylactic antibiotic therapy for 48h.
- No role for acid suppression drugs (e.g. H_2 blockers, PPIs) before endoscopy.
- Endoscopy alongside resuscitation if haemodynamically unstable, otherwise within 24h of bleeding. Band ligation or sclerosant injection will be used for variceal haemorrhage and adrenaline may be injected around ulcers.

Salvage interventions

- Interventional radiology may be indicated for patients who rebleed after endoscopic treatment if appropriate expertise is available. TIPSS is the treatment of choice.
- Balloon tamponade with an oesophageal and gastric balloon should be used only where endoscopic and drug treatments have failed. There is a high risk of fatal complications (aspiration, oesophageal tear/rupture and airway obstruction) and therefore, this should be used only in HDU/ICU.

Stabilisation and prevention of rebleeding

- Consider ICU/HDU admission in situations described above or according to usual physiological criteria.
- Prophylactic β-blockade (propranolol 40–160mg bd) reduces portal pressure and decreases rebleed rate from 70% to 50%.

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Anaesthesia for transjugular intrahepatic portosystemic shunt procedure

- TIPSS: percutaneous creation of a vascular connection between portal and systemic circulations, reducing portal venous pressure.[5,6]
- Indicated in treatment of portal hypertension and its complications (refractory variceal bleeding and diuretic-resistant ascites).
- A stent is placed radiologically between the hepatic and portal veins, allowing blood to bypass dilated oesophageal and gastric veins.
- May be elective (for ascites) or emergent (variceal bleeding).
- Anaesthesia requires good IV access and invasive arterial line monitoring to aid cardiovascular stability. Inotropes, vasopressors and blood components should be easily available.

Preoperative considerations

- Assess the hepatic functional impairment, haemodynamic stability, risk of aspiration, presence of heart failure and encephalopathy.[7]
- TIPSS is contraindicated if there is severe pulmonary hypertension, severe tricuspid regurgitation or severe heart failure.

Conduct of anaesthesia

- Usually undertaken in an angiography suite; hence, all anaesthetic issues related to remote-site anaesthesia apply.
- Prophylactic broad-spectrum antibiotics should be administered before the procedure and should be continued for 24h.
- Conscious sedation or GA are acceptable; if the patient is at high risk, it is safer to give GA with RSI and ETT.
- Either inhalational agents or TIVA technique using short-acting hypnotic agents, muscle relaxants and opioids are appropriate in order to ensure rapid post-procedure recovery.
- The internal jugular vein is cannulated to access the hepatic vein via a sheath. A cutting-edged catheter is introduced through the liver parenchyma into a branch of the portal vein under fluoroscopy with contrast. The tract is dilated and a metallic stent is placed.
- Intraoperative complications can include those related to accessing the internal jugular vein (or femoral, if this route is used). Procedure-specific complications include massive bleeding due to inadvertent hepatic artery puncture or hepatic capsular tear.
- TIPSS may precipitate acute cardiac failure, as the shunt leads to an ↑ venous return and preload. Post-procedural worsening of jaundice or encephalopathy may be seen in 20% of patients and 20% fail to control variceal bleeding, even after the procedure.[8]
- Post-procedure sepsis is possible.
- Patients are managed either in critical care or on gastroenterology/hepatology wards according to unit criteria (conscious level, haemodynamics, etc.).
- Evidence suggesting that early TIPSS in variceal bleeding improves outcome requires further study.[9,10,11]

Liver transplantation

Procedure	Transplantation of entire liver
Time	4–10h
Pain	Variable, but less than other comparable procedures (e.g. gastrectomy, thoracotomy). PCA; avoid NSAIDs
Position	Supine, one or both arms out
Blood loss	Extremely variable. 0–10 000mL, X-match 6 units and consider need for thawing other components before surgery. Cell salvage and point-of-care coagulation management vital. Use donated blood and components as necessary
Practical techniques	ETT, IPPV

Introduction

The majority of patients who present for liver transplantation have end-stage liver disease. The commonest indication worldwide is post-hepatitis C cirrhosis,[12] but alcohol-related cirrhosis and hepatic cancers are the commonest aetiologies in the UK.[13] Other conditions requiring transplantation include: 1° sclerosing cholangitis, viral hepatitis (B, C), polycystic liver disease, 1° biliary cirrhosis and metabolic liver disease.

Transplants are performed semi-electively as graft availability is the rate-limiting step. Patients with end-stage liver disease are ranked in order of priority according to a pre-agreed allocation system, then matched to the 1st suitable organ based on considerations including compatibility and graft size. A minority with fulminant or subacute liver failure are listed 'super-urgently' and receive priority over end-stage liver disease patients. In the UK, offers are allocated nationally based on the United Kingdom model for End-Stage Liver Disease (UKELD) and Transplant Benefit Score (TBS) that considers both risk of death without, and life expectancy gained after liver transplantation.[14]

Liver transplantation is performed under GA guided by invasive monitoring, with resuscitation facilities (including fluid and donated and/or salvaged blood components) and vasoactive drugs being readily available. Key anaesthetic objectives are to maintain normal systemic physiology and to stabilise vital organ function (including graft function) despite unique challenges at different surgical stages.

Preoperative

Potential recipients are assessed by a multidisciplinary team, including, as a minimum, a hepatologist, a transplant surgeon and an anaesthetist. Opinions are sought from others as required (intensivist, dietitian, psychologist, physiotherapist, substance misuse specialist and medical specialties) before listing. Individual decisions are made based on regularly updated national selection policies and risk scoring systems (e.g. UKELD/TBS in the UK or MELD in the US).[15,16] Preoperative assessment includes investigation/treatment of the following.

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Issues related to the liver disease

- Jaundice, hyponatraemia, ascites, pleural effusions, renal failure 2° to liver disease (hepatorenal syndrome), hepatopulmonary syndrome, portopulmonary syndromes (associated severe portal and pulmonary hypertension, leading to RV failure and potential cardiac arrest intraoperatively), coagulopathy (prolonged PT, low platelet count, fibrinolysis), varices (oesophageal, gastric, rectal, abdominal wall), hepatic encephalopathy, systemic vasodilation with hypotension and cardiac failure and poor nutritional state.
- Haemodynamic instability can result from cardiac consequences of the underlying pathology (e.g. alcoholic cardiomyopathy), from pericardial effusions and from circulatory failure due to vasodilation and low SVR. Anaemia resulting in low plasma viscosity further reduces effective tissue perfusion.

Comorbidity

- Smoking, DM (a common comorbidity with hepatitis C), IHD, pre-existing renal impairment of other aetiology.

Screening investigations

- As for any major abdominal surgery (FBC, U&E, clotting, ECG, CXR). Detailed abdominal imaging as requested by the surgeon. Resting echocardiography and spirometry.

Evaluation of physiological reserve is essential

- History and bedside assessment of frailty (e.g. Liver Frailty Index) for all patients. CPET and/or pharmacological/myoview stress echocardiography for specific indications.
- Preoperative fluids are not routinely administered, except in patients with renal impairment and hyperacute liver failure (glucose-based solutions).

Perioperative

- Venous ± arterial access before induction.
- Anaesthesia: intubate the trachea. Gastric emptying often delayed in end-stage liver disease; consider the need for RSI. Vasopressors may be required.
- Establish arterial, central venous and wide-bore access if not done preinduction. CO monitoring is often necessary to guide fluid and vasopressor administration. Pulse contour analysis or TOE are used and in patients with suspected pulmonary hypertension, PA catheterisation is considered.
- Broad-spectrum antibiotic prophylaxis, large-bore NGT and core temperature monitoring. Forced air warming.
- Ventilate to normocapnia, using O_2-enriched air and volatile agent (isoflurane, sevoflurane, desflurane). Establish infusion of an opioid agent (alfentanil, remifentanil, fentanyl). Paralysis is maintained with either intermittent boluses or infusions.
- Patients undergoing transplantation for fulminant liver failure are at risk of raised ICP. Controversy exists whether volatile agents worsen ICP, even where hypercapnia is avoided. Measures to reduce intracranial hypertension are advisable.

Surgical techniques vary but share common features.

Pre-anhepatic/dissection phase

Bilateral subcostal ('Mercedes')/reverse L subcostal incision. The liver, porta hepatis and surrounding structures are exposed, its anatomy defined and slings placed around the major vessels. Haemorrhage from dissection, varices and pre-existing coagulopathy is common. Most bleeding is venous and can be limited by judicious maintenance of low CVP, as for liver resection.

Anhepatic phase

Portal and hepatic veins divided. Explantation of native liver and IVC preparation for implantation. New liver inserted. Caval and portal anastomoses fashioned. Two main techniques are used for hepatectomy and implantation of the donor liver (Fig. 27.1):

- Cava-cavostomy: division of the hepatic veins with caval preservation, followed by a 'piggy back' implant where the new liver, with its own attached vena cava, is anastomosed cava-to-cava with the recipient's IVC either side-to-side or end-to-side. Surgery is usually performed with the native vena cava side-clamped, so that venous return is relatively preserved.
- Classical technique: liver explant with its included portion of the IVC. Anastomosis of the donor vena cava above and below the liver ('caval replacement'). Now less popular as it requires caval cross-clamping with reduced venous return and consequent refractory hypotension. Venovenous bypass is employed in some centres to facilitate venous return (femoral vein to right internal jugular or brachiocephalic vein). Venovenous bypass uses heparin-bonded extracorporeal circuitry; systemic anticoagulation is unnecessary.
- Anastomosis fashioned between donor and recipient portal vein.
- While anhepatic, patients with acute liver failure may become profoundly hypoglycaemic. This is less common in end-stage liver disease.
- Hypocalcaemia, lactic acidosis, coagulopathy and hypoglycaemia progressively occur during the anhepatic phase. Regular monitoring and prompt treatment are essential.

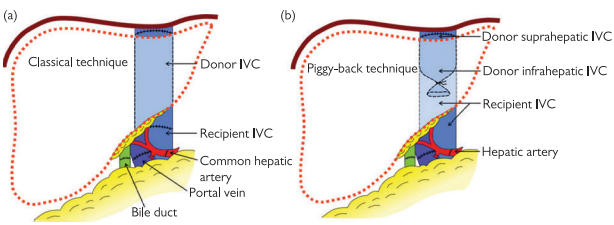

Fig. 27.1 (a) Classical technique; (b) 'Piggy back' technique of orthotopic liver transplantation. This image was published in *BJA Education*, **17**(1), Kashimutt S, Kotze, A. Anaesthesia for liver transplantation, 35–40, Copyright © The Author 2016.

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Neohepatic stage

Graft reperfusion, hepatic artery anastomosis and biliary reconstruction.

Reperfusion phase

- Begins with the re-establishment of blood flow through the liver (portal vein to vena cava). Reperfusion syndrome occurs, with cytokines release, complement activation and transient reduction in core temperature. Immediately after reperfusion, there is a rapid elevation in plasma K^+, as it is washed out of the previously ischaemic graft along with other products of hepatocyte breakdown. At the start of reperfusion, a bolus dose of 10mmol calcium chloride helps protect against the cardiac effects of sudden K^+ flux. Arrhythmias are common and cardiac arrest occasionally occurs. Profound hypotension is common unless the circulation is actively supported with appropriate fluid and vasoactive drug therapy (e.g. noradrenaline up to 2 micrograms/kg/min, adrenaline 10 micrograms or phenylephrine boluses of 500 micrograms to 1mg aliquots). As graft hepatocyte function returns, electrolyte gradients are restored. Hypotension at this stage results from myocardial depression and subsequently vasodilation. Myocardial depression usually resolves within 2 or 3min, but vasodilation may persist for several hours. In severely ill patients, an infusion of noradrenaline may subsequently be required. Some centres use a prophylactic vasopressin analogue, such as terlipressin, before reperfusion.
- The haemodynamic and biochemical mayhem of reperfusion resolves rapidly if the graft is functioning. Persisting acidosis or hypocalcaemia are suggestive of graft 1° non-function, which represents a transplantation emergency. This may necessitate urgent retransplantation. Early promising signs of graft function include a rise in body temperature due to ↑ metabolism, hyperglycaemia from enhanced gluconeogenesis, normalisation of coagulopathy, bile production and lactate clearance. Coagulopathy is managed with haemostatic agents (tranexamic acid/protamine) and blood or blood product transfusion guided by POCT (TEG®/ROTEM®/point-of-care INR/ABG).
- Following reperfusion, the hepatic artery is reanastomosed, and finally the bile duct reconstructed by direct duct-to-duct anastomosis/Roux-en-Y loop.
- Other considerations:
  - Induction immunosuppression (e.g. methylprednisolone 0.5–1g) is administered before graft reperfusion in some centres.
  - K^+ and Ca^{2+} should be monitored regularly during surgery and supplemented, when required, to maintain normal values. Some centres use Ca^{2+} infusion (3–5mmol/h) from the dissection phase.
  - There is no proven strategy for avoiding renal failure, other than optimising fluid balance and avoiding nephrotoxins. In patients at particularly high risk, avoidance of nephrotoxic immunosuppressants (such as ciclosporin, tacrolimus) in the early postoperative period may have a role.

Postoperative

- Patients should be managed in ICU. Early extubation is often feasible and further facilitates improvement of early graft blood flow due to negative intrathoracic pressure during SV, reduced ICU stay and ↓ incidence of nosocomial infections.
- Analgesia: PCA/epidural/paravertebral blocks have all been described. However, regional techniques are discouraged in many centres because of coagulopathy, instead relying more on regular systemic analgesics (opioid PCA, paracetamol) and occasionally wound catheters. Avoid NSAIDs (interaction with calcineurin inhibitors to induce renal failure).
- Postoperative fluids: maintenance fluid/NG feed at 1.5mL/kg/h.
- Bleeding postoperatively is relatively uncommon.
- Graft 1° non-function/initial poor function due to vascular issues (hepatic artery thrombosis, portal vein thrombosis) or the graft itself occurs in up to 5–10% of cases,[17] requiring retransplantation.
- Other postoperative problems include sepsis, AKI and acute rejection. These are managed medically with good results.
- Immunosuppression is usually started with standard triple therapy (steroid/mycophenolate/tacrolimus) and then tailored to the individual, guided by unit policy. Other drugs in current use include basiliximab, ciclosporin, azathioprine and sirolimus.
- Long-term results of liver transplantation are continually improving. One-year survival figures in major centres now run between 85% and 97% (risk-adjusted),[13] with a good long-term quality of life.

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Hepatic resection

Procedure	Resection of liver tissue
Time	2–6h
Pain	As for transplantation. Epidural more common
Position	Supine, arms out, reverse Trendelenburg position
Blood loss	Minimal to 900mL, X-match 5–10 units
Practical techniques	ETT, IPPV

The major indication for hepatic resection is metastatic colorectal adenocarcinoma, for which it improves 5y survival by 30%, compared to no intervention.[18] Other indications are benign and malignant 1° hepatobiliary tumours, donor hepatectomy for liver transplantation and occasionally liver trauma. The principles underlying anaesthesia are similar to those for any patient undergoing a major laparotomy.

- Major liver resection usually involves removal of 30–75% of functional hepatic tissue. Remaining hepatocytes function poorly for some days; short-acting drugs should be used.
- Increasingly, surgeons are using minimally invasive techniques.
- Laparoscopic liver resection decreases intraoperative bleeding, transfusion requirements and postoperative ascites.[19] It presents different anaesthetic challenges due to pneumoperitoneum, with consequent haemodynamic instability.
- Drugs that might compound postoperative hepatic encephalopathy or which rely on hepatic metabolism should be avoided, e.g. benzodiazepines.
- Most resections are accomplished with minimal blood loss, but catastrophic haemorrhage may occur.
- Resection commences with perihepatic dissection and identification of vascular anatomy.
- Intraoperative diagnostic ultrasound is often used to pinpoint lesions requiring resection.
- Bleeding occurs from either vascular inflow (portal vein, hepatic artery) or venous back bleeding. Branches of the hepatic artery and portal vein to the segment of the liver to be resected have usually been ligated, so inflow bleeding should not be a major problem. In practice, the line of resection often passes through a watershed area between vital and devitalised tissue. Maintaining a low CVP reduces venous back bleeding (often difficult in laparoscopic resections). Remaining inflow bleeding may require intermittent portal vein cross-clamping (the 'Pringle manoeuvre'). This results in a degree of ischaemia–reperfusion injury to the remaining liver tissue, and potentially poor postoperative liver function. This can be minimised by intermittent (rather than continuous) clamping. A brief period of vascular occlusion followed by reperfusion before sustained ischaemia ('preconditioning') may be beneficial.

- Resection of tumours located in critical sites of the liver and very radical liver resections are now possible. The liver is totally excised and dissected *ex vivo/ex situ* following perfusion with an ice-cold preservation solution. Healthy parts of the liver are then attached to the vena cava and reimplanted. This is a prolonged and difficult procedure and anaesthetically similar to a liver transplant. Venovenous bypass is sometimes required.[20]

Preoperative

- Most patients are otherwise relatively fit unless they have parenchymal liver disease.
- Those who have undergone neoadjuvant chemoradiotherapy are at higher risk as it can deplete functional cardiorespiratory reserves.
- The degree of planned resection plays a major role in modifying the anaesthetic technique as well as monitoring due to risk of bleeding and postoperative liver failure.[18]
- Preoperative assessment should thus be tailored. Apart from routine investigations for any major surgery, particular attention should be paid to liver function (including coagulation), Child–Pugh scoring and cardiorespiratory function.

Perioperative

- Large-bore venous access. Arterial line. CVP monitoring may be used, depending on bleeding risk and predicted need for vasoactive therapy. Active warming.
- Regular monitoring of blood glucose is essential as there is a risk of hypoglycaemia.
- Thoracic epidural analgesia is utilised to good effect postoperatively, though there is controversy (but little data) on the risks posed by postoperative coagulopathy.[21] Alternatives are intrathecal opioids, PCA and wound catheters (or a combination).
- Aim to preserve hepatic blood flow, thus minimising liver injury. Intubation and IPPV with a volatile agent are the commonest way of achieving this.
- Isoflurane and sevoflurane may enhance ischaemic preconditioning and help preserve hepatic function where the Pringle manoeuvre is used.

Fluid and haemodynamic management

- Maintaining a low CVP (<5cmH_2O) substantially reduces bleeding. This approach has dramatically reduced transfusion requirements, with no reported adverse consequences, despite the theoretically ↑ risk of an air embolus. A higher CVP target may be necessary to reduce the risk of renal ischaemia in patients at risk or in those with poor cardiac reserve.
- Techniques to lower the venous pressure include pre-resection fluid restriction, epidural boluses, head-up tilt and nitrate or diuretic infusions.
- Surgical manipulation of the liver can cause significant reductions in venous return with hypotension. Close communication between the anaesthetist and the surgeon is essential.

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- Early tranexamic acid decreases transfusion requirements.
- Blood components are required in massive haemorrhage or prolonged hepatic inflow obstruction or where very little hepatic tissue remains. Point-of-care coagulation testing allows rational treatment. However, intraoperative coagulopathy is relatively uncommon.
- Peak disturbances in clotting are seen on postoperative d2–3.

Postoperative

- Patients who have undergone >50% resection should initially be managed in an HDU for monitoring of liver function and to manage pain with either epidural or PCA.
- Postoperative liver insufficiency: coagulopathy or encephalopathy may develop in those who have undergone very major resections. This has practical implications for the timing of removal of epidural catheters, etc. which may require FFP cover.
- Postoperative renal dysfunction, hypoglycaemia, sepsis and intra-abdominal infections occur more commonly in extensive resections or high-risk patients.
- The overall results of radical hepatic resection are very encouraging, with many cases treated that were previously considered inoperable. Many remain disease-free 5y following resection. In those cases where recurrences arise, further hepatic resection is often possible.

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Chapter 28

Endocrine surgery

Pete Ford and Peter Valentine

Thyroidectomy *710*
Parathyroidectomy *714*
Phaeochromocytoma *716*
Carcinoid tumours *720*

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Thyroidectomy

Procedure	Removal of all or part of the thyroid gland
Time	1–2h, depending on complexity
Pain	+/++
Position	Shoulder bolster and head ring. Head-up tilt
Blood loss	Usually minimal. Potentially major if retro-sternal extension
Practical techniques	IPPV + reinforced ETT

General considerations

(See also ➔ pp. 223–5.)

- Complexity can vary from removal of a thyroid nodule to removal of a long-standing retrosternal goitre to relieve tracheal compression.
- Retrosternal goitre is usually excised through a standard incision, but occasionally a sternotomy is required.
- Recurrent laryngeal nerves and parathyroid glands may be damaged or removed.
- Straightforward unilateral surgery can be performed under superficial and deep cervical plexus block, but GA is usual (see ➔ p. 1118).

Preoperative

- Ensure that the patient is as near euthyroid as possible (see ➔ pp. 223–5).
- Check for complications associated with hyperthyroidism: AF, tachycardia, proptosis.
- Acute preparation of thyrotoxic patients involves iodine and glucocorticoid—both inhibit the conversion of T4 to T3 and narrow the window (7–10d) for surgery, necessitating joint management with the surgeon and endocrinologist.
- Check biopsy histology for malignancy.
- Ask about duration of goitre. Long-standing compression of the trachea may be associated with tracheomalacia.
- Ask about positional breathlessness. Assess the airway.
- Examine the neck. How big is the goitre? Consistency: malignant goitres are hard. Can you feel below the gland (retrosternal spread)? Is there evidence of tracheal deviation (check the radiograph)?
- Look for signs of SVC obstruction: distended neck veins that do not vary with the respiratory cycle.
- Listen for stridor.
- Check the range of neck movements preoperatively, and do not extend them outside of their normal range during surgery.
- Preoperative paracetamol/NSAIDs (PO or PR) help postoperative pain control.

Investigations

- FBC, U&E, Ca^{2+} and thyroid function tests are routine.
- Chest radiograph. Check for tracheal deviation and narrowing. Thoracic inlet views may be necessary if retrosternal extension is suspected,

and to detect tracheal compression in the anterior–posterior plane (retrosternal enlargement may be asymptomatic).
- CT scan accurately delineates the site and degree of airway encroachment or intraluminal spread. Advisable if there are symptoms of narrowing (e.g. stridor, positional breathlessness) or >50% narrowing on the radiograph. Plain radiographs overestimate diameters, due to magnification effects, and cannot be relied on when predicting ETT diameter and length. Furthermore, a CT scan will help assess the degree of retrosternal extension.
- ENT consultation to document cord function for medicolegal purposes is not routine in all units, unless an abnormality is likely, e.g. previous surgery and malignancy. Pre-existing cord dysfunction may be asymptomatic. Fibreoptic examination also defines any possible laryngeal displacement (useful in airway planning). Look for photographs or diagrams in notes.

Airway planning

- The majority of cases are straightforward, even when there is some tracheal deviation or compression. A reinforced ETT will negotiate most distorted tracheas and permit optimal head positioning. Tracheal compression by a benign goitre will often accommodate an ETT beyond the predicted size, as the gland is soft. Preoxygenation should be followed by IV induction and a neuromuscular-blocking drug (after checking that the lungs can be inflated manually).
- The following features should lead to a more considered approach and may require discussion with the surgeon and radiologist:
  - **Malignancy**. Cord palsies are likely. Distortion and rigidity of surrounding structures. Possibility of intraluminal spread. The larynx may be displaced. The tumour can produce obstruction anywhere from the glottis to the carina.
  - Significant **respiratory symptoms** or >50% narrowing on chest radiograph or lateral thoracic inlet view.
  - Coexisting predictors of **difficult intubation**.

Options to secure the airway for complicated thyroid surgery

- Teamwork between the anaesthetist and the surgeon is the key to successful and safe airway management.
- **Inhalational induction** with sevoflurane in patients with stridor and a suspected difficult upper airway. Stridor and ↓ minute ventilation delay the onset of sufficiently deep anaesthesia for intubation. Topical LA may be useful.
- **Fibreoptic intubation** (see ➲ pp. 393–6). Attempts to pass a fibreoptic bronchoscope in an awake patient with stridor are difficult, as the narrowed airway may become obstructed by the instrument. May be useful where there is marked displacement of the larynx or coexisting difficulties with intubation, e.g. ankylosing spondylitis.
- An **SGA** may be difficult to place in patients with laryngeal displacement.

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- **Tracheostomy** under LA. This will only be possible if the tracheostomy can be easily performed below the level of obstruction.
- Ventilation through a **rigid bronchoscope** is a backup option when attempts to pass an ETT fail. The surgeon and necessary equipment should be immediately available for complex cases, particularly those involving significant mid- to lower tracheal narrowing.
- 'Plan C' of the difficult airway algorithm (perform a cricothyroid puncture) may not be an option.

Perioperative

- Eye padding, lubrication and tape are important, especially if the patient has exophthalmos.
- Tracheal manipulation during surgery can be very stimulating. Full relaxation or use of remifentanil should be used to prevent coughing during surgery.
- Electrophysiological monitoring of the recurrent laryngeal nerves is now common intraoperatively, using specialised reinforced ETTs with EMG capability. Neuromuscular-blocking drugs should therefore be avoided during maintenance of anaesthesia.
- Securely fix the ETT with tape, avoiding ties around the neck. Access to check the tube is difficult during the procedure.
- Head and neck extension with slight head-up tilt.
- Consider a superficial cervical plexus block for postoperative analgesia. Some surgeons infiltrate SC with LA and adrenaline before starting. LA at the end can produce spurious nerve palsies (see ➔ p. 1118).
- Arms to sides, IV extension.
- Communicate with the surgeon if there are excessive airway pressures during manipulation of the trachea. Obstruction may be due to airway manipulation distal to the tube or the bevel of the tube abutting on the trachea.
- Monitor muscle relaxation on the leg.
- In cases of long-standing goitre, some surgeons like to feel the trachea before closing to assess tracheomalacia. They may ask for partial withdrawal of the ETT, so that the tip is just proximal to the operative site.
- At the end of surgery, reverse the muscle relaxant and extubate with the patient sitting up to reduce venous compression. Use an extubation technique that minimises coughing to reduce early 2° haemorrhage. This might include waking up on remifentanil. Any respiratory difficulty should lead to immediate reintubation. The traditional practice of inspecting the cords immediately following extubation is difficult and unreliable. Possible cord dysfunction and postoperative tracheomalacia are better assessed with the patient awake and sitting up in the recovery room.

Postoperative

- Intermittent opioids with PO/PR paracetamol and NSAIDs.
- The opioid requirement is reduced with SC infiltration and superficial cervical plexus block.
- Use fibreoptic nasendoscopy if there is doubt about recurrent laryngeal nerve injury.

Postoperative stridor

- **Haemorrhage** with tense swelling of the neck. Remove clips from the skin, and sutures from the platysma/strap muscles to remove the clot. In extremis, this should be done at the bedside. Otherwise return to theatre without delay. A haematoma will affect lymphatic and venous drainage of the upper airway, causing laryngeal and pharyngeal oedema. Removing the haematoma will not always restore airway patency immediately. IV dexamethasone and nebulised adrenaline may help acutely.
- **Tracheomalacia**. Long-standing large goitres may cause tracheal collapse. This is a very rare complication. Immediate reintubation, followed by tracheostomy, may be necessary.
- **Bilateral recurrent laryngeal nerve palsies**. This may present with respiratory difficulty immediately postoperatively or after a variable period. Stridor may only occur when the patient becomes agitated. Assess by fibreoptic nasendoscopy. May require tracheostomy.

Other postoperative complications

Hypocalcaemia

- Hypocalcaemia from parathyroid removal is rare. Serum Ca^{2+} should be checked at 24h, and again daily if low.
- Presentation: may present with signs of neuromuscular excitability, tingling around the mouth or tetany. May progress to fits or ventricular arrhythmias.
- Diagnosis: carpopedal spasm (flexed wrists, fingers drawn together) may be precipitated by cuff inflation (Trousseau's sign). Tapping over the facial nerve at the parotid may cause facial twitching (Chvostek's sign). Prolonged QT interval on ECG.
- Treatment: serum Ca^{2+} below 2mmol/L should be treated urgently with 10mL of 10% calcium gluconate over 3min plus alfacalcidol 1–5g PO (calcium gluconate is preferable, as calcium chloride will cause tissue necrosis if extravasation occurs). Check the level after 4h, and consider Ca^{2+} infusion if still low. If hypocalcaemic, but level above 2mmol/L, treat with PO Ca^{2+} supplements (see also ➲ p. 227).

Thyroid crisis

(See ➲ pp. 224–5.)

Pneumothorax

Pneumothorax is possible if there has been retrosternal dissection.

Further reading

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Parathyroidectomy

Procedure	Removal of solitary adenoma or four glands for hyperplasia
Time	1–3h
Pain	+/++
Position	Shoulder bolster and head ring. Head-up tilt
Blood loss	Usually minimal
Practical techniques	IPPV + ETT

General considerations

(See ➔ p. 226.)

- Usual indication for operation is 1° hyperparathyroidism from parathyroid adenoma.
- With preoperative localisation, removal of simple adenoma has been described using sedation and LA. GA is more usual.
- Carcinoma may require en bloc dissection.
- Total parathyroidectomy may also be performed in 2° hyperparathyroidism associated with CKD.
- Hypercalcaemia may produce significant debility, particularly in the elderly.

Preoperative

Hypercalcaemia is usual. With moderate elevation, ensure adequate hydration with 0.9% sodium chloride. Levels over 3mmol/L should be corrected before surgery, as follows:

- Urinary catheter
- One litre of 0.9% sodium chloride in the 1st hour, then 4–6L over 24h
- Pamidronate 60mg in 500mL of 0.9% sodium chloride over 4h
- Watch for fluid overload—CVP measurement may be necessary. Monitor electrolytes, including Mg^{2+}, phosphate and K^{+}.

Severe hypercalcaemia may occasionally necessitate emergency surgery. It may cause arrhythmias and may antagonise the effects of NDMRs.

- Preoperative imaging using ultrasound and technetium-99m sestamibi scanning may be used to localise parathyroid adenomas, allowing a minimal access or targeted approach with a 2cm incision over the suspected gland.
- 2° hyperparathyroidism occurs 2° to low serum Ca^{2+} in CKD. In this situation:
  - Total parathyroidectomy may be required. Control afterwards is easier if no functioning parathyroid tissue is left.
  - Dialysis will be required preoperatively.
  - The risk of bleeding is ↑.
  - Alfacalcidol is usually started preoperatively.
- 1° hyperparathyroidism has been associated with an ↑ risk of death from CVS disease, hypertension, LVH, valvular and myocardial calcifications, impaired vascular reactivity, alterations in cardiac conduction, impaired glucose metabolism and dyslipidaemia. PTH has serious consequences on cardiac function in renal failure.

- A less utilised technique these days is to use methylthioninium chloride (methylene blue) to highlight the parathyroid glands. Most useful in four-gland hyperplasia; parathyroids are highly vascular and take up the dye faster than surrounding tissues. If given too early, however, the effect is lost, as the surrounding tissue colours. The usual dose is 5mg/kg, diluted in 500mL, given over 1h prior to surgery. Complications include restlessness, paraesthesiae, burning sensation, chest pain, dizziness, headache and mental confusion. SpO_2 will not be accurate if the infusion is too fast.

Perioperative

- Similar anaesthetic considerations to thyroid surgery. Airway encroachment is not usually a problem.
- Operation times may be unpredictable, especially if frozen section or parathyroid assays are performed. Consider active heat conservation.
- Point-of-care PTH assays are available, making intraoperative measurement of PTH possible in minimally invasive or targeted surgery, thereby allowing a rapid assessment of success intraoperatively.
- Extubation requires a cough-free technique, reducing the incidence of early 2° haemorrhage.

Postoperative

- Serum Ca^{2+} checked at 6h and 24h. Hypocalcaemia may occur (for diagnosis and treatment, see ➲ p. 713). Continuation of alfacalcidol in 2° hyperparathyroidism lessens the chance of hypocalcaemia postoperatively.
- Perform fibreoptic nasendoscopy if recurrent laryngeal nerve damage is suspected.
- Pain not usually severe, especially with LA infiltration or superficial cervical plexus blocks. PR paracetamol is useful. Avoid NSAIDs in patients with poor renal function.

Further reading

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Phaeochromocytoma

Procedure	Removal of one or two adrenals or extra-adrenal tumour
Time	1–2h open, possibly longer if laparoscopic
Pain	+/++ (depending if open or laparoscopic)
Position	Lateral or supine for open, lateral for laparoscopic
Blood loss	Variable
Practical techniques	IPPV + ETT, arterial and CVP lines ± CO monitor

- Tumours of chromaffin cells secreting noradrenaline (commonest), adrenaline or dopamine (least common). May secrete >1 amine.
- May secrete other substances, e.g. VIP, ACTH.
- Ninety-nine per cent occur in adrenals; 10% are bilateral; may be anywhere along the sympathetic chain from the base of the skull to the pelvis.
- Most are benign; a few are malignant.
- Occur in all age groups, less commonly in children.
- Can occur in association with MEN2A (medullary thyroid carcinoma, parathyroid adenomas) and MEN2B (medullary thyroid carcinoma and Marfanoid features). Both have abnormalities of the *RET* oncogene on chromosome 10.
- Also found in patients with neurofibromatosis 1 and von Hippel–Lindau syndrome.

Presentation

- Hypertension can be constant, intermittent or insignificant.
- Association of palpitations, sweating and headache with hypertension has a high predictive value.
- Anxiety, nausea and vomiting, weakness and lethargy are also common features.
- Acute presentations include pulmonary oedema, MI and cerebrovascular episodes.
- Can present perioperatively. Unless the diagnosis is considered and appropriate treatment instituted, the mortality rate is high—up to 50%.

Diagnosis

- Clinical suspicion.
- With ↑ genetic testing of families, more patients are being diagnosed before they become symptomatic (up to 30% familial).
- Urinary catecholamines or their metabolites (metadrenaline and normetadrenaline) measured either over 24h or overnight.
- CT radiocontrast may provoke phaeochromocytoma crises, and its use must be avoided in unblocked patients. Modern contrast agents may be used.
- MIBG (meta-iodobenzylguanidine) scan: a radiolabelled isotope of iodine taken up by chromaffin tissue.
- MRI.
- Search in the abdomen first, and widen the search if tumour not located. MIBG is particularly helpful in revealing unusual sites.

Investigations relevant to anaesthesia

- Echocardiography: patients with a history of ischaemia or signs of heart failure require cardiac echocardiography. Rarely, patients can present with a catecholamine cardiomyopathy.
- Blood glucose: excess catecholamines result in glycogenolysis and insulin resistance; some patients become frankly diabetic.

Preoperative

- Refer the patient to an experienced team. It is not acceptable to manage on an occasional basis.
- Usual management is sympathetic blockade with first α- and then β-blocker, if required, for tachycardia (**phenoxybenzamine**, a non-competitive, non-selective α-blocker, and then **atenolol/propranolol/metoprolol**).
- Phenoxybenzamine causes postural hypotension, lethargy and nasal congestion.
- Whereas α-blockade is generally considered a necessity, the use of β-blockade is more controversial and some anaesthetists will actively avoid them at the time of surgery.
- Preoperative blockade:
  - Allows safe anaesthesia for removal of the tumour
  - Prevents hypertensive response to induction of anaesthesia
  - Limits surges in BP seen during tumour handling.
- Avoid unopposed β-blockade; there is a theoretical risk of increasing vasoconstriction and precipitating a crisis. Although this has been reported, many patients will already have received β-blockers for hypertension before presentation, without adverse effects.
- Prazosin and doxazosin have been used. These are competitive, selective α1-blockers. They do not inhibit presynaptic noradrenaline reuptake and thus avoid the tachycardia seen with non-selective α-blockade. The literature contains reports both in favour and against the use of selective blockade.
- Calcium channel blockers (particularly nicardipine) have been used. They inhibit noradrenaline-mediated Ca^{2+} influx into smooth muscle but do not affect catecholamine secretion by the tumour.
- Metirosine is an inhibitor of catecholamine synthesis. It is toxic and not widely used.
- There are no absolute criteria for fitness for surgery.

Assessment of sympathetic blockade

- Patients will often be admitted a few days prior to surgery to observe BP control and/or have had outpatient 24h ambulatory BP monitoring.
- Aim for BP <140/90mmHg, with HR <100bpm.
- Erect and supine BP and HR. Should exhibit a marked postural drop >20mmHg, with compensatory tachycardia.
- The duration of blockade is determined by the practicalities of tumour localisation and scheduling of surgery.
- Blockade is started to treat symptoms, as well as to prepare for surgery.

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Perioperative

- Laparoscopic or open adrenalectomy through a midline, transverse or flank incision (introduction of gas for laparoscopic resection can result in hypertension in normal subjects, and this may be exaggerated in patients with phaeochromocytomas).
- Premedication PRN (e.g. temazepam 20–30mg).
- Monitoring to include direct BP and CVP (triple lumen to allow drug infusions). Consider CO monitoring in patients with CVS disease and catecholamine cardiomyopathy.
- Large-bore IV access.
- Monitor and maintain temperature, particularly during laparoscopic resection which can be prolonged.
- Induction: avoid agents that release histamine, and thus catecholamines (use propofol, alfentanil or remifentanil and vecuronium or rocuronium).
- Hypotension is unlikely at induction and can be treated with either ephedrine, metaraminol or phenylephrine. Due to preoperative α-blockade, doses will likely need to be ↑. Rarely, dilute adrenaline may be required.
- Maintenance: if using volatile, use isoflurane or sevoflurane. Desflurane should be avoided, as it can cause sympathetic nervous system activation.
- Consider epidural with opioid and LA for open procedures (sympathetic blockade will not prevent catecholamine-induced vasoconstriction); otherwise fentanyl/alfentanil/remifentanil until tumour removal, when morphine (10–20mg) can be substituted.
- Nicardipine and Mg^{2+} are also useful (block catecholamine release, block receptors and provide direct vasodilation and possibly myocardial protection).
- Mg^{2+} is started prior to induction, given as a bolus of 2–4g, and then continued at a rate of 1–2g/h. It is normal for the patient to feel nauseated with the Mg^{2+} bolus.
- Surges in BP can occur at induction, with formation of the pneumoperitoneum and with tumour handling. The fluctuations in BP tend to be transient, and medication needs to respond in a similar fashion. Hypertension can be treated in a number of ways: intermittent 2g boluses of Mg^{2+}, boluses of remifentanil, phentolamine, sodium nitroprusside or labetalol (if associated with tachycardia).
- Control HR at <100bpm with the β-blocker of choice.
- Once the tumour is resected, BP takes several minutes to decline. Prevent hypotension by ensuring an adequate preload. Maintain a high CVP of 10–15mmHg. Several litres of a crystalloid may be needed.
- Hypotension following resection can be due to low CO or a low SVR. Treat the former with low-dose adrenaline, and the latter with metaraminol or phenylephrine. Vasopressin has been used in resistant hypotension. Terlipressin 1mg bolus, followed, if required, by vasopressin, starting at 0.04 units/min, then titrated to effect.
- It is unusual to require inotropic support by the time the patient is ready to leave theatre, unless there are coexisting medical problems.

Postoperative

- Patient should be nursed in ICU/HDU for 12h.
- Monitor blood glucose. The withdrawal of catecholamine excess can lead to severe hypoglycaemia.
- If both adrenals are resected, the patient will require steroid support immediately. Hydrocortisone 100mg bolus in theatre, decreasing to maintenance dose after surgical stress. Fludrocortisone 0.1mg daily may be commenced with oral intake.
- Even when only one adrenal is removed, patients may occasionally be relatively hypoadrenal and require support. If this is suspected (e.g. unexpectedly low BP), a small dose of hydrocortisone (50mg) will do no harm, while the result of cortisol estimation is awaited.

Special considerations

Pregnancy

- There are many reports of the combination of a newly diagnosed phaeochromocytoma and pregnancy. Overall mortality is up to 17%.
- Phenoxybenzamine and metoprolol are safe.
- If phaeochromocytoma is diagnosed before mid trimester, it should be resected at this stage.
- There is high mortality associated with normal delivery; consider lower-segment Caesarean section (LSCS), with or without resection of the phaeochromocytoma at the same procedure.

Management of an unexpected phaeochromocytoma

- Any patient who has unexplained pulmonary oedema, hypertension or severe unexpected hypotension should prompt consideration of the diagnosis; however, it can be very difficult. There is no quick available test to support the diagnosis in the acute situation.
- Once the diagnosis has been considered, if possible, surgery should be discontinued to allow acute treatment, investigation and blockade prior to definitive surgery. Attempts to remove the tumour during a crisis may result in significant morbidity, or even mortality.
- Treatment acutely should consist of vasodilators and IV fluid; this may be counterintuitive in a patient with severe pulmonary oedema. The circulating volume in patients with phaeochromocytoma may be markedly reduced, and vasodilation will result in a profound drop in BP. GTN can usually be successfully titrated in this situation.
- Patients who present with hypotension have an acutely failing heart due to profound vasoconstriction. These are the most difficult patients in whom to make the diagnosis and to treat. Additional catecholamines in this situation merely fuel the fire but are difficult to resist. The mortality rate is very high.

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Carcinoid tumours

Carcinoid tumours are derived from argentaffin cells and produce peptides and amines. They occur in the GI tract (75%), bronchus, pancreas and gonads. Surgery may be performed to remove the 1° tumour and debulk liver metastases in carefully selected patients.

Conduct of anaesthesia in patient with carcinoid syndrome

Best managed by centres familiar with the difficulties. Major complications anticipated include profound swings in BP, fluid and electrolyte shifts and bronchospasm.

- Preoperatively treat symptomatically with antidiarrhoeal agents, bronchodilators, correction of dehydration/electrolyte imbalance and treatment of heart failure.
- Preoperatively, some centres will use continuous infusions of octreotide 50–100 micrograms/hour; others give 100 micrograms SC tds. Although schedules vary from weeks (SC) to hours (IVI), both are only of benefit even if given before surgery to suppress basal amine turnover.
- Avoid factors that may trigger carcinoid crises: anxiety and drugs that release histamine, e.g. morphine.
- Premedication: anxiolytic (benzodiazepine) and octreotide 50–500 micrograms SC 1h preoperatively, if not already treated; otherwise continue with preoperative regime.
- Monitoring should include invasive BP preinduction (both induction and surgical manipulation of the tumour can cause large swings), CVP, regular blood glucose and blood gases. CO monitoring will guide fluid therapy and help in managing hormone-induced preload and afterload variations, particularly if cardiac complications present.
- Right heart failure increases hepatic venous congestion which predisposes to bleeding in liver surgery. Limiting IV fluid initially in these patients and permitting controlled hypotension may reduce bleeding.
- Consider an epidural. Benefits include a reduced risk of a carcinoid crisis with ↓ stress response 2° to good analgesia; however, low doses of LA should be used.
- Give octreotide 100 micrograms IV diluted to 10 micrograms/mL, slowly at induction. Prevent pressor response to intubation.
- Suxamethonium has been used safely for RSI, although fasciculations may theoretically stimulate hormone release by increasing the intra-abdominal pressure, so consider rocuronium.
- Maintenance: both TIVA and inhalation techniques have been used successfully.
- Octreotide (10–20 microgram boluses IV) to treat severe hypotension.
- Avoid all histamine-releasing drugs (atracurium, morphine) and catecholamines (release serotonin and kallikrein, which activate bradykinins).
- Labetalol, esmolol or ketanserin (5-HT_2 receptor blocker) can be used for hypertension.
- ICU or HDU is required.

- Patients may wake very slowly (thought to be due to serotonin).
- Hypotensive episodes may occur, as surgery may have reduced, rather than eliminated, the tumour, thus requiring further IV boluses of octreotide (10–20 micrograms).
- Wean octreotide over 7–10d following tumour resection.

Further reading

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Chapter 29

Urological surgery

Mark Catolico

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Open simple prostatectomy and radical prostatectomy *732*
Nephrectomy and partial nephrectomy *733*
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See also

Circumcision/hypospadias repair p. *941*
Orchidopexy p. *942*

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Cystoscopic procedures

- A large number of urological procedures are performed via the cystoscope. These include cystoscopy, transurethral resection of the prostate (TURP), bladder neck incision, transurethral resection of bladder tumour, ureteroscopy and/or stone removal or stent insertion.
- The majority of patients are undergoing procedures for benign prostatic hypertrophy or carcinoma of the bladder. The incidence of both these conditions increases markedly over 60y, and bladder cancer is smoking-related, so patients frequently have CAD and COPD.
- FBC, creatinine and electrolytes should be checked preoperatively, because bladder cancers can bleed insidiously. Both bladder cancer and benign prostatic hypertrophy can cause an obstructive uropathy/renal impairment, and drugs and the technique should be chosen accordingly.
- **Flexible cystoscopy** is largely used for diagnostic purposes, does not require full bladder distension and can normally be performed under LA. Biopsies can be taken this way, with only a small amount of discomfort, and skilled surgeons can perform retrograde ureteric catheterisations. Occasional patients insist on sedation/GA for flexible cystoscopy. Midazolam or propofol is ideal.
- Rigid cystoscopy requires GA, due to the scope diameter and the use of an irrigating solution to distend the bladder and allow visualisation of the surgical field. If large volumes of irrigant are absorbed, systemic complications due to fluid overload can result (see pp. 729–30).
- Spinal anaesthesia works well for rigid cystoscopic procedures and is commonly used for TURP. Sensory supply to the urethra, prostate, bladder neck and bladder mucosa is from S2 to S4. Pain from bladder distension, however, is carried by T10–L2, so a higher block is required. Many patients will request sedation. In the elderly population, 1–2mg of midazolam is usually adequate. Higher doses may result in loss of airway control, confusion and restlessness. A low-dose propofol infusion is an alternative. Spinal anaesthesia is advantageous for patients with severe COPD, as long as the patient can lie flat without coughing.
- Either hyperbaric or isobaric bupivacaine can be used. Hyperbaric bupivacaine usually produces a higher block than the isobaric solution, especially when the injection is performed with the patient in the lateral position and then turned supine; 2.5–3mL of 'heavy bupivacaine' 0.5% usually gives a block to T10. Do not tilt the patient head-down, unless the block is not sufficiently high.
- Other short-acting drugs increase options for effective ambulatory spinal anaesthesia (level T10 and below); 40–50mg (4–5mL) of plain chloroprocaine 1% for procedures of up to 40min, or 40–60mg (2–3mL) of hyperbaric prilocaine 2% for procedures lasting up to 90min. These options provide more predictable spinal anaesthesia, compared with bupivacaine, and quicker readiness for discharge (see Fig. 18.2).
- Patients with chronic chest disease tend to cough on lying flat. During surgery under regional block, coughing can seriously impair surgical access. Sedation can help to reduce the cough impulse.
- Patients with spinal cord injuries (see pp. 303–8) often require repeated urological procedures. Bladder distension during cystoscopy is very stimulating and prone to cause autonomic hyperreflexia, so a GA or spinal is advisable—check previous anaesthetic charts.

- Take particular care in positioning elderly patients in lithotomy, especially those with joint replacements.
- **Permanent pacemakers** are not normally a problem, even with the almost continuous diathermy required for TURP, as long as the diathermy plate is positioned caudally, usually on the thigh.
- **Implantable defibrillators** can be triggered by the diathermy, so need to be switched off preoperatively (see pp. 161–2).
- **Penile erection** can make cystoscopy difficult and surgery hazardous. It usually occurs due to surgical stimulation when the depth of anaesthesia is inadequate, and can usually be managed by deepening anaesthesia. If the erection still persists, small doses of ketamine can be useful.
- **Antibiotic prophylaxis** (single dose of an agent with Gram-negative cover) is often required, particularly if the patient has an indwelling urinary catheter, an obstructed ureter and positive results on preoperative midstream urine (MSU). Gentamicin 3mg/kg is popular.
- **DVT prophylaxis**: graduated compression stockings or pneumatic calf compression devices are generally considered adequate in low-risk patients undergoing cystoscopic procedures, as most will mobilise rapidly following surgery. However, LMWH should also be used in patients with additional risk factors or those who have recently undergone other surgery and a period of immobility (see pp. 273–4).

Postoperative complications of rigid cystoscopic procedures

- **Perforation of the bladder** can occur and can be difficult to recognise, especially in the presence of a spinal block, which may mask abdominal pain. Perforations are classified as extraperitoneal when pain is said to be maximal in the suprapubic region, and intraperitoneal when there is generalised abdominal pain, shoulder tip pain due to fluid tracking up to the diaphragm and signs of peritonism. Intraperitoneal perforations need fluid resuscitation and urgent surgery to prevent progressive shock.
- **Bacteraemia** can have a very dramatic onset with signs of profound septic shock. If the diagnosis is made quickly, there is usually a rapid response to IV fluids and appropriate antibiotics (e.g. gentamicin single dose of 3–5mg/kg, followed by cefuroxime 750–1500mg 8-hourly; modify according to sensitivities on preoperative MSU). Always suspect this diagnosis with unexplained hypotension after a seemingly straightforward urinary tract instrumentation.
- **Bladder spasm** is a painful, involuntary contraction of the bladder occurring after any cystoscopic technique, most commonly in patients who did not have an indwelling catheter preoperatively. Diagnosis is supported by failure of irrigation fluid to flow freely in and out of the bladder. It responds poorly to conventional analgesics but is often eased by small doses of IV benzodiazepine, e.g. diazepam 2.5–5mg or hyoscine butylbromide 20mg slow IV or IM.
- Bleeding and fluid overload are dealt with under anaesthesia for TURP (see pp. 728–30).

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Miscellaneous urological procedures

Table 29.1 presents the important information for those procedures which are not covered in the individual topics within this chapter.

Table 29.1 Other urological procedures

Operation	Description	Time (min)	Pain (+ to ++++)	Position	Blood loss/ X-match	Notes
Uretero-scopy	Investigate obstruction, remove stones	20–60	+	Lithotomy	Nil	LMA + SV. Check renal function. Possible antibiotic prophylaxis
Insert ureteric stent	To relieve ureteric obstruction, using image intensifier	20	+	Lithotomy	Nil	LM + SV. Possible antibiotic prophylaxis
Remove ureteric stents	Cystoscopy to retrieve stent	10–20	+	Lithotomy	Nil	Awake or LMA + SV. Often possible with flexible scope and LA
Insert suprapubic catheter	Trans-cutaneous insertion of catheter into full bladder	15	+	Lithotomy or supine	Nil	Sedation + LA or LMA + SV. Often frail patients with advanced neurological disease
Bladder neck incision	Transurethral diathermy incision of prostate at narrowed bladder neck	15–30	++	Lithotomy	Nil	LMA + SV. Younger patients than TURP
Urethro-plasty	Recon-struction of urethra narrowed by trauma or infection—very variable procedure	90–240	++++	Lithotomy	300–2000mL	ETT + IPPV ± epidural. Beware prolonged lithotomy. Consider epidural for postoperative pain
Nesbit's procedure	Straightening of penile deformation from Peyronie's disease	60–120	+++	Supine	Nil	LMA + SV. Consider caudal or penile block

Operation	Description	Time (min)	Pain (+ to ++++)	Position	Blood loss/ X-match	Notes
Circumcision	Excision of foreskin	20	++	Supine	Nil	LMA + SV + LA. Penile block or caudal useful. Topical lidocaine gel to take home. LA alone possible in frail elderly
Urethral dilation	Stretching of narrowed urethra with serial dilators	10	+	Lithotomy or supine	Nil	LMA or spinal. Possible antibiotic prophylaxis
Urethral meatotomy	Incision to widen urethral meatus	10	+	Supine	Nil	LMA + SV
Orchidectomy	Removal of testis—through groin or scrotum, depending on pathology	20–45	++	Supine	Nil	LMA + SV + ilioinguinal block. Need to block to T9/10 if using regional technique due to embryological origins
Vasectomy	Division of vas deferens via scrotal incision	20–40	++	Supine	Nil	LMA + SV. Often under LA
Pyeloplasty	Refashioning of obstructed renal pelvis via loin incision. Children and young adults	90–120	++++	Lateral 'kidney position'	300–500mL	ETT + IPPV + epidural/ PCA. Similar considerations as nephrectomy. May be significant blood loss in children
Prostatic urethral lift 'UroLift®' implant	Minimally invasive treatment for benign prostatic hypertrophy	15–30	+	Lithotomy	Nil	LMA + SV. LA ± sedation is possible

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Transurethral resection of the prostate

Procedure	Cystoscopic resection of the prostate using diathermy wire—monopolar/glycine irrigation being replaced by bipolar resectoscopes with 0.9% sodium chloride irrigation
Time	30–90min, depending on size of the prostate
Pain	+
Position	Lithotomy ± head-down
Blood loss	Very variable (200–2000mL), can be profuse and continue postoperatively; G&S
Practical techniques	Spinal ± sedation is method of choice GA, LMA and SV GA, ETT and IPPV

Preoperative

- Patients are frequently elderly with coexisting disease and on multiple medications.
- Check creatinine and serum Na^+; suggest postponing surgery if Na^+ is significantly low, as this is likely to fall further with absorption of irrigant.
- Heart failure or uncontrolled AF is a particular risk due to fluid absorption. Aim for optimal medical control preoperatively.
- Assess the mental state and communication—spinal anaesthesia is difficult if the patient is confused or deaf.

Perioperative

- Insert a large cannula, and use warmed IV fluids.
- **Spinal anaesthesia**: in theory, it is easier to detect changes in the mental state and signs of fluid overload (see pp. 729–30); shown in some, but not all, studies to reduce blood loss; 2.5–3mL of bupivacaine (plain or hyperbaric) or consider alternative LA solutions (see Fig. 18.2); frequent BP check—hypotension unusual with the above doses but can occur suddenly; check BP at the end when the legs are down (unmasks hypotension).
- **GA**: consider intubation if the patient is very obese or has a history of reflux; intraoperative fentanyl or morphine plus multimodal analgesia (paracetamol, NSAID and tramadol/codeine) are usually adequate; unusual to need opioids postoperatively.
- **Blood loss** can be difficult to assess. In theory, can be calculated from measuring Hb and the volume of discarded irrigation fluid. In practice, it is more common to visually assess the volume and colour, but this can be misleading. Checking the patient's Hb with a bedside device (e.g. HemoCue®) is useful. Blood loss is generally related to the size and weight of prostatic tissue excised (normally 15–60g), the duration of resection and the expertise of the operator.
- Antibiotic prophylaxis usually required.
- Obturator spasm (see p. 731).
- Fluid therapy: crystalloid can be used initially. Bear in mind that a significant volume of hypotonic irrigating fluid may be absorbed, so do not give excessive volumes and never use glucose. Be ready to transfuse if the Hb falls below the transfusion trigger.

Postoperative

- Bladder irrigation with 0.9% sodium chloride via a three-way catheter continues for ~24h, until bleeding is reduced; inadvertent slowing of irrigation can lead to clot retention.
- There is generally little pain, but discomfort from the catheter or bladder spasm may be a problem (see ➲ p. 725).
- Severe pain suggests clot retention, bladder spasm or bladder perforation (see ➲ p. 725).
- Clot retention can give a very distended painful bladder and vagal symptoms. It requires washout, sometimes under anaesthetic.
- Bleeding can continue and require further surgery; resuscitation may be necessary.
- Measure FBC, creatinine and electrolytes the day following surgery.

Special considerations

- Hypothermia may result when large volumes of irrigation fluid are used (the fluid should be warmed to 37°C).
- If the prostate is very large (>100g), a simple open prostatectomy may carry fewer complications.
- The risk of complications increases with resection times of >1h. If a resection is likely to take longer than an hour, consider limiting the resection to one lobe only, leaving the other to be done at a later date.
- Bipolar TURP has been shown to reduce the overall complication rate, transfusion rate and TURP syndrome.

Laser TURP and transurethral vaporisation of the prostate

- Several 'minimally invasive' techniques using lasers and other forms of heat have been developed which reduce the prostate size. These generally cause less bleeding and absorption of fluid so are sometimes chosen for patients perceived to be at higher risk from conventional TURP.
- Laser vaporisation techniques can be done under LA on an outpatient basis. These may be preferred in patients on anticoagulation because of their haemostatic effect on prostate tissue.

Brachytherapy for localised prostate carcinoma

- This consists of insertion of radioactive pellets through rods positioned in the prostate under ultrasound control. This is usually done outside of theatre in the radiotherapy department.
- The patient may require two or more procedures in the same day. Repeated GAs are possible, but a spinal catheter topped up before each procedure works well. Consider risks of remote site anaesthesia.
- For a single treatment, a spinal, using 0.5% bupivacaine with fentanyl 15 micrograms, is effective and allows safe transfer to radiotherapy.

TURP syndrome

- A combination of fluid overload and hyponatraemia, which occurs when large volumes of irrigation fluid are absorbed via open venous sinuses.
- Classic TURP uses monopolar cautery. Therefore, non-conductive (non-electrolyte) irrigation fluid must be used to allow diathermy current to be focused and avoid thermal burns. The most commonly used irrigant is glycine 1.5% in water, which is hypotonic (osmolality 220mmol/L).

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- Glycine is a non-essential amino acid which functions as an inhibitory neurotransmitter, and it is unclear whether glycine toxicity plays a part in the syndrome. Ammonia is a metabolite of glycine and may also contribute to CNS disturbance.
- Bipolar TURP has become a more common procedure, which allows use of isotonic irrigation fluid. Hyponatraemia is far less likely; however, absorption of large volumes causing overload may still occur. A meta-analysis comparing monopolar vs bipolar TURP found that the bipolar technique eliminated the 'TURP syndrome' and had a lower overall complication rate.
- The most effective way of managing TURP syndrome is through early detection and prevention.
- Prevention includes avoiding monopolar cautery (non-isotonic irrigation), minimising fluid absorption by using low infusion pressures (bladder pressure <15mmHg), monitoring the quantity of irrigation fluid absorbed (consider terminating the procedure if >2L fluid deficit) and limiting the duration of surgery.
- Signs of *pulmonary oedema*, *cerebral oedema* and *hyponatraemia* are the usual presenting features. Choosing regional anaesthesia allows for early detection.
- *Early symptoms* include restlessness, headache and tachypnoea, and these may progress to respiratory distress, hypoxia, frank pulmonary oedema, nausea, vomiting, visual disturbances, confusion, convulsions and coma. In the anaesthetised patient, the only evidence may be tachycardia and hypertension. Rapid absorption of a large volume can lead to reflex bradycardia. Hypotension can also occur. The diagnosis can be confirmed by low serum Na^+. An acute fall to <120mmol/L is always symptomatic. A quick check of Na^+ is often possible by checking an ABG (use venous blood, unless concerned about acid/base balance).
- If detected intraoperatively, notify the surgeon; bleeding points should be coagulated, surgery terminated as soon as possible and IV fluids stopped. Give furosemide 20–40mg, and check serum Na^+ and Hb. Support respiration with O_2 or intubation and ventilation, if required. Administer IV anticonvulsants, if fitting.
- Both severe acute hyponatraemia and overly rapid correction of chronic hyponatraemia can result in permanent neurological damage (most commonly central pontine myelinolysis).
- If serum Na^+ remains >120mmol/L, fluid restriction should suffice. If <120mmol/L and associated with neurological signs, consider giving hypertonic sodium chloride (1.8–3%) to restore Na^+ to around 125mmol/L (see ➲ p. 242). Give 1.2–2.4mL/kg/h of 3% sodium chloride until symptomatic improvement. Correction should ideally not be faster than 1.5–2mmol/L/h for 3–4h, then 1mmol/L/h until symptomatic improvement or Na^+ >125mmol/L. Maximum rise should not exceed 12mmol/L in 24h.
- Beware of compounding effects on Na^+ by other simultaneous treatments (diuretics, colloids, etc.).

Transurethral resection of bladder tumour

Procedure	Cystoscopic diathermy resection of bladder tumour
Time	10–40min
Pain	+/++ and bladder spasm
Position	Lithotomy
Blood loss	0 to >500mL
Practical techniques	GA with LMA; spinal ± sedation

Preoperative

- Slow-growing tumours of the bladder epithelium are common in the elderly. They are best managed by periodic resection of the tumours within the bladder, rather than aggressive complete removal.
- Commonest in smokers; check for CAD and COPD.
- Check Hb; chronic blood loss is common.
- Check renal function.
- Refer to previous anaesthetic charts; many patients have repeated surgery.

Perioperative

- Obturator spasm occurs when the obturator nerve, which runs adjacent to the lateral walls of the bladder, is directly stimulated by the diathermy current. It causes adduction of the leg and can seriously impair surgical access and increase the risk of bladder perforation. It can usually be controlled by reducing the diathermy current or intermittent use of NMBAs.
- Antibiotic prophylaxis.

Postoperative

- Pain can be a problem with extensive resections; NSAIDs are useful (check renal function) and oral opioids may be needed.
- Bladder spasm is common (see p. 725).

Special considerations

- If using a spinal anaesthetic, ensure block to above T10 (see pp. 724–6).

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Open simple prostatectomy and radical prostatectomy

Procedure	Simple retropubic (Millen procedure): open excision of grossly hypertrophied benign prostate shelled out of capsule; radical retropubic: open complete excision of malignant prostate and pelvic lymph nodes with anastomosis
Time	60–120min (simple); 120–180min (radical)
Pain	+++/++++
Position	Supine
Blood loss	300–1000mL (simple); 500–2000mL (radical) X-match 2 units, and use cell salvage if available
Practical techniques	ETT, IPPV, remifentanil ± rectus sheath catheters ± PCA

Preoperative

- Simple prostatectomy is usually carried out in elderly men and can be treated as TURP.
- Radical: patients are selected if relatively young and medically fit.
- Check renal function.
- Consider COX-2 selective inhibitors, gabapentinoids and dexamethasone as preoperative analgesic interventions.
- Consider HDU bed for radical prostatectomy, depending on local practice and medical factors.

Perioperative

- Prepare for major blood loss with a large IV cannula, blood warmer, forced air warming blanket, etc.
- A Pfannenstiel-type incision is used for simple, and lower midline for radical, prostatectomy.
- Consider using an arterial line and CVP line or CO monitoring, particularly in patients with CVS disease.
- Ensure blood is available, and reorder intraoperatively, as necessary.
- Cell salvage techniques can be useful where blood loss is expected to be substantial (radical prostatectomy).
- Air embolism is a possible complication.
- Epidural analgesia is no longer recommended (see the European Society of Regional Anaesthesia and Pain Therapy's PROSPECT reference, ➲ Further reading, p. 744).
- Consider using remifentanil infusion intraoperatively.

Postoperative

- For open prostatectomy, LA wound infiltration administered at the end of surgery is recommended. Lidocaine infusion is recommended for radical prostatectomy. PCA may be a useful alternative.

Nephrectomy and partial nephrectomy

Procedure	Excision of kidney for tumour, other pathologies or live donor
Time	1–2.5h
Pain	+++/++++
Position	Supine or lateral (kidney position)
Blood loss	Depends on pathology, 300 to >3000mL. G&S/X-match, as required
Practical techniques	ETT + IPPV ± thoracic epidural or wound infiltration catheter

Preoperative

- Ascertain the pathology and surgical incision planned before deciding on the technique and monitoring.
- Check Hb; renal tumours can cause anaemia without blood loss.
- Check BP and renal function; 'non-functioning' kidney or renovascular disease is associated with renal impairment and hypertension.
- Consider cell salvage intraoperatively.
- Check serum electrolytes; renal tumours can cause inappropriate ADH secretion.
- Check the chest radiograph if there is a tumour; there may be metastases, pleural effusions, etc.
- Radiofrequency ablation (RFA) is an option for patients with small cortical tumours (<3cm), and those who present a high surgical risk or have compromised renal function.

Perioperative

- Nephrectomy and partial nephrectomy can be carried out via open, laparoscopic or robot-assisted laparoscopic approaches.
- For large tumours and polycystic kidneys, surgical practice in the UK is an open laparotomy via a paramedian or transverse incision for a tumour, and a loin incision with a retroperitoneal approach for other pathologies or donor nephrectomy.
- Loin incision requires the 'kidney position', i.e. lateral with the patient extended over a break in the table. A marked fall in BP is common on assuming this position due to reduced venous return from the legs and possible IVC compression. Further compression during surgery may result in a severe reduction in venous return and CO.
- Ask the surgeon about the predicted extent of surgery; a large tumour may necessitate extensive dissection, possibly via a thoracotomy, or opening of the IVC to resect tumour margins, in which case sudden, torrential blood loss is possible. Occasionally, the IVC is temporarily clamped to allow dissection and to control haemorrhage; this gives a sudden fall in CO. Inform the surgeon if BP falls suddenly; have fluids and blood checked and available to infuse immediately under pressure, and have a vasoconstrictor or an inotrope, such as metaraminol or ephedrine, prepared.

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- Use large IV cannulae, blood warmer, central venous line and arterial line if the procedure is anything other than an uncomplicated, non-malignant nephrectomy or a small isolated tumour.
- If an epidural is used, a high block will be required postoperatively, but use it cautiously intraoperatively until bleeding is under control.

Postoperative

- All approaches are painful; epidurals are useful but need to cover up to T7/8 for a loin incision. Rectus sheath catheters may be useful for the anterior approach. PCA or an opioid infusion is an alternative.
- Intercostal blocks will give analgesia for several hours after a loin incision.
- Wound infiltration catheters have also proved very effective.
- NSAIDs are useful if renal function is good postoperatively and the patient is not hypovolaemic. Use cautiously.
- Monitor hourly urine output.

Partial nephrectomy

- Recommended as the preferred option in organ-confined tumours measuring up to 7cm.
- Blood loss can be large, as vessels are more difficult to control.
- Some surgeons suggest the administration of mannitol 12.5g, furosemide 10mg and/or heparin 3000 units before clamping of the renal artery in an attempt to maintain renal perfusion and minimise ischaemia. Cooling with ice can also be used.
- If the renal function is markedly impaired preoperatively, optimisation of fluid balance throughout the perioperative period is extremely important. Admission to HDU postoperatively should be considered.

Radical cystectomy

Procedure	Excision of bladder plus urinary diversion procedure (e.g. ileal conduit) or neobladder reconstruction (orthotopic bladder formation)
Time	3–5h (longer with bladder reconstruction)
Pain	++++
Position	Lithotomy plus head-down
Blood loss	700 to >3000mL, X-match 4 units, use cell salvage
Practical techniques	ETT, IPPV, arterial line ± oesophageal Doppler/CVP ± rectus sheath catheters/PCA/epidural

Preoperative

- Consider the use of an 'enhanced recovery pathway'.
- Check for IHD/COPD, plus renal function and FBC.
- Book an HDU bed, depending on local practice/coexisting problems.
- Ensure thromboprophylaxis is prescribed.

Perioperative

- The commonest postoperative problem is prolonged ileus, which contributes significantly to morbidity and mortality, and several of the measures recommended are thought to reduce its incidence.
- Prepare for major blood loss; large IV cannulae, blood warmer, CVP or oesophageal Doppler CO and direct arterial monitoring are routine. Ensure blood is available, and reorder intraoperatively, as necessary. Consider autotransfusion intraoperatively. If blood salvage is used, discontinue it when the bowel is opened.
- If rectus sheath catheters are to be used, insert after induction. Surgical incision is subumbilical midline and does not permit easy placement of catheters during surgery.
- Remifentanil infusion gives stable anaesthesia and controllable BP; mild hypotension can aid surgery by reducing blood loss.
- Epidurals are now used infrequently, but if placed, use cautiously intraoperatively; there will be plenty of time after the main blood-losing episode to establish an adequate block.
- Take measures to prevent heat loss, e.g. forced air warming blanket.
- Antibiotic prophylaxis as for bowel resection.
- Use of NGTs is now rare; ask the surgeon if specifically required.
- Blood loss can be insidious from pelvic venous plexuses; consider weighing swabs.
- Air embolism is a possible complication, as in any major pelvic surgery.

Postoperative

- Rectus sheath catheters (for up to 5d), with or without a PCA, have proven very effective. Visceral pain tends to last for 24–36h postoperatively, requiring parenteral opioids. This technique enables early mobilisation, return of bowel function and ↓ postoperative ileus/length of hospital stay. There is some evidence that anastomotic leak is ↑ with the use of NSAIDs, so use cautiously after checking the renal function.

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- Use CVP/urine output to guide fluid replacement; requirements are usually large due to intraperitoneal loss and ileus.
- Urine output via a new ileal conduit is difficult to monitor, as drainage tends to be positional. Following orthotopic reconstruction, urine drains from a number of different catheters so needs to be calculated each hour to monitor output.
- Early feeding, as part of enhanced recovery, may be associated with a reduced incidence of certain postoperative complications.
- Leakage from a ureteric anastomosis may present as urine in the abdominal drain; confirm by comparing biochemistry of the drainage fluid and urine from the conduit.

Robot-assisted laparoscopic prostatectomy

(See ➲ pp. 480–2.)

Procedure	Robot-assisted laparoscopic prostatectomy
Time	120–180min
Pain	+/++
Position	Lithotomy with steep Trendelenburg
Blood loss	Minimal
Practical techniques	ETT, IPPV, remifentanil, arterial line

Preoperative

- The commonest use of surgical robots to date is in urology, mostly for radical prostatectomy (robot-assisted laparoscopic prostatectomy).
- Advantages to the patient may include a reduction in blood loss, pain and length of stay ± a reduced incidence of incontinence and erectile dysfunction.
- Advantages to the surgeon over conventional laparoscopy include provision of 3D vision, filtration of any hand tremor, scaling of hand movements, greater range of movements within the patient and a comfortable and stable position.

Perioperative

- Positioning of the patient is important. Long operative times with steep head-down tilt have been associated with:
  - Neurapraxia (especially brachial). Take care with positioning, and use shoulder brace. Do not hyperextend the legs in lithotomy.
  - Facial/airway oedema and stridor.
  - Acid burns to eyes and oral ulceration due to reflux of gastric acid. Premedicate with a PPI (omeprazole 40mg); consider an OGT (not NGT due to epistaxis risk) and throat pack, and protect the eyes with lubricating ophthalmic ointment/pads.
  - ↑ ICP (exacerbated by hypercapnia) and ↑ IOP (beware patients with glaucoma).
- Insert ETT as short as possible (risk of endobronchial intubation), and tape in position to minimise cerebral venous obstruction.
- Access to the patient is poor, so ensure reliable, large-bore venous access on the left side because of robot arm positioning.
- Robotic equipment is locked in position once inserted into the abdomen, so any inadvertent patient movement can cause grave surgical complications. A remifentanil infusion works well and allows intermittent boluses of muscle relaxant only as required.
- Avoid using N_2O.
- Communicating with the surgeon may be difficult due to the bulk and space required for the equipment. The team needs to be familiar with the audio equipment and also able to undock and remove the robot quickly in case of an emergency requiring resuscitation.
- Large urine output can interfere with the surgical field; use minimal IV fluid (<1000mL) until anastomosis is complete (may also reduce the risk of airway oedema).

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Postoperative

- Perform leak test prior to extubation to assess airway swelling.
- Cerebral oedema may be problematic. A short-acting volatile agent with remifentanil allows rapid assessment of the conscious level postoperatively. Most patients experience a degree of cerebral irritation or agitation initially on wakening.
- Postoperative pain is considerably less than for open procedures but can still be severe enough to require opioid analgesia for a short period, in addition to simple analgesics. In some institutions, use of intrathecal diamorphine up to 1mg is commonly used.

Percutaneous stone removal

Procedure	Endoscopic excision of renal stone via nephrostomy
Time	60–90min
Pain	++/+++
Position	Prone oblique
Blood loss	Variable, 0–1000mL
Practical techniques	ETT and IPPV

Preoperative

- Usually healthy young and middle-aged adults, but stones may be due to an underlying metabolic problem or due to bladder dysfunction from a neurological disability.
- Check renal function.

Perioperative

- Patient initially in the lithotomy position to insert ureteric stents, then turned semi-prone to place nephrostomy posterolaterally below the 12th rib, under radiographic control—be aware of the potential to dislodge lines and for pressure area damage.
- Consider a reinforced ETT to prevent kinking, and secure well. The head needs to be turned toward the operative side, so it is best to position the ETT in the same side of the mouth.
- Support the chest and pelvis to allow abdominal excursion with ventilation.
- Support and pad the head, arms and lower legs, and pad the eyes.
- Check ventilation during and after position changes.
- Ventilation may need to be temporarily interrupted for radiographs.
- Antibiotic prophylaxis may be required.

Postoperative

- Pain from nephrostomy is variable.
- Multimodal: paracetamol, NSAIDs (check renal function), PCA morphine or PO tramadol.

Special considerations

- Hypothermia can occur if large volumes of irrigation fluid are used.
- Insertion of nephrostomy is often close to the diaphragm, with the possibility of breaching the pleura, causing a pneumothorax or hydrothorax—if in doubt, perform a CXR postoperatively.
- Rupture of the renal pelvis is a recognised complication, when large volumes of irrigant may enter the retroperitoneal space.
- Postoperative Gram-negative septicaemia is a significant risk after any urinary tract surgery for stones (see ➔ pp. 1035–9).

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Extracorporeal shock wave lithotripsy

Procedure	Non-invasive fragmentation of renal stones using pulsed ultrasound
Time	20–40min
Pain	+/++
Blood loss	Nil
Practical techniques	Sedation for adults. GA/LMA for children

In the early days of extracorporeal shock wave lithotripsy, patients were suspended in a water bath in a semi-sitting position, which produced a number of problems for the anaesthetist. Developments in the 1980s meant that a water bath was no longer required, and more recent refinements of the ultrasound beam have made it less uncomfortable, so that, with most current lithotripters, only few patients need anaesthesia or sedation.

Preoperative

- Patients often undergo repeated lithotripsy, so refer to previous treatment records where possible.
- Premedication with paracetamol/NSAIDs (note renal function) is usually effective for treatment.

Perioperative

- Lateral position with arms above the head.
- Renal stones are located using ultrasound or an image intensifier, and the shock wave focused on the stones.
- Antibiotic prophylaxis may be required.

Postoperative

Mild discomfort only; oral analgesics or NSAIDs are adequate.

Special considerations

- Shock wave can cause occasional dysrhythmias, which are usually self-limiting. If persistent, the shock waves can be delivered in time with the ECG (refractory period). Judicious use of anticholinergics (glycopyrronium 200 micrograms) will increase the HR and increase the frequency of delivered shock.
- Pacemakers can be deprogrammed by the shock wave; seek advice from a pacemaker technician.
- Energy from shock waves is released when they meet an air/water interface. It is advisable to use 0.9% sodium chloride, rather than air, for 'loss of resistance' if siting an epidural.

Testicular surgery

Procedure	Removal/biopsy of testis, marsupialisation of hydrocele, vasectomy, testicular torsion
Time	30min to 1h
Pain	++/+++
Position	Supine
Blood loss	Not significant
Practical techniques	GA, LMA, spermatic cord block. RSI/ETT if emergency (e.g. torsion). Spinal or LA infiltration

Preoperative

- Often suitable for day surgery.

Perioperative

- Beware vagal responses—have atropine ready.

Special considerations

- Innervation of testes and scrotum: somatic innervation is via the ilioinguinal, genitofemoral, pudendal and posterior scrotal nerves (branches of the posterior cutaneous nerve of the thigh) with nerve root contributions from L1 to S3. Autonomic innervation is from the sympathetic chain T10–L4 and the parasympathetic plexus S1–S3. Local techniques therefore need to cover T10–S3.
- A spermatic cord block can be used as an adjunct to GA or as part of a local technique for scrotal surgery. The block covers all nerves, except the pudendal and posterior scrotal branches. If used as part of an LA technique, supplemental infiltration of the scrotal skin is also required.
- The spermatic cord is best blocked under direct vision by the surgeon. However, if a local technique is planned, feel for the spermatic cord as it enters the top of the scrotum, and infiltrate 5–10mL of LA around it.

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Renal transplant

Procedure	Transplantation of cadaveric or live donor organ
Time	90–180min
Pain	++/+++
Position	Supine
Blood loss	Not significant to 500mL
Practical techniques	ETT and IPPV, CVP

Preoperative

- Usual problems relate to CKD and uraemia (see pp. 193–5).
- Chronic anaemia is common (Hb usually around 80-100g/L). Do not transfuse to normal levels.
- IHD is common and should be assessed and treated during workup for transplantation.
- There has often been recent haemodialysis. Therefore, some degree of hypovolaemia is common.
- Check post-dialysis bloods, including K^+.

Perioperative

- Avoid A–V fistulae when placing IV cannulae and BP cuff. Avoid using antecubital veins or veins on the radial side of the wrist, if possible (they may be needed for future fistulae).
- Gently fluid-load prior to induction; wide swings in arterial pressure may occur.
- Commonly used agents that are safe in renal failure include fentanyl, remifentanil, propofol, atracurium and sevoflurane.
- Consider a central line under ultrasound guidance and monitor CVP. Avoid the subclavian vein (risk of subclavian stenosis which precludes a future fistula). Consider an arterial line.
- Prior to graft reperfusion, administer fluids to increase CVP to 12–15mmHg and MAP to >80mmHg to optimise perfusion. Ephedrine and/or metaraminol may be used cautiously to increase BP if the patient is optimally filled.
- Maintain normothermia.
- Many centres administer diuretics (e.g. mannitol/furosemide) and immunosuppressants (e.g. methylprednisolone) prior to graft reperfusion. Check local protocols.
- Avoid blood transfusion if possible, although it may be needed if there is surgical bleeding or Hb <80g/L.

Postoperative

- Regular paracetamol and PCA is usually adequate. A fentanyl PCA is preferable. Be cautious with morphine due to reduced clearance and risk of respiratory depression. An epidural/spinal is not usually necessary; there is a risk of bleeding on insertion (residual anticoagulation from haemodialysis, poor platelet function, etc.) and may cause hypotension. Consider a TAP block.
- Avoid NSAIDs.
- Monitor CVP and urine output hourly. Maintain mild hypervolaemia to promote diuresis. Many units have protocols.

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Further reading

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Chapter 30

Gynaecological surgery

Claire Todd

General principles *746*
Evacuation of retained products of conception/suction or vacuum termination of pregnancy *749*
Diagnostic laparoscopy/laparoscopic sterilisation *750*
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Ectopic pregnancy *753*

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General principles

Many gynaecological patients are fit and undergo relatively minor procedures as day cases. Others are inpatients undergoing more major surgery. Elderly patients often require operations to relieve pelvic floor prolapse.

- Many patients are apprehensive, with corresponding higher induction doses required.
- PONV is a particular problem. With high-risk patients, use appropriate techniques; avoid N_2O, give prophylactic antiemetics and consider TIVA.
- Pelvic surgery is associated with DVT. Ensure that adequate prophylactic measures are taken.
- Prophylactic antibiotics reduce postoperative wound infection rates for certain operations. Check your hospital protocol.
- Vagal stimulation may occur during cervical dilation, traction on the pelvic organs or the mesentery or during laparoscopic procedures.
- Take care during patient positioning. Patients are often moved up or down the table, when airway devices can be dislodged and disconnections can occur. Pre-existing back or joint pain may be worsened in the lithotomy position, and if the legs are supported in stirrups, there is a potential for common peroneal nerve injury.
- It may be reasonable to ask the gynaecologist to administer analgesic drugs rectally during anaesthesia. Ensure that you have the patient's permission to do so.
- Ensure patients are kept warm during longer cases.
- Major gynaecological surgery can incur considerable blood loss and may be prolonged. Cell salvage should be available for such cases.
- Many gynaecological operations formerly done through an open approach (e.g. hysterectomy, tubal pregnancy repair) are now done primarily using laparoscopic techniques.

Miscellaneous gynaecological procedures

Table 30.1 presents the important information for those procedures which are not covered in the individual topics within this chapter.

Table 30.1 Other gynaecological procedures

Operation	Description	Time (min)	Pain	Position	G&S/ X-match	Notes
Colpo-suspension	Abdominal procedure for stress incontinence	40	+++	Supine	Nil	ETT, IPPV
Cone biopsy/ LLETZ*	Removal of the terminal part of the cervix through the vagina	30	++	Litho-tomy	Nil	May bleed post-operatively. LMA, SV
Hystero-scopy	Inspection of endometrial cavity Biopsy ± removal of polyps	20	+	Litho-tomy	Nil	LMA, SV
Laparotomy, investigative	Abdominal assessment of pelvic mass	120	++++	Supine	2units	Ovarian tumours may be adherent to adjacent structures. Potentially large blood loss ETT, IPPV
Myo-mectomy	Abdominal excision of fibroids from uterus	60	+++	Supine	G&S	Blood loss may be greater than expected. ETT, IPPV
Oophorec-tomy	Removal of ovaries	40	+++	Supine	Nil	ETT, IPPV
Repair, anterior	Repair of anterior vaginal wall	20	++	Litho-tomy	Nil	Often combined with vaginal hysterectomy. LMA, SV
Repair, posterior	Repair of posterior vaginal wall	20	++	Litho-tomy	Nil	Often combined with vaginal hysterectomy LMA, SV
Sacro-colpopexy	Abdominal repair of vault prolapse	60	+++	Supine	G&S	ETT, IPPV
Sacrospinous fixation	Vaginal operation for vault prolapse	40	++	Litho-tomy	Nil	LMA, SV

(Continued)

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Table 30.1 *(Contd.)*

Operation	Description	Time (min)	Pain	Position	G&S/ X-match	Notes
Shirodkar suture	Insertion of suture around cervix to prevent recurrent miscarriage	20	++	Litho-tomy	Nil	May need antacid prophylaxis and ETT (see p. 896)
Thermo-ablation	Thermal obliteration of endometrium	20	++	Litho-tomy	Nil	May require opiods. LMA, SV
TCRE*	Endoscopic resection of endometrium	30	+	Litho-tomy	Nil	Systemic absorption of water may occur from the glycine solution. Treat as for TURP syndrome. LMA, SV
Vulvectomy, simple	Excision of vulva	90	+++	Litho-tomy	G&S	LMA, SV
Vulvectomy, radical	Excision of vulva and lymph nodes	150	++++	Litho-tomy	2units	Consider regional anaesthesia for analgesia. ETT, IPPV

* LLETZ, large loop excision of the transformation zone; TCRE, transcervical resection of endometrium

Evacuation of retained products of conception/suction or vacuum termination of pregnancy

Procedure	ERPC; STOP/VTOP
Time	10–20min
Pain	+
Position	Lithotomy
Blood loss	Usually minimal
Practical techniques	LMA, SV, day case

Preoperative

- ERPC: remaining products of conception may have to be surgically removed after an incomplete miscarriage. This usually occurs between 6w and 12w gestation. Substantial blood loss may have occurred preoperatively and may continue perioperatively. IV access and crystalloid infusion are required if the haemorrhage appears anything more than trivial.
- Suction or vacuum termination of pregnancy (STOP/VTOP) is a procedure undertaken at up to 12w gestation.

Perioperative

- LMA or face mask. Intubate unfasted emergency patients. Avoid high concentrations of volatile agents due to the relaxant effect on the uterus. Propofol induction followed by intermittent boluses or propofol TCI and an opioid (alfentanil or fentanyl) is appropriate.
- A drug to help contract the uterus and reduce bleeding may be requested. Oxytocin 3–5 units can be given slowly.

Postoperative

Oral analgesics and antiemetics.

Special considerations

- Pregnancies beyond 12w can be terminated surgically by dilation and evacuation. The procedure is similar to a STOP/VTOP, but there is greater potential for blood loss.
- If there are symptoms of reflux oesophagitis, ranitidine or PPI premedication and intubation are indicated.
- If a pregnancy has gone beyond 16w, it may be terminated medically with prostaglandin. These patients may still require an ERPC and are more likely to suffer significant blood loss.

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Diagnostic laparoscopy/laparoscopic sterilisation

Procedure	Intra-abdominal examination of gynaecological organs ± treatment ± clips to Fallopian tubes
Time	30–60min
Pain	+/++
Position	Lithotomy
Blood loss	Nil
Practical techniques	LMA/ETT, IPPV, day case

Preoperative

- Usually young and fit. Give oral analgesics preoperatively.

Perioperative

- Use a short-acting opioid (e.g. fentanyl).
- Administer antiemetics to ensure same-day discharge.
- Encourage infiltration of the skin incisions with LA.
- The traditional airway management is endotracheal intubation with minimal doses of muscle relaxants, as cases are generally quick. Monitor with a nerve stimulator, using reversal agents accordingly.
- An alternative technique for uncomplicated short procedures is to use an LMA. This is only suitable for non-obese patients; the potential for gastric regurgitation and aspiration must be assessed carefully. Surgeons may find SV difficult and request paralysis.

Postoperative

Further opioids or antiemetics may be required.

Special considerations

- Bradycardias are common due to vagal stimulation. Atropine should be readily available; ask surgeons to release the pneumoperitoneum.
- Shoulder pain is common postoperatively due to diaphragmatic irritation. Although self-limiting, it can be difficult to treat and is ↓ by expelling as much CO_2 as possible at the end of the procedure.
- If significant endometriosis is found and excised, postoperative pain can be severe and same-day discharge is likely to be difficult.
- Very rarely, CO_2 gas may be inadvertently injected intravascularly, resulting in VAE (see pp. 584–5). This results in V/Q mismatch, with a fall in $ETCO_2$, impaired CO, hypotension, arrhythmias and tachycardia. Alert the surgeon and resuscitate the patient.
- If an LMA is used, consider premedication with PO ranitidine or a PPI, and the use of a device with a gastric channel (e.g. ProSeal™).

Hysterectomy

Procedure	Removal of uterus (may also include ovaries ± Fallopian tubes as bilateral salpingo-oophorectomy)
Time	1–3h, depending on surgical approach
Pain	+ to +++
Position	Supine, head-down or lithotomy
Blood loss	250–1500mL, G&S
Practical techniques	LMA, SV suitable for vaginal hysterectomy ETT, IPPV required for laparoscopic or open cases Spinal diamorphine useful for analgesia

Preoperative

- Indications for hysterectomy vary from vaginal/uterine prolapse, menorrhagia and uterine fibroids to gynaecological malignancies.
- Patients may be anaemic if they have had menorrhagia or postmenopausal bleeding.
- Renal function may be abnormal if an abdominal mass has been compressing the ureters.
- PONV is common.
- Those who have been treated with preoperative chemotherapy to debulk tumours are frequently anaemic, frail and malnourished. They should be considered high-risk patients.
- Cell salvage should be used for all open and radical hysterectomies.
- Ensure prophylaxis for DVT has been initiated.

Perioperative

- Airway management as above.
- Antibiotic prophylaxis is usually required.
- Steep, head-down positioning is required for a laparoscopic approach.
- Blood loss is variable; some hysterectomies bleed more than expected. X-match blood early if bleeding appears to be a problem.
- Heat loss through the abdominal incision can be significant. Use a forced air warming blanket over the upper body during the operation.

Postoperative

- Pain is usually reasonably well controlled with oral analgesics for vaginal and laparoscopic cases. These patients are usually ready for discharge within 48h.
- Open cases will require additional postoperative analgesia. Options include PCAs, rectus sheath catheters and central neuraxial blockade. Spinal anaesthesia with diamorphine is now preferred to epidurals.

Vaginal hysterectomy

- Often supplemented by an anterior ± posterior repair which reduces bladder and bowel prolapse through the vagina. It is usually not possible to remove the Fallopian tubes and ovaries during a vaginal hysterectomy because of the restricted surgical field.

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- Laparoscopically assisted vaginal hysterectomy (LAVH) is designed to enable the uterus, Fallopian tubes and ovaries to be removed through the vagina. The operation begins with a laparoscopy, during which the broad ligament is identified and detached. Once satisfactory mobility of the gynaecological organs has been achieved at laparoscopy, they are removed vaginally.

Total laparoscopic and abdominal hysterectomy

- With advanced laparoscopic techniques, the number of open cases being performed has ↓. They are now normally reserved for advanced malignancies and cases where it is felt that the uterus is too large to remove via a laparoscopic approach.
- Both laparoscopic and open procedures require good muscle relaxation.
- Laparoscopic hysterectomies being performed for cervical or uterine malignancies are increasingly being combined with sentinel lymph node biopsies. Blue dye is injected around the cervix which allows sentinel lymph node sampling intraoperatively. There is a risk of anaphylaxis.
- Radical hysterectomy is undertaken in patients who have cervical and uterine malignancies. In addition to the uterus, the Fallopian tubes, and often the ovaries, are removed and the pelvic lymph nodes are dissected out. These operations take much longer, and there is a potential for substantial blood loss. An arterial line and goal-directed fluid therapy should be considered. Spinal anaesthesia for postoperative pain relief is useful.

Ectopic pregnancy

Procedure	Laparoscopy (less commonly laparotomy) to stop bleeding from ruptured tubal pregnancy
Time	60min
Pain	++/+++ (depending on surgical approach)
Position	Lithotomy
Blood loss	Can be massive, X-match 2 units
Practical techniques	ETT, IPPV

Preoperative

- The presentation is variable. A stable patient may have ill-defined abdominal pain and amenorrhoea; others may present with life-threatening abdominal haemorrhage. At least one large-bore IV cannula should be inserted prior to theatre, and crystalloids or blood products infused, according to the clinical picture.
- FBC, X-match and possibly a clotting screen should be requested on admission.
- Seek help from a 2nd anaesthetist if the patient is unstable.

Perioperative

- RSI
- Careful IV induction as significant blood loss can be concealed.
- Continue IV fluid resuscitation and actively warm the patient.

Postoperative

- Clotting abnormalities are not uncommon if large volumes of blood have been lost. Send a clotting screen or perform point-of-care coagulation testing.
- Analgesia requirements will vary, depending on surgical approach. May require a PCA.

Special considerations

- Patients are young and generally compensate for significant volumes of blood loss, meaning the preoperative clinical picture can be falsely reassuring.
- In most centres, the operation is performed laparoscopically and only converted to a laparotomy if there are complications. Be aware that the pneumoperitoneum may impede venous return, resulting in hypotension.

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Chapter 31

Ear, nose and throat surgery

Grant Turner

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See also

➲ Cleft lip and palate pp. *943–4*

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General principles

Airway problems are the major concern in ENT surgery, related to both the underlying clinical problem and the shared airway.
Presenting pathology may:
- Produce airway obstruction
- Make access difficult or impossible.

Surgeons working in, or close to, the airway can:
- Displace, obstruct or damage airway equipment
- Obscure the anaesthetist's view of the patient
- Limit access for the anaesthetist during operation
- Produce bleeding into the airway (intra- and postoperatively)
- Potentiate postoperative airway swelling/obstruction.

The surgeon and anaesthetist should plan together to use techniques/equipment that provide good conditions for surgery, while maintaining a safe, secure airway. Whenever an airway problem is suspected intraoperatively, correcting it is the first priority, stopping the surgery, if necessary. Other structures around the head are inaccessible during surgery and need protection, especially the eyes. Ensure they are kept closed with appropriate tape, padded as necessary, and that pressure from equipment is prevented, especially for long cases.

Airway/ventilation management

Tracheal tube or laryngeal mask airway
- Traditionally, an ETT has been used for airway protection for the majority of ENT work.
- Preformed RAE tubes provide excellent protection with minimal intrusion into the surgical field.
- An oral, south-facing RAE tube is used for nasal and much oral surgery, although a nasal tube (north-facing) allows better surgical access to the oral cavity.
- An LMA, often of the reinforced flexible type, offers an alternative approach. It provides adequate protection against aspiration of blood or surgical debris and avoids complications of tracheal intubation/extubation. It restricts surgical access to a greater degree, however, and is more prone to displacement during surgery (with potentially catastrophic results).

Spontaneous ventilation or intermittent positive pressure ventilation
- Continuous NMB is not required for most ENT surgery and facial nerve monitoring is commonly utilised.
- Ventilator advances mean many ENT anaesthetists favour SV or pressure-supported SV. This allows titration of analgesia and improves success for deep extubation techniques.
- The use of remifentanil/opioid infusions to optimise surgical conditions (and avoid NMBs) commonly requires IPPV through either ETTs or LMAs.
- Spontaneously ventilating patients with an IV anaesthetic technique is now commonplace. Multiple induction techniques are described.

Deep or light extubation

- Many ENT procedures create bleeding into the airway. Suction (and pack removal) under direct vision before extubation is essential in such cases, taking care not to traumatise any surgical sites.
- One particular danger site for blood accumulation is the nasopharynx behind the soft palate, an area not readily visible. Blood pooling here can be aspirated following extubation, with fatal results ('coroner's clot'). It is best cleared using a nasal suction catheter or a Yankauer sucker rotated so its angled tip is placed behind the uvula.
- Excessive coughing may exacerbate bleeding risk and contributes to extubation technique selection.
- Laryngospasm can follow extubation, particularly in children, from recent instrumentation of the larynx or irritation by blood. The risk is minimised by extubating either deep or light (not in between).

Deep extubation

- Deep extubation is best suited to SV/supported SV. At the end of surgery, continue, or even increase, the anaesthetic agent concentration, but change gases to 100% O_2 to increase the FRC store. After careful suction, insert a Guedel airway; turn the patient left lateral/head-down (tonsil position); check respiration is regular (turning can produce transient coughing/breath-holding), then extubate.
- Check airway/respiration are intact, and keep the patient in this position until airway reflexes return. Since the patient remains unconscious initially in recovery, care from appropriately skilled recovery staff is essential, with an anaesthetist immediately available in case of airway complications.

Light extubation

- Light extubation is recommended in all patients with a difficult airway or significant respiratory compromise.
- After careful suctioning, any residual NMB is reversed, anaesthetic agents discontinued and the trachea extubated after laryngeal reflexes have returned.
- Light extubation often produces a brief period of coughing/restlessness initially. This is less frequent with the use of opioids.

Throat packs

- A throat pack[1] (wet gauze or tampon) is often used around the ETT/LMA to absorb blood that might otherwise pool in the upper airway.
- It is particularly useful during nasal operations (where bleeding can be substantial and is not cleared during surgery) and during dental surgery.
- The pack must be removed before extubation, as it can lead to catastrophic airway obstruction if left. Recognised systems to ensure removal include:
  - Tying or taping the pack to the ETT
  - Placing an identification sticker on the ETT or patient's forehead
  - Including the pack in the scrub nurse's count
  - Standardising whether removal of the throat pack at the end of surgery is a surgical or an anaesthetic task, but always performing direct laryngoscopy prior to extubation regardless.

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Nasal vasoconstrictors

- Topical vasoconstrictors are routinely used to reduce bleeding in nasal surgery, administered by spray, gel or soaked swabs. Cocaine-containing solutions (e.g. Moffett's solution: cocaine, sodium bicarbonate and adrenaline) are declining in usage, largely due to concerns over drug storage/availability and systemic toxicity (hypertension, arrhythmias, euphoria). Maximum topical cocaine dose is 3mg/kg.
- Proprietary decongestants are a commonly used alternative such as pseudoephedrine or phenylephrine, which are equally effective.
- Systemic absorption can result in a transient sympathomimetic response.
- Infiltration with adrenaline-containing solutions may be used in addition, with greater risk of systemic effects.

Remifentanil

- The intense opioid action of remifentanil, combined with its rapid recovery profile, has led to its widespread use in ENT, particularly for major cases.
- Normally given by infusion, clinical applications include:
  - Middle ear surgery/major head and neck resections (controlled arterial pressure reduces bleeding)
  - Parotidectomy (facilitates IPPV without relaxant)
  - Laryngoscopy/pharyngoscopy (attenuates hypertensive response)
  - Procedures requiring nerve monitoring (NMBs contraindicated).
- Beware of bradycardia/hypotension when used at induction, particularly in the elderly.
- Interpatient variability greatly limits the value of predetermined infusion schemes.
- For major surgery, to prevent postoperative rebound hypertension/agitation in recovery, continue remifentanil at a low infusion rate or give longer-acting opioid (morphine) 15–20min before the end of surgery; clonidine up to 2 micrograms/kg IV or dexmedetomidine may also be of use.

Miscellaneous ear, nose and throat procedures

Table 31.1 presents the important information for those procedures which are not covered in the individual topics within this chapter.

References

1 NHS Improvement (2018). *Recommendations from National Patient Safety Agency alerts that remain relevant to the Never Events list 2018*. https://improvement.nhs.uk/documents/2267/Recommendations_from_NPSA_alerts_that_remain_relevant_to_NEs_FINAL.pdf

Table 31.1 Miscellaneous ENT procedures

Operation	Description	Time (min)	Pain	Position	Blood loss	Notes
Mastoidectomy	Clearance of cholesteatoma from mastoid cavity	90–120	++	Head-up tilt, head tilted to side on ring	Minimal	RAE tube or LMA, SV or IPPV. Bloodless field needed (see stapedectomy). If disease close to facial nerve, surgeon may request no relaxant used (see parotidectomy)
Drilling of ear exostoses	Excision of external auditory ('swimmer's') exostoses	60–90	++	Head-up tilt, head tilted to side on ring	Minimal	RAE tube or LMA, SV or IPPV
Bone-anchored hearing aid (BAHA)	Application of BAHA	90–120	++	Head-up tilt, head tilted to side on ring	Minimal	LA plus sedation or GA with RAE tube or LMA, SV or IPPV
Cochlear implant	Insertion of cochlear implant	120–180	++	Head-up, head tilted to side on head ring		GA, ETT/LMA, nerve monitoring, microscopic surgery, vertigo/PONV risk
Manipulation under anaesthesia (MUA) nose	Correction of nasal fracture	1–15	+	Supine	Small	If quick, preoxygenate plus propofol only. If longer, RAE tube or reinforced LMA plus throat pack. Occasionally bleeds dramatically
Removal of foreign body from nose	Removal of foreign body from nose, usually in child	5–10	Nil	Supine, head ring	Nil	Gas induction, RAE tube or LMA, throat pack, SV. Avoid positive pressure face mask ventilation if possible (risk of pushing foreign body down into lower airway)

(Continued)

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Table 31.1 *(Contd.)*

Operation	Description	Time (min)	Pain	Position	Blood loss	Notes
Rhinoplasty	Cosmetic alteration or reconstruction of nose using bone/cartilage graft	60–90	++	Head-up tilt, head ring	Small	RAE tube or reinforced LMA, SV or IPPV, throat pack. Moderate hypotension useful to decrease bleeding: remifentanil ideal
Lateral rhinotomy	Resection of nasal tumour via lateral rhinotomy	90	++	Head-up tilt, head ring	Moderate	RAE tube or reinforced LMA, SV or IPPV, throat pack. Moderate hypotension useful to decrease bleeding
Uvulopalato-pharyngoplasty (UPPP)	Excision of uvula and lax tissue from soft palate, sometimes using laser	20–30	+++	Supine, pad under shoulders	Small	RAE tube or reinforced LMA, SV or IPPV. Laser-proof tube if needed. Regular postop NSAID plus paracetamol. OSA precautions if indicated
Submandibular gland excision	Excision of blocked/diseased submandibular gland	45–60	++	Supine, pad under shoulders, head ring	Small	RAE tube or reinforced LMA on opposite side, SV or IPPV
Tracheobronchial foreign body removal	Removal of inhaled foreign body using rigid bronchoscope, usually in child (see also p. 947)	20–30	Nil	Supine, pad under shoulders	Nil	Deep inhalational anaesthesia using O_2 and sevoflurane, allowing surgeon intermittent access. LA spray. Glycopyrronium useful to prevent bradycardia and dry secretions, improving surgical view
Laryngoscopy in child	Examination of larynx in child, usually for recurrent stridor or aspiration	15–20	Nil	Supine, pad under shoulders	Nil	Inhalational induction, LA spray to larynx. Either SV via rigid surgical laryngoscope (circuit connected to scope) or LMA and fibreoptic laryngoscopy through it (ideal for small child and enables larynx to be viewed during emergence)

Direct pharyngoscopy	Examination of pharynx using rigid pharyngoscope	10–15	+	Supine, pad under shoulders	Nil	Check for reflux. Small (6–7) oral RAE tube secured on left, IPPV, mivacurium or intermittent suxamethonium. Risk of bleeding if biopsies done
Endoscopic stapling of pharyngeal pouch	Division of opening to pharyngeal pouch using staple gun endoscopically	15–20	+	Supine, pad under shoulders	Nil	Preoxygenate, avoid face mask ventilation, small (6–7) oral RAE tube secured on opposite side, IPPV. NGT at end and IV fluids as nil by mouth postoperatively
Excision of pharyngeal pouch	Excision of pharyngeal pouch via external approach	45–60	+	Supine, pad under shoulders, head ring	Nil	Preoxygenate, avoid face mask ventilation, small (6–7) oral RAE tube secured on opposite side, IPPV. Surgeon may want oesophageal bougie inserted to help recognise anatomy. Antibiotic cover, NGT at end and IV fluids as nil by mouth postoperatively
Insertion of speaking valve (e.g. Provox®)	Insertion of speaking valve via trache-oesophageal puncture following laryngectomy	15	+	Supine, pad under shoulders, head ring	Nil	Microlaryngoscopy tube inserted via tracheostomy, IPPV, mivacurium or intermittent suxamethonium, remifentanil or alfentanil to reduce CVS response
Pharyngolaryn-gectomy	Extended resection of larynx and pharynx for tumour of hypo-pharynx, using oesophagectomy and stomach pull-up or free jejunum transfer. Involves laparotomy ± thoracotomy	6–10h	++++	Supine, pad under shoulders, head ring	Major, X-match 4-6 units	No access to patient whatsoever! Prepare as for laryngectomy with all lines, plus DLT if doing thoracotomy. Consider epidural analgesia for laparotomy/thoracotomy (using plain LA) with PCA morphine to cover remaining surgical sites. ICU mandatory postoperatively (see also pp. 551–2)

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Preoperative airway obstruction

(See also ➲ pp. 384–7.)

Assessment

- Patients with preoperative airway obstruction usually present for surgery either to establish the diagnosis or to relieve the obstruction.
- Obstructions may be supraglottic, glottic or subglottic. The commonest level for obstruction is the larynx, which classically produces stridor (high-pitched, inspiratory).
- In adults, tumours are the commonest cause of acute upper airway obstruction, though a haematoma or an infection (including epiglottitis) is also possible. In children, an infection (croup) or a foreign body is more likely; in the UK, Hib vaccination has virtually eliminated childhood epiglottitis.
- OSA considerations are addressed elsewhere (see ➲ pp. 73–5).
- Extreme airway obstruction will cause obvious signs of respiratory distress at rest. Exhaustion or an obtunded conscious level are late signs and indicate the need for immediate intervention.
- If the obstruction has a gradual onset, patients can compensate very effectively, and moderately severe obstruction can develop without gross physical signs. Features to help recognise a substantial degree of upper airway obstruction include:
  - Long, slow inspirations, with pauses during speech
  - Recent marked deterioration in exercise tolerance
  - Worsening stridor during sleep (history from spouse).
- Oropharyngeal lesions rarely present with airway obstruction, and assessment is normally straightforward on preoperative examination. Important features are limitation of mouth opening and tongue protrusion, and identification of any masses compromising the airway.
- Useful information may come from radiographs (plain films, CT/MRI) or ENT clinic flexible or indirect laryngoscopy.

Management: assessment and holding measures

For life-threatening airway obstruction, emergency intervention may be needed, but usually surgery will be a planned procedure. Determine how time-critical airway management is. Is there time for further investigations? For emergencies, avoid undue delays. While preparing theatre, consider whether holding measures may be of benefit.

High-flow nasal oxygen

- O_2 delivery via high-flow nasal cannulae has revolutionised airway management—multiple devices, including Optiflow™, provide low-level positive pressure and improve airway patency, improve oxygenation and decrease work of breathing and distress. May be incorporated into awake intubation technique and prolong apnoeic oxygenation.

Steroids

- Useful if associated oedema/inflammation, unlikely to cause harm in acute setting and may be beneficial. Little direct evidence of benefit in adult airway obstruction, but improve symptoms in children with croup and improve laryngeal oedema and post-extubation stridor in adults.[2]

Nebulised adrenaline

- Appears to be useful in reducing stridor associated with various obstructive aetiologies in case reports (better evidence lacking). Widely utilised, 1–5mg of adrenaline nebulised in O_2.

Helium

- Medical Heliox (mixtures may be 21/70 or 30/70 O_2/helium). A similar viscosity to air, but lower density increases tendency to laminar flow and decreases resistance in turbulent flow. Limited by reduced O_2 content of any mixture useful for flow characteristics.

Continuous positive airway pressure

- Splints any collapsing airway segment. Variable availability in emergency setting and high-flow nasal cannulae likely better tolerated.

Investigations

Consider potential benefit of further investigations:

- **Nasendoscopy:** quick and relatively low risk of airway trauma, may offer significant information; anaesthetist should be present if not performing.
- **CT:** rapid and accurate assessment, usually well tolerated, may be performed lateral or even supine if necessary. Reconstructions allow 3D assessment. No information on any dynamic or postural contribution to airway patency.
- **MRI:** gold standard for soft tissue imaging; however, limited availability, prolonged scan times and expected intolerance of supine position with coil over airway render MRI largely impractical in acute setting.

Management: approach to securing airway

The main problems in securing airway access are:

- Airway obstruction likely to be worsened by lying the patient flat, GA (all techniques) or instrumenting the airway (laryngospasm, bleeding).
- Identifying the laryngeal inlet may be difficult because of anatomical distortion (especially supraglottic lesions), secretions or blood.
- Severe stenosis may make passage of the tube difficult (particularly glottic or subglottic tumours).

There is little evidence to support any one particular anaesthetic technique. Patient presentations and anaesthetic experience vary widely. However, the use of IV induction agents and/or NMBAs carries the catastrophic risk of CICO in a patient unable to breathe spontaneously. Advances in O_2 delivery techniques and VL are transforming planned approaches to the obstructed airway, and high-flow nasal cannulae may augment any chosen technique and offer ongoing oxygenation and potentially provide some airway splinting during securing of the airway.

Traditionally, the three main options for establishing secure access in an obstructed airway are:

- Direct laryngoscopy and intubation under deep inhalational anaesthesia (SV): increasing consideration of SV under IV anaesthesia.
- AFOI under LA
- Tracheostomy under LA (or deep inhalational GA with face mask or LMA in less severe cases).

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Whichever technique is used, a full range of equipment should be prepared, including different laryngoscopes, cricothyroidotomy kit and tubes in various sizes. A small ETT kept in ice will be stiffer, useful to get past a tight stenotic lesion.

- AFOI under LA is generally most useful for supraglottic lesions where anatomical orientation is the main problem; for stenotic lesions of the glottis/subglottis, the scope may block the airway completely.
- Deep inhalational anaesthesia, best with sevoflurane in O_2, may be slow because of reduced minute ventilation. A moderate degree of CPAP is effective at keeping the airway patent, as anaesthesia deepens. Once deep, LA spray to the larynx extends the available time for laryngoscopy before reflexes return.
- In children, deep inhalational anaesthesia is the only realistic option; best undertaken with the child sitting, comforted by a parent, and may be safer to site IV cannula after induction in small children to minimise upset. Avoid delays because of rapid and unpredictable decline in condition.
- In childhood epiglottitis, distortion of the epiglottis can make recognition of the glottis very difficult; a useful tip is to press on the child's chest and watch for a bubble of gas emerging from the larynx.
- Mason and Fielder[3] reviewed the merits of each technique for airway obstruction at different levels but concluded none is universally certain, safe and easy, and the final decision in each case will be strongly influenced by the particular skills and experience of the anaesthetist and surgeon concerned.
- If complete airway obstruction occurs and all conventional attempts to secure the airway fail, emergency surgical access to the airway is the only option. Surgical cricothyroidotomy is preferable to tracheostomy for emergency airway access, as it is quicker to perform, more superficial and less likely to bleed (above the thyroid gland) (see ➔ p. 381).

References

2 Fan T, Wang G, Mao B, *et al*. (2008). Prophylactic administration of parenteral steroids for preventing airway complications after extubation in adults: meta-analysis of randomised placebo-controlled trials. *BMJ*, **20**, a1841.

3 Mason RA, Fielder CP (1999). The obstructed airway in head and neck surgery. *Anaesthesia*, **54**, 625–8.

Jet ventilation

Jet ventilation describes the delivery of a jet stream of gas from a high-pressure source, delivered into an open airway and generally relying on passive exhalation. Jet ventilation carries significant risks, ↑ by unfamiliarity with the technique. Jet delivery via misplaced/misdirected cannula can have catastrophic consequences. Inadequate gas exchange is common.

- Low-frequency jet ventilation, usually achieved using an injector system, such as the manually driven adjustable-flow Manujet®, but also describes rescue techniques for CICO scenarios utilising more simply constructed systems. This may be connected via:
  - Cricothyroidotomy needle/cannula placed through the cricothyroid membrane under LA before induction and aimed towards the carina. Commercial versions available or a Tuohy needle can be used.
  - Jet catheter (rigid, non-compliant) with the tip placed midway down the trachea. Multiple catheters available with gas sampling/pressure monitoring port or made from laser-resistant material.
  - Injector needle attached to the proximal end of the operating laryngoscope/rigid bronchoscope, and ventilation started when correctly aligned with larynx/trachea. Various needle sizes available with different flow rates. Technique not suitable if good view of larynx is unobtainable and has disadvantage of blowing debris/smoke into trachea with ventilation.
- High-frequency jet ventilation requires specialised ventilators capable of performing high frequencies (typically 1–10Hz) and experience with the technique. Warmed and humidified inspiratory jets are electronically controlled. Air is entrained along with inspiratory jet but smaller VTs utilised. Frequency, driving pressure, inspiratory time and gas composition are usually adjustable; however, the degree of air entrainment means delivered FiO_2 may vary.
  - Most modern systems monitor airway pressures and feature cut-offs and improved safety features. Inspiratory pauses are required for more accurate CO_2 sampling.
- Indications for jet ventilation importantly include:
  - Emergency airway management via a transtracheal/cricothyroid membrane cannula
  - Airway surgery (including laser) for laryngeal/tracheal stenosis, major conducting airway surgery
  - Thoracic surgery, including non-dependent lung ventilation during lung isolation techniques.
  - Lung-protective ventilation strategies/rescue in ICU.

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Grommet insertion

Procedure	Myringotomy and grommet insertion, usually bilateral
Time	5–15min
Pain	+
Position	Supine, head tilted to side, head ring
Blood loss	Nil
Practical techniques	Face mask or LMA, SV using T-piece or paediatric circle

Preoperative

- Usually children (1–8y), normally day case.
- Repeated ear infections; check for recent URTI.
- Paracetamol/NSAID PO.

Perioperative

- LMA commonly used.
- Face mask suitable if surgeon happy to work round it, but assistant needed to adjust vaporiser, etc. Insert Guedel airway before draping, and ensure reservoir bag visible throughout (T-piece ideal if face mask used).

Postoperative

- Need for additional analgesia unlikely.

Special considerations

- If face mask airway difficult, change early to LMA.
- Reflex bradycardia occasionally seen related to partial vagal innervation of tympanic membrane.

Tonsillectomy/adenoidectomy: child

Procedure	Excision of lymphoid tissue from oropharynx (tonsils) or nasopharynx (adenoids)
Time	20–30min
Pain	+++
Position	Supine, pad under shoulders
Blood loss	Usually small, can bleed postop
Practical techniques	South-facing RAE tube or reinforced LMA, placed in groove of split blade of Boyle–Davis gag; SV or IPPV

Preoperative

- Careful history to exclude/define OSA or active infection. Consider overnight bed if history/sleep study indicates moderate/severe OSA.
- Topical LA on hands (mark sites of veins).
- Consider paracetamol/NSAID PO.
- Consent for PR analgesia if to be used.

Perioperative

- IV or inhalational induction (sevoflurane ± N_2O): oropharyngeal airway useful if nasopharynx blocked by large adenoids.
- Intubate (RAE) using relaxant or deep inhalational/IV anaesthesia, or insert LMA using propofol/opioid or deep inhalational anaesthesia.
- Secure in midline, no pack (obscures surgical field).
- Beware surgeon displacing/obstructing tube intraoperatively, particularly after insertion or opening of Boyle–Davis gag.
- T-piece ideal for SV, but ensure reservoir bag always visible.
- Reliable IV access essential, IV fluids routine.
- Analgesia with morphine or fentanyl titrated IV plus paracetamol/NSAID (if not given preoperatively). Consider tramadol.
- Antiemetic: at least one recommended—dexamethasone or ondansetron.
- Careful suction of oropharynx and nasopharynx at end under direct vision (generally done by surgeon).
- Extubate left lateral/head-down (tonsil position), with oropharyngeal airway. Refer to deep or light extubation discussion (see ➔ p. 757).

Postoperative

- Keep patient in tonsil position until airway reflexes return.
- High-quality recovery care essential.
- Analgesia with IV morphine/fentanyl initially, then PO paracetamol/NSAID/morphine. Dexmedetomidine has been used.
- Leave IV cannula (flushed) in place in case of bleeding.

Special considerations

- In small children, a pillow/roll under the shoulders can be used to provide the necessary tilt.
- Avoid blind pharyngeal suction with a rigid sucker, as this may start bleeding from the tonsil bed.

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- Best available evidence unable to demonstrate NSAIDs increase bleeding risk.[4] Heterogeneity of drug/timing/surgical technique noted.
- LA infiltration of the tonsil bed is not recommended.
- Beware continual swallowing in recovery, a sign of bleeding from the tonsil/adenoid bed.
- Adenoidectomy/tonsillectomy now widely done as a day case with an extended (5–6h) postoperative stay; morphine still suitable for analgesia.
- Concern about possible transmission of vCJD via contaminated equipment used in adenotonsillectomy led to a single-use policy in the UK in 2001, which was subsequently lifted in the light of further data.

Bleeding after adenotonsillectomy

- May be detected in recovery or many hours later.
- Loss may be much greater than readily apparent (swallowed blood).
- Senior anaesthetist must be involved.
- Problems include:
  - Hypovolaemia
  - Risk of aspiration (fresh bleeding and blood in stomach)
  - Difficult laryngoscopy because of blood in the airway or oedema
  - Residual anaesthetic effect.
- Resuscitate preoperatively; check Hb (HemoCue® ideal); X-match, and give blood, as needed. Note: Hb will fall as IV fluids administered (dilution).
- Options:
  - RSI: enables rapid airway protection, but laryngoscopy may be difficult (blood, swelling)—generally preferred
  - Inhalational induction left lateral/head-down: allows time for laryngoscopy but takes longer, and unfamiliar technique to many.
- Use wide-bore gastric tube to empty stomach after bleeding stopped.
- Extubate fully awake.
- Extended stay in recovery for close monitoring.
- Nasopharyngeal pack occasionally needed (secured via tapes through nose) if bleeding from adenoids cannot be controlled. Usually very uncomfortable: patient may need midazolam/morphine to tolerate.
- Check postoperative Hb.

References

4 Lewis SR, Nicholson A, Cardwell ME, *et al*. (2013). Nonsteroidal anti-inflammatory drugs and perioperative bleeding in paediatric tonsillectomy. *Cochrane Database Syst Rev*, **7**, CD003591.

Tonsillectomy in adults

As for child, except:

- Usually more painful postoperatively in adult: give morphine/long-acting opioid in theatre. Consider tramadol.
- IPPV with relaxant technique used more commonly. Deep extubation may be more reliant on airway adjuvant.
- Occasionally, patients present with peritonsillar abscess (quinsy). Now normally treated with antibiotics, and tonsillectomy performed later. If drainage essential because of airway swelling, pus usually aspirated with syringe and large needle under LA infiltration.

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Myringoplasty

Procedure	Reconstruction of perforated tympanic membrane with autograft (usually temporalis fascia)
Time	60–90min
Pain	++
Position	Supine, head tilted to side, head ring, head-up tilt
Blood loss	Minimal
Practical techniques	South-facing RAE tube or LMA (usually reinforced); SV or IPPV

Preoperative

Usually young, fit patients.

Perioperative

- Ensure coughing avoided during surgery; LA spray to larynx; monitor NMB if IPPV–relaxant technique used.
- Dry field improves the surgical view, though not as important as for stapedectomy; head-up tilt and avoiding hypertension/tachycardia normally sufficient.
- Remifentanil infusion suitable.
- Routine antiemetic useful.

Postoperative

- PRN paracetamol or NSAID PO/IV; may need morphine.
- PRN antiemetic.

Special considerations

Using N_2O may produce diffusion into the middle ear and risk the graft lifting off; either avoid or discontinue 20min before the end of the case.

Stapedectomy/tympanoplasty

Procedure	Excision/reconstruction of damaged middle ear structures
Time	2–4h
Pain	++/+++
Position	Supine, head tilted to side, head ring, head-up tilt
Blood loss	Minimal
Practical techniques	South-facing RAE tube or LMA (usually reinforced). IPPV normally. Arterial line often used

Preoperative

- Check for CVS disease, as this will limit the degree of hypotension possible.
- Oral premedication options include benzodiazepines, β-blockers and clonidine.

Perioperative

- If surgical use of nerve integrity monitoring, avoid NMBs.
- Bloodless field enables greater surgical accuracy. Simple measures include: potent opioid preinduction; ensuring coughing avoided at intubation (LA spray to larynx helpful); head-up tilt to reduce VP.
- Further benefit achieved by lowering arterial BP (mean of 50–60mmHg in healthy patients) and HR (<60bpm).
- Remifentanil infusion ideal to achieve this. Alternatively, use IV labetalol (combined α-/β-blocker, 5mg increments) or IV β-blocker (metoprolol 1mg increments, esmolol infusion) plus vasodilator (isoflurane, hydralazine 5mg increments). Arterial line strongly advised with CVS disease or if potent vasodilators used; head-up tilt further reduces perfusion pressure to the brain.
- Give at least one antiemetic routinely. Consider prochlorperazine (discuss vertigo risk with surgeon).

Postoperative

- Regular antiemetic for 24–48h.
- PRN paracetamol or NSAID PO/IV/PR; may need morphine.

Special considerations

- N_2O diffusion into the middle ear may disrupt surgery, though less important than in myringoplasty. Either avoid or discontinue 20min before the end of the case.

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Nasal cavity surgery

Procedure	Submucous resection of septum, septoplasty, turbinectomy, polypectomy, functional endoscopic sinus surgery
Time	20–100min
Pain	++
Position	Supine, head ring, head-up tilt
Blood loss	Usually minor
Practical techniques	South-facing RAE tube or LMA (usually reinforced); SV or IPPV. Throat pack

Preoperative

- Obstructive airways disease often associated with nasal polyps.
- Combination of procedures mentioned in the box above frequently performed.

Perioperative

- Face mask ventilation often needs Guedel airway due to blocked nose.
- Nasal vasoconstrictor usually applied (topical or infiltration).
- Leave eyes untaped for polypectomy (the optic nerve can be close, and the surgeon needs to check for eye movement).
- TIVA and remifentanil ideal for improved surgical conditions; aim to control arterial BP (mean 50–60mmHg in healthy individuals).
- Suction pharynx (particularly behind soft palate for the 'coroner's clot'; see ➲ p. 757) before extubation; less easy with LMA.

Postoperative

- Analgesia with PRN paracetamol or NSAID PO/IV/PR.
- Nose usually packed, producing obstruction of nasal airway; if disturbing to patient, or in cases of OSA, nasopharyngeal airway(s) can be incorporated into the pack.
- Sit patient up as soon as awake to reduce bleeding.

Special considerations

- Leave IV cannula in overnight, as can bleed postoperatively.

Microlaryngoscopy

Procedure	Examination of larynx using operating microscope (plus excision/biopsy; may use laser)
Time	10–30min
Pain	+/++
Position	Supine, pad under shoulders, head extended
Blood loss	Nil
Practical techniques	Microlaryngeal tube and conventional IPPV. TIVA and jet ventilation using injector system (O_2 plus entrained air) via: • Injector needle on the operating laryngoscope • Semi-rigid tracheal catheter • Cricothyroidotomy needle/cannula

Ventilation during microlaryngoscopy

Spontaneous ventilation/apnoeic oxygenation with unsecured airway

- Discuss with surgeon if laryngeal function to be visualised (uncommon); requires SV technique.
- High-flow nasal cannulae and TIVA technique allow uninterrupted surgical access (in appropriate patients). Poor airway protection. If resection to occur, aerosolisation of blood/pathogens is a consideration. Gas blender-compatible equipment may facilitate laser surgery in experienced centres. (See pp. 476–9.)
- Prolonged apnoeic oxygenation with HFNC well described if immobile field essential.[5]
- May be preferred surgical airway technique if posterior lesion.

Microlaryngeal tube and conventional intermittent positive pressure ventilation

- Microlaryngeal tubes are longer ETTs with a small internal and outer diameter and a high-volume/low-pressure cuff, and available typically in sizes 4 and above. They allow greater surgical access than conventional ETTs.
- Enables maintenance of anaesthesia with inhalational agents, conventional IPPV strategies and monitoring of gas exchange and airway pressures.
- Protects against aspiration of blood/surgical debris but obstructs up to one-third of posterior glottis from view.
- Use long, slow inspiration for IPPV because of high resistance of tube. Measured inflation pressure will be high, but the patient's airway pressures distal to tube will be lower.
- Laser-compatible microlaryngeal tubes are available (all with significantly ↑ external diameter).

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Jet ventilation
(See ➲ p. 765.)
- Ventilation achieved using an injector system, and delivered usually via rigid jet catheter.
- Induce in theatre, or use an LMA or a microlaryngeal tube initially; then remove, place jet catheter and commence jet ventilation when surgical team ready.
- Ensure the anaesthetic machine in theatre is situated close to enable easy face mask ventilation at induction/recovery.
- TIVA needed for maintenance (propofol/remifentanil infusion).
- Ventilation techniques vary, depending on device and surgery. Adjust inspiratory flow (alter injector settings, or change needle size) to produce appropriate degree of chest expansion.
- Accurate flow/pressure measurement not easy; barotrauma a potential risk, oxygenation often exceeds CO_2 clearance.
- Stop ventilation intermittently during surgical work (clear communication essential).
- Provides minimal obstruction to surgical view.
- At end of the case, may continue jet ventilation until SV re-established or more commonly discontinue and ventilate by alternate airway until SV recommences.

Preoperative

- Patients often elderly and usually smokers; CVS/respiratory system problems common.
- Carefully assess the airway for evidence of obstruction. History, examination, ENT clinic assessment, plain films and CT scan may all help, but if any degree of stridor present, obstruction must be substantial (see ➲ pp. 384–7).
- Ensure all equipment is ready before induction, including cricothyroidotomy kit, and surgeon is prepared for emergency tracheostomy, if required.

Perioperative

- If airway obstruction suspected, secure airway initially, using principles outlined (see ➲ Emergency management of the obstructed airway, pp. 384–7). Inserting a cricothyroid cannula under LA preinduction provides a route for ventilation in the event of total obstruction. Detailed airway and rescue preplanning essential.
- Give short-acting opioid (alfentanil, remifentanil) to attenuate hypertensive response. High-dose infusion may prevent need for muscle relaxation if surgical field immobility required.
- If muscle relaxation planned, use of rocuronium and reversal with sugammadex may be an option.
- LA spray to larynx reduces risk of laryngospasm, though this impairs airway protection, so recover left lateral, head-down.

Postoperative

- Analgesia with PRN paracetamol or NSAID PO/IV/PR.
- May develop stridor postoperatively from oedema of an already compromised airway. Dexamethasone 8–12mg IV sometimes used to prevent this. Consider nebulised adrenaline.

Special considerations

- Careful planning required if airway laser to be utilised.
- Airway laser surgery carries significant risk of airway fire, as well as collateral tissue damage/injury to both patient and staff. Excellent teamwork and adherence to local health and safety policies essential.
- Microlaryngoscopy can be used to inject inert material (e.g. silicone) into paralysed vocal cords to improve phonation, though this can lead to airway obstruction if overdone.

References

5 Patel A, Nouraei SAR (2015). Transnasal Humidified Rapid Insufflation Ventilatory Exchange (THRIVE): a physiological method of increasing apnoea time in patients with difficult airways. *Anaesthesia*, **70**, 323–9.

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Tracheostomy

Procedure	Insertion of an ETT via neck incision
Time	30min
Pain	++
Position	Supine, pad under shoulders, head ring, head-up tilt
Blood loss	Normally small, though can bleed from thyroid vessels
Practical techniques	IPPV, ETT with tubing going 'north', changed to tracheostomy tube during case. LMA if airway not a problem, IPPV or SV. Can be done under LA

Preoperative

- Normally done for long-term ICU ventilation or airway obstruction.
- ICU patients almost certainly already intubated. If ventilation difficult and oxygenation critical, set up ICU ventilator in theatre, using TIVA, rather than inhalational agents.
- Stop NG feeds, if applicable.
- If tracheostomy is for airway obstruction, secure airway initially, using principles outlined (see Preoperative airway obstruction, pp. 762–4).
- Before induction, ensure all equipment prepared (including cricothyroidotomy kit) and the surgeon ready for emergency tracheostomy, if required.

Perioperative

- Secure ETT with tape to allow easy removal during case, with pilot cuff readily accessible.
- Aspirate NGT (if present), and clear oropharynx of secretions before draping.
- Drape patient to allow anaesthetist access to ETT for tube change.
- Long tubing needed for breathing circuit and gas sampling.
- Before changing to tracheostomy tube, preoxygenate for 3–4min (increasing volatile agent as necessary), and check NMB is adequate.
- Ensure scrub nurse has correct tracheostomy tube and sterile catheter mount.
- Deflate ETT cuff before surgeons incise trachea, so it can be reinflated and ventilation continued if problems occur.
- Withdraw ETT slowly into upper trachea (do not remove from trachea until tracheostomy secure and certain), and connect breathing circuit and capnograph to new tracheostomy tube via sterile catheter mount.
- Beware false passage created during tracheostomy tube insertion, especially in the obese; check position with fibreoptic endoscopy, if any doubt.
- If problems occur, remove tracheostomy tube and advance ETT back down trachea.

Postoperative

- Regular suction to new tracheostomy (blood, secretions).
- Humidify inspired gases.
- Analgesia in recovery with paracetamol or NSAID IV/PR or morphine IV. Usually little analgesia required thereafter.

- A new tracheostomy often produces protracted coughing—morphine, benzodiazepines or low-dose propofol useful for control.
- Antiemetic, as required.
- If tracheostomy tube comes out, reinsertion can be very difficult in first few days; orotracheal intubation often more practical. Two retraction sutures left in tracheal incision are useful for identifying and opening the stoma.

Special considerations

- Can be done under LA, though difficult in a dyspnoeic, struggling patient.
- In ICU, tracheostomy is now usually done percutaneously, using dilational technique; theatre cases are likely to be the difficult ones.
- Tracheostomy is not the ideal route of approach for emergency airway access; cricothyroidotomy is more accessible and less likely to bleed (see ➔ p. 381).
- LMA can be used if tracheostomy is done at start of larger procedure and upper airway normal.

Tracheostomy tubes

- Specific features available include:
  - Inner tube (e.g. Shiley®): permits removal for cleaning
  - Adjustable flange: length can be modified for short trachea or deep stoma
  - Channel in obturator for guide-wire
  - Fenestration: allows speech by patient occluding the lumen with a finger and exhaling through hole in back wall of tube. Alternatively, speaking valves such as the Passy Muir® Valve can be added.
- Tube change:
  - New tube must be inserted with obturator in place to prevent stomal damage.
  - May be difficult to find trachea in new tracheostomy; guide-wire may be useful.
  - Prepare for orotracheal intubation in case of problems.
  - Cannot be left in place longer than 28d (classified as an implant thereafter).

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Laryngectomy

Procedure	Excision of larynx (epiglottis and glottis) with creation of an end-stomal tracheostomy
Time	3–4h
Pain	+++
Position	Supine, pad under shoulders, head ring, head-up tilt
Blood loss	Moderate to substantial; X-match 2 units
Practical techniques	IPPV, ETT with tubing going 'north', changed to tracheostomy during case Arterial line, urinary catheter, CVP line for long/complicated surgery or if indicated by cardiac disease

Preoperative

- Some degree of airway obstruction likely. Patient likely to have had recent GA (for diagnosis) to guide airway management; beware if some time has elapsed.
- If no recent GA, assess the airway as for microlaryngoscopy (see pp. 773–5).
- Usually smokers; CVS/respiratory system problems and malnutrition common.
- Discuss implications of tracheostomy preoperatively (communication, secretions, coughing produced by tube). Speech therapist will do much of this.

Perioperative

- Insert fine-bore NG feeding tube at induction, and fix securely (can be sutured to nasal septum).
- Warming blanket and fluid warmer.
- Long tubing needed for breathing circuit and gas sampling tube.
- Remifentanil infusion ideal.
- Substantial blood loss can accumulate under drapes at back of neck and may not be apparent until end of case.
- For CVP access, all neck lines hinder surgery; femoral best, though antecubital fossa (ACF) or subclavian can be used.
- Antibiotic prophylaxis for at least 24h.
- When changing to tracheostomy tube, see precautions for tracheostomy (see pp. 776–7), though end-stoma makes tracheal access safer and easier.
- During surgery, long tube (armoured or special preformed) via tracheostomy is useful to enable surgical access round stoma, then changed for standard tracheostomy tube at end.

Postoperative

- HDU ideal.
- Humidification and regular suction essential (blood, secretions).
- New tracheostomy produces protracted coughing—morphine, benzodiazepines or low-dose propofol useful for control.
- Analgesia with PRN morphine IV/NG, plus PRN paracetamol or NSAID NG/IV/PR. Analgesic requirements usually surprisingly low.
- Antiemetic, as required.

Special considerations

- Beware of air emboli (see ➲ pp. 584–5) during dissection—early detection by sudden fall in $ETCO_2$.
- For previous laryngectomy patients presenting for surgery, to ventilate via stoma, use paediatric face mask turned through 180°, LMA applied to neck or intubate awake after LA spray to stoma. Tracheostomy tube insertion is usually easy, though check stoma for stenosis or tumour recurrence, and always preoxygenate.
- Partial laryngectomy, with laryngeal reconstruction and temporary tracheostomy, favoured by some as alternative to radiotherapy in early laryngeal tumours.

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Radical neck dissection

Procedure	Excision of sternomastoid, internal and external jugular veins and associated lymph nodes. Modified or selective neck dissection preserves some of these structures (notably internal jugular vein)
Time	2–4h
Pain	+ (selective neck dissection +++)
Position	Supine, pad under shoulders, head on ring tilted to side, head-up tilt
Blood loss	Moderate to substantial, X-match 2–4 units
Practical techniques	IPPV, ETT with tubing going 'north' or south-facing RAE tube on opposite side. Arterial line, urinary catheter, CVP line if surgery likely to be long/complicated or with cardiac disease

Preoperative

- Assess airway carefully, as may be an associated head and neck tumour or previous major surgery.
- May be performed with another procedure, e.g. laryngectomy.

Perioperative

- Forced air warming blanket and fluid warmer.
- Long tubing is needed for the breathing circuit and gas sampling.
- Remifentanil infusion ideal.
- Can bleed briskly from large neck vessels, with substantial accumulation of blood under drapes (may not be apparent until end of case).
- For CVP access, femoral is best. Must avoid remaining jugulars, as head and neck venous drainage dependent on them.

Postoperative

- Head and neck oedema likely for several days (impaired venous drainage). Keep head up as much as possible, and avoid excessive IV fluids.
- To reduce chance of agitation/rebound hypertension and wound haematoma in recovery, continue remifentanil at a low infusion rate, or give morphine 15–20min before end of surgery; clonidine up to 2 micrograms/kg IV or dexmedetomidine also very useful. Treat any hypertension early.
- Analgesia with PRN paracetamol or NSAID PO/IV/PR, morphine PO/IV. Surprisingly low analgesic requirements normally.
- Antiemetic, as required.

Special considerations

- Beware of air emboli during dissection—early detection by sudden fall in $ETCO_2$ (see ➔ pp. 584–5).
- Surgical manipulation of carotid sinus can produce marked bradycardia.
- If neck dissection previously done on other side, oedema is usually worse and can raise ICP. Dexamethasone 8–12mg IV preoperatively (then 4mg IV 6-hourly) is used by many to reduce this.

Parotidectomy

Procedure	Excision of parotid gland, usually preserving facial nerve
Time	2–5h
Pain	++/+++
Position	Supine, head ring, head tilted to side and moderately extended, head-up tilt
Blood loss	Small/moderate, G&S. Greater for malignancy
Practical techniques	South-facing RAE tube and IPPV normally used, though SV possible for suitable patients. Reinforced LMA and IPPV or SV also possible. No NMB during dissection around facial nerve

Preoperative

- Check if suitable for SV—not if elderly, obese or respiratory disease.
- Check mouth opening, especially if malignant.

Perioperative

- Forced air warming blanket, fluid warmer ± urinary catheter.
- Avoid NMB after initial dose (check recovery with PNS) to allow surgical testing for facial nerve.
- Remifentanil infusion ideal to allow IPPV without NMB and also reduce blood loss.
- Alternatively, suppress respiratory drive with other opioid, volatile agent or propofol infusion, plus moderate hyperventilation.
- LA spray to larynx useful to prevent coughing.
- If SV used, ensure patient settled initially using high level of volatile agent.

Postoperative

- To reduce chance of agitation/rebound hypertension and wound haematoma in recovery, continue remifentanil at a low infusion rate or give morphine 15–20min before end of surgery; keep head up, and treat hypertension early; clonidine up to 2 micrograms/kg IV is very useful.
- Antiemetic, as required.
- Analgesia with PRN morphine IV/PO, paracetamol or NSAID PO/IV/PR.

Special considerations

- Surgeon normally uses nerve stimulator to identify facial nerve during dissection and may wish to leave ipsilateral eye exposed to monitor response.
- Large-bore IV access at start, as occasionally bleeds substantially (especially malignant tumours).

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Chapter 32

Maxillofacial and dental surgery

Alastair Martin and **John Bowden**

Oral/maxillofacial surgery *784*
Fractures of the orbitozygomatic complex *786*
Mandibular fractures *787*
Maxillary/mandibular osteotomy *788*
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See also

➲ Radical neck dissection p. *780*
➲ Parotidectomy p. *781*
➲ Tracheostomy pp. *776–7*

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Oral/maxillofacial surgery

General principles

Anaesthesia for intraoral/maxillofacial procedures requires management of a shared (often difficult) airway. Nasal intubation is frequently used to improve surgical access to the mouth, facilitate X-rays and allow assessment of bite alignment. At the preoperative visit, check nostril patency and ask about epistaxis and the use of anticoagulants. Discuss the choice of airway with the surgeon.

- Simple intraoral procedures are usually possible using a reinforced LMA. For unilateral intraoral procedures, an oral ETT (e.g. RAE tube) placed on the opposite side of the mouth may be acceptable. Oral airways may be dislodged, and vigilance is required (particularly LMAs which tend to obstruct as they migrate forwards).
- If the nasal route is chosen for intubation, use LA and/or a vasoconstrictor mixture (lidocaine 5%/phenylephrine 0.5% or xylometazoline). There are many varieties of nasal tube; the 'Ivory Preformed North-Facing Nasal' from Portex® is ideal. These preshaped tubes are made of soft material and cause little nasal trauma. Sizes of 6.0, 6.5 and 7.0mm should be available. Place in warm water before use to soften the material even further. The tube should be padded with gauze to protect the patient's forehead. Consider fixing the ETT, NGT and temperature probe with clear adhesive film. Avoid excessive tension/pressure on the alar margin which risks causing necrosis. The surgeon will need to recheck periodically.
- Patients who have had previous surgery and/or radiotherapy may have thick, fixed ('woody') soft tissues and poor neck mobility. Intubation may be harder than predicted by bedside tests. Consider VL techniques (e.g. CMAC®/Glidescope® or fibreoptic intubation).
- Protect the eyes with tape and eye pads or surgically positioned plastic contact lenses (e.g. the Crouch Corneal Protector®).
- Position the patient with the head at the opposite end to the anaesthetic machine. A long breathing circuit and gas analysis/spirometry lines are normally required. Secure the breathing circuit with a tube holder. Ensure the pilot cuff is accessible and clear from the eyes.
- Stabilise the head with a horseshoe or head ring. For operations on the roof of the mouth, use a bolster under the shoulders to extend the neck further. Positioning the patient slightly head-up will reduce bleeding.
- Routine use of throat packs is not recommended. If they are used, they should be placed by the surgeon and a robust system should be in place to ensure that they are not inadvertently left *in situ*. This should include:
  - Discussion at 'Time Out'
  - A visual reminder (e.g. throat pack sticker and an entry on the swab/sharps board)
  - Confirmation of removal in the 'Sign Out' swab count
  - Clear documentation.

Extubation

- There is a risk of aspiration of blood, pus and debris. The oropharynx and larynx should be suctioned at the end of the case (preferably under direct vision). Patients should be extubated sitting at 30–45° to reduce bleeding from venous congestion (or in the left lateral position with head-down tilt if there is a high risk of airway soiling).
- Some anaesthetists extubate the patient using a 'deep', spontaneous breathing technique. Some exchange the ETT for an LMA and allow the patient to wake slowly in recovery. Others prefer to extubate awake.
- Whichever technique is used, the aim is to avoid bleeding and swelling caused by coughing and straining. The use of a nasotracheal tube or LMA, which do not stimulate the gag reflex as much as an oral tube, facilitates a smoother extubation. Consider also spraying the vocal cords with lidocaine at intubation.
- If a nasal tube has been used, it is possible to convert it into a nasopharyngeal airway by withdrawing it until the tip lies in the oropharynx, inserting a safety pin (to prevent the tube from slipping back into the nostril) and cutting at the 15cm mark.

ALGRAWANY

Fractures of the orbitozygomatic complex

Procedure	Elevation of fractured zygomatic complex ± fixation
Time	10–180min
Pain	+/++
Position	Supine, with head-up tilt, head ring
Blood loss	Minor (may be more with internal fixation)
Practical techniques	Oral RAE tube and IPPV. LMA/SV for simple elevation

Fractures may occur in isolation or be associated with damage to other parts of the facial skeleton. Interference with movement of the coronoid process of the mandible by the depressed zygomatic complex may limit mouth opening. Following elevation, the fracture may be stable or unstable and require internal fixation. Unstable fractures require plating or wiring via skin or intraoral incisions.

Preoperative

- Assess for associated injuries, particularly head and neck injuries. Treatment of these fractures does not have high clinical priority (unless there is ocular compromise from retrobulbar haemorrhage). The operation is often easier 5–7d later when facial swelling has reduced.
- Make a careful airway assessment.

Perioperative

- Intubate the patient with an oral RAE tube. For simple fracture elevations, a flexible LMA may be used, but discuss with the surgeon whether open fixation of the fracture is planned.
- Lubricate and protect the eye on the non-operative side.
- Give antibiotics according to local protocols if metalwork is to be inserted (e.g. co-amoxiclav 1.2g or clindamycin 600mg), and steroids (e.g. dexamethasone 6.6mg IV) as requested.
- Be prepared for potential bradycardia (vagally mediated) when the zygoma fracture is reduced (Gillies' lift).
- Extubate with the patient breathing spontaneously. Do not apply excessive pressure over the zygoma with the face mask post-extubation.

Postoperative

- IV opioids may be required in recovery.
- Prescribe oral analgesia for the ward.
- Eye observations in recovery and on the ward to detect postoperative retrobulbar haemorrhage (which would require a return to theatre).

Mandibular fractures

Procedure	Reduction and fixation of a fractured mandible
Time	1–3h
Pain	+
Position	Supine, with head-up tilt, head ring
Blood loss	Variable.G&S usually not required
Practical techniques	Nasal tube and IPPV. Fibreoptic intubation may be required

Mandibular fractures can be treated by closed reduction and indirect skeletal fixation (using interdental wires, arch bars or splints) or open reduction and direct skeletal fixation using bone plates. Direct skeletal fixation is more common. Indirect skeletal fixation is now rarely used, when the patient's teeth may be wired together at the completion of surgery. On occasion, external fixation may be required.

Preoperative

- Ensure careful assessment for associated injuries.
- Make a meticulous assessment of the airway. There may be severe trismus and marked soft tissue swelling.
- Assess nostril patency. Check for evidence of basal skull fracture and CSF leak, as these contraindicate nasal intubation and require discussion with a specialist neurosurgical centre.

Perioperative

- Nasal intubation, tracheostomy or submental intubation. The surgeons cannot work around an oral airway.
- Acute trismus makes intubation look potentially difficult preoperatively, as mouth opening may be markedly limited, but this tends to relax following induction. Patients with older fractures and those complicated by infection tend not to relax as much.
- Marked swelling may make intubation more difficult, and an AFOI may occasionally be required.
- Bilateral mandibular fractures allow ↑ anterior jaw displacement after induction, but airway maintenance by face mask may not always be easy due to swelling and loss of jaw structure. An RSI may be appropriate with suxamethonium or rocuronium (have sugammadex available).
- Gas induction and effective preoxygenation may be more difficult due to pain when applying the face mask.

Postoperative

As for patients having maxillary/mandibular osteotomies (see ➲ pp. 788–9).

ALGRAWANY

Maxillary/mandibular osteotomy

Procedure	Surgical realignment of facial skeleton
Time	3–6h
Pain	++
Position	Supine, with head-up tilt, head ring
Blood loss	Variable. Occasionally can be severe. G&S
Practical techniques	Nasal tube and IPPV. Consider arterial line

Patients presenting for orthognathic surgery may have malformations isolated to one or both jaws or have multiple craniofacial deformities as part of a syndrome. Patients are usually in their late teens or early twenties and are generally fit and healthy. When an osteotomy is performed, the bone is plated and often transiently stabilised by wiring the maxilla and mandible together. Rarely the patient remains 'wired' at the end of the case. If vomiting or bleeding were to occur postoperatively, fatal airway obstruction may ensue, unless the fixation can be quickly removed. This requires expertly trained staff and adequate postoperative facilities.

Preoperative

- Assess the airway carefully. Check the nostrils for patency.
- Check Hb and G&S (as per local guidelines).
- Thromboembolic prophylaxis: compression stockings; consider the use of intermittent pneumatic compression boots in theatre.

Perioperative

- Intubate nasally using a preformed nasal tube (see p. 784). Spraying the cords and larynx with lidocaine may reduce coughing on extubation.
- Good venous access. Consider invasive pressure monitoring due to the length of surgery.
- Put lubricating ophthalmic ointment into the eyes and protect them with pads and tape or plastic contact lenses.
- Position the patient carefully on the operating table. Place the head on a ring and tilt the table head-up.
- Mild induced hypotension is useful to help minimise blood loss. Avoid hypertension. Consider remifentanil infusion (0.1–0.75 micrograms/kg/min, or TCI 2–6 nanograms/mL, although >12 nanograms/mL may occasionally be needed). Other choices include GTN infusion (2–10mg/h) and β-blockade (e.g. labetalol 5–10mg boluses or by infusion). As a general guide, maintain the MAP at 60mmHg or above.
- Give IV antibiotics as per local protocol (e.g. co-amoxiclav 1.2g or clindamycin 600mg) and steroids (e.g. dexamethasone 6.6mg IV) to minimise swelling.
- Administer prophylactic antiemetics (ondansetron plus cyclizine) to minimise the risk of nausea and vomiting. Dexamethasone is also effective.
- Keep the patient warm. Measure the core temperature; warm IV fluids and use a heating mattress and/or hot air blower. These may need to be reduced after 2–3h, as patients tend to overheat.

- Monitor blood loss carefully. HemoCue® is a useful way of tracking Hb concentration in theatre.
- The patient's jaws will rarely be wired together on completion of surgery. Ensure that throat packs are removed and the oropharynx is cleared of blood and debris before this is done.
- Extubate the patient once fully awake. Withdraw the nasal tube (15cm mark at the nostril); insert safety pin and cut to leave as a nasopharyngeal airway.
- Use multimodal analgesia, including opioid, intraoperatively.
- Ensure that you and the nursing staff are familiar with the position of any wires that hold the jaws together. Make sure wire cutters are always with the patient if the jaws remain wired.

Postoperative

- Some units send these patients to HDU. Others send them to the ward after an extended period in recovery.
- Administer humidified O_2.
- Ensure all oral analgesics are prescribed in a soluble form. PCA or IM opioids should also be prescribed.
- Continue prophylactic antibiotics and steroids postoperatively, as per your unit's protocol (usually 24–48h).
- Prescribe IV fluids. Encourage the patient to take fluid by the oral route as soon as possible.

ALGRAWANY

Maxillofacial tumour surgery

(See also ➲ pp. 784–5.)

Procedure	Resection of head and neck tumour. May include tracheostomy formation, access surgery, neck dissection, ablative surgery and graft or flap reconstruction
Time	2–10+ h
Pain	++
Position	Supine, head ring, head-up tilt, pad under shoulders if neck dissection or roof of mouth surgery
Blood loss	Moderate. Group and save is usually sufficient
Practical techniques	IPPV with north-facing preformed nasal ETT, or RAE on the other side. May need tracheostomy. Arterial line, urinary catheter for long cases with reconstruction

Preoperative

- Assess airway, paying attention to tumour site and size, relevant imaging, obstructive symptoms, mouth opening and neck mobility, anaesthetic records, history of radiotherapy and previous maxillofacial/neck surgery. Discuss with the surgeon.
- Patients frequently have a history of smoking and high alcohol intake. Look for cardiovascular and respiratory comorbidity.
- Prepare long breathing circuit.

Perioperative

- Position and drape to allow access to head, neck and donor sites.
- Apply warming blanket and insert a temperature probe. Warming may need to be reduced after 2–3h, as patients tend to overheat.
- Fine-bore feeding NGT should be inserted before surgery begins if likely to be needed postoperatively.
- Ensure pressure areas are padded. Check periodically—pressure damage and tissue loss can occur with prolonged surgery (especially heels, elbows, nose and forehead).
- Give appropriate antibiotic prophylaxis as per local protocol (e.g. co-amoxiclav 1.2g or clindamycin 600mg).
- Give steroids (e.g. dexamethasone 6.6mg) to reduce postoperative swelling and for analgesia.
- If the surgery involves laser resection, minimise risks to the patient and staff by reducing FiO_2, using a laser tube if possible, inflating the cuff with 0.9% sodium chloride (± methylthioninium chloride (methylene blue)), using smoke-filtering masks and wearing eye protection. The surgeon should use protective 0.9% sodium chloride-soaked swabs, non-reflective instruments and smoke extraction (see ➲ pp. 476–9).
- Avoid hypertension (see ➲ pp. 1068–9). Normalise BP before wound closure to check for bleeding.

Reconstructive flap surgery

- Maxillofacial reconstructions are performed using local (e.g. nasolabial), regional (e.g. pectoralis major) or free flaps (particularly from the radial forearm/fibula/anterolateral thigh).
- Major operations may be lengthy, 6–10+ h.
- The same principles apply as for plastic surgery free flaps (see ➔ pp. 659–61), with the added complication of a potentially difficult airway, both pre- and post-surgery.
- Keep the patient warm. Hypothermia increases surgical site infection rates, and vasoconstriction and vascular spasm may threaten flap survival.
- Avoid hypertension. Allow lower BP during tumour resection and raising of the flap to reduce blood loss. Thereafter, aim for near preoperative BP. Consider advanced haemodynamic monitoring if familiar (e.g. LiDCO®). Avoid excessive IV crystalloid administration as oedema and swelling may compromise flap blood flow.
- Surgical stimulation varies widely during these operations. Remifentanil provides good titratable opioid cover and easier BP control. Ensure a longer-acting opioid is administered before waking.
- Consider regional blocks or LA infusions for peripheral donor sites.
- HDU or ICU care is usually indicated postoperatively for fluid balance, BP control, flap monitoring and rapid airway rescue if needed.
- Traditionally, surgical tracheostomies were often performed because of the risk of postoperative airway compromise. Sedation and overnight ventilation on the ICU with a nasal ETT are becoming increasingly common and may avoid tracheostomy.
- Avoid heavy-handed systemic analgesia as this may lead to postoperative hypotension when the surgical stimulus has abated. Sedation should be titrated down to minimise the need for fluid boluses and vasopressors.
- The patient should be nursed with the head and neck in a neutral position to avoid tension on, or compression of, newly anastomosed vessels.

Postoperative

- Ensure throat packs are removed before transfer to recovery or ICU.
- Assess airway for oedema and swelling. Plan the extubation carefully; it is often riskier than intubation.
- Keep head-up to minimise oedema, venous congestion and pain.
- Analgesia with regular paracetamol, NSAID and PO/IV morphine (these procedures are often less painful than they look). PCA is sometimes required and may be useful for donor site pain.

ALGRAWANY

Sedation for dentistry

Patients who are unable to tolerate dental treatment under LA can often be managed by a combination technique using sedation. These procedures are usually performed by the dentist in the dental clinic. PO or IV sedation can be provided by short-acting benzodiazepines such as midazolam. Inhalational sedation can be provided by subanaesthetic concentrations of N_2O (up to 50%) in O_2 using a nasal mask—termed 'relative analgesia'. Whichever route of administration is used, it is important to ensure that the patient remains conscious throughout. The patient must be monitored by a trained member of staff (and not the operator who may be distracted by the procedure).

General considerations

- Patients should be ASA 1 or 2. (Patients with significant comorbidities should have their procedures in the hospital setting with an anaesthetist present.)
- Patients will require an escort for the procedure and to care for them afterwards.
- Written instructions should be provided regarding limitations on driving (as for GA) and operating machinery postoperatively. The patient should be told to avoid a heavy meal/alcohol prior to treatment. Patients should follow standard starvation guidelines when an anaesthetist is present and conversion to GA is a possibility.
- Inhalational sedation cannot be used in patients with nasal obstruction or those unable to cooperate with breathing through a nasal mask.
- LA is used in all patients after sedation has been established.
- The patient should be able to communicate throughout the procedure.
- Resuscitation equipment must be available.

Suitable regimes

- Single-agent regimes are safer.
- For adults, midazolam 2mg IV; wait 90s, then give 1mg every 30s until sedated. Expect to give 6–10mg (Society for Advancement of Anaesthesia in Dentistry guidelines). Reduce the dose in the elderly.
- Low-dose propofol infusion (only with suitable training).
- O_2 (100%) via nasal mask; add 10% N_2O for 1min, then 20% for 1min. Continue increments of 5% until sedated (up to 50%).

Special considerations

- Have flumazenil available for reversal of midazolam.
- Allow at least 1h for recovery following IV sedation.
- Following N_2O sedation, 100% O_2 must be administered to prevent diffusion hypoxia.
- The patient can be discharged once they are able to stand and walk unaided.

General anaesthesia for dentistry

General considerations

- GA for dental procedures should be undertaken in hospital and reserved for patients unable to tolerate LA (i.e. young children, adults with learning difficulties, impacted/buried teeth).
- Patients with learning difficulties may have trouble understanding the procedure and are often anxious. A short-acting anxiolytic agent, such as midazolam, and a topical anaesthetic cream may help.
- Patients may have a more complex medical disorder such as Down's syndrome or other congenital abnormality. It is important to exclude any significant cardiac pathology.
- **Antibiotic prophylaxis** against infective endocarditis should not routinely be used for dental patients. For high-risk patients, prophylaxis may be appropriate. NICE recommends that clinicians should apply clinical judgement on an individual basis, explaining risks and benefits and involving the patient in the decision.
- Patients requiring extensive work can be treated in a day case unit but may require overnight stay if they have major comorbidities.
- **Positioning**. There is no longer a place for 'dental chair' anaesthesia. Postural hypotension can easily be overlooked. It is now standard practice to keep patients supine or slightly head-up.
- **LA infiltration** should be used whenever possible. Care with very young children where it may lead to accidental biting/laceration.
- **Dental labelling**. Deciduous teeth are assigned letters A–E in each quadrant, and adult teeth are numbered 1–8. Roots are indicated by 'x', supernumerary (extra) teeth by '$' and buried or unerupted teeth by a circle around the letter/number. These are drawn with the patient 'facing you' and may be written as a complete mouth grid (as shown in Fig. 32.1) or as quadrants.
- **Simple extractions** may be very quick procedures, lasting a few minutes. LMAs (flexible) are preferable for multiple extractions. A prop/gag is inserted by the surgeon to facilitate surgical access; ensure that it does not obstruct the airway. During extractions, airway patency must be maintained and may require jaw support. When extractions are complete, a pack is positioned over the dental sockets to absorb any oozing blood.
- **Restoration work** can take over an hour and often requires intubation and ventilation. To facilitate X-rays and bite assessment, a nasal tube may be required.

ALGRAWANY

Dental extractions

Procedure	Dental extractions
Time	2–30min (much longer for additional restorative work in adults)
Pain	+/++
Position	Supine
Blood loss	Nil
Practical techniques	LMA/nasal mask (nasal ETT for prolonged restorative work)

Preoperative

- Usually children 3–12y, dental phobics or patients with learning difficulties.
- Beware of undiagnosed pathology, e.g. heart murmurs.
- Obtain consent for analgesic suppositories, if required.
- Give pre-emptive oral analgesia, e.g. paracetamol and ibuprofen.
- If a sedative premedication is needed, consider buccal or nasal midazolam (500 micrograms/kg, max 10mg). Buccal absorption is more rapid than oral. Effects are variable, and the patient should be closely observed. Higher doses of up to 20mg (off product licence) have been used successfully in larger adults.
- Ketamine, dexmedetomidine and clonidine are alternatives.
- Apply a topical anaesthetic for cannulation if IV induction planned.

Perioperative

- Give propofol for IV induction, and sevoflurane for inhalational induction.
- Tape the eyes closed.
- Maintenance with volatile agent or IV agent.
- Use LA infiltration (by dentist/oral surgeon); opioids are not usually needed for short day cases.
- Simple extractions do not usually require antibiotic cover.
- Stabilise the head and neck manually during the procedure.

Postoperative

- Place young children in the lateral position, slightly head-down at the end.
- Regular paracetamol and ibuprofen.

Special considerations

- The operator may apply considerable pressure during extraction, often resulting in an airway-obstructing 'reverse jaw thrust'. The anaesthetist should apply counterpressure to support and stabilise the head and airway.
- Beware of potential hypoxia. Give 100% O_2 for maintenance, if necessary.
- Children with blocked noses can be safely anaesthetised using an LMA (provided there is no URTI).
- (See Fig. 32.1 for dentition labelling nomenclature.) X-rays should be displayed during surgery to ensure the correct teeth are removed.

Quadrant labelling:		Full set of deciduous teeth:		Full set of adult teeth:	
Upper right	Upper left	EDCBA	ABCDE	87654321	12345678
Lower right	Lower left	EDCBA	ABCDE	87654321	12345678

1: central incisor
2: lateral incisor
3: cuspid/canine
4: 1st premolar
5: 2nd premolar
6: 1st molar
7: 2nd molar
8: 3rd molar (wisdom teeth)
X: roots
$: unerupted teeth

8	8
8	13_{xx} 8

Example documentation

Fig. 32.1 Dentition labelling.

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Extraction of impacted/buried teeth

Procedure	Removal of teeth
Time	3–45min
Pain	+
Position	Supine, head ring, bolster under shoulders if upper teeth to be extracted
Blood loss	Minimal
Practical techniques	LMA and SV, nasal tube and IPPV (extubate awake or deep)

Preoperative

- Careful assessment of the airway. Check nostrils for patency.
- If the patient has a dental abscess, there may be marked swelling of the face and severe trismus. AFOI may be necessary (see pp. 393–6).

Perioperative

- Consider an LMA/oral tube for simple/unilateral extractions.
- For more complex procedures, consider a preformed nasal tube.
- Protect the eyes with tape and pads.
- The surgeon should anaesthetise the appropriate terminal branches of the maxillary division (infraorbital, greater palatine, nasopalatine) and mandibular division (inferior alveolar, lingual, buccal, mental) of the trigeminal nerve with LA.
- Give an opioid and NSAID/paracetamol pre- or intraoperatively.
- IV antibiotics may be administered to minimise the risk of infection (e.g. co-amoxiclav 1.2g or clindamycin 600mg for patients with penicillin allergy). Check your local antibiotic policy.
- Steroids (e.g. dexamethasone 6.6 mg IV) may be given for antiemesis and to minimise swelling.

Postoperative

- Balanced analgesia with regular paracetamol and NSAIDs. Prescribe rescue analgesia with PRN tramadol or codeine phosphate. (Codeine is no longer advised for children.)

Special considerations

- Talk to the surgeon to ascertain the likely length of surgery. Remember that some patients require GA only because they are 'dental-phobic'. The surgical extractions may be simple, and the operative time consequently very short. A short-acting muscle relaxant may be required.

Further reading

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ALGRAWANY

Chapter 33

Ophthalmic surgery

Peter B Williamson

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ALGRAWANY

Relevant anatomy and physiology

The orbit

The bony orbit (Fig. 33.1) is a 40–50mm deep, pyramid-shaped recess in the cranial bones (with the apex slightly angulated nasosuperiorly) which contains the globe and ocular muscles surrounded by loose connective tissue. Nerves and vessels enter via the superior and inferior orbital fissures and mostly remain inside 'the cone' of muscles. Globe movement is controlled by the extraocular muscles, innervated by cranial nerves (CN) III, IV and VI (III + LR_6SO_4 is a useful aide memoire). The sensory components are via the 1st and 2nd divisions of the trigeminal nerve (CN V).

The globe

In relation to the orbit, the globe sits anteriorly, high and closer to the zygomatic bone on the temporal side. Needle access is therefore safest either at the medial canthus or inferotemporally. The 'axial length' quantifies the depth the globe reaches in the orbit, with higher values suggesting a longer, 'sausage-shaped' eye which may preclude peribulbar blocks. The sclera is the fibrous exterior of the globe, protecting the deeper choroidal, middle and inner retinal layers. Immediately superficial to the sclera is the potential space bounded by the white and avascular Tenon's capsule, which can

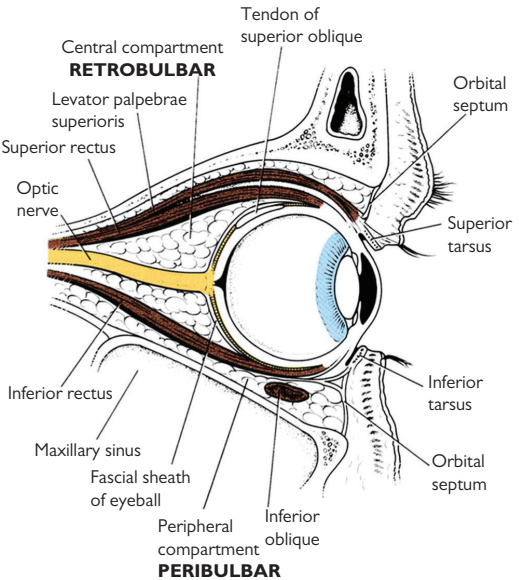

Fig. 33.1 Vertical section through the orbit. Reproduced with permission of Oxford Publishing Limited through PLSclear from McLeod G, *et al*. (2012) *Principles and Practice of Regional Anaesthesia*, with permission from Oxford University Press. Copyright © Oxford University Press, 2012.

be accessed to administer a sub-Tenon's block. The most superficial layer visible is the conjunctiva, which is loose to allow eye movement and inserts into the limbus of the eye (the boundary of the cornea).

The cone

Delineated by the four rectus muscles, the cone contains both key sites for the action of anaesthetic drugs (sensory nerves, the ciliary ganglion and the insertion of CN III, IV and VI into their respective extraocular muscles) and structures best avoided (the optic nerve, and retinal artery and vein). The aim of a peribulbar block is to deposit LA outside the cone and allow it to spread gradually to intraconal structures, whereas with retrobulbar block, the needle enters the cone itself and a smaller volume of LA is directly injected to the area of interest.

Intraocular pressure

The IOP is made up of aqueous/vitreous humour volume, choroidal blood volume and extraocular tone. It is akin to ICP in many ways and the normal range is 10–20mmHg in the healthy intact globe. The IOP can increase dramatically during coughing, vomiting or laryngospasm, resulting in catastrophic expulsive haemorrhage if the globe is open. Preventing these events and controlling IOP is key and can be achieved through:

- Head-up position and GA with muscle relaxants
- Avoidance of direct eye compression and the pressor response during airway management
- Physiological manipulation of choroidal blood volume with modest hyperventilation to an $ETCO_2$ of 3.5–4.0kPa and avoidance of hypertension/tachycardia
- Pharmacological manipulation with acetazolamide 500mg IV (reduces aqueous production) and mannitol 0.5mg/kg IVI (increases vitreous drainage), while mindful of the uncatheterised patient due to excessive diuresis on the operating table
- Minimising LA volume during blocks.

Oculocardiac reflex

Unique to ophthalmic surgery, and most commonly seen during squint and glaucoma surgery, the oculocardiac reflex is triggered by pressure on the extraocular muscles or the globe itself and results in a powerful vagal response via the short and long ciliary nerves. Clinically, this manifests as a sudden bradycardia (and, in extreme cases, asystole), sometimes accompanied by apnoea and vomiting. Prophylaxis against the reflex is via:

- LA techniques which block the ciliary afferent nerves (sub-Tenon's and peribulbar blocks or intracameral LA injection)
- Avoidance of hypercapnia
- Administering glycopyrronium bromide 200–400 micrograms.

Treatment of the bradycardia is via removal of surgical traction or pressure, appropriate vagolytics (glycopyrronium 200–600 micrograms or atropine 300–600 micrograms) and support of CO.

ALGRAWANY

Preoperative considerations

Preoperative assessment

Although classically described as the 'extremes of age', all age groups can present for ophthalmic surgery:

- Paediatric: treatment of congenital pathology and strabismus
- Adult: treatment of refractive errors, floaters, penetrating eye injury and ptosis
- Elderly: treatment of cataracts, retinal detachment and glaucoma.

The patient population tends to share some common features:

- DM or hypertension: predisposition to retinal pathology and detachment; relevant as risk factors for cerebrovascular disease and IHD
- COPD: predisposition to retinal vascular disease, more likely to cough during surgery
- Thyroid disease: predisposition to ocular pathology; relevant as risk factors for OSA and difficult airway
- Systemic diseases with ophthalmic presentations such as multiple sclerosis, inflammatory bowel disease, sarcoidosis and connective tissue disease
- Anxiety: very few people are relaxed about the notion of a surgeon performing microsurgery on one of their most valued organs, let alone awake with a drape over their head.

In addition to standard preoperative assessment and in-depth questioning regarding the issues above, make special note of:

- The patient's ability to lie flat and still
- Axial length (if available) on cataract biometry—if >26mm, it may preclude peribulbar block
- INR/APTT/anticoagulant usage—peribulbar blocks may be contraindicated with a certain degree of coagulopathy (exact cut-offs may be dictated by local protocols)
- Tamsulosin—predisposes to 'floppy iris syndrome' which can make a routine cataract more difficult.

Anaesthetic strategy

Determining conduct of anaesthesia (LA, sedation or GA) is one of the most crucial strategic decisions to be made preoperatively.

LA techniques can be used in conjunction with sedation or GA as appropriate and each combination will present a different risk/benefit analysis. LA techniques are usually preferred due to minimising cardiovascular instability and drug load, providing good operating conditions and maintaining high list turnover. They can also be performed by most ophthalmic surgeons. Patient cooperation during block insertion is highly indicative of their likely behaviour on the operating table.

Consider:

- Patient factors: level of cooperation/hearing/comprehension, degree of anxiety and claustrophobia, movement disorders precluding remaining still and flat, predilection for coughing, ASA grade and factors which increase the risk of GA
- Anaesthetic factors for converting to GA: ease of IV access, degree of shared airway

- Surgical factors: length and complexity of procedure, degree of surgical stimulation/postoperative pain, need for normal muscle tone (e.g. for blepharoplasty), degree of akinesia required and likely impact of patient movement, multiple site surgery (e.g. donor site surgery for oculoplastics), need for vision in operative eye following surgery
- Logistical factors: impact on list booking and turnover, level of postoperative care required.

ALGRAWANY

Local anaesthetic techniques

Topical anaesthesia

Topical anaesthesia is the least invasive LA modality and is quick, simple, very safe, cheap and suitable for most superficial procedures, including cataracts. There are virtually no contraindications beyond being able to lie flat for the procedure, and IV access is not needed. Vision is also intact postoperatively which is especially important if you are operating on the only functioning eye. Topical anaesthesia is limited by the fact that it confers only simple surface analgesia and no akinesia, and is relatively ineffective in the inflamed eye.

- Simple instillation of drops (proxymetacaine 0.5%, tetracaine 1%, oxybuprocaine 0.4%) is all that is required.
- The surgeon will often supplement topical anaesthesia by blocking the iris and ciliary body with an injection of LA into the anterior chamber (intracameral) once surgery is under way.

Drugs for blocks

- Lidocaine 2%: more than sufficient for cataract surgery.
- Bupivacaine 0.5%: suitable for block under GA for postoperative analgesia, or longer surgery awake if time allows the block to evolve.
- A 1:1 mixture of 2% lidocaine and 0.5% bupivacaine provides balance between onset, duration and quality of the block for longer procedures.
- Hyaluronidase 2–30 units/mL is sometimes added to aid block spread and reduce IOP, although this must be balanced against the risk of occasional severe allergy or anaphylaxis.

Volume of LA for blocks

- Higher volumes of LA improve onset and quality of the block.
- High volumes will also deliberately proptose a 'deep eye' and facilitate surgical access.
- However, high volumes will also increase IOP and make anterior chamber surgery difficult, especially with glaucoma.
- Sub-Tenon's blocks usually require ~3.0–5.0mL of LA.
- Peribulbar blocks usually involve a total of 5.0–10.0mL of LA, depending on the number of injections and desired effect.

Sub-Tenon's block

(See Fig. 33.2.) Performing a sub-Tenon's block is simple and relatively safe, conveys modest analgesia and akinesia and can be performed in most patients, including those with long axial lengths and/or taking anticoagulants. Its principle disadvantages are disruption and oedema of the conjunctiva (chemosis), which makes some surgical procedures more challenging (e.g. trabeculectomy, port insertion for vitrectomy), and performing the block becomes more challenging with successive procedures due to scarring of the planes (although an alternative superotemporal approach can be used), and vision is blocked postoperatively. The eye can be unsightly and irritated at 24–48h, but this is usually mild and treated with simple analgesia and LA drops. IV access is only usually required if sedation is indicated.

- Position the patient supine.
- Apply proxymetacaine 0.5% and iodine drops to the operative eye.

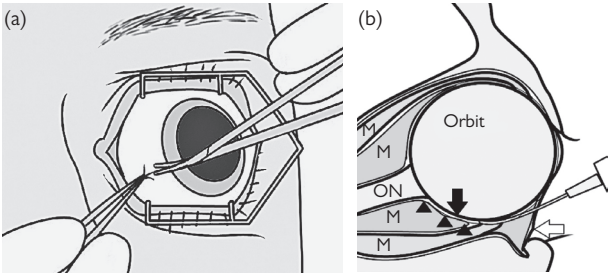

Fig. 33.2 Sub-Tenon's block. (a) Making the incision in the inferior nasal quadrant of the conjunctiva for a sub-Tenon's block. (b) Cross-section through the orbit showing the position of the needle during a sub-Tenon's block. M, extraocular muscles; ON, optic nerve. Black triangles, Tenon's fascia; black arrow, sub-Tenon's space; white arrow, conjunctiva .Reproduced with permission of Oxford Publishing Limited through PLSclear from Warman P. *et al*. (2014) *Oxford Specialist Handbook of Regional Anaesthesia, Stimulation and Ultrasound Techniques*. Copyright © Oxford University Press, 2014.

- Clean the lids, eyelashes and eyebrows with iodine prep solution, then retract the lower lid with a speculum.
- Ask the patient to look 'up and out' or to follow your fingers to the correct position.
- Identify an avascular area about 5mm away from the limbus in the inferonasal quadrant.
- Use Moorfields forceps to lift the conjunctiva.
- Make a small incision with blunt-ended Westcott's scissors; you should see the plain white, relatively avascular Tenon's capsule.
- In some cases, you can proceed straight to insertion of a blunt sub-Tenon's cannula in an inferonasal direction and then injection; in others, further blunt dissection of the plane with the scissors is required.
- Once the cannula is past the equator of the globe (syringe almost vertical, perpendicular to the patient), you can administer LA injectate—warn the patient they will experience some pressure behind the eye at this point.
- Usual volume of injectate is 3.0–5.0mL of LA; then tape the blocked eye closed.
- Complications: if you see considerable chemosis, you might be in the wrong plane (subconjunctival); more serious complications are extremely rare.

Peribulbar block

(See Fig. 33.3.) An extraconal injection of LA; quick to perform either via the conjunctiva (cleaner and easier to anaesthetise) or transcutaneously (if the patient cannot keep eye open) without requiring a speculum; provides good akinesia and analgesia with minimal disruption of conjunctiva. However, as a sharp needle technique, it introduces the risk of globe perforation (<0.1%) and retrobulbar haemorrhage (0.07%). It is more dangerous in the long eye (axial length >26mm increases the risk of globe

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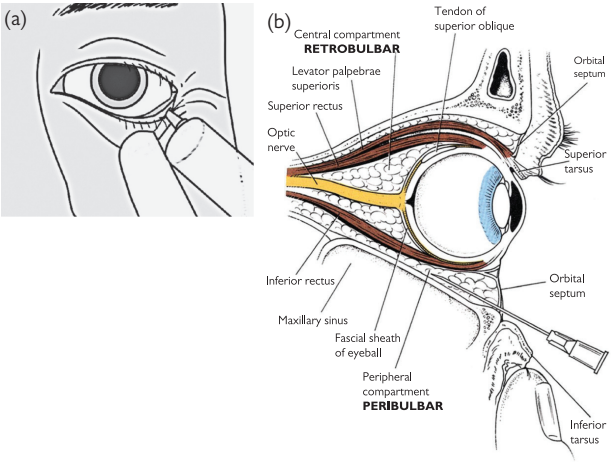

Fig. 33.3 Peribulbar (extraconal) block. (a) An inferotemporal injection for a peribulbar block. (b) Needle position for peribulbar block. Reproduced with permission of Oxford Publishing Limited through PLSclear from a) Warman P. *et al*. (2014) *Oxford Specialist Handbook of Regional Anaesthesia, Stimulation and Ultrasound Techniques* © Oxford University Press, 2014; b) McLeod G *et al*. (2012) *Principles and Practice of Regional Anaesthesia*. © Oxford University Press, 2012.

perforation) and is relatively contraindicated with anticoagulants (but safe with aspirin) and absolutely contraindicated in the perforated or infected eye. Vision is also blocked for a prolonged period postoperatively. IV access is a risk/benefit analysis but suggested, given the risk of systemic complications (e.g. bradycardia or seizure).

- Establish IV access if required and attach basic monitoring.
- Prepare LA injectate in a 10mL syringe.
- Position the patient either supine or sitting up.
- Apply proxymetacaine 0.5% and iodine drops to the operative eye.
- Ask the patient to look straight ahead.
- Establish needle entry point: junction of middle and lateral thirds of a horizontal line across the orbit, 1mm above the inferior orbital rim either through the conjunctiva or the skin.
- Insert the 25G (orange) 25mm hypodermic needle with slight inferior intent away from the globe (in case patient moves forwards).
- If the patient remains still, advance the needle perpendicularly to the inferior orbit floor (if you make contact with the orbital floor, a slight superior adjustment allows you to stay on target), aiming for the needle tip to be at the same depth as the posterior pole of the globe (usually most, if not all, of your needle length is utilised).
- Aspirate before administering 5.0–8.0mL of LA injectate; warn your patient of a pressure behind the eye.
- If using an integrated two-injection technique, or top-up is required due to poor akinesia, proceed with a medial canthus injection.

- Establish needle entry point: at the medial canthus, clear of the lacrimal apparatus and medial to the caruncle at the very edge of the skinfold.
- Insert 25G 25mm hypodermic needle perpendicular to the patient's face, parallel to the medial wall of the orbit, to a depth of 10–20mm and administer a further 2.0–4.0mL of LA injectate, then tape the blocked eye closed.
- Look for: akinesia of the eye, ptosis and block of orbicularis oculi.
- Complications: local complications include lower lid filling (which can be remedied with firm digital massage), patient complaining of numb teeth, globe perforation (if you see involuntary movement of the globe, stop the block immediately and inform your surgeons) and retrobulbar haemorrhage (rapidly proptosing eye); systemic side effects include bradycardia and vasovagal syncope, or seizures in very rare cases.

Retrobulbar block

(See Fig. 33.4.) Retrobulbar block (intraconal injection of LA) is no longer routinely performed due to the higher incidence of retrobulbar haemorrhage, globe perforation, LA toxicity and total spinal. It is still of clinical relevance as rapid ptosis and block onset with only a few mL of LA during a peribulbar block could indicate inadvertent retrobulbar block—either stop injecting or adjust the dose to 2.0–4.0mL. Inform your surgeon of likely retrobulbar injection.

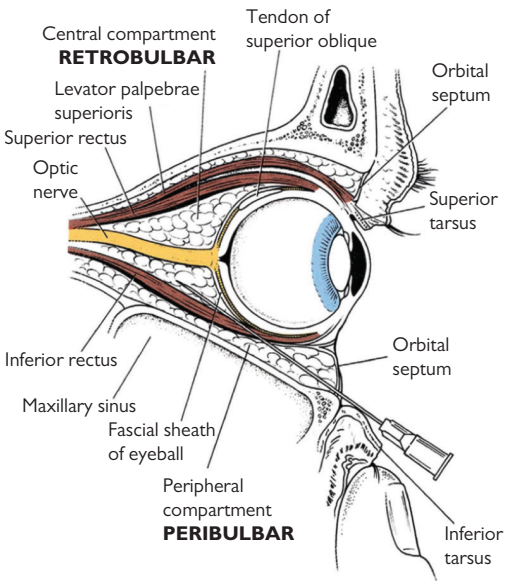

Fig. 33.4 Retrobulbar (intraconal) block. Reproduced with permission of Oxford Publishing Limited through PLSclear from McLeod G, *et al*. (2012) *Principles and Practice of Regional Anaesthesia*, with permission from Oxford University Press. Copyright © Oxford University Press, 2012.

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General anaesthesia and sedation

The main anaesthetic objective with GA is to eradicate any form of movement during surgery (even inconsequential movement, such as leaning on the table, is perceived by the surgeon down the microscope), with the 2° objective being lowering IOP via a combination of $PaCO_2$ control and inducing a 'tranquil circulation'.

Fortunately, there are some factors which make ophthalmic anaesthesia slightly easier than one would initially expect, namely:

- The minimal degree of tissue trauma, stress response and blood loss
- LA techniques reducing the need for long-acting opioids in most cases.

However, the challenge is:

- Having the patient deep and/or paralysed enough to prevent any movement right up until the end of surgery, but having them awake and maintaining their own airway straight afterwards in the context of a high turnover day case list in a remote site.
- Balancing the best way of controlling IOP via $PaCO_2$ (intubating and ventilating) vs the technique least likely to cause coughing on emergence (SGA).
- The patient population is usually ASA 3.

Induction

A standard induction with propofol and a short-acting opioid (remifentanil, alfentanil or fentanyl) is ideal, in anticipation of analgesia being provided either by topical drops or an LA block.

- Even small doses of non-depolarising neuromuscular blockers (e.g. rocuronium) are useful in abolishing laryngospasm at induction and movement of the eye during surgery (the extraocular muscles are exquisitely sensitive to NMB drugs) but are best avoided in very short surgery, such as cataract extractions, unless use of sugammadex is planned.
- Glycopyrronium bromide 200 micrograms is useful to obtund the oculocardiac reflex and dry up secretions to prevent laryngospasm on emergence.
- Tape the non-operative eye (note this is the opposite of cases done under block).
- Induction usually takes place on a pillow for patient comfort and to facilitate neck movement for airway management, but before surgery is usually swapped for a rigid support pillow (e.g. Rubens pillow) to prevent head movement during surgery.
- Standard antiemetics are indicated to prevent PONV.

Airway

In many ways, SGAs are ideal as they eliminate the need for laryngoscopy and are more likely to facilitate a coughless emergence; however, this needs to be weighed against the risk of aspiration and/or losing the airway without immediate access to the head.

- Depending on the degree of access the surgeon requires, either a reinforced LMA or a 2nd-generation supraglottic device would be sufficient in most cases.
- If intubating, a south-facing RAE tube is ideal in most cases, although reinforced tubes can also be used.

Maintenance and ventilation

- Propofol/remifentanil TIVA is more likely to confer a coughless emergence without emergence delirium or PONV; however, depth of anaesthesia monitoring is often confounded by surgical pressure on the forehead.
- Volatile maintenance with sevoflurane facilitates a spontaneously breathing patient being transferred to recovery to wake in their own time, but can be associated with coughing during the procedure due to secretions.
- N_2O is best avoided, as some ophthalmic procedures involve injection of gas of precise density into the eye.
- SV via an SGA is suitable for most short cases such as cataracts; however, some patients will naturally settle at a $PaCO_2$ which significantly raises IOP and makes anterior chamber work difficult, so be prepared to take over ventilation if required.
- Controlled ventilation allows best control of IOP but often requires additional drug load, which makes rapid wake-up after a 10min case challenging.
- If utilising controlled ventilation, avoid high airway pressures and watch for subtle changes in the P_{AW} waveform which suggest the patient is recovering from NMB or is about to obstruct, and manage these before they fully manifest.
- Non-depolarising neuromuscular blockers are useful as part of a balanced anaesthetic technique to help abolish movement during surgery—regular boluses throughout a longer case (e.g. 10–20mg of rocuronium or atracurium) guided by the length of surgery and time to finish are usually used, rather than infusions; standard neuromuscular monitoring and appropriate reversal at the end of the case are both required.
- Intraoperative hypertension causes IOP to rise and should be treated with IV agents, with common drugs of choice being opioids (if pain is the cause), magnesium, clonidine, esmolol/labetalol (although caution with COPD and DM) and occasionally phentolamine, depending on the degree of hypertension.

Analgesia

Many GA ophthalmic procedures do not require long-acting opioids for postoperative analgesia as the patients can be given topical LA drops or a block under GA by the surgeons.

- Notable exceptions are some oculoplastics and vitreoretinal procedures which require modest doses of a longer-acting opioid such as fentanyl, morphine or diamorphine, depending on the degree of stimulation which breaks through the LA block.
- A bolus of a short-acting drug such as alfentanil at induction to facilitate airway instrumentation usually suffices if a surgeon is going to insert a block before or shortly after surgery commences.
- If a block is going to be performed at the end of the procedure (e.g. trabeculectomy), then remifentanil TCI can be used intraoperatively to obtund surgical stimulation but prevent narcosis in recovery.

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Emergence

The key objective at emergence is to prevent coughing, which raises IOP and strains ophthalmic sutures.

- Optimising the conditions and timing for the removal of the airway are both important.
- Additional techniques sometimes employed to remove the airway include: deep extubation and/or exchange to an SGA/oropharyngeal airway, spraying the cords with lidocaine and IV injection of lidocaine (1mg/kg).

Sedation

- If surgery is proceeding under topical or block, it is not uncommon for the patient and/or surgeon to request sedation.
- There is still a widespread belief that sedation is universally 'safer than GA' when, in fact, the patient can be disinhibited, uncooperative and free to move and/or hypoventilate intraoperatively.
- Careful patient selection is vital.
- IV access, supplementary O_2 and standard monitoring are required—capnometry attached to either nasal cannulae or a face mask modified to avoid impinging on the surgical field is ideal.
- Midazolam 0.5–2.0mg has a role despite the risk of POND, given the need for both cooperation and anxiolysis.
- Very low doses of propofol (10–30mg) can also provide anxiolysis and gentle suppression of consciousness during block insertion.
- Boluses of opioids such as alfentanil (100–200 micrograms) or fentanyl (25–50 micrograms) can be titrated to effect if the patient experiences pain, but must be offset against the risk of inducing nausea and vomiting.
- Allow the patient to wake after block insertion, but before surgery begins—patients who regain consciousness after this are likely to be distressed by their change in situation (different room, drapes, light shining in their eye, etc.) and reach for their eye almost as a reflex.
- Reassure patients verbally; hold their hand, and regain their full cooperation before surgery.
- Have a plan to convert to GA if required.

Vitreoretinal surgery

Procedures	Intraocular posterior chamber retinal surgery, including vitrectomy, cryo/laser therapy, removal/insertion of oil/gas, scleral banding and retinopexy
Time	90–180min
Pain	++/+++
Position	Supine
Blood loss	None
Practical techniques	Reinforced LMA + IPPV + LA block ETT (RAE or reinforced) + IPPV + LA block LA block ± sedation

Preoperative

- Past medical history often includes COPD, hypertension, DM and IHD as these are also risk factors for retinal disease.
- Check axial length if possible—patients may have long eyes (hence the retinal detachment) which may preclude peribulbar block.
- Patients sometimes come for repeat admissions and are used to having ophthalmic procedures (it can be difficult to site a sub-Tenon's because of scarring of the tissue planes).
- 'Mac-on' retinal detachments (where the macula remains undetached) are urgent sight-saving procedures.

Perioperative

- Surgery is long, stimulating and performed in the dark.
- Many require a GA due to length and complexity of the procedure.
- Additional LA block is highly advised to smoothe out the intense stimulation, and is often inserted by the surgeon following port insertion (either a sub-Tenon's or careful medial peribulbar).
- Ensure adequate opioids on board to cover the extremely stimulating port insertion (alfentanil bolus or remifentanil infusion).
- IPPV via an SGA with either TIVA or a volatile is ideal.
- Avoid N_2O as the surgeons use gases of specific densities and postoperative posturing to hold retinal detachments in place.
- Modest doses of fentanyl, morphine or diamorphine may be required for postoperative analgesia in very painful cases, e.g. cryotherapy.
- Ensure adequate depth of anaesthesia during the final phases of surgery—the ports are removed last, so the globe is 'open' right to the end.

Postoperative

- The LA block is usually sufficient.
- Some patients might require simple analgesics, e.g. paracetamol and tramadol, especially if cryotherapy was part of surgery.

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Cataract and anterior chamber surgery

Procedures	Cataract extraction and intraocular lens insertion, photocoagulation of ciliary body, insertion of aqueous shunt, trabeculectomy
Time	+/+++
Pain	+
Position	Supine
Blood loss	None
Practical techniques	Topical LA LA block + sedation Reinforced LMA + IPPV + LA block ETT (RAE or reinforced) + IPPV + LA block

Preoperative

- Ensure the patient can be still and flat for cataract extraction.
- Highlight any manual handling issues which might disrupt a high-turnover list.

Perioperative

- Cataract extractions are almost always performed under topical LA or sub-Tenon's block.
- Requests for sedation are not uncommon (see ➲ p. 810).
- Tape a swab over the ear on the operative side to catch irrigation fluid.
- GA cataracts can usually be undertaken with the patient spontaneously breathing through an SGA—opioids at induction are to be used cautiously, given the 10–15min procedure time.
- Patients with active inflammation, e.g. due to glaucoma, often need a block as topical LA is less effective.
- Shunt insertion and trabeculectomy are longer procedures, which usually require a GA and can be blocked by the surgeons towards the end (owing to the operation being on the conjunctiva itself).
- For longer procedures, IPPV is recommended; using an LMA to facilitate coughless emergence must be balanced against the risk of aspiration.

Postoperative

- Postoperative analgesic requirements are negligible as topical LA is very effective.

Strabismus surgery

Procedures	Extraocular squint correction
Time	60min
Pain	++
Position	Supine
Blood loss	None
Practical techniques	Reinforced LMA + IPPV/SV ETT (RAE or reinforced) + IPPV

Preoperative

- Common paediatric day case.
- Patients are usually otherwise well. Strabismus can occasionally be part of a syndrome.
- Consider a sedative premedication if the child is distressed (they may have visual impairment).

Perioperative

- Requires a GA (IV or inhalational induction as indicated).
- Avoid suxamethonium as it alters extraocular muscle tone and there may be some association between squints and MH.
- Depth of anaesthesia should facilitate neutral gaze; TIVA with propofol is useful as it also reduces PONV.
- Be vigilant for the oculocardiac reflex—avoid hypercapnia and be prepared to treat with vagolytics.
- SV via an LMA is usually sufficient; IPPV may be required if $ETCO_2$ is high; intubate if indicated.
- Multimodal PONV prophylaxis (ondansetron 0.1 mg/kg, dexamethasone 0.1 mg/kg).
- Sub-Tenon's block performed by the surgeon, paracetamol 15mg/kg and diclofenac 1mg/kg for postoperative analgesia (avoid opioids if possible).

Postoperative

- There is a high incidence of PONV, especially with opioids.
- Moderate analgesic requirements, e.g. PO morphine or tramadol.
- Standard paediatric day case concerns.

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Dacryocystorhinostomy

Procedures	EUA tear duct, insertion of drainage tube, dacryocystorhinostomy
Time	45min
Pain	+/++
Position	Supine + head-up
Blood loss	Low, but near airway
Practical techniques	ETT (RAE or reinforced) + IPPV Reinforced LMA + IPPV

Preoperative

- Usual concerns for eye and ENT surgery apply.
- Establish if the patient is likely to tolerate hypotension.

Perioperative

- Standard anaesthetic induction of choice as indicated.
- Using an SGA avoids complications of intubation, but blood may soil the airway in some difficult procedures.
- Multimodal approach to minimising blood loss: Moffett's solution/co-phenylcaine to the nose after induction, IPPV desirable as moderate hypocapnia reduces blood loss, head-up tilt to improve venous drainage, moderate induced hypotension (remifentanil can be useful).
- Traditionally, a throat pack was used for all cases, but this is falling out of favour based on risk/benefit.
- Analgesia usually consists of paracetamol, an NSAID and remifentanil infusion plus a bolus of longer-acting opioid.
- Magnesium can also be useful both to reduce BP and as an analgesic.
- Ask your surgeon to give LA to the ducts if possible.

Postoperative

- Postoperative analgesia can be provided by paracetamol, an NSAID and an oral opioid such as tramadol or codeine.

Penetrating globe injury

Procedures	EUA, repair of globe rupture, closure of punctum, enucleation
Time	++/+++
Pain	+++
Position	Supine
Blood loss	Minimal
Practical techniques	ETT (RAE or reinforced) + IPPV

Preoperative

- In addition to standard anaesthetic concerns, adults with penetrating eye injury are often extremely anxious, in pain and unfasted.
- Children represent a particular problem as they are usually also uncooperative, crying and rubbing their eye.
- Surgery is an emergency, as coughing or straining can result in expulsive haemorrhage of globe contents, but must be balanced against fasting and optimising for GA.
- Use preoperative opioid analgesia with extreme caution (as vomiting is catastrophic) and give pre-emptive antiemetics.

Perioperative

- Patients should generally be treated as being at risk of aspiration, rendering SGAs less useful.
- Depending on the clinical scenario, induction can be 'ophthalmic' or an RSI for rapid airway control in the unfasted patient, or have elements of both.
- RSI dose of rocuronium (1.0–1.2mg/kg) with rescue dose of sugammadex (16mg/kg) on standby in case of difficult airway is ideal.
- Suxamethonium raises IOP (vs induction agents which lower it) and familiarity with its use is falling; however, it may still be used.
- Intubating with VL as 1st-line reduces the pressor response and removes the need for multiple laryngoscopies with difficult airways.
- LA techniques are not effective (or even possible) with these cases; long-acting IV opioids are commonly required.

Postoperative

- Recovery analgesia can be provided with topical LA drops and boluses of fentanyl, morphine or diamorphine as required.
- Regular ward analgesia can be paracetamol, an NSAID of choice and a PRN weak opioid given PO.

Further reading

Anker R, Kaur N (2017). Regional anaesthesia for ophthalmic surgery. *BJA Educ*, **17**, 221–7. https://doi.org/10.1093/bjaed/mkw078

ALGRAWANY

Chapter 34

Anaesthesia for radiology and cardiology

Alex Wickham

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Anaesthesia for MRI *820*
Anaesthesia for interventional radiology *823*

Katherine Holmes, Craig Dunlop and **David Tomlinson**

Anaesthesia for cardiology procedures *826*
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Anaesthesia for angiography *829*
Anaesthesia for cardiac device insertion and removal *830*
Anaesthesia for electrophysiology procedures (AF and VT ablation) *832*
Anaesthesia for transcatheter aortic valve implantation *834*

See also

- Endovascular treatment of intracranial aneurysms p. *580*
- Endovascular thrombectomy p. *582*
- Endovascular stenting of elective or emergency abdominal aortic aneurysm p. *597*
- Resuscitative endovascular balloon occlusion of the aorta p. *1000*

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Anaesthesia for diagnostic imaging

Anaesthetists are increasingly working within medical imaging departments. However, the environment remains potentially hazardous, and the equipment unfamiliar. Ensure that experienced, trained assistance and full monitoring are available. Familiarise yourself with the staff and surroundings. Locate the nearest resuscitation facilities (self-inflating bag/mask, portable O_2, 'crash' trolley and defibrillator) and confirm that your assistant and the radiographers also know where these are located.

Indications for anaesthesia

- Young or uncooperative children. Infants (under 2mo) may sleep through a scan if given a feed and wrapped up well.
- Older children or adults with psychological, behavioural or movement disorders.
- Acute trauma patients or patients receiving intensive care.
- Interventional procedures under ultrasound, CT or MRI guidance that require analgesia, sedation or anaesthesia.

Anaesthesia: general points

- Patients requiring anaesthesia for elective scans have a range of problems. Check the indications for the scan and the nature of the underlying pathology, e.g. developmental delay, epilepsy, malignancy, psychiatric disease or movement disorders. Beware the 'undiagnosed' paediatric patient and syndromes with CVS manifestations.
- Choice of sedation or GA depend upon individual patient needs, the nature of the scan and the skills of the anaesthetist.
- Check whether the anaesthetic machines are using piped gases or cylinders. If using cylinders, confirm that a full spare O_2 cylinder is immediately available.
- Plan the location of the anaesthetic machine, suction, monitoring and the configuration and routing of the breathing system with radiographers in advance. Ensure the breathing circuit is sufficiently long for any gantry movement.
- Decide where to induce the patient—a dedicated induction area may not be available or may be very small. It is usual to induce on a tilting trolley, then transfer to the scanner when anaesthetised.
- Certain equipment configurations (e.g. anaesthetic machine in the scan room and monitors in the control room) may require two anaesthetists to manage the patient safely.
- Satisfactory recovery facilities must be available, i.e. an appropriately equipped recovery bay staffed by an experienced recovery nurse near the scanner. If not available, arrangements for safe transfer of the patient to an operating department recovery room must exist.
- Intensive care patients requiring diagnostic imaging should be managed with full transport monitoring and ventilatory support. Ideally, the ICU medical team should supervise the patient and review the scan with the reporting radiologist before return to the ICU.

Anaesthesia for CT

- CT scanning does not restrict the type of equipment used, but space can be limited, so compact machines and monitors are ideal.
- The patient, anaesthetic machine and monitors must all be visible from the control room.
- The patient's head is usually accessible during CT scanning, so an SGA may be used if airway protection is not required.
- Anaesthesia or sedation sufficient to produce immobility and lack of awareness is all that is required for diagnostic procedures.

Hazards

- CT scanning uses ionising radiation, so it is preferable for the anaesthetist to monitor the patient from outside the scan room. If it is necessary to remain near the patient, wear appropriate radiation protection and use barriers if available.
- Cannulae, catheters, drains and ETTs can pull out during movement of the patient through the scanner—check nothing snags beforehand.

Contrast media

- IV contrast media for X-ray imaging are usually iodine-based, non-ionic, water-soluble compounds. Agents may trigger allergic reactions (ask about iodine sensitivity).
- Radiographers will usually give IV contrast, but you may be asked to administer it in anaesthetised/paediatric patients. The radiographers should ensure the correct volume (dependent on preparation, investigation, age and weight) is provided according to local policy.
- Some 'dynamic' investigations (e.g. aortography) require contrast to be administered while the scan is occurring.
- Contrast is viscous and can be difficult to inject through small cannulae or injection ports.
- Automated contrast injectors should not be connected to standard central venous lines. The high pressure developed by the rapid injection of viscous medium down a long, narrow lumen can burst the line. PowerLine® catheters allow power-injection of contrast media.
- Contrast media may cause kidney injury in patients with dehydration or impaired renal function, so ensure patients are adequately hydrated. Lactic acidosis can be precipitated in patients taking biguanides (metformin)—ideally avoid for 48h before and after the scan.

Practical considerations

- Move metal-containing objects (e.g. ECG leads, pressure transducer cables) away from the area being scanned to prevent X-ray artefact.
- Thoracic and abdominal scans may require 'breath-holds' of a few seconds to reduce respiratory movement artefact. Both paralysed and spontaneously breathing patients can be ventilated manually, and their lungs held in inspiration for each individual scan.
- The patient's arms ideally need to be positioned above the head during thoracic and abdominal scans. Soft Velcro straps attached to the gantry or wide adhesive tape are useful for securing the limbs.

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Anaesthesia for MRI

MRI uses superconducting electromagnets to generate powerful magnetic fields. Computers create images from the radiofrequency signals generated by hydrogen nuclei that move in and out of alignment with the high-frequency magnetic pulses. Most scanners use 1.5–3.0T magnets, but higher-strength machines are entering clinical practice. MRI produces detailed images, particularly of soft tissues, and is free from the dangers of ionising radiation. Increasingly, invasive procedures are being performed within MRI scanners, including some operations. Provision of safe anaesthesia for MRI requires specialised equipment and careful organisation.

The MRI scanner is an unpleasant claustrophobic environment—the patient must lie in a narrow, noisy tunnel, with the imaged body part surrounded by a radiofrequency coil (a frame-like device). Typical scans last 15–25min, but complex scans may take much longer. Up to 3% of adults cannot tolerate scanning without sedation or anaesthesia.

Hazards

- Displacement from static magnetic field: small ferromagnetic materials within the 3mT field can become projectiles which may injure or kill, while large objects can crush or trap. Foreign bodies (some heart valves, aneurysm clips, steel splinters in the eye) can move, causing haemorrhage or blindness. Implanted devices (cardiac pacemakers or neurological stimulators) may move or be inactivated or reprogrammed. Electric motors in syringe pumps may run erratically, and magnetic media (credit cards, mobile phones) will be erased. Beyond the 0.5mT boundary is considered safe.
- Noise: rapid magnetic field changes can cause >80dB noise and damage hearing. Use MRI-safe earplugs or defenders during scan.
- Oscillating radiofrequency fields can cause heating, rapidly causing severe burns. Remove or limit conductive materials, e.g. metal in clothing. Pacemaker wires may also heat—ensure they are MRI safe.
- Helium escape: in the event of an emergency magnetic field shutdown (a 'quench'), liquid helium coolant rapidly expands to a gas. This should vent outside the building, but some may enter the MRI suite, causing a hypoxic environment, requiring urgent evacuation.
- Gadolinium contrast agents are used in up to 30% of scans. Side effects include headache, nausea, dizziness, local burning and wheals. Severe hypotension/anaphylactic reactions are rare (~1:100 000). Newer agents are associated with lower risk of nephrogenic sclerosing fibrosis.

Patient and staff safety

- To avoid injury, all patients having an MRI scan must complete a screening/consent form. For children or sedated ICU patients, such forms must be completed on their behalf by relatives or staff.
- Staff must also complete a screening questionnaire and leave metallic objects, mobile phones, pagers, wallets, etc. outside the room.
- Patients and staff with cardiac pacemakers must remain outside the 0.5mT boundary.

Equipment safety

All equipment and implants are classified into one of three categories:

- MRI Safe: contains no material that would cause a hazard. Can be taken right into the scanner.
- MRI Conditional: safe for use under conditions specified by the manufacturer. Do not place MRI conditional kit on the moving table as it might move beyond the 5mT line.
- MRI Unsafe: must not be taken into the MRI scanner.

The scan room is usually shielded to stop external electrical interference swamping the MRI signals. All electrical equipment in the scan room must also be fully shielded. Electrical conductors entering the room (e.g. monitoring cables) require special radiofrequency filters.

Two alternative approaches are feasible

- Specialised 'MRI Conditional' equipment within the scan room
- Conventional kit outside the magnetic field in the control room.

Departments should standardise on one approach, depending on space, funds and frequency of use. Each approach has its pros and cons.

Typical setup

- Induction and recovery area adjacent to, but outside the scan room, equipped with conventional anaesthetic machine and monitoring.
- Non-magnetic tipping trolley for patient transfer into the scanner.
- Piped gases, scavenging and suction in induction and scan areas.
- **Either** a compact anaesthetic machine and ventilator in the control room with a 10m coaxial (Bain) breathing system and a gas/agent side-stream analyser with capnograph display fitted with an extended sampling tube (increases the response time by 5–10s).
- **Or** an 'MRI Conditional' anaesthetic machine in the scan room with circle circuit.
- 'MRI Conditional' monitoring devices: fibreoptic pulse oximeter probe with shielded cable; ECG with carbon fibre leads and electrodes; NIBP cuffs with an extended hose and non-metallic connectors.
- Multiple manufacturers produce 'MRI Conditional' monitor units within the scan room, with a slave unit in the control room.

Practical considerations and techniques

Airway

- Patient access is restricted physically and 'magnetically'. Ensure the airway and vascular access are well secured.
- Intubate and ventilate: babies and small children (<10kg), patients with raised ICP (or suspected raised ICP) or patients needing a protected airway. A RAE tube keeps the breathing circuit clear of the coil in patients having head scans.
- SV via an SGA can be considered in larger children and adults with no risk of raised ICP. Do not use a flexible LMA containing a metal wire spiral. i-gels have no metallic components.
- Tape the pilot balloon of a cuffed ETT or LMA outside the coil to avoid image distortion by the metal spring.

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Sedation

- Benzodiazepine sedation (PO or IV) may be used for healthy, but claustrophobic adults. Strong analgesia may be required for patients with severe back or root compression pain to tolerate positioning.
- The role of sedation for MRI scanning in children is unclear. Some children's centres have reported successes with structured sedation programmes, e.g. using dexmedetomidine. However, the safety of having heavily sedated children in the medical imaging department without direct anaesthetic supervision has been questioned.

Tips for IPPV through a 10m breathing system

- Use a system that functions as a 'T-piece' (Mapleson D or E), so dead space is unaffected by the length. Ayre's T-piece and Bain systems work well. Both can be used for babies and small children.
- Airway pressures measured near the ventilator may not accurately represent distal pressures at the ETT.
- V_T delivered to the lungs will be reduced by 'compression losses' of the gas within the system and by expansion of the tubing during inspiration, making it difficult to compensate for significant leaks around uncuffed ETTs—use a slightly larger tube to minimise leaks.
- As a result of these effects, IPPV using a simple pressure generator (e.g. Penlon Nuffield 200 with a Newton valve) may not be effective in children weighing >15kg.
- ↑ expiratory resistance of some long systems (e.g. Ayre's T-piece) generates a PEEP which increases with FGF.

Intensive care patients

- MRI scans in ICU patients confer greater risk and require detailed planning. The risk/benefit balance should be assessed by senior clinicians. Avoid scanning patients who are haemodynamically or otherwise unstable unless this will have a substantial impact on outcome.
- As the patient may lack capacity, full checks (including, if necessary, plain radiographs) must be performed to confirm there are no hazardous metallic implants or foreign bodies present.
- Conventional monitoring (including ICP transducers and temporary pacing wires) should be removed or replaced with 'MRI Safe' equipment before the patient enters the scan room.
- Infusion lines must be long enough to allow pumps to be located at a safe distance from the magnet—ideally outside the scan room. Prepare duplicate pumps in the control room, with extended infusion lines threaded with the breathing system into the scan room. Connect the patient to the running infusions while outside the room. Be especially cautious with infusions such as noradrenaline—check the patient is stable, then move into the scanner.

Cardiac arrest

- Start basic life support (BLS) with a non-metallic self-inflating bag. Rapidly remove the patient from the scan room on a non-magnetic trolley and continue ALS outside the 0.5mT boundary.
- Do not attempt ALS in the scan room and do not allow the cardiac arrest team into the scan room.

Anaesthesia for interventional radiology

Interventional radiology uses a variety of imaging modalities (fluoroscopy, CT, ultrasound or MRI) to guide minimally invasive diagnostic or therapeutic procedures. Interventional radiology procedures avoid open surgery, thereby reducing post-procedure pain and shortening recovery times. Interventional radiology suites are usually 'remote', in the X-ray department, but hybrid radiology/theatre suites are increasingly common.

Indications for anaesthesia

- Patients required to be very still for long periods of time
- Painful procedures
- Paediatric patients.

Anaesthesia for interventional radiology: general points

- Hazards are as previously described for CT and MRI.
- Interventional radiology suites are often 'isolated'—depending on the workload, invest in an anaesthesia trolley to securely store familiar equipment and drugs.
- Anaesthetic technique and airway choice depend on the patient's condition, planned procedure, positioning required, fasting status and the need for airway protection. Options include monitored care, sedation, GA and regional techniques or a combination.
- Scavenging may not be possible, so an activated charcoal absorber (e.g. Aldasorber) may be required or TIVA may be used. Induction and emergence generally take place within the radiology suite, while recovery may be in main theatres.
- The interventional radiology table usually does not tilt, will be controlled by a radiographer and may move during procedures. It is recommended to induce anaesthesia on an anaesthetic trolley and transfer the patient after induction.
- Ensure lines and tubes are secured and that there is sufficient circuit length to account for table and fluoroscopy C-arm movements.
- Access to the patient may be limited by radiological equipment—ensure you have IV access with a three-way tap and extension tubing.

Vascular procedures: angioplasty/stenting

- A balloon-tipped catheter is inserted into a narrow or blocked vessel, and the balloon inflated. A stent may be placed to keep it open. Some vascular, cardiac and trauma-related procedures do not require GA but require an anaesthetist for monitored care. Balloon dilation/temporary occlusion of certain vascular structures can cause vagal responses.
- Endovascular repair of AAA (see ➲ pp. 597–8) is associated with a lower mortality and may be favoured in significantly comorbid patients at high perioperative risk from open surgery. This may be done under regional, neuraxial anaesthesia (± sedation) or GA. Such procedures may now take place in hybrid interventional radiology–theatre suites.
- Patients may be required to lie supine for several hours after the procedure to prevent puncture site bleeding.

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Embolisations

- Blood vessels are selectively occluded to treat or prevent bleeding and stop tumour growth. A variety of agents, including metal coils, special foams, plugs and microbeads, are used to induce the embolus.
- Superficial procedures, the use of alcohol for embolisation or procedures involving AVMs can be very painful and require sedation or GA.
- Interventional radiology treatment of intracranial aneurysms requires GA because the patient must be completely still. Similar anaesthetic techniques as for craniotomy (see ➲ pp. 561–2; ➲ pp. 580–1), with an ETT, invasive monitoring, avoidance of changes in CPP and maintenance of normocapnia and normothermia, should be used.
- Uterine artery embolisation is included in NICE guidance [IPG367] for fibroid disease. Balloon occlusion of uterine arteries can be considered prior to CS in a patient anticipated to bleed, or can be inserted emergently in the management of 1° postpartum haemorrhage (though the logistical challenges to facilitate this transfer are often considerable).
- Chemoembolisation: combination of delivering cancer treatment directly to a tumour and then blocking its blood supply.
- Patients who require emergency embolisation for trauma-related haemorrhage (e.g. spleen, kidney, liver, intercostal arteries) require the presence of an anaesthetist, even if not having sedation or GA, to manage the transfusion and all other aspects of their care.

Radiofrequency ablation

- RFA destroys tissue by heating, e.g. a tumour. Although the ablation of the tumour is fairly quick, the localisation can take time and the procedure is painful and stimulating, so requires a GA.
- RFA is commonly used to treat hepatic and renal tumours—either metastases, difficult-to-reach tumours, or tumours in those patients who are too frail for an open procedure.

Thrombolysis and thrombectomy

- Minimally invasive dissolution, or removal, of blood clots to improve blood supply. Contrast media is used to help define the clot, which is then dissolved by medication or removed by a mechanical device. This can be used in the acute management of CVE, to treat arteries in diseased vascular beds, DVT, coronary emboli, PEs and thromboses in fistulae.
- Anaesthetic technique for acute CVE thrombectomy should be determined on an individual patient basis. Whether LA only, sedation or GA is chosen, adherence to standard physiological targets (see ➲ pp. 558–60; ➲ pp. 578–9) is required.

Transjugular intrahepatic portosystemic shunt

(See ➲ p. 698.)

- A stent, inserted via the jugular vein, is used to connect the portal vein and hepatic vein, thereby reducing portal hypertension and bleeding risk in patients with end-stage liver disease.
- GA with ETT and invasive monitoring is the preferred technique due to procedure duration and the patient's physiological condition and comorbid disease. Coagulopathy is common, may be profound, and should be corrected.
- Peri-procedural risks include haemorrhage, heart failure and encephalopathy. Postoperative care may require HDU.

Other procedures

- Vertebroplasty/cementoplasty: injection of cement into bone to reduce pain in tumours and fractures. Can be painful and requires sedation or GA.
- Cryoablation: destruction of tissue by freezing. Typically not painful, and often sedation is all that is required.
- Vascular catheter placement: sedation and LA are suitable for most patients; GA will be required for children.
- GI viscera can be dilated and stented. Oesophageal dilation can be painful and requires analgesia and consideration of requirement for airway protection in case of regurgitation.
- IVC filter insertion: not painful.

Fluid aspiration, biopsies and percutaneous drain placements

- Usually performed under ultrasound guidance, but may require CT for deeper structures. Typically not painful.
- Discuss positioning and apnoeic periods with the radiologist pre-procedure.

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Anaesthesia for cardiology procedures

These procedures generally take place in the cardiac catheterisation laboratory (the 'cath lab') and many of the additional considerations of anaesthesia for radiology apply. Establishing good team communication from the start is essential.

Indications for anaesthesia

- There has been a move away from 'single operator' procedures (cardiologist managing both procedure and sedation simultaneously) to regular anaesthetic support, with the aim of improving safety, efficiency and efficacy.
- Ablation procedures are facilitated by GA as this provides an immobile patient and ventilation control.
- Transcatheter aortic valve implantation (TAVI) avoids operative aortic valve replacement (AVR) and is done under either LA or GA in the cath lab.
- Angioplasties can be done under LA, but sedation may be required for patients agitated after out-of-hospital cardiac arrest.
- Cardioversions usually require just a brief face mask GA. Airway control may be needed for TOE for atrial clot.
- While some permanent pacemakers and devices can be inserted under LA (± minimal sedation), subpectoral chest wall insertion sites require GA. Wire removal always requires a GA as these procedures can be lengthy and complicated, with a risk of significant blood loss.
- Otherwise fit and well patients for electrophysiology studies may prefer a GA.

Anaesthesia: general points

- Choice of sedation or GA and the type of GA depends upon the needs of the patient and the nature of the procedure. Patients range from fit, well and coming in from home to those unwell on the coronary care unit, those septic from infected devices and those with critical AS deemed unsuitable for cardiac surgery.
- Before starting, check you have all the drugs drawn up that you anticipate using for anaesthesia and those you may want in an emergency (such as metaraminol, ephedrine, atropine and suxamethonium). Access to opioids or muscle relaxants can be delayed by your remote location.
- GAs may require several infusion pumps. Other equipment such as depth of anaesthesia monitoring or a jet ventilator (e.g. for atrial ablations) may be required and are likely to be stored centrally.
- Check the anaesthetic machine and suction and that monitoring is available, and consider the positioning with respect to both during induction of anaesthesia and afterwards. Consider the path of the C-arm and location of electrophysiology screens, defibrillator trolley, drip stands and pumps and also your own location in relation to this.
- Close monitoring and access to the patient will need to be balanced against operator access and your own radiation safety. A lead apron and thyroid screen is mandatory and regular visitors to the cath lab should wear a radiation monitoring device.

- Induction of the patient is most easily performed in the lab itself, but this comes with noise and distraction—be prepared to manage this. Make sure there is good access to the patient's airway, your drug and airway trolleys and anaesthetic machine when inducing anaesthesia on the procedure table. Move the C-arm out of the way, if possible.
- In some situations (difficult airway, risk of regurgitation, morbid obesity), it may be better to induce the patient on a trolley or bed to allow for a more head-up position or the ability to tilt the trolley.
- Maintaining normothermia can be challenging—use a fluid warmer and an underbody forced air warming device.
- Many patients are anticoagulated. For those who are not (or have had their anticoagulation reversed for the procedure), remember to consider VTE prophylaxis such as calf pumps.
- Ensure recovery facilities are available with an appropriately equipped recovery bay and experienced recovery nurses. In some situations, it may be necessary to transfer the patient to a theatre recovery area.
- Some patients will need to remain monitored on the cardiology ward overnight. The need to apply compression to groin access sites may limit the degree of head-up positioning in the early postoperative period.
- In the event of unexpectedly prolonged and complicated procedures, maintain contact with colleagues in theatres such as the duty anaesthetist or the on-call anaesthetic team. Breaks, advice and/or immediate help may be required. Anaesthetic activities running over time in remote sites can be easily overlooked.

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Anaesthesia for cardioversion

- Cardioversions can be elective or emergency and the former are most likely to be performed in the cath lab.
- Despite coming in from home, and therefore being relatively stable, patients often have documented poor ventricular function that has been worsened by their dysrhythmia. Other comorbidities are common. Patients are usually day case and in AF.
- A single synchronised shock is usual (maximum of three).
- Check anticoagulant compliance or adequacy of INR.
- May need definitive airway management for associated TOE (used to check if suspicion of atrial appendage clot), otherwise position head-up with face mask and preoxygenation. Have a backup airway management plan prepared.
- Propofol ± midazolam are usually sufficient for a face mask GA. Although cardioversion is very stimulating, it rarely requires opioids afterwards. Atropine may be needed for sinus bradycardia post-cardioversion.
- Placing pads front to back is most effective, especially in the obese or if previous repeated shocks. Consider *in situ* devices (e.g. permanent pacemakers) when placing pads.
- Before commencing anaesthesia, check the team is ready and discuss defibrillator safety; the operator may be anyone from a cardiology specialist nurse practitioner to an inexperienced junior doctor.
- If the patient has an ICD *in situ*, an electrophysiologist should be present. The device may be used to perform the shock; however, the battery drainage outweighs the potential risk of damage to the ICD, so an external defibrillator is preferred.
- Emergency cardioversions can be for atrial or ventricular dysrhythmias and performed anywhere in the hospital, depending on urgency. Unstable patients in the resuscitation bay will need very little in the way of sedation; unfasted patients may need RSI if high risk.
- Even in cases of urgency, skilled airway assistance for the anaesthetist must be available and all drugs and equipment should be checked.

Anaesthesia for angiography

Angiograms and angioplasty are most commonly performed under LA by the cardiologist, but some situations require a GA or sedation from the anaesthetist.

- Elective and urgent ward angiography patients will usually have their procedure under LA ± minimal sedation from the cardiologist.
- Patients who have survived an out-of-hospital cardiac arrest may need emergency angiography and revascularisation. They can be unstable but distressed and agitated, needing careful sedation. Aim for avoidance of further cardiorespiratory disruption or loss of consciousness, but prepare for the possibility of airway intervention and vasopressor/inotropic support. These patients will go to the coronary care unit post-procedure if stable enough.
- Unresponsive patients will need intubation and ventilation and transfer to ICU post-procedure. Conversely, patients already admitted to ICU after an arrest may require transfer to the lab for investigation.
- Challenges involve an unfamiliar environment and team, alongside the time pressures for revascularisation.
- Good communication between the anaesthetist and the cardiologist is essential to balance the need for safe patient monitoring against swift intervention and avoid risks such as exposure to radiation of a distracted anaesthetic team.
- Good venous access needs to remain easily accessible to the anaesthetist and if the patient is awake and agitated, they will need to hear reassurance. A plan and equipment for airway management must be in place if they are not already intubated. There should be dedicated skilled airway assistance available.

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Anaesthesia for cardiac device insertion and removal

- Devices may be permanent pacemakers, ICDs or have a combined function. Cardiac resynchronisation therapy (CRT) devices have three venous leads to mimic normal physiology while pacing and can be CRT-P (pacemaker) or CRT-D (defibrillator).
- S-ICD pacemakers are extravascular (subcutaneous) and placed in the chest wall between the serratus anterior and the latissimus dorsi, with tunnelling of leads towards the sternum.
- ICDs are placed in patients who are at risk of sudden death due to malignant cardiac arrhythmias. Patients range from young and otherwise fit adults with normal cardiac contractility to extremely compromised cardiac patients.
- Device insertion can be under LA alone or LA with sedation from the cardiologist, LA with deeper sedation (such as propofol TCI) from the anaesthetist, or GA.
- Fluoroscopy/X-rays will be used and so radiation protection is mandatory.
- Relevant factors to consider in anaesthetic planning are: indication, cardiac function, procedure requirements and duration and patient preference (Table 34.1).

Device insertion

- Pacemakers and ICDs that are not being tested are usually inserted by the cardiologist in the pacing lab under LA without the need for anaesthetic support.
- ICD insertion can be for 1° or 2° prevention and the latter group of patients are likely to have greater comorbidity.
- If the ICD requires testing (e.g. for patients with hypertrophic obstructive cardiomyopathy, complex anatomy or right-sided devices), this necessitates either deep sedation or a GA.

Table 34.1 Procedures and appropriate anaesthetic

Procedure	GA/LA/airway	Note
Permanent pacemaker insertion	LA ± sedation	Usually by cardiologist
ICD insertion	LA ± sedation	Can be 1° or 2° prevention
ICD insertion	GA (LMA/ETT)	If device test planned. Arterial line
S-ICD insertion	GA (with LA infiltration)	± regional blockade
CRT insertion	GA or LA + sedation	Poor ventricular function
Permanent pacemaker box change	LA ± sedation	GA if deep/wound revision
Lead/system extraction	GA (ETT)	May be infected. Risk of major haemorrhage

- If the device implantation is likely to be more stimulating than usual (sited in a deeper location, e.g. subpectoral or within chest wall S-ICDs), a GA is indicated.
- Chest wall regional blockade could be considered (instead of GA) for the insertion of SC ICDs in high-risk grown-up congenital heart (GUCH) patients.
- Insertion of devices for CRT can take longer and need anaesthetic support. These patients will, by definition, have more comorbidities and be at higher risk.
- Prophylactic antibiotics are given for device insertion.

Device extraction

- Devices/systems are usually extracted for infection, lead migration or breakage. Box changes alone should be relatively straightforward, but lead extraction can be high risk, depending on the number and location of leads, duration *in situ* and likely degree of scarring and extraction difficulty.
- In all lead extraction cases, major haemorrhage should be prepared for and difficult cases should have a cardiac surgeon and cardiac bypass standing by.
- Although a single-lumen ETT is standard, consider a DLT if very high concern (e.g. in extractions with a previous midline sternotomy for access to the SVC and brachiocephalic vein if needed.)
- Patients should not be anticoagulated or anaemic, and should have blood available and large venous access *in situ*, preferably right-sided.
- Although the cardiologist can gain arterial access femorally at the start of the procedure, a dedicated radial arterial line can be used for induction and emergence/recovery, leaving femoral access free for bypass intervention.
- Team brief should include discussion of potential adverse events and a clear plan for what happens should they occur.
- Infected leads and systems will require antibiotics at induction if not already given, e.g. teicoplanin. Patients may be systemically unwell and/or chronically septic.
- If the system is infected, a new device is not usually inserted immediately as the infection needs to be treated first, so a temporary pacing wire may be needed in the interim, depending on the underlying dysrhythmia. Pacing access may be femoral or via the neck veins.

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Anaesthesia for electrophysiology procedures (AF and VT ablation)

With advances in technology, mapping and ablation procedures are becoming more frequent. Ablation is now commonly performed as a 2nd-line therapy for both paroxysmal and persistent AF. Ablation can also be performed for atrial flutter and ventricular arrhythmias such as VT. Common ablation techniques use radiofrequency energy (burning) or cryothermy (freezing).

Preoperative considerations

- Most patients come in from home on the day and are functionally well, in either sinus rhythm or rate-controlled AF.
- However, there may be those who have poor cardiac function destabilised by their dysrhythmia and unstable inpatients with resistant VT on the coronary care unit.
- While solely right-sided procedures may be day cases, most will require an inpatient bed overnight.
- Anxious patients may require GA for electrophysiology studies.

Procedural points

- Long procedures (2–4h) requiring minimal patient movement and/or ventilation control mean that GA is preferable.
- The main target for atrial ablation is isolation of the four pulmonary veins. For persistent AF, left atrial 'substrate' ablation may also be needed, prolonging the procedure.
- RFA requires mapping systems to identify arrhythmia trigger sites and pathways, necessitating careful positioning of multiple electrodes by the lab electrophysiologists (usually applied prior to anaesthetic monitoring).
- There will be ionising radiation used for much of the procedure.

Ventilation and access

- There is respiratory excursion compensation within the mapping systems, but mechanical ventilation should also be optimised.
- IPPV with a low V_T and high RR can be used throughout or in conjunction with periods of jet ventilation via a jet catheter placed inside the ETT (Monsoon/Mistral ventilator, frequency 150, for 30–60min). Some centres use jet ventilation throughout; however, lung disease or obesity may limit this. Use a TIVA anaesthetic with EEG/BIS® monitoring if jet ventilation is used.
- Only a large accessible peripheral access is required for simple atrial ablation, unless other patient comorbidities make an arterial line advisable.
- Ventricular ablations usually require an arterial line either at induction or prior to the procedure. The cardiologist can gain arterial access (and central venous, if needed) in the groin and a pressure waveform can be slaved from this.

Other considerations

- The procedure table is narrow and does not tip or go head up. Table controls may be distant and unfamiliar—the radiographer can assist. Patients with a difficult airway or obesity may be better anaesthetised on a trolley and transferred once asleep.
- Use an 'underbody' forced air warmer with temperature monitoring.
- Femoral venous access is used to access the heart; remifentanil infusion as part of the anaesthetic technique can reduce coughing at extubation and subsequent groin haematoma.
- Sodium chloride 0.9% irrigation around the ablation catheter tip results in a significant fluid load (1.5L). Balance with restricted fluids ± vasopressors if needed, as urinary catheterisation is not usually performed.
- In the cath lab, the WHO sign-in is performed with the whole team.
- Once the patient is asleep, wires, monitors, drip stands and the anaesthetic machine need to be positioned to avoid the C-arm and allow visualisation of the monitor and patient access as required.

During the procedure

- Relatively unstimulating procedure—BP support may be required.
- TOE can be needed to visualise trans-septal left atrial access when challenging with fluoroscopy alone.
- Heparin is always given for left-sided procedures, even when the patient is anticoagulated. ACT target usually >300s and reversed by protamine at the end; this is usually managed by the cardiologist.
- Induced arrhythmias may cause haemodynamic instability and it can be necessary to cardiovert the patient during the procedure.
- Isoprenaline and adenosine may be given at the discretion of the cardiologist towards the end of the procedure and will cause tachyarrhythmias and brief asystole, respectively.
- Analgesia requirements are minimal—LA at access sites, IV paracetamol and antiemetics are sufficient.
- An in–out catheter can improve comfort in recovery.

Complications

- Include vascular injury/pseudoaneurysms/haematomas/A–V fistulae (1:100).
- The cardiologist will routinely perform a TOE at the end of the procedure to check for tamponade; a sudden unexplained drop in BP during the procedure could indicate tamponade and should be communicated (1:100–1:200 risk).
- Oesophageal injuries such as perforation or atrio-oesophageal fistula are serious complications but typically present 7–10d post-AF ablation.
- Phrenic nerve injury is usually transient and is particularly common during cryoballoon ablation of AF.
- Pericardial effusion (1:100–1:200).
- CVE (1:250).

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Anaesthesia for transcatheter aortic valve implantation

- Increasingly common procedure to avoid operative AVR. Initially performed in high-risk patients (frail/elderly/comorbidities), indications now extending to lower-risk patients.
- Establish extent of appropriate intervention in case of complication, e.g. suitability for sternotomy/bypass/CPR.
- Most commonly performed transfemorally (percutaneous); alternatively, transapically (mini-thoracotomy) where there are issues with femoral access. Less common approaches include transaxillary, transaortic and transcaval.
- Transfemoral approach usually performed under LA with sedation.
- Requires good peripheral access. Arterial and central venous pressures can be measured from procedural access. Sedation requirements can be minimal and depend on the individual. Prepare patient for intervention points, i.e. LA infiltration, device insertion and valve deployment.
- Transapical/transaortic approach—set up as for CPB: arterial line, CVP, large-bore IV access, urinary catheter, external defibrillator pads.
- Typically TCI for GA (e.g. remifentanil, propofol, relaxant; intubate and ventilate with O_2/air).
- Have emergency drugs prepared for all TAVI procedures. Consider noradrenaline infusion to maintain systemic BP.
- Procedure involves rapid atrial pacing to permit valvotomy and placement of the prosthetic valve—maintain BP (>100mmHg) before this starts, and have adrenaline (1:10 000) drawn up, as the heart may be stunned immediately following this. Ventilation does not need to stop for valve positioning.
- Have hypotensive agents (e.g. esmolol, GTN) available post-valve insertion, as hypertension can be marked.
- Analgesia: infiltrate all wounds with LA and give IV paracetamol for postoperative pain relief; consider paravertebral block for mini-thoracotomy.
- For the transapical approach: more analgesia is required; blood should be available in the angiography suite for LV apical ventriculotomy, and consider cell salvage. Early extubation is ideal.
- Recover supine (femoral access closure), with supplemental O_2, and recover with full monitoring as for any anaesthetic.
- Be aware of TAVI complications—these include arrhythmia/heart block/tamponade/rupture/valve malposition or migration/coronary occlusion/CVE.

Special considerations

- Digoxin increases the risk of arrhythmia—omit on the day.

Further reading

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Chapter 35

Obstetric anaesthesia and analgesia

James Eldridge, Nicola Cox, Alisha Allana and Heidi Lightfoot

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See also

➲ Obstetric anaesthesia in the patient with a spinal cord injury pp. *307–8*
➲ Pregnancy and trauma p. *988*

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Physiology and pharmacology

From early in the 1st trimester, a woman's physiology changes under the influence of increasing progesterone and oestrogen production. The effects are widespread.

- CO increases by ~50%. Diastolic BP falls in early pregnancy and is at its lowest at 20w gestation, returning to prepregnant levels by term. Systolic BP follows the same pattern but is less affected. CVP and pulmonary capillary wedge pressure are not altered.
- CO increases further during labour, peaking immediately post-delivery. Preload and afterload change rapidly at delivery. This is a period of high risk for women with impaired myocardial or valvular function.
- Uteroplacental blood flow is not autoregulated and so is dependent on uterine artery BP.
- Aortocaval occlusion occurs when the gravid uterus rests on the aorta or the IVC. Near term, complete caval occlusion in the supine position is almost universal. Most women have sufficient collateral circulation, so only 10% develop overt supine hypotensive syndrome. Even if normotensive, placental blood supply may be compromised in the supine position. After the 20th week of gestation, a left lateral tilt or uterine displacement should be employed. If either mother or fetus is symptomatic, the tilt should be increased.
- Plasma volume increases by 50% by term while red cell mass only increases by 30%, resulting in physiological anaemia of pregnancy.
- Pregnant women become hypercoagulable early in the 1st trimester and it is at this time deaths from PE occur most commonly. Plasma concentrations of factors I (fibrinogen), VII, VIII, IX, X and XII increase, but antithrombin III levels decrease. All pregnant women undergoing surgery should be risk-assessed for thromboprophylaxis.
- $PaCO_2$ decreases to ~4.0kPa (30mmHg) in early pregnancy. FRC decreases by 20% at term, resulting in airway closure in 50% of supine women. This, combined with a 60% increase in O_2 consumption, renders pregnant women at term vulnerable to hypoxia when supine, especially if combined with a raised BMI.
- In labour, painful contractions and excessive breathing of Entonox® can result in further hyperventilation and marked alkalosis may occur. An arterial pH in excess of 7.5 is common.
- While whole gut transit time is increased in pregnancy, gastric emptying and acidity are little changed. However, gastric emptying is slowed in established labour and almost halted by systemic opioids. Barrier pressure (the difference in pressure between the stomach and the lower oesophageal 'sphincter') is reduced, but the incidence of regurgitation into the upper oesophagus during anaesthesia in otherwise asymptomatic individuals is not significantly different in the 1st and 2nd trimesters
- By 48h postpartum, intra-abdominal pressure, gastric emptying, volume and acidity are all similar to non-pregnant controls. Lower oesophageal sphincter tone may take longer to recover, but in the absence of other indications for intubation, mask anaesthesia is acceptable 48h after delivery.

- Renal blood flow increases by 75% at term and GFR by 50%. Both urea and creatinine plasma concentrations fall.
- Neurological tissue has a greater susceptibility to the action of LA during pregnancy and 'MAC' is also reduced.
- The volume of distribution increases by 5L, affecting predominantly polar (water-soluble) agents. Lipid-soluble drugs are more affected by changes in protein binding. The fall in albumin concentration increases the free active portion of acidic agents, while basic drugs are more dominantly bound to α-1 glycoprotein. Some specific binding proteins, such as thyroxine-binding protein, increase in pregnancy.
- Plasma cholinesterase concentration falls by about 25%, but due to the increased volume of distribution, the duration of action of suxamethonium is little changed.

ALGRAWANY

Analgesia for labour

- The three most commonly used analgesics in labour are inhaled N_2O, opioids and CNB techniques—spinals, combined spinal/epidurals (CSEs) and epidurals.
- For many women, acceptable analgesia in labour does not mean complete absence of all sensation and there are numerous practices that can help mothers through labour. These include prepared childbirth, massage, acupuncture, warm water baths and transcutaneous electrical nerve stimulation (TENS). Although the evidence that these alter pain scores is weak, they are all useful techniques.
- N_2O/O_2 (Entonox®) is the most commonly used inhalational agent and is possibly more efficacious and safer than a single dose of pethidine. However, complete analgesia is never attained with it. Entonox® can induce nausea and presyncope, and with prolonged exposure, N_2O has adverse effects on the haematological and immunological systems. N_2O is a greenhouse gas.
- Worldwide, pethidine remains one of the most popular opioids for labour analgesia. However, it has a long half-life in the fetus (18–23h), reduces fetal HR variability, is associated with changes in neonatal neurobehavioural scores and can affect breastfeeding. If regional analgesia is contraindicated, a fentanyl or remifentanil PCA may be more beneficial (see pp. 1162–3). IM diamorphine is also used in the UK, although it may prolong labour by over an hour.
- CNB analgesia remains the most effective form of analgesia.
- Uterine pain is transmitted in sensory fibres which accompany the sympathetic supply to T10–L1, while vaginal pain is transmitted via the pudendal nerve to S2–S4 nerve roots. Paracervical and pudendal nerve blocks can block the pain of labour, but CNB techniques are more effective and have largely replaced them.
- CNB techniques can be expected to provide effective analgesia in over 85% of women. CNB analgesia is associated with hypotension, ↑ oxytocin use and ↑ incidence of maternal pyrexia, but is not associated with an increase in CS rates. With low concentrations of epidural LA (i.e. ≤0.1% bupivacaine), there is no increase in the instrumental delivery rate. Fetal umbilical pH is marginally improved with epidural analgesia.
- Using 'low-dose' epidural techniques can reduce the incidence of hypotension and motor blockade and increase maternal satisfaction. The instrumental delivery rate can be reduced by:
  - Decreasing the total dose of LA administered by establishing, as well as maintaining, CNB analgesia with low-dose LA and opioid
  - Using patient-controlled epidural analgesia (PCEA) or intermittent top-ups to maintain analgesia. In general, infusions deliver a greater total dose of LA than intermittent top-ups, while PCEA delivers the smallest total dose.

Central neuraxial blocks for labour analgesia

See also pp. 1114–17.

Indications

- Maternal request.
- Expectation of operative delivery.
- Conditions which might make GA difficult or life-threatening (e.g. morbid obesity, difficult airway).
- Maternal comorbidities, in particular those where sympathetic stimulation might cause maternal or fetal deterioration, specific CVS disease (e.g. regurgitant valvular lesions), severe respiratory disease (e.g. CF), specific neurological disease (e.g. intracranial AVMs, spinal cord injury) and some obstetric disease (e.g. pre-eclampsia without coagulopathy or thrombocytopenia).

Absolute contraindications

- Maternal refusal or allergy.
- Local infection or severe systemic sepsis.
- Uncorrected hypovolaemia or uncontrolled haemorrhage.
- Coagulopathy: expert opinion on precise values when CNB cannot be used varies; always consider risk and benefit for individual patients. Spinal analgesia is probably safer than epidural analgesia. Generally, in the absence of pharmacological agents that affect clotting, a platelet count >75 × 10^9/L with a normal clotting screen is considered safe. However, a slightly lower platelet count may be acceptable in patients at high risk from GA. Tests of coagulation and platelet count should usually be within 6h of the procedure, but also consider the rate at which the platelet count is falling. The AoA guidelines can be accessed online.[1]
- Raised ICP (excluding the majority of individuals with communicating idiopathic intracranial hypertension).

Relative contraindications

- Expectation of significant haemorrhage.
- Untreated systemic infection.
- Specific cardiac disease (e.g. severe valvular stenosis, Eisenmenger's syndrome, peripartum cardiomyopathy).
- Previous back surgery with scarring of the epidural space may cause difficulty in placement of an epidural, increase the risk of a dural puncture and may reduce the analgesic efficacy. Intrathecal techniques would be expected to work normally.

Consent

Most UK anaesthetists do not take written consent before inserting an epidural for labour analgesia, but an 'appropriate' explanation must be given. Many women do not accurately recall information given in labour. The explanation and all the adverse effects discussed should be documented. The Obstetric Anaesthetists' Association has produced an information website and card for mothers, available in multiple languages, which includes a quantitative estimate of the incidence of a variety of potential complications, including neurological injury (https://www.labourpains.com).

ALGRAWANY

Epidural analgesia for labour

- Make sure a trained midwife is available to provide one-to-one care.
- Establish IV access. In the absence of previous haemorrhage or dehydration, when low-dose LA techniques are used, fluid loading is unnecessary.
- Sterile technique: mask, hat, gown and gloves should be worn.
- Position either in full lateral or sitting (finding the midline may be easier when sitting).
- Record fetal HR before and during the establishment of analgesia.
- Skin sterilisation with 0.5% chlorhexidine is common in the UK. Most sterilising solutions, including chlorhexidine, are neurotoxic. Avoid contamination of the CNB equipment or the anaesthetist's gloves. It is sensible to complete skin sterilisation before the neuraxial equipment is unwrapped. Chlorhexidine must be allowed to dry before the skin is touched.
- Locate the epidural space using a Tuohy needle. Loss of resistance to 0.9% sodium chloride may slightly reduce the incidence of accidental dural puncture and 'missed segments' when compared to loss of resistance to air.
- The chance of puncturing a vessel with the catheter is reduced if 10mL of 0.9% sodium chloride is flushed into the epidural space before inserting the catheter. Warn about discomfort when threading the catheter.
- Leave 3.5–5cm of catheter in the epidural space; longer increases risk of unilateral block and shorter increases risk of catheter pulling out of the epidural space.
- Aspirate the catheter to check for blood or CSF. If blood is aspirated, withdraw the catheter, leaving a minimum of 3cm in the space. If blood is still present, remove the catheter and reinsert.
- Give a test dose. The dose should be sufficient to produce a significant but not dangerously high block within 5min if the catheter is unexpectedly in CSF. Testing for IV placement of the catheter using 1:200 000 adrenaline has both high false positive and negative rates. Many anaesthetists will use 8mL of 0.1% bupivacaine with a dilute opioid (2 micrograms/mL fentanyl) as the test dose.
- After 5min, load with further (dilute) LA to establish analgesia (12mL of 0.1% bupivacaine with 2 micrograms/mL fentanyl).
- The complete absence of a detectable block after a loading dose should raise the suspicion of a malpositioned catheter. Remember 'every dose is a test dose'!
- Measure BP every 5min for at least 20min after every bolus dose.
- Epidural analgesia can be maintained by one of four methods:
  - PCEA. A variety of regimens have been proposed. In general, larger volumes of low-concentration bupivacaine with opioid produce more effective analgesia, e.g. 5–10mL boluses of 0.1% bupivacaine with 2 micrograms/mL fentanyl and a 15–20min lockout period.
  - A continuous infusion of LA (5–12mL/h of 0.1% bupivacaine with 2 micrograms/mL fentanyl).
  - Intermittent top-ups of LA administered by midwife (rare in the UK—superseded by PCEA).
  - Programmed intermittent epidural boluses. These have a reduced risk of breakthrough pain compared to continuous infusions, without increasing the risk of an instrumental delivery.

Combined spinal/epidural analgesia for labour

- Low-dose subarachnoid LA and/or opioid, together with subsequent top-ups of weak epidural LA, produces rapid onset of analgesia with minimal motor block (10–15min quicker than epidural alone).
- CSEs can be advantageous when re-establishing analgesia for women who have had a failed epidural or for women who are so distressed by pain that they are unable to remain still while an epidural is sited. They are used routinely in some centres.
- There is some evidence of improved epidural analgesia after the initial spinal analgesia has receded.

CSEs can be performed as a needle-through-needle technique or as separate injections in the same or different intervertebral spaces.

Needle-through-needle

- Locate epidural space at the L3/4 interspace or below, with a Tuohy needle. The level of the iliac crests usually corresponds to the spinous process of L4 (Tuffier's line), although there is variation between individuals. Pass a 25–27G pencil-point needle through the Tuohy needle to locate the subarachnoid space and inject subarachnoid solution (e.g. 0.5–1.0mL of 0.25% bupivacaine with 5–25 micrograms fentanyl).
- Without rotating the epidural needle, insert an epidural catheter.

Separate injection technique

- Perform spinal at L3/4. Inject subarachnoid solution (e.g. 0.5–1.0mL of 0.25% bupivacaine with 5–25 micrograms fentanyl).
- Insert an epidural catheter at a different interspace.
- This technique is particularly helpful when women are too distressed to cooperate. Once spinal analgesia is established, an epidural can be performed with a more compliant patient.

After 15min, check the sensory and motor block and then give the epidural test dose. If the epidural has been unintentionally sited intrathecally, the block will change significantly within 5min.

Continue maintenance as per epidural analgesia alone (see pp. 855–6).

Dural puncture epidural

The dural puncture epidural is a modification of the combined spinal epidural.[2] A dural puncture is performed with a 25G (not 27G) pencil-point needle. No intrathecal agents are injected and then an epidural is inserted and run in the usual fashion. The analgesia obtained is slightly quicker in onset than a standard epidural and the quality of analgesia is slightly better. This is probably a result of epidural solution leaking into the CSF through the hole created by the 25G needle. The incidence of maternal hypotension, pruritus and uterine hyperstimulation is lower than with a CSE.

ALGRAWANY

The poorly functioning epidural

Look for the pattern of failure (Table 35.1). Remember that a full bladder may cause breakthrough pain. It is important to be confident that the epidural could be topped up for a CS if required. Therefore, if in doubt, resite the epidural. (See also ➔ p. 1164.)

Table 35.1 Solutions for the poorly functioning epidural

Pattern of failure	Remedy
No detectable block despite at least 10mL of 0.25% bupivacaine (or equivalent)	Resite epidural
Unilateral block Assess the block: Are both feet symmetrically warm and dry? Is the pattern of sensory blockade consistent with where the pain is felt?	1. Top up in a lateral position, with the painful side down. Use dilute, large-volume LA and opiate (this is usually more effective than high-concentration, low-volume solutions) 2. Withdraw the catheter 2–3cm and give a further top-up 3. Resite epidural
Missed segment True missed segments are rare. Commonly, a 'missed segment' felt in the groin is a partial unilateral block	1. Top up with opioid (i.e. 50–100 micrograms fentanyl). The intrathecal mode of action will minimise segmental effects 2. Continue as per 'unilateral block'
Back pain Often associated with an occipito-posterior position of the fetus and may require a dense block to establish analgesia	1. Top up with more LA and opioid. To achieve a sufficiently dense block, higher concentration of LA is occasionally required
Perineal pain	1. Check sacral block and that the bladder is empty 2. Top up with more LA in sitting position 3. Continue as per unilateral block

Complications of CNB analgesia

Around 25% of labouring women in the UK receive epidural analgesia, and the obstetric population as a whole account for 45% of all CNBs. Long-term complications after CNB occur less frequently in the obstetric population than in the general population, probably because the duration of epidural analgesia is relatively short and the population is generally healthy and younger.[3]

Hypotension

Hypotension is common after CNB **anaesthesia**, but less so after CNB **analgesia**. Aim to maintain systolic BP >90% of baseline. Uterine blood flow is not autoregulated, so prolonged or severe hypotension will cause fetal compromise. Fluid loading is not routinely required when using low doses of LA, but if hypovolaemia is suspected, this should be corrected before starting CNB analgesia.

If hypotension occurs, treat quickly:

- Position patient fully laterally or sitting up. Avoid aortocaval occlusion.
- Give an IV fluid bolus of crystalloid solution.
- Give vasopressor (e.g. 6mg IV ephedrine) and repeat as necessary.
- If fetal distress occurs, call obstetrician. Consider O_2 via face mask.

Remember that brachial artery pressure may not reflect uterine artery blood flow. If fetal distress is detected and is chronologically related to a CNB procedure, treat as above, even in the absence of overt hypotension.

Subdural block

Subdural block occurs when the epidural catheter is misplaced between the dura mater and the arachnoid mater. In obstetric practice, the incidence of clinically recognised subdural block is <1:1000 epidurals. However, subdural blocks may be clinically indistinguishable from epidural blocks. Definitive diagnosis is radiological. The classical characteristics of a subdural block are:

- A slow onset (20–30min) of a block that is inappropriately extensive for the volume of LA injected. The block may extend to the cervical dermatomes and a Horner's syndrome may develop.
- The block is often patchy and asymmetrical. Sparing of motor fibres to the lower limbs may occur.
- A total spinal may occur with top-up doses. This is probably due to an increase in volume causing the arachnoid mater to rupture.

If a subdural placement is suspected, resite the epidural catheter.

Total spinal

Total spinals are the commonest cause of maternal cardiac arrest in UK delivery suites. The incidence of unexpectedly high blocks or total spinal is variously reported to be between 1:100 to 1:100 000 CNBs, depending on definition. Symptoms and signs are usually immediate, although delayed onset may occur, possibly caused by a change in maternal position or a subdural catheter placement. Spinal blocks performed in the presence of a partially functioning epidural block have a high risk of causing unexpectedly high blocks or a total spinal.

ALGRAWANY

The onset of a total spinal usually follows a sequence of initial difficulty in coughing (commonly seen during CNB anaesthesia for CS), then loss of hand and arm strength, followed by difficulty with talking, breathing and swallowing, then loss of consciousness.

If a block is rising abnormally rapidly, think about the likely cause:

- If the high block followed a subarachnoid injection of hyperbaric LA (e.g. most spinals), or followed an excess dose into a correctly sited epidural catheter, position the patient head up.
- If the high block followed either an intentional or an unintentional injection of plain LA into the subarachnoid space (e.g. with an unrecognised spinal catheter, rather than an epidural catheter), remember that plain LA is hypobaric compared to CSF, so a head-up position may encourage the block to spread further. Gently position the mother in a left lateral position to minimise dural compression through epidural vein engorgement (which occurs with caval occlusion) and observe very closely. Sudden movements may cause CSF to move further cephalad.
- Make sure that the equipment for ventilatory and cardiovascular support are immediately available. Respiratory paralysis, cardiovascular depression, unconsciousness and fixed, dilated pupils may ensue.
- Call for help and follow the ABC approach. Maintain airway and ventilation; avoid aortocaval compression and provide CVS support.
- Even if consciousness is not lost, intubation may be required to protect the airway. A reduced induction dose is often necessary.
- Careful maternal and fetal monitoring is essential and, if appropriate, delivery of the fetus. In the absence of fetal distress, CS is not an immediate requirement.
- Ventilation is usually needed for 1–2h until the block recedes. Before extubation, check the patient has good handgrip strength.

Accidental IV injection of local anaesthetic

At the time of siting, 5% of epidurals catheters are completely or partially in a blood vessel. While these are usually detected and corrected, remember that 'every dose is a test dose'. The maxim is to avoid injecting any single large bolus of LA intravenously.

- Always checking for blood in the catheter
- Always being alert to symptoms of IV injection with every dose of LA, even when previous doses were uncomplicated
- Dividing all large doses of LA into small aliquots
- Using appropriate LAs
- Maintaining vigilance for incorrect connection of IVIs to epidural catheters. NAP3 reported a higher incidence of epidural to IV connections in obstetric patients. Using non-interchangeable CNB connectors (i.e. NRFit® systems) will reduce this risk.
- If neurological or cardiovascular symptoms occur, stop injecting the LA. Treat according to BLS/ALS protocols and administer 20% lipid emulsion (see pp. 1092–3).

Neurological damage

- Neurological sequelae following delivery under GA is as common as delivery under CNB anaesthesia, suggesting that obstetric causes from nerve compression (e.g. pressure from fetal head, prolonged labour or poor maternal positioning) are probably more common than any effects from the CNB technique.
- Direct nerve root damage may be associated with pain on insertion of a CNB. Temporary neurological deficits occur in roughly 1:3000 procedures. Prolonged neurological deficit is much rarer (~1:15 000) and permanent major neurological damage probably occurs in <1:80 000 CNBs in the obstetric population.
- Neurological damage from potentially reversible causes, such as infection (e.g. vertebral canal abscess), ischaemia or haemorrhage (e.g. subdural haematoma), may be immediate or delayed.
- It is unusual to have a dense motor block (an inability to straight leg raise) in the context of low-concentration LA epidural infusions or if >4h have passed since the last high dose of spinal or epidural LA. Immediate anaesthetic review is indicated.
- If an epidural haematoma is suspected and an epidural catheter is *in situ*, this should remain until the diagnosis has been excluded as removal may worsen potential bleeding. Removal of epidural catheters in any patient should be done with consideration of coagulation status.
- Symptoms from a vertebral canal abscess can occur anytime between a few days to several months after the CNB procedure. Diagnosis can be difficult, with fever, back pain, neurological deficit and signs of localised infection variably present. A high index of suspicion is required.
- Reversibility is time-critical. Depending on the context, urgent MRI with neurosurgical review and/or nerve conduction studies may be required.

Dural puncture and postdural puncture headache

(See ➲ pp. 848–51.)

ALGRAWANY

Dural puncture and postdural puncture headache

- When loss of CSF through a dural tear is greater than production, CSF pressure falls and the brain sinks, stretching the meninges. This stretching is thought to cause headache, sometimes with neck stiffness and cranial nerve involvement. Compensatory vasodilation of intracranial vessels may further worsen symptoms.
- Dural puncture may be intentional (as in CSE or spinal anaesthesia) or unintentional (as a complication of an epidural), and both may cause headache. Inadvertent dural puncture should occur in <1% of epidurals, although up to 40% of inadvertent dural punctures may not be recognised at the time of the procedure. If a dural puncture occurs with a 16G Tuohy needle, the incidence of postdural puncture headache (PDPH) is >50%.
- The risk of PDPH following spinal anaesthesia with a 25G pencil-point needle is 2–10%. These headaches are usually mild, self-limiting and often not recognised. The incidence of severe headache requiring blood patching is ~1:650. Symptoms may not develop for several days.
- All midwives, as well as obstetric and anaesthetic staff, should be alert to the signs of PDPH, as symptoms may not develop for several days. If untreated, headaches are not only unpleasant but, very rarely, can be life-threatening. Intracranial haemorrhage, subdural haematoma, cerebral venous thrombosis and coning of the brainstem have all been reported.

Management of accidental dural puncture can be divided into immediate management of an inadvertent dural puncture and late management if a PDPH develops.

Immediate management of inadvertent dural puncture

The aim is to achieve effective analgesia without further complication. If a dural puncture occurs, a catheter can be either threaded and left in situ (now an intrathecal catheter) or removed and an epidural catheter reinserted. Both techniques are described below. The patient should be informed at the earliest opportunity that a dural puncture has occurred and of the likely sequelae. All patients at risk of developing a PDPH should be reviewed daily by an anaesthetist until resolution.

Intrathecal catheter technique

- After a dural puncture, do not move the Tuohy needle. Pass 2–3cm of catheter into the subarachnoid space.
- Label the catheter clearly as an intrathecal catheter and only allow anaesthetists to perform top-ups.
- Give through the catheter intermittent top-ups of 1.0–2.5mg bupivacaine ± 5–25 micrograms fentanyl. Tachyphylaxis may occur with prolonged labour. Make sure that the dead space of the catheter and filter is known and accounted for (usually ~1.1mL). The dead space can either be left with a known solution within it or flushed with the appropriate volume of 0.9% sodium chloride after each top-up.
- Communicate and document which technique is being used.

The principle advantage of this technique is that the chance of performing another dural puncture is removed. Also, the need for epidural blood patch (although not the incidence of PDPH) may be reduced. While the quality of analgesia may be excellent, unfamiliarity with dosing, tachyphylaxis and movement of the intrathecal catheter out of the intrathecal space may compromise this.

The greatest risk of this technique is that the catheter may be mistaken for an epidural catheter.

Removal of the Tuohy needle/epidural catheter

- Reinsert the epidural at a different interspace, usually one space higher. Consider asking a senior colleague to help.
- Run the epidural as normal, but beware of intrathecal spread of LA causing high blocks.
- All top-ups should be given cautiously by an anaesthetist.

Beware... that when large top-ups are given for an operative procedure, intrathecal spread through the dural tear can result in an unexpectedly high block.

Late management of postdural puncture headache

Headaches in the postnatal period are common and other causes of headaches should be considered (dehydration, tiredness, migraines, anaemia, meningitis, SAH, venous sinus thrombosis, pituitary bleeds). Common features of a PDPH include:

- A history of a dural puncture (although in one-third of PDPHs following epidural analgesia, the original dural puncture was not recognised at the time of insertion).
- Onset 24–48h postdural puncture but can be up to 5d. Untreated, they are said to last 7–14d, but the evidence is poor.
- Headache made worse by standing. Headache is often absent after overnight bed rest but returns after mobilising. However, 5% of PDPHs are not postural.[4]
- The headache is usually fronto-occipital and may be associated with neck stiffness and muffled hearing.
- The headache may be relieved by tight abdominal compression which can be useful diagnostically.
- Photophobia and difficulty in accommodation are common. Hearing loss, tinnitus and CN VI nerve palsy with diplopia are possible. If these signs develop, women should be encouraged to have a blood patch as these are an indication of a more severe headache and the nerve injuries may not recover if the dural puncture is not treated promptly.

Treatment is either conservative, alleviating symptoms while waiting for the dural tear to heal, or actively attempting to 'seal' the puncture. Epidural blood patching is the only commonly used method of sealing dural tears, although neurosurgical closure has been performed.

Prophylactic treatment

There is a high incidence of bacteraemia shortly after delivery. This, combined with poor efficacy, means that prophylactic blood patching has fallen out of favour.

ALGRAWANY

Symptomatic treatment

- Bed rest alleviates symptoms, but the effect is usually transient and increases the risk of VTE. If immobile for ≥24h, consider VTE prophylaxis (time LMWH doses to avoid delaying blood patch if needed).
- Adequate fluid intake (usually oral) should be encouraged, although there is no evidence that hydration reduces the incidence of PDPH. Excessive fluid intake may be harmful.
- Simple analgesics are the mainstays of symptomatic treatment. They should always be offered, even though they are unlikely to completely relieve a PDPH.
- Caffeine reduces intracranial vasodilation which is partially responsible for the headache. There is limited evidence that the severity of PDPH is reduced and some concern that seizures may be increased with high-dose caffeine. A maximum of 900mg of caffeine is recommended per day, reduced to 200mg if breastfeeding. A high-energy drink or coffee contains ~150mg of caffeine.
- There is insufficient evidence currently to recommend routine use of any of the following: sphenopalatine ganglion block, greater occipital nerve blocks, theophylline, ACTH analogues, steroids, triptans, gabapentin, acupuncture, epidural opioid or fluid.[4]
- Definitive treatment is with epidural blood patching.

Epidural blood patch

Only a third of postpartum women with PDPH will have complete resolution of symptoms after an epidural blood patch, although 50–80% will report some benefit. Relapse can occur and the need for a 2nd blood patch is common.[5] Lower success rates occur if blood patches are performed <48h after the dural puncture.

The proposed mechanism of action of blood patch is twofold—blood injected into the epidural space compresses the dural sac and raises the ICP, which can produce almost instant improvement in pain. Secondly, blood forming a clot over the dural tear seals the CSF leak and the dural sac gradually refills.

Although serious complications after blood patching are rare, backache is common—35% of women experience some discomfort 48h post-patch and 16% have prolonged backache (mean duration 27d). Repeated dural puncture is possible, and neurological deficits, arachnoiditis, infection, epileptiform fits and cranial nerve damage have all been reported.

Technique

- Written information should be offered to the patient and written consent obtained. Consent should include reference to the success rate (as above), and that repeat dural puncture is possible, that back pain during and for several days after epidural blood patch is common and that there can be significant and rare complications, including nerve damage, bleeding and infection.
- The patient should be apyrexial with a normal WCC.
- Two operators are required. One should be an experienced 'epiduralist', and the other is required to take blood in a sterile manner.
- The patient may benefit from a period of bed rest before performing the patch to reduce the CSF volume in the epidural space.

- Aseptic technique must be meticulous at both the epidural site and the site of venepuncture.
- Blood injected into the epidural space predominantly spreads cephalad, so blood patches should be performed at the same or lower interspace as the dural puncture, with the woman in the lateral position to minimise CSF pressure in the lumbar dural sac.
- When the epidural space has been identified, 20mL of blood is obtained.
- Inject the blood slowly through the epidural needle until either a maximum of 20mL has been given or pain develops (commonly in the back or legs). If pain occurs, pause and if the pain resolves, try continuing a slow injection. If the pain recurs, then stop.
- Maintain bed rest for 2h to allow a clot to form.
- As far as possible, the patient should avoid straining, lifting or excessive bending for 48h.
- Follow-up is still required and every woman should have clear instructions to contact the anaesthetists again if symptoms recur.

ALGRAWANY

Remifentanil for labour analgesia

Remifentanil is an ultrashort-acting, potent mu-agonist synthetic opioid, which is broken down by non-specific tissue and plasma esterases. It has a half-life of about 6min and a rapid onset time of 30–60s. Although remifentanil readily crosses the placenta, it is also rapidly metabolised in the fetus. These features make remifentanil a useful analgesic agent in labour.[6] Like all opioids, it does not produce complete analgesia but is more effective than IM pethidine. Although its analgesic effect may be similar to fentanyl PCA, remifentanil is associated with a decreased requirement for fetal resuscitation, but an increased incidence of maternal respiratory depression (5–93%). Remifentanil should only be used with direct (in the room) supervision. There have been numerous case reports of maternal respiratory arrests.

As there is a significant risk of respiratory depression, a training programme for all midwifery and anaesthetic staff must be completed before remifentanil is introduced to a labour ward.

The ideal PCA regimen has not been established. Many proposed techniques are based on body weight, but the technique below is based on a fixed dose which has the advantage of simplicity and hence safety.

Technique

- Of critical importance is establishing one-to-one care with a trained individual (midwife) who must be continuously present in the room.
- No opioid should have been used in the previous 4h.
- Establish dedicated IV access.
- PCA bolus dose of 20–40 micrograms and lockout of 2min.
- Monitor with continuous SpO_2.
- Give O_2 if SpO_2 <94% on air.
- Thirty-minute observations of RR, sedation score and pain scores.
- Always flush the cannula when a PCA is discontinued.
- Call the anaesthetist if:
  - The patient is not rousable to voice
  - RR <8 breaths/min
  - SpO_2 <94% despite O_2 supplementation.

Caesarean section

With all CS, it is vital that the obstetrician clearly communicates the degree of urgency to all staff. The classification in Table 35.2 is a modification of that originally proposed by Lucas.

Table 35.2 Categories of urgency of Caesarean section

Category 1	Maternal or fetal compromise with immediate threat to the life of mother or fetus
Category 2	Maternal or fetal compromise that is not immediately life-threatening
Category 3	No maternal or fetal compromise but requires early delivery
Category 4	No maternal or fetal compromise. Delivery timed to suit mother and maternity services

- Any emergency CS requires transfer of the patient to theatre as rapidly as possible. Fetal monitoring should be continued until abdominal skin preparation starts.
- Category 1 CS: aim to deliver the fetus as quickly as possible while not compromising maternal safety. While it is the obstetrician's responsibility to call the urgency of the CS, it is the anaesthetist's responsibility to choose a method of anaesthesia that is safe. While in many centres, GA is common for Category 1 sections, CNB anaesthesia is a valid alternative. Do not be pressured into choosing a form of anaesthesia that is inappropriate for the mother.
- Category 2–4 CS: usually performed under CNB anaesthesia.
- Remember that the classification of urgency should be continuously reviewed. Category 1 sections can become Category 2, and vice versa.
- Remember to document the following times: time informed, time into theatre, time anaesthetic was ready, time of knife to skin and time of delivery.

Central neuraxial blockade for Caesarean section

(See also ➲ pp. 1114–17.) CNB anaesthesia for CS was initially driven by maternal preference. It was subsequently found to be safer than GA, although with good-quality training and modern anaesthetic standards and equipment, the difference in maternal mortality appears to be less than it was in the past.

Advantages of CNB include:

- Both mother and partner can be present at the delivery
- Minimal risk of aspiration and lower risk of anaphylaxis
- The neonate is more alert, promoting early bonding and breastfeeding
- Fewer drugs are administered, with less 'hangover' than after GA
- Postoperative analgesia is better with earlier mobilisation.

Mothers who are nervous about having a CS under CNB should be given a clear explanation of the advantages and disadvantages of CNB and GA but should not be coerced into having either technique.

ALGRAWANY

There are three principal techniques for CNB: epidural, spinal and CSE. Epidural anaesthesia is mostly used for women who already have labour epidural analgesia. Spinal anaesthesia is the most popular technique for elective CS, although in some centres, CSE is preferred.

The speed of onset of sympathectomy that occurs with spinal anaesthesia (as opposed to epidural) results in a greater fall in maternal CO and BP (see ➔ p. 861) and may be associated with a more acidotic neonate at delivery. When there is particular concern about the speed of onset of a block, a CSE approach can be used, injecting only a small dose of intrathecal LA and extending the block if required using the epidural catheter. Spinal anaesthesia generally provides a better quality of analgesia than epidural anaesthesia.

Preparation for CS

Whatever anaesthetic technique is chosen, careful history should be taken and appropriate examination performed. This should include checking:

- Blood group and antibody screen. Routine X-matching of blood is not required, unless haemorrhage is expected or antibodies that interfere with X-matching are present.
- Ultrasound reports to establish the position of the placenta. A low-lying anterior placenta puts a woman at risk of major haemorrhage, particularly if associated with a scar from a previous CS (see ➔ pp. 884–5).

An explanation of the technique should be offered. Although CS under CNB becomes routine for the anaesthetist, it can be intimidating for the mother. Reassurance and support are important. The possibility of complications must be mentioned, including the risk of intraoperative discomfort and its management. Pain during CNB remains a leading obstetric anaesthetic cause of maternal litigation. Document all complications that are discussed.

Caesarean section: epidural

Advantages	Disadvantages
• A functioning labour epidural is easy to top-up • More stable BP (than spinal) • Intraoperative top-up possible • Epidural can be used for postoperative analgesia	• Slow onset • Large doses of LA • Poorer quality of block than spinal anaesthesia

Indications for CS under epidural anaesthesia

- Women with a functioning epidural for labour analgesia.
- Some specific maternal diseases where rapid changes in SVR might be problematic (e.g. some cardiac disease), although commonly these individuals will have a careful CSE.

Technique

- Preparation relevant for all CS:
  - History/examination/explanation and consent.
  - Ensure that antacid prophylaxis has been given.
  - Establish 16G or larger IV access. Start crystalloid co-load.
  - Give antibiotic prophylaxis as per local guidelines.
- Insert epidural catheter at the L2/3 or L3/4 vertebral interspace.
- Position patient in the supine position with left lateral tilt/wedge.
- Test dose, then incrementally top-up the epidural with LA and opioid:
  - Boluses of 5–8mL of 2% lidocaine with 1:200 000 adrenaline ± sodium bicarbonate every 2–3min up to a maximum of 7mg/kg (~20mL). If bicarbonate is used, add 2mL of 8.4% bicarbonate to 18mL of 2% lidocaine with 1:200 000 adrenaline,

  OR
  - 5mL of 0.5% bupivacaine/levobupivacaine or 0.75% ropivacaine every 4–5min up to a maximum of 2mg/kg in any 4h period. Single-enantiomer LAs may offer some safety advantage; however, lidocaine is still safer than either ropivacaine or levobupivacaine.
- Opioid (e.g. 100 micrograms fentanyl or 2.5mg diamorphine) improves the quality of the analgesia.
- Establish an S4–T4 block (nipple level). A slightly lower dermatomal block height may be accepted if epidural opioid is given with epidural LA (e.g. T6 to light touch). Always check the sacral dermatomes, as epidural LA occasionally does not spread caudally. Anaesthesia to light touch is more reliable at predicting adequacy of block than loss of cold sensation. Document the level of block obtained and the adequacy of perioperative analgesia.
- Hypotension is much less common than with intrathecal techniques. However, if hypotension does occur, treat hypotension with the following (see also ➲ p. 861):
  - Boluses of 500mL of crystalloid
  - Phenylephrine 50–100 micrograms IV bolus (expect a reflex bradycardia) or ephedrine 6mg IV
  - Increasing the left uterine displacement.

ALGRAWANY

- At delivery, give 2–5 units of oxytocin as a slow IV bolus. If tachycardia must be avoided, then an IVI of 40 units of oxytocin in 500mL of crystalloid given over 4h is an acceptable alternative.
- At the end of the procedure, give an NSAID unless contraindicated (e.g. 100mg diclofenac PR).
- Epidural diamorphine (2.5mg) given at the time of surgery improves postoperative analgesia, while epidural fentanyl has little postoperative analgesic benefit.
- Prescribe postoperative analgesia and VTE prophylaxis as per local protocol.

Caesarean section: spinal

Advantages	Disadvantages
• Quick onset • Good-quality analgesia • Easy to perform • Use of opioids provides postoperative analgesia	• Limited duration • Inadequate analgesia is difficult to correct • Rapid changes in BP and CO

Spinal anaesthesia is the most commonly used technique for elective CS.

Technique

- Preparation as per CS under epidural (see ➔ pp. 855–6).
- A sitting position usually makes finding the midline easier, which may be helpful with obese patients and may be associated with a faster onset, although the height of block is less predictable. A lateral position is associated with a slower onset of block, particularly if a full lateral position is maintained until the block has fully developed. The block height may be slightly more consistent, and women sometimes find it more comfortable than sitting.
- Perform spinal anaesthetic at L3/4 interspace or lower using a 25G or smaller pencil-point needle. The level of the iliac crests usually corresponds to the spinous process of L4 (Tuffier's line), although there is variation between individuals.
- With the orifice pointing cephalad, inject the anaesthetic solution, e.g. 2.5mL of 0.5% hyperbaric bupivacaine with 300 micrograms diamorphine or 15 micrograms fentanyl. Intrathecal diamorphine improves postoperative analgesia, while intrathecal fentanyl has little postoperative analgesic benefit.
- After injection of the solution, move the woman to a supine position with left lateral tilt or wedge. When hyperbaric LA solutions are used, it is important that the cervical spine is kept elevated (pillow) to prevent LA spreading to the cervical dermatomes.
- Hypotension is more common with spinal anaesthesia than with epidural anaesthesia. Try to prevent hypotension, rather than treating it after it has occurred. When possible, a continuous infusion of pressor agent should be started at the time of the injection of spinal LA (see ➔ p. 861).
- Continue as for epidural anaesthesia for CS (see ➔ pp. 855–6).

ALGRAWANY

Caesarean section: CSE

Advantages	Disadvantages
• Quick onset	• Rapid change in BP and CO
• Good-quality analgesia	• Technically more difficult
• Intraoperative top-up possible	• Higher failure rate of spinal
• Epidural can be used postoperatively	• Untested epidural catheter

In some centres, CSE is the technique of choice for CS. Indications include:

- Limiting the speed of onset of a block. A small initial intrathecal dose of LA can be supplemented through the epidural as required.
- Expectation of prolonged surgery.
- Ability to use the epidural catheter for postoperative analgesia.

Technique

- Preparation as per CS under epidural (see pp. 855–6).
- There are two commonly used techniques—a needle-through-needle technique where the spinal needle is passed through the epidural needle, and a separate injection technique. The needle-through-needle technique is associated with a higher incidence of failure to locate CSF with the spinal needle but only involves one injection. If the two-injection technique is used, the epidural is usually sited first at L2/3 or above and then the spinal injection is performed at L3/4 or below. There is a theoretical risk of damaging the catheter with the spinal needle.
- If the spinal block is inadequate, inject LA or 10mL of 0.9% sodium chloride through the epidural catheter. (0.9% sodium chloride works by compressing the dural sac, causing cephalad spread of intrathecal LA.)

Needle-through-needle technique

- Use either a dedicated CSE set or a Tuohy needle and a long 25G (or smaller) pencil-point needle. Locate the epidural space at L3/4 or below with a Tuohy needle and then pass the long spinal needle through the Tuohy needle into the intrathecal space. Inject anaesthetic solution with the needle orifice pointing cephalad (e.g. 2.5mL of 0.5% hyperbaric bupivacaine with 300 micrograms diamorphine or 15 micrograms fentanyl).
- Insert the epidural catheter. Aspirate the catheter carefully for CSF.

Two-needle technique

- Insert an epidural at L2/3 or above. After the catheter is in position, perform a spinal injection at L3/4 or below with a 25G or smaller pencil-point needle.

With either technique, testing the catheter with LA before the intrathecal dose has receded may be unreliable. However, using the catheter intraoperatively is reasonable, as the anaesthetist is continuously present to deal with the consequences of an intrathecal injection. This may not be true if opioids are given through the catheter for postoperative analgesia at the end of the procedure before the block has receded.

Continue as for spinal anaesthesia for CS (see p. 857).

Special considerations

- Although the incidence of major complications of CNB, as identified by the Third National Audit Project of the RCoA, was higher when a CSE technique was used, the numbers were very small (two or four patients, depending on whether an optimistic or a pessimistic analysis was used) and the study cautions against overinterpretation of these results.

ALGRAWANY

Inadequate anaesthesia

Every patient should be warned of the possibility of intraoperative discomfort and this should be documented. Of attempted CNB anaesthetics for CS 1-5% are inadequate for surgery. The majority should be identified before the operation commences.

Inadequate block identified before surgery has started

Epidural

- If no block develops, then the catheter is incorrectly positioned. It may be reinserted or a spinal performed.
- If a partial but inadequate block has developed, the epidural may be resited or withdrawn slightly. If the toxic limit for the LA agent has been reached, elective procedures can be abandoned, but for urgent procedures, a GA or a spinal anaesthetic will be required. Be very cautious about converting a partially functioning epidural anaesthetic to a spinal anaesthetic. Although a normal spinal dose of hyperbaric LA is commonly used, aggressive control of the spread of LA is needed to prevent a high or total spinal occurring. This is done by positioning the head up initially and then slowly lowering the head to achieve the required level of block.

Spinal

- If no block develops, a repeat spinal may be performed.
- If a partial but inadequate block develops, an epidural may be inserted and slowly topped up.
- Use a GA if required.

Intraoperative inadequate block

In this situation, good communication with the mother and surgeon is essential. If possible, stop surgery. Identify the likely cause of pain (e.g. inadequately blocked sacral nerve roots, peritoneal pain). Try to give the mother a realistic expectation of continued duration and severity of pain. If the pain has occurred before the delivery of the fetus, it is very likely that a GA will be required.

- If the patient requests GA, in all but exceptional circumstances, comply.
- If the anaesthetist feels that the severity of pain is not acceptable, persuade the patient that GA is required.

Spinal

Reassure and treat with:

- Inhaled N_2O
- IV opioid (e.g. 25–50 micrograms fentanyl, repeated as necessary)—inform the neonatologists that opioid has been given
- Surgical infiltration of LA (care with total dose)
- GA.

Epidural/CSE

- Treat as per spinal anaesthesia, but in addition, epidural opioid (e.g. 100 micrograms fentanyl) and/or more epidural LA can be given.

Hypotension

Hypotension is common with CNB anaesthesia, especially spinal anaesthesia.[7] Preventing hypotension, rather than treating it after it has occurred, is associated with better fetal and maternal outcomes. Aim to maintain the systolic BP at ≥90% of baseline.

Prophylactic pressor agents are key, but it is also important to minimise aortocaval occlusion (i.e. lateral tilt) and a fluid co-load should be routine unless fluid is being restricted.

Pressor agents

Using prophylactic pressor agents is beneficial for both mother and fetus. α-agonists (phenylephrine and metaraminol) should be considered as the 1° agents. Ephedrine (6mg bolus) is an acceptable alternative, despite possibly causing marginally more fetal acidosis than phenylephrine. Noradrenaline, which has both α and β action, has been shown to have marginal advantage over phenylephrine, but currently its use as a peripheral infusion remains controversial. Phenylephrine bolus doses of 50–100 micrograms can cause reflex bradycardias, so if possible, use a phenylephrine or metaraminol infusion instead. A reduction in HR is associated with a reduced CO, so treat bradycardia with either ephedrine or an anticholinergic agent. A simple regime for phenylephrine infusion is outlined below.

- Put 20mL of 100 micrograms/mL of phenylephrine in a syringe driver.
- Start infusion of 30mL/h as the spinal solution is injected.
- Titrate to response in increments of 10mL/h.
- Expect the HR to slow—give anticholinergic agents as required.
- Reduce and stop the infusion post-delivery.
- In pre-eclamptic and hypertensive individuals, start at a lower infusion rate.

Fluid

Crystalloid preloading is ineffective at preventing hypotension, and in women with severe pre-eclampsia, large preloads are harmful, predisposing to pulmonary oedema. Using colloids as a preload is more effective, but colloids are associated with a variety of problems, including anaphylaxis and clotting abnormalities.

While preloading with crystalloid is ineffective, co-loading crystalloid (giving fluid as the block is establishing) is more effective.

A co-load should:

- Be given immediately before or during the onset of the CNB technique to minimise redistribution
- Be limited to 10–15mL/kg of crystalloid. Larger volumes should be avoided as they offer little advantage and may be harmful. (More fluid may be given intraoperatively if clinically indicated.)

Do not delay an emergency CS to allow a fluid preload to be administered.

ALGRAWANY

Caesarean section: general anaesthesia

Elective GA for CS is now uncommon in the UK. Safety remains a concern as many of the major anaesthetic complications still relate to difficulties with the airway. Failed intubation is much more frequent in obstetric than in non-obstetric anaesthesia (see ➲ p. 864). All obstetric anaesthetists should be familiar with a failed intubation drill.

Indications for GA

Include:

- Maternal request
- Category 1 CS—while GA is the most commonly used technique, CNB is only marginally slower with a well-drilled anaesthetic team
- Failed CNB or CNB contraindicated (e.g. coagulopathy, maternal hypovolaemia)
- Additional/long surgery planned which may make CNB less acceptable.

Technique

- History and examination, including airway assessment.
- Using a GA checklist can be very helpful to reduce stress and errors.
- Antacid prophylaxis—see details below.
- Position supine with left lateral tilt/wedge. Consider head-up position to improve view at laryngoscopy (especially in obese patients).
- Preoxygenate for 3–5min or with 4–8 VC breaths with a high flow through the circuit and a good face mask seal. At term, women have a reduced FRC and a higher RR and O_2 consumption. This reduces the time required for denitrogenation but also reduces the time from apnoea to arterial O_2 desaturation. The use of nasal O_2 should be considered.
- Perform RSI. The risk of awareness without recall may be increased if the dose of induction agent is reduced. Consider using a short-acting opioid. (Be aware that opioids administered before delivery may increase neonatal respiratory depression, so always inform the neonatologist.)
- Induction is usually performed with either propofol or thiopental. Ketamine can be used if clinically indicated.
- A 7.0mm ETT is adequate and may make intubation easier.
- Ventilate with 50% O_2 in N_2O. If severe fetal distress is suspected, then 75% O_2 or higher may be appropriate. Maintain $ETCO_2$ at 4.0–4.5kPa (30–34 mmHg).
- Rapidly increase the end-tidal concentration of anaesthetic agent to at least 0.75 of MAC.
- At delivery, give 2–5 units of oxytocin IV bolus (or IVI of 40 units of oxytocin in 500mL of crystalloid, infused over 4h if tachycardia to be avoided), opioid (e.g. 10–15mg morphine) and IV paracetamol.
- At the end of the procedure, consider surgical infiltration of LA or performing regional blocks (e.g. TAP, ilioinguinal, quadratus lumborum or rectus sheath).
- An NSAID can be given PR or IV.
- Extubation is a high-risk procedure—extubate awake.
- Midwives may be less familiar with recovery and airway care. The same standard of recovery staff should be available to women on labour wards as in a normal theatre recovery unit.

Antacid prophylaxis

Aspiration of particulate matter, blood or bile is associated with worse outcomes than aspiration of gastric fluid. Fluid aspiration is commonly associated with chemical pneumonitis and the severity of this is, in turn, dependent on the volume and acidity of the aspirated fluid. Use of antacids and prokinetic agents can elevate the gastric pH and reduce the intragastric volume. A suggested regime is the following.

Elective surgery

- Ranitidine 150mg or omeprazole 40mg PO 12h and 2h before surgery.
- Metoclopramide 10mg PO 2h before surgery.
- 30mL of 0.3M sodium citrate immediately before induction of GA. (Gastric pH >2.5 is maintained for only 30min after 30mL of 0.3M sodium citrate. If a GA is required after this, a further dose of citrate is required.)

Emergency surgery

If prophylaxis has not already been given:

- Ranitidine 50mg by slow IV injection immediately before surgery. Remember this will not alter the risk if aspiration occurs during induction but may offer benefit by the time of extubation.
- Metoclopramide 10mg IV injection immediately before surgery.
- 30mL of 0.3M sodium citrate PO immediately before induction of GA.

Effect of general anaesthesia on the fetus

Lower fetal Apgar scores at 1 and 5min are more common when GA is used for CS. Most anaesthetic agents, except for muscle relaxants, rapidly cross the placenta. Thiopental can be detected in the fetus within 30s of administration, with peak umbilical vein concentration occurring at around 1min. Umbilical artery to umbilical vein concentrations approach unity at 8min. Opioids administered before delivery may cause fetal depression. If there is a specific indication for opioids before delivery, they should be given and the neonatologist informed. Hypotension, hypoxia, hypocapnia and excessive maternal catecholamine secretion may all be harmful to the fetus.

ALGRAWANY

Failed intubation

(See also ➲ pp. 368–72.)

Failed intubation occurs much more commonly in the obstetric population (~1:400).[8] If a failed intubation occurs, the mortality is ~1:90. Causes of failed intubation include obesity, increased fatty tissue, pharyngeal/laryngeal oedema, large tongue, large breasts, incorrect cricoid pressure, complete dentition and the experience and training of anaesthetic staff.

The Obstetric Anaesthetists' Association and the DAS produced three algorithms in 2015: (1) for safe obstetric GA, (2) for obstetric failed intubation and (3) for CICO scenarios (https://www.oaa-anaes.ac.uk/assets/_managed/cms/files/Clinical%20Guidelines/Guideline_Algorithms_2015.pdf).

Before embarking on a GA, it is important to have thought about alternative plans if intubation is difficult or if intubation fails, and to have discussed these plans with the anaesthetic assistant. GA checklists can greatly assist with this.

In the event of a failed intubation, deciding on whether to continue surgery or to wake the mother can be difficult. Factors to consider include the mother's condition, the fetal condition (while waiting for the muscle relaxant to wear off, fetal monitoring can be reapplied and this may give useful additional information), the experience of the anaesthetist, the anticipated difficulty of the surgery, patient factors, including obesity and starvation status, and the difficulty of alternative methods of anaesthesia. Additional factors that can only be established after the airway has been rescued after a failed intubation include the method by which the airway is rescued (i.e. 2nd-generation SGA device or difficult mask ventilation) and whether any airway trauma has occurred. Remember that when a failed intubation occurs, the priority is oxygenation.

If the decision is to continue surgery, ideally a 2nd-generation SGA device would be inserted (release cricoid pressure before insertion), and empty the stomach with an OGT. The anaesthetist will have to decide on whether to opt for spontaneous respiration or paralyse (ideally with rocuronium if sugammadex is readily available) and ventilate. Remember to ask the obstetricians to avoid fundal pressure at delivery if possible, because fundal pressure can increase intragastric pressure to >70mmHg. Try to avoid further instrumentation of the airway, but be prepared for front of neck access, and if familiar, have a fibreoptic scope immediately to hand.

Postoperative analgesia

Most postpartum women are very well motivated and mobilise quickly. However, effective analgesia does allow earlier mobilisation. The mainstays of postoperative analgesia are opioids, NSAIDs and paracetamol. The route that these are given is dependent on the intraoperative anaesthetic technique.

Opioids

Intrathecal/epidural opioid

- CNB fentanyl lasts little longer than CNB LA and has little postoperative analgesic benefit. If an epidural catheter is left *in situ*, epidural fentanyl may be given as an infusion or as intermittent postoperative boluses (50–100 micrograms up to 2-hourly for two or three doses). However, this is rarely done.
- Intrathecal diamorphine (300–400 micrograms) can be expected to provide 6–18h of analgesia. More than 40% of women will require no other postoperative opioid. Higher doses have been recommended but are associated with an increased incidence of side effects. Pruritus is very common (60–80% of cases), although only 1–2% have severe pruritus. The evidence that antihistamines reduce pruritus caused by CNB opioids is poor.
- Epidural diamorphine (2.5mg in 10mL of 0.9% sodium chloride) provides 6–10h of analgesia after a single dose. Intermittent doses may be given if the epidural catheter is left *in situ*.
- Intrathecal preservative-free morphine (100 micrograms) provides long-lasting analgesia (12–18h). However, pruritus and nausea are common. Doses above 150 micrograms are associated with increased side effects without improved analgesia. The low lipophilicity of morphine may increase the risk of late respiratory depression. Epidural morphine (2–3mg) provides analgesia for 6–24h, but pruritus is again common and nausea occurs in 20–40% of cases. Diamorphine is used much more commonly in the UK than morphine.

IV patient-controlled analgesia or oral opioids

- IV PCA or PO opioids can be used, although these are not as effective as CNB analgesia.

Neonatal effects of maternal opioids

- A small quantity of opioid may be transferred to the neonate through breast milk (see pp. 867–8). Rarely, this can be associated with neonatal respiratory depression.

NSAIDs

- NSAIDs are very effective postoperative analgesics, reducing opioid requirements. They should be administered regularly whenever possible, but beware renal impairment in severe pre-eclampsia, and care with significant haemorrhage.

ALGRAWANY

Summary of dosing regimes

(See Table 35.3.)

Table 35.3 Summary of dosing regimens

Procedure	Technique	Suggested dose
Labour	Epidural loading	20mL of 0.1% bupivacaine with 2 micrograms/mL fentanyl in divided doses
	Epidural infusion	10mL/h of 0.1% bupivacaine with 2 micrograms/mL fentanyl
	Top-ups	10–20mL of 0.1% bupivacaine with 2 micrograms/mL fentanyl
	CSE	Intrathecal: 0.5–1mL of 0.25% bupivacaine with 5–25 micrograms/mL fentanyl
		Epidural: top-up and infusion as above
	PCEA	10mL boluses of 0.1% bupivacaine with 2 micrograms/mL fentanyl with a 15–20min lockout
LSCS*	Spinal	2.5mL of 0.5% bupivacaine in 8% glucose ('heavy') + 300 micrograms diamorphine
	Epidural	15–20mL of 2% lidocaine with 1:200 000 adrenaline (± 0.5–2mL of preservative-free 8.4% sodium bicarbonate)
	CSE	Normal spinal dose (reduce if slow onset of block is required)
		If needed, top up the epidural with 5mL aliquots of 2% lidocaine with 1:200 000 adrenaline or 0.5% (levo)bupivacaine
Post-LSCS analgesia	GA	Bilateral ilioinguinal nerve blocks, rectus sheath blocks, TAP blocks or local infiltration at end of surgery
		IV aliquots of morphine until comfortable
		Further opioid can be given PO or IV (PCA is commonly used)
		75mg diclofenac IV and 1g paracetamol IV before extubation, followed by 50mg diclofenac PO 8-hourly
	CNB and GA	Regular paracetamol analgesics PRN
	CNB	100mg diclofenac PR at end of surgery, followed by 50mg diclofenac PO 8-hourly
		Epidural diamorphine (2.5mg) in 10mL of 0.9% sodium chloride. On rare occasions the epidural catheter is left *in situ* and doses can be repeated up to 4-hourly

* LSCS, lower-segment Caesarean section.

Breastfeeding and drug transfer

For drugs to be transferred to a neonate through breastfeeding, they must be secreted in the milk and absorbed in the neonatal GI tract and must not undergo extensive 1st-pass metabolism in the neonatal liver.

In general, for breastfed infants, the neonatal serum concentration of a drug is <2% of maternal serum concentration, resulting in a subtherapeutic dose. Most drugs are therefore safe. There are some exceptions, either because transfer is much higher or because transfer of even small quantities of a drug is unacceptable. Drugs with high protein binding may displace bilirubin and precipitate kernicterus in a jaundiced neonate.

Factors that make significant transfer more likely include low maternal protein binding, lipophilicity or, with hydrophilic drugs, a molecular weight of <200Da and weak bases which increase the proportion of ionised drug in the weakly acidic breast milk, leading to 'trapping'.

Most opioids have a milk-to-maternal plasma ratio of ~2, while NSAIDs have ratios of ~0.1 or below, and paracetamol of 1.

Table 35.4 Drug safety in breastfeeding

Drug	Comment
Opioids	Small amount delivered to neonatal serum. Minor concern about the long duration of action of pethidine's metabolite norpethidine. Avoid codeine, and care with other oral opioids if mother or neonate excessively drowsy (see ➲ p. 868)
NSAIDs	Most NSAIDs are considered safe in breastfeeding. Some would advise caution with aspirin because of unsubstantiated concerns about causing Reye's syndrome in the neonate
Antibiotics	Penicillins and cephalosporins are safe, although trace amounts may be passed to the neonate Tetracycline should be avoided (although absorption is probably minimal because of chelation with calcium in milk) Chloramphenicol may cause bone marrow suppression in the neonate and should be avoided Ciprofloxacin is present in high concentrations in breast milk and should be avoided
Antipsychotics	Generally suggested that these are avoided, although the amount excreted in milk is probably too small to be harmful. Chlorpromazine and clozapine cause neonate drowsiness
Cardiac drugs	Amiodarone is present in milk in significant amounts and breastfeeding should be discontinued Most β-blockers are secreted in minimal amounts. Sotalol is present in larger amounts While enalapril and captopril have no known adverse effects, other ACE inhibitors, ARBs and amlodipine should be avoided
Anticonvulsants	While carbamazepine does not accumulate in the neonate, phenobarbital and diazepam may. Neonates should be observed for evidence of sedation

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Timing of administration just after breastfeeding can reduce drug transfer. Breastfeeding constitutes a metabolic and fluid stress for the mother, so keep the mother well hydrated and try to minimise periods of nil by mouth. Treat nausea and vomiting promptly.

Codeine, oxycodone, dihydrocodeine and oral morphine

Small quantities of opioid are transferred in breast milk, but the risk of neonatal respiratory depression is very small. However, there have been case reports of neonatal respiratory depression after maternal administration of PO morphine, oxycodone and dihydrocodeine and one neonatal death following prolonged use of postoperative codeine. Postnatal women taking opioids should be observed and if they, or their offspring, appear excessively drowsy, the opioid should be stopped. Breastfeeding women should not be discharged with opioid analgesia.

Table 35.4 gives information on some agents. A more comprehensive list of drugs compatible with breastfeeding can be found at:

The Breastfeeding Network: https://www.breastfeedingnetwork.org.uk/drugs-factsheets

Drugs and Lactation Database (LactMed): https://www.ncbi.nlm.nih.gov/books/NBK501922/

Retained placenta

- IV access with 16G or larger cannula.
- Assess total amount and rate of blood loss and cardiovascular stability. Blood loss may be difficult to accurately assess. If rapid blood loss is continuing, then urgent X-match and evacuation of placenta under GA is required.
- CNB anaesthesia is safe, provided estimated blood loss is <1000mL, but if there are signs suggesting hypovolaemia, GA may be required.
- Remember antacid prophylaxis.
- For GA, use an RSI technique with a cuffed ETT.
- CNB anaesthesia can be performed either by topping up an existing epidural or with a spinal (e.g. 2–2.5mL of 0.5% hyperbaric bupivacaine plus 15 micrograms fentanyl or 300 micrograms diamorphine intrathecally). A T7 block reliably ensures analgesia.
- Occasionally, uterine relaxation is required. Under GA, this can be produced by increasing the halogenated vapour concentration. Under CNB anaesthesia, a sublingual GTN spray is usually effective, although expect transient hypotension.
- On delivery of the placenta, give 5 units of oxytocin ± an infusion of oxytocin (e.g. 40 units in 500mL of crystalloid over 4h).
- At the end of the procedure, give an NSAID unless contraindicated.

ALGRAWANY

Fetal neuroprotection

Magnesium

- Magnesium should be considered for all women expected to deliver before 34w gestation to reduce the incidence of cerebral palsy in the baby.[9]
- Dose of magnesium for neuroprotection is 4g IV bolus of magnesium sulfate over 15min, followed by an IVI of 1g/h until birth or for 24h, whichever is sooner.
- Patients receiving magnesium should be monitored every 4h for signs of toxicity, including recording pulse, BP, RR and deep tendon reflexes.
- If the patient develops oliguria or renal failure, monitoring for magnesium toxicity should be performed more frequently and a reduction in the dose of magnesium should be considered.

In utero fetal death

- The death *in utero* of a formed fetus is emotionally devastating for a family. The delivery can also be very traumatic, and it is important to be as supportive as possible during this period. The cause of fetal loss is varied and often uncertain. Remember that *in utero* deaths can be associated with concealed abruption, sepsis and DIC.
- Sometimes women will have to be delivered by CS, but the majority undergo a vaginal delivery.
- Analgesia for labour includes all the normal techniques. Patient-controlled IV opioids are often used, as is CNB analgesia. However, before any CNB techniques are performed, check that maternal clotting is normal. Clotting derangement can occur even when the fetal death is thought to have occurred very recently.
- Delivery by CS can be performed under regional anaesthesia or GA. This should be guided by maternal preference, as well as maternal safety.

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Pregnancy-induced hypertension and pre-eclampsia

Pre-eclampsia remains a leading cause of worldwide maternal deaths. Estimates of incidence vary geographically but are of the order of 2–8% of all pregnancies. Although a systemic disorder, its pathogenesis is primarily placental. The precise aetiology is complex and incompletely understood. Immunological factors, genetic factors and endothelial dysfunction, as well as abnormalities in placental implantation, fatty acid metabolism, coagulation and platelet factors, are all implicated. Early-onset and late-onset pre-eclampsia are increasingly recognised as separate, although interrelated, subtypes. Early onset is primarily of placental cause, while late onset is not only related to placental factors but also with maternal genetic predisposition to cardiovascular and metabolic disease. Early-onset disease is usually more severe.

Definitions are arbitrary. Often diagnosis is made by considering the whole picture. However, commonly used definitions are:

Hypertension

Sustained systolic BP >140mmHg or diastolic BP >90mmHg.

Chronic hypertension

Hypertension that existed before the 20th week of pregnancy or those already taking antihypertensive medication prepregnancy.

Gestational hypertension

Hypertension that develops after the 20th week of pregnancy.

Pre-eclampsia

- New-onset hypertension with one or more other features:
  - Proteinuria (urine protein:creatinine ratio ≥30mg/mmol or albumin:creatinine ratio ≥8mg/mmol, or ≥1g/L [2+] on dipstick)
  - Other maternal organ dysfunction (renal, hepatic or neurological)
  - Haematological involvement
  - Uteroplacental dysfunction (fetal growth restriction or abnormal uteroplacental blood flow on ultrasound).[10]
- Pre-eclampsia with any of the following features all have a greater potential for poor outcome: severe hypertension that does not respond to treatment or is associated with ongoing or recurring severe headaches, visual scotomata, nausea or vomiting, epigastric pain, oliguria, progressive deterioration in laboratory blood tests such as rising creatinine or liver transaminases, a falling platelet count or failure of fetal growth or abnormal Doppler findings.
- The distinction between early and late forms of pre-eclampsia is important as early onset is commonly associated with a higher risk. Onset before the 35th gestational week is considered 'early onset'.
- **Eclampsia** (see ➔ p. 877) refers to convulsions occurring in pregnancy or the puerperium in the absence of other causes. Signs of pre-eclampsia may not be manifest until after a fit.

Pathophysiology

Cardiorespiratory

- Hypertension and ↑ sensitivity to catecholamine and exogenous vasopressors
- Reduced circulating volume, but ↑ TBW
- SVR is usually ↑ and CO reduced
- ↑ capillary permeability which may result in:
  - Pulmonary oedema—be very careful to avoid fluid overload
  - Laryngeal and pharyngeal oedema. Voice change may give an indication of this. Stridor is a very worrying sign.

Haematological

- Reduced platelet count with ↑ platelet consumption and hypercoagulability with ↑ fibrin activation and breakdown—DIC may result
- ↑ Hct due to reduced circulating volume.

Renal function

- Reduced GFR
- ↑ permeability to large molecules resulting in proteinuria
- Oliguria in severe disease.

Cerebral function

- Headache, visual disturbance and generalised hyperreflexia
- Cerebrovascular haemorrhage
- Eclampsia (resulting from cerebral oedema or cerebrovascular vasoconstriction). Clonus indicates a high risk of imminent eclampsia.

Fetoplacental unit

- Reduced fetal growth with associated oligohydramnios
- Poor placental perfusion and ↑ sensitivity to changes in maternal BP.
- Non-reassuring CTGs.
- Changes in umbilical arterial blood flow. A reduction of umbilical arterial diastolic blood flow, and particularly reverse diastolic flow, is indicative of poor fetal outcome and an indication for early intervention.

Management of pre-eclampsia

Prevention

- Antiplatelet agents (aspirin 75–150mg) should be prescribed for patients at high risk (such as chronic hypertension, type 1 or type 2 diabetes or previous hypertensive disease) or with two or more moderate risk factors (such as booking BMI >36kg/m^2 and age >40y).

Early detection

- There are a variety of cardiovascular and biochemical markers that can predict pre-eclampsia, including placental growth factor (the Triage PlGF test) and the sFlt-1/PlGF ratio.[11] These tests can help to exclude pre-eclampsia in women between 20w and 35w of gestation.

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Symptom control

- Further management is aimed at controlling the symptoms of pre-eclampsia and preventing major harm to mother or fetus, until the placenta is delivered. After delivery of the placenta, severe symptoms will usually start to resolve within 24–48h.
- In general, delivery is not indicated before 37w gestational age, unless BP cannot be controlled to below 160/90mmHg or there are other medical indications.
- If the fetus is preterm at the time of delivery, when possible, delivery should be delayed for administration of steroids to promote fetal lung maturation. Magnesium may also be administered, not only to reduce the risk of eclampsia, but also for fetal neuroprotection.
- After 37w gestational age, timing of the delivery should be agreed between a senior obstetrician and the mother.

Antihypertensive therapy

- BP should be controlled to below 135/85mmHg to reduce maternal morbidity, particularly from intracranial haemorrhage, encephalopathy and myocardial ischaemia/failure. If the BP is above 160/110mmHg, urgent treatment is required. BP should be measured every 15–30min until it is below 160/110mmHg. Once below 160/110mmHg, the aim is to control BP to <135/85mmHg.
- In the UK, the 1° antihypertensive agent is the combined α- and β-blocker labetalol. If contraindicated or additional agents are required, nifedipine or methyldopa can be used. In severe or resistant cases, IV agents may be required (as below).
- ACE inhibitors should be avoided as they are associated with oligohydramnios, stillbirth and neonatal renal failure.
- Rapid control of severe hypertension can be achieved with:
  - Labetalol (5–10mg IV every 10min)
  - Hydralazine (5mg IV aliquots to a maximum of 20mg). The 1st IV dose of hydralazine is sometimes given with a 500mL crystalloid fluid bolus.
  - PO nifedipine (10mg). Sublingual nifedipine should be avoided because of rapid changes in placental circulation, which may compromise fetal condition.

Protection from eclampsia

- Magnesium sulfate reduces the incidence of eclampsia. Magnesium treatment should be considered if any of the following are present: clonus, persistent or recurrent headaches, visual scotoma, nausea and vomiting, epigastric pain, oliguria, severe hypertension or progressive deterioration in renal or liver function tests.
- For magnesium dosing, see ➲ p. 877.

Fluid management

Fluid management in severe pre-eclampsia is critical. Intravascular volume is depleted, but TBW is increased. Excessive fluid load may result in pulmonary oedema, but underfilling may compromise fetal circulation and renal function. General principles are:

- Fluid management protocols should be followed, and a named individual should have overall responsibility for fluid therapy.
- Measure hourly urine output.

- Usually total fluid intake is limited to 80mL/h. Beware of fluid loads being delivered with drugs. It may be necessary to increase the concentrations of agents such as oxytocin or magnesium to stay within the 80mL/h limit.
- Avoid fluid loading before CNB analgesia.
- Be cautious with fluid during CS; aim for neutral balance at the end.
- Invasive BP monitoring is indicated in severe pre-eclampsia for:
  - Monitoring the response to laryngoscopy and surgery during GA
  - Taking repeated ABGs
  - Monitoring rapidly acting hypotensive agents.
- CVP monitoring is rarely indicated, even in severe pre-eclampsia.

Analgesia for vaginal delivery

- Effective epidural analgesia controls excessive surges in BP during labour and is recommended.
- Check platelet count before performing an epidural. The 'acceptable' level of platelet count is debatable and based on little evidence. However, common general guidelines are:
  - If the platelet count is $<100 \times 10^9$/L, a clotting screen is required.
  - If the platelet count is $>75 \times 10^9$/L and the clotting screen is normal, then CNB techniques are acceptable.
  - With a platelet count of $<75 \times 10^9$/L, a careful assessment from a senior individual is required and the potential risks and benefits should be discussed with the patient.
  - Usually a platelet count within 6h of insertion is adequate, but if maternal condition is deteriorating or if the platelet numbers are rapidly falling, then a count must be performed immediately before placement.
- Fluid loading before CNB analgesia is not required, but monitor BP and fetus carefully and treat changes in BP promptly with cautious doses of ephedrine or phenylephrine.

Anaesthesia for CS in pre-eclamptic patients

GA or CNB may be used. GA is indicated if significant thrombocytopenia or coagulopathy have developed.

General anaesthesia

- Assess the airway carefully. Sometimes partners may be better able to assess the onset of facial oedema. A history of stridor is of major concern. A selection of small tube sizes must be available. Consider AFOI in severe cases.
- Obtund the hypertensive response to laryngoscopy, e.g. alfentanil 1–2mg (inform paediatrician that opioids have been used) and/or labetalol 10–20mg before induction. Remifentanil TCI or bolus may be useful if the anaesthetist is familiar with its use. In very severe pre-eclampsia, intra-arterial pressure monitoring is required before induction.
- If magnesium has been used, expect prolongation of action of NDMRs. Use a reduced dose and assess neuromuscular function with a nerve stimulator.
- Magnesium can also cause uterine relaxation, particularly with GA; an oxytocin infusion may be helpful.
- Ensure adequate analgesia before extubation. The hypertensive response to extubation may also need to be controlled with antihypertensive agents (e.g. labetalol 10–20mg).

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CNB anaesthesia

Despite the depleted intravascular volume that occurs with severe pre-eclampsia, pre-eclamptic patients are less prone to the hypotensive consequences of CNB anaesthesia than normal individuals. Spinal anaesthesia consistently produces better analgesia than epidural anaesthesia and should not be avoided.

- Platelet count and, if necessary, a clotting screen should be assessed.
- A reduced volume of fluid co-load should be used. By the end of the procedure, aim to have given no more crystalloid than measured blood loss.
- Use ephedrine or phenylephrine as indicated. However, be cautious because they may have an increased effect.

Effective postoperative analgesia is required, but avoid NSAIDs as these patients are prone to renal impairment and may have impaired platelet count or function. When the proteinuria has resolved, which is often within 48h, NSAIDs may be introduced.

Continue care in HDU or ICU.

Eclampsia

Incidence

Around 2:10 000 pregnancies in the developed world,[12] but there are wide international variations. Remember most seizures in pregnancy are not due to eclampsia. Always be alert to the differential diagnosis.

- Eclamptic fits occur most commonly in the 3rd trimester or within 12h of delivery.
- Eclampsia is a life-threatening event.
- Management is aimed at immediate control of the fit and 2° prevention of further fits.

Management

- Airway (left lateral position with jaw thrust), breathing (bag and mask ventilation and measure O_2 saturation), circulation (obtain IV access and measure BP when possible; avoid aortocaval compression).
- Control fits with magnesium:
  - Load with 4g IV over 5min, followed by 1g/h for 24h.
  - Recurrent seizures should be treated with 2–4g IV bolus over 5min.
- The therapeutic magnesium plasma concentration is 2–4mmol/L. Magnesium levels may be monitored clinically or with laboratory monitoring. Loss of reflexes occurs at concentrations of >5.0mmol/L, reduced RR at concentrations of 6.0-7.0mmol/L and cardiac arrest at concentrations of >12.0mmol/L. Reduce infusion rate with oliguria.
- Patients on calcium channel antagonists are at particular risk of toxicity.
- Toxicity can be treated with IV calcium (e.g. 10mL of 10% calcium chloride or calcium gluconate).
- If eclampsia occurs before delivery, once the fit has been controlled, think about the urgency of the delivery. In general, providing the fetus is not distressed, eclampsia is not an indication for emergency CS. The patient should be stabilised on magnesium and then consideration given to vaginal or operative delivery. Care should be continued on HDU or ICU.

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HELLP syndrome

Haemolysis, elevated liver enzymes and low platelets comprise the HELLP syndrome. It is usually associated with pre-eclampsia or eclampsia, but these are not a prerequisite for diagnosis. Severe HELLP syndrome has a 5% maternal mortality. HELLP rarely presents before the 20th week of gestation, but one-sixth of cases present before the 3rd trimester and a further third present postnatally (usually within 48h of delivery). Symptoms are sometimes of a vague flu-like illness, which may delay diagnosis. Maintain a high index of suspicion.

Features of HELLP

- Evidence of haemolysis (a falling Hb concentration without evidence of overt bleeding, haemoglobinuria, elevated bilirubin in serum and urine, elevated lactate dehydrogenase (LDH)). Haemolysis is the least common element of HELLP and is an indication of severe disease.
- Elevated LFTs: AST or ALT. Epigastric or RUQ abdominal pain is present in 90% of women with HELLP. Liver failure and hepatic rupture may occur. Elevation of AST to >150 units/L is associated with a poorer maternal outcome. Consider the differential diagnosis of acute fatty liver of pregnancy. Remember the potential for hypoglycaemia. Most women with RUQ pain and a platelet count of $<20 \times 10^9$/L have had an intrahepatic or subcapsular bleed.
- A falling platelet count: platelets $<100 \times 10^9$/L are of concern, while a count $<50 \times 10^9$/L is indicative of severe disease.
- Hypertension and proteinuria are present in 80% of women with HELLP and 50% suffer nausea and vomiting. Convulsions and GI haemorrhage are occasional presenting features.

The only definitive treatment is delivery of the placenta. Steroids do not alter disease progression, but if maternal condition allows, delivery may be delayed, allowing administration of steroids to promote fetal lung maturity if needed.

- The method of delivery depends on maternal condition and the likelihood of successfully inducing labour. Severe HELLP syndrome may require an urgent CS.
- Coagulation abnormalities may preclude the use of CNB. Consideration must be given to both the absolute platelet number as well as its rate of fall. All patients require a clotting screen.
- Be prepared for major haemorrhage.

Further management is supportive, with appropriate replacement of blood products as required.

- Invasive monitoring is dictated by clinical condition of the patient.
- ARDS, renal failure and DIC may develop.
- After delivery of the placenta, recovery starts within 24–48h.

Massive obstetric haemorrhage

At term, the gravid uterus receives 10–20% of the CO and when haemorrhage occurs, blood loss can be rapid. In the developing world, haemorrhage is the leading cause of maternal death. 'Normal' blood loss after vaginal delivery is of the order of 250–400mL and around 500–1000mL after CS. Blood loss is usually underestimated.

Although various definitions have been proposed for massive haemorrhage, acute blood loss of >1000mL should prompt full resuscitation measures. Beware of women with low body weight where any loss can proportionally be more significant.

Protocols for major haemorrhage should be available in every delivery suite and training should include team simulation drills. Patients with risk factors for bleeding should be actively managed, including counselling regarding place of delivery, pre-emptive IV access and active management of the 3rd stage. Antenatal iron deficiency anaemia, which affects more than a third of pregnancies; is associated with increased bleeding at delivery and should be treated with supplementation. Consider IV iron for women who fail to respond to PO iron.

Aetiology of obstetric haemorrhage

Antenatal

- Placental abruption. Bleeding is often associated with pain. Blood loss may be concealed with retroplacental bleeding. Fetal compromise is common. While small bleeds may be treated conservatively, significant bleeds have a fetal mortality as high as 35%.
- Placenta praevia/accreta (see ➲ pp. 884–5). Usually associated with small, painless bleeds, but bleeding may be catastrophic.
- Uterine rupture. Fetal distress is almost universal. Usually happens in the presence of a previous uterine scar and is classically said to be painful, but painless dehiscence can occur.

Postnatal: the four 'T's

Tone

Uterine atony is associated with chorioamnionitis, long labour and uterine distension (e.g. polyhydramnios, multiple gestation). Uterine inversion is a rare complication and is also associated with atony. To enable resiting of the uterus paradoxically further tocolysis may be required. After the uterus is resited, uterotonics should be given.

Tissue

Retained placentas can cause massive haemorrhage, but often bleeding is minimal. Retained products of conception are the leading cause of late haemorrhage.

Trauma

Genital tract trauma can cause significant blood loss. Surgical control is usually achievable. Significant retroperitoneal haematomas, while rare, may be extensive and life-threatening.

Thrombin

The risk of haemorrhage is increased by any factor that reduces the efficacy of coagulation. AFE and abruption are the two commonest obstetric causes of hypofibrinogenaemia.

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Diagnosis

Diagnosis of haemorrhage is usually self-evident, although concealed bleeding can occur, especially with placental abruptions. In addition, signs of cardiovascular decompensation may be delayed, as women are usually young and fit and start with a pregnancy-induced expansion of their intravascular volume. Beware the woman with cold peripheries—this is abnormal in pregnancy. Hypotension is a late and worrying sign.

Management

- In the event of a major haemorrhage requiring surgery, do not delay operation until X-matched blood is available.
- Clear communication with the patient, relatives and other members of the health care team is vital.
- Ongoing blood loss calculation should be contemporaneously communicated between team members and recorded.
- Call for help. Senior anaesthetic and surgical staff should be present. Blood transfusion services should be alerted.
- Follow ABC principles.
- Give supplemental O_2. If laryngeal reflexes are obtunded, intubate and ventilate.
- In antenatal patients, avoid aortocaval compression.
- Insert two 14G cannulae and take blood for X-matching, FBC and clotting screen, including fibrinogen. Bedside measurements of Hb, lactate and clotting, when available, are very useful, but a single normal bedside Hb estimate in the face of acute massive bleeding should not delay giving of blood.
- Fluid resuscitate initially with crystalloid (ideally warmed).
- If required, give Group O rhesus-negative blood (i.e. ongoing blood loss of 2–3L and/or the presence of ECG abnormalities).
- Start appropriate monitoring. Urine output and invasive monitoring of central venous and arterial pressures may be indicated, depending on rate of blood loss and maternal condition. However, early monitoring of CVP is not essential, as hypotension is almost always due to hypovolaemia.
- Treat the cause of haemorrhage (see below).
- If surgery is required:
  - Do not perform CNB if the patient is hypovolaemic.
  - Beware coagulopathies in the presence of concealed abruption.
- With continuing haemorrhage, further equipment, including warming devices and rapid transfusion devices and lines, should be available. Temperature should be monitored and optimised to aid coagulation.
- Fibrinogen is important and levels of <2.0g/dL after 1L of blood loss are associated with major haemorrhage. Aim to maintain fibrinogen >2.0g/dL. This can be done with fibrinogen concentrate or cryoprecipitate. Two pools of cryoprecipitate will increase fibrinogen concentration by ~1.0g/dL, depending on the rate of consumption. FFP, although containing some fibrinogen, may not be able to elevate fibrinogen concentration above 2.0g/dL, as plasma from non-pregnant donors generally have fibrinogen concentrations below this level and so may conversely contribute to dilution.[13]

- Goal-directed therapy using bedside monitoring of coagulation (TEG®/ROTEM®) is used in some centres, following agreed treatment algorithms.
- In the absence of clotting results, in cases where hypofibrinogenaemia may be suspected (e.g. massive abruption, AFE), empiric use of cryoprecipitate or fibrinogen concentrates may be considered.
- If cryoprecipitate or fibrinogen concentrates are not available, after 4 units of red cells and ongoing bleeding, give ~15mL/kg of FFP. Larger volumes may be required if the PT/APTT ratio is >1.5.
- Platelets should be transfused to maintain a level of $>75 \times 10^9$/L.
- The antifibrinolytic agent tranexamic acid (1g slow IV repeated after 30min if bleeding continuing) has been shown to reduce death due to bleeding from postpartum haemorrhage, with no increase in complications. It should be given as soon as possible in haemorrhage of over 1000mL.[14]
- Cell salvage use is now well established in obstetric practice. Although most recent evidence shows no significant reduction in allogeneic blood transfusion when used routinely during CS, it may be useful in situations of haemorrhage or where haemorrhage is expected.[15] To reduce the reinfusion of fetal tissue, do not collect fluid that is rich in amniotic fluid. The use of leucocyte depletion filters may reduce contamination, although more recent data indicate the filtration process alone is effective without the risk of filter-related bradykinin-mediated hypotension and the increased processing time. With major rapid blood loss, allogeneic blood will still be needed.
- During massive transfusion, calcium supplementation may be needed.
- Once blood loss has been controlled, before extubation, make sure the patient is warm, has a normal lactate and is passing urine. Continue care on HDU or ICU.

Specific treatment for haemorrhage

Think of the cause of the bleeding. Often treatment is surgical, by either removing tissue or repairing trauma. Uterine atony can be treated with uterotonics and, in some circumstances, can also be treated surgically with uterine brace sutures. Applying direct pressure to the bleeding site can be done manually, or with balloons or packs. Ultimately, hysterectomy may be required to control bleeding.

- Firm bimanual pressure can temporarily control postpartum haemorrhage due to uterine atony.
- Manual or mechanical, external or internal pressure on the aorta may be lifesaving.
- Uterotonics can only be used in the postnatal period. A common sequence is 2–5 units oxytocin bolus, oxytocin infusion, ergometrine 500 micrograms slow IV or IM, carboprost 250 micrograms IM repeated every 15min (max 2mg). There are contraindications to all these agents, so consider the appropriate uterotonic on an individual basis (see ➔ p. 883).
- Uterine compression brace sutures (B-Lynch suture, etc.) may be helpful.

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- Intrauterine balloon tamponade (Bakri® balloons, Rusch balloons, condom catheters, Sengstaken–Blakemore or Foley catheters have all been advocated). Usually they are left *in situ* for up to 24h and then deflated over a period of hours.
- Surgical ligation of blood vessels, direct pressure on bleeding points and firm manual pressure can all help control blood loss, while a circulating volume is re-established and/or coagulopathy is corrected. Consider leaving packs *in situ* and then re-exploring the abdomen 6–24h later.
- Interventional radiology is especially useful when major haemorrhage is anticipated. It may not reduce the incidence of Caesarean hysterectomy but probably reduces blood loss. Balloon catheters can be prophylactically placed in the anterior division of the internal iliac vessels before delivery (and then inflated after delivery). Interventional radiology can also be used during an unexpected major bleed, but be very cautious of moving a patient to a radiology suite if they are cardiovascularly unstable. The relative risk and benefit of interventional radiology is still being assessed.
- Ultimately, hysterectomy may be required. The decision to perform a hysterectomy should not be delayed until the patient is in extremis.

Commonly used uterotonics and doses

There are substantial differences in the dose requirements, particularly of oxytocin. In women undergoing elective CS, the dose of oxytocin required to produce effective uterine contraction is substantially lower than for intrapartum women. Either oxytocin or carbetocin can be used as 1st-line drugs. Second-line drugs are either ergometrine or a prostaglandin.[16] (See Table 35.5.)

Table 35.5 Uterotonics

Drug	Dose	Comment
Oxytocin (e.g. Syntocinon®)	2–5 unit bolus 30–50 units in 500mL of crystalloid and titrated as indicated	Synthetic hormone causing uterine contraction and peripheral vasodilation 5 units can cause a temporary drop in systolic BP of 30%; tachycardia is common. Give slowly Dose requirement for elective CS is lower than that for intrapartum women Has mild ADH action
Carbetocin	100 micrograms IV over 1min	Long-acting analogue of oxytocin (half-life 40min vs 5–10min). Experience still limited in pre-eclampsia, eclampsia and epilepsy; manufacturer suggests to avoid it in these groups. Relatively expensive
Ergometrine	0.5mg IM or slow IV injection	An ergot alkaloid derivative Effective uterine constriction Nausea and vomiting are very common. Systemic vasoconstriction may produce dangerous hypertension; avoid in at-risk groups (e.g. pre-eclampsia and specific cardiac diseases)
Carboprost (e.g. Hemabate®) (15-methyl prostaglandin F2α)	0.25mgIM every 15min to a max of 2mg	Uterine constrictor Causes nausea, vomiting and diarrhoea. May produce pyrexia and severe bronchospasm, alter pulmonary shunt fraction and induce hypoxia (caution in asthmatics)
Misoprostol	0.8–1mg PR	Uterine constrictor. Same cautions as carboprost

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Placenta praevia and accreta

Placenta praevia

The term placenta praevia is used when the placenta partially or completely covers the internal os, and 'low-lying' describes when the placenta sits within 20mm of it. A low-lying placenta resolves in many cases as the lower segment of the uterus develops during pregnancy. Diagnosis is usually made by ultrasound. Repeat ultrasounds are offered for assessment nearer term. The incidence is ~1:200 pregnancies, but higher with previous uterine scars and multiparity.

Obstetric management usually involves preserving the pregnancy until the 36th gestational week. The risk of haemorrhage increases rapidly after this. Premature labour, bleeding or fetal distress may necessitate earlier delivery. If at 36w gestation, a vaginal delivery is not possible, a CS is performed.

Patients with placenta praevia are at risk of haemorrhage: firstly because the lower uterine segment does not contract as effectively as the body of the uterus, so after separation of the placenta, the placental bed may continue to bleed following delivery; secondly because the obstetrician may have to divide the placenta at LSCS to deliver the fetus; thirdly because the placenta may have grown into the uterine wall (placenta accreta—see below), preventing the placenta from separating and the uterus from contracting.

Three questions can be used to evaluate the implications of placenta praevia:

- Is a vaginal delivery possible? This is unlikely if the placenta extends to within 2cm of the os, especially if the fetal head is not engaged in the pelvis.
- If not, does the placenta cover the anterior lower segment of the uterus? If it does, the obstetrician may have to divide the placenta at LSCS to deliver the fetus and increased blood loss is likely.
- Is there a uterine scar from previous surgery? Placenta accreta is more common if the placenta overlies a uterine scar and the risk of significant haemorrhage increases further.

Placenta accreta, increta and percreta

Placenta accreta occurs when the placenta implants abnormally into the uterine wall.

- In **placenta accreta**, the placenta grows through the endometrium to the myometrium.
- In **placenta increta**, the placenta grows into the myometrium.
- In **placenta percreta**, the placenta invades through the myometrium into the uterine serosa and surrounding structures.[17]

The endometrium usually provides a cleavage plane between the placenta and the myometrium. Because this plane is absent, the placenta fails to separate from the uterus post-delivery and uterine contraction is impaired. This can result in life-threatening haemorrhage. The risk of haemorrhage increases sequentially with placenta accreta, increta and percreta.

The incidence of accreta is rising, possibly because of the increasing numbers of CS. The risk of an adherent placenta increases with the number of uterine scars. With three uterine scars and an overlying placenta, the chance of an accreta is 40%, increasing to 61% and 67% with four or five

scars respectively. Previous uterine surgery, the presence of uterine fibroids and previous uterine compression sutures are also associated with placenta accreta. Diagnosis of percreta may be made by ultrasound, but if still equivocal, MRI may be helpful. However, placenta accreta and increta are often diagnosed at surgery.

Anaesthetic management

- Anaesthetic management is dictated by the likelihood of major haemorrhage, maternal preference and experience of the team.
- Although the sympathectomy that occurs with CNB may make control of BP more difficult, practical experience shows that CNB can be safely used for placenta praevia, providing the patient is normovolaemic before starting. However, if significant haemorrhage does occur, hypotensive and bleeding patients require reassurance which may divert the anaesthetist's attention and the experience is unlikely to be pleasant for the mother.
- CNB anaesthesia should therefore only be undertaken by experienced anaesthetists with additional help available. If CNB anaesthesia is considered appropriate, mothers should be warned that a GA may be required, usually in the first 10min after delivery.
- If intubation is anticipated to be difficult, it should be undertaken at the start of the operation, so that the anaesthetist is not dealing with bleeding and a difficult intubation at the same time.
- For bleeding patients, a GA is the preferred choice.

Technique

- Experienced obstetricians and anaesthetists are essential.
- Interventional radiology may be considered (see pp. 881–2).
- Two to 8 units of blood should be X-matched, depending on the anticipated risk of haemorrhage, and equipment for massive haemorrhage must be present.
- Cell salvage should be used if available (see pp. 458–9).
- Obstetric staff experienced in Caesarean hysterectomy should be immediately available.
- Two 14G cannulae should be inserted.
- If CNB anaesthesia is used, a CSE may offer advantages as the surgery may be prolonged.
- Have a selection of uterotonics to hand. Even if massive haemorrhage is not encountered, an oxytocin infusion is advantageous.
- If massive bleeding does occur, follow massive obstetric haemorrhage guidelines (see pp. 879–82). Remember hysterectomy may be the only method of controlling bleeding. Excessive delay in making this decision may jeopardise maternal life. Close communication with the surgical team is crucial.
- Even if no significant bleeding occurred intraoperatively, continue to observe closely in the postnatal period as haemorrhage may still occur.

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Amniotic fluid embolism

AFE is recognised by the abrupt onset, during labour, delivery or immediately postpartum, of hypoxia, cardiovascular collapse (usually right heart failure initially, followed by left heart failure and pulmonary oedema), seizures or DIC.[18,19] It is thought to be caused by fetal tissue entering maternal circulation and causing abnormal activation of immunological mechanisms.

Because the diagnosis is commonly one of exclusion, the estimate of the incidence varies considerably but is of the order of 1:12 000 to 1:40 000 deliveries, with a mortality of 10–40%.

- AFE is associated with:
  - Age >35y
  - Multiparous women
  - Obstructed labour, particularly associated with uterine stimulants
  - Multiple pregnancies
  - Short labours
  - Operative deliveries.
- AFE should be considered if any of the following occurs in the absence of other potential causes:
  - Sudden collapse with acute hypotension and fetal distress
  - Pulmonary oedema (>90% of cases) and cyanosis (80%)
  - Coagulopathy (80%)—fibrinogen commonly falls precipitously; remember that haemorrhage may be concealed
  - Seizures (50%)
  - Cardiac arrest (occurs in nearly 90% of severe cases).

Care with the use of uterine stimulants and timely diagnosis of obstructed labour may help to reduce the incidence.

Once AFE has occurred, treatment is purely supportive:

- ABC
- Senior staff should be present
- Maternal and fetal survival are dependent on early delivery of the fetus:
  - Alert haematology and activate the major haemorrhage plan.
  - Hypofibrinogenaemia is very common in AFE. Measure coagulation profile regularly. If bedside measurement of clotting is not available (TEG® or ROTEM®), consider giving prophylactic cryoprecipitate or fibrinogen concentrate and platelets.
  - ICU will be required for those who survive the initial insult.

Obesity and pregnancy

(See also ➔ pp. 70–81.)

In high-income countries, obesity is increasing. Adult obesity prevalence in England has almost doubled between 1993 and 2019.[20]

Maternal obesity is a major risk factor for death from thromboembolic disease and cardiac disease. It is also associated with gestational diabetes, pre-eclampsia, postpartum haemorrhage, wound infections, operative deliveries and shoulder dystocia. Fetal effects include an increased incidence of miscarriage, neural tube defects, macrosomia and admission to neonatal intensive care.

Anaesthetic concerns include difficult IV access, difficulty in performing CNB techniques, a propensity for desaturation due to a combination of reduced FRC and ↑ O_2 consumption, an ↑ difficulty in intubation and high inflation pressures. OSA can develop or be made worse by pregnancy.

Management is geared to reviewing women in the antenatal period, assessing the level of risk, especially if GA is required, and having a plan documented in the patient's notes.

In many units, a BMI of 40 at booking is the cut-off for reviewing women in an anaesthetic antenatal clinic, although internationally, recommendations vary from BMI >30 to BMI >45.

Key points to consider are:

- Is intubation or GA likely to be problematic?
  - If yes, women should be encouraged to have an epidural early in labour and they should be warned that in the event of a GA, there may be delay to ensure that a senior anaesthetic team is present. This delay may have implications for the fetal health if fetal distress is present.
  - There should be a written plan documenting who needs to be present for an anaesthetic.[21] In general, this should be a senior anaesthetist, but individual assessment should be made. One or even two consultants may be required for some individuals.
- Are the lumbar spinous processes palpable?
  - It is harder to site epidurals in larger women, with failure of epidural analgesia reported to be up to 25%. There is also an ↑ risk of dural puncture if the depth to the epidural space is >6cm. When the spinous processes are difficult to palpate, it is more likely that CNB will be difficult.
  - Advise women of the advantage of an early epidural. This allows time for senior anaesthetists to be summoned and for resiting if the epidural fails.
  - There should be written guidance as to who should site the epidural.
  - Some centres recommend ultrasound to assist in siting epidurals. However, experience is needed. If ultrasound is used, having the probe in a longitudinal orientation gives a more accurate prediction of the depth to the epidural space than when the probe is transverse.
- Do patients suffer from sleep apnoea?
  - If so, are they using CPAP machines? If they do, remind patients to bring their CPAP machine to the delivery suite.
  - Look for signs of right heart failure (ECG/echocardiography as indicated).
  - Remember these patients may become very sedated by opioids. Especially in the event of a GA, think about whether HDU care is needed postoperatively.

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- Is venous access likely to be difficult?
  - If so, early venous access should be obtained. Ultrasound may be of assistance. Very rarely, central venous lines may be needed.
- At the antenatal visit, some discussion about limiting weight gain in pregnancy and weight loss after pregnancy may be helpful but needs to be done diplomatically.

Consideration should also go into the equipment available in the delivery suite, maternity theatres and maternity wards. Most units have the appropriate BP cuffs, theatre tables, lateral transfer equipment such as hover mattresses and a selection of airway devices. Some units use ramps to position patients on theatre tables. Many units do not have the appropriate hoists, wide wheelchairs and seated scales for the supermorbidly obese.

When admitted in labour, make sure that all members of the team are aware of the plan and that appropriate equipment is to hand.

- If an operative procedure is required, remember that epidural and, to a lesser extent, spinal anaesthesia will often spread higher than expected. Watch blocks as they develop very closely and divide epidural top-ups into small aliquots.
- If a GA is required, consider HFNO as part of the preoxygenation process. Be very careful to optimise maternal position. Consider steep head-up position at both intubation and extubation.
- Make sure that appropriate airway equipment is immediately to hand.
- Make sure that the surgical team for operative procedures is also appropriately senior.
- Beware a large panniculus (excessive fatty tissue in the lower abdomen). This can worsen aortocaval occlusion and make surgery very difficult. Sometimes, rather than reflecting the panniculus, the surgical incision is performed above it.
- Remember that recovery for these individuals, especially those with sleep apnoea, is potentially dangerous. Think about the appropriate site and level of observation required.

Maternal sepsis

Maternal sepsis is a life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion or the postpartum period and accounts for 15% of maternal deaths worldwide. The pathophysiology remains incompletely understood, but sepsis is associated with a dysregulated host response to infection. Organ dysfunction can be considered to be an acute increase in the Sequential Organ Failure Assessment (SOFA) score by ≥2 points and reflects an overall mortality risk of ~10% in a general hospital population with suspected infection.[22] Patients with suspected sepsis can be stratified as high risk using the qSOFA (a quick SOFA or 'HAT') which is two or more of:

- **Hypotension**: systolic BP ≤100mmHg
- **Altered mental status**: any GCS <15
- **Tachypnoea**: RR ≥22.

- Septic shock is defined as sepsis with a vasopressor requirement to maintain a MAP >65mmHg plus a lactate level >2mmol/L in the absence of hypovolaemia.[23]
- Maternal deaths are often associated with a failure to recognise sepsis quickly and inadequate or inappropriate early treatment. Early recognition and treatment of the signs and symptoms of maternal sepsis are crucial.
- Recognition of sepsis may not be easy. Use of 'track and trigger' systems, such as Modified Early Obstetric Warning Scoring (MEOWS) charts, is recommended. Pregnancy causes modulation of the immune system to accept foreign proteins and this increases the risk of infection. Remember women can appear well, despite widespread inflammation. Usually HR, BP, temperature and/or RR give an indication of early stages of sepsis. Once suspected, a key element to the management is early review by a senior doctor and instigation of urgent management and resuscitation.
- Some or all of the following may be present:
  - Temperature >38°C or <36°C. Hypothermia is a significant finding that may indicate severe infection
  - Persistent HR >100bpm
  - Tachypnoea (RR >22 breaths/min)
  - Leucopenia or leucocytosis (WCC <4 × 10^9/L or >12 × 10^9/L)
  - Sore throat, flu-like symptoms or a productive cough
  - Diarrhoea and/or vomiting
  - Abdominal, pelvic or loin pain
  - Premature rupture of membranes ± offensive vaginal discharge (offensive suggests anaerobes; serosanguinous suggests streptococcal infection)
  - Abnormal or absent fetal heartbeat
  - Rash
  - Impaired mental state/confusion/lethargy
  - Headache/neck stiffness
  - Urinary symptoms
  - Wound infection: spreading cellulitis or discharge

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- Hypotension
- Arterial hypoxaemia
- Raised lactate
- Acute oliguria (urinary output <0.5mL/kg/h)
- Deranged renal function
- Deranged liver function
- Altered mental status
- Coagulation abnormalities
- Hyperglycaemia in the absence of DM.

Management of patients with suspected/confirmed sepsis

- Management of sepsis is aimed at stabilising the patient while diagnosing and treating the underlying cause. Treatment is more likely to be effective if appropriate therapy is started early.
- A multidisciplinary team approach is required and should include obstetricians, midwives, anaesthetists, microbiologists and critical care staff. Critically ill patients should be cared for in level 2 or 3 facilities with the capability for invasive techniques of monitoring and experienced nursing/midwifery staff.
- A protocoled approach is recommended for early resuscitation with goal-directed treatment.
- While the 'sepsis six' is no longer the sole goal, following its principles remains useful for early treatment. Within 1h, aim to achieve:
  - O_2 therapy (maintain saturations >94%)
  - Bloods, blood cultures and septic screen
  - IV antibiotics
  - Fluid therapy
  - ABG (monitoring pH and lactate)
  - Continuous monitoring, including urine output.
- Reassess the patient regularly; involve critical care as necessary and ensure the consultant obstetrician and anaesthetists are informed and updated. Source control should also be considered a priority.

Cardiac disease and pregnancy

(See also ➔ Chapter 5.)

Cardiac disease remains the leading cause of maternal death in the UK.[24] Cardiomyopathies and IHD are each responsible for ~25% of the total deaths, with sudden arrhythmic death and aortic dissection making up another 25%. Valvular disease accounts for a little under 10% of cardiac deaths.

Pregnancy and labour present a significant cardiac stress to women. As a generality, if women are symptomatic with minimal activity before pregnancy, particularly if symptomatic at rest, the course of pregnancy is likely to be stormy, with mortality of the order of 20–30%. Prepregnancy counselling is crucial in high-risk individuals.

Pregnancy is not recommended in women with the following:

- Pulmonary arterial hypertension
- Severe ventricular dysfunction (EF <30% or NYHA classes III–IV)
- Previous peripartum cardiomyopathy with residual impairment
- Severe MS or severe symptomatic AS
- Systemic RV with moderate/severely reduced function
- Severe aortic dilation or severe (re-)coarctation
- Vascular Ehler–Danlos syndrome
- Fontan with any complication.

General management is covered in this section as an outline only—each patient should have an individualised plan; assess early and involve a multidisciplinary team consisting of a combination of obstetricians, anaesthetists, cardiologists, midwives and neonatologists. Investigations should be performed as indicated. The risk to the fetus from procedures such as chest radiographs is minimal.

Have a written plan for the delivery and ask the woman to keep a copy on her at all times.

- Consider site (ranging from a normal delivery suite to a cardiac theatre in a tertiary centre) and modality of delivery. Is vaginal delivery acceptable? Is epidural analgesia indicated? Should pushing in the 2nd stage be limited? With each condition, consider the effects of vasodilation, vasoconstriction and positive and negative inotropic and chronotropic agents. Have written guidance on the acceptability of CNB analgesia/anaesthesia or GA, as well as the use of oxytocin (potent vasodilator) and ergometrine (potent vasoconstrictor). Think about the appropriate treatment for hypotension.
- Consider anticoagulation. Patients with mechanical heart valves are at high risk in pregnancy. Warfarin, despite its known teratogenicity, is the anticoagulant of choice for women with mechanical heart valves if the daily dose is <5mg/day.
- In most situations, rapid changes in pre- or afterload should be avoided, so always use oxytocin with extreme caution and preferably only as an infusion.
- Expect the period of highest risk to be in the 1–2h post-delivery (vasoactive uterotonics are given; there is unpredictable blood loss and unpredictable volume of autotransfusion and CO usually peaks).
- Continue management on ICU if appropriate

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General considerations for specific conditions

- Pulmonary hypertension has a very high maternal mortality.
- Fixed CO states—avoid sudden changes in afterload.
- Cyanotic heart lesions (i.e. right-to-left shunts) will not tolerate reductions in SVR; nevertheless, epidural analgesia is sometimes used to minimise the stress of labour. Onset of analgesia must be slow, and use phenylephrine to maintain afterload. GA is probably the technique of choice for CS.
- AS may become symptomatic during pregnancy. Serial echocardiography is often used. GA or slow-onset CNB have both been advocated for CS. The technique is probably less important than the skill with which it is applied. Avoid tachycardia and reduction in afterload. Loss of sinus rhythm should be treated promptly.
- Valvular insufficiencies are usually well tolerated during pregnancy.
- Women with symptomatic Marfan's disease (particularly if the aortic root is dilated >50mm or 27mm/m² body surface area) or type 4 (vascular) Ehlers–Danlos syndrome have a high risk of aortic dissection. They are usually maintained on β-blockers. Unexplained severe chest pain is an indication for CXR and echocardiography.
- MI during pregnancy has 20% mortality. Infarction occurs most commonly in the 3rd trimester. If possible, delivery should be delayed at least 3w after infarction. Both elective CS and vaginal delivery have been advocated. In either case, cardiac stress should be minimised with effective analgesia.
- Peripartum cardiomyopathy is a dilated cardiomyopathy that occurs between the last month of pregnancy and 5mo postpartum. The diagnosis is based on echocardiography and is a diagnosis of exclusion. The incidence has marked geographic variation, ranging from 1:100 in parts of Nigeria to 1:4000 in the US. Estimates of mortality range from 7% to 50%. The treatment should be multidisciplinary, with the expectation of severe LV dysfunction. Bromocriptine may be used to stop lactation and enhance LV recovery, but is controversial. The cardiomyopathy can recur and if the cardiomyopathy did not completely resolve, the mortality in subsequent pregnancies is very high. Preconceptual counselling is crucial.

Surgery during pregnancy

One to 2% of women require incidental surgery during pregnancy. Remember that although various fetal risks (such as fetal loss, premature labour, low birthweight (LBW) and teratogenicity) are attributed to surgery during pregnancy, fetal wellbeing is intimately tied to maternal wellbeing. In general, what is good for the mother is good for the fetus.

General considerations

- When possible, delay surgery until the postnatal period or alternatively into the 2nd trimester, when teratogenic risks to the fetus are reduced (the fetus is at greatest risk of major teratogenesis during the first 12w of gestation). Risks must be discussed (including miscarriage) with the patient and documented.
- Make sure that the obstetric team are aware that surgery is planned.
- Remember gastric acid and VTE prophylaxis. Pregnant women are hypercoagulable from the 1st trimester.[25,26]
- Consider regional anaesthesia. The combination of a mother maintaining her own airway together with minimal fetal drug exposure is desirable. However, data demonstrating that regional anaesthesia is safer than GA are lacking.
- In asymptomatic women with no other indication for intubation, it is acceptable not to perform an RSI up to 18w gestation. However, be aware that lower oesophageal sphincter tone is reduced within the first few weeks of pregnancy and intra-abdominal pressure rises in the 2nd trimester. Use RSI if patients are symptomatic or have additional risk factors for regurgitation.
- Every effort must be made to maintain normal maternal physiological parameters throughout the perioperative period.
- From the 20th week of gestation, use left lateral tilt to reduce aortocaval compression. Uterine blood flow may be compromised in the supine position.
- Light anaesthesia is associated with increased catecholamine release, which reduces placental blood flow.
- The tocolytic effect of inhalational agents is advantageous.
- Fetal monitoring may be beneficial. In general, in the UK, fetal HR is monitored pre- and post-surgery, but not intraoperatively.
- Treat haemorrhage and avoid hypovolaemia and anaemia which impact on fetal oxygenation.
- The 1° risk to the fetus is premature labour in the postoperative period. Detection and suppression of premature labour are vital. Women should be told to report sensations of uterine contractions so that appropriate tocolytic therapy can be instituted.
- Effective postoperative analgesia is required to reduce maternal catecholamine secretion. Regional analgesia with LA agents may be preferential. Simple analgesics such as paracetamol and codeine can be used. NSAIDs should be avoided.

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Teratogenicity

The fetus is at greatest risk of major teratogenesis during the period of organogenesis, predominantly in the first 12w of gestation. However, minor abnormalities may occur after this. Causes of teratogenicity are diverse, including infection, pyrexia, hypoxia and acidosis, as well as the better recognised hazards of drugs and radiation. Establishing whether drugs are teratogens can be difficult. Current information on the risk of exposure to many medications (but excluding most anaesthetic agents) can be found at the UK Teratology Information Service (http://www.uktis.org) or the European Network of Teratology Information Services (ENTIS) (https://www.entis-org.eu).

Premedication

- Benzodiazepine exposure just before delivery may cause neonatal drowsiness and hypotonia. Case reports associating benzodiazepines with cleft lip formation have not been substantiated and a single dose has never been associated with teratogenicity. Chronic administration may cause neonatal withdrawal symptoms post-delivery.
- Ranitidine and omeprazole are not known to be harmful.

Induction agents

- Thiopental. Clinical experience with thiopental suggests that this is a very safe drug to use.
- Propofol is safe to use during CS at term, but use in early pregnancy has not been formally investigated, although it is not teratogenic in animal studies.
- Etomidate is an inhibitor of cortisol synthesis, and if used for CS, neonates have reduced cortisol concentrations. It is not teratogenic in animal studies.
- Ketamine should be avoided in early pregnancy as it increases intrauterine pressure, resulting in fetal asphyxia. This increase in intrauterine pressure is not apparent in the 3rd trimester.

Inhalational agents

- Halogenated inhalational agents have been used extensively in pregnancy and are safe. While theoretical concern has been expressed about an increase in neuroapoptosis with these agents, a single relatively short exposure is unlikely to have negative effects.[27] At high concentrations, maternal BP and CO fall, resulting in a significant reduction in uterine blood flow. These agents also cause uterine relaxation, which may be beneficial.
- Despite early concerns, epidemiological studies suggest that N_2O is safe. However, given that anaesthesia can be easily delivered without N_2O, it is sensible to avoid this agent.
- Muscle relaxants: because these agents are not lipophilic, only very small quantities cross the placenta and so fetal exposure is limited. These agents are safe to use.
- Anticholinesterase inhibitors: these agents are highly ionised and so, like muscle relaxants, do not readily cross the placenta and are safe to use. Chronic use of pyridostigmine to treat myasthenia gravis may cause premature labour.

Analgesics

- Opioids readily cross the placenta, but brief exposure is safe. Long-term exposure will cause symptoms of withdrawal when the fetus is delivered. Animal studies suggest possible fetal teratogenicity if prolonged hypercapnia or impaired feeding develop as side effects of opioid exposure.
- Chronic exposure to NSAIDs in early pregnancy may be associated with increased fetal loss and in the 3rd trimester may cause premature closure of the ductus arteriosus and persistent pulmonary hypertension of the newborn. Single doses are unlikely to be harmful.
- Bupivacaine and lidocaine are safe. When used near delivery, bupivacaine has no significant neonatal neurobehavioural effects, while lidocaine may have a mild effect. Cocaine abuse during pregnancy increases fetal loss and may increase the incidence of abnormalities in the genitourinary tract.

Antiemetics

- Concern has been expressed about an association between chronic exposure to ondansetron during the 1st trimester and cleft lip. Where possible, ondansetron should be avoided in the 1st trimester; however, single doses are unlikely to increase risk significantly.

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Cervical cerclage (cervical stitch)

(See ➔ p. 893 for surgery during pregnancy.)

Procedure	Surgical treatment of incompetent cervical os
Time	20min
Pain	+
Position	Lithotomy
Blood loss	Nil
Technique	Spinal, epidural or GA

Cervical cerclage is performed to prevent premature cervical dilation and loss of fetus (usually in 2nd trimester). It is one of the commonest surgical procedures during pregnancy. Causes of an incompetent cervix include: congenital abnormalities, cervical scarring/surgery and hormonal imbalance. Usually performed between 14w and 26w gestation, rarely preconception. Emergency cerclage may be required in the face of a dilating cervix and bulging membranes, although this is less successful than prophylactic cerclage.

Type of surgery

- Transvaginal procedures are commonest (Shirodkar and McDonald procedures). They require anaesthesia for insertion but can be removed by obstetricians at around the 38th week gestation without anaesthetic.
- The transabdominal procedure requires two operations, one for insertion and another for a CS for delivery and removal of the suture. It also carries a greater risk of ureteric involvement.

Preoperative

- The risks of cerclage include membrane rupture (more common if the membranes are already bulging), infection, haemorrhage and inducing premature labour.
- As per surgery during pregnancy.

Perioperative

- Both CNB and GA may be used.
- If GA is used and uterine relaxation is required to allow bulging membranes to be reduced, the halogenated vapour concentration can be increased.
- For regional anaesthesia, a T8–T10 level is required for intraoperative comfort. If uterine relaxation is required, 2–3 puffs of sublingual GTN spray may be used and repeated as necessary, although transient hypotension is to be expected.

Postoperative

- As per surgery during pregnancy.

Maternal resuscitation

Maternal cardiac arrest is fortunately rare. All the normal resuscitation drugs should be used as indicated and defibrillation is safe for the fetus. Adrenaline is also the drug of choice in major anaphylactic reactions. Severe hypotension associated with anaphylaxis results in very poor fetal outcomes.

The basic algorithms for adult resuscitation (see pp. 1052–7) are appropriate for maternal resuscitation, with several important differences:

- After 20w gestation, attempts must be made to minimise aortocaval obstruction while performing effective cardiac compressions. The fetus can be displaced with firm lateral pressure (manual uterine displacement).
- After 4min, if CO has not been re-established, the fetus should be delivered. This improves the chance of maternal, as well as fetal, survival.
- Pregnant women have reduced oesophageal sphincter tone and both cricoid pressure and intubation should be performed as early as possible.

Consideration should be given to the diagnosis and treatment of obstetric causes of maternal arrest, including:

- Cardiac events
- Intracranial events
- Sepsis
- Haemorrhage
- PE
- AFE
- Iatrogenic events:
  - Hypermagnesaemia: treat with 10mL of 10% calcium chloride or gluconate
  - High or total spinal (which is the commonest cause of maternal cardiac arrest on delivery suites in the UK)[28]—supportive treatment
  - LA-induced arrhythmia—treat with 20% lipid emulsion (see pp. 1092–3).

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Chapter 36

Paediatric and neonatal anaesthesia

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➲ Tonsillectomy/adenoidectomy: child pp. *767–8*
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Neonatal/infant physiology

Paediatric anaesthesia embraces patients from the premature neonate to the adolescent. Major differences exist between the anatomy, physiology and pharmacological response of children and adults (Tables 36.1, 36.2 and 36.3). In anaesthetic terms, special considerations apply to the neonate.

Definitions

- Neonate: first 44w of post-conceptual age
- Infant: 1mo to 1y
- Child: 1–16y
- Young person: 16–18y
- Premature infant: <37w gestational age
- SGA: small for gestational age
- Low birthweight (LBW): <2500g
- Very low birthweight (VLBW): <1500g
- Extremely low birthweight (ELBW): <1000g.

Respiratory considerations

- At birth, each terminal bronchiole opens into a single alveolus instead of fully developed alveolar clustering. The 20–50 million alveoli are thick-walled and alveolar growth continues by multiplication to reach 300 million by adolescence.
- Cartilaginous ribs are horizontally aligned, so that the 'bucket handle' action of the adult thorax is not possible. Intercostal muscles are poorly developed, with a lower proportion of type 1 muscle fibres and fatigue more easily. The diaphragm has a more horizontal attachment, reducing mechanical advantage.
- Ventilation is essentially diaphragmatic and rate-dependent. Abdominal distension may cause splinting of the diaphragm, leading to respiratory failure.
- Chest wall compliance is high because of the cartilaginous thorax; intercostal or sternal recession is common with ↑ work of breathing or airway obstruction.
- Closing volume occurs within tidal breathing in the neonate. Minor decreases in FRC increase the pulmonary shunt and lead to lung collapse. The application of CPAP improves oxygenation and reduces the work of breathing.
- Narrow airways result in ↑ resistance, up to the age of 8y. Nasal resistance represents almost 50% of total airway resistance, accentuating the problem of children with nasal congestion who are obligate nasal breathers. An NGT can increase resistance by 50% in neonates.
- Apnoea is a common postoperative problem in preterm neonates. It is significant if the episode exceeds 20s or induces cyanosis or bradycardia. CPAP may be helpful, with the distending pressure triggering stretch receptors in the chest wall.
- Due to the higher metabolic rate and alveolar minute volume, volatile agents achieve a more rapid induction and emergence than with adults. They are profound respiratory depressants; most anaesthetised neonates require intubation and controlled ventilation.
- Respiratory parameters of the neonate are summarised in Table 36.1.

Table 36.1 Respiratory parameters in the neonate and adult

Parameter	Neonate	Adult
V_T (spontaneous) (mL/kg)	7	7–10
V_T (IPPV) (mL/kg)	7–10	10
Dead space (mL/kg)	2.2	2.2
V_D:V_T ratio	0.3	0.3
RR (breaths/min)	30–40	15
Compliance (mL/cmH_2O)	5	100
Resistance (cmH_2O/L/s)	25	5
Time constant (s)	0.5	1.1
O_2 consumption (mL/kg/min)	7	3

Parameters for children over 2y approximate to adult values.
Estimate RR from the formula = 24 – age/2.

Cardiovascular considerations

- PVR falls at birth, in response to a rise in PaO_2/pH and a fall in $PaCO_2$. Subsequent closure of the foramen ovale and ductus arteriosus may reverse with hypoxia and acidosis, leading to pulmonary hypertension and right-to-left shunt (transitional circulation).
- The neonate has small ventricles with reduced contractile mass and poor ventricular compliance. CO is higher than in adults (200mL/kg/min) and rate-dependent. Normal systolic pressure is 70–90mmHg with low SVR.
- HR of up to 200 bpm can be tolerated. Bradycardia occurs in response to hypoxia and should be treated with O_2, rather than atropine. Neonatal and infant HR <60 bpm require external cardiac compression.
- Autonomic and baroreceptor control is fully functional at term, but vagally mediated parasympathetic tone predominates.
- Incidence of CHD is 7–8 per 1000 live births; 10–15% have associated non-cardiac pathology. All neonates with midline defects should be assessed for related cardiac lesions.
- CVS parameters in children are summarised in Table 36.2.

Gastrointestinal considerations

- The liver is immature. Enzyme systems have matured by 12w, but some drugs are metabolised more slowly and others by different enzyme pathways from adults. The action of barbiturates and opioids in the neonate is prolonged and enhanced.
- Bilirubin metabolism is affected by a poorly developed glucuronyl transferase system. Rises in unconjugated bilirubin may lead to neonatal jaundice and kernicterus by crossing the blood–brain barrier. Some drugs (e.g. diazepam, vitamin K) displace bilirubin from plasma proteins and can exacerbate jaundice.
- Glycogen stores are reduced in neonates. The premature baby and stressed neonate are vulnerable to hypoglycaemia.

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Table 36.2 Cardiovascular parameters in children

Age (y)	HR (bpm)	Mean systolic BP (mmHg)	Mean diastolic BP (mmHg)
Neonate	80–200	50–90	25–60
1	80–160	85–105	50–65
2	80–130	95–105	50–65
4	80–120	95–110	55–70
6	75–115	95–110	55–70
8	70–110	95–110	55–70
10	70–110	100–120	60–75
12	60–110	110–130	65–80

Mean systolic BP over 1y = 80 + (age in y × 2).

- Vitamin K-dependent factors are low at term. Routine administration of vitamin K 1mg IM may prevent haemorrhagic disease of the newborn and is recommended before surgery in the 1st week of life.
- Gastro-oesophageal reflux is common in neonates.

Renal considerations

- Nephron formation is complete at term but contains only 20% of the adult cellular component.
- GFR reaches adult values by 2y as renal blood flow increases with decreasing vascular resistance.
- Tubular function is immature, reaching adult values by 6–9mo.
- Initially, neonates are unable to excrete a large water or Na^+ load.
- Glucose and Na^+ reabsorption are less efficient in premature infants.
- Because of the high metabolic rate and insensible losses, infants are more susceptible to dehydration.
- It may be necessary to reduce drug dosages or extend frequency intervals.

Haematological considerations

Circulating blood volume is estimated as shown in Table 36.3.

- Post-delivery Hb concentrations range from 130 to 200g/L (average 180g/L), depending on the degree of placental transfusion. Subsequently, Hb concentration falls, as the increase in circulating volume exceeds growth in bone marrow activity and erythropoietin levels fall. The resulting 'physiological anaemia of infancy' varies from 100 to 120g/L.
- The predominant Hb type at term is HbF (80–90%). By 4mo, this has fallen to 10–15% and been replaced by HbA. HbF has a higher O_2 affinity due to reduced 2,3-diphosphoglycerate levels.
- Preoperative Hb <100g/L is abnormal and should be investigated.

Table 36.3 Estimating paediatric circulating blood volume

Neonate	90mL/kg
Infant	85mL/kg
Child	80mL/kg

Central nervous system

- Neurones are completely formed at term, but the total number of brain cells is reduced.
- Dendritic proliferation, myelination and synaptic connections develop in the 3rd trimester and first 2y of life.
- The blood–brain barrier is more permeable in neonates—barbiturates, opioids, antibiotics and bilirubin all cross more readily.
- Autoregulation of the cerebral circulation is present from birth.
- The brain contains a higher proportion of fat, which may allow volatile agents to reach higher concentrations more rapidly.
- All neonates, however immature, feel pain. The premature neonate may be hypersensitive due to a relative increase of transmitters mediating nociception with the later development of descending inhibitory pathways.
- Dose requirements of volatile agents vary with age. The neonatal MAC is comparable to adult values and decreases with prematurity. MAC peaks at 1y (~50% greater than adult values), then declines to reach adult levels by the onset of puberty (see pp. 411–12).

Weight

- Weight estimation formulae (Table 36.4) provide a guide for calculations before the arrival of a child to the ED. They should be quickly replaced by using a Broselow tape or weighing the child.
- All paediatric patients should be weighed preoperatively.

Table 36.4 Estimating the ideal weight of paediatric patients

Birth	3–3.5kg
0–1y	Weight = [age (mo)/2] + 4
1–5y	Weight = [age (y) × 2] + 8
6–12y	Weight = [age (y) × 3] + 7

Thermoregulation

- Poorly developed thermoregulatory mechanism. High surface area to volume ratio with minimal SC fat and poor insulation. Vasoconstrictor response is limited, and the neonate is unable to shiver.
- **Non-shivering thermogenesis** is achieved by metabolism in brown fat found in the back, shoulders and legs, and around the thoracic vessels. This considerably increases O_2 consumption and may worsen pre-existing hypoxia. Brown fat is deficient in premature infants.

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- Neonates lose heat during surgery by conduction, convection and evaporation, but predominantly by radiation.
- GA depresses the thermoregulatory response. Heat is lost from the core to the cooler peripheral tissues. Prolonged hypothermia can lead to a profound acidosis, with impaired perfusion. Platelet function is impaired, but clotting factors are unaffected above 32°C. The duration of opioids and muscle relaxants is prolonged.

Measures to conserve heat loss

- Theatres should be heated before surgery to warm the walls and raise the ambient temperature; 21°C is adequate for larger children, but infants and neonates may require 26°C. In practice, this is too hot; theatre temperature of 21°C is an adequate compromise if active measures are taken to reduce heat loss and maintain the 'microclimate' around the patient. Close doors to avoid draughts.
- Avoid exposure of the child; this applies particularly in the anaesthetic room. The head is relatively large in infants and should be covered with a hat, Gamgee or polythene. The rest of the body can also be insulated with warm Gamgee and transparent drapes.
- Use active warming devices: warming mattress, convective warm air blanket, overhead radiant heaters.
- Humidify and warm anaesthetic gases. Heated water vapour humidifiers are available, but disposable HMEs are usually satisfactory.
- IV fluids, blood, surgical prep and wash fluids should be warmed.
- Temperature measurement is essential in neonatal surgery, paediatric surgery of intermediate to long duration and where major fluid and blood loss is expected.

Fluid management

Eighty per cent of neonatal total body weight is water; the value is higher in the preterm infant and reaches an adult level of 60% by 2y. Extracellular water constitutes 45% of TBW at term (over 50% in the preterm) but attains an adult value of 35% by early childhood. Plasma volume tends to stay constant at 5% of total body weight, independent of age.

- Turnover of water is over double that of the adult; 40% of extracellular water is lost daily in infants as urine, faeces, sweat and insensible losses. A small increase in loss or reduction in intake can rapidly lead to dehydration.
- Daily fluid maintenance is calculated from the calorie requirement: 100kcal/kg for the infant, with older children requiring 75kcal/kg, and adults 35kcal/kg. Each kcal requires 1mL of water for metabolism.

Neonatal fluid requirements

- Fluid is initially given cautiously, as the kidneys cannot easily excrete a water or Na^+ load. Newer 'cold-light' phototherapy does not require ↑ fluid intake.
- The fluid of choice is 10% glucose. This is adjusted in increments of 2.5% to achieve normoglycaemia. A blood sugar below 2.6mmol/L is treated with 2mL/kg of 10% glucose.
- Routinely added electrolytes are Na^+ 3mmol/kg/d and K^+ 2mmol/kg/d. Other electrolytes, including Ca^{2+}, are added as indicated.
- The first 5d of neonatal fluid requirements are given in Table 36.5.[1] Preterm requirements are dependent on birthweight and may be proportionally higher.

Table 36.5 First 5d neonatal fluid requirement (mL/kg/d)

Days of life	Term
Day 1	50–60
Day 2	70–80
Day 3	80–100
Day 4	100–120
Day 5	120–150

Paediatric fluid requirements

- Maintenance is calculated using the '4–2–1' regime.
- The fluid of choice is 0.45% sodium chloride/5% glucose:
  - 4mL/kg/h (100mL/kg/d) for each of the first 10kg
  - 2mL/kg/h (50mL/kg/d) for each of the second 10kg
  - 1mL/kg/h (25mL/kg/d) for each subsequent kg.

Maintenance requirement makes no allowance for extra losses from gastroenteritis, intestinal obstruction and insensible loss from pyrexia. Additional Na^+ and K^+ may also be required.

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Perioperative fluid management

Perioperative fluids comprise the basic maintenance requirement plus replacement of other observed fluid losses. These are replaced by isotonic crystalloid, i.e. 0.9% sodium chloride, Hartmann's solution, albumin or blood, according to the clinical need. Glucose 1% or 2.5%/Hartmann's solution (add 10mL or 25mL of 50% glucose to 500mL of Hartmann's solution) is a useful perioperative fluid for infants. Regular blood glucose measurement is essential in neonatal surgery.[1]

- Transfusion is required after 15% of blood loss or use a transfusion trigger of 70g/L.
- Estimated blood volume (EBV) (Table 34.3) and maximal allowable blood loss (MABL) (Box 36.1) should be calculated prior to surgery.
- Swabs should be carefully weighed, and suction volumes recorded. Cell salvage can be utilised for children >10kg.
- Postoperatively, use 0.45% sodium chloride/5% glucose (or Hartmann's solution for children >8–10y) at two-thirds of maintenance.

Box 36.1 Maximal allowable blood loss calculation

MABL = (Hb initial − Hb low)/Hb low x EBV

Postoperative hyponatraemia

- Postoperative hyponatraemia (serum Na^+ <135mmol/L) is uncommon, but more likely with the administration of hypotonic solutions, e.g. 0.18% sodium chloride/4% glucose.
- Symptoms are often non-specific, including nausea, vomiting and headache (a common early sign). It may also present as seizure or respiratory arrest.
- Hyponatraemic seizures respond poorly to anticonvulsants, and initial management should be to administer an infusion of 3% sodium chloride. Plan to increase serum Na^+ to >125mmol/L or until symptoms improve (1mL/kg of 3% sodium chloride should raise serum Na^+ by 1mmol/L).
- Asymptomatic hyponatraemia can be managed with 0.9% sodium chloride. If hypervolaemic, restrict fluids to 50% of maintenance.

Fluid resuscitation

Shock is the clinical state in which delivery of O_2 and metabolic substrates is inadequate for cellular demand.

- In compensated shock, oxygenation of the vital structures (brain and heart) is maintained by sympathetic reflexes at the expense of non-essential tissues. BP remains normal, with an increase in SVR.
- In decompensated shock, hypotension develops and vital organ perfusion is compromised.
- With irreversible shock, there is cyanosis, bradycardia and gasping respiration. This is a preterminal event.
- Hypovolaemia is the commonest cause of circulatory failure in children. Other causes of shock include pump failure (cardiogenic), distributive (sepsis, anaphylaxis, neurogenic) and obstructive (cardiac tamponade, tension pneumothorax).

Assessment of dehydration and hypovolaemia

Assessment of dehydration and hypovolaemia is made predominantly on clinical signs (Table 36.6).

- ↑ capillary refill time ≥2s, cold and blue peripheries and an increasing core–peripheral temperature gap with a thready pulse are early signs of hypovolaemia.
- Rising HR may reflect pain, anxiety or fever.
- Oliguria and a reduced level of consciousness are late signs.
- Hypotension does not occur until >35% of blood volume is lost (Table 36.7).

Management

- Administer fluid boluses of 20mL/kg crystalloid, either 0.9% sodium chloride or Hartmann's solution, and then reassess.
- Give blood when 15% of the circulating volume is lost (Table 36.3) or if no improvement after 40mL/kg. Aim for Hb of 70g/L or packed cell volume (PCV) of 25%.[2]
- **4mL/kg of blood raises the Hb concentration by 10g/L.** Transfused blood should be fresh, if possible, warm, filtered and CMV-negative for neonates. It can be rapidly transfused using a syringe and a three-way tap.
- The 'swing' of the arterial or SpO_2 trace is a valuable aid in assessing intravascular loss. CVP may be less sensitive in smaller children because of the greater venous capacitance.

Table 36.6 Clinical assessment of dehydration in paediatrics

Sign	5% dehydration	10% dehydration
Skin	Loss of turgor	Mottled, poor capillary return
Fontanelle	Depressed	Deeply depressed
Eyes	Sunken	Deeply sunken
Peripheral pulses	Normal	Tachycardia, weak pulse
Mental state	Lethargic	Unresponsive

Replacement volume (mL) = weight (kg) × % loss, e.g. a 10% loss in a 5kg infant requires a replacement volume of 50mL.

Table 36.7 Clinical assessment of hypovolaemia in paediatrics

Sign	Compensated	Uncompensated	Irreversible
HR	↑	↑↑	↑↓
Systolic BP	Normal/↑	Normal/↓	↑↓
Pulse volume	Normal/↓	↓	↓↓
Capillary refill	Normal/↑	↑	↑↑
Skin colour	Pale	Mottled	White/grey
Skin temperature	Cool	Cold	Cold
Mental status	Agitated	Lethargic	Unresponsive
RR	Normal/↑	↑↑	Sighing
Fluid loss	<25%	25–40%	>40%

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Anaesthetic equipment

See also ➲ Breathing systems, pp. 350–2; ➲ Airway equipment, pp. 355–8.

Oropharyngeal airway

- Range in size from 000 to 4 (4–10cm in length).
- Rarely useful in neonates who are obligate nasal breathers but may be advantageous in older children or in mask ventilation to prevent gastric distension.
- Estimating the size of the airway is crucial. Incorrect size will worsen the airway obstruction. Correct length is equal to the distance from the incisors to the angle of the jaw.
- The airway should not be inverted during insertion in infants, as this may damage the palate.

Nasopharyngeal airway

- Limited application in paediatric practice. Tolerated at lighter levels of anaesthesia than an OPA and may be of use during induction/recovery of some congenital airway problems or OSA.
- Well lubricated prior to insertion; bleeding is possible from mucosal or adenoidal trauma, especially in younger children.
- Appropriate length is equal to the distance from the tip of the nostril to the tragus of the ear.
- If an ETT is used as a modified nasopharyngeal airway, the size is calculated by: (age/4 + 3.5).

Face masks

- Clear plastic masks with an inflatable rim provide an excellent seal for SV and assisted ventilation.
- Manufactured in a round or teardrop shape; the round shape is suitable for neonates and infants. Also available as 'flavoured' masks.
- Transparent design allows for observation of cyanosis/regurgitation and the presence of breathing.
- Size is estimated to fit an area from the bridge of the nose to the cleft of the chin.

Supraglottic airway devices

- Tables 36.8 and 36.9 describes how to choose the appropriate size of SGA.
- Indications and insertion techniques are similar to adult use. An alternative method of insertion is to advance it partially inflated and upside down behind the tongue before rotating through 180°.
- Smaller sizes have ↑ complication rates. The effectiveness of these smaller masks is not established for resuscitation.
- Intubating LMA (iLMA) available in size 3 which is potentially useful for older children.
- Both the LMA ProSeal™ and i-gel® are available in a full range of paediatric sizes.[3]
- LMA is best secured in slight flexion, and i-gel® in slight extension.

Table 36.8 Estimating LMA sizes in children

Size of LMA	Weight (kg)	Cuff volume (mL)
1	0–5	2–5
1.5	5–10	5–7
2	10–20	7–10
2.5	20–30	12–14
3	>30	15–20

Table 36.9 Estimating i-gel® sizes in children

Size of i-gel®	Patient size	Patient weight guidance (kg)
1	Neonate	2–5
1.5	Infant	5–12
2	Small paediatric	10–25
2.5	Large paediatric	25–35
3	Small adult	30–60

Laryngoscopes

- Laryngoscope blades available in different lengths from size 0 to 3.
- Straight-blade preferable for infants ≤6mo due to high anterior larynx (see p. 909 for paediatric intubation).
- Polio and McCoy blades are also available.
- Many video laryngoscopes are available in paediatric sizes.

Endotracheal tubes

- Traditionally uncuffed paediatric ETTs have been used and are available from 2.0mm to 7.0mm ID. Table 36.10 describes how to choose an appropriate size of uncuffed tube.
- Uncuffed ETT ID (mm) may also approximate to the length of the child's middle finger (cm).[4]
- Standard cuffed tubes are available from 3.0mm ID. Microcuff® tubes have a more distal, high-volume, low-pressure cuff. Use of a cuffed ETT reduces intubation attempts to correctly size a tube and improves ventilation characteristics without increasing the incidence of post-extubation stridor.[5]
- Specific indications include children at high risk of aspiration, poor lung compliance and facial burns. Cuff pressure should be limited to 20cmH_2O and continuously monitored. Tube sizes are a half size lower than uncuffed tube.
- Paediatric versions of the RAE, armoured, and laser tubes all exist. A north-facing uncuffed preformed tube has been developed for routine paediatric surgery.

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Table 36.10 Paediatric endotracheal tube sizes

Weight or age	ETT ID (mm)
>2kg	2.5
2–4kg	3.0
Term neonate	3.5
3mo to 1y	4.0–4.5
Over 2yr	Tube size = age/4 + 4 Cuffed tube size = age/4 + 3.5 (also check packaging as manufacturer variation exists)

- The paediatric trachea is conical. The narrowest part is at the level of the cricoid ring, the only part of the airway completely surrounded by cartilage. If the ETT is too large, it will compress the tracheal epithelium at this level, leading to ischaemia with consequent scarring and the risk of subglottic stenosis.
- A correctly sized tube is one in which ventilation is adequate, but a small audible leak of air is present when positive pressure is applied at 20cmH_2O.
- Paediatric 8.5mm connectors can be used as an alternative to the standard 15mm connector. Catheter mounts should be avoided because of the large dead space involved.
- Tube length in cm can be calculated as:
  - Oral tube: age/2 + 12 (or tube size × 3)
  - Nasal tube: age/2 + 15.
- Tube placement needs to be meticulous to avoid endobronchial intubation or inadvertent extubation.
- To assess the length of tube to be passed below the vocal cords, use the black guide line at the distal end of the tube or the tube size in cm. Ultimately, the position must be confirmed clinically by auscultation.

Anaesthetic breathing systems

Ayre's T-piece with Jackson–Rees modification

- The Jackson–Rees modification of the Ayre's T-piece (Mapleson F) is a commonly used breathing system in paediatric anaesthetic practice. Suitable for all children up to 20kg, beyond which it becomes inefficient. Low-resistance, valveless, lightweight circuit. The expiratory limb exceeds the V_T to prevent entrainment of room air during SV. The open-ended 500mL reservoir bag or Jackson–Rees modification allows:
  - Assessment of the V_T
  - The ability to partially occlude the bag for CPAP or PEEP
  - The potential for assisted or controlled ventilation
  - Qualitative appreciation of lung compliance
  - Reduction in dead space during SV (FGF washes out expired gas during the expiratory pause).
- Scavenging is limited. However, some versions of the T-piece incorporate a closed bag with an expiratory valve and a scavenging attachment. Requirements for FGF are higher in SV than in controlled ventilation. Recommendations are 2–3 times the alveolar minute

volume for spontaneous breathing, or 1000mL plus 200mL/kg in controlled ventilation. FGF is dependent on the respiratory pattern. A rapid RR requires a higher FGF. Conversely, an end-expiratory pause during controlled ventilation will help reduce the FGF.

- Most children require a minimum FGF of 3L, which can then be adjusted to achieve normocapnia and an inspired CO_2 concentration of <0.6kPa (4.5mmHg). Partial rebreathing allows conservation of heat and humidification.
- $ETCO_2$ concentration may be underestimated in children below 10kg from dilution of expired gases. Sampling should be distal in the circuit.

Circle absorption systems

- Paediatric circle systems using 15mm lightweight hose are suitable for children over 5kg. The unidirectional valves may increase resistance to breathing and should not be allowed to become damp.
- During controlled ventilation, the leak around the ETT may require gas flows to be ↑.

Bain system

The coaxial Mapleson D system is unsuitable for children under 20kg due to resistance of the expiratory valve.

Mechanical ventilation

- Standard adult ventilators are suitable down to 20kg.
- Below 20kg, a paediatric ventilator should be capable of delivering small V_T, rapid RR, variable inspiratory flow rates and different I:E ratios.
- Calculation of a small V_T may be compromised because of compression of gases in the ventilator tubing and a variable leak around the ETT. More sophisticated ventilators may be capable of measuring expired V_T, which is of more practical value.
- Some ventilators are designed to work with specific breathing systems. The Newton valve converts the Penlon Nuffield 200 ventilator from a time-cycled flow generator to a time-cycled pressure generator and can be attached directly to the expiratory limb of the Ayre's T-piece. It is suitable for neonates and children up to 20kg.
- New anaesthetic workstations incorporate integral ventilators attached to circle systems suitable for paediatric practice.
- Pressure-controlled ventilation is commonly used and reduces the risk of barotrauma/pneumothorax. This mode will compensate for a leak around the ETT, but not for changes in lung compliance, partial or complete tube obstruction or bronchospasm.
- Volume control can make an allowance for changes in lung compliance, but at a potential cost of high peak airway pressures. It does not compensate for leaks around the ETT.
- The use of volume guarantee/pressure control provides a balance.
- Ultimately, setting ventilator parameters is based on clinical observation. Inspiratory flow, pressure or volume is gradually ↑ until adequate chest movement is observed. Measurement of capnography and SpO_2 confirms normocapnia and adequate oxygenation. The peak airway pressure is kept to a minimum.

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- Most children can be ventilated adequately with inspiratory pressures of 16–20cmH_2O and an RR between 16 and 24 breaths/min. Normally, inspiratory pressure should not exceed 30cmH_2O. The rate can be adjusted accordingly to achieve normocapnia. A minimum PEEP of 4cmH_2O is advisable for infants and neonates to maintain FRC.
- The immediate ability to hand-ventilate using the Ayre's T-piece is essential. It should always be available in the event of unexpected desaturation or ventilator failure (plus a bag–valve–mask). Mechanical ventilation may be unsuitable for the small premature neonate.
- With gastroschisis and exomphalos, hand ventilation facilitates assessment of changes in lung compliance to help determine how much of the abdominal contents should be reduced back into the abdominal cavity. Hand ventilation during repair of a tracheoesophageal fistula can allow the surgeon maximum exposure and time to perform the repair.

Conduct of anaesthesia

See ➲ Chapter 16 for general information on conduct of anaesthesia.

Preoperative assessment

(See ➲ Chapter 2 for general information on preoperative considerations.)

The preoperative visit is essential in establishing a rapport with both parents and children and in helping to dissipate anxiety. Communication should be simple, informative and truthful.

- Involve the parents, but try to question the child directly when appropriate and stay at eye level if possible.
- A preadmission visit reduces parental anxiety and is beneficial to children over 6y. Play therapists can help provide an informal setting and prepare the child by describing the course of events from the ward to induction of anaesthesia. A collection of photographs, video or virtual-reality tools may be helpful.[6]

Preoperative investigations

Routine preoperative Hb is indicated for:

- Neonates and ex-premature infants under 1y
- Children at risk of SCD (see ➲ pp. 257–9)
- Children for whom intraoperative transfusion may be necessary
- Children with systemic disease.

A preoperative Hb of <100g/L is abnormal and needs to be investigated. It does not necessarily entail cancellation if the child is haemodynamically stable and otherwise well.

Routine biochemistry is required for:

- Children with metabolic, endocrine or renal disease
- Children receiving IV fluids.

The child with an upper respiratory tract infection

The preschool child develops 6–8 URTIs per year. Almost 25% of children have a chronic runny nose due to seasonal rhinitis or adenoidal infection.

- Anaesthesia in the presence of an intercurrent URTI is associated with a higher risk of complications in younger children, particularly <1y. There is an ↑ incidence of coughing, breath-holding, desaturation, excess secretions, airway obstruction, laryngospasm and bronchospasm. This risk is ↑ if the child is intubated.
- Children with moderate to severe chest infections should be postponed. This will include those with productive cough, purulent nasal discharge, pyrexia, abnormal chest auscultation and signs of viraemia or constitutional illness, including diarrhoea and vomiting.
- The child with a mild URTI is a difficult problem. The history in these cases is crucial. It is important to decide whether the child is at the beginning or at the end of the URTI. Other members of the family or children at school may have already experienced the same infection, and this can provide useful information.
- A child deemed to be post-viral, apyrexial, with no chest signs and constitutionally well is probably fit for surgery, even if they have a runny nose.
- The decision to proceed is not always clear and requires careful discussion with the parents. Level of urgency, complexity of surgery, informed consent, good clinical judgement and experience are key factors in these decisions.

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- Preoperative bronchodilators may reduce adverse airway events. Propofol induction is associated with fewer complications than sevoflurane. Awake extubation is the safer option.
- Significant URTI requires postponement for a minimum of 2w and at least 4w if lower respiratory tract involvement is suspected. Bronchiolitis warrants a delay of at least 6w.

The child with a murmur

The majority of pathological murmurs are diagnosed perinatally. These children will already be under the care of paediatric cardiology.

- Previously unreported murmurs are commonly heard at 2–4y. The majority are functional.
- A systolic murmur with normal heart sounds and palpable peripheral pulses in a child with normal O_2 saturations and no limitation in exercise tolerance can be assumed to be innocent. If any doubt, defer surgery until a formal assessment has been made.
- NICE guidelines no longer recommend routine antibiotic prophylaxis for surgery in patients at risk of infectious endocarditis. However, if a child requires prophylactic antibiotics for a GI or genitourinary procedure, these should also include drugs effective against organisms that cause infectious endocarditis.

The anxious or uncooperative child

Two-thirds of children have significant anxiety at induction. This may be due to fear of pain, e.g. cannulation, or general anxiety about anaesthesia and the operation.

- Children with behavioural issues, such as autism or ADHD, may cause particular problems.[7,8] If the problem can be anticipated, a multidisciplinary approach should be adopted and a plan instituted for the anaesthetic room. (See ➲ pp. 95–6 for patients with learning disabilities and/or autism.)
- Some general tips:
  - Agree a plan with the parents beforehand and involve the parents as much as possible.
  - Medical equipment can be frightening. Keep the amount on display to a minimum and draw up drugs in advance.
  - Minimise the number of people in the anaesthetic room and maintain a calm, quiet atmosphere.
  - Only one person at a time should speak to the child, at eye level if possible.
  - Adapt the technique to the child's personality and developmental level.
  - Support coping strategies or distraction techniques, e.g. book, bubbles or digital aids.
- Premedication can be useful, and older children may choose this option. PO or buccal midazolam is commonly used, with dexmedetomidine or ketamine as an alternative, either alone or in combination with midazolam. Clonidine may be a useful option for autistic children.
- A decision on whether to proceed should centre on the best interests of the child.
- There should be a clinical holding policy as a guideline to facilitate clinical procedures.

Child protection

- Child protection training is mandatory for all hospital staff who work with children.[9]
- Anaesthetists may become suspicious of child abuse during resuscitation, on PICU, in the anaesthetic room, during the course of a surgical procedure or rarely by direct disclosure.[10]
- In these situations, it is essential to act in the best interests of the child.
- If there is concern about suspected abuse, the first point of contact should be the named clinical lead for safeguarding children or the consultant paediatrician on call.

Consent

(See also ➲ p. 42.)

Allow time at the end of the preoperative assessment for parents and children to ask questions. Adopt the principle of shared decision-making and discuss the risks associated with GA. Discuss the options of IV or inhalational induction and plans for postoperative pain relief.

- Obtain consent for suppository, neuraxial blockade or regional/peripheral nerve block, if indicated.
- A young person is deemed competent to consent from 16y.
- Children under 16y may have the capacity to decide, depending on their ability to understand what is involved (Gillick competence). A Gillick-competent child can consent to treatment against parental wishes but cannot refuse it.

Anaesthetic neurotoxicity

- There is evidence from infant animal models that neuronal apoptosis and neurodegeneration may be caused by prolonged or multiple anaesthetics.
- To date, results from studies in the human infant have failed to show adverse effects on cognitive development from a single anaesthetic episode of short duration <1h.
- Parents/carers can be advised that surgery is carried out in infants only when necessary and that currently there is no indication of a long-term neurological effect from a single anaesthetic exposure.[11]

Preoperative fasting

(See ➲ pp. 57–8.)

Fasting instructions (Table 36.11) are designed to minimise the risk of regurgitation of gastric contents with consequent pulmonary aspiration.

- Fasting reduces the gastric volume but does not guarantee an empty stomach. Prolonged fasting does not further reduce the risk of aspiration and, in infants, can lead to dehydration and hypoglycaemia.
- Infants may be at greater risk of regurgitation due to reduced lower oesophageal sphincter tone and a tendency for stomach distension during mask ventilation. However, the incidence of pneumonitis following aspiration in children is much lower than in adults.[12]
- One-hour fluid fasting for clear fluids does not significantly alter gastric pH or residual volume, compared with 2h.[13]
- Clear fluids can be given safely up to 1h preoperatively,[14] and the intake of fluids (either water or a fruit squash drink) should be encouraged. Children are less irritable at induction, and there may be a reduction in PONV.

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Table 36.11 Guidelines for preoperative fasting periods

Ingested material	Minimum fast (h)	
	Infants <1y	Children >1y
Clear liquids	1	1
Breast milk	4	4
Formula milk	6	6
Light meal and all milk	6	6

Source: data from Thomas M, *et al*. (2018). Consensus statement on clear fluids fasting for elective pediatric general anesthesia. *Pediatr Anesth*, **28**, 411–14; however, there may be unit variability.

- Contraindications to reduced fasting times include gastro-oesophageal reflux disease, oesophageal stricture, achalasia, gastroparesis and severe cerebral palsy.
- The data for milk and solid food are less clear. Breast milk is cleared from the stomach more rapidly than formula milk in infants.
- Every unit should have fasting guidelines. Close liaison with ward staff ensures that children receive adequate clear fluid preoperatively and that milk feeds for neonates and infants are appropriately timed.

Topical anaesthetics

Topical LA preparations reduce the pain of venepuncture and facilitate IV induction.

- EMLA® cream is a eutectic mixture of 2.5% lidocaine and 2.5% prilocaine in a 1:1 ratio. It should be applied for at least 45min and can produce vasoconstriction. The duration of action is 30–60min. EMLA® should be avoided in premature infants <37w and used with caution in children <1y receiving medication that may predispose to methaemoglobinaemia.
- Ametop® is a 4% gel formulation of tetracaine (amethocaine). The onset time is 30min for venesection and 45min for cannulation. There is a prolonged duration of action (4–6h) after the gel has been removed. It is licensed from 4w of age and has vasodilating properties. There may be a higher incidence of allergic reactions. The gel should be applied for no longer than 90min and removed earlier if a rash or itchiness develops.
- LMX 4® is topical 4% lidocaine and should be applied at least 30min before procedure. It should not be left on the skin >60min in a 1–2mo old, 4h if 3–11mo and 5h if 1–17y.
- It is important to identify the veins to be anaesthetised and not blindly apply the LA preparation to the dorsum of each hand. Keep the area bandaged to prevent removal or licking of the preparation!
- Ethyl chloride is a cryoanalgesic. It is useful when topical LA preparations are either contraindicated or forgotten.

Premedication

(See ➲ Premedicants, pp. 66–8; ➲ Sedation, p. 419.)

Routine sedative premedication is not necessary ('parents are often the best premedication').

- Sedative premedication is used to reduce anxiety and facilitate compliance at induction (Table 36.12).[15]
- It may also reduce PONV and postoperative delirium. Postoperative amnesia with midazolam may reduce postoperative behavioural changes, including nightmares, bedwetting and eating disorders, especially in the preschool child.
- Recovery time may be prolonged.
- Indications for premedication include excessive anxiety or non-compliance, previous distress, learning disability and behavioural issues.
- Contraindications include an anticipated difficult airway, OSA, reduced conscious level, raised ICP and ↑ risk of aspiration.
- Infants have not yet developed a fear of strangers and appear relatively undisturbed when separated from their mothers. The preschool child is vulnerable to separation anxiety in a strange environment, but without the ability to reason. Children from 3 to 6y may require a simple explanation and children from 6 to 12y will need a more detailed explanation and a sense of control. Older children or adolescents may request premedication.
- PO midazolam is commonly used. It acts within 20–30min to reduce anxiety, leading to a more cooperative child, but with minimal delay in recovery. The IV formulation is often used but is extremely bitter and should be diluted in fruit juice or paracetamol syrup. The newer preparation of buccal midazolam (Buccolam®) is tasteless and requires little cooperation from the child. It does not affect fasting and has a quicker onset of 10–15min. Midazolam can also be given intranasally where it has a rapid onset of action within 5–15min but is poorly tolerated because of the burning sensation in the nasal mucosa. Use a mucosal atomiser device and divide the dose between each nostril.
- Ketamine causes excessive salivation and postoperative emergence delirium (although these may be less frequent in children). It should be given PO in combination with midazolam or IM as a last resort (use the 50mg/mL preparation). The latter option would require careful discussion with the parents.
- Clonidine is tasteless. It may reduce the postoperative analgesic requirement. It has a slow onset and recovery time and may cause bradycardia and hypotension; there is no amnesic effect.
- Dexmedetomidine may be useful intranasally as a 3rd-line drug if other options have failed.
- An antisialogogue may be required for children with excessive secretions, e.g. Down's syndrome and cerebral palsy, and for the suspected difficult airway in younger children and co-administration with ketamine. Absorption of oral atropine is variable. To be certain of efficacy, administer atropine 10 micrograms/kg IM 30min preoperatively or glycopyrronium bromide (5 micrograms/kg) IM or IV.

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Table 36.12 Sedative premedication

Drug	Dose	Route	Onset	Duration
1st line				
Midazolam	0.5mg/kg Max: 20mg	PO	20–30min	1–2h
	0.2mg/kg Max: 10mg	Buccal	10–15min	1–2h
	0.3mg/kg Max: 10mg	Nasal	10–15min	1–2h
2nd line				
Midazolam + ketamine	0.5mg/kg + 3mg/kg	PO	20min	1–3h
Clonidine	4micrograms/kg	PO	45–60min	6h
Midazolam + clonidine	0.5mg/kg + 2 micrograms/kg	PO	30min	6h
3rd line				
Ketamine	5–10mg/kg	PO	20–30min	3–4h
	3mg/kg	IM	5–10min	3–4h
Dexmedetomidine	2–4micrograms/kg Max: 200 micrograms	Nasal	45min	90min

Parents in the anaesthetic room

In the UK, a parent is routinely allowed into the anaesthetic room, while their child is anaesthetised. Enforced separation disempowers the parent and is an emotionally traumatic experience for both parent and child.

- Parents are naturally anxious over the loss of control, a strange environment and the possibility of adverse events. Unfortunately, this parental anxiety may communicate itself to the child.
- Parental presence should not be compulsory. It is not always beneficial and may even be counterproductive with a very anxious parent. Evidence of benefit has only been demonstrated for children older than 4y with a calm parent attending the induction.
- Preschool children are especially at risk of behavioural disturbance, probably because of difficulties in reasoning. In contrast, some adolescents may not wish their parents to accompany them.
- Anaesthetic induction appears to be the most distressing event experienced by parents. Separation from the child after induction, watching the child become unconscious and the degree of stress experienced by the child before induction are all important factors.
- The parent should always be accompanied by a nurse or play therapist who can support and escort them out of the theatre suite after induction of anaesthesia. It is less common to allow both parents into the anaesthetic room; be aware of the space constraints.

Induction of anaesthesia

(See also ➔ pp. 406–9.)

- Induction should occur in a child-friendly environment. A dedicated paediatric theatre is not always an option. In these circumstances, a customised paediatric anaesthetic trolley incorporating a comprehensive range of airway and vascular equipment is important.
- Prepare drugs and equipment before the child arrives. Recheck the weight (Table 36.4).
- Online calculators or smartphone apps can be useful to check doses.
- Precalculate the dose (and volume) of atropine and suxamethonium in prepared syringes, as it may be given by your assistant in an emergency (see Table 36.15). A 10mL syringe of propofol is useful as a 1st-line treatment for laryngospasm.
- SpO_2 is the minimum monitoring acceptable in the anaesthetic room, although it will not read accurately on the agitated child. Many children will tolerate an ECG and a BP cuff prior to induction.

Inhalational induction

(See also ➔ pp. 391–2.)

- It is important to learn more than one method. Not all children are susceptible to the same technique.
- Sevoflurane is rapidly acting, enabling a smooth induction. It is not odourless but is relatively non-irritant. For the suspected difficult airway, use sevoflurane in 100% O_2; otherwise 50% N_2O/O_2 is satisfactory, and anecdotally N_2O may obtund the child's sense of smell, facilitating induction.
- Emergence delirium has a reported incidence of 20% with sevoflurane. There is a strong association with ENT surgery, preschool ♂, parental and patient anxiety, rapid awakening and inadequate analgesia. A single dose of propofol 1mg/kg at the end of surgery may be of benefit.
- Involve the parent as much as possible. This may involve holding the child or even helping with the induction.
- Position the child either supine on the trolley or across the lap of the parent, so that the parent or anaesthetic assistant can gently restrain the arms, if necessary. Warn the parent that the child's head will become floppy and need support.
- For smaller children, a cupped hand method is useful. Occlude the end of the bag to direct all the FGF towards the patient's mouth and nose.
- A face mask is often tolerated by older children. This can be held by the parent, child or anaesthetist, and the child can be encouraged to blow up the bag 'like a balloon'. A flavoured face mask may be useful initially, but the volatile agent rapidly becomes the dominant smell.
- The parent should be warned of abnormal movements when the child is nearly anaesthetised.
- Once anaesthesia is achieved and the eyelash reflex is absent, anaesthesia can be maintained with another volatile agent, if desired.

Intravenous induction

- The smaller child sits across the parent's lap, and the arm is placed under the parent's axilla, thereby obstructing the child's view. The older child will usually lie on the trolley, with the parent on one side holding the child's hand, while the other is cannulated.
- The induction agent of choice is propofol 3–5mg/kg, with 1% lidocaine (1mL/10mL propofol) added to reduce pain on injection.

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Propofol is licensed for children over 1mo.

- If using a small vein, the dilution of propofol with an equal volume of 0.9% sodium chloride significantly reduces pain on injection.
- The Paedfusor TCI system is approved for >1mo or >5kg.[16] The dose is age-dependent and proportionally greater than the adult dose due to the higher volume of distribution and clearance in children.
- Thiopental 4–6mg/kg is a suitable alternative and is licensed for neonates (2mg/kg).
- Ketamine 2mg/kg is useful for haemodynamically compromised patients, usually in conjunction with fentanyl 1–2 micrograms/kg. Emergence phenomena are less common in children, especially in combination with midazolam, but the incidence of PONV and salivation is higher.

Comparison of intravenous and inhalational induction

- IV induction is simple and safer but is associated with more hypoxia, possibly because children are rarely preoxygenated.
- Inhalational induction produces more coughing and laryngospasm.
- In practice, it seems prudent to opt for IV induction, if possible, unless the child actively chooses an inhalational method.

Tips for cannulation

Securing IV access can be difficult, even for paediatric anaesthetists! It is important to realise this; relax and send for help, if necessary. Good lighting, patience, competent anaesthetic assistance and a selection of cannulae with prepared 0.9% sodium chloride flush syringes are all essential.

- Neonates often have surprisingly good superficial veins on the hand and wrist. Conversely, healthy children between 3mo and 2y can be notoriously difficult because of the fat pads over the hands and feet. Filling a knotted disposable surgical glove with warm water and placing over the dorsum of the hand may be helpful.
- Compression of a limb by the assistant should be gentle to act as a venous, rather than an arterial, tourniquet. The skin is often mobile and should be gently stretched. In neonates, it may be easier for the anaesthetist to flex and squeeze the wrist with their non-cannulating hand.
- Examine the wrists and dorsum of the feet for superficial veins. Scalp veins are possible in neonates. Long saphenous and cephalic veins may be palpated.
- In some children, most commonly in the feet, the skin is surprisingly tough and a small nick in the skin with a 21G needle may be necessary. Loosening the cap of the cannula or priming with 0.9% sodium chloride will enhance flashback of blood in small veins.
- Transfixion is possible in smaller children. It is potentially useful for all veins, but especially in 'blind' long saphenous and femoral vein cannulation. Slowly pull back the cannula until in the vein, and then gently advance.
- If cannulating the femoral vein, a small support under the pelvis and slight external rotation may be useful.
- A handheld near-infrared device (e.g. AccuVein®) can be very useful for identifying veins on the dorsum of the hands and feet that can be neither visualised nor palpated.

- Ultrasound is useful for difficult access. A small 'footprint' probe of 7–10MHz is suitable for most ages. It is particularly useful for central venous and peripheral arterial access, but veins in the upper arm and the long saphenous vein can also be visualised.[16]
- If all else fails, intraosseous access can be an invaluable alternative. Observing aseptic precautions, prepare an area of the skin over the anteromedial aspect of the tibia, 1cm below and medial to the tibial tuberosity. The IO needle is inserted perpendicularly to the skin and advanced in a twisting, pushing movement against the bone, until there is a sudden loss of resistance. The position is confirmed if the needle remains upright without support and marrow can be aspirated, and fluid can be administered without SC swelling around the entry site. Children can be successfully anaesthetised via this route and the IO route is particularly useful in fluid resuscitation of the shocked child before definitive IV access can be gained. Routine blood samples, including X-match, can be taken from this site before induction. Battery-powered devices are available (EZ-IO®).
- Surgical cut-down is rarely needed and often technically difficult, and should be reserved as a last resort.

Airway management

Airway complications, including coughing, laryngospasm and upper airway obstruction, are more common in children.

- Use **chin lift** and **jaw thrust** to maintain a clear airway.
- Hyperextension of the neck in the neonate often occludes the airway, and a neutral position is usually more successful. A small shoulder roll (towel or gauze roll) may be beneficial to achieve this. For older children, the adult 'sniffing the morning air' position should be adopted.
- Smaller children do not require a pillow; this may lead to unwanted head flexion.
- The paediatric face mask should be accurately sized and held gently, but firmly, on the face with the thumb and forefinger. The other fingers should curl around and grip the mandible. It is important to avoid pressing on the floor of the mouth, which will push the tongue forward and obstruct the airway.
- Early use of an OPA may be useful in older children, especially if the child has grossly enlarged tonsils. Caution in a lightly anaesthetised child, as this risks laryngospasm.
- A nasopharyngeal airway may be helpful. This should be well lubricated. It is indicated in cases of micrognathia and can be inserted at lighter levels of anaesthesia.
- The most important technique in the management of the airway is judicious use of CPAP. Ensure a good seal with the face mask, and then partially occlude the bag of the Ayre's T-piece.

Laryngospasm

(See also ➔ pp. 370–1.)

Laryngospasm is more common in children. Additional risk factors include inhalational induction, asthma, URTI and chronic lung disease. Children become cyanotic more rapidly than adults because of ↑ metabolic rate/O_2 consumption and reduced FRC.

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- Bradycardia is a premorbid event, indicating an inadequate CO and a significant risk of cerebral hypoxia.
- Partial laryngospasm management:
  - 100% O_2
  - CPAP
  - Gentle assisted ventilation
  - Propofol incremental 0.5mg/kg boluses.
- Complete laryngospasm management:
  - 100% O_2
  - CPAP
  - Assisted ventilation may exacerbate the condition by inflating the stomach and forcing the arytenoids and false cords against the true vocal cords
  - Early administration of suxamethonium:
    1–2mg/kg IV
    4mg/kg IM to thigh with massage
    3mg/kg submental (extraoral approach), intralingual with massage is described, but not preferred due to less familiarity with this route.
  - Atropine 10–20 micrograms/kg may be necessary. Precalculate these doses and volumes.

Intubation

(For ETT size, see ➔ p. 910.)

- Neonatal intubation is not normally difficult, only different. The neonate has:
  - Proportionately larger head, shorter neck, larger tongue and smaller mandible
  - Larynx that is more anterior/superior (C3–C4, compared with C5–C6)
  - Epiglottis that is large, floppy, U-shaped and with obliquely angled vocal cords.
- In the absence of recognised medical conditions with associated airway complications, paediatric intubation is usually straightforward.
- Below 6mo of age, a straight-bladed laryngoscope can improve the view of the glottis. The head should be in a neutral position, and the shoulders supported if necessary. Advance the straight blade past the larynx, then withdraw slowly until the larynx becomes visible, i.e. the blade is posterior to the epiglottis. (The blade is used to lift the large, floppy, U-shaped epiglottis out of the way.) Gentle cricoid pressure is often helpful. If nasal intubation is required, use a laryngoscope blade with minimal guttering to allow more room for instrumentation in the oropharynx. Over 6mo of age, a curved blade is usually easier. Intubation can be performed with the blade resting in the vallecula, as in adults.
- Most intubated neonates will also require an NGT (8–10Fr).
- Always have a range of ETTs available, including a half size above and below the original estimation.
- Complications are common. Oesophageal and endobronchial intubation, extubation, kinking of the tube and disconnection should all be anticipated. Secretions are far more likely to cause obstruction because of the smaller tube sizes involved, and periodic suction may be necessary. **Confirm the length of the tube by auscultation before securing.**

- Intubation increases the work of breathing. The reduction in the cross-sectional area of the neonatal trachea with a size 3.5 tube *in situ* increases airway resistance by a factor of 16. Most intubated infants should undergo controlled ventilation as part of the anaesthetic technique.

Tube fixation

Tube fixation is crucial. The neonatal trachea is only 4cm in length. Inadvertent extubation and endobronchial intubation are common.

- Secure with a '**three-point fixation**' to prevent movement of the tube in all three planes.
- Two pieces of trouser-shaped Elastoplast® may be used, with one 'leg' across the upper lip while the other 'leg' is wrapped around the tube. An OPA helps splint the tube.
- There are numerous other methods of fixation, all equally valid. The tube should be secured to the maxilla, rather than to the more mobile mandible.

Difficult intubation

(See also ➔ p. 367; ➔ Unanticipated difficult airway in adults, pp. 368–72.)

The key to difficult intubation is to identify the at-risk patient and plan accordingly with appropriate assistance and equipment.

- Some conditions are well known to be associated with airway problems (e.g. Pierre–Robin, Treacher Collins and Goldenhar syndromes). Other patients can be identified by assessment of the airway preoperatively, specifically the presence of micrognathia and retrognathia.[18]
- Optional premedication with atropine 20 micrograms/kg IM or glycopyrronium 5 micrograms/kg IM 30min preoperatively to dry secretions.
- The traditional method is deep inhalational anaesthesia with CPAP and IV access. Laryngoscopy and intubation are attempted with the patient breathing spontaneously.
- Halothane is now rarely available, and sevoflurane is the agent of choice. A propofol infusion can be used to supplement this technique.
- HFNO can double apnoea time for desaturation <90% in well children.[19]
- A blind nasal approach to intubation is possible, but experience in the technique is declining and there is a risk of trauma.
- An SGA will often secure the airway adequately, without the need for intubation.
- If intubation is still necessary, it may be possible to pass a bougie through an SGA into the trachea and then railroad an ETT. A size 3 iLMA is available and may be suitable for a larger child. An FOB can also be used via the LMA.
- Videolaryngoscopy allows a magnified, high-resolution view of the airway, with visual confirmation of intubation. The blade is usually inserted in the midline without a tongue sweep. Most are now available in a range of neonatal and paediatric sizes.[20]
- FOB intubation is rarely necessary. Children need to be anaesthetised, but a propofol infusion is an alternative method to volatile anaesthesia. Smaller-size neonatal and paediatric bronchoscopes do not all have a suction channel and should be checked to confirm that the selected ETT will fit over them.

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- Conventional ETTs may be difficult to railroad, and armoured tubes should be considered. Alternatively, a guide-wire can be inserted into the trachea using the suction channel. An exchange catheter is passed over the wire, and then the ETT railroaded over the exchange catheter.
- A tracheostomy is rarely required. It is exceedingly difficult as an emergency procedure. Paediatric percutaneous cricothyroidotomy cannulae are available in 18G and 16G sizes and should be available in the anaesthetic room.
- Paediatric DAS guidelines for the difficult airway should be readily available.[21]

Rapid sequence induction

(See also ➲ pp. 388–90.)

- Ranitidine and metoclopramide are not routinely prescribed.
- Preoxygenation does not usually present problems with infants and older children but may be more difficult in preschool children.
- Inhalational induction may be necessary, after which cricoid force can be applied while breathing spontaneously.
- Suxamethonium should be preceded by atropine to prevent bradycardia. Rocuronium 1.2mg/kg is a suitable alternative.
- Cricoid force may reduce aspiration and stomach inflation but, especially if excessive, can impede face mask ventilation, laryngoscopy and passage of the ETT. It should be reduced if difficulties with ventilation or laryngoscopy are encountered.
- There should be a low threshold for using an NGT in neonates and small infants. If already *in situ* in the neonate, it should remain in place, rather than being removed. There is no consensus for older children.
- The child should be extubated awake in the left lateral position.

Maintenance

- Position the infant and smaller child with both arms raised at the level of the head. Exposure of the hand allows assessment of the pulse, peripheral temperature, colour and capillary refill. A blocked IV may be more easily cleared and a new cannula may be easier to site.
- The pulse oximeter probe should be sited on the same arm as the IV infusion, and the contralateral arm to the BP cuff. Avoid oximeter probes on the feet, as the trace is often lost once abdominal surgery commences.
- Check that the ETT is still *in situ* and securely fixed, and that the lungs are being ventilated adequately and equally.
- The connections should all be secure, and the breathing system supported, if necessary.
- Confirm that cannulae are secure, working and accessible; extension tubing may be necessary. Three-way taps allow the administration of drugs and fluid volume when necessary. Neonatal surgery requires a minimum of two cannulae (maintenance and volume).
- Blood sugar should be checked regularly.
- Even small air bubbles in IV fluids can be potentially harmful to infants, especially in the presence of an ASD or VSD.

- Theatre temperature should be about 21°C and preheated. The child's head should be covered, and body exposure reduced to a minimum. It is easier to prevent hypothermia than to treat it. Both IV fluids and surgical wash should be warmed.
- Minimum monitoring should include ECG, BP (with appropriately sized cuff), SpO_2, capnography, temperature, inspired and expired anaesthetic agent (if used) and gas monitoring, airway pressure and peripheral nerve stimulation (if an NMBA has been used). The ventilator alarm should be set to appropriate values.
- The width of the BP cuff should be 20% greater than the diameter of the arm to avoid artefactually raised BP.
- Electronic monitoring is often unreliable with the sick or shocked neonate. It should support, but not replace, clinical observation.
- **Do not** let surgery start until you are ready and the preoperative checks have been completed.
- Anaesthetic complications in paediatric practice are as common during maintenance as at induction or in the postoperative period.

Reversal

Following surgery, the child should be warm, well oxygenated, normocarbic and pain-free. A cold, acidotic neonate will not breathe postoperatively.

- SGAs can be removed, either deep or awake. If an armoured LMA is *in situ*, a bite block will be needed. There is often a stage shortly before waking when the mouth opens slightly to mimic a small yawn; this is an ideal opportunity to deftly remove the LMA.
- Most children should be extubated awake. If warm and with adequate analgesia, this is tolerated well. Exceptions include tonsillectomy and other procedures when coughing is to be avoided. In these cases, deep extubation may be preferable.
- Neonates should be extubated awake, preceded by an assisted ventilation to preoxygenate the lungs.

Postoperative nausea and vomiting

(See also ➔ pp. 442–6.)

PONV is unusual under the age of 3y and more common in post-pubertal girls. Predictors include type of procedure (adenotonsillectomy, squint surgery), duration of surgery, travel sickness and previous PONV.

- Opioids increase the risk of PONV. Regional anaesthesia and other opioid-sparing techniques should be encouraged.
- There is some evidence of reduced PONV with TIVA in children. N_2O does not appear to be associated with an ↑ risk of PONV in children.
- In children at high risk of PONV, consider combination therapy of IV ondansetron 150 micrograms/kg up to 4mg IV and dexamethasone 150 micrograms/kg up to 8mg.[22]
- Cyclizine is no longer recommended for reducing PONV in children.

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Postoperative pain relief

Children feel pain as much as adults. Underdosage is common due to inadequate knowledge and fear of side effects. Poor-quality pain relief can lead to prolonged hospital stay, maladaptive behaviour (temper tantrums, bed-wetting and nightmares) and chronic pain issues.

- Similar principles to adult practice apply (see pp. 1152–3), including the application of multimodal analgesia and specialised pain charts.[23]
- Pain assessment can be challenging with infants and neonates. Use physiological/behavioural pain scales, e.g. the Children's Revised Impact of Event Scale (CRIES) is used for infants ≥38w of gestation. Characteristics of crying, O_2 requirement, ↑ vital signs, facial expression and sleep state are scored. A maximal score of 10 is possible. If the CRIES score is >4, further pain assessment should be undertaken. Analgesic administration is indicated for a score of ≥6.
- The FLACC Scale is used for children who are unable to communicate their pain (ages 2–7). Each of the five categories (**f**ace, **l**egs, **a**ctivity, **c**onsolability and **c**ry) is scored from 0 to 2. The total score will lie in the range of 0–10, where 0 represents no pain and 7–10 represents severe discomfort/pain.
- Older children may be able to self-report using faces charts, e.g. Wong–Baker or visual analogue scales.
- Use non-pharmacological methods such as explanation, reassurance and distraction (stories, play, music).
- Paracetamol and NSAIDs (Table 36.13) are widely prescribed for minor cases/day surgery and for their opioid-sparing effects. Drugs should be given regularly. Single doses of IV perioperative analgesics should be documented on the ward drug chart to avoid duplicate doses being given postoperatively.
- Codeine is no longer recommended for children under 12y.
- Opioids: morphine infusions can be administered cautiously to neonates, and as nurse-controlled analgesia (NCA) for smaller children with a background infusion. PCA can be used effectively in children as young as 6y, some with a low background infusion. Bolus function must not be activated by parents (Table 36.14).
- Caudal analgesia and PNBs are extremely useful for day cases.
- Epidural blockade is of proven benefit in abdominal and orthopaedic surgery. Below 6mo, it is technically easier, and possibly safer, to insert the catheter via the caudal route (see pp. 929–30).
- Ultrasound-guided blocks are common in children using the same principles and landmarks as adults.
- Nerve infusion catheters and wound catheters are gaining popularity.
- Liaison with ward staff is crucial, and a standardised multidisciplinary team approach, preferably with an acute pain service, is ideal.
- Drugs doses may need to be modified in obese or underweight patients.

Regional anaesthesia

Successful regional blockade provides conditions for light and haemodynamically stable GA. The stress response is attenuated and early, pain-free emergence is possible, leading to a smooth postoperative recovery.

- Few children tolerate these techniques awake, and the majority of regional blocks are performed on anaesthetised patients.
- Motor blockade is unnecessary and low concentrations of LA can be used. The most widely used solutions are 0.25% bupivacaine/levobupivacaine and 0.2% ropivacaine.

Table 36.13 An example of analgesia prescribing guidance in children

Mild to moderate pain			
Paracetamol			
Neonates and infants <10kg	10mg/kg	IV	8-hourly
	15mg/kg	PO	6-hourly
10–50kg	15mg/kg	PO/IV	6-hourly
>50kg	1g	PO/IV	6-hourly
Ibuprofen[*]			
1–3mo	5mg/kg	PO	8-hourly
>3mo	10mg/kg	PO	6-hourly
Diclofenac[*]			
>6mo	1mg/kg	PO/PR	8-hourly
Ketorolac[*]			
	0.5mg/kg	IV	6-hourly
Morphine			
	100micrograms/kg	PO	6-hourly
Moderate to severe pain			
Morphine			
Neonate to 3mo	100micrograms/kg	PO	3–4 hourly
3mo to 1y	200micrograms/kg	PO	3–4 hourly
>1y	300micrograms/kg	PO	3–4 hourly

[*] NSAIDs: caution with renal impairment, asthma and low platelets. If possible, administer with food.

[**] Note that there may be slight variation in guidance between different institutions.

Do not exceed adult doses.

Source: data from Mason DG *et al*. Guidance for prescribing pain relief in children. Paediatrics at the Nuffield Department of Anaesthetists (P@NDA), Oxford University Hospitals NHS Trust, 2019.

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Table 36.14 Typical IV morphine options for severe postoperative pain in children

Morphine	50–100 micrograms/kg IV incremental boluses
Morphine infusion	Morphine 1mg/kg in 50mL of 0.9% sodium chloride, i.e. 20 micrograms/kg/mL Rate: 1–2mL/h (20–40 micrograms/kg/h)
Morphine NCA	Morphine 1mg/kg in 50mL of 0.9% sodium chloride, i.e. 20 micrograms/kg/mL Rate: 0.5–1mL/h. Bolus: 1mL. Lockout: 15–20min
Morphine PCA	Morphine 1mg/kg in 50mL of 0.9% sodium chloride, i.e. 20 micrograms/kg/mL Rate: 0–1mL/h. Bolus: 1mL. Lockout: 5min

Caudal block

Caudal extradural analgesia has a wide application in children. It is suitable for all surgery below the umbilicus, including general surgery, urology and orthopaedics. The technique is easier than with adults, with a higher success rate of ~95%. Epidural fat is less dense and less tightly packed in children, with the result that LA can spread more easily and quickly. Therefore, caudals can achieve a higher dermatomal block.[24]

Technique

- Position the patient in the left lateral position, with the legs flexed at the hip. Aseptic technique is a prerequisite.
- Identify the sacral hiatus as the apex of an equilateral triangle with the base formed by a line joining the posterior superior iliac spines (Fig. 36.1).
- Alternatively, with the hips flexed at 90°, a line extended along the midline of the right femur will intersect with the sacral hiatus. The natal cleft does not always correspond to bony midline structures.
- Define the boundaries of the sacral hiatus. This is again a triangle with the base formed by a line joining the sacral cornua and the apex representing the lower part of the 4th sacral vertebra. The sacral hiatus is covered by the sacrococcygeal membrane.
- Direct a short-bevelled 22G or 20G cannula at 60° to the skin from the midpoint of the line joining the sacral cornua. A small 'give' indicates penetration of the sacrococcygeal membrane. Flatten the cannula or needle slightly, then advance. If using a cannula, withdraw the stylet to just behind the cannula before advancing the cannula into the caudal space. Do not advance the needle or cannula any more than is necessary. Advancement of a cannula, rather than a needle, may reduce the incidence of inadvertent dural or vascular puncture. Easy progression of the cannula is a good prognostic indicator of success.
- Draw up your medications now—this ensures patience to observe any blood or CSF flow passively up the cannula.
- Use a double-aspiration technique; if the cannula is in a small vein, the vein may simply collapse with the 1st aspiration. Aspiration should be repeated during injection of the LA. The commonest reason for a failed attempt is positioning the needle too caudally.
- Ultrasound can be used to assess the caudal anatomy and to confirm the spread of the injectate in the caudal extradural space.[25]
- Levobupivacaine 0.25% is commonly administered. Current guidelines recommend that doses should not exceed 2.5mg/kg for caudal bupivacaine, and the recommended volumes are 0.5mL/kg for blockade of sacral dermatomes, 1.0mL/kg for lumbar dermatomes or 1.25mL/kg for lower thoracic dermatomes.[24] Duration of the block averages 4–8h.
- Caudal blockade can be extended with clonidine 1–2 micrograms/kg but can cause intraoperative hypotension and postoperative sedation.
- Adrenaline has been implicated in cases of spinal ischaemia and should be avoided.
- Morphine and diamorphine increase the incidence of urinary retention and should be reserved for surgery in which catheterisation is required.
- Ketamine is no longer recommended, especially in younger children, because of the potential risk of neuronal apoptosis.

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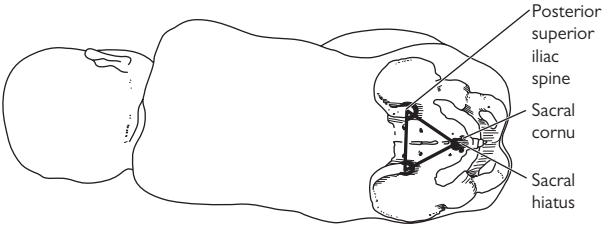

Fig. 36.1 Anatomy for caudal block.

Advantages/complications of caudal analgesia

- Simple, safe and successful, with a wide range of indications.
- Motor block, paraesthesiae, hypotension, urinary retention, inadvertent dural puncture and intravascular injection can all occur. All these complications are rare using a single-shot caudal technique.

Continuous caudal epidural analgesia

Caudal injection is restricted in its duration of action. A catheter can be introduced into the epidural space via the caudal route. It is a safe and effective method of administering epidural analgesia in infants. The single curve of the back allows the catheter to thread into the epidural space; the tip of the catheter should be close to the level of the dermatomes that need to be blocked.

- Over 2y of age, the development of a lumbosacral curvature tends to lead to a higher failure rate. However, some authors claim comparable success rates.
- Because of the proximity of the perineum, a caudal catheter should not be left *in situ* for longer than 36h.

Epidural/subarachnoid block

(See also ➔ pp. 1114–17.)

Epidural block

(See also ➔ pp. 1164–6.)

Epidural blockade is technically more difficult in children and requires experience. The ligamentum flavum is less well developed, and the intervertebral spaces are narrower. In infants, the epidural space is rarely located at a depth >15mm and often as superficially as 10mm from the skin. The technique is similar to that used in adults. Either a midline or a paramedian approach is acceptable. The NAP3 study demonstrated that paediatric epidurals resulted in fewer complications than adults.[26] Severe neurological complications, including fatalities, have been reported in association with using air to find the epidural space in neonates. The caudal route may represent a safer alternative with this group.

- Epidural needle: 18G for infants/children, 19G for neonates/infants (catheter 'end-hole' only).
- For suitable doses, see Table 36.15.

Subarachnoid block

Paediatric spinal anaesthesia can be useful for herniotomy in neonates. The procedure requires training specific to paediatrics and an experienced assistant.

- The infant needs to be firmly gripped in the lateral or sitting position. The needle should be directed at right angles to the skin in the midline below L3, with L5–S1 reported as the safest approach. Prior infiltration of LA into the skin will help prevent patient movement.
- The block has a rapid onset, but duration is rarely >40min. If sedation is required during the surgery, the incidence of postoperative apnoea is comparable with a GA technique.
- Spinal needle: 5cm, 22G.
- For suitable doses, see Table 36.15.

Table 36.15 Regional analgesia doses in children

Caudal extradural blockade	Sacral: 0.5mL/kg of 0.25% bupivacaine Lumbar: 1mL/kg of 0.25% bupivacaine Thoracolumbar: 1.25mL/kg (0.25% bupivacaine diluted with 0.9% sodium chloride)
Supplements to extend duration of caudal	Clonidine 1–2 micrograms/kg
Lumbar epidural (intraoperative)	0.75mL/kg of 0.25% bupivacaine
Thoracic epidural (intraoperative)	0.5mL/kg of 0.25% bupivacaine
Epidural infusion	0.1% bupivacaine + fentanyl 2 micrograms/mL (sterile premixed bag) Rate: 0.1–0.4mL/kg/h
Spinal block	0.1mL/kg of 0.5% 'heavy' bupivacaine + 0.06mL for needle dead space
Nerve infusion catheters	0.1–0.4mL/kg/h
Wound catheters	0.1–0.4mL/kg/h
Wound infiltration	1mL/kg of 0.25% bupivacaine

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Peripheral nerve blocks

PNBs are covered in depth in ➲ Chapter 42. Only additional paediatric-specific information is covered here.[27] Refer to ➲ Chapter 42 for advice on safety and performance of regional nerve blockade. Be advised that paediatric LA doses are quoted as mL/kg of a specific concentration of a specific LA, as the volume is important. Do not confuse with maximum doses which still apply and must be calculated to ensure they are not exceeded (see ➲ p. 1102).

Ilioinguinal and iliohypogastric nerve block

(See ➲ pp. 1131–2.) Useful alternative to caudal blockade in herniotomy, hydrocele and orchidopexy. It does not block pain from traction of the spermatic cord or visceral peritoneum and is unsuitable for a high orchidopexy. There is a 10% incidence of femoral nerve block. Should be avoided for neonatal herniotomy, as the LA may obscure the operating field. The block can be performed easily under direct vision by the surgeon. Of note, the entry point is 1cm medial to the ASIS, not 2cm as in adults.

- Dose: 1mL/kg of 0.25% bupivacaine (retain 1–2mL for an SC fan injection laterally, medially and inferiorly).

Dorsal nerve block of penis

(See ➲ p. 1132.) This block is indicated for distal surgery to the penis, including circumcision, meatoplasty and simple hypospadias repair.

- Also consider ring block at base of penis with 25G or 27G needle, as penile block does not always block the ventral surface of the penis.
- Dosage: 0.5mL/kg of 0.5% bupivacaine.

Transversus abdominis plane block

(See ➲ p. 1133.) Indicated for lower abdominal surgery, including herniotomy, appendicectomy and some laparoscopic surgery.

- Dosage: 1mL/kg of 0.25% bupivacaine.

Rectus sheath block

(See ➲ pp. 1133–4.) Indicated for midline hernias and single-port laparoscopic surgery.

- Bilateral block with total dose: 1mL/kg of 0.25% bupivacaine.

Femoral nerve block

(See ➲ p. 1136.) This block is indicated for surgery to the knee or femur.

- Dose: 0.5mL/kg of 0.5% bupivacaine.

Sciatic nerve block

(See ➲ pp. 1138–40.) This block is indicated for ankle surgery or lower limb procedures, in combination with a femoral nerve block.

- Dosage: 0.5mL/kg of 0.5% bupivacaine.

Axillary block

(See ➲ p. 1122.) This block is indicated for hand and lower arm surgery.

- Dosage: 0.5mL/kg of 0.5% bupivacaine.

Down's syndrome/trisomy 21

Down's syndrome is the commonest congenital abnormality (1.6 per 1000 deliveries). It is associated with a higher morbidity and mortality and characteristic dysmorphic features, including:

- Impaired global development
- Congenital cardiac defects (40%; predominantly endocardial cushion defects/VSD)
- Eisenmenger's syndrome (especially if there is associated OSA)
- Recurrent respiratory tract infection (relative immune deficiency and a degree of upper airway obstruction from tonsillar/adenoidal hypertrophy)
- Atlantoaxial instability (30%, but frequently asymptomatic; routine X-ray not indicated; avoid excessive neck movement)
- Epilepsy (10%)
- Obesity and potentially difficult venous access
- Hypothyroidism (40%)
- ↑ incidence of gastro-oesophageal reflux
- Prone to hypoventilation (consider IPPV).

Assessment

- Perform careful airway assessment: relatively large tongue, crowding of mid-facial structures, high arched narrow palate, micrognathia and short, broad neck.
- Perform careful cardiorespiratory assessment (including investigation).
- Beware asymptomatic disease; optimise where possible, and have a reduced threshold for postoperative HDU/ICU.
- Hypotonia (up to 75%) may compromise the airway.
- These patients are prone to atelectasis and respiratory tract infections; consider humidified O_2 ± physiotherapy.

Preoperative

- Often uncooperative (sedative premedications are often helpful; caution if airway obstructed).
- Drying agents may be useful if hypersalivation (⚠ caution: may have exaggerated sensitivity to mydriatic/cardiac effects of atropine).

Postoperative

- Pain management may be problematic; consider regional blocks/LA. PCA possible in selected patients.
- Parents/carers often indispensable in managing postoperative agitation.

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Diaphragmatic hernia

Procedure	Repair of defect in diaphragm either by suturing to abdominal wall or with a synthetic graft
Time	1–2h
Pain	+++
Position	Supine
Blood loss	Usually minimal to moderate
Practical techniques	GA plus IPPV, arterial line

Preoperative

- 1:3000–4000 deliveries; 85% left-sided; cardiac anomalies in 20%.
- Diagnosis usually made antenatally on ultrasound. Present in respiratory distress: ↑ RR, cyanosis, with a scaphoid abdomen. CXR diagnostic.
- Overall mortality of 30% from lung hypoplasia, abnormal pulmonary vasculature and pulmonary hypertension.
- Usually already intubated and ventilated. Ventilatory support can include high-frequency oscillation (HFO) and NO.
- For high-risk cases, surgery may need to take place on the special care baby unit if conventional ventilation is not possible.
- An NGT is essential preoperatively to prevent the stomach and small bowel in the chest cavity from compressing the lung.

Perioperative

- Cautious ventilation via a face mask; avoid N_2O.
- NGT, 2 × IV cannulae, right radial arterial line (preductal sampling).
- Keep airway pressures <25cmH_2O (pulmonary hypoplasia and consequent risk of pneumothorax). Use RR to improve ventilation.
- High-dose fentanyl (25 micrograms/kg) to reduce pulmonary vasoconstriction response to surgical stress.

Postoperative

- Postoperative ventilation for at least 24h, then attempt to wean.
- Infant may deteriorate within 12h due to pulmonary hypertensive crises. Pulmonary vasculature is reduced and abnormal → exaggerated vasoconstrictive response to hypoxaemia and acidosis.
- Rarely, if minimal defect, extubate immediately.

Special considerations

- Pulmonary hypertension may be significant. Initial management includes assisted hyperventilation with 100% O_2 and fluid boluses. Other therapies include epoprostenol, sildenafil and N_2O.
- ECMO is a last resort but has been used.
- To assist weaning, a thoracic epidural may be of benefit, inserted either conventionally or via the caudal route.

Gastroschisis/exomphalos

Procedure	Replacement of abdominal contents into the abdominal cavity
Time	2h
Pain	++/+++
Position	Supine
Blood loss	Moderate
Practical techniques	GA + IPPV; NGT; arterial line

Preoperative

- Usually diagnosed *in utero*. Incidence of 1:3000–4000.
- Gastroschisis: defect in the anterior abdominal wall, usually on the right, causing herniation of abdominal contents without a covering sac; associated with LBW and thickened bowel wall due to exposure to amniotic fluid; associated with younger maternal age and lower socioeconomic status. Repair is an urgent procedure.
- Exomphalos: failure of the gut to return to the abdominal cavity during fetal development → persistent herniation through the umbilical cord which covers it. May include other abdominal organs. ↑ incidence of associated anomalies, including cardiac disease.
- Exposed abdominal contents result in large evaporative heat and water losses and predispose to infection. They should initially be covered with cling film or equivalent.

Perioperative

- May already be intubated. Otherwise intubate conventionally.
- Intraoperative analgesia: fentanyl 1.5–10 micrograms/kg or epidural if extubation within 48h is contemplated.

Postoperative

- Ventilate in the head-up position, especially if the abdomen is tense.
- Assiduous attention to fluid balance. There may be large abdominal losses of crystalloid and protein.

Special considerations

- Lines should be sited in the arms, as abdominal distension may impair venous return from the lower body.
- Consider inserting a percutaneous long line or central line for parenteral feeding—postoperative oedema makes cannulation difficult.
- Manual ventilation is useful to assess the effect of replacement of abdominal contents on lung compliance to determine the correct degree of abdominal reduction before closure.
- Complete reduction is not always possible. A silo is then created around the extra-abdominal contents to be gradually reduced on the ICU. Fluid loss and infection are major issues in these cases.

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Tracheoesophageal fistula

Procedure	Ligation of fistula plus anastomotic repair of oesophageal atresia
Time	2h
Pain	+++
Position	Left lateral for right thoracotomy
Blood loss	Moderate
Practical techniques	GA + IPPV; arterial line

Preoperative

- Incidence of 1:3500; 85% comprise oesophageal atresia with distal fistula. Majority diagnosed *in utero*; always exclude in cases of polyhydramnios.
- High incidence of prematurity (30%) and cardiac disease (25%).
- Presents with choking, cyanosis on feeding and inability to pass NGT.
- Constant risk of pulmonary aspiration. A double-lumen Replogle tube in the oesophagus allows irrigation and suction.

Perioperative

- Inhalational or IV induction. Gentle mask ventilation to minimise gastric distension via a fistula.
- Careful ETT placement. Confirm symmetrical ventilation with the tube distal to the fistula. Manual ventilation may be necessary to assess lung compliance after ligation of the fistula, to assist in repair of the oesophagus and to periodically reinflate the left lung. Surgical retraction may impede ventilation.
- Intraoperative access will be needed to pass the transanastomotic tube nasally to facilitate oesophageal repair.
- Intraoperative analgesia: fentanyl (5–10 micrograms/kg) or epidural by either the thoracic or caudal route, if early weaning is anticipated.
- The operation is performed via thoracoscopic repair or via a right thoracotomy extrapleural approach (unless right-sided aortic arch present; 5% of cases; necessitates left thoracotomy).
- Thoracoscopic compression of the ipsilateral lung makes desaturation common; direct compression of vascular structures (IVC and the right atrium) reduces venous return. CO_2 absorption → hypercarbia and acidosis which may be poorly tolerated by these neonates.[28]

Postoperative

- Majority are ventilated postoperatively, especially if oesophageal repair is under tension. Critical to secure the NGT or transanastomotic tube.

Special considerations

- The fistula is normally situated on the posterior aspect of the trachea, just proximal to the carina. An FOB can be used to confirm ETT position.

Patent ductus arteriosus

Procedure	Ligation or clipping of ductus arteriosus
Time	1h
Pain	+
Position	Left thoracotomy
Blood loss	Usually minimal. Occasionally massive if the vessel is torn
Practical techniques	IPPV, fentanyl

Preoperative

- Small premature babies: 25% of premature infants <1.5kg recovering from hyaline membrane disease have a patent ductus arteriosus. Associated with other cardiac anomalies.
- Indications are for failure of medical treatment which include ibuprofen, indometacin or paracetamol, ventilator dependence and risk of developing bronchopulmonary dysplasia.

Perioperative

- High-risk group. Operation may be undertaken on the special care baby unit.
- Patient is usually already ventilated with full monitoring.
- Adequate IV access for transfusion. Arterial monitoring.
- IPPV with O_2/air and fentanyl up to 10 micrograms/kg with a low dose of volatile agent.
- Active heat conservation.
- Keep saturations < 96% to avoid retinopathy of prematurity.
- Local infiltration for analgesia, interpleural block by surgeon or thoracic epidural if early weaning considered.

Postoperative

- Postoperative ventilation until stable, then attempt to wean.

Special considerations

- Sudden ligation of the ductus may precipitate an acute rise in systemic BP and increase the risk of intraventricular haemorrhage. The duct should be clamped gently, or alternatively the concentration of the volatile agent can be temporarily ↑.
- Older children requiring patent ductus arteriosus occlusion tend to be fit, although some present with cardiac failure. The procedure can be performed percutaneously as a day case using a coil device.

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Pyloric stenosis

Procedure	Splitting the pylorus muscle longitudinally down to the mucosa
Time	30min
Pain	+
Position	Supine
Blood loss	Minimal
Practical techniques	GA + IPPV, ? RSI

Preoperative

- Incidence of 1:350 births; more common in first-born ♂ ; 80% are ♂ ; 10% are premature.
- Present with biochemical abnormalities, notably hypochloraemic alkalosis. May be dehydrated. Operation is never urgent, and full resuscitation should occur.
- Electrolytes, particularly chloride and HCO_3^-, and pH should be within normal limits, with chloride ≥100mmol/L.

Perioperative

- No complete agreement, but there is a risk of pulmonary aspiration from gastric outflow obstruction.
- An NGT is mandatory and will be *in situ*. Aspirate, and do not remove. It does not reduce the effect of cricoid pressure and may act as an escape valve if mask ventilation increases intragastric pressure.
- IV access is usually in place. Consider RSI ± cricoid pressure if there is excessive NG loss (>2mL/kg/h).
- Fentanyl (1 microgram/kg) plus paracetamol IV. Local infiltration (up to 1mL/kg of 0.25% bupivacaine ± adrenaline) pre-incision.
- Can be performed laparoscopically, in which case a rectus sheath block may be useful.
- Extubate awake in the left lateral position.

Postoperative

- Remove the NGT at the end of the procedure unless mucosa breached.
- Give paracetamol regularly and PO morphine PRN.
- Feed within 6h, but maintain IV fluids until feeding is established.
- Apnoea alarm overnight.

Special considerations

- Resuscitate with 5% glucose/0.45% sodium chloride plus 20mmol/L potassium chloride (HCO_3^- <32mmol/L). More severe cases will require 0.9% sodium chloride. Replace NG loss with 0.9% sodium chloride.

Intussusception

Procedure	Reduction of invaginated bowel
Time	1–3h
Pain	+++
Position	Supine
Blood loss	Moderate, may be large
Practical techniques	RSI + IPPV

Preoperative

- Intussusception is the commonest cause of obstruction in infants over 2mo of age; incidence of 2:1000 births.
- Invagination of the bowel into an adjacent lower segment, usually at the terminal ileum or ileocaecal valve. Rarely caused by a polyp or Meckel's diverticulum (5% of cases).
- Presents with paroxysmal pain, blood and mucus in stool (redcurrant jelly stool), and a sausage-shaped mass in the right abdomen.
- 70% of cases are reduced by air or barium enema.
- Child may be profoundly shocked. Urgent fluid resuscitation with gastric decompression and electrolyte correction will be needed and blood may be required. Delay can result in perforated or necrotic bowel. Fluid loss may be greater than expected.

Perioperative

- RSI. Retain the NGT *in situ*.
- Fentanyl 2–5 micrograms/kg plus volatile agent.
- Two cannulae of adequate size. CVP line in severe cases.
- Routine monitoring, temperature measurement and urinary catheter.
- Prolonged intussusception with ischaemic gut requiring resection often leads to metabolic acidosis and septic shock. Admission to a paediatric ICU will be required.

Postoperative

- TAP block, wound catheter or local wound infiltration, and morphine NCA.

Special considerations

These children can be very challenging. They will have had unsuccessful barium enema in the district general hospital and air enema in the regional centre, both with sedation. They may be inadequately resuscitated. Venous access is often challenging and a significant resuscitation is often necessary.

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Herniotomy

Procedure	Excision of patent processus vaginalis
Time	30min
Pain	++
Position	Supine
Blood loss	Minimal
Practical techniques	LMA/SV or ETT/IPPV; spinal, caudal or local infiltration

Preoperative

- Majority are fit ASA 1 children. More common in boys.
- 20% of preterm babies present for surgery at ~40w post-conceptual age or when ready to leave the special care baby unit.

Perioperative

- Children: inhalational or IV induction with LMA, then caudal or ilioinguinal block and intraoperative opioids if necessary.
- Infants: intubate with controlled ventilation. With neonates, avoid ilioinguinal block, as spread of LA may obscure the surgical field. Use either caudal or postoperative infiltration.
- Paracetamol IV or diclofenac suppository.

Postoperative

- Day case: regular paracetamol and ibuprofen.
- Admit term babies <4w and ex-premature infants <60w post-conceptual age overnight for SpO_2 and apnoea alarm monitoring.

Special considerations

- No consensus on the most appropriate regional block. Caudal blockade is indicated for bilateral herniotomy repair and children <20kg. Ilioinguinal block is effective in children >5kg.
- Laparoscopic repair becoming common. Local infiltration adequate.
- The ex-premature baby may be small and O_2-dependent with chronic lung disease. Postoperative apnoea and bradycardia are documented risks associated with GA for this group. Avoid hypocarbia and hypothermia; O_2 saturations of 90–95% are acceptable. Caffeine (10mg/kg IV) given at induction reduces the risk of apnoea by 70%.
- To avoid a GA in the high-risk premature infant, a spinal technique may be used. This may be technically difficult and complicated by a bloody or dry tap. It is too short-acting for bilateral repair. Single-shot caudal is an alternative method. Supplementary sedation results in the same risk of postoperative apnoea as with GA.
- A strangulated hernia that does not reduce is an emergency and requires fluid resuscitation and an NGT. Precautions should be taken against regurgitation and aspiration.

Circumcision/hypospadias repair

Procedure	Removal of prepuce (foreskin)/restoration of urethral opening to the tip of the penis
Time	Circumcision: 20min; hypospadias: 1–3h
Pain	++
Position	Supine
Blood loss	Minimal
Practical techniques	LMA + SV/IPPV, caudal/penile block/ring block

Preoperative

- Circumcision: common day case procedure; move towards conservative management, including simple stretch or preputioplasty.
- Hypospadias is usually an isolated problem, but there may be an association with certain rare dysmorphic syndromes.
- Obtain consent for a regional block and suppository.

Perioperative

- Inhalational or IV induction.
- If procedure <1h, LMA plus SV.
- If procedure >1h, LMA or ETT plus IPPV.
- Regional block: caudal or penile block (if hypospadias is distal) (see ➲ pp. 929–30; ➲ p. 931; ➲ p. 1132).
- Paracetamol IV and diclofenac PR.

Postoperative

- Regular paracetamol and NSAIDs. Hypospadias: consider morphine NCA (not always necessary).
- Topical lidocaine gel can be applied frequently, without exceeding the toxic dose.

Special considerations

- Hypospadias repair: may be a simple procedure, e.g. meatal advancement and glanduloplasty, or extensive involving a buccal mucosa graft. Adjust anaesthetic technique accordingly.
- A regional block should be performed prior to the surgery. Avoid erection with a regional block plus an adequate depth of anaesthesia.
- There is no consensus as to the optimal strategy for pain relief. Caudal is technically easier in infants, and penile block may be more suitable in children over 10kg. Ring block is easier in boys >5kg. All methods are effective.
- Circumcision is one of the most painful day case procedures; parents should be warned and advised to apply topical gel regularly and continue paracetamol for several days.

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Orchidopexy

Procedure	Release of undescended testis into scrotum
Time	45min
Pain	++
Position	Supine
Blood loss	Minimal
Practical techniques	SV, LMA + regional block

Preoperative

- Boys, usually over 2y (2% of population).
- Common day case procedure.
- Obtain consent for a suppository and regional block.

Perioperative

- Inhalational or IV induction. LMA.
- Regional technique: caudal, ilioinguinal block or local infiltration.
- Paracetamol IV or diclofenac PR.
- Give supplementary opioids, if indicated.

Postoperative

- Regular paracetamol and ibuprofen. PO morphine if necessary.

Special considerations

- Adequate analgesia is difficult if the testis is high. In this case, aim for a high-volume, low-concentration mid-thoracic caudal block. Use 1.25mL/kg volume of 0.25% bupivacaine with 0.9% sodium chloride (observing maximum bupivacaine dose) (see ➲ pp. 929–30; ➲ p. 931).
- If an ilioinguinal block is used, only the anterior part of the scrotum is anaesthetised; use local infiltration for the scrotal incision (see ➲ p. 932; ➲ pp. 1131–2).
- Testicular traction, even with a seemingly adequate blockade, may lead to intraoperative bradycardia or laryngospasm, especially with an ilioinguinal block. Surgery should be stopped, and anaesthesia deepened; supplementary opioids may be required.
- Suspected torsion of the testis is a surgical emergency, and the need for an RSI will have to be considered. Local infiltration is adequate.
- A high testis may need surgery in two stages. The 1st procedure is to identify the testis and, if possible, bring it down to the inguinal ring. This is usually performed laparoscopically and will require intubation, controlled ventilation with intraoperative opioids, IV paracetamol and diclofenac PR.

Cleft lip and palate

Procedure	Repair of defect in upper lip and palate
Time	2–3h
Pain	++
Position	Supine, head ring, shoulder support
Blood loss	Minimal for cleft lip. Moderate for cleft palate
Practical techniques	IPPV. Armoured or RAE tube

Preoperative

- Incidence of 1:300–600 births but can be 1:25 with a family history.
- Both the lip and palate are involved together in 50% of cases.
- Isolated cleft palate incidence of 1:2000 live births. ↑ incidence of congenital abnormalities.
- Associated syndromes often involve a difficult airway, e.g. Pierre–Robin, Treacher Collins and Goldenhar syndromes. Therefore, make a careful assessment of the airway.
- Discuss risks and complications.
- Administer IM atropine 20 micrograms/kg 30min preoperatively if a difficult airway is suspected.

Perioperative

- Inhalational or IV induction. If difficult airway is suspected, consider inhalational induction with CPAP if necessary. Intubate deep with the child breathing spontaneously or following muscle relaxant. Videolaryngoscopy may be necessary.
- Caution when surgeon inserts gag. A preformed RAE tube may become obstructed or kinked, especially the smaller sizes.
- Use a throat pack, and make sure the eyes are protected.
- Encourage LA infiltration to improve analgesia and ↓ blood loss.
- Fentanyl (2–4 micrograms/kg) before LA, then morphine 50–150 micrograms/kg plus paracetamol IV.
- Dexamethasone 0.25mg/kg + three postoperative doses to prevent postoperative swelling.
- Consider an infraorbital nerve block for cleft lip repair.

Postoperative

- Extubate awake. Suction the pharynx early and carefully to prevent damage to the repair.
- Nasal stents may be inserted to maintain patency of the airway. A tongue stitch is rarely used.
- Regular paracetamol and ibuprofen. IV morphine may be required initially.

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Special considerations

- Intubation is usually uncomplicated.
- The laryngoscope blade rarely lodges in the cleft. If there is a problem, a roll of gauze can fill the gap.
- Prolonged surgery may cause a swollen tongue from pressure of the mouth gag.
- Cleft palate repair can produce upper airway obstruction, and extreme care is needed for extubation.
- With airway problems, opioids should be given cautiously. Postoperative monitoring should include SpO_2 and apnoea alarm.
- Cleft lip is usually repaired at 3mo, and cleft palate at 6–9mo. The lip may be repaired at the neonatal stage to improve the scar and assist maternal bonding. There is little evidence to support this.

Congenital talipes equinovarus

Procedure	Correction of club foot abnormality
Time	45–90 min
Pain	++
Position	Supine, sometimes prone for posterior release
Blood loss	Minimal
Practical techniques	SV + LMA or ETT + IPPV, caudal If prone, then ETT + IPPV, caudal

Preoperative

- Occurs in 1:1000 births.
- Usually an isolated anomaly but may occur in association with some myopathic diseases, hence ↑ theoretical risk of MH.
- Obtain consent for a suppository and regional block.

Perioperative

- Inhalational or IV induction. If prone, intubate and ventilate. Give additional opioids, if indicated.
- Extended caudal blockade: 1mL/kg of 0.25% levobupivacaine + clonidine (see pp. 929–30; p. 931).
- Paracetamol IV and diclofenac PR.

Postoperative

Regular paracetamol and ibuprofen plus PO morphine PRN.

Special considerations

For prolonged pain relief, either top up the caudal at the end of the procedure by using an indwelling epidural catheter or extend the duration of the block by adding clonidine (1–2 micrograms/kg) to the initial dose of bupivacaine.

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Femoral osteotomy

Procedure	Stabilising the hip in congenital dislocation by re-aligning the proximal femur
Time	2h
Pain	+++
Position	Supine
Blood loss	Moderate/potentially large
Practical techniques	SV + LMA or ETT + IPPV, caudal/epidural

Preoperative

- Usually an isolated defect. More common in girls or where there is a family history.
- Obtain consent for a regional block and suppository.

Perioperative

- Inhalational or IV induction. Adequate IV access.
- Caudal block plus clonidine (see ➲ pp. 929–30; ➲ p. 931) if hip spica to be applied.
- Alternatively, femoral nerve block + LA to skin incision.
- Lumbar epidural if high osteotomy (see ➲ p. 931).
- Intraoperative opioids if regional block not possible (➲ pp. 926–8).
- Paracetamol IV and diclofenac PR.
- Employ heat conservation measures.
- Attention to blood loss.

Postoperative

- A hip spica provides support and helps with pain relief.
- Extended caudal or morphine NCA, with regular NSAIDs and paracetamol.
- Epidural infusion (Table 36.15) with regular paracetamol, NSAIDs + PO morphine PRN.

Special considerations

- Blood loss may be extensive if revision surgery. Consider cell saver.
- A hip spica complicates urinary retention in girls.

Inhaled foreign body

Procedure	Removal of foreign body from bronchial tree
Time	30–60min
Pain	+
Position	Supine
Blood loss	Nil
Practical techniques	SV or IPPV

Preoperative

- Commonest reason for bronchoscopy in the 1–3y age group.
- May present as an emergency acute airway obstruction.
- Presentation of lower airway obstruction may occur several days after a history of coughing. Peanut oil is an irritant and leads to mucosal oedema and chemical pneumonitis. CXR shows characteristic hyperinflation of affected side during expiration, but a foreign body is not always visible.
- Treat symptoms as indicated, e.g. dehydration, pneumonia, wheeze.

Perioperative

- Inhalational induction is usual to avoid displacing the object further. Use 100% O_2 with sevoflurane.
- TIVA is becoming a more popular maintenance technique.
- Apply topical anaesthesia to the vocal cords (4% lidocaine, up to 3mg/kg), and consider a drying agent (atropine 20 micrograms/kg IM 30min preoperatively or 10 micrograms/kg IV at induction, or glycopyrronium 5 micrograms/kg IM or IV).
- Prior to bronchoscopy, maintain airway with a face mask or LMA.
- Rigid bronchoscopy: the Storz bronchoscope has an attachment for a T-piece. Check compatibility before the procedure.
- For foreign objects in the upper airways, maintain SV. If tracheal/ball–valve obstruction suspected, IPPV is contraindicated.
- If the foreign body is in the lower airway, then IPPV with a muscle relaxant is acceptable, since the object will be pushed distally by the bronchoscope until it can be grasped by forceps. Give assisted ventilation via a T-piece or high-frequency jet ventilation.
- This may be a difficult surgical procedure.

Postoperative

- If bronchoscopy is traumatic, give dexamethasone 0.25mg/kg IV, then two doses 8-hourly of 0.125mg/kg.
- Consider physiotherapy, bronchodilators and antibiotics as indicated.

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Sedation

- The expansion of imaging techniques and diagnostic and therapeutic interventions has led to a rise in demand for sedation services.[29]
- The safe sedation of children for procedures requires a systematic approach. Compared with GA, sedation is neither cheaper nor safer.
- Anaesthetists are not always available to administer sedation, and other medical or nursing personnel may be involved. Sedation guidelines are essential[29] and monitoring of SpO_2 is mandatory. With appropriate planning, nurse-led sedation services have been developed at several centres following strict protocols. The children are fasted conventionally but allowed unrestricted clear fluids.
- Safety is paramount—facilities must include an airway trolley and monitoring and resuscitation equipment, together with all personnel necessary to sedate the child and carry out the specific procedure. All standard anaesthetic equipment should be available for resuscitation.
- Each sedated child must be supervised by an appropriate nurse or doctor trained in paediatric resuscitation. Experienced medical staff must be immediately available to assist with sedation problems or resuscitation. There must be a contingency for overnight admission if recovery is prolonged.
- The adult concept of sedation with verbal contact maintained is not practical in children. There may be little difference between deep sedation, as defined by the American Academy of Pediatrics, and uncontrolled anaesthesia. Ideal conditions achieve depression of the nervous system, allowing the procedures to occur, with preservation of airway reflexes. In practice, this is difficult to achieve.
- It is important not to confuse sedation with analgesia. Painful procedures may require a topical anaesthetic cream, infiltration with LAs and occasionally systemic opioids.
- Contraindications include children with potential airway issues, apnoeic episodes, respiratory disease, raised ICP, risk of pulmonary aspiration and epilepsy.
- Medications used:
  - Chloral hydrate 50–100 micrograms/kg for younger children.
  - Midazolam (0.5mg/kg PO or incremental bolus doses of 0.05–0.1mg/kg IV, up to a maximum of 0.4mg/kg) can produce good conditions for sedation; additional property of amnesia.
  - Ketamine (6–10mg/kg PO or 0.5–1mg/kg IV boluses every 10–15min) is indicated for short, painful procedures and may be used in combination with midazolam. Emergence delirium is less of a problem with children, but a drying agent is often required.
  - Propofol can be used alone or in combination with remifentanil for endoscopy sedation, but the use of these drugs should be reserved for anaesthetists.[29]
  - Dexmedetomidine 1 microgram/kg and/or infusion 0.2–1 micrograms/kg/h. Can cause bradycardia.
- Surprisingly young children can tolerate scans awake with encouragement, careful explanation and parental presence.

Medical problems

Acute laryngotracheobronchitis (croup)

- Croup occurs predominantly in epidemics in autumn and early spring. The peak age of incidence is 6mo to 2y. It is viral in aetiology. The majority of cases are due to parainfluenza, but influenza and respiratory syncytial virus are possible.
- Symptoms are coryzal for the first few days but then progress to a characteristic barking cough/hoarseness with profuse secretions and occasional dysphagia. Pyrexia is mild or absent.
- The larynx, trachea and bronchi are all involved and become oedematous, leading to the onset of stridor. An anxious child will exacerbate the condition, as the trachea will tend to collapse on inspiration.
- The majority of children respond to conservative measures and reassurance. In severe cases, steroids (dexamethasone 0.25mg/kg IV followed by two further doses 8-hourly of 0.125mg/kg) and nebulised adrenaline (0.5mg/kg, up to a maximum of 5mg) are required.
- 10% of children are admitted, and 1% will require intubation.
- The majority of children have a single isolated episode.

Acute epiglottitis

- This is an acute life-threatening infection caused by Hib. It most commonly presents at 2–3y.
- There is a rapid onset of oedema of the epiglottis and aryepiglottic folds. The child has a high temperature, usually >39.5°C, and presents sitting or leaning forwards, with drooling saliva, and unable to swallow, with the tongue pushed forwards. Inspiratory and expiratory stridor is rapidly progressive and is a late sign.
- Acute epiglottitis is a medical emergency. The antibiotic of choice is ceftriaxone (50mg/kg IV once daily). Intubation is indicated in 60% of cases. In some centres, all children are routinely intubated.
- Following the introduction of the Hib vaccine, this condition is now rare.

Anaesthetic management of croup and acute epiglottitis

- The differential diagnosis between croup and epiglottitis is not always obvious. If epiglottitis is even remotely suspected, there must be liaison with an ENT surgeon at consultant level.
- Induction occurs in the anaesthetic room or operating theatre, with the full range of appropriate equipment and monitoring available.
- During anaesthesia, the ENT surgeon should be scrubbed in theatre, with the tracheostomy set open.
- Traditionally, IV access has been contraindicated prior to induction because of the risk of acute glottic closure. However, the use of a topical cream facilitates an atraumatic venepuncture. Unless access is obviously difficult, cannulation should proceed before anaesthesia.
- Inhalational induction is performed in the sitting position, with sevoflurane in 100% O_2. Once anaesthetised, the child can be moved to a more recumbent position and maintained with sevoflurane, up to concentrations of 8%, if needed. CPAP should be routinely applied, but the airway is not usually difficult to maintain.

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- In **croup**, laryngoscopy is usually straightforward, but the ETT size required may be surprisingly small. Start with one size smaller than normal. Older children may require a tube that has been cut to a longer length. If possible, once the airway is secure, the child should be reintubated nasally since this is better tolerated. Profuse secretions are always a problem and frequent suction is necessary. Intubation is usually required for at least 2–3d, and bronchoscopy is indicated if an air leak around the tube fails to develop.
- With **epiglottitis**, intubation may be exceedingly difficult. Laryngoscopy often reveals an abnormal anatomy with no obvious glottic opening. Careful inspection or pushing gently on the chest may reveal movement of small amounts of mucus, indicating tidal flow. The child should be intubated, using a stylet, so that the ETT can be immediately railroaded, if necessary. The tube size will be smaller than predicted.
- Once intubation has been achieved, oedema rapidly settles. Following demonstration of a leak around the tube, extubation is normally possible within 36h. Dexamethasone is often given prior to extubation to reduce laryngeal oedema.

Paediatric advanced life support

Cardiopulmonary arrest in children is usually 2° to decompensated respiratory or circulatory failure rather than cardiac in origin. The commonest arrest scenario in children is bradycardia proceeding to asystole—a response to severe hypoxia and acidosis. Oxygenation, ventilation and good-quality chest compressions should therefore be a priority of management (Fig. 36.2).

- Performing chest compression is tiring; if possible, rotate team members every 2min.
- VF is relatively uncommon but may complicate hypothermia, tricyclic poisoning and children with pre-existing cardiac disease (Fig. 36.3).
- Many parents wish to be present at resuscitation. A team member should be available to explain the process and provide support.
- Adopt a **SAFE** approach:
  - **S**hout for help.
  - **A**pproach with care.
  - **F**ree the patient from immediate danger.
  - **E**valuate the patient's ABC.
- Assess the airway and breathing first. Give five **rescue** breaths (chest seen to rise and fall) before assessing the circulation. Each breath should take about 1–1.5s.
- Check the carotid pulse in a child, but use the brachial pulse in an infant. Feel for no more than 10s.
- Commence basic life support using a ratio of 15 compressions:2 ventilations at 100–120 compressions/min (five cycles). Each compression should depress the sternum one-third of the anteroposterior (AP) diameter (~4cm in infants and 5cm in children).
- Chest compressions should be started if a central pulse cannot be palpated or the child has a pulse rate <60bpm with poor perfusion.
- Where no vascular access is present, immediate IO access is recommended.
- Once the airway has been secured, chest compressions should be continued at 100–120/min uninterrupted, with breaths administered at a rate of 10–12/min.
- Once restoration of spontaneous circulation (ROSC) is achieved, ventilate the child at 12–20 breaths/min to normalise $PaCO_2$.

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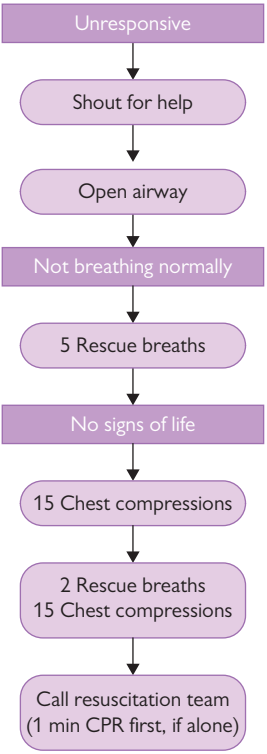
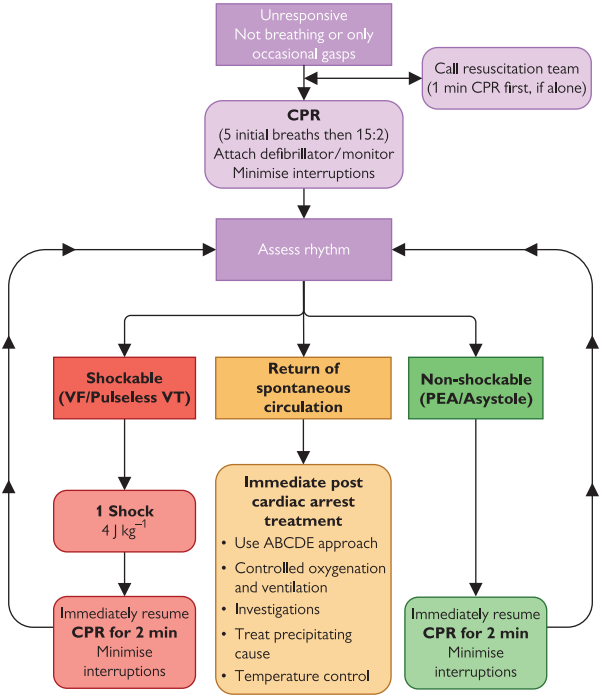


Fig. 36.2 Paediatric basic life support. Reproduced with the kind permission of Resuscitation Council UK.



During CPR

- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3–5min
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Correct reversible causes
- Consider amiodarone after 3 and 5 shocks

Reversible causes

- Hypoxia
- Hypovolaemia
- Hyper/hypokalaemia, metabolic
- Hypothermia
- Thrombosis (coronary or pulmonary)
- Tension pneumothorax
- Tamponade (cardiac)
- Toxic/therapeutic disturbances

Fig. 36.3 Paediatric advanced life support. Reproduced with the kind permission of Resuscitation Council UK.

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Asystole and pulseless electrical activity

This is a more common arrest scenario in children (Fig. 36.4).

- Continue CPR using 15 compressions:2 breaths with high-flow O_2.
- If intubated, continue uninterrupted chest compressions and ventilate at 10–12 breaths/min.
- Secure IO access if vascular access not possible.
- **Give adrenaline 10 micrograms/kg** (0.1mL/kg of 1:10 000 solution). Repeat at alternate cycles (3–5min).
- Review rhythm every 2min.

If no improvement, consider reversible causes (Box 36.2).

Box 36.2 Reversible causes (4Hs and 4Ts)

Hypoxia	Thromboembolism
Hypovolaemia	Tension pneumothorax
Hyper-/hypokalaemia	Tamponade (cardiac)
Hypothermia	Toxins

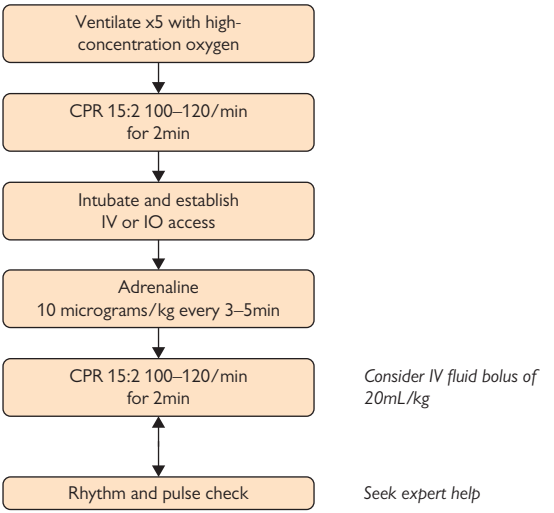

Fig. 36.4 Asystole or pulseless electrical activity.

Post-resuscitation care

- Titrate O_2 for O_2 saturations of 94–98%.
- Maintain CO_2 4.5–5.0kPa.
- Avoid hypotension. Consider fluids and/or inotropes.
- Suggested, but no statistical evidence for, mild hypothermia 32–34°C.[30]
- Moderate glucose control.

Ventricular fibrillation or pulseless ventricular tachycardia

(See Fig. 36.5.)

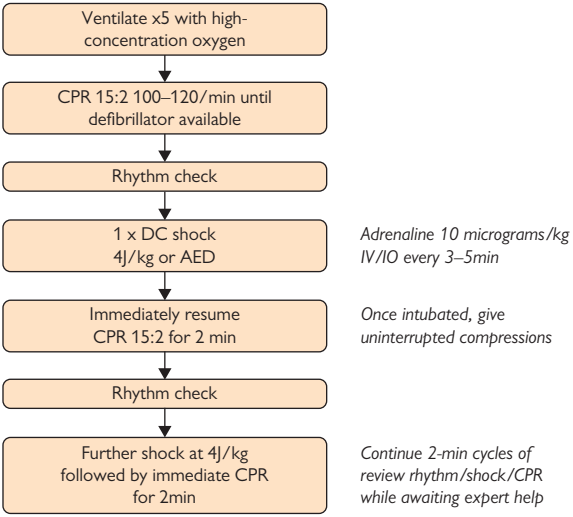

Fig. 36.5 Ventricular fibrillation or pulseless ventricular tachycardia.

Notes

- Continue shocks 4J/kg every 2min. Resume CPR immediately after defibrillation without checking output.
- Standard automated external defibrillators (AEDs) may be used in children over 8y. Purpose-made paediatric pads are recommended for children aged 1–8y, if available. Below 1y, a manual defibrillator should be used if available, but an adult AED can be used.
- Give **adrenaline 10 micrograms/kg** (= 0.1mL/kg of 1:10 000 solution) and amiodarone 5mg/kg after a 3rd shock, once compressions have been resumed.
- Repeat adrenaline every alternate cycle (3–5min).
- Further antiarrhythmic agents:
  - Repeat amiodarone 5mg/kg IV after 5th shock.
  - Torsade de pointes/hypomagnesaemia: magnesium sulfate 25–50mg/kg.
  - There is no evidence that atropine confers any benefit in asphyxial bradycardia or asystole.
- Calcium: routine use associated with ↑ mortality. Indicated for hyperkalaemia, hypocalcaemia and calcium channel blocker overdose.
- Sodium bicarbonate 1mmol/kg: routine use not recommended. Indicated for hyperkalaemia and tricyclic overdose.

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Neonatal resuscitation

Condition	Acute neonatal asphyxia during the birth process
Presentation	Floppy, blue or pale, HR <60bpm, diminished respiratory effort
Immediate action	Delay cord clamping for at least 1min. Dry, wrap and warm the baby. Open and clear airway, five inflation breaths with air (2–3s at 30cmH_2O)
Follow-up action	Cardiac compressions (3:1) at 120/min if <60bpm, review ventilation
Investigations	Record Apgar scores (Table 36.16), take cord gases
Also consider	Hypovolaemia, diaphragmatic hernia, pneumothorax. Consider therapeutic hypothermia

Risk factors

- Known fetal distress; Category 1 emergency CS; meconium-stained liquor; <35w gestation.
- Prolonged delivery; vaginal breech; instrumental delivery; shoulder dystocia; multiple births.
- Maternal drugs: opioids, GA for CS.
- Preterm delivery (prognosis is very poor if gestation <23w).

Diagnosis

- A normal newly delivered baby is pink, breathes spontaneously within 15s, has a HR >100bpm and good muscle tone and is vocal.
- A baby requiring resuscitation is floppy, silent, blue or pale, has a HR <100bpm and gasping, diminished or absent respiratory effort (see Apgar scores in Table 36.16).

Table 36.16 Apgar scores

	0	1	2
Colour	Pale/blue	Blue extremities	Pink
HR (bpm)	Absent	<100	>100
Response to stimulation	Nil	Movement	Cry
Muscle tone	Limp	Some flexion	Well flexed
Respiratory effort	Absent	Poor effort/weak cry	Good

Source: data from Apgar A (1953) A proposal for a new method of evaluation of the newborn infant. *Anesth Analg*, **32**; 250–9.

Immediate management

- Keep the theatre warm. Dry and wrap the baby. Actively maintain temperature between 36.5°C and 37.7°C. Keep warm under a radiant heater. Use polythene wrapping for preterm babies <30w.
- Consider ECG and preductal SpO_2 (right hand). Maintain saturations <95%.

- Open and clear the airway, but keep the neck in a neutral position.
- Routine suctioning is not recommended.
- Give five effective inflation breaths (2–3s at 30cmH_2O) initially with air.
- HR should increase; continue ventilating at 30–40 breaths/min, until spontaneous effort is adequate.
- If HR remains <60bpm, commence chest compressions with thumbs around the chest, at a compression rate of 120/min and a ratio of 3:1 breaths. Compress the chest diameter by one-third.
- Reassess HR every 30s.

(See Fig. 36.6.)

Subsequent management

- In the neonate that remains unresponsive despite oxygenation, consider intubation and drugs (Table 36.17). This is seldom required.

Table 36.17 Resuscitating the neonate

	40/40 gestation	35/40 gestation	30/40 gestation
Weight (kg)	3.5	2.5	1.5
ETT ID (mm)	3.5	3.0	2.5
ETT length (cm)	9.5	8.5	7.5
Adrenaline 1:10 000 (IO/IV) (mL)	0.35–1.0	0.25–0.75	0.15–0.45
Sodium bicarbonate 4.2% IV (mL)	3.5–7	2.5–5	1.5–3
Glucose 10% IV (mL)	17–35	12–25	7–15
Volume IV (O-negative/0.9% sodium chloride) (mL)	35–70	25–50	15–30

Other considerations

- If response to resuscitation is prompt (required support breaths only and now making good, spontaneous respiratory effort), return the baby to the parents.
- For ventilatory depression thought to be due to maternal opioids, give naloxone 200 micrograms IM.
- Therapeutic hypothermia is indicated for term or near-term infants with moderate to severe hypoxia.

Further reading

Resuscitation Council (UK) (2015). *Guidelines: Resuscitation and support of transition of babies at birth.* https://www.resus.org.uk/library/2015-resuscitation-guidelines/resuscitation-and-support-transition-babies-birth

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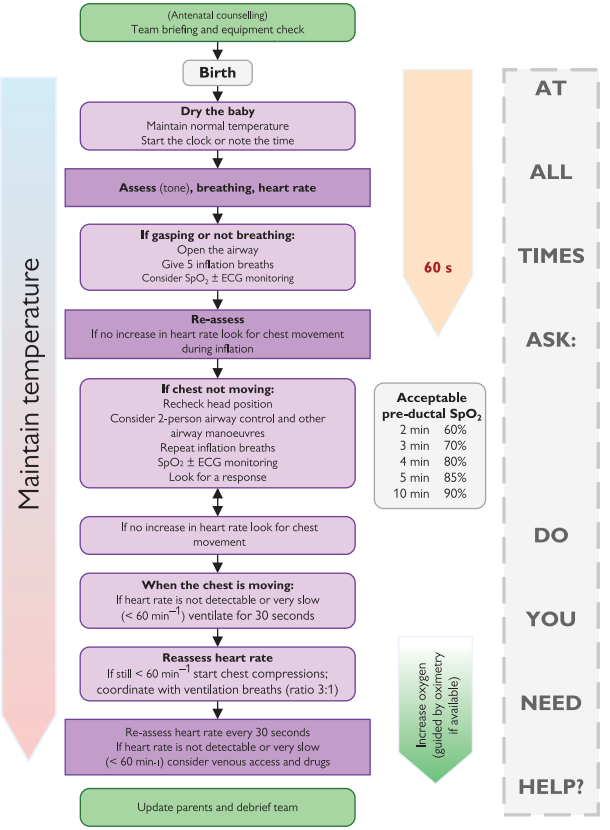

Fig. 36.6 Neonatal life support. Reproduced with the kind permission of Resuscitation Council UK.

The collapsed, septic child

Condition	Sepsis with hypoperfusion and/or respiratory failure progressing to capillary leak and multiorgan failure
Presentation	Irritability, inappropriate crying, drowsiness, confusion, poor interaction with parents, lethargy or becoming unrousable, along with other features such as tachypnoea and reduced urine output
Immediate action	High-flow O_2, fluid resuscitation, antibiotics
Follow-up action	Referral to a specialist paediatric critical care unit
Investigations	Blood cultures, lactate, urine output FBC, clotting, U&E, glucose
Also consider	Hypovolaemia/blood loss, anaphylaxis, poisoning, cardiac abnormality

Risk factors

- Prematurity, immune deficiency, chronic cardiorespiratory disease, endocarditis, CNS infections.
- Exposure to MRSA, Gram-negative bacteria, nosocomial infection and fungi.

Recognition of at-risk child

- Suspected or proven infection plus two out of: temperature <36.5°C or >38.5°C, tachycardia, altered mental state, prolonged capillary refill time (>2s), immunocompromise and hypotension.
- Look for signs of warm or cold shock:
  - Warm shock: vasodilation, bounding peripheral pulses, wide pulse pressure
  - Cold shock: cool peripheries, capillary refill time >2s, narrow pulse pressure.

Red flag signs

- Any one sign of the following: lactate >2mmol/L; extreme tachycardia or tachypnoea; SpO_2 <90%, grunting, cyanosis, apnoea; reduced level of consciousness, persistent hypotension and non-blanching rash/mottled skin.

Immediate management

- Aggressive early management improves prognosis. Hypovolaemia is severe and often under-resuscitated.
- **ABC**: maintain/support airway; high-flow O_2 via a non-rebreathing mask; consider ventilatory support ± intubation.
- **Rapid fluid boluses** of 20mL/kg of crystalloid/albumin: 80–100mL/kg may be required to restore normovolaemia.
- **Correct hypoglycaemia**: 2mL/kg 10% glucose.
- **Inotropic support**: if no significant response to fluid, start adrenaline 0.1–0.5 micrograms/kg/min.
- Antibiotics: cefotaxime 50mg/kg IV 1st line; for 2nd line, discuss with local microbiology team.
- Early referral to specialist centre.

Subsequent management

- Ventilation under sedation and paralysis: target V_T 4–7mL/kg, initial PEEP 5cmH_2O. Saturation >95%.
- Inotropes:
  - Cold shock: adrenaline 0.1–0.5 micrograms/kg/min IV
  - Warm shock: noradrenaline 0.1–0.5 micrograms/kg/min IV.
- Risk of adrenal failure: hydrocortisone 1mg/kg qds IV.
- Consider the possibility of meningitis/encephalitis and raised ICP.
- Stabilise for transfer and prepare handover documentation. A retrieval service may be provided by the receiving unit.

Other considerations

- Significant fluid shift from capillary leakage may result in pulmonary oedema 2° to fluid resuscitation. If drugs are required to intubate the child, anticipate an exaggerated fall in BP and adjust the dose accordingly. Use ketamine, fentanyl, rocuronium and cuffed ETT. It is wise to start inotropes and fluid resuscitation before induction.

Further reading

NHS Children's Acute Transport Service (CATS) (2020). *Clinical guidelines: septic shock*. https://cats.nhs.uk/wp-content/uploads/guideline-sepsis.pdf

Randolph AG, McCulloh RJ (2014). Paediatric sepsis: important considerations for diagnosing and managing severe infections in infants, children and adolescents. *Virulence*, **5**, 179–89.

Stabilisation of the sick child (prior to PICU transfer)

- Anaesthetists may be required to assist with the stabilisation of critically ill children, including difficult airway management, prior to surgery or transfer.
- Seek early contact and advice from the local paediatric intensive care retrieval team. Initial resuscitation remains the responsibility of the local medical teams.
- Frequently involves the coordination of multidisciplinary teams, including paediatricians, intensivists, anaesthetists and surgeons. Management of sick children should follow a systematic, evidence-based approach.
- These cases will often take place away from theatres and familiar surroundings. The availability of a trained assistant is essential.
- Checklists for drugs and equipment, as well as emergency 'grab bags', will improve readiness.
- 'Run-throughs' or simulation will improve familiarity and identify potential problems with working in unfamiliar locations.

General principles

Airway and breathing

- Indications for intubation:
  - Protection of airway (low conscious level, facial trauma, burns)
  - Control of ventilation (respiratory failure, head injury).
- Cuffed tube, if possible (see ➲ p. 360). Avoid precutting smaller tubes. Capnography should be used.
- If equipment, monitoring or personnel are not optimal, consider risk/benefits of moving location, e.g. theatres.
- Induction: ketamine (0.5–2mg/kg) with rocuronium (1.2mg/kg). Must be fluid-resuscitated first. Use reduced induction doses if unstable.
- Ventilation: pressure control ventilation usually appropriate. Aim for normocapnia and O_2 saturations of 94–98%. PEEP is helpful if volume-resuscitated.
- Sedation: various regimens. Morphine and midazolam infusions are popular (Table 36.18). Propofol is acceptable for short transfers (remember analgesia).

Circulation

- IV access. Consider IO, if difficult. Central access may be needed.
- Isotonic fluid for boluses: 20mL/kg repeated. Be aware that intubation more likely after 40mL/kg. Blood transfusion may be required after 40mL/kg isotonic fluids (see ➲ p. 978 for fluid resuscitation).
- If blood loss is the cause, give blood products early. While waiting for blood products to arrive, give up to 20mL/kg fluid in 10mL/kg fluid boluses.
- Inotropes may be needed in addition to ongoing fluids/blood products to improve haemodynamics. Discuss with your local PICU or transfer team (Table 36.18). Adrenaline and dopamine can be given peripherally.
- Arterial line may be useful, but rarely essential.

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Table 36.18 Infusion regimes of selected PICU drugs

Medication	Dosing details
Morphine infusion (sedation) If <50kg, 1mg/kg in 50mL If >50kg, 50mg in 50mL	20–80 micrograms/kg/h 1mL/h = 20 micrograms/kg/h
Midazolam infusion (sedation) If <10kg, 5mg/kg in 50mL If >10kg, 50mg in 50mL	50–200 micrograms/kg/h 1mL/h = 100 micrograms/kg/h
Propofol	2–5mg/kg/h; titrate to effect
Dopamine infusion <15kg	2–10 micrograms/kg/min 15mg/kg in 50mL (central access) 1mL/h = 5 micrograms/kg/min of this mix Halve concentration if giving peripherally
Dopamine infusion >15kg	2–10 micrograms/kg/min 200mg in 50mL (central access) 80mg in 50mL (peripheral access)
Adrenaline infusion <15kg	0.1–0.5 micrograms/kg/min 0.3mg/kg in 50mL 1mL/h = 0.1 micrograms/kg/min of this mix
Noradrenaline infusion >15kg	0.1–0.5 micrograms/kg/min 4mg in 50mL

Further reading

Many local retrieval service websites include guidelines and drug calculators. For example: Wales & West Acute Transport for Children service (WATCh). https://www.watch.nhs.uk/about

Managing Emergencies in Paediatric Anaesthesia (MEPA). [Simulation-based training] https://mepa.org.uk

Spotting the Sick Child. [Education and case studies] http://www.spottingthesickchild.com

Roberts S, ed. (2019). *Paediatric Anaesthesia*, 2nd edn. Oxford: Oxford University Press.

Paediatric doses and equipment

(See Table 36.19.)

Table 36.19 Paediatric quick reference guide

Age	Approximate weight (kg)	Body surface area (m^2)	Percentage of adult drug dose (approximate)	ETT size (mm)	ETT length (cm)	LMA size	Suxamethonium dose (mg) IV	Atropine dose (micrograms) IV
Term	3.5	0.23	12.5(1/8th)	3.5	9	1	7	35
1mo	4.2	0.26	14.5	3.5	10	1	8	40
3mo	6	0.33	15	3.5	10	1.5	12	60
6mo	7.5	0.38	22	3.5/4.0	11	1.5	15	75
1y	10	0.47	25(1/4)	4.0	12	1.5/2	20	100
2y	12	0.53	30	4.5	13	2	24	120
3y	14	0.61	33	4.5/5	13/14	2	28	140
5y	18	0.73	40	5.0/5.5	14.5	2.5	36	180
7y	22	0.86	50(1/2)	6.0	15.5	2.5	44	220
10y	30	1.10	60	6.5cuffed	17	3	60	300
12y	38	1.30	75(3/4)	7.0cuffed	18	3/4	75	380

Note: weights are approximations only. Patients should be weighed accurately.

Weight/blood pressure estimation (1–10y)

- Child's weight in kg (see ➲ p. 903)
- Normal systolic BP in mmHg = (age in y × 2) + 80.

Airway

- ETT ID in mm = (age in y/4) + 4
- ETT length (oral) to lips in cm = (age in y/2) + 12
- ETT length (nose) to lips in cm = (age in y/2) + 15
- LMA#1 (cuff volume 4mL): <6.5kg
- LMA#2 (cuff volume 10mL): 6.5–20kg
- LMA#2.5 (cuff volume 14mL): 20–30kg
- LMA#3 (cuff volume 20mL): >30kg.

Estimated drug doses

(See also ➲ p. 963.)

- Adrenaline 10 micrograms/kg
- Aminophylline 5mg/kg
- Amiodarone 5mg/kg
- Atropine 10–20 micrograms/kg
- Bicarbonate 1mmol/kg (use 4.2% <1y)
- Calcium chloride 0.2mL/kg of 10% solution slowly
- Calcium gluconate 0.6mL/kg of 10% solution

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- Cefotaxime 50mg/kg
- Diazepam 0.1mg/kg IV; 0.5mg/kg PR
- Glucose (10%) 2mL/kg
- Ketamine 2mg/kg
- Lorazepam 0.1mg/kg for status epilepticus (repeatable after 10min)
- Magnesium 25–50mg/kg
- Naloxone 0.1mg/kg
- Neostigmine 50 micrograms/kg
- Paraldehyde 0.1mg/kg
- Phenytoin 10–20mg/kg
- Salbutamol 2.5mg nebuliser.

Circulation

- Blood volume: 75mL/kg (1–10y), 70mL/kg (>10y)
- Fluid bolus: 20mL/kg.

These estimations are not valid for premature infants and are intended as rough guides only.[31]

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Chapter 37

The major trauma patient

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The patient journey

Trauma

Trauma is injury caused when physical energy is transferred to the body from an outside force. Kinetic energy may be blunt or sharp in nature. Other mechanisms include pressure (blast injuries) and thermal (hot/cold/electrical/chemical burns).

Penetrating injuries result from an object entering the body (and sometimes exiting the body), causing damage along the path. They can be classified as low velocity (knife/stabbing) or high velocity (gunshot wound). The range of injury is vast and depends on what instrument is used, how much force is applied and in which direction and which body cavity/organ/tissue is penetrated.

Blunt trauma are injuries that occur when an object does not enter the body. It may be caused by an impact (road traffic collision, fall from height, crush) or assault. It produces a spectrum of effects from minor, single-system to life-threatening, multisystem injuries. Typically, these include contusions, abrasions, lacerations, tearing (shear forces) and fractures to the affected body parts. The degree of damage depends on the mass and speed of the moving object.

The major trauma system

Severely injured patients who are treated in a major trauma centre (MTC) have better outcomes than those treated in smaller trauma units. Since 2010 in London, and 2012 in the rest of the UK, trauma networks have been set up whereby ambulances and prehospital services can bypass smaller trauma units to take trauma patients directly to an MTC.[1]

The MTC provides specialists in the management of major trauma and ensures critically injured patients are treated by the right people in the right place in the right time frame. Paediatric MTCs offer specialist treatment for patients below 16y old.

Prehospital care

The patient journey begins at the point of injury. Prehospital services have adapted and grown over time to improve patient management and outcome prior to arrival at the MTC. Prehospital emergency anaesthesia for intubation, blood administration and resuscitative thoracotomy are among the procedures that can now be provided in the field by appropriately trained prehospital clinicians. These teams are supported by specialist ground and air ambulance services which transport these patients to the most appropriate centre.

Advance warning and concise communication using an ATMIST[2,3] pre-alert to the ED will allow the team to prepare (Box 37.1). This ensures essential resuscitation drugs, blood, staff and equipment are available before the patient's arrival. Code words given by the prehospital team allow organisational systems to be activated, e.g. blood products to be present on arrival, specialist surgical teams pre-alerted and theatres to be made immediately available.

Box 37.1 ATMIST pre-alert communication tool

- Age and name
- Time of incident
- Mechanism of injury
- Injuries top to toe
- Vital Signs (initial and significant changes)
- Treatment.

Also include estimated time of arrival, mode of transport and specialist resources required to be standing by.

Arrival in the resus room

Trauma teams

Resuscitation of the major trauma patient is most effective when undertaken by a team of specifically trained doctors and nurses with the leadership of a trauma team leader (TTL). Allocated roles include: TTL, 1° survey doctor, nurse, scribe, runner and blood monitor (if transfusion anticipated). Team members work in parallel to simultaneously perform assessment, investigations, procedures and treatments under the direction of the TTL.

As an anaesthetist presenting to the ED prior to arrival of the patient, it is important to identify and introduce yourself (name and grade) to the TTL. The TTL will brief the team using ATMIST and allocate roles. As the anaesthetist, you are likely to be allocated airway management and neurological assessment. Check your equipment; draw up medications if appropriate and prepare for arrival of the patient.

Handover

Critical information from the prehospital phase of the patient journey must be handed over to the trauma team on arrival. Prior to handover, a rapid assessment is made by the TTL to ensure the patient has:

- A patent airway
- A pulse
- No uncontrolled, external, catastrophic haemorrhage.

This rapid assessment should take <10s and, once complete, is verbalised to the team. Alternatively, the TTL can ask the prehospital team if they have any immediate concerns. Only once the TTL is satisfied no immediate action needs to be taken, can patient handover start.[4] Handover should be concise and completed within a minute. Avoid distractions, such as placing monitoring, during this time to ensure you pay attention to the handover. The team can then commence the next phase of the management.

Resuscitation room management

Traditional teaching by the well-known Advanced Trauma Life Support® (ATLS®)[5] framework considers the initial management in four phases:

- 1° survey
- Resuscitation
- 2° survey
- Definitive care.

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The first two phases are carried out simultaneously, during which there is multidisciplinary discussion and planning for the next steps of patient care, e.g. theatre, CT, ICU or ward. The 2° survey (see p. 980) is not started until the patient has been adequately resuscitated and stabilised. Clear documentation should be made of each stage of the process: examination findings, investigations and treatment, including medications and transfusion. Interruptions in care for investigations and handover between teams (e.g. ED to theatre) are common, and outstanding tasks must be recorded (e.g. 2° survey not complete) so they are not forgotten. As the anaesthetist, you may accompany the patient from the ED, via imaging, to theatre and onwards to the ICU, so you are uniquely placed to provide continuity of care, ensuring that tasks are completed and details retained.

Primary survey and resuscitation

- During the 1° survey, the TTL should have a hands-off approach, standing at the foot of the bed directing the team, while simultaneously maintaining an overview and good situational awareness. Closed loop questions and clear objectives from the TTL allow for a smooth transition through the phase of immediate management.
- The principles of the 1° survey are to identify and resolve life-threatening injuries in order of priority (Table 37.1).

Table 37.1 Principles of the primary survey and resuscitation

C	Catastrophic haemorrhage control	Control via direct pressure and tourniquet
A	Airway with C-spine control	Maintain airway patency and control the C-spine
B	Breathing	Identify and treat chest injuries to ensure oxygenation and ventilation
C	Circulation	Identify causes of shock and treat appropriately
D	Disability	Perform a rapid assessment of neurological function
E	Exposure	Ensure full exposure of the patient while preventing hypothermia

Management of catastrophic haemorrhage

- Life-threatening bleeding will lead to exsanguination and death unless prompt treatment is instigated. Sequential management involves use of elevation, direct pressure, indirect pressure to a proximal artery, trauma pressure dressings, topical haemostatic agents (e.g. Celox™) and tourniquets.
- ⚠ If the trauma patient is >20w pregnant, aortocaval compression from the gravid uterus will exacerbate hypotension. A left lateral tilt or manual left displacement of the gravid uterus should be instituted as early as possible and maintained throughout resuscitation to optimise preload. Consider the possibility of pregnancy in any ♀ trauma patient of childbearing age. (See also ➲ pp. 460–1.)

Airway with cervical spine control

- The priority during resuscitation of any severely injured patient is to ensure a patent airway and maintain oxygenation. In doing so, strategies to avoid exacerbating any C-spine injury must be employed.
- Assume the presence of a spinal injury in any patient who has sustained significant blunt trauma, until clearance procedures have been completed.

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- Exclusion of C-spine injury must be undertaken by an experienced clinician after clinical examination and imaging have been completed. A reliable clinical examination cannot be obtained if the patient:
  - Has sustained a significant closed head injury
  - Is intoxicated
  - Has a reduced conscious level from any other cause
  - Has significant pain from an injury, which 'distracts' attention from the neck.[6]
- Practical considerations need to be taken into account when applying a rigid collar and blocks, especially to agitated patients or elderly patients who may have pre-existing spinal conditions.
  - Jaw thrust is the preferred technique to open an airway in trauma patients, as it minimises movement of the C-spine.
  - The use of direct visual suction (taking care not to stimulate vomiting in the obtunded patient) and adjuncts such as an oropharyngeal airway can be used.
  - Avoid instrumenting the nasopharynx in head injury patients due to the possibility of base of skull fractures allowing access to the cranium. ⚠ Nasopharyngeal airways and NGTs should be used with extreme caution.
  - In the spontaneously breathing patient, O_2 is delivered with a reservoir (non-rebreathing) mask which delivers $FiO_2 \approx 0.85$.
- A non-exhaustive list of indications for intubation are listed in Box 37.2. Prior to intubation, it is extremely important to document a precise GCS and pupil assessment.

Induction of anaesthesia and intubation of the major trauma patient
(See ➲ p. 388.)

- **Teamwork** is key to 1st-time success. There needs to be clear communication between the TTL and the clinician performing the intubation.
- **Prepare**: prepare and check all drugs and equipment. Refamiliarise yourself with the equipment available (e.g. VLs or emergency cricothyroidotomy equipment).

Box 37.2 Indications for endotracheal intubation

- Airway obstruction unrelieved by basic airway manoeuvres
- Impending airway obstruction (e.g. inhalational injury and burns)
- Airway compromise (e.g. soiling from maxillofacial injuries)
- Respiratory failure: consider intubation for head-injured patients to maintain normocapnia and $PaO_2 \geq 8$kPa
- Damage control or resuscitative surgery
- Expedient intubation for analgesic/humanitarian reasons
- Combative/uncooperative patient, compromising further investigation or treatment
- Reduced or rapidly reducing GCS or loss of laryngeal reflexes. Historically GCS ≤8; however, patients with a higher GCS may still require intubation

- **Allocate clear roles**: 1st intubator, 2nd intubator (if available), airway assistant, drug administrator, cricoid pressure, MILS of the C-spine.
- **Vocalise the airway plan** and strategy for failed intubation. An intubation checklist should be used.
- **MILS of the C-spine**: unless the trauma is an isolated penetrating injury, full MILS C-spine immobilisation should be used during intubation. MILS can be maintained from the side (ideal as the team member then does not interfere with the intubator) or head-end of the patient. It should be instituted prior to removal of the collar or blocks and continues until they are replaced at the end of intubation. Traction must not be applied.
- **Preoxygenate**: consider augmenting Mapleson C/circle circuit high FiO_2 and 5cmH_2O PEEP with nasal O_2 cannulae at 15L/min placed pre-intubation if SpO_2 <90%. If the patient's ventilatory effort is inadequate, gentle assisted ventilation, avoiding inflating the stomach, can be used.
- **Cricoid pressure**, if used, should be two-handed (anterior and posterior) to prevent displacement of any C-spine injuries.
- **Induce anaesthesia**. All induction drugs have the potential to cause vasodilation and compound hypotension.
  - Ketamine, fentanyl and rocuronium have become the drug combination of choice in major trauma, for both prehospital and in-hospital emergency use. The dose regimens should be sensitive to the patient's physiology.
  - For the trauma patient without physiological compromise: 3 micrograms/kg fentanyl, 2mg/kg ketamine and 1mg/kg rocuronium is appropriate (a '3-2-1 induction').
  - For physiologically unstable patients, reduce the doses to 1 microgram/kg fentanyl, 1mg/kg ketamine and 1mg/kg rocuronium (a '1-1-1- induction').
  - If the patient is critically unwell, ketamine in reduced doses and rocuronium alone may be used.
  - Alternative agents: thiopental can be used for normotensive head injuries. Suxamethonium 1.5mg/kg can be used in the immediate aftermath of trauma, even in patients with burns or spinal injury; however, in patients with severe muscle injury or if there is a suspicion of raised K^+ from other causes, it should be avoided.
  - Blood/fluid should be connected to a large-bore cannula and volume given to compensate for the effects of anaesthesia.
- **Laryngoscopy**:
  - Anticipate difficult laryngoscopy. Full C-spine immobilisation with collar, blocks and tape limits neck movement to 5% of the normal range, resulting in Cormack and Lehane grade III or IV views in 64% of patients. Removing the anterior portion of the hard collar and using assistant-provided MILS makes it easier, but 22% of patients will still have grade III views at laryngoscopy.[7]
  - VLs with hyperangulated blades are the preferred method for intubation to reduce movement of the atlanto-occipital and atlantoaxial joints.

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  - If there is airway or maxillofacial trauma, direct laryngoscopy is ideal as a small amount of blood on a VL camera will rapidly obscure any view. A size 4 Macintosh blade with a short handle is suitable.
  - Some advocate use of bougie on 1st attempt.
  - If intubation is impossible, an SGA will provide a temporary airway but may not prevent aspiration. Refer to Fig. 15.4 (see ➲ p. 369) for DAS algorithm.
- **ETT**. A large, cuffed single-lumen ETT should be used. Confirm ETT placement with $ETCO_2$ (preferably waveform). This needs to be verbalised clearly to the TTL.

Breathing problems and thoracic trauma

The aim is to identify and correct the immediately life-threatening conditions which can follow blunt or penetrating thoracic trauma:

- Tension pneumothorax
- Open pneumothorax
- Massive haemothorax
- Flail chest
- Airway disruption/obstruction
- Cardiac tamponade.

Other non-life-threatening conditions will be discussed later in the chapter.

- A systematic approach, including inspection, palpation, percussion and auscultation, paying specific attention to the axillae, neck, upper abdomen and back will help to identify these pathologies.
- Early use of thoracic ultrasound as part of the extended focused assessment with sonography for trauma (eFAST) will pick up cardiac tamponade or massive haemothorax and is more sensitive in picking up pneumothorax than CXR in the supine patient. It can also give a crude, but useful appreciation of fluid status by looking at the IVC.

Tension pneumothorax

- Suspect if: thoracic trauma, hypotension, tachycardia and respiratory distress.
- Signs: reduced air entry and chest wall movement on the affected side with resonant percussion note. Tracheal deviation is a late and inconsistent finding.
- Treatment: decompression by lateral thoracostomy in the 4th or 5th intercostal space (in pregnant patients, the 4th intercostal space is preferred), anterior to the mid-axillary line. This is followed by chest tube insertion. Following thoracostomy, an audible rush of air may be heard and accompany prompt improvement in physiology.
- Needle decompression in the 2nd intercostal space, mid-clavicular line, should be considered a technique of last resort if no one is available to perform tube thoracostomy.

Open pneumothorax

An open pneumothorax should be treated by covering the exterior wound with a non-occlusive, one-way valve dressing, such as an Asherman Chest Seal™, followed by a chest drain inserted at a separate site on the same side of the chest.

Massive haemothorax

- Defined as 1000–1500mL of blood in the thoracic cavity in an adult. It causes both hypovolaemia and impaired ventilation.
- It usually occurs 2° to a laceration of the intercostal artery or vein and less commonly from a mediastinal vessel.
- Signs: reduced chest wall movement and stony dull percussion can be supported by thoracic ultrasound which reveals a large collection of blood above the diaphragm.
- Immediate treatment includes:
  - High-flow O_2
  - Large-bore IV access × 2
  - Chest drain insertion
  - Blood product resuscitation via a rapid infusor device (see ➔ p. 460). Start the transfusion as the haemothorax is released.
- Caution is needed with chest drain insertion as this procedure may dislodge a clot and cause further active bleeding.
- Ongoing drainage of blood from the intercostal drain of 100–200mL/h over the subsequent hours will indicate the need for thoracotomy or interventional radiology.

Flail chest

- A flail segment occurs when two or more adjacent ribs are fractured in two or more places.
- Patients will present with pain and, if conscious, shallow and painful respiration.
- Signs: paradoxical movement of the chest wall may not always be present (some flails are only seen radiologically), but there will be tenderness, bruising, crepitus and underlying lung contusion ± pneumothorax.
- The principles of treatment are as per pulmonary contusion (see ➔ pp. 995–6.
- Rib fixation within 3–5d of injury has demonstrated shorter length of hospital stay and better analgesic outcomes.[8]

Airway disruption

- Patients with major airway disruption will often asphyxiate at the scene. Survivors may present with severe surgical emphysema, pneumothorax, haemothorax, pneumomediastinum, pneumopericardium and pneumoperitoneum. These injuries are often the consequence of high-energy transfer and shear forces.
- Diagnosis is often made by the anaesthetist when trying to manage blood in the airway, difficult intubation and, once intubated, a major air leak.
- Fibreoptic bronchoscopy or, if stable enough, CT are diagnostic.
- Depending on the level of disruption, management may be surgical or, for some small bronchopleural fistulae, conservative.

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Cardiac tamponade

- Cardiac tamponade should be considered in any trauma patient who has sustained a penetrating injury to the chest, neck or upper abdomen. Cardiac tamponade can also occur in blunt trauma, especially in patients taking oral anticoagulants, but this is rare.
- Beck's triad (hypotension, distended neck veins and muffled heart sounds) is unreliable, especially in a noisy ED. Thoracic ultrasound will demonstrate diastolic RV collapse and blood in the pericardial space. This is diagnostic of cardiac tamponade.
- The initial management of cardiac tamponade involves improving preload through volume resuscitation (blood products), while the TTL arranges definitive surgical intervention.
- If the patient deteriorates in the ED despite this, a resuscitative bilateral anterior ('clamshell') thoracotomy and pericardiotomy with repair of the myocardium should be undertaken (see ➔ pp. 1010–11).

Circulation and shock

During the 1° survey, always suspect and actively look for haemorrhage and shock. Place pregnant patients in the left lateral position or manually displace the uterus to the left. Shock is defined as inadequate delivery of oxygenated blood to tissues. Causes are shown in Table 37.2.

- Suspicion of circulatory compromise may come from the pre-alert and mechanism of injury, while recognition of shock should begin at the rapid assessment and handover.
- The TTL will coordinate simultaneous:
  - Circulatory assessment and identification of hypovolaemia
  - Haemostasis
  - Treatment of haemorrhage.
- During the 1° survey, HR, BP, RR, capillary refill time, conscious level and pallor may help to identify patients with shock.
- Traditional ATLS® teaching classifies shock into grades I–IV; however, this has been widely scrutinised, with fewer than half of European ATLS® instructors declaring they would use this tool.[9] Certain drugs, being cold and some pre-existing medical conditions may lead to overestimation of blood loss, while the tool underestimates blood loss in children, pregnancy, blunt trauma and athletes.[9]
- A shock index (HR/systolic BP) of >1 can help identify shock in patients with apparently relatively normal physiology.[10]

Table 37.2 Shock

Type of shock	Example causes
Hypovolaemic	Haemorrhage, burns, interstitial losses, dehydration
Cardiogenic	1° myocardial event, including injury and ischaemia
Obstructive	Cardiac tamponade, tension pneumothorax, massive PE
Distributive	Neurogenic, usually caused by a spinal injury above T6
	Septic, due to systemic inflammatory response
	Anaphylactic, type 1 hypersensitivity reaction

- Bedside use of ultrasound may help identify the presence of blood and/or the cause of shock (e.g. pericardial fluid, an empty IVC, blood in the abdomen or chest).
- Control of haemorrhage begins with direct pressure, indirect pressure, tourniquets, pelvic and long bone splints.
- Large-bore IV (14 or 16G), IO or central access is attained in major trauma patients as early as possible.
- Send blood samples for: venous blood gas (lactate), FBC, G&S or X-match, electrolytes, coagulation profile (TEG® or ROTEM® if available). POCT can be extremely useful.
- If significant bleeding is suspected or evident, tranexamic acid is given within 3h of injury (1g over 10min), if not already given by the prehospital team, followed by 1g over 8h.
- If the patient is known to be on anticoagulants with ongoing bleeding, use of factor concentrates or reversal agents may be indicated.
- Major trauma patients are at risk of the 'lethal triad' of acidosis, coagulopathy and hypothermia. Pay attention to keeping the patient warm—measure temperature, limit exposure to that which is required for assessment/procedures and use active warming and warmed blood products.

Resuscitation goals

- The goals of resuscitation are to optimise tissue oxygenation (Box 37.3).[11,12] Acute shocked trauma patients are in a dynamic situation and should be reassessed regularly.
- Patients with haemorrhagic shock will respond in one of three ways to blood product administration:
  - Improve
  - Transiently improve, then deteriorate, requiring further fluid resuscitation
  - Not improve.
- Practically, until major bleeding has been stopped, volume resuscitation should be titrated to maintain palpable central pulses,[8] systolic BP of 80–90mmHg, MAP of 50–60mmHg[3] and diastolic pressures of 25–35mmHg (to achieve coronary perfusion).[1] Conscious level is a good indicator of cerebral perfusion in awake patients. Young, healthy patients may tolerate a lower BP, while elderly patients may need a higher BP—this is up to the clinical judgement of an experienced anaesthetist/TTL on an individual basis[13] (see ➲ p. 1026).
  (See ➲ p. 1020 for paediatric parameters.)
- In the complex patient with traumatic brain or spinal cord injuries and ongoing haemorrhage, the BP target depends on the dominant condition. If haemorrhagic shock is the predominant problem, restrictive volume resuscitation should be employed. If traumatic brain injury is the predominant issue, a less restrictive approach, targeting SBP of 100–110mmHg and MAP of ≥80mmHg, should be used to maintain cerebral perfusion prior to surgery.[3,11,14,15]
- Patients who do not have a sustained improvement require further blood product resuscitation and definitive management of their haemorrhage. This can be achieved by surgery (see ➲ pp. 982–3) or interventional radiology (see ➲ p. 824) with ongoing correction of coagulopathy.

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- If the source of bleeding is unknown, the TTL, in conjunction with the trauma/general surgeon, must decide if the patient will go straight to theatre or undergo CT first. This will depend on the stability of the patient, but unless lifesaving surgery is required immediately, patients generally benefit from CT scans.[16]
- Deterioration in the resuscitation room despite fluid resuscitation may lead to traumatic cardiac arrest (see ➲ pp. 1009–11).

Box 37.3 Goals of resuscitation

- Systolic BP 80–100mmHg (head-injured patient: MAP >80mmHg) (see caveats above)
- HR <120bpm
- SpO_2 >95%
- Hb 70–100g/L
- INR <1.6
- Lactate <4mmol/L
- Base excess > –6
- Fibrinogen >1.5g/L
- Platelets >50 × 10^9/L (aim for >75 × 10^9/L)
- pH >7.2
- Core temperature >35.0°C
- Ionised calcium >1.1mmol/L

Source: data from Patient Blood Management Guidelines: Module 1 Critical Bleeding Massive Transfusion (2009) National Blood Authority Australia Updated 2011.

Fluid administration in major trauma

- The use of crystalloid in traumatic haemorrhage is limited to prehospital teams that do not carry blood and patients who will not receive blood products (e.g. Jehovah's Witnesses).[3]
- For full guidance on haemostatic resuscitation, see ➲ pp. 983–4.
- Initial balanced resuscitation, using warmed PRBCs/FFP/platelets in a 1:1:1 ratio, is recommended.[17]
- In the UK, all hospitals should have a massive transfusion protocol (MTP), which can be initiated based on local thresholds, for significant haemorrhage. The MTP alerts the transfusion laboratory of a major haemorrhage and guarantees the delivery of certain blood products, which may vary between hospitals. Typically, there is an initial pack containing PRBCs and FFP and a 2nd pack that includes PRBCs, FFP, cryoprecipitate (or fibrinogen concentrate) and platelets.
- See Box 37.4 for initial dosage of blood products.[18] Further administration is guided by laboratory testing or POCT, e.g. ABG and TEG® or ROTEM®.
- Consider calcium chloride after 2–4 units of blood.

Disability

- Assess pupils for size, equality and reactivity to light.
- A relative afferent pupillary defect with facial trauma may alert the team to a retro-orbital haematoma which needs immediate decompression with lateral canthectomy.

- Formal assessment of GCS (see ➲ Table 37.4, p. 990) and gross motor function bilaterally will alert the TTL to major spinal and intracranial injuries. This needs to be accomplished prior to intubation.
- If not already administered, tranexamic acid should be given in isolated head-injured patients with reduced GCS (9–12) prior to CT brain if within 3h of the injury.[19]
- Ensure that blood glucose level and analgesia have been addressed by this point.

Box 37.4 Initial dosage of blood products

Hb <70g/L	Rate of administration guided by rate of blood loss. Aim for 70–90g/L (>80g/L if cardiac history)
INR >1.5	Give FFP 15–20mL/kg
Fibrinogen <1.0g/L	Give two packs (2 × 5 unit pools)
Platelets <50 × 10^9/L	Give one adult therapeutic dose

Source: data from Hunt B *et al.* (2015) A practical guideline for the haematological management of major haemorrhage *British J Haematology* **170**(6), 788–803.

Exposure

- It is important to expose the patient completely and inspect the axillae, back and groin for penetrating injuries (a 'stab check'), while making attempts to avoid hypothermia.
- Often a log roll is not performed prior to a trauma CT (if indicated), but the TTL should have an appreciation of all major obvious wounds.

Imaging

(See ➲ Table 37.3 and Box 37.5.)

- Imaging should aid in decision-making and the early planning phase of the patient journey, as well as provide diagnoses.
- X-rays of the chest and pelvis may be performed as part of the 1° survey.
- Ultrasound can be performed quickly in the ED to identify free fluid (blood) in the perihepatic and perisplenic regions indicating abdominal haemorrhage. eFAST is a structured assessment looking for:
  - Blood in the perihepatic, perisplenic, pericardial and pelvic areas
  - Air (pneumothoraces) via anterior thoracic windows.
- Ultrasound can be used to identify blood in the abdomen but should not be used to rule out the presence of blood, as there is significant operator dependency and, even in experienced hands, retroperitoneal and small volumes of blood can be missed.[20] Even if no fluid is identified, this does not rule out other abdominal injuries which may require surgical intervention.

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- eFAST has become controversial in some units where expedient trauma CT is preferred as the initial investigation of choice.
- A trauma CT (see ➔ p. 819) images the head, neck, chest, abdomen and pelvis. It is often prioritised after the 1° survey in adults. The aim is to gain as much information of the injuries as quickly as possible. The injection of contrast allows for greater diagnostic information. The Camp Bastion Protocol for trauma CT is commonly used. (See ➔ pp. 1022–3 for guidance in paediatric patients.)
- The Primary Assessment Report[21] is a method by which the radiologist can rapidly provide information that will change immediate management and disposition of the patient. A formal report will be issued subsequently when the radiologist has fully reviewed the films.

Table 37.3 Imaging

	Ultrasound	CT scan
Indication	Screening for free fluid in unstable patients	Diagnosis of organ injury in haemodynamically stable patients
Advantages	Fast, sensitivity 83–87%, can detect free fluid and some solid organ injury	Most specific for injury (92–98%), will also demonstrate perforation
Dis-advantages	Operator-dependent; misses diaphragm, bowel and some pancreatic injuries May delay CT	Takes time and involves transfer away from resus room

Log roll

If the patient is stable, a log roll may be appropriate prior to moving on to the 2° survey. The purpose of the log roll is to ensure the back has been visualised and if a full trauma scan has not been done, the TTL will assess the spine for tenderness and sensation.

The secondary survey

The 2° survey requires a systematic head-to-toe examination of the patient. It will also include management of peripheral injuries, an ECG, complete neurological examination and, if required, a formal orthopaedic review, a urinary catheter and American Spinal Injury Association (ASIA) chart completion. The 2° survey often occurs outside the ED in major trauma, after resuscitation, surgery or transfer to critical care, and formal documentation of this step, when it occurs, is important.

Box 37.5 Summary of NICE guidance on imaging in major trauma[3]

- **Patients with suspected chest trauma or suspected haemorrhage**: perform imaging urgently; images should be interpreted immediately by a health care professional with training and skills in this area.
- **Patients with suspected haemorrhage and haemodynamic instability who are not responding to volume resuscitation**: limit diagnostic imaging (CXR and pelvis X-ray or FAST) to the minimum needed to direct intervention.
- **Patients with suspected haemorrhage who are responding to resuscitation or have normal haemodynamic status**: consider immediate CT.
- Use **whole-body CT** in adults (≥16y) with blunt major trauma and suspected multiple injuries.
- **FAST in major trauma patients**:
  - Do not use FAST or other diagnostic imaging before immediate CT or as a screening modality to determine the need for CT.
  - A negative FAST does not exclude intraperitoneal or retroperitoneal haemorrhage.

FAST, focused assessment with sonography for trauma.

Source: data from National Institute for Health and Care Excellence (2016) Major trauma: assessment and initial management NICE guideline 39 https://www.nice.org.uk/guidance/ng39

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Damage control resuscitation

(See also ➲ pp. 460–1.)

Overview

- Haemorrhage is the leading cause of preventable death in the first 24h after trauma, accounting for 50% of deaths.[15]
- Damage control resuscitation (DCR) is a treatment strategy that aims to preserve life in bleeding trauma patients by stopping haemorrhage and proactively countering the 'lethal triad' of hypothermia, acidaemia and coagulopathy.[22]
- The key elements of DCR include:
  - Haemorrhage control and restoration of perfusion via damage control surgery (DCS)
  - Haemostatic resuscitation to prevent and treat trauma-induced coagulopathy (TIC)
  - Permissive hypotension.
- DCR consists of four phases:
  - Phase 0: recognition, rapid transport to hospital/theatre and start of resuscitation
  - Phase 1: DCS and continued DCR
  - Phase 2: ICU-based physiological and biochemical stabilisation
  - Phase 3: return to theatre for definitive repair of injuries.

Damage control surgery

- DCS involves an abbreviated operation to control life-threatening injuries and restore physiology, rather than correct anatomy. Initially described by Rotondo,[23] the goal is to preserve life.
- DCS may be essential in non-trauma centres to stabilise a patient prior to transfer to a tertiary centre.
- The decision to use DCS should be made early, but not lightly. Timely selection of appropriate patients for DCS ensures maximal benefit; the later DCS is applied, the less successful the outcome. Around 10% of major trauma patients require DCS, but the risk of morbidity and mortality is significantly higher and there are resource implications.[22] If the patient has normal physiology or responds promptly to resuscitation, then definitive repair should be attempted. Surgery should be matched to the physiology—if a patient deteriorates during their procedure, DCS should be utilised.
- There is no single threshold for DCS, rather a 'global' view should be used to select patients. When the severity of surgical bleeding exceeds the patient's capacity to respond, DCS should be considered. Indications of physiological exhaustion include:[11,21,22]
  - Preselected, based on mechanism of injury, e.g. penetrating trauma or high-energy blunt trauma
  - Metabolic compromise: pH <7.25, base excess < –6, lactate >2.5
  - Massive transfusion, i.e. >10 units of PRBCs
  - Coagulopathy on POCT/lab results or clinical evidence
  - Hypothermia <34.0–35.0°C.
- Once the need for DCS has been identified, the patient should be rapidly transported to theatre. Investigations should only help identify which body cavity to open—CXR, pelvic X-ray and eFAST.

- Typical DCS procedures include resuscitative laparotomy, resuscitative thoracotomy, pelvic packing, junctional surgery (i.e. neck/groins/axillae) and external fixation of fractures. More than 1 surgical team may operate simultaneously.
- The patient should be positioned and the skin prepared to enable extension of the initial incision into another body cavity or junctional zone, e.g. in the crucifix position.
- DCS consists of four key principles:
  - Haemorrhage control: using packs, vessel ligation, suturing, organ excision (e.g. splenectomy, nephrectomy, linear resection of lung tissue), shunts and haemostatic adjuncts
  - Control contamination: debridement and removal of contaminants followed by closure of bowel injuries—small injuries may be simply sutured, while larger injuries should be stapled and the bowel ends left discontinuous
  - Protect the patient from further injury
  - Temporary closure.
- DCS procedures should last <60–90min. Resuscitation and correction of physiology should continue in ICU thereafter.

Haemostatic resuscitation

- Haemostatic resuscitation[11,17,21,22,24] is the aggressive treatment of TIC.
- TIC is caused by multiple factors and is associated with ↑ morbidity and mortality:[21]
  - Hypoperfusion, resulting from the injury itself, causes an endogenous coagulopathy.
  - Formation of clots leads to a consumptive coagulopathy.
  - Hypothermia and acidaemia (together with TIC make the 'lethal triad') exacerbate dysfunction further.
  - Stored blood is physiologically imperfect, particularly so if >22d old. Even if you provide excellent resuscitation, you will be providing resuscitation with fluid that has inferior clotting and O_2-carrying ability to the patient's own.
- Transfusion should follow the local major haemorrhage/massive transfusion protocol. The major haemorrhage protocol should be initiated to alert the transfusion laboratory and portering services, in addition to clinical teams, that significant haemorrhage is expected. Typical major haemorrhage protocol triggers include blood given by prehospital services, at TTL discretion based on pre-alert information, blood loss of ≥150mL/min or actual/anticipated blood loss of ≥30% of circulating volume in 3h (~1500mL). PRBCs and blood products are often issued as emergency 'packs', which enable abbreviated ordering of the products. The 'packs' are virtual and may not arrive together, depending on what is immediately available.
- 'Shock/trauma packs' often contain 4 PRBCs and 4 FFP (prethawed, if possible). O+ blood can be given to ♂ patients. Further 'packs' may contain similar products and quantities but also include cryoprecipitate (or fibrinogen concentrate) and platelets.
- **Initial transfusion ratios** target the empirical optimum transfusion ratio of 1:1:1 PRBCs to FFP to platelets, which was demonstrated to improve

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outcomes in the PROPPR trial.[17] A 'pool of platelets' contains 4–6 units, so giving one pool every 4–6 PRBCs maintains the ratio balance.

- Use a rapid transfusor device to warm and deliver all blood products, apart from platelets. The rate of transfusion should be guided by an estimation of the deficit and the rate of ongoing blood loss.
- Tranexamic acid (1g or 15mg/kg in children) should be given IV over 10min as soon as possible within the first 3h after injury to counter hyperfibrinolysis. A 2nd dose of 1g should be infused over the next 8h.
- Calcium chloride 10%: give 10mL approximately every 4 units of PRBCs, or if ionised calcium ≤1.0mmol/L, to treat hypocalcaemia from consumption and citrate chelation.
- Early and regular blood gas analysis and viscoelastic assays (TEG®/ROTEM®) should be used, in addition to clinical information, to target further transfusion to the individual patient's need. Base excess, lactate, pH, viscoelastic assay values and rate of ongoing blood loss guide the resuscitation. In acute haemorrhage, Hb is unreliable, but targeting 70–100g/L and platelets >50 × 10^9/L is recommended.[11,18] ⚠ Do not wait until Hb has dropped to these values before starting transfusion, as you will struggle to catch up.
- **Switch to type-specific or X-matched blood** at the earliest opportunity.
- Replace fibrinogen. Target TEG®/ROTEM® values or Clauss fibrinogen levels (>2g/L preferably, and at least >1.5g/L). Two pools of cryoprecipitate will provide ~1g of fibrinogen. Up to 3–4g may be needed initially. Alternatively, use fibrinogen concentrate, which does not need to be thawed.
- Correct hyperkalaemia: 50mL of 50% glucose and 15 units of insulin infusion over 20–30min to maintain K^+ 3.5–4.5mmol/L.
- **Crystalloid and colloid fluids should be avoided** to prevent worsening the dilutional coagulopathy, unless there is no other option. Small volumes, e.g. 250mL boluses, should be used.
- **Reverse anticoagulated patients**: PCC, e.g. Octaplex®, or other appropriate reversal agents.
- **Further management**: PCC, recombinant factor VIIa and warm whole blood (if available) may be considered for resistant coagulopathy, in conjunction with haematology advice.

Permissive hypotension

- Permissive hypotension is a temporarily tolerated passive process until the bleeding is stopped definitively by surgery or interventional radiology.[21]
- There is little high-quality evidence for permissive hypotension. But studies have shown that attempting to normalise BP in active haemorrhage with fluids or vasopressors is counterproductive, creating a cycle of bleeding from ↑ hydrostatic pressure followed by requirement for further fluids/vasopressors.[21,25,26] However, there is ongoing debate about the validity of this research, as most was conducted before modern balanced transfusion and DCR-based approaches.
- Practically, until major bleeding has been stopped, volume resuscitation should be titrated to maintain palpable central pulses,[3] systolic BP of 80–90mmHg, MAP of 50–60mmHg[11] and diastolic pressures of 25–35mmHg (to achieve coronary perfusion).[21] Conscious level is a

good indicator of cerebral perfusion in awake patients. A lower BP in a young, healthy patient who is cerebrating may be acceptable, but this is up to the clinical judgement of an experienced anaesthetist/TTL on an individual basis. Elderly patients may need a higher systolic pressure (see ➔ p. 1026).

- In complex patients with both haemorrhagic shock and traumatic brain injury (GCS ≤8)/spinal cord injury, the BP targets depend on the dominant condition. If haemorrhagic shock is the predominant problem, restrictive volume resuscitation should be employed. If traumatic brain injury is the predominant issue, a less restrictive approach, targeting systolic BP of 100–110mmHg and MAP of ≥80mmHg, should be used to maintain cerebral perfusion.[3,11,14,15]
- Transient use of vasopressors, targeting the above values, can be considered in life-threatening hypotension while volume replacement is ongoing.[11]
- Note, the above BP values are population-based rules of thumb. Elderly patients may need higher MAP and systolic BP.
- When haemorrhage is controlled, carefully resuscitate to more normotensive BPs to optimise organ perfusion. The shorter the period of permissive hypotension, the better for organ perfusion and morbidity. The concept of 'hybrid resuscitation'[27] acknowledges the need to reduce the period of hypotension, partially restoring the BP after 1h, even if definitive haemostasis is not achieved. This provides a greater degree of tissue perfusion prior to complete surgical haemostasis.

Additional considerations

Vascular access

- Two 14–16G cannulae in a large peripheral vein.
- Large-bore central access 8.5Fr (PAFC introducer/Arrow® MAC™) into the subclavian or internal jugular vein. Avoid insertion distal to a source of haemorrhage, i.e. avoid the femoral vein in abdominal trauma.
- Resuscitation, theatre transfers and surgery should not be delayed for central or arterial lines (there is no such thing as a 'quick' line!). Position the patient to allow insertion once surgery has started, e.g. in the crucifix position.
- Temporary vascular access can be achieved by IO cannulae in the humeral heads, if necessary.

Hypothermia management

- Target a core temperature >36°C.
- Monitor temperature with an orally placed oesophageal temperature probe.
- Warm all blood products/fluids.
- Use both underbody and overbody forced air warming blankets and cover other exposed areas with Gamgee/blankets.
- Warm humidified breathing circuit.

Human factors

- Providing DCR for severely injured major trauma patients is challenging. Diagnoses and multiple interventions need to occur simultaneously. Senior clinicians should be present throughout.

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- Clear communication, calm leadership, active followership and situational awareness from team members are key.
- Roles should be assigned, including: team and subteam leader(s), blood and transfusion monitors and scribe.
- Regular communication between surgical teams and anaesthetists is recommended every 15–20min during surgery. 'SNAP-Chat'[28] is a mnemonic that aids covering:
  - **S**urgical progress/systolic BP
  - **N**umber of blood products
  - **A**BG, coagulation and temperature
  - **P**lan until next interval.
- A hot debrief when the patient has left theatre is extremely useful both as a learning tool and to support staff wellbeing. Further debriefing and support may be needed after on-table deaths, extreme injuries and mass casualty events.

Damage control anaesthesia

(See ➲ pp. 972–4.)

- **Preoxygenation** should start using 15L/min via a non-rebreathing face mask while en route to theatre if the patient is awake.
- In theatre, if inducing for lifesaving haemorrhage control surgery, the patient should have their skin prepared and draped and the surgical team ready to put knife to skin immediately after intubation.
- A primed rapid infusor should be connected to a large-bore cannula giving volume to compensate for anaesthesia and release of any tamponade.
- DLTs are not recommended, even for thoracic cases, due to the additional complexity (and lack of familiarity for most anaesthetists) in an already difficult situation. A bronchial blocker can be used, or a single lumen tube advanced endobronchially, if required.
- **Ventilation**: initially, limit intrathoracic pressure to avoid further compromising a shocked circulation. Suggested starting parameters are FiO_2 100%, RR 10 breaths/min, V_T 5–6mL/kg and PEEP 0–4cmH_2O, aiming for oxygenation. As the circulating volume expands, minute ventilation and PEEP can be ↑ and FiO_2 reduced to more normal levels.
- **Maintenance of anaesthesia**: volatile maintenance is recommended over TIVA to avoid additional workload in changing syringes. Target a MAC of 0.4–0.6, with 50-microgram fentanyl boluses (2–3mg total dose for case) to provide simple, CVS-stable anaesthesia. Fentanyl provides a degree of vasodilation to aid the replacement of lost circulating fluid and lactate clearance. Larger doses can cause haemodynamic instability.
- **Antibiotics**: determined by local policy, dependent on the incision.
- **Analgesia**: fentanyl boluses as above. Further small doses of ketamine and paracetamol may be used as appropriate. Regional anaesthesia may be considered but is often impractical.
- **Constantly re-evaluate**: when bleeding is controlled, slow down the rate of blood product infusion to avoid fluid-overloading the patient.
- **Intraoperative problems**:
  - Unexplained ↓ BP and ↑ HR: consider hypovolaemia, pneumothorax, pericardial tamponade or retroperitoneal vessel injury.
  - Unexplained hypoxia and rise in inflation pressures: consider tension pneumothorax.

- Unexplained ↑ BP: consider pain, ↑ ICP (search for associated neurological signs), or rarely traumatic disruption of the thoracic aorta (causing a pseudocoarctation effect).

Tips

- If pH <7.1, consider sodium bicarbonate.
- Transthoracic echocardiography can be useful to assess the fluid status of the heart chambers for those trained in its use.
- Pass an OGT to empty the stomach prior to extubation if this is intended.
- Although patients initially may be hypocoagulable, they often become progressively hypercoagulable. Veno-thromboprophylaxis should be instigated early when warranted.

Stages II and III: restoration of normality and definitive repair

- The ICU team will aim to reverse the physiological and biochemical effects of injury and hypotension. They will use invasive monitoring and POCT to optimise blood flow, continue correction of coagulopathy and rewarm the patient. A tertiary survey will occur and any further imaging be undertaken (if the patient is 'stable' after surgery, a CT scan can be completed en route to ICU).
- Restoration of normal physiology may take up to 48h, though this may occur quicker. Some patients may require an unplanned return to theatre to manage further bleeding, a shunt occlusion or abdominal compartment syndrome.
- Definitive surgical repair may require several trips to theatre, particularly in the case of polytrauma patients. Procedures should be performed 48–72h after the initial surgery when the patient's physiology has been corrected; the patient should be normothermic and have normal clotting and haemodynamics.
- Procedures should be planned such that appropriate specialist surgical teams are prepared. It is possible, and more efficient, if surgical teams from different specialties work simultaneously to achieve >1 procedure per trip to theatre. However, major trauma patients at 48–72h post-injury are still finely balanced physiologically and can still deteriorate if surgery is prolonged. Plans should be in place for staging surgery.
- Be prepared for further blood loss and fluid losses. Invasive monitoring, frequent POCT and correction with blood, fluids and inotropes are appropriate.
- ALI: trauma patients are at high risk of hypoxia from ALI. This may be 2° to direct pulmonary contusion or due to fat embolism from orthopaedic injuries (see ➔ p. 614). Lung-protective ventilation and advanced ventilatory modes may be required to maintain appropriate oxygenation.

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Pregnancy and trauma

Trauma affects 7% of pregnancies,[29] typically road traffic collisions, falls and domestic violence. Standard assessment and management principles should be followed as optimal resuscitation of the mother also benefits the fetus.[30]

Considerations in the pregnant woman

- The anatomical and physiological changes of pregnancy (see pp. 838–9) mean injury patterns may differ, depending on the stage of pregnancy, and the woman's response can be altered.[29,30,31]
- After 12w gestation, the uterus expands out of the pelvis and displaces intra-abdominal organs which may be relatively protected.
- Later in pregnancy, the raised diaphragm means 'chest trauma' could also involve abdominal structures.
- Injuries to the uterus can lead to placental abruption, preterm labour (most commonly), premature rupture of membranes and uterine rupture.
- Blunt trauma is more likely to cause placental abruption, while penetrating abdominal trauma is more likely to involve the uterus itself (which provides some protection to abdominal viscera). The gravid uterus can complicate laparotomy (see p. 893).
- Pregnant women have a larger circulating blood volume and compensate for bleeding well; fetal distress (on cardiotocography) may be the 1st sign of physiological instability.

Adaptations to the initial assessment and management

- Visibly pregnant women (typically >20w gestation) should be placed in a 20° left lateral tilt or the uterus displaced.
- Perform a pelvic examination; take an obstetric history, and obtain a cardiotocography trace.
- X-rays and CT scans should not be withheld if indicated, but shield the fetus where possible.
- Additional teams who should attend, in addition to the standard trauma team, include obstetrics/midwifery and neonatal.
- Ensure VTE prevention is prescribed.
- Test for fetomaternal haemorrhage (affects ~30%) with a Kleihauer test and use anti-D immunoglobulin in all rhesus-negative women.
- Chest drains, if required, should be inserted 1–2 intercostal spaces above normal.
- A perimortem CS may improve maternal resuscitation if started within 4min of the cardiac arrest starting (see p. 897).

Head and traumatic brain injury

Head injury is defined as trauma to the cranium, which may include a traumatic brain injury. Facial injury is also discussed here.

Head injuries may not be suspected at scene, so head-injured patients often present to non-specialist centres and rely on 2° transfer to a neurosurgical centre. In the UK, trauma networks have helped to ensure patients with moderate and severe head trauma are transferred directly to an MTC with a neurosurgical facility.

Head injuries and traumatic brain injury are often graded by GCS (see ➲ Table 37.4, p. 990) into mild (GCS 13–14), moderate (GCS 9–12) and severe (GCS ≤8). There is an increasing probability of poor outcome with decreasing GCS in a stepwise manner. Overall, 30d mortality following traumatic brain injury is ~20%. If the initial GCS is reliably obtained and not tainted by medications or intubation, ~20% of the patients with GCS ≤5 will survive and <10% of these will have a functional survival.[32] Age is a strong factor influencing mortality and morbidity, with children doing better than adults and adults >60y having significantly worse outcomes.[32]

There are various ways to define brain injuries. In this text, they are categorised as 1° or 2° brain injury. 1° injury occurs at the time of impact and 2° brain injury refers to the changes that occur hours to days after the 1° event.

Primary brain injury

Occurs at the time of the traumatic event and is associated with cranial fracture, parenchymal contusion, axonal shearing/laceration and vascular injury. It can be diffuse or focal.

Secondary brain injury

Occurs after the traumatic event due to the pathophysiological consequences of the 1° injury. It can be exacerbated by reduced cerebral perfusion, ischaemia (global or local) and ↑ metabolic requirements 2° to:

- Hypoxia
- Hypotension
- Intracranial hypertension
- Haemorrhage (intraparenchymal, subdural and extradural)
- Seizures.

The Glasgow Coma Scale and AVPU scores

Calculating the GCS (Table 37.4) accurately is important for planning care and aiding prognostication after head trauma and traumatic brain injury. For paediatric patients, see Table 37.5 or use AVPU. Reliable calculation must occur prior to intubation and without the effect of medications, which is not always possible if therapy has been started prehospital.

Management

Medical therapy cannot affect the impact of 1° traumatic brain injury but can influence the degree of the 2° insult, potentially salvaging watershed areas. The principles of managing head-injured patients therefore are to optimise cerebral perfusion and oxygenation and reduce ongoing bleeding.

During the 1° survey, management of the airway, control of ventilation and oxygenation and maintenance of BP are key components to reducing 2° injury. Anaesthesia to facilitate diagnostic and therapeutic interventions in the agitated patient with reduced GCS may also be essential.

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Table 37.4 Glasgow Coma Scale

	Response	Score
Best motor response	Obeys commands	6
	Localises pain	5
	Normal flexion withdrawal (stimulus to supraorbital notch)	4
	Abnormal flexion to pain	3
	Extension to pain	2
	No motor response	1
Best verbal response	Orientated	5
	Confused	4
	Inappropriate words	3
	Inarticulate sounds	2
	No verbal response	1
Eye opening	Eyes open	4
	Eyes open to speech	3
	Eyes open to pain	2
	No eye opening	1

Reprinted from *The Lancet*, **304**:7872, Teasdale G, Jennett B, Assessment of coma and impaired consciousness, pp. 81–84, Copyright (1974), with permission from Elsevier.

Table 37.5 Modification of GCS for children under 5y

	Response	Score
Best motor response	Obeys commands (>2y)	6
	Localises to pain (<2y)	5
	Normal flexion to pain (>6mo)	4
	Abnormal flexion to pain	3
	Extension to pain	2
	No motor response	1
Best verbal response	Orientated (>5y)	5
	Words (>1y)	4
	Vocal sounds (>6mo)	3
	Cries (<6mo)	2
	No verbal response	1
Eye opening	Eyes open	4
	Eyes open to speech	3
	Eyes open to pain	2
	No eye opening	1

Using this scoring system, the maximum GCS is 9 at 0–6mo, 11 at 6–12mo, 13 at 1–2y and 14 at 2–5y.

Source: data from Reilly PL, *et al.*, Assessing the conscious level in infants and young children: a paediatric version of the Glasgow Coma Scale, *Childs Nerv Syst*, 1988:**4**(1):30–3.

Airway

Patients with reduced GCS may have impaired laryngeal reflexes and be unable to protect their airway. Indications for intubation are as per major trauma (Box 37.2) but, in addition, may include:

- Spontaneous hyperventilation causing $PaCO_2$ <4.0kPa
- To enable CT scan
- Recurrent seizures uncontrolled by simple benzodiazepines.

The ETT should be secured with tape, rather than with ties, to prevent compromising cerebral venous drainage and risk increasing ICP.

Breathing

Adequate oxygenation and ventilation will reduce the risk of 2° brain injury. CBF is affected by extracellular pH which is, in turn, related to $PaCO_2$ (see ➲ p. 559).

Once intubated, initial ventilatory strategies should aim for:

- Normocapnia: $PaCO_2$ 4.5–5.0kPa. Be mindful that $ETCO_2$ will be ~0.5kPa lower than $PaCO_2$[33]—take note of the ABG value. Reducing $PaCO_2$ will result in vasoconstriction and ↓ cerebral perfusion. Hyperventilation to $PaCO_2$ of 4.0kPa (avoid lower) is recommended as a temporising measure to reduce ↑ ICP, but should be avoided, if possible, during the first 24h after injury as CBF is often critically reduced.[34]
- Normoxia: SpO_2 >95% and PaO_2 ≥8kPa. Recent AoA guidance for transfers recommends PaO_2 ≥13kPa.[35]
- V_T of 6–7mL/kg. While lung-protective strategies are preferred, V_T can be ↑, if needed, to prevent hypoxia or hypercapnia.[36,37]
- PEEP ≤5cmH_2O: changes to intrathoracic pressure (PEEP) should be minimised to reduce the effects on venous drainage and MAP, and thus ICP and CBF, respectively.

Circulation

- The injured brain can lose the ability to autoregulate blood flow and is dependent on systemic pressures. ⚠ A single episode of hypotension (systolic BP <90mmHg) confers a doubling of mortality and an increase in morbidity during acute management.
- In isolated head injury, aim for MAP of >90mmHg. Mortality may be reduced and outcomes improved if systolic BP is maintained at:
  - ≥100mmHg in patients 50–69y
  - ≥110mmHg in patients 15–49y and >70y.[34]
- The recommended target for CPP (CPP = MAP – ICP) is 60–70mmHg.[34]
- Trauma patients often present clinical conundrums with opposing treatment strategies for different injuries. Treatment of the most life-threatening injury takes precedence. If a trauma patient has uncontrolled haemorrhage, a systolic BP of 100mmHg is preferred until haemorrhage control. A systolic BP <90mmHg is associated with poor outcomes.[33]
- The correct fluid of resuscitation will depend on other injuries, but avoidance of hyponatraemia is important. If haemorrhagic shock is the dominant condition, continue restrictive volume resuscitation; if traumatic brain injury is the dominant condition, use a less restrictive volume resuscitation approach to maintain cerebral perfusion. Be

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mindful that significant blood loss can occur from isolated scalp wounds and during operative management of isolated head injuries. Be prepared to transfuse these patients.
- In isolated head injury with GCS of 9–12, 1g tranexamic acid should be given if within 3h of injury and any anticoagulants should be reversed promptly.[19]

Disability

The Glasgow Coma Scale, pupillary examination and motor function are key to neurological assessment and prognostication in traumatic brain injury.
- Eyes should be examined for:
  - Signs of orbital trauma
  - Pupillary response to light
  - Pupillary size (>4mm is recommended as a measure of dilated pupil).[34]
- Anaesthetists are well positioned at the trauma call to look in the ears for haemorrhage and CSF indicative of base of skull fracture.
- Analgesia, sedation and adequate NMB are essential in the management of traumatic brain injury to reduce cerebral metabolism and straining. TCI propofol is acceptable for maintenance of anaesthesia.
- Keep the neck in the midline and tilt the bed/ trolley to a 30° head-up position to reduce ICP by improving venous return.

Other considerations

- The use of high-dose barbiturates is only recommended for control of raised ICP refractory to standard medical and surgical treatment and can cause significant haemodynamic instability.[34]
- Post-traumatic seizures are common (up to 12%). Seizure prophylaxis with phenytoin or levetiracetam should be started, dependent on local guidelines.
- Seizures in awake patients are managed as normal but would highlight the possible need for intubation.
- Hyperglycaemia should be avoided.
- Maintain normothermia. There is no evidence supporting early hypothermia.[38] Hyperthermia should be managed aggressively.
- Ensure laxatives are prescribed.

Emergent management of raised ICP

- ICP should be kept <22mmHg. Prior to direct measurement (intraventricular or intraparenchymal), elevated ICP may be identified by clinical signs or at head CT with ventricular and sulcal effacement, compression of basal cisterns and herniation.
- Raised ICP is associated with pupillary and haemodynamic changes. In severe traumatic brain injury, abnormalities of pupillary response or pupil size asymmetries are often associated with neurological deteriorations and are correlated with poor neurological outcome.[39]
- Classically, the Cushing reflex is described as hypertension, bradycardia and apnoea in response to intracranial hypertension. The bradycardia is often preceded by tachycardia.[40]

- Patients who develop signs of worsening raised ICP should be managed immediately (see p. 560). Temporising therapeutic interventions include:
  - Hyperventilation to 4.0kPa
  - Mannitol 0.25g/kg (repeated up to max 1g/kg). Can drop BP
  - Hypertonic sodium chloride 3–6mL/kg of 3% (or 2–6mL/kg of 5%).

Imaging

Head and C-spine CT may form part of the trauma CT. In isolated head injuries, NICE guidelines indicate the cohort of adult patients that should be imaged (Box 37.6).[41]

Surgically significant abnormalities seen on CT should be discussed with a neurosurgical unit.

Regardless of imaging, other reasons for discussing a patient's treatment plan with a neurosurgeon include:

- GCS <8 after initial resuscitation
- Unexplained confusion >4h
- Deterioration in GCS and progressive focal neurology
- A seizure without full recovery
- Definite or suspected penetrating injury
- A CSF leak.

Referral and disposition will depend on local guidelines. Transfer of critically ill patients with traumatic brain injury must be accompanied by a doctor with suitable competencies and experience in brain injury transfer.[35,42]

Box 37.6 Summary of NICE indications for head CT in trauma patients

CT head within 1h of assessment in ED

- GCS <13 on admission; or GCS <15 2h after injury
- Depressed, open or basal skull fracture
- Post-traumatic seizure
- Focal neurological deficit
- Vomiting > once

*CT head within 8h of assessment in ED**

- ≥65y
- History of bleeding disorder
- Dangerous mechanism of injury
- >30min retrograde amnesia
- Patients on anticoagulant treatment, with no other indications, should also undergo a CT head scan within 8h

* If patient has had some loss of consciousness or amnesia since injury, a report should be available within 1h.

Source: data from National Institute for Health and Care Excellence (2019). Head injury: assessment and early management NICE guideline 176. https://www.nice.org.uk/guidance/cg176

Secondary survey

As part of the 2° survey, it is important to check the skull and eyes for other injuries. Give antibiotics if open skull fractures are present. Base of skull fractures, although treated conservatively, need to be highlighted such that NGTs are not inserted.

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Clinical signs of base of skull fracture

- Early signs: haemotympanum, clear fluid and blood from the nose or ear, scleral haemorrhage with no posterior margin.
- Late signs, 12–24h post-injury: bruising over the mastoid (Battle's sign), orbital bruising (raccoon or panda eyes).

Maxillofacial trauma

- Severe intractable bleeding from maxillofacial injuries, although uncommon, can be life-threatening and is usually managed as part of the 1° survey. Managing the airway and ongoing bleeding prior to interventional radiology or surgical repair is essential. Early decision-making, involvement of other specialists (maxillofacial and ENT) and clearly verbalising the airway plan are important.
- Intubation using VL may be difficult due to bleeding. Direct visualisation with high-volume suction kit can aid ETT placement.
- Front of neck access should be anticipated and planned for (see p. 381).
- Bleeding points are often hard to localise. Severe haemorrhage may require packing, manual reduction of fractures, balloon tamponade, angiography with embolisation or direct external carotid artery ligation to obtain haemostasis. In intubated patients, mid-facial haemorrhage can be reduced by splinting and stabilising the mid-facial fractures. Providing a line of fixation from the clavicles, through the rigid cervical collar, bite blocks and nasal balloons, allows a degree of haemostasis to be achieved.[43]

Thoracic injury

In the prehospital environment, severe thoracic injury accounts for 25% of trauma deaths.[44] Thoracic trauma can occur after:

- Direct injury: e.g. seat belts, intrusion of vehicle wreckage, penetrating trauma
- Rapid deceleration, resulting in shearing forces that may lead to traumatic aortic and lung injuries.

The immediate life-threatening conditions (tension pneumothorax, open pneumothorax, massive haemothorax, flail chest, airway disruption/obstruction and cardiac tamponade) need to be identified and treated rapidly during the 1° survey; these are addressed on ➲ pp. 974–6. There are six further potentially life-threatening injuries, which can occur after thoracic trauma:

- Pulmonary contusion and rib fractures
- Cardiac contusion
- Traumatic aortic injury
- Oesophageal injury
- Ruptured diaphragm
- Rupture of the tracheobronchial tree.

Pulmonary contusion and rib fractures

Pulmonary contusions and rib fractures are common following blunt thoracic trauma and are associated with a high risk of respiratory complications. Mechanisms include impact, deceleration and seat belt injuries. Examination may reveal external bruising and focal points of tenderness, indicating likely underlying rib fractures. Young adults and children have ↑ chest wall compliance which may result in significant pulmonary contusion without rib fractures, while elderly patients may suffer significant rib injuries after a simple fall; these can be associated with considerable morbidity and mortality.[45]

Pulmonary contusions

The earliest indicator of pulmonary contusion is hypoxia.

- CXR may show patchy infiltrates over the affected area but is often initially normal. It may, however, reveal other pathology which would raise the suspicion of pulmonary contusion, e.g. rib fractures, flail segment, haemo- or pneumothorax. Less than 50% of pulmonary contusions are apparent on admission, compared to 92% at 24h.[46]
- Typically, hypoxia from lung contusion progresses over 24–48h, associated with reduction in lung compliance and worsening respiratory distress.
- If these patients are ventilated, they may require higher levels of PEEP to maintain oxygenation. Lung-protective strategies and careful fluid resuscitation are also important to avoid worsening lung injury.

Rib fractures

- Rib fractures (see ➲ p. 553) are painful; they reduce chest wall excursion, leading to hypoventilation, and impair coughing ability, leading to sputum retention. These combine to cause atelectasis. Additionally, gas exchange is impaired in injured lung tissue, causing V/Q mismatch. ⚠ Inadequate analgesia worsens hypoxia associated with pulmonary contusion and rib fractures by ↓ ventilation, ↑ atelectasis and ↑ risk of superimposed infection.

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- Patients with rib fractures should be managed using a pathway that incorporates analgesia, respiratory support and physiotherapy.
- Chest trauma scores[47] help stratify injury severity and type of analgesia (e.g. PO/PCA/regional nerve catheters/thoracic epidural) and guide level of care (i.e. ward/HDU/ICU). They include parameters such as age, number of ribs fractured, comorbidities, concomitant anticoagulant use and SpO_2, and sometimes include a frailty score.
- Indications for admission, critical care involvement, type of respiratory support (i.e. mask O_2/HFNO/non-invasive ventilation/intubation) and further imaging (e.g. chest CT) and indication for referral for rib fixation can also be included in these protocols.
- Prompt multimodal analgesia, including paracetamol, NSAIDs if appropriate, oral or PCA opioids, and regional analgesia are essential to improve the ability to breathe and cough, thereby reducing complications.
- There are numerous regional analgesia options, each supported by relatively low-quality evidence. The most widely advocated in contemporary practice are epidural or paravertebral catheters and ultrasound-guided erector spinae (ESP) (see ➲ p. 1130) or serratus anterior plane blocks. ESP and serratus anterior plane are both relatively simple, have a lower risk of pneumothorax (compared with paravertebral) and are safer in anticoagulated patients.
- Serratus anterior plane and ESP catheters can be bolused with 30–40mL of 0.25% levobupivacaine (weight-dependent) ± 1:200 000 adrenaline, followed by an infusion of 10mL/h of 0.125% levobupivacaine.
- Paravertebral catheters and epidurals have the best available evidence base. Paravertebrals can provide a reliable unilateral block with a reduced side effect profile, compared with thoracic epidurals (see ➲ p. 1128).
- Operative fixation may be indicated if there is significant deformity, ongoing air leak, uncontrollable pain in spite of optimal analgesia or flail segments with paradoxical movements.
- Humidified O_2, nebulised 0.9% sodium chloride and respiratory physiotherapy also help reduce respiratory complications. Non-invasive ventilation or high-flow O_2 reduce atelectasis and improve the paradoxical movement of a flail segment. IPPV should be avoided, if possible.

Cardiac contusion

- Cardiac contusion should always be suspected in patients with deceleration injuries and/or sternal fractures.
- A 12-lead ECG should be performed as part of the 2° survey.
- There is a range of injury from minor myocardial contusion with raised troponin to free wall or valvular rupture.
- Patients who have an abnormal ECG (ST-segment changes, arrhythmia such as PVC, SVT or heart block) and raised cardiac markers should be admitted for 24–48h with ECG monitoring.
- Echocardiography is a useful adjunct when evaluating hypotensive patients or patients with other clinical signs, including chest pain, ECG abnormalities and elevated cardiac enzymes. Experience is needed to interpret the subtle signs associated with cardiac contusion.[48]

- Often patients with severe cardiac contusion will have other injuries mandating their admission to HDU/ICU.
- Severe myocardial contusion may require the use of inotropic support, and although late complications including heart failure, arrhythmia and aneurysm have been reported, they are rare.

Traumatic aortic injury

Around 80% of patients with traumatic aortic injury will die at the scene. Although deceleration and falls account for the majority of cases, penetrating trauma is also a recognised cause.[49]

A high index of suspicion and a low threshold for CT and angiography are the best diagnostic tools. 'Cardinal' CXR signs such as widened mediastinum, pleural capping and blunting of the aortic knuckle are not always present. Checking bilateral BP is of limited value.

The site of the injury will dictate where the blood may collect; damage to the intrapericardial portion of the aorta will lead to a cardiac tamponade, and injuries to the extrapericardial aorta will lead to mediastinal haematoma and haemothorax.

Grade of traumatic aortic injury

- Type 1: intimal tear
- Type 2: intramural haematoma
- Type 3: pseudoaneurysm
- Type 4: rupture (e.g. periaortic haematoma, free rupture).

Management of traumatic aortic injury

- Type 1 injuries: non-operative management consisting of aggressive HR and BP control and serial imaging. Systolic BP should be maintained at 100mmHg and HR <100bpm with β-blockers and vasodilators if not contraindicated.[50]
- Type 2, 3 and 4 injuries: repair is recommended. Endovascular intervention is becoming the mainstay of treatment if the anatomy is favourable due to the significant morbidity associated with open repair. While awaiting repair, control BP and HR as per type 1 injuries. Delayed repair may be appropriate for patients who are haemodynamically stable with severe coexisting injuries.[51]

Oesophageal injuries

Oesophageal trauma is uncommon due to the relative protection afforded by the chest wall. Oesophageal injuries normally occur in two places:

- Lower portion (commonest) associated with severe blunt trauma to the abdomen
- Cervical region (less common) 2° to penetrating trauma.

Oesophageal rupture leads to mediastinitis, pneumomediastinum and a left-sided pneumothorax or pleural effusion. Severe pain out of proportion to the apparent injuries, pain on swallowing and shock should alert the clinician to the presence of an oesophageal injury. Oesophageal rupture carries a high mortality and early use of antibiotics and surgical repair is recommended[52] (see ➲ p. 553 for repair of oesophageal injuries).

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Ruptured diaphragm

Diaphragmatic rupture can be caused by both blunt and penetrating trauma to the abdomen. Up to 15% of penetrating thoracoabdominal trauma will involve the diaphragm. The commonest cause of blunt diaphragmatic trauma is due to seat belt injuries and is more common from side impact than frontal collision.[4] The left side is more commonly involved, but when the right hemidiaphragm is involved, hepatothorax can occur. Underlying vascular damage can initially be masked due to the effect of the liver in the chest.

Some diaphragmatic injuries are asymptomatic, with the diagnosis made after CT. However, it is not uncommon for bowel to be felt on finger thoracostomy. Subsequent herniation and strangulation are a significant complication. Diaphragmatic rupture requires surgical repair (see ➔ p. 553 for repair of diaphragmatic injuries).

Rupture of the tracheobronchial tree

- Tracheobronchial injuries surviving to hospital treatment are rare.[53] Blunt trauma causes include deceleration, hyperextension and shear forces, while penetrating injuries usually occur in the cervical trachea.
- Although airway management is key to the 1° survey, tracheobronchial injuries can be missed in 25–68% of patients.[54] There should be raised suspicion for tracheobronchial injury when SC emphysema is seen over the neck, a pneumothorax fails to reinflate after insertion of a chest drain or there is evidence of excessive air leakage.
- Patients with distress and clinical suspicion of airway injury should be immediately intubated, preferably under FOB guidance to place the ETT distal to the injury or facilitate one-lung ventilation. Methods of intubation include orotracheal (direct or VL), fibreoptic, through open neck wound or tracheostomy.
- Injuries of the intrathoracic airway are more challenging. The use of long ETTs bypassing the injury for single-lung ventilation has been recommended.

(See ➔ p. 554 for repair of tracheobronchial injuries.)

Abdominal and pelvic injuries

Abdominal trauma

The 2nd commonest, and leading preventable, cause of death in the first 24h after trauma is major haemorrhage.[55] The abdomen is largely unprotected and susceptible to both blunt and penetrating trauma. Solid (e.g. liver, spleen, kidney) and hollow (e.g. small and large bowel, bladder) abdominal viscera can be injured, as well as retroperitoneal vessels, diaphragmatic rupture and trauma to the abdominal wall. Road traffic collisions account for the majority of blunt abdominal trauma, while stabbing accounts for most penetrating cases in the UK.

Abdominal trauma may cause major haemorrhage or soiling of the peritoneum and may be intra- or retroperitoneal. Early haemodynamic compromise from abdominal injuries arises solely from bleeding; the peritoneum can accommodate nearly all of a patient's circulating blood volume and represents an uncontrollable bleeding source. Eviscerated bowel and peritoneal blood can both stimulate a vagal response. Patients with serious injuries who appear physiologically compensated can deteriorate without warning. Watch for isolated BP drops, unexplained persistent tachycardia and raised base deficit.

Abdominal trauma can pose a diagnostic and management challenge due to the spectrum of injury and treatments available. A common dilemma during management of a trauma patient is deciding if they are stable enough for CT or if they need to go straight to theatre/interventional radiology for immediate treatment. CT imaging can help target surgery, but the delay in surgical control may be deleterious. Attention to the mechanism and pattern of injury will suggest likely injuries and appropriate resuscitation, while careful examination of the abdomen, use of eFAST and response to initial resuscitation will help guide the team. The priority is deciding on the next course of action, not the definitive diagnosis.

Treatment options

Indications for urgent laparotomy include ongoing blood loss or gross haemodynamic instability, with or without a positive eFAST examination, significant solid/hollow viscus injury, generalised peritonitis, foreign body *in situ* or evisceration. If the patient is physiologically well, then a definitive surgical procedure can be performed; otherwise DCS should be started.

Interventional radiology techniques, such as angiography and embolisation, can be used to manage arterial bleeding from solid organs, e.g. spleen, liver and kidneys, dependent on the grade of injury. These approaches can preserve organ function, in addition to avoiding surgical morbidity. Interventional radiology can also be used to control bleeding from abdominal/thoracic wall vessels. The anaesthetic team must accompany the patient for any acute interventional radiology procedure, as if the patient had gone to theatre, to provide continued resuscitation (± DCR), analgosedation and/or anaesthesia.[56] Interventional radiology procedures may take place in a remote location, so the usual considerations about remote site anaesthesia apply (see ➲ pp. 823–5).

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Laparoscopy may be used in selected patients with either blunt or penetrating trauma as a diagnostic tool and therapeutic technique. The rationale for this mode of surgery is to reduce the burden of 'negative laparotomy' and complications of laparotomy. Indications include suspected hollow viscus or diaphragmatic injury, free fluid from unknown source/mesentery and screening for peritoneal breach.[57] Contraindications include haemodynamic instability, absence of indications for open surgery and brain injury. The patient should be positioned as per a laparotomy in case conversion is necessary, which occurs in ~10–20% of cases.[57]

Non-operative management comprising monitoring of vital signs, serial abdominal examinations (more frequently than 4-hourly) and regular FBC/blood gases is also increasingly being used. Surgical intervention should occur if there is haemodynamic instability, signs of peritonitis, a drop in Hb >30g/L or a rise in WCC.[58]

Damage control surgery

(See ➲ pp. 982–3.)

Resuscitative endovascular balloon occlusion of the aorta

Resuscitative endovascular balloon occlusion of the aorta (REBOA) involves inserting an endovascular balloon, typically via an open femoral artery cutdown, into the aorta and advancing it to predetermined 'landing zones'. The balloon is then inflated to control bleeding below this level. It is a temporising measure for non-compressible, uncontrolled torso haemorrhage.

Three distinct aortic 'landing zones' are described:[59]

- Zone 1: between left subclavian and coeliac artery. Useful for intra-abdominal haemorrhage. Leads to the greatest physiological changes
- Zone 2: between coeliac artery and the most caudal renal artery. No-occlusion zone
- Zone 3: between the most caudal renal artery and aortic bifurcation. Useful for pelvic fractures/haemorrhage and injuries of the groin/femoral junction.

The literature is heterogeneous in regard to the indication and patient population of most benefit; however, growing levels of evidence support the use of REBOA in selected cases.[60]

REBOA is a temporising measure, rendering the structures below ischaemic; therefore, minimising the inflation duration is key. Patients should be taken rapidly to theatre where surgical repair of the bleeding source can be performed. REBOA deflation can lead to hypotension from reduced afterload and returning cold blood, emboli, ischaemic metabolites and haemorrhage; it should be done slowly and the balloon reinflated if more volume resuscitation is needed. The anaesthetic team should be prepared with volume replacement, drugs and means of managing a cardiac arrest. Sheath removal may need formal vascular repair.[59]

Pelvic injuries

- Pelvic ring injuries occur in 8–9% of all blunt force trauma and mortality rates range from 10% to 50%, depending on the extent of haemorrhage and associated injuries.
- Life-threatening haemorrhage may result. Most bleeding is usually from tearing and shearing forces on the venous plexus at the back of the pelvis. A small proportion is from arterial injury (<10%) and bleeding from the surface of the fractures themselves.
- Associated urological injuries can result in severe long-term disability and are potentially fatal.
- Open fractures of the pelvis involving the vagina and rectum are associated with a mortality of 30–50%. These injuries may be difficult to diagnose initially.

Injury classification

The Young and Burgess classification of pelvic fractures uses three mechanistic descriptions, each with degrees of severity.

- AP compression ('open book') I–III. Associated with frontal collisions.
- Lateral compression I–III. Associated with lateral collisions.
- Vertical sheer. Associated with falls from height.

Management

- Immediate management of the unstable patient with pelvic trauma is placement of a pelvic binder, which reduces the potential space within the pelvic cavity.[61]
- The pelvic binder can be left in place for several hours and, if an injury is identified, should only be removed when personnel are available who can repair the injury.
- After a pelvic binder is removed, repeat a pelvic X-ray.
- Haemorrhage control can be achieved by:
  - Pelvic external fixation—will clamp both sides of the pelvis together, reducing pelvic volume and tamponading bleeding. This will take precedence over laparotomy in an open book or vertical shear fracture.
  - Pelvic packing to manage venous bleeding in the unstable patient.
  - Selective angiography and embolisation to manage arterial bleeding.
- A single gentle attempt at catheterisation by an experienced doctor is permissible, even if the CT findings suggest urethral injury.
- Note, multiple surgical teams may be operating at once in pelvic injuries.

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Spinal trauma

Between 250 000 and 500 000 patients around the world suffer a spinal cord injury each year. Of these, ~90% are due to traumatic events such as road traffic collisions, falls and violence. Outcome after a spinal cord injury depends on the severity and location of the lesion. Below the level of injury, there may be complete or partial loss of sensory/motor function. The commonest level affected is C5.[62] Patients with spinal cord injuries are 2–5 times more likely to die prematurely.[63]

The 1° spinal cord injury results from the initial traumatic event, directly damaging ascending and descending pathways and blood vessels. The commonest 1° mechanism of spinal cord injury is an impact, followed by continued compression.[64] The acute phase of 2° injury occurs within minutes of the 1° event and results from the consequences of the 1° injury, including: local haemorrhage, hypotension, vasospasm, hypoperfusion, ischaemia, cytokine release, oedema and neurotransmitter accumulation.[63,64]

The principles of management of a spinal cord injury are similar to those of traumatic brain injury. The aims are to preserve neurological function and reduce further injury by minimising 2° injury. The most effective treatment in this case is early surgical decompression within 8h of injury.[65]

Primary survey

- On arrival in the ED, the patient may be on a vacuum mattress, 'scoop' or spinal board. Historically, spinal boards have been used for transport, but these are extrication devices only. They should be removed as soon as possible in the ED to avoid pressure areas developing using a 'log roll' technique (see ➲ p. 1003). Every movement of the patient should be well coordinated.
- The mechanism of injury, signs and symptoms will alert the team to the presence of a spinal cord injury. Polytrauma patients should always be considered as having a spinal cord injury until formal assessment, including radiological investigations, has been completed.

Airway and cervical spine control

- Patients should arrive in the ED with C-spine control and spinal motion restriction; however, this is not always the case. When performing MILS or placing a hard collar, no patient should have their head or neck forced into a neutral spine position and no deformity should be reduced.[4]
- Early intubation and ventilation may be required as high cervical injury above C3 leads to apnoeic respiratory arrest. Patients with spinal cord injury are also at a higher risk of aspiration.[66] (See ➲ pp. 972–4 for information regarding intubating the immobilised patient.)

Breathing

Supported ventilation may be required in the patient who has a thoracic injury or cervical injury sparing the diaphragm (C3–5) due to reduced chest excursion. Respiratory effort may deteriorate over time in the supine patient.

Circulation

Neurogenic shock, due to sudden loss of autonomic tone, may develop in a patient with a cervical or high thoracic injury. Bradycardia and hypotension (systolic BP <90mmHg) in the presence of cervical or high thoracic trauma should be managed with fluid and vasopressor infusion to maintain a spinal cord perfusion pressure >60–65mmHg,[67] which in practice entails maintaining a MAP >85–90mmHg for 5–7d after injury.[62] Metaraminol is a simple 1st-line choice, but central venous access to facilitate noradrenaline is recommended. When the injury is associated with polytrauma, every effort must be made to avoid ongoing haemorrhage. It may not be possible to differentiate between the two causes of shock, especially when there is loss of sensation below the injury level.

Disability

- The ASIA chart is currently the most widely accepted and employed clinical record for spinal cord injury. The ASIA chart is repeated at 48h to ascertain if the injury is incomplete or complete. A complete spinal cord injury is suspected when there has been no change in neurological deficit in this time.
- The ASIA chart is performed as part of the 2° survey, and perianal sensation and tone should be included as this may demonstrate sacral sparing. Prognostication may be complicated by the presence of spinal shock and should not be discussed as part of the initial assessment and management.

Log roll

'Log rolling' a patient requires a minimum of five people: three to control the patient's body and limbs, ensuring minimal movement of the spine; one to control the C-spine and lead the log roll; and one to inspect the back. Often formal examination of the back is delayed until after the trauma CT. Removing the trauma scoop should be done using a modified log roll, utilising the smallest tilt/brace required to remove the split board.

Spinal shock

Generally, this is a reversible condition where there is complete loss of sensation, muscle tone, power, autonomic activity and areflexia below the level of the injury. This can last anywhere from hours to a number of weeks. (See also ➔ p. 303.)

Autonomic dysreflexia

Rarely seen in the acute stage of spinal cord injury, but occasionally occurs in the following days and weeks. Most often occurs with lesions above T6. Defined as episodic hypertension and associated bradycardia (baroreceptor-mediated) due to disorganised autonomic reflexes. Life-threatening hypertension can occur, precipitated by noxious stimuli below the level of the injury, most commonly bladder or bowel distension. Other symptoms include headache, sweating, flushing and anxiety. Management is focused around removing the stimulus and acutely managing the hypertension. Head-up positioning and short-acting agents such as GTN sublingual spray (400 micrograms) and sublingual nifedipine (5–10mg) are all treatment strategies.

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Limb and extremity injuries

- Extremity injuries are very common in the polytrauma patient. Assessment of extremity and soft tissue trauma usually falls into the 2° survey, unless there are features of significant uncontrolled haemorrhage or closed long bone fractures of the femur.
- Management of open fractures and extremity lacerations should follow the principles of haemorrhage control using tourniquets and splinting. Femoral fractures can account for 1–1.5L of blood loss. Alignment of a displaced fracture and traction splinting should occur in the 1° survey if there are features of paraesthesiae, poor circulation or cardiovascular instability.
- Limb-threatening soft tissue injuries requiring urgent intervention include:
  - Vascular injuries proximal to the elbow or knee
  - Major joint dislocations
  - Crush injury
  - Compartment syndrome
  - Open fracture
  - Fracture with neurovascular injury.
- Arterial injuries associated with fracture or dislocation are rare, but the limb must be revascularised as a surgical emergency, as after 3–4h, irreversible tissue damage can occur. Common sites of arterial involvement include knee and elbow dislocations and severe tibial and femoral fractures. Diagnosis of an ischaemic limb can be aided by waveform SpO_2.[68]
- Manipulation and reduction of orthopaedic injuries can occur in the ED. Common agents used for provision of analgesia ± anaesthesia in this setting include ketamine, fentanyl and methoxyflurane (Penthrox™).
- Relocation or realignment and immobilisation of fracture sites significantly reduces pain and may reduce bleeding.
- A careful examination of the peripheral nervous and vascular systems must be performed and clearly recorded for all injuries. This must be repeated and recorded after any manipulation or surgery.
- For open fractures, IV antibiotics should be given within 1h of injury[69] and local guidelines will dictate the choice.
- In the UK, hind- or mid-foot fractures and compound fractures of long bones need to be discussed with, and possibly transferred to, a centre which can provide orthoplastic care.
- Fat embolism (see ➔ p. 614) is associated with long bone injury and manipulation, particularly compound femoral fractures. Although it is generally rare, it can be fatal in up to 15% of patients.
- Extremity trauma can be extremely painful and may progress to chronic pain.[70] Aggressive multimodal analgesia is warranted, including early use of antineuropathic agents.
- Regional nerve blocks, e.g. fascia iliaca blocks (see ➔ p. 1135), are routinely placed in the ED for pain control. Documentation of the block must be clear. There is controversy over their use in patients at risk of compartment syndrome, compounded by a lack of high-quality evidence. There are case reports illustrating that peripheral nerve blocks both do, and do not, mask the pain of compartment syndrome. A recent

systematic review concluded that peripheral nerve blocks do not affect the pain perception from acute compartment syndrome,[71] although epidurals may delay the diagnosis.[72] The risk, in part, depends on the type and location of the fracture and the planned surgical management, e.g. if fasciotomies are planned as part of immediate surgical repair, compartment syndrome is unlikely to be a problem. It is wise to discuss the use of regional anaesthesia with the orthopaedic/trauma team on a case-by-case basis prior to performing any procedure.

Compartment syndrome

- Is a limb- and potentially life-threatening condition that occurs when injured muscle groups contained in poorly compliant osseofascial compartments expand, creating a tissue pressure greater than the perfusion pressure, leading to ischaemia and tissue necrosis. If prolonged, permanent muscle and nerve damage can occur.
- It is most commonly caused by trauma (soft tissue, with or without fracture), typically closed fractures of the tibia and forearm,[73] crush injuries and burns.
- Pulses are often present early and it is the presence of 'out-of-proportion pain' and pain on passive movement which will raise the clinical suspicion. Swelling, skin turgor, sensory deficits and paresis may also occur.
- Diagnosis is clinical and notoriously difficult; therefore, a high index of clinical suspicion is required. Compartment pressures can be measured in sedated patients—a difference of 30mmHg between the diastolic BP and the compartment pressure suggests an ↑ risk of compartment syndrome. An absolute compartment pressure of 40mmHg, with clinical symptoms, indicates a consideration for urgent fasciotomy for decompression.
- When diagnosed, fasciotomy should occur within <1h.

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Gunshot, blast and crush injuries

Gunshot injury

Gunshot wounds are associated with extensive tissue damage caused by the transfer of kinetic energy. Low-velocity bullets, such as from a pistol, result in crush and laceration injuries. Secondary projectile, e.g. bone or bullet fragments, may move on unpredictable trajectories. High-velocity rounds, such as fired by a rifle, create a rapidly expanding and collapsing vacuum in the wake of the bullet's trajectory, sucking in debris. This results in cavity formation and significant tissue destruction not just in the path of the bullet, but also in surrounding tissues. The degree of tissue destruction and cavitation is affected by the yaw and tumble motion of the bullet along the trajectory within the body. A small entry wound can belie the extent of tissue injury. The unpredictable path of the bullet and contamination throughout its course are responsible for significant injury. Surgical debridement of all devascularised or soiled tissue is imperative to avoid sepsis.

Blast-related injury

Injuries result from exposure to pressure waves caused by explosions and can result in unique injury patterns, which are not commonly seen, in multiple patients at the same time. Injury severity is determined by the type of explosive device, the geography of the explosion (enclosed spaces reflect the blast wave) and the proximity of the victim. The predominant injury patterns in survivors are a combination of standard penetrating and blunt trauma. Blast injuries can be defined by five mechanisms (Table 37.6).

Primary blast lung injury

Primary blast lung injury (often called 'blast lung') is the commonest, fatal, 1° blast injury among initial survivors.[74] The lungs are particularly prone to damage, with widespread disruption of the alveolar–capillary membrane, leading to air embolus and hypoxia. Patients are at high risk of pulmonary oedema. It is rare for this injury to exist in isolation. 'Blast lung' is a clinical diagnosis and characterised by V/Q mismatch, hypoxaemia and dyspnoea. Pulmonary injuries can range from scattered petechiae to confluent haemorrhage, sometimes with associated pneumothoraces. The pattern of infiltration produces a classic butterfly appearance on CXR; however, initial imaging may not demonstrate the extent of the damage. An inflammatory phase follows the initial injury.

Embolic events

Air embolism is thought to be an early cause of death for non-survivors at the scene. Pulmonary fat embolism may be detected in as many as 75% of casualties.[75]

Management

Management of these patients is as for all trauma patients; however, specific mention should be made of the following:

- Always consider tension pneumothoraces which need to be treated urgently.
- Confusion and agitation are common and may be due to the blast wave itself or to cerebral air embolism.

Table 37.6 Mechanisms of trauma in blast injuries

Type	Characteristics
Primary	**Mechanism**: overpressurisation by the blast wave causes rapid compression and expansion of gas-filled structures **Affects**: gas-filled structures **Examples**: blast lung, bowel haemorrhage and perforation from shear forces, tympanic membrane rupture and middle ear damage, globe rupture, traumatic brain injury without signs of head injury
Secondary	**Mechanism**: flying debris and bomb fragments **Affects**: any part **Examples**: penetrating ballistic (fragments) or blunt injuries, eye penetration (± occult)
Tertiary	**Mechanism**: individual thrown by, or crushed by structures thrown by, the blast wind **Affects**: typically blunt injuries to any body part **Examples**: fractures and traumatic amputation, closed and open traumatic brain injury
Quaternary	**Mechanism**: all explosion-related injuries, illnesses or diseases not due to the above. Includes exacerbation or complications of existing conditions **Affects**: any part **Examples**: burns, inhalational injuries, crush injuries, closed and open traumatic brain injury, respiratory disease from dust, smoke or toxic fume inhalation, angina, hyperglycaemia, hypertension
Quinary	**Mechanism**: tissue contamination from post-detonation environment **Affects**: any part **Examples**: nuclear, biological, chemical, bloodborne viruses, human remains

- Lung injury is often heterogeneous and lung-protective ventilation strategies should be employed to avoid overdistension of unaffected alveoli. Note, permissive hypercapnia may have deleterious effects in the polytrauma patient with head injury.
- Lung compliance is often reduced and ↑ PEEP may be required.
- Hypoxia may be a consequence of inhalational injury or toxic fumes, as well as 'blast lung'.
- Appropriate personal protective equipment and strategies to protect personnel must be employed if there is suspicion of toxic contamination.
- The tympanic membrane is the most vulnerable structure to 1° blast injury and should be visualised during the 2° survey, if not before.

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Crush injury

Crush injuries are caused by physical compression leading to direct injury or ischaemia.[76] The limbs are most commonly involved (legs > arms), but multiple injury patterns are possible. Injury severity is determined by the magnitude and duration of the crush.

- Fractures and open injuries will require standard treatment of fixation, debridement, tetanus toxoid and antibiotics.
- Compressive forces to the torso can cause a wide spectrum of blunt or penetrating injuries, including organ contusions/rupture, vascular damage, fractures, haemorrhage and evisceration. Standard treatments, including lung-protective ventilation, pelvic binders and prompt multimodal analgesia, are required.

Crush syndrome: if significant muscle mass is crushed, potentially life-threatening systemic manifestations, known as 'crush syndrome', may occur. Crush syndrome results from traumatic rhabdomyolysis and the release of intracellular contents (K^+, myoglobin, phosphate, urate and CK) into the circulation following reperfusion of the injured muscle/organ.

The systemic effects and their management include:

- Reperfusion can lead to acute hypovolaemia and hypotension. Significant volume/fluid replacement may be needed to compensate for fluid sequestration into the interstitium, to restore BP and to maintain renal perfusion. Infusions and boluses should be titrated to the individual patient and regularly reassessed.
- Rhabdomyolysis:
  - AKI may occur from myoglobinuria. Mannitol may be needed initially to encourage diuresis, but haemofiltration/dialysis may also be required later.
  - Electrolyte disturbances/metabolic abnormalities—hyperkalaemia and hypocalcaemia. Observe for cardiac arrhythmias and correct as required.
  - Metabolic abnormalities: lactic acidosis resulting from ischaemic tissues can exacerbate the electrolyte abnormalities.
  - Sodium bicarbonate 8.4% 1mL/kg slow bolus, with further doses titrated to effect, can help.
- Compartment syndrome can occur as injured tissues swell. Prophylactic fasciotomies may be needed.
- In addition to the specific nuances of crush syndrome, multiorgan failure, ARDS, TIC and sepsis can also occur.

Traumatic cardiac arrest

A cardiac arrest 2° to trauma is managed differently to a cardiac arrest due to a medical cause. Importantly, should the mechanism of injury and collateral history suggest a 1° medical arrest, followed by trauma (e.g. single-vehicle accident without brakes being applied or low-impact trauma leading to cardiac arrest), then standard ALS should be instituted. VF and pulseless VT are usually associated with non-traumatic cardiac arrest; however, PEA and asystole are more common in traumatic cardiac arrest. The 1° goals in traumatic cardiac arrest are to stop exsanguination, relieve obstructive causes of shock and optimise oxygenation/ventilation.

Survival after traumatic cardiac arrest is poor. Overall survival rates with penetrating trauma are 5–10%. Patients with isolated cardiac wounds treated by expert teams can achieve greater survival rates. Patients with blunt trauma have a worse outcome, and resuscitative thoracotomy is most likely to be successful if the patient has vital signs on admission and CPR has been performed for <15min[77] (Fig. 37.1).

Indicators of the potential for favourable outcome after traumatic cardiac arrest include:

- Penetrating injury, especially single puncture with a knife (rather than a bullet) and RV injury
- Presence of signs of life at any time since medical contact
- Cardiac arrest <10min
- Visible cardiac activity on eFAST.

Management priorities for traumatic cardiac arrest

The below interventions should be performed concurrently by a trauma team.

Deprioritise chest compression

- Chest compressions are unhelpful in extreme hypovolaemia and potentially in tamponade. They also impede the ability to deliver potentially lifesaving procedures, may worsen injuries, add iatrogenic injuries and can slow transfusion from rapid infusors.
- Once a reversible cause has been corrected, chest compressions may help to support organ perfusion while blood volume resuscitation continues.

Optimise oxygenation and ventilation

- Give 100% O_2 and intubate the patient. If the intubation is difficult, consider using an SGA.
- Perform bilateral open thoracostomies in the 4th to 5th intercostal space, anterior axillary line, to release tension pneumothoraces.

Stop bleeding

- Temporising methods include direct and indirect pressure, haemostatic agents, tourniquets, pelvic binders, REBOA and resuscitative thoracotomy.
- Transfer to an operating theatre, interventional radiology or hybrid suite for definitive management should occur as soon as practicable for definitive haemorrhage control.

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Actively restore physiology

- The principles are as per DCR (see ➔ pp. 982–7)—obtaining vascular access, aggressive restoration of circulating volume using haemostatic principles and warming the patient.

Resuscitative thoracotomy

A resuscitative thoracotomy is a bilateral anterior ('clamshell') thoracotomy performed prehospital, in the ED or in the operating theatre as part of the immediate lifesaving resuscitation.

Indications

Accepted	Penetrating chest/epigastric injury associated with cardiac arrest (any rhythm) or periarrest (e.g. decompensating tamponade/persistent hypotension) within last 10min
Relative	Penetrating intra-/extrathoracic injury, pulseless and absent signs of life*, CPR <15min Blunt thoracic injury with cardiac arrest and previous signs of life
Contra-indications	CPR for >15min or >10min without response for penetrating and blunt trauma injury, respectively Unsurvivable concomitant injuries

* Signs of life = pulse, movements, GCS >3, eye or gag reflexes and cardiac activity on ECG or ultrasound.

Preparation

Achieving a good outcome from resuscitative thoracotomy relies on a number of components:

- A clear goal:
  - Release of cardiac tamponade
  - Control of cardiac, vascular or pulmonary haemorrhage
  - Occlusion of descending thoracic aorta to reduce haemorrhage below the diaphragm and increase blood flow to heart and brain
  - Control of air embolism
  - Provision of open cardiac massage.
- Personnel should have the expertise to perform the procedure.
- It needs a team approach with clear direction. If the TTL is the only individual skilled in resuscitative thoracotomy, they should temporarily hand over team leadership to another senior team member to allow them to task-focus. The anaesthetist is responsible for securing the airway and may be required to gain large-bore or central venous access.
- There needs to be a clear chain of survival such that when ROSC is achieved, ongoing care is planned and there is no hiatus in the flow of treatment.
- It is important to recognise that once initiated, the ongoing management of resuscitative thoracotomy and traumatic cardiac arrest will use a large number of personnel and resources.
- TCA should not be attempted if:
  - There are no signs of life within the previous 15min
  - Trauma is incompatible with life, e.g. decapitation or brain tissue loss.

Procedure[78]

- Stop CPR.
- Ensure the patient has an ETT or SGA in place.
- Perform bilateral finger thoracostomy in the 4th or 5th intercostal space.
- Connect the two thoracostomies with a clamshell incision, using heavy-duty (e.g. TuffCutt®) scissors or similar, to cross the sternum.
- Insert a retractor to open the chest cavity to the maximal extent.
- Lift the pericardium with forceps and make a midline longitudinal inverse 'T' incision to the pericardium. This avoids the phrenic nerves which pass laterally on the pericardial sac.
- Deliver the heart from the pericardial sac.
- Close any wounds to the cardiac muscle with staple or interrupted sutures. If you are unsure what to do, you can put a finger in the hole temporarily.
- A 2nd operator can provide pressure on the descending aorta.
- Concurrently infuse blood and blood products. The right atrial appendage can be used if vascular access is not possible to obtain.
- Control any bleeding that may be evident after ROSC, especially the internal mammary arteries.
- Resuscitation should be terminated as a team decision and should be considered if:
  - There is no ROSC after reversible causes have been addressed
  - There is no evidence of cardiac activity in the absence of a tamponade (if ultrasound is used prior to thoracotomy).

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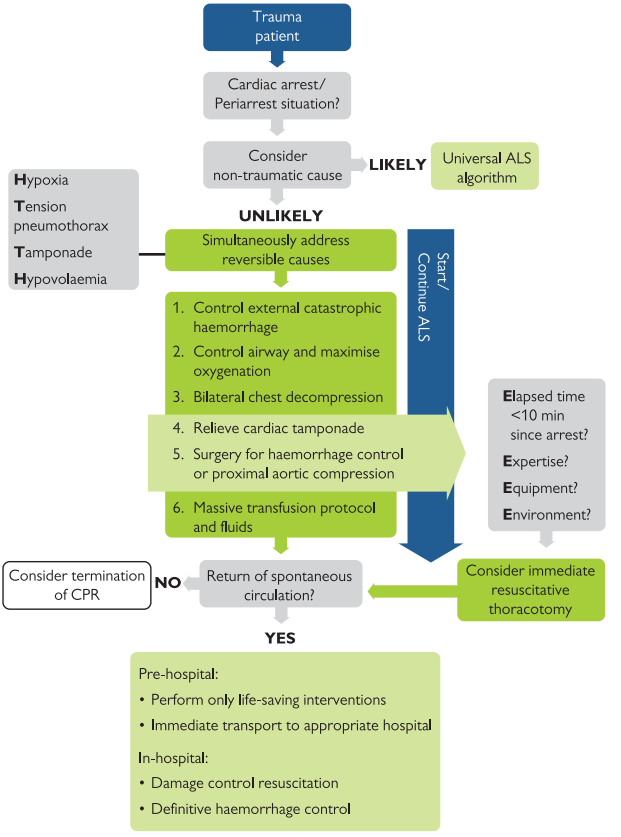

Fig. 37.1 Traumatic cardiac arrest algorithm. Local guidelines may be adapted to include the use of ultrasound and variations for the gravid patient. Reprinted from Resuscitation, **95**, Truhlar A *et al.*, European Resuscitation Council Guidelines for Resuscitation 2015, Section 4. Cardiac arrest in special circumstances, 148–201, Copyright © 2015, with permission from the European Resuscitation Council and Elsevier.

Burns: early management

(See also ➲ p. 665.)

General considerations

- Major burns account for 5% of traumatic injuries across the UK.[79]
- Treat life-threatening injuries; traumatic injuries and burns often coexist.
- Fire is the commonest cause of burns in adults; scalding is the commonest cause in children. Most injuries occur at home.
- Burns may be associated with alcohol intoxication, epilepsy or psychiatric illness.
- Consider the possibility of non-accidental injury in children.
- Mortality is related to age, total body surface area (TBSA) burnt, burn depth, presence of inhalational injury and hypothermia. Commonly used burns prognostic scoring systems, e.g. BOBI (Belgian Outcome of Burn Injury) score, FLAMES (Fatality by Longevity, APACHE II score, Measured Extent of burn and Sex), revised Baux score and ABSI (Abbreviated Burn Severity Index), use these parameters with various weightings. Use Box 37.7 to guide decision about transfer to a major burns centre.

Airway (with cervical spine control)

- Burns to the head and neck may rapidly cause airway obstruction from oedema. Inhalation of hot gases usually causes airway injury above the larynx.
- Signs of potential airway compromise: singed nasal hairs, hoarse voice, productive cough and soot-stained sputum.
- Clinical judgement will determine the need for immediate intubation. Maximum wound oedema occurs 12–36h after injury when fluid resuscitation is well under way, although the airway may be compromised much earlier. ⚠ **If in doubt, intubate early using a large, uncut ETT; subsequent oedema can be considerable.**[80]
- Clinical features shown to correlate with intubation include: full-thickness facial burns, stridor, respiratory distress, oedema on laryngoscopy, smoke inhalation and singed nasal hairs.[81]
- When considering a surgical airway with overlying burns, a longitudinal incision is advised.

Breathing

- Give O_2 15L/min, using a face mask with a reservoir bag, aiming for saturations of 94–98%.[82]
- Intubation may be required in patients who are:
  - Unconscious from coexisting trauma or from inhalation of toxic substances such as carbon monoxide (CO)
  - Developing acute respiratory failure due to smoke inhalation or blast injury
  - In need of extensive resuscitation, sedation and analgesia following a major burn.

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Circulation (with haemorrhage control)

- Burns >25% TBSA produce a marked systemic inflammatory response, accompanied by an increase in capillary permeability and generalised oedema.
- Insert cannulae through intact skin, wherever possible. Start IV fluids for burns:
  - >15% TBSA in adults (some units advocate 20%)
  - >10% TBSA in children.
- Hartmann's solution is the preferred resuscitation fluid for burns.
  - The fluid requirement in the first 24h in adults and children is 2–4mL/kg/% TBSA. Give half the calculated fluid in the first 8h from the time of injury, and give the remainder in the next 16h.
  - Maintenance fluids are required, in addition to the calculated resuscitation fluid. Ensure fluid is warmed.
- These calculated values are an estimate. Volume required will be guided by the urine output (>0.5–1.0mL/kg), blood gas values and CVS response.
- Test the urine for haemochromogens (myoglobin/Hb) arising from muscle damage and red cell breakdown. If positive:
  - Increase the urine output to 1–2mL/kg/h.
  - Alkalinise urine: infuse 1.25% sodium bicarbonate solution.
  - Promote diuresis: add 12.5g of mannitol to each litre of Hartmann's solution.

Neurological deficit

- Head injury is common in burns associated with road traffic collisions, falls and blasts.
- CO poisoning and alcohol intoxication are common causes of altered consciousness.

Exposure (with temperature control)

- Remove all clothing to assess the extent of burn injury. If clothing is stuck to the skin, cut around the area, leaving the adherent fabric in place. Keep the patient warm.
- Assess the percentage TBSA burnt. The 'rule of nines' conveniently divides the adult body surface into multiples of 9%; this is inaccurate for small children. The palmar surface of a patient's hand and fingers is ~1% TBSA. Mobile phone applications such as Mersey Burns (https://merseyburns.com) can be useful in TBSA estimation. Detailed assessment of the burn area is made by referring to a Lund and Browder chart (Fig. 37.2; Table 37.7).
- Assess the burn depth; burns may be superficial or deep; in practice, most injuries are a mixture of both.
  - Superficial: affecting the epidermis only (sunburn, flash burns) or involving the superficial part of the dermis (producing a blister). These burns are painful and pinprick sensation is preserved. Healing will occur without the need for grafting.
  - Deep: consisting of deep dermal burns (no capillary refill beneath the blister, since blood vessels are destroyed) or full-thickness burns (involving the entire epidermis and dermis, possibly including underlying structures). Burns may have a white, waxy appearance. Pinprick sensation is lost.

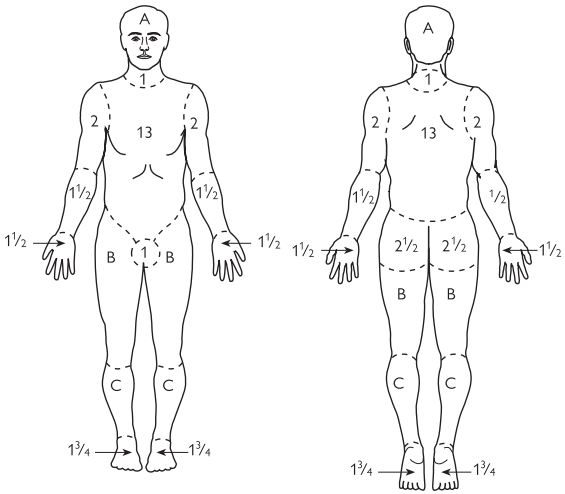

Fig. 37.2 Lund and Browder chart.

Table 37.7 Percentage of area affected by growth

	1mo	1y	5y	10y	15y	Adult
Half of head	9½	8½	6½	5½	4½	3½
Half of thigh	2¾	3¼	4	4½	4½	4¾
Half of leg	2½	2½	2¾	3	3¼	3½

Immediate wound care

- Cool the burn wound with cold running water (12°C is optimal);[83] this helps reduce the production of inflammatory mediators and reduces tissue damage. Continue cooling for at least 20min. Cooling the burn is an effective analgesic and can be done up to 3h after the initial injury.[84] **⚠ Monitor for hypothermia which is independently associated with ↑ mortality.**[85]
- Burns are initially sterile. Cover with 'cling film' to limit evaporation and heat loss and reduce pain.

Monitoring

- Monitor SpO_2, ECG, urine output, core temperature and NIBP. The pulse oximeter cannot detect COHb and will over-read the O_2 saturation of Hb; use a co-oximeter to obtain an accurate estimation of the percentage of oxyhaemoglobin.
- Arterial lines for patients with major burns or inhalational injury.
- Gastroparesis is common, so insert an NGT if major burns.

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- Check FBC (including Hct), urea/creatinine, electrolytes, glucose and COHb; X-match blood and consider trace elements (copper, selenium, zinc) in severe burns.

Analgesia

- Analgesia is important. Although skin sensation is lost over deep burns, the surrounding area is very painful.
- There is no evidence to support the use of any particular opioid. Morphine, fentanyl and methoxyflurane are equally efficacious.[86] Ongoing analgesia with PCA may be necessary.

Escharotomy

- Eschar is the coagulated dead skin of a full-thickness burn; it cannot expand, as tissue oedema progresses. Circumferential burns to limbs may result in limb ischaemia; circumferential burns to the trunk may reduce chest wall compliance and impede ventilation.
- Escharotomy, the release of the burn wound by incision down to SC fat, is performed in the operating room. Incisions are made longitudinally along the medial and lateral sides of the limbs; on the trunk, incisions are made along the anterior axillary line down to the upper abdomen. Ensure blood is available; bleeding can be extreme.
- Patients are often already sedated and ventilated. Lightly sedated patients will need additional sedation and analgesia.

Special circumstances

Inhalation of toxic substances

CO poisoning is common—check COHb. The severity of symptoms may not correlate with the percentage of COHb and may mimic alcohol intoxication (Table 37.8).

- CO reduces the capacity of blood to carry O_2, causing tissue hypoxia. PaO_2 is normal. CO also binds avidly to other haem-containing compounds, especially the cytochrome system. The half-life of COHb is 250min when breathing room air; this is reduced to 40min when breathing 100% O_2. O_2 therapy should be continued, since a 2° peak of COHb occurs after 24h and is attributed to washout of CO from cytochromes.
- Hyperbaric O_2 therapy reduces the half-life of COHb to just 15–30min; however, the precise role of hyperbaric O_2 is controversial[87] and is highly subject to the availability of hyperbaric facilities. Indications for discussion with a hyperbaric facility include:
  - Any loss of consciousness, neurological abnormality or cognitive impairment
  - Chest pain, abnormal ECG and cardiac enzymes
  - Pregnancy
  - Inability to assess adequately.
- Other toxic products of combustion may include cyanide; ammonia; phosgene; hydrogen chloride, fluoride or bromide; and complex organic compounds. These toxic substances may produce:
  - A chemical burn to the respiratory tract
  - Interstitial lung oedema, impaired gas exchange and ARDS
  - Systemic acid–base disturbances
  - Rising lactate without hypoxia.
  - Hydrofluoric acid binding serum Ca^{2+} and causing hypocalcaemia.

Table 37.8 Symptoms associated with CO poisoning

COHb (%)	Symptoms
0–15	None (smokers)
15–20	Headache, mild confusion
20–40	Nausea and vomiting, disorientation, fatigue
40–60	Hallucinations, ataxia, fits, coma
>60	Death

Chemical burns

- Hands and upper limbs are the most frequently affected areas.
- Staff must protect themselves with gloves, apron and face mask.
- Remove contaminated clothing as early as possible and place in a secure container for disposal.
- Industrial or household alkalis and acids are commonly used chemicals, e.g. bleach, washing powder, disinfectants, drain cleaner, paint stripper. Immersion in complex hydrocarbons (petrol, diesel) without ignition may cause systemic toxicity. Phosphorus burns may result from fireworks or military applications.
- Tissue damage continues until the chemical is neutralised or diluted with water. Early, continuous and prolonged (1h) irrigation with cold water is vital for burns (except elemental Na, K and lithium).
- Specific treatments include:
  - **Hydrofluoric acid**: used in the glass industry, highly toxic. Burns of 2% TBSA can be fatal. Tissue penetration by fluoride ions causes deep chemical burns. Inactivate toxic fluoride ions by application of topical calcium gluconate burn gel and 10% calcium gluconate injections into the burn wound (0.5mL/cm² of surface burn), extending 0.5cm beyond the burn margin. Do not use calcium chloride; it is an irritant. Consider intra-arterial (10mL of 10% calcium gluconate in 40mL of 5% glucose over 4h) or IV (Bier's block; 10–15mL of 10% calcium gluconate plus 5000 units of heparin, diluted up to 40mL in 5% glucose).
  - **Phosphorus**: white phosphorus ignites spontaneously when exposed to air; it can be extinguished by water. Apply copper sulphate solution, converting phosphorus to black cupric phosphide.
  - **Bitumen**: common injury in the UK from road maintenance. It is liquid at 150°C and causes thermal burns. Cool with water; remove the bitumen with vegetable or paraffin oil.
  - **Cyanide**: hydroxocobalamin 5mg IV or 12.5g sodium thiosulfate should be considered in patients with features of cyanide poisoning. These treatments are controversial but have fewer side effects than dicobalt edetate which should be reserved for confirmed cases of cyanide poisoning.

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Electrical burns

- Low voltage (<1000V) causes a local contact burn. The 50Hz alternating current (AC) domestic supply is particularly likely to cause cardiac arrest. Muscle spasm may prevent release of the electrical source. There is no associated deep tissue damage.
- High voltage (>1000V) causes flash burn or deep tissue damage due to current transmission. High-voltage cables carry 11 000 or 33 000V; electric shock produces an entrance and exit wound, which may require fasciotomy under GA. Haemochromogens released from muscle and damaged red cells may cause AKI.
- A direct strike by lightning (ultrahigh voltage, high current) has a very high mortality. Current may flow up one leg and down the other, producing an entry and exit wound. Respiratory arrest is common. Side flash (nearby lightning strike producing current that flows over the surface of the victim) causes superficial burns.

Box 37. 7 British Burn Association criteria for transfer to a burns centre

- Burn >10% TBSA (adult) or >5% TBSA (child), or full-thickness burn >5% TBSA
- Burn to face, hands, feet, genitalia, perineum or major joints
- Electrical and chemical burns
- Inhalational injury
- Circumferential burn to the limbs or chest
- Patients at extremes of age
- Patients with medical conditions which may complicate treatment

Major trauma in children

Paediatric major trauma encompasses a diverse and heterogeneous group of pathologies and problems. Few health care professionals have clinical expertise exclusively in this area, making the team approach to the assessment and management of children essential. This section provides an overview of the management of major trauma in children, in particular focusing on the differences from adult care.

Data from the UK's Trauma Audit and Research Network[88] show the mean age of the most severely injured children is 6.5y, with a peak in the 1st year of life and another after 6y of age. The most frequently reported mechanisms were road traffic collisions (42%), falls (32%) and non-accidental injury (10% in <2y). Of the most severely injured children, the most frequently injured body part was the head (74%), followed by the thorax (20%).

Catastrophic haemorrhage

Catastrophic haemorrhage is usually managed in the prehospital setting and general principles apply such as direct pressure to active bleeding, use of haemostatic dressings, pressure over proximal vessels or applications of tourniquets when required. See Table 37.9 for a guide to sizing of chest drains, catheters and NGTs, and Table 37.10 for normal systolic BP and HR dependent on age.

The estimated blood volume in children is dependent on age:

- 90–100mL/kg for premature infants
- 80–90mL/kg for the term infant to 3mo
- 70mL/kg in children older than 3mo
- 65mL/kg in obese children.

(For information on maximum allowable blood loss calculation, see ➲ p. 906.)

Airway

(See ➲ p. 900 for differences in assessment and management of the paediatric airway.) In the context of major trauma, there is a high risk of airway obstruction, particularly in significant head injury with reduced GCS or severe multisystem trauma.

- Indications for intubation include:
  - Reduced GCS
  - Significant chest injury resulting in impaired oxygenation
  - To facilitate assessment and imaging such as CT or MRI in a distressed or agitated child.
- Suggested rapid anaesthetic for paediatric trauma patients:
  - Ketamine 1–2mg/kg ± fentanyl 1microgram/kg, rocuronium 1mg/kg
  - Suxamethonium 1.5mg/kg as an alternative (not in renal failure, burns >12h, rhabdomyolysis or high spinal cord injuries).
- Continued sedation after intubation is typically with morphine and midazolam (propofol can be used if sedation is for a short period only). Example regime: morphine 20–80 micrograms/kg/h and midazolam 120–360 micrograms/kg/h or propofol 2–5mg/kg/h.

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Table 37.9 Chest drain, NGT and urinary catheter size by weight

	3–5kg	6–9kg	10–11kg	12–18kg	19–22kg	23–30kg	>30kg
Chest drain	10–12Fr	12–16Fr	16–20Fr	20–24Fr	24–28Fr	28–32Fr	32–42Fr
NGT	8–10Fr	10Fr	10Fr	10–12Fr	12Fr	12–14Fr	14–16Fr
Foley catheter	6Fr	6Fr	6–8Fr	8–10Fr	10–12Fr	12Fr	12–14Fr

Table 37.10 Normal vital signs in children

	HR (bpm)	Systolic BP (mmHg)	RR (breaths/min)
Preterm	120–180	40–60	50–70
0–1mo	100–160	50–70	35–55
1–12mo	80–140	70–100	30–40
1–3y	80–130	70–110	20–30
3–6y	80–110	80–110	20–30
6–12y	70–100	80–120	18–24
12+y	60–90	100–120	14–22

Cervical spine

C-spine injuries in children are rare, but when present, they more frequently occur in the upper cervical spine (C1–4) due to the relatively higher head-to-body size ratio and higher fulcrum. With increasing age, the fulcrum lowers and lower cervical spine injuries become more common. The spinal skeleton is relatively mobile and can be significantly distracted without an underlying spinal fracture; this leads to SCIWORA. If plain X-rays or CT scans do not reveal a bony injury, but the neurological exam is abnormal, SCIWORA should be suspected and imaging with MRI is required.

Immobilisation

The cervical spine must, wherever possible, be stabilised until a C-spine injury is ruled out.[89]

If attempts at immobilising the cervical spine are causing distress and agitation, the risks and benefits of continued attempts must be assessed.

- In conscious children, use MILS whenever possible.
- In unconscious children or when MILS cannot be maintained, immobilisation should be performed with a properly fitting collar, blocks and tape.
- If no properly fitting collar is available, towels or blankets, etc. should be used to improvise an immobilisation device.

Breathing and chest assessment

- In young children, the chest wall is soft and pliable. The ribs are aligned horizontally and the intercostal muscles are weak. Rib fractures are much less common in children and, when present, represent significant traumatic force. Tension pneumothoraces are poorly tolerated as children develop hypoxia more rapidly than adults due to diminished FRC and a higher metabolic rate.
- The management of significant chest injuries is the same as for adult patients in many cases; the use of adequate analgesia and supplemental O_2 may be all that is needed. An intercostal drain must be considered in large haemothoraces or pneumothoraces causing respiratory compromise or if the patient is being placed on positive pressure ventilation.

Circulation

Children initially compensate very well for blood volume loss; tachycardia and hypotension are late signs. Mild agitation, sweating and confusion may all suggest impending circulatory collapse.

Abdominal assessment

Children have a relatively thin abdominal wall, a more horizontal diaphragm and altered organ proportion and placement, and in infants, the bladder is intra-abdominal. Therefore, the intra-abdominal organs are more exposed than in adults, rendering them more vulnerable to injury.

Visceral injuries are managed conservatively where possible, with surgery reserved for those children who remain unstable despite aggressive resuscitation.

Pelvis and long bones

Pelvic fractures are rare in children, but more likely in high-speed road traffic collisions with lateral side impact or pedestrian vs vehicle. An appropriately sized pelvic binder, or a folded sheet, wrapped around the child at the level of the greater trochanters (if a binder is unavailable) should be used for haemorrhage control if there is a suspicion of pelvic injury.

Femoral injuries are relatively more common. When assessing the skeleton, the margins of the pelvis should be palpated gently, as should the full length of the femurs, looking for swelling, tenderness, bruising and crepitus.

Managing haemorrhage

- IV access can be difficult in a child who is shocked and cold. If initial attempts at peripheral IV access are unsuccessful and the child requires immediate resuscitation, IO access should be gained, usually at either the proximal tibia or the distal femur.
- If a child is actively bleeding, resuscitate with blood (consider O-negative blood initially until X-matched blood is available). Crystalloid 10mL/kg can be used initially, but this is not recommended if blood is available. Balanced blood product resuscitation should be delivered following the principles described in DCR (see pp. 982–7). (See Table 37.11 for transfusion targets.)
- In haemorrhage, give IV tranexamic acid 15mg/kg (max 1g) within 3h, followed by a maintenance dose of 2mg/kg/h over the next 8h.[90]
- A transfusion of 40 mL/kg of blood products within the first 24h indicates a critically unwell child who is at ↑ risk for early and in-hospital death.

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Table 37.11 Transfusion targets in children

Blood product	Target	Dosage
FFP	1:1 ratio with PRBCs	5–20mL/kg For children born after 1996, FFP is virally inactivated (e.g. octaplasLG®)
Platelets	≥75 × 10^9/L	10–15mL/kg
Cryoprecipitate	Fibrinogen >1.5g/L	10mL/kg

Disability

Pupils should be assessed and a measure of consciousness, such as AVPU, documented and repeatedly assessed.

Exposure and environment

Full exposure and examination of the child while limiting heat loss are critical. Underbody forced air warmers and paediatric fluid warmers with reduced dead space are useful.

Primary survey imaging

- Children are believed to be more sensitive to high doses of radiation and this, together with a growing body of evidence suggesting a relationship between exposure to ionising radiation and a risk of developing malignancy, leads to a more conservative approach to imaging, compared to adult patients. The 'as low as reasonably achievable' principle leads to an imaging strategy built around judicious use of plain films and targeted CT + MRI. (See Box 37.8 for a useful framework for imaging of children in major trauma.)[6,91]
- Whole-body CT scan is still appropriate in children with severe injuries or injuries affecting >1 body region, where the overall risks and benefits have been carefully considered. Imaging should not be prioritised at the expense of careful examination, observation and stabilisation.

Imaging guidelines

Indications for CT head within 1h of ED presentation for children sustaining a head injury are as per adults (Box 37.8), but with a number of additional indications:[41]

- Suspicion of non-accidental injury
- GCS <14 (<15 in under 1y) on initial ED assessment
- Tense fontanelle
- Children <1y with presence of bruise, swelling or laceration of >5cm on the head
- More than one of the following:
  - Loss of consciousness lasting >5min
  - Abnormal drowsiness
  - ≥3 discrete episodes of vomiting
  - Dangerous mechanism of injury
  - Amnesia (anterograde or retrograde) lasting >5min.

The Royal College of Radiologists suggests the following guidance as a decision tree for paediatric imaging.[91]

Box 37.8 When to perform imaging of the cervical spine in children <16y

- Perform MRI for children if there is strong suspicion of cervical spinal cord injury, as indicated by the Canadian C-spine rule and/or clinical assessment and/or abnormal neurological signs or symptoms.
- Consider plain X-rays in children who do not fulfil the criteria for MRI but clinical suspicion remains, and discuss the findings of the plain X-rays with a consultant radiologist.
- For children who have sustained a head injury, perform CT cervical spine if any of the following apply:
  - GCS <13 on initial assessment
  - The patient has been intubated
  - Focal peripheral neurological signs
  - Paraesthesiae in the upper or lower limbs
  - Definitive diagnosis of C-spine injury is needed urgently
  - Other areas scanned for head injury or multiregion trauma
  - There is strong clinical suspicion despite normal X-rays
  - Plain X-rays are technically difficult or inadequate
  - Plain X-rays identify a significant bony injury.

Is the mechanism of injury consistent with major trauma?

- No: image as per clinical suspicion.
- Yes: perform 1° survey and CXR; if the CXR suggests significant intrathoracic injury, perform a CT thorax.
- Yes, but suspicion of isolated C-spine injury only: C-spine plain films normally enough to rule out bony injury.

Does the child meet NICE criteria for CT head?

- Yes: request head and if there is suspicion of severe multisystem trauma, consider C-spine CT.

Is there severe multisystem trauma, a lap-belt injury, abdominal ecchymosis, distension or tenderness, persistent hypovolaemia or PR/NG bleeding?

- Yes: abdominal CT (pelvic and abdominal X-rays are not indicated in paediatric trauma).

Concern of, or abnormal neurology consistent with, spinal injury?

- Yes: request plain spinal films and MRI spine.

MRI is preferable in children due to the ↑ incidence of SCIWORA and avoidance of ionising radiation.

The role of FAST

Generally, focused abdominal sonography in trauma (FAST) is not recommended in paediatric trauma. The technique is operator-dependent and has only a modest sensitivity in detecting haemoperitoneum. If imaging is needed, CT should be requested.

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Paediatric cardiac arrest

The evidence around specific adjustments required to standard ALS in children with traumatic cardiac arrest is limited.[92] The mechanism of injury helps predict possible injury patterns and potential reversible causes—interventions should focus on the treatment of the underlying cause. Thoracotomy should be considered if the skill exists within the team, for appropriate cases, e.g. penetrating thoracic or abdominal trauma, where intervention post-thoracotomy is more likely to be possible.

The adult traumatic cardiac arrest algorithm (see ➲ Fig 37.1) is similar to that for children, produced by Vassallo *et al*.[92] The authors noted that before assuming a traumatic cause of cardiac arrest in children, causes of hypoxia, e.g. drowning, asphyxiation and impact apnoea, must be excluded.

Paediatric considerations for the trauma team

- Paediatric trauma covers a broad range of emotional intelligence, but it is likely that the patient will be more anxious than an adult patient. Accordingly, the trauma call should be adapted.
- Rationalise the team. Paediatric trauma teams can be large and appear daunting. Staff who are not immediately needed should be asked to step away.
- Allocate one person to do the 1° survey and one person to be at the head end, talking to and calming the child.
- Encourage the parents to be present and visible. They should also have a chaperone explaining what is happening.

Safeguarding

A child presenting to hospital as a major trauma call can be considered a red flag for potential safeguarding issues. Rarely, the trauma call may be due to non-accidental injury, but more often injuries result from neglect or wider social stressors within a family. Older children and adolescents may become injured because of their own risk-taking behaviour or peer group behaviours.

All children presenting to trauma services should have these issues explored and local safeguarding services should be involved in their care. Where necessary, concerns will need to be escalated beyond the local system.

Analgesia in paediatric trauma

(See ➲ p. 926 for further detail on analgesia for children.) One of the biggest contributors to fear in paediatric trauma patients is pain, and this should be addressed early on in the patient journey. If surgery or imminent intubation is thought to be likely, then oral medications should be avoided. Suggested strategies include:

- Paracetamol (IV)
- Entonox® in older, awake patients
- Intranasal or IV opioids (diamorphine or fentanyl)
- Ketamine 250–300 micrograms/kg IM, IV, PO
- Morphine IV 50–200 micrograms/kg (max 10mg).

Silver trauma

'Silver trauma' generally describes trauma in patients ≥60y. A fall from standing is the commonest mechanism, and the commonest injury is a fractured neck of femur. However, rib and fragility fractures, head injury and major haemorrhage are also commonly seen.

Frailty, lack of physiological reserve, sarcopenia, comorbid disease and medication all impact on the type and severity of traumatic injuries and their presentation.[93,94,95,96] Older people are at significant risk of losing their pretrauma level of function. Improving outcomes and the process of re-enablement begins at triage. The Trauma Audit and Research Network (TARN) data consistently show that elderly patients are undertriaged, assessed by more junior team members, may be denied interventions and face delays to treatment and transfer to trauma centres.[97] Use of comprehensive geriatric assessment and frailty screening tools is recommended to facilitate more informed early decision-making in older trauma patients.[98]

The falls history

'A mechanical fall' is commonly documented as the mechanism of injury in silver trauma. This overlooks an important consideration: what precipitated the fall? Assessment, examination and investigation of these patients should aim to determine both the reason for the fall, the trauma caused by the fall and any associated injuries (e.g. burns, rhabdomyolysis caused by periods of immobility)—the input of orthogeriatricians is invaluable. Establishing the cause of the fall can be aided by the following sieve:

- Cardiac: chest pain, new breathlessness, collapse on exercise?
- Neurological: preceding loss of consciousness? Symptoms of CVE, headache, seizures?
- Drug-related: alcohol, medication changes?
- Orthostatic: PD, DM, bleeding, sepsis, medication?
- Environment: ill-fitting shoes, postprandial or post-micturition, emotional state?

Emergency assessment

The salient differences in injury and physiological patterns are summarised below. Be mindful that major life-changing injuries to the head, neck and chest can result from low-energy falls from standing.

Airway with C-spine control

Can be compromised or complicated by lack of, or displaced, dentures and restrictions in neck movement due to arthritic or spondylytic changes.

- Presence of severe degenerative disease of the cervical spine puts elderly patients at risk of worsening neurological outcome with poorly fitting hard collars. Padding and tape may be more appropriate and help avoid hyperextended positions and pressure sores.[99]
- Upper C-spine injuries are more common.
- Degenerative disease makes spinal contusion and central cord syndrome more likely and interpretation of images more difficult.[100]
- Have a low threshold for C-spine imaging. NICE guidance indicates that even without signs or symptoms of cervical spine injury, if there is the potential for C-spine injury in a person >65y, then a CT head should be performed.[6] If there is a head or face injury, think neck injury too. If CT head is indicated, so is CT cervical spine.

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Breathing

Multiple rib fractures, flail segments and haemo-/pneumothoraces may result from simple falls.

Chronic lung disease and reduced muscle mass may impair the patient's ability to increase respiratory effort, making ↓ pO_2 and ↑ pCO_2 more likely.

Circulation

- Anticoagulation for cardiac and vascular disease is common in the 'silver trauma' patient, turning what may have been a small haemorrhage into a major haemorrhage. Anticoagulant and antiplatelet history should be specifically sought for all patients. POCT of coagulation and platelet function (e.g. TEG® or ROTEM®) can be useful. Where bleeding is suspected, reversal of anticoagulation should be initiated promptly if possible (see ➲ p. 272).
- Hypertension is common, so physiological parameters for activation of major haemorrhage protocols should be adjusted. Systolic BP <110mmHg and/or a pulse of >90bpm should raise suspicion of hypovolaemia/haemorrhage. β-blocker use prevents/reduces the tachycardia response to hypovolaemia, which can reduce clinical suspicion of haemorrhage and also reduce the physiological method by which cardiac output and tissue perfusion are maintained.
- Invasive monitoring and repeated measurement of lactate and base deficit help guide resuscitation. Base deficit changes more acutely, whereas lactate can lag behind the clinical picture.

Neurological assessment

- Cerebral atrophy increases the relative skull vault space such that significant intracranial bleeding may go unnoticed, without an initial decrease in GCS. Repeat re-evaluation and a low threshold for CT head are necessary.
- Patients on antiplatelet or anticoagulant medications with a head injury and a normal initial CT head may require a repeat scan.

Temperature

- Elderly patients have reduced metabolism and muscle mass and are often discovered, having fallen some time ago, lying on the floor. These considerations render them more susceptible to hypothermia. Active warming begins in the ED and should continue until the patient is normothermic.

Electrolyte disturbance

- Elderly patients are more likely to have deranged electrolytes due to comorbidities (e.g. CKD) or medicines (e.g. diuretics).

Analgesia

Undertreated pain is a significant cause of delirium, so timely assessment and provision of multimodal analgesia are especially important in this at-risk group. NSAIDs are relatively contraindicated. Conversely, opioids can also cause delirium; regular oxycodone is preferred over morphine because it has better oral bioavailability and no active metabolites and does not accumulate in renal impairment. Laxatives should be co-prescribed. Regional anaesthesia (single shot or via catheters), where suitable, is ideal for reducing opioid needs.

Delirium

Delirium is a disorder in which there is an acute confusional state, usually with a fluctuating course, characterised by disturbed consciousness, cognitive function or perception. In older people, especially those with pre-existing cognitive impairment, it is usual to find several factors contributing to delirium.[101]

The 'PInCH ME'[102] mnemonic (Table 37.12) is a useful aide memoire to use when reviewing patients, in order to screen for and treat causes of delirium. The 4AT[103] (https://www.the4at.com) is a validated tool that quickly and easily detects delirium in routine clinical practice. Patients should be given their usual visual and auditory aids to allow them to engage with their environment as soon as possible.

Table 37.12 Risk factors for delirium

		Risk factors
P	Pain	Fractures and injuries
In	Infection	Skin and chest wall injuries, surgical site
C	Constipation	Opioids and immobility
H	Hydration	Nil by mouth, unable to reach drinks or hold cups, delirium
M	Medication	Polypharmacy and alcohol or substance withdrawal
E	Electrolytes	Glucose, Na^+ and K^+ derangement

Anaesthesia and surgery

- Time in theatre and under anaesthesia is a tiny proportion of the overall hospital journey, but its conduct can significantly impact remobilisation, re-enablement and rehabilitation.
- Ceilings of care/DNAR orders and their adaptation in the perioperative period should be discussed with the patient and multidisciplinary team. The ReSPECT tool may aid this.[95]
- Providing a homeostatic anaesthetic is of key importance. Proactively maintain a systolic BP within 20% of normal[104] and a MAP of ≥65mmHg.[105] A vasopressor infusion is ideal for this (but ensure euvolaemia).
- Consider inhalational induction or co-induction and use age-adjusted MAC ± depth of anaesthesia monitoring.
- Limit LA dose in spinal anaesthesia (e.g. hip fractures, ≤2mL of 0.5% heavy/plain bupivacaine) and avoid adjunctive opioids, or at most restrict to 20 micrograms of fentanyl.
- Maintain Hb around 90–100g/L.[106,107]
- Regional anaesthesia improves analgesia, reduces opioid requirement and reduces delirium.
- Avoid 'deliriogenic' drugs, e.g. ketamine, cyclizine, atropine, midazolam. For sedation, propofol as a TCI has a better side-effect profile compared to midazolam.
- POCT should be employed to direct transfusion and fluid requirements in theatre and on the ward.

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Recovery

Surgery care bundles are helpful in silver trauma patients. These prompt consideration of common issues during recovery and the inpatient stay, and provide appropriate parameters for early warning scores (e.g. BP, HR, Hb).

Elder abuse

Can take many forms, e.g. physical, financial, sexual, neglect, emotional and discriminatory. Be vigilant for the signs and if suspected, adhere to local reporting mechanisms.

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Chapter 38

The emergency patient

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Tracheal intubation of critically ill adults

(See also pp. 368–9 and p. 373–8.) (See Fig. 38.1.)

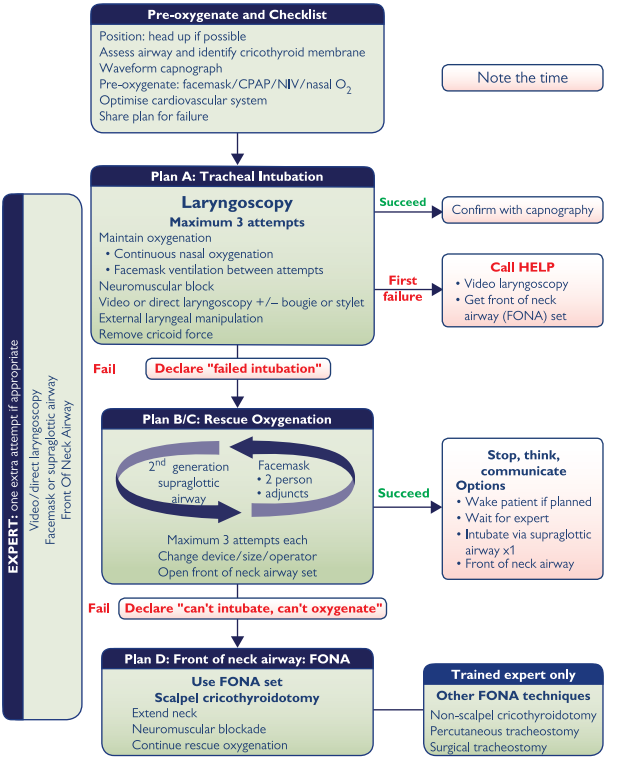

Fig. 38.1 Tracheal intubation of critically ill adults. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6): 827–48 (2015) doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

Sepsis and septic shock

Definitions

- The current definition of sepsis is life-threatening organ dysfunction due to a dysregulated host response. Historic distinctions between a systemic inflammatory response and infection have been removed.[1]
- qSOFA is used as a rapid clinical screen for sepsis; >2 of the following are required:
  - Hypotension (SBP <100mmHg)
  - Altered mental status (GCS <15)
  - Tachypnoea (RR >22 breaths/min).
- Septic shock is defined as the presence of sepsis with hypotension requiring vasopressors to achieve a MAP >65mmHg and lactate >2mmol/L despite adequate fluid resuscitation.
- Even with vigilance and treatment, sepsis and septic shock carry a high mortality.

Pathology

- The response to sepsis is complex and involves many pro- and anti-inflammatory mediators. The severity of illness is determined more by the nature of the individual inflammatory response than by the infection.
- Pathological effects caused by these mediators include vasodilation, impairment of the glycocalyx, capillary permeability, impaired tissue O_2 utilisation and septic cardiomyopathy.
- Tissues may become hypoxic for several reasons, including hypotension (vasodilation, hypovolaemia, myocardial depression), microvascular thrombosis (activation of coagulation), tissue oedema (acting as a barrier to O_2 diffusion) and shunting past some capillary beds. Hypoxia drives anaerobic metabolism and lactic acidosis and may occur despite adequate O_2 delivery, due to mitochondrial dysfunction. Reperfusion of previously hypoxic tissues can cause further release of damaging reactive O_2 species.

Resuscitation

- The resuscitation of a septic patient is started as soon as the condition is recognised. International guidelines for the treatment of sepsis and septic shock are frequently updated.[2] Current goals within the 1st hour of resuscitation are:[3]
  - To measure lactate (and remeasure if initial lactate is >2mmol/L)
  - To obtain blood cultures before administering antibiotics
  - To administer broad-spectrum antibiotics, guided by local microbiology policies
  - Rapid administration of 30mL/kg crystalloid for hypotension or lactate levels of >4mmol/L. Caution in patients with LV failure and consider need for fluid warming.
- Administer vasopressors to achieve a MAP >65mmHg if hypotension persists after fluid resuscitation.
- Induction of anaesthesia in the septic patient is hazardous, and every effort should be made to resuscitate the patient preoperatively. However, this must be balanced against the urgency of the surgery and the need for source control. Surgical source control may initially worsen the clinical state by precipitating a septic shower or haemorrhage.

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- Establish vascular access and monitoring as soon as possible and before induction of anaesthesia:
  - Secure large-bore IV cannulae
  - Arterial line
  - Urinary catheter (can be done post-induction)
  - CVP line if a need for centrally administered vasoactives/inotropes/parenteral nutrition is anticipated (can be performed post-induction)
  - In the presence of advanced sepsis, consider non-invasive CO monitoring using the preferred local method.
- Preoxygenate patients with 100% O_2 to increase SpO_2, ideally >92%[4] prior to induction.
- A higher MAP target may be appropriate in those with AKI and a background of hypertension.[5]
- Ensure blood is available—maintain Hb >70g/L. A higher transfusion threshold may be needed in acute haemorrhage. Transfusion should be balanced with factor replacement and utilisation of adjuncts (calcium, warming to normothermia, tranexamic acid) (see ➔ p. 454).
- Consider IV hydrocortisone (50mg 6-hourly) if hypotension remains poorly responsive to adequate fluid resuscitation and vasopressors.[6,7]
- If the patient requires high-level postoperative monitoring or support, discuss with critical care staff.

Interpretation of investigations in the septic patient

- FBC: expect a high WCC with neutrophilia, although a low WCC may be evidence of overwhelming sepsis. A low platelet count is common due to septic coagulopathy and thrombocytosis is also common in sepsis.
- U&E: elevated creatinine indicates AKI and an elevated urea:creatinine ratio may indicate dehydration.
- Coagulation: non-iatrogenic ↑ INR suggests septic coagulopathy.
- Blood glucose: stress-induced hyperglycaemia is common and might be exacerbated by catecholamines (classically adrenaline). Low glucose is concerning and supports the presence of advanced sepsis or hepatic dysfunction.
- ABGs:
  - Metabolic acidosis; early respiratory compensation may not be maintained as the patient becomes obtunded.
  - Hypoxia is multifactorial and common.
  - High-anion gap metabolic acidosis may be due to tissue hypoperfusion and hyperlactatemia, but other causes include blood ketones due to diabetic ketoacidosis, accumulation of toxins/drugs (i.e. metformin) and AKI. Lactate will also rise due to catecholamines, particularly adrenaline. If a normal-anion gap metabolic acidosis is present, consider common causes such as excessive 0.9% sodium chloride administration (chloride will be high) or GI bicarbonate loss.
- The CXR may show evidence of non-cardiogenic pulmonary oedema, indicating the development of ARDS. The PaO_2:FiO_2 ratio can used to determine the onset and severity of ARDS.[8]

Induction and maintenance of anaesthesia

- ↓ anaesthetic, sedative and opioid dose usually required. Carefully titrated induction drugs will achieve greater CVS stability, but the risk of aspiration may necessitate a more rapid administration of medication (RSI). The use of ketamine 1–2mg/kg IV is increasingly popular as an induction drug in the critically ill. Although cardiovascularly stable, etomidate is associated with ↑ mortality in septic patients.
- If acidotic and needing RSI, consider low-pressure ventilation to avoid hypercapnia worsening acidosis.
- Anticipate and prepare for cardiovascular collapse on induction due to vasodilation and sympatholysis. Ensure vasopressor drugs are available (e.g. adrenaline 1:10 000) and prepared (e.g. metaraminol, phenylephrine and ephedrine). Metaraminol or phenylephrine infusion can be useful at induction (see ➲ p. 1209), but ensure infusion lines primed before inducing anaesthesia.
- Insert and suction an NGT, if not already in place.
- Neuraxial blockade should be used cautiously due to risks of infective complications, sympatholysis with exaggerated hypotension and septic coagulopathy which might have developed since the most recent blood tests. If an epidural catheter is inserted, analgesia can be titrated once CVS stability is achieved or postoperatively (i.e. in the ICU). IV antibiotics should be given prior to a neuraxial procedure.
- A depth of anaesthesia monitor allows for titration of maintenance anaesthesia and reduces the risk of awareness.[9]
- The effects and duration of action of all commonly administered anaesthetic agents are unpredictable due to an altered volume of distribution, metabolism, organ blood flow and function.
- Avoid NSAIDs (including COX-2-selective agents) due to the risk of AKI.
- Blood glucose control should be implemented, aiming for a blood sugar level of <10mmol/L while avoiding hypoglycaemia.[10]

Maintaining tissue oxygenation in the operating room

Fluids

- Use balanced salt solutions.[11] Albumin might be considered when large volumes of crystalloid are used, but there is no evidence for improvement of patient-centred outcomes.[12] Starches are harmful and discouraged.[13]

Inotropes/vasoconstrictors

(See ➲ p. 1209 for doses.) After adequate fluid resuscitation, noradrenaline infusion titrated to a MAP >65mmHg is 1st line for its vasopressor effects. Occasionally, and cautiously, vasopressin might be used as an additional vasoactive drug, but evidence for patient-centred benefit is lacking. If inotropy is required, adrenaline or dobutamine should be used. If the cause for hypotension is unclear, an intraoperative echo (transthoracic echocardiography or TOE, dependent on access) can help direct management.

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Induction, oxygen and positive end-expiratory pressure

- Desaturation with apnoea is rapid and should be anticipated due to factors such as shunt and ↑ basal metabolic rate. Preparation with preoxygenation, aiming for an ETO_2 >90% (if possible), and consideration of apnoeic oxygenation (i.e. with high-flow nasal cannulae) can be lifesaving. Equipment used to oxygenate and initiate ventilation should be able to provide PEEP.
- Commence ventilation with an FiO_2 of 1.0 and titrate downwards to achieve an SaO_2 >90%, with PEEP starting at 5cmH_2O.
- If the patient remains on a high FiO_2 (>0.5) due to hypoxia, further titration of PEEP can be useful (increasing the PEEP to 10cmH_2O and greater). There needs to be careful attention to haemodynamics (as venous return will decrease and right heart afterload will increase, and occasionally the shunt may be exacerbated).
- Blind recruitment manoeuvres are harmful in the ICU (risks include cardiovascular collapse and ventilator-induced lung injury) and should be used very cautiously.[14]
- Persistent difficulty with oxygenation should prompt postoperative prone positioning and consideration of ECMO referral.[15]

Ventilation

- Intra- and extraparenchymal pathology and ↑ capillary permeability in sepsis decrease lung compliance and produce high plateau airway pressures, with a risk of ventilator-induced lung injury.
- In patients with early ARDS, every effort should be made to provide lung-protective ventilation, limiting the peak airway pressure to 30cmH_2O and V_T to 6–8mL/kg of the IBW.[16] Permissive hypercapnia should be tolerated to a lowest pH of 7.15, with anticipation of resistance to catecholamines and a difficulty in oxygenation met with PEEP titration. Airway pressure targets should only cautiously be raised with expert titration using tools like the driving pressure (Pplat – PEEP = <15cmH_2O).[17]
- Ongoing difficulty with ventilation may benefit from NMB, alongside sedation (though more recent evidence is conflicting).[18,19]

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Emergency laparotomy

(See also ➲ pp. 1035–9; ➲ pp. 1044–5.)

Indications for laparotomy include: bowel obstruction, perforation, infection, ischaemia and bleeding. Physiological derangement and organ dysfunction are associated with both the underlying disease process and the surgery itself:

- CVS instability due to vasoplegia and altered CO due to ↓ preload, depressed myocardial function and arrhythmias exacerbated by electrolyte dysfunction and acidosis.
- Impaired oxygenation from V/Q mismatching due to atelectasis, ↓ FRC, fluid extravasation into pulmonary interstitium, reduced lung compliance, abdominal distension and/or pain, leading to ↑ work of breathing or ↑ inspiratory pressures.
- Renal hypoperfusion, ↑ abdominal pressures and septic mediators can cause AKI.
- Coagulopathy which may be related to sepsis or major bleeding. Anaemia and bleeding might be exacerbated by acidosis, dilution and hypothermia.
- Stress-induced hyperglycaemia and altered drug metabolism. Tissue hypoxia is a common cause of metabolic acidosis and demonstrated by a high blood lactate and a high anion gap (>14).

The recurrent UK National Emergency Laparotomy Audit highlighted that the 30d mortality of emergency laparotomy remains ~10%. Outcomes are worse in the elderly. The mortality benefit from standardisation of care has plateaued and wider organisational changes may be required to achieve further reductions.[20]

Multiple risk factors associated with mortality have been identified (including age, ASA, emergency surgery and peritoneal soiling).[21]

Preoperative assessment

- While a period of preoperative resuscitation may benefit some patients, this should not delay surgery which should be performed by an experienced team.
- A validated risk scoring tool (e.g. the NELA risk calculator https://data.nela.org.uk/riskcalculator) and assessment of frailty should be conducted preoperatively. This allows for discussion of individualised benefit vs risk with the patient and family, mobilisation of consultant-led services and ICU/HDU admission for those with an estimated mortality of >5%.
- O_2 should be administered preoperatively to all hypoxic patients.
- Peritonitis and sepsis are common. Prompt antibiotic cover within the 1st hour in line with local guidelines should be given to cover Gram-positive, Gram-negative and anaerobic bacteria.[22]
- An NGT should be inserted in patients with intestinal obstruction.
- Administration of balanced crystalloids is encouraged to minimise acidosis and AKI.[23]
- Investigations: FBC, electrolytes (including Mg^{2+}), LFTs, amylase, clotting, lactate, G&S, ECG and CXR where appropriate. Reported CT imaging can assist surgical planning.
- Electrolytes will often be deranged and should be corrected as soon as possible perioperatively.

- Aim for a blood sugar level <10mmol/L (avoid hypoglycaemia).[24]
- Metabolic lactic acidosis is common and may improve with titrated fluid and CVS support.
- Bleeding should be anticipated and reversal of anticoagulant therapy must be considered (see ➲ pp. 269–76).

Intraoperative care

- A high-risk patient warrants the presence of a consultant anaesthetist and surgeon.[20]
- IABP monitoring should be established prior to induction.
- Secure a large-bore IV cannula prior to induction and connect warm fluid/fluid warmer.
- Aspirate the NGT prior to induction.
- Anticipate major hypotension following induction. Have vasopressors (ephedrine, metaraminol, phenylephrine and/or adrenaline) and vagolytics (atropine, glycopyrronium) drawn up and to hand. Consider vasoconstrictor infusions from the start.
- A CVC may be required for vasopressor and inotrope infusions, administration of electrolytes and postoperative parenteral nutrition.
- Perform maximal preoxygenation and anticipate rapid desaturation. High-flow nasal cannulae may provide apnoeic oxygenation.
- Careful consideration of the risk of CVS collapse from induction drug dosage vs the risk of aspiration is essential (see ➲ p. 388), as all agents can precipitate life-threatening hypotension. Ketamine (1–2mg/kg IV) or midazolam may be useful in the severely compromised patient.
- Rocuronium at RSI dosage (0.9–1.2mg/kg) or suxamethonium.
- Depth of anaesthesia monitoring can allow for titration of anaesthesia and may reduce both the risks of hypotension and awareness.[25]
- Ventilation can be challenging. Raised intra-abdominal pressure leads to ↑ airway pressures. Employ a lung-protective ventilation strategy with PEEP titration.
- Fluid assessment:
  - Enquire about any underlying cardiac conditions.
  - Know how much and which fluids have been administered preoperatively.
  - Goal-directed fluid technologies may help (e.g. oesophageal Doppler, pulse wave analysis, CVP), although there is limited evidence that they improve outcomes in abdominal surgery.
  - A restrictive fluid strategy has been shown to increase AKI in non-urgent abdominal surgery.[26]
  - In addition to balanced crystalloids, ensure that balanced blood products are readily available, with consideration of adjuncts (calcium, temperature control and tranexamic acid). Coagulation studies and viscoelastic studies can be used to guide transfusion.
- Patients who require repeated bolus doses of vasopressors, despite what is judged to be adequate volume administration, should be commenced on a vasopressor infusion early. Noradrenaline via CVC is the 1st choice for the septic patient. Adrenaline or dobutamine may be useful in patients with measured or suspected low CO.[27]
- Measure core temperature and maintain normothermia to minimise coagulopathy and altered drug pharmacokinetics.

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- Analgesia:
  - Opioids remain the mainstay for intraoperative analgesia in most institutions.
  - Caution with remifentanil and associated bradycardia,
  - Neuraxial blockade should be used cautiously due to risks of infective complications, sympatholysis and septic or haematological coagulopathy.
  - Rectus sheath catheters can be inserted at the end of the procedure, and LA administered postoperatively (see ➔ p. 1133).
- Monitor lactate and ABGs throughout the case to help guide perioperative response to therapies and aid decision-making on the postoperative disposition.

Postoperative care

- Consider ICU/HDU in high-risk or at-risk patients. Elderly and frail patients have a higher mortality and ideally would be admitted to critical care. If an ICU/HDU bed is unavailable, patients should be kept in recovery for ongoing observation, and the same level of care provided with ongoing reassessment of organ dysfunction.[20]
- Assess for the risk of abdominal compartment syndrome. High ventilatory pressures (>30cmH_2O) with hypoxia at the time of closure are concerning and should be discussed with the surgeon and intensivist (laparostomy may be helpful).
- Urine output should target >0.5mL/kg/h. In patients with persistently low urine output, assess fluid balance and reconsider vasoactive support to minimise AKI. AKI might be multifactorial (e.g. hypoperfusion, septic AKI, nephrotoxins, abdominal compartment syndrome). Continuous renal replacement therapy might be required.
- Optimise analgesia and provide chest physiotherapy to reduce the risk of hospital-acquired pneumonia (with early commencement of antibiotics if suspected).
- VTE prophylaxis when surgeon happy.
- Consider antacid prophylaxis if patient not able to eat.
- Consider parenteral nutrition if patient to remain nil by mouth.
- Specialist geriatrician input is recommended for elderly and frail patients.
- Timely communication with the family is important as the patient's care progresses.

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The ICU patient going to theatre

Care of a critically ill patient needing surgery can be challenging, and senior anaesthetists and surgeons must be involved to facilitate timely and appropriate perioperative planning. Routine aspects of anaesthesia preoperative assessment remain relevant to the critically ill patient and must not be overlooked.

Preoperative assessment

A system-based patient evaluation of active life-threatening problems, organ function and current level of support can be gained at the bedside, with discussion with the critical care medical and nursing teams. The patient may not be able to give consent. This should be obtained from the medical enduring power of attorney (usually next of kin), advance directives respected and if required or treatment guided by the best interests standard.

- Respiratory: note the patient's current level of respiratory support and recent trends, and formulate a perioperative ventilation strategy. Knowledge of additional respiratory therapy (secretion load, bronchodilators) can be valuable to troubleshoot intraoperative problems.
- Cardiovascular: note volume and type of fluids administered and vasopressor/inotrope infusions rates. Recent echocardiography, CVP and CO monitors may help guide further therapy.
- Confirm IV access. Know which lines are available for use without compromising other therapies. Know which line is best for rapid fluid administration.
- Check recent FBC, U&E, coagulation studies, glucose and ABG. Ensure a valid G&S ± X-matched blood is available.
- Intubated patients are often lightly sedated to allow tube tolerance and comfort, with the risks of prolonged sedation. Ensure adequate level of anaesthesia prior to muscle relaxation.
- Check if any scheduled drugs will be due in theatre (e.g. antibiotics).
- Fasting: evidence is emerging for non-airway surgery that in intubated patients, a shorter fasting period may be acceptable to reduce delays and extended caloric deficits. Aspirate NGTs prior to theatre.

Conduct of anaesthesia

- If the patient is not intubated, the decision of where to intubate (ICU vs theatre) is influenced by available personnel, need for equipment, familiarity with environment and clinical stability.
- Exercise vigilance when transferring between bed and operating table, as there is a risk of losing lines and tubes. Where the patient is on multiple infusions, transferring and maintaining the pumps on a single infusion stack will reduce tangling of lines, disconnections and inadvertent drug errors. Transfer the patient towards, not away, from lines.
- Disconnect the ETT from the anaesthesia circuit prior to transfer; the ETT can be clamped prior to disconnection if there is concern about infection, atelectasis or derecruitment.
- Increasing depth of anaesthesia can be achieved with any combination of opioids, sedatives/analgesia, TIVA or inhalational agents.
- NMBAs may be needed to facilitate intubation, ventilation or surgery. Ensure you hand over to the ICU team the timing of their delivery.

- Use of a processed EEG may reduce the incidence of awareness. Maintenance of lighter depths of anaesthesia may not reduce morbidity or mortality as previously thought.
- Fluid therapy should reflect whether the patient may still be in the resuscitative or stabilisation and optimisation phase of their illness. The choice of fluid will be influenced by surgical losses, the metabolic status and the 1° pathology.
- Analgesic strategies depend on whether the patient will be waking up postoperatively or not. Liaise with the ICU team to formulate a plan.
- Ensure a full verbal and written handover is given to the ICU medical and nursing staff, and clearly communicate any important postoperative events and outcomes.
- Contact next of kin if appropriate.

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ALGRAWANY

Transferring the critically ill

Transfer of a critically ill patient within or between health care facilities may be needed to access specialist investigations or procedures or for repatriation and rehabilitation. Transferring patients requires trained staff and careful preparation in order to not compromise the level of care delivered and to ensure patient and staff safety. Specialised transport teams may improve outcomes but are not always available. Before transferring any critically ill patient, the risks of transfer should be weighed against the potential benefits of transfer.

Chain of command and planning

- Once an indication for transfer and the receiving unit have been identified, one coordinator assumes overall responsibility for the whole transfer process. This person should understand the transport modalities available and their advantages and disadvantages, as well as the experience of the staff involved.
- The initiation of a transfer should follow clear guidelines and protocolled channels of communication.
- The transfer should not adversely affect the provision of care at the transferring hospital by depleting staff numbers or available equipment.

Transfer modality

- Patients may be transferred by road or air.
- Different facilities are available in different vehicles and craft, and the choice depends on the requirements of the patient. Ideally, for ICU transfer, there should be reasonable access to the patient, electrical supply, O_2 supply, adequate lighting and ability to communicate within the vehicle and the hospitals.
- Factors influencing the choice of transport modality include: effect on patient's condition, availability, staff experience/training, locations involved, distance, road situation (closures/rush hour), weather and cost.
- Whatever the modality, respect the staff of the vehicle or craft in which you are; they know it intimately and can advise you on the best way to avoid difficulties en route.

Aeromedical transfer (helicopter and fixed-wing aircraft)

- Often used for longer distances where time is of the essence.
- Most aircraft are pressurised to 1500–2000m above sea level, but pressure inside the helicopter is dependent on the height they fly. Height of helicopter transfer may be dependent on the flight path. Reduced barometric pressure can have deleterious effects on patients. Air-filled cavities expand as atmospheric pressure decreases with altitude. Pneumothoraces, obstructed bowel or air emboli can worsen. Pre-emptive chest drains and NGTs may be required. Endotracheal cuffs may expand.
- There is a decrease in O_2 partial pressure at altitude, so non-ventilated patients may require additional O_2 support.
- Both fixed-wing aircraft and helicopters may be located at a nearby airport, so that the 1° mode of transport may be preceded or followed by an ambulance transfer. More often hospitals have on-site helipads allowing for site–site transfer.

- Helicopter transfer involves greater vibratory forces and noise, compared to fixed-wing aircrafts or ambulances, and carrying capacity, patient access, air speed and range are lower than with fixed-wing aircrafts, making it less suitable over longer distances.

Ambulance transfer

- Road transport is cheaper and often faster to mobilise and presents fewer physiological disturbances and easier patient access.
- The standards for an emergency ambulance stipulate the presence of a minimal O_2 supply of 2000L (usually 2F-sized cylinders), direct current/AC power inverters and basic monitoring equipment. However, it is best to assume that everything will be supplied by the transfer team.
- When considering road safety, the goal is to facilitate smooth and rapid transfer, with minimum acceleration and deceleration. High-speed travel is rarely necessary. The driver will decide on the need for lights and sirens, depending on the road conditions and information from the transfer team.

Preparation

Familiarity with the transfer environment aids planning and preparation for the transfer and potential emergency scenarios.

Patient preparation

- Dependent on urgency of transfer (ruptured AAA vs stable ICU patient). Stabilisation and interventions must be weighed against urgency of transfer. Even if stable, prepare for clinical deterioration en route.
- The airway is usually secured with an ETT if deterioration is anticipated. Ventilated patients should be safely established on the transport ventilator before departure. Check ABG if possible.
- IV access must be robustly secured, reliable and readily accessible. Use extension lines.
- Arterial lines may be more reliable than NIBP which can be affected by movement and vibration.
- Chest drains, NGTs and catheters, if sited, must be secured.
- Ensure sedation is adequate for ↑ stimulation during transit and administer NMBA if needed.
- Protect pressure areas, while ensuring patient and equipment are securely fastened to the transport stretcher.
- Have means to keep the patient warm—children are extremely susceptible to hypothermia.
- Inform the receiving team that you are leaving the base hospital.
- Inform relatives about the transfer and advise them what to do at the receiving hospital.

Drugs, equipment and monitors

- The range of drugs and equipment available should allow maintenance of current management, treatment of predictable changes in the patient's condition and the treatment of acute medical emergencies (Table 38.1).

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Table 38.1 Checklist for transfer equipment and medications

Airway and breathing	Suction equipment, tracheal tubes, connectors, ties, stethoscope, tracheostomy tubes (if appropriate), face masks, airways, self-inflating bag with reservoir, laryngoscopes, spare batteries, gum elastic bougie
Circulation	Cannulae plus IV dressings and tape, appropriate IV fluids/blood and associated giving sets. Syringes, needles and safe sharps disposal container
Resuscitation drugs	Adenosine, adrenaline, amiodarone, atropine, calcium chloride, furosemide, glucose, GTN spray, hydrocortisone, lidocaine, lorazepam, metoprolol, naloxone, noradrenaline, salbutamol (with appropriate delivery equipment), 0.9% sodium chloride, sodium bicarbonate Drug labels
Sedation and NMDAs	Propofol, midazolam Atracurium, rocuronium or suxamethonium
Neurological	Mannitol, hypertonic sodium chloride
Additional paediatric equipment	Paediatric O_2 mask with reservoir bag, tracheal tubes, small cannulae, appropriately sized self-inflating bag with reservoir, laryngoscope and stylets IO needle Magill forceps and suction catheters. Paediatric drug doses calculated and written down 10% glucose for correction of hypoglycaemia

- Gravity-fed drips are unreliable in moving vehicles, so pumps and pressure bags should be used and mounted at, or below, the level of the patient to enable access to the patient.
- O_2 and drugs supply should be in excess of estimated transport requirements. Drugs that will be affected by interruptions should be preconnected, with scope to 'double pump'.
- Know if your vehicle has its own O_2 supply.
- ECG and BP are susceptible to motion artefact, and NIBP may rapidly deplete the battery.
- Ensure all battery-dependent equipment is charged and plugged into a power source in the transport vehicle, if possible.

You and your team

- Transfers can happen near the end of your shift. Recognise your limitations and fatigue, and do not go on a transfer if you do not feel comfortable to do so.
- Ensure you have the appropriate medical indemnity to go on a transfer. Consider non-sedating antiemetic prior to transfer if you have a history of travel sickness.
- Wear warm clothes and take money with you.
- Understand how you and your equipment (e.g. ICU monitors, infusion pumps) will get back to the base hospital. This may be by taxi (know how this will be paid for).

Table 38.2 Checklist for preparation to transfer a patient

Airway	Is the airway safe? If in doubt, intubate Cervical spine control
Breathing	Portable ventilator settings. Check ABG after 15min on the portable ventilator, before departure Self-inflating bag–valve device in the event of a ventilator/ O_2 failure Suction Adequate O_2 reserves Insert a chest drain if there is a possibility of a pneumothorax (e.g. fractured ribs)
Circulation	Stable circulation with good access Controlled external bleeding Invasive BP when indicated Inotropes: if in doubt, have them prepared and ready to run Pumps and batteries Consider inserting a urinary catheter, and monitor output
Disability	Adequate sedation, analgesia and NMBA GCS, pupillary signs NGT/OGT
Exposure	Avoid hypothermia Splint long bones
Forgotten	All notes, referral letter, results, radiographs (including CT scans) and blood products Inform the receiving unit that you are leaving the base hospital Inform relatives Take contact numbers Take warm clothing, mobile phone, food and credit card/ money for the team! Plan for the return journey Medical indemnity and insurance for death or disability of transfer staff

- Ensure you have enough personal protective equipment (specialist equipment may be required).
- Take a charged telephone with the numbers of the base and receiving hospital contacts.
- Relatives, especially of paediatric patients, often request to travel with the patient. For air transfers, the pilot decides who can be transported as weight restrictions apply. Always discuss with the vehicle/aircraft crew prior to agreeing to a travel request, and ensure everyone (including yourself) is briefed before boarding.
- Whatever the mode of transport, try to position yourself so you have a view of the patient and monitoring.
- Equipment can become a missile in the event of a sudden deceleration, so ensure it is secured correctly.

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- Helicopters and, less so, ambulances are noisy and you may be required to wear ear protectors. Alarms are unreliably heard, even at their maximum setting, so monitoring should be visible. If there is a paramedic more familiar with the environment than you are, ask them to help en route, such that you are able to remain hands-off, if possible, and maintain situational awareness.

Paperwork

- Take: a concise handover, photocopy of the patient's notes and drug charts, pertinent imaging and lab results.
- The transport record should document the patient's clinical status before, during and after transport, relevant medical conditions, environmental factors, therapy given, adverse logistical events and procedures undertaken.
- Perform a predeparture checklist (Table 38.2).

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Chapter 39

Anaesthetic emergencies

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See also

➲ Airway assessment and management p. *361*
➲ Paediatric emergencies pp. *951–64*

Adult basic life support

(See Fig. 39.1.)

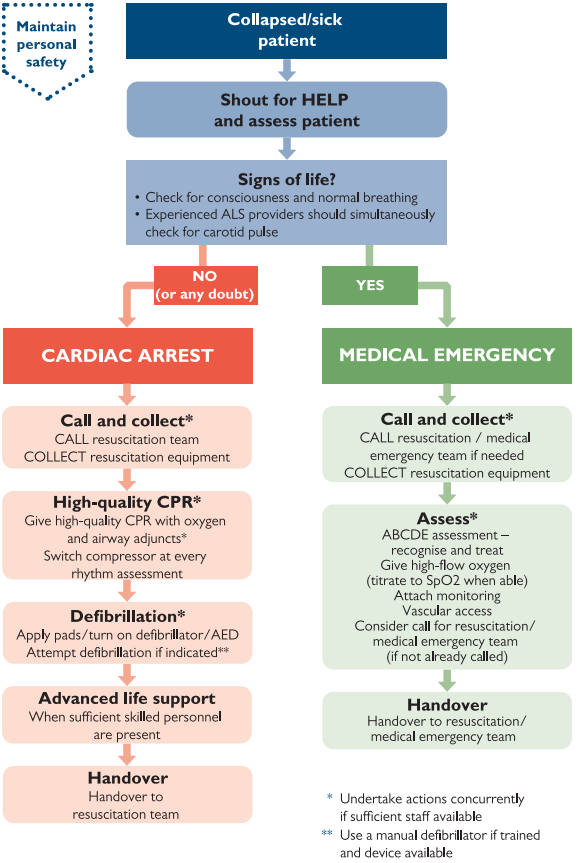


Fig. 39.1 Resuscitation Council UK adult in-hospital cardiac arrest algorithm. Reproduced with the kind permission of the Resuscitation Council UK.

- Shout for help early.
- **Look** for chest movement, **listen** for air flow and **fee**l for a carotid pulse.
- If absent or uncertain, declare cardiac arrest and immediately call the resuscitation team.
- Gasping, agonal breaths and seizure-like movements are common in the early moments after cardiac arrest and should not be mistaken for signs of circulation.
- Immediately start CPR.
- Apply defibrillation pads, check the rhythm, and give a shock if VF or pulseless VT (VF/pVT).
- Follow the ALS algorithm.

Chest compressions

- Give compressions at about 120/min at a depth of 5–6cm in the lower half of the sternum.
- Allow full recoil of the chest, with equal time in compression and decompression.
- Change the person doing compressions about every 2min.
- Use a mechanical compression device if manual compressions are impractical, prolonged or during transport.

Breathing

- Give 30 chest compressions, before giving two ventilation breaths.
- Use an inspiratory time of 1s and achieve a normal chest rise.
- Give the maximum inspired O_2 feasible.
- When the patient's trachea is intubated, give continuous compression at about 120/min and ventilate the lungs at ten breaths/min without pausing compressions. This can also be done with an SGA when there is a good seal.

Application of defibrillation pads

- Analyse the rhythm using self-adhesive pads as soon as is possible.
- Continue chest compressions while placing the pads.
- Place one pad below the right clavicle and the other in the left mid-axillary line at the position of the V_6 ECG electrode (lateral to any breast tissue). Ensure good adhesive contact with both pads.
- Alternative pad positions are AP or biaxillary.
- Pause briefly (<5s) to assess the rhythm.
- If shockable (VF/pVT), restart compressions and charge the defibrillator during compressions. Once charged, pause the compressions and give shock. Immediately resume compressions.
- Check rhythm after 2min of compressions.

Suspected cervical spine injury

- Do not delay starting CPR when there is a risk of spinal cord damage. Untreated cardiorespiratory arrest will kill the patient.
- Minimise neck movement and try and keep the neck in a neutral position, but do not compromise resuscitation interventions.

At-risk patients

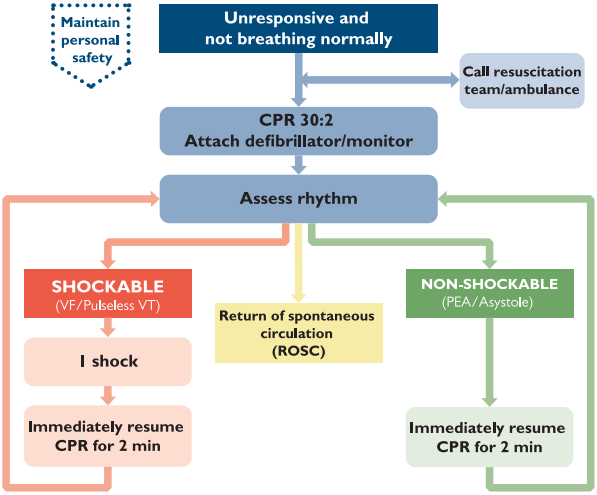
- All patients should have prior decisions about CPR to prevent inappropriate CPR attempts.
- Use an early warning scoring system (e.g. National Early Warning Score 2 (NEWS2)) to identify deterioration and escalate care.

Further reading

European Resuscitation Council. https://www.erc.edu
Resuscitation Council UK. https://www.resus.org.uk

Adult advanced life support

(See Fig. 39.2.)



Give high-quality chest compressions, and:
- Give oxygen
- Use waveform capnography
- Continuous compressions if advanced airway
- Minimise interruptions to compressions
- Intravenous or intraosseous access
- Give adrenaline every 3–5 min
- Give amiodarone after 3 shocks
- Identify and treat reversible causes

Identify and treat reversible causes
- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypo/hyperthermia
- Thrombosis – coronary or pulmonary
- Tension pneumothorax
- Tamponade – cardiac
- Toxins

Consider ultrasound imaging to identify reversible causes

Consider
- Coronary angiography/percutaneous coronary intervention
- Mechanical chest compressions to facilitate transfer/treatment
- Extracorporeal CPR

After ROSC
- Use an ABCDE approach
- Aim for SpO_2 of 94–98% and normal $PaCO_2$
- 12-lead ECG
- Identify and treat cause
- Targeted temperature management

Fig. 39.2 Resuscitation Council UK—adult advanced life support algorithm. Reproduced with the kind permission of the Resuscitation Council UK.

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High-quality CPR

- Ensure high-quality chest compressions with minimal interruption.
- Aim for <5s for all pauses for interventions.

Defibrillation

- When a shockable rhythm (VF/pVT) is seen, restart chest compressions and charge the defibrillator.
- Pause to deliver the shock and immediately resume compressions.
- Ensure everyone is clear when shock is delivered.
- Use at least 150J in an adult. Follow defibrillator manufacturer's instructions. Escalate shock energy if initial shocks fail.
- Remove O_2 face mask or bag–valve–mask to >1m.
- Leave O_2 circuit connected for an SGA or tracheal tube.
- Continue compressions for 2min until the next rhythm check.
- Use three 'stacked' shocks for a witnessed VF/pVT where a defibrillator is immediately available, e.g. cardiac catheter lab.
- Precordial thump has a low chance of success if no defibrillator available.
- Change pad position (e.g. to anterior–posterior) if VF/pVT persists.

Airway management and ventilation

- Assess and open the airway; use head-tilt, chin-lift and/or jaw thrust.
- Use an oropharyngeal or nasopharyngeal airway if needed.
- Use suction to clear secretions and gastric contents.
- Give 100% inspired O_2 during CPR. After ROSC, target SpO_2 94–98%.
- Use a stepwise approach (bag–valve–mask, SGA or tracheal tube). Use whichever technique works best to deliver high-quality CPR with minimal interruption to compressions.
- When the patient's trachea is intubated, give continuous compressions at about 120/min and ventilate the lungs at ten breaths/min without pausing compressions. This can also be done with an SGA when there is a good seal.
- Use waveform capnography to confirm correct tracheal tube placement. Remember even in cardiac arrest, 'No trace = wrong place'.
- Use front of neck access for airway (see pp. 381–3) if unable to oxygenate.

Drugs in cardiac arrest

- Give 1mg of adrenaline IV or IO every 3–5min (10mL of 1:10 000 solution) during cardiac arrest.
- For VF/pVT, delay 1st dose of adrenaline until after the 3rd shock.
- Give amiodarone 300mg IV or IO after three shocks for VF/pVT.
- Lidocaine 100mg can be used if amiodarone is not available.

Treat reversible causes

- Use the '4 Hs and 4Ts' approach: hypoxia, hypovolaemia, hyperkalaemia (and other metabolic disturbances), hypothermia, thrombosis, tension pneumothorax, tamponade and toxins.
- Consider anaesthesia-specific causes: malignant hyperthermia, LA toxicity, bone cement implantation syndrome, CO_2/air/amniotic fluid embolism, anaphylaxis, adverse drug reactions.

- Use ultrasound/echocardiography if skilled to do so.
- If available, the use of extracorporeal circulatory support (ECMO) can be used to restore circulation and buy time to treat the underlying cause.

Modifications during anaesthesia

- Alert the theatre team and call for help early.
- Start chest compressions immediately.
- Discontinue surgery if no surgical cause.
- Initially give small incremental doses of IV adrenaline (50 micrograms) and use standard dosing of 1mg every 4min if required.
- Rapidly identify and treat reversible causes (e.g. airway problems, retraction and vagal tone, haemorrhage).
- Chest compressions can be started by compressing between the scapulae for patients in the prone position.
- If the patient is not supine, emergency repositioning is required into the supine position to continue resuscitation.

Further reading

Resuscitation Council UK (2014). *Management of cardiac arrest during neurosurgery in adults*. https://www.resus.org.uk/sites/default/files/2020-05/CPR_in_neurosurgical_patients.pdf
European Resuscitation Council. https://www.erc.edu
Resuscitation Council UK. https://www.resus.org.uk

Post-resuscitation care

Following cardiac arrest, ROSC is the 1st step in what may be a prolonged period of treatment. Unless the duration of cardiac arrest is very short, the patient will be unconscious and is likely to develop the post-cardiac arrest syndrome, which is associated with a marked systemic inflammatory response. The anaesthetist may be expected to initiate treatment in the ED, the operating room, the critical care unit or on the general ward. The aims of post-resuscitation care are to:

- Prevent a further cardiac arrest
- Define and treat the underlying disease process
- Limit organ damage
- Predict non-survivors.

Prevention of further cardiac arrest

- Optimise oxygenation. If fully conscious following a short-duration cardiac arrest, give O_2 via a face mask.
- Most patients will require assisted ventilation via a tracheal tube.
- After ROSC, once SpO_2 can be measured reliably or ABG values are obtained, titrate the inspired O_2 to achieve a saturation of 94–98% or PaO_2 of 10–13kPa.
- Provide ventilation to maintain normocarbia. Excessive ventilation will cause hypocarbia and may cause cerebral ischaemia from cerebral vasoconstriction.
- Maintain sedation with a propofol infusion, combined with a short-acting opioid.
- Avoid hypotension (MAP <65mmHg). Target MAP to achieve adequate urine output (>0.5mL/kg/h) and normal or decreasing lactate.
- Correct electrolyte disturbances, particularly K^+, Mg^{2+} and Ca^{2+}.
- Control blood glucose—treat blood glucose with insulin if it exceeds 10mmol/L; maintain in the range of 4–10mmol/L.

Define and treat the underlying disease process

- Establish the patient's pre-arrest medical condition.
- Confirm correct placement of ETT and exclude pneumothorax with CXR ± ultrasound.
- Perform early echocardiography in all patients in order to detect any underlying condition and quantify myocardial dysfunction.
- Consider CT brain and CT pulmonary angiography.

Limit organ damage

- ST-elevation on the 12-lead ECG is an indication for urgent coronary angiography and PCI if indicated. In patients without ST-elevation on the ECG, consider urgent coronary angiography if there is a high probability of acute coronary occlusion (e.g. patients with haemodynamic and/or electrical instability).
- Treat patients remaining comatose after ROSC with targeted temperature management. Maintain a constant target temperature between 32°C and 36°C for at least 24h.
- Avoid fever for at least 72h after ROSC in patients who remain in coma.
- Control seizures with propofol, benzodiazepines, levetiracetam or sodium valproate.

Prediction of non-survivors

- About 8% of all those sustaining an out-of-hospital cardiac arrest will survive to hospital discharge; the figure for in-hospital cardiac arrest is about 23%.
- Short duration of cardiac arrest/CPR achieves better neurological outcomes.
- Prognostication in unconscious patients is unreliable for at least 72–96h after ROSC.
- Ensure that sedative drugs have cleared and use multiple modalities: clinical examination, imaging (CT and/or MRI), biomarkers (e.g. neuron-specific enolase) and electrophysiology (EEG, somatosensory evoked potentials).
- Myocardial, neurological and other organ function may all improve slowly, given appropriate support over a period of time—at least 3–7d of intensive care should be considered in the comatose patient with ROSC following cardiac arrest.

Further reading

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Severe bradycardia

(See Fig. 39.3.)

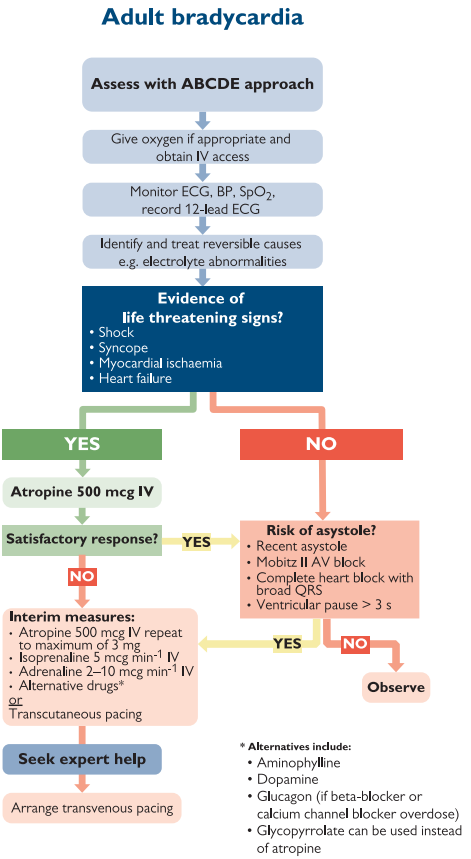

Fig. 39.3 Resuscitation Council UK—adult bradycardia algorithm. Reproduced with the kind permission of the Resuscitation Council UK.

- During anaesthesia, stop surgical stimulus (e.g. eye retraction/pneumoperitoneum).
- If bradycardia persists, give atropine 0.5mg IV increments up to 3mg. Repeat atropine every 3–5min up to 3mg.
- Alternatively, use glycopyrronium in 50–100 microgram increments.
- If ineffective, consider other drugs (e.g. adrenaline 2–10 micrograms/min IV or isoprenaline 5 micrograms/min) or temporary pacing.
- If transcutaneous pacing, check for electrical capture on the ECG and that there is an associated pulse. The patient may need analgesia and/or sedation.
- Start chest compressions and follow ALS algorithm if brady-asystole that is unresponsive to treatment.
- Discuss early with cardiology regarding temporary transvenous pacing and/or urgent permanent pacemaker insertion if immediately available.
- Even without adverse features, a period of recent asystole, Mobitz type 2 block, complete heart block and ventricular pauses >3s is high risk. These patients need monitoring and consideration of pharmacological treatment or pacing.

Consider reversible causes of bradycardia

MI, AF, post-cardiac surgery, drugs/toxins, electrolyte abnormalities, hypothermia, hypothyroidism, hypovolaemia, hypoxaemia and infections (e.g. Lyme).

Specific antidotes

Ca^{2+} channel blocker overdose

- Give 10% calcium gluconate 1–2g or 10% calcium chloride 3–6g over 10–20min.

β-blocker overdose

- Give glucagon 3–10mg IV, then 3–5mg/h IVI. Consider high-dose insulin 1 unit/kg bolus, followed by 0.5 units/kg/h.

Digoxin toxicity

- Give digoxin antibody fragment; one vial binds ~0.5mg of digoxin.

Further reading

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European Resuscitation Council. https://www.erc.edu

Resuscitation Council UK. https://www.resus.org.uk

Tachycardia

(See Fig. 39.4.)

Unstable tachycardias

- For sinus tachycardia, ensure adequate anaesthesia and analgesia.
- Check ABCDE. Rapidly correct hypoxia, hypovolaemia and electrolyte abnormalities (K^+, Mg^{2+}).
- In otherwise normal patients, a ventricular rate of up to 150/min is normally well tolerated, but patients with impaired function may decompensate at lower rates.
- For all tachycardias, assess for life-threatening adverse features: syncope, shock, myocardial ischaemia and heart failure. If present, perform synchronised direct current cardioversion. Patient may need sedation/GA.
- For direct current cardioversion, use 70–120J for atrial flutter and narrow complex tachycardia, and 120–150J for broad complex tachycardia and AF.
- If direct current cardioversion fails, give 300mg IV amiodarone over 10–20min, then reattempt direct current cardioversion. Follow this with 900mg IV amiodarone over 24h.
- If cardiac arrest or pulseless VT, start ALS algorithm.
- If the patient is stable, aim to use pharmacological methods 1st line. There is usually time to seek expert help (see ➲ pp. 147–51).

Stable, regular broad complex tachycardia

- This is likely to be VT or a supraventricular rhythm with a bundle branch block. Treat with amiodarone. Seek expert help.

Stable, irregular broad complex tachycardia

- Likely AF with a bundle branch block. Most cases should be treated as AF (see ➲ pp. 149–51).
- This may be polymorphic VT (i.e. torsade de pointes), but this is likely to be seen with adverse features or cardiac arrest. Treat with magnesium sulfate 2g IV and seek expert help.

Stable, regular narrow complex tachycardia

- Sinus tachycardia is a physiological response. Identify the underlying cause and treat.
- Paroxysmal SVT (AV nodal re-entry tachycardia and AV re-entry tachycardia) represents abnormal conduction pathways allowing rapid ventricular rate. Atrial activity is often not visible on the ECG.
- Atrial flutter with variable AV conduction presents as a narrow complex tachycardia. The atrial flutter rate is classically 300/min, and so a ratio of 2:1 conduction gives a ventricular rate of 150/min.
- Try vagal manoeuvres.
- If ineffective, give IV adenosine, starting with 6mg, then 12mg (repeat 12mg; if necessary, consider 18mg).
- Differentiating narrow from broad complex tachycardia can be difficult, especially at high ventricular rates. Vagal manoeuvres or adenosine should slow AV conduction of an SVT, but not a VT.

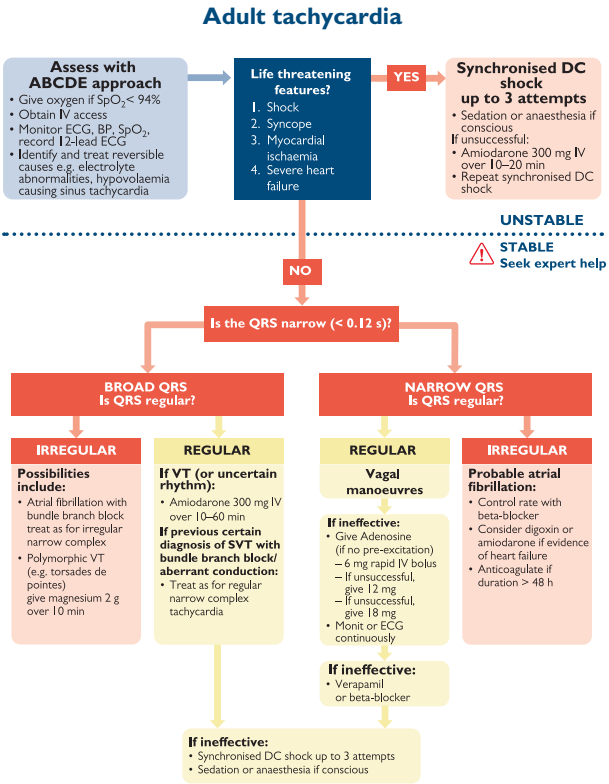

Fig. 39.4 Resuscitation Council UK—adult tachycardia algorithm. Reproduced with the kind permission of the Resuscitation Council UK.

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- Theophylline interacts with adenosine and tends to block its effect.
- Dipyridamole and carbimazole potentiate the effects of adenosine.
- Adenosine should be used with caution in WPW syndrome and should be avoided in asthmatics. Warn patients of the transient unpleasant sensation before adenosine administration.
- If adenosine is ineffective, try IV verapamil 5–10mg or short-acting IV β-blocker (esmolol or metoprolol).
- Do not use verapamil and a β-blocker together.

Stable, irregular narrow complex tachycardia

- Narrow complex tachycardia in an unstable patient.
- Correct rate with a short-acting β-blocker IV.
- Consider amiodarone or digoxin in heart failure.
- AF may require anticoagulation of the patient prior to cardioversion (patients with AF >48h). Discuss with cardiologists.

Further reading

European Resuscitation Council. https://www.erc.edu
European Society of Cardiology. https://www.escardio.org/
Resuscitation Council UK. https://www.resus.org.uk

Severe hypotension in theatre

Patient factors	Hypovolaemia Obstructed venous return ↑ intrathoracic pressure Anaphylaxis Embolus Cardiac failure and dysrhythmia Severe sepsis or septic shock
Anaesthetic factors	Measurement error Excessive depth of anaesthesia High regional block Drug error
Action	O_2 Look for cause; A, B, C, D Trendelenburg if appropriate IV fluid challenge Vasoconstrictors/inotropes Treat cause
Investigations	ECG, ABGs, echocardiography, troponin, CXR

Risk factors

- Preoperative fluid deficit (dehydration, diarrhoea and vomiting, blood loss).
- Mediastinal/hepatic/renal surgery (blood loss and caval compression).
- Pre-existing myocardial disease/dysrhythmia.
- Multiple trauma.
- Sepsis.
- Carcinoid syndrome with liver or lung tumour/metastases (bradykinin).
- ACE inhibitors and ARBs.

Differential diagnosis

- Measurement error: clinical assessment and manual palpation of the distal pulse, while repeating NIBP. Check when pulsation returns against the monitor deflation figure. If using intra-arterial BP, check the transducer height is at the level of the heart.
- Check peripheral perfusion; warm peripheries may suggest sepsis.
- Raised intrathoracic pressure can be caused by a pneumothorax or capnothorax (during laparoscopic surgery where the diaphragm is breached by insufflated gas). If tension pneumothorax is suspected, (particularly following central line insertion and IPPV), the trachea will be shifted away from a hyperresonant lung field, which will have diminished breath sounds. Neck veins may be engorged. Treat immediately by decompressing the pleural cavity with a large-bore cannula placed in the 2nd intercostal space in the mid-clavicular line, or a thoracostomy.

- Suspect hypovolaemia if the patient has HR >100bpm, RR >20 breaths/min, capillary return >2s, cool peripheries, collapsed veins, a narrow and peaked arterial line trace or marked respiratory swing on either CVP or arterial line trace. Dehydration may be indicated by thirst, a dry tongue and dark, concentrated urine, and elevated blood cell counts, urea, creatinine and electrolytes.
- Suspect cardiac failure if the patient has HR >100bpm, RR >20 breaths/min, engorged central veins, capillary return >2s, cool peripheries, pulmonary oedema or worsening SpO_2 with fluid challenge.
- Suspect an embolic event (e.g. gas/thrombus/cement/fat/amniotic embolus) if sudden ↓ $ETCO_2$, ↓ SpO_2, loss of palpable pulse, PEA and rise in CVP. Air or gas embolus likely if there is an open venous bed above the level of the heart. Suspect cement or fat embolus if temporarily related to intramedullary surgery or cementing.
- Suspect fat embolus or cement reaction in the presence of multiple bony injuries or long bone.
- Drug-related: iatrogenic drug responses (e.g. in patients with porphyria—see ➔ pp. 260–2; or histamine release) and drug errors (e.g. wrong dilution or LA toxicity).
- High central neural blockade (including unexpected central spread from peribulbar/interscalene). May be heralded by Horner's syndrome (small pupil, ptosis, stuffy nose, anhidrosis).
- Anaphylaxis: hypotension 1st feature in 46%, bronchospasm/high airway pressure (18%), tachycardia (9.8%), cyanosis/O_2 desaturation (4.7%), bradycardia (3%), reduced/absent capnography trace (2.3%) and cardiac arrest (1.2%).

Immediate management

ABCDE

- Check what the surgeons are doing (caval compression/blood loss/high pneumoperitoneal pressure); prevent further blood losses by clamp or direct pressure.
- Administer high FiO_2.
- Maintenance of organ perfusion and oxygenation is more important than achieving BP alone. BP = SVR × CO; therefore, improvement in CO (CO = stroke volume × HR) may help ameliorate low perfusion pressure.

Call for help early

- Including asking for massive haemorrhage protocol if bleeding.

Optimise preload

- If CVC sited, observe change in CVP from baseline and response to fluid challenges. Trend is more informative than actual number.
- Lifting the legs to 20° (passive leg raise), or the Trendelenburg position, returns blood into the central venous compartment, thus increasing stroke volume.
- Fluid challenge of 10mL/kg of crystalloid/colloid.
- Using a vasopressor infusion.
- Give blood and blood products for massive haemorrhage. Correct coagulopathy. Give tranexamic acid.

- After each intervention, assess response (BP/HR/CVP) and repeat if appropriate.

Increase contractility

- Ephedrine 6mg IV (mixed direct and indirect action); adrenaline 10 micrograms IV (β1,2 and α activity).
- Consider calcium slow IV (up to 10mL of 10% calcium chloride).

Systemic vasoconstriction

- Note: α-agonists increase perfusion pressure but may reduce CO. Metaraminol 1–2mg IV; phenylephrine 0.25–0.5mg IV; adrenaline 10 micrograms IV, noradrenaline infusion.

Subsequent management

- 12-lead ECG and troponin to check for myocardial injury.
- FBC/ABG/HemoCue® to check for anaemia.
- Correct acidosis to improve myocardial response to inotropes. Check ABGs, and correct respiratory acidosis first. If a severe metabolic acidosis exists (arterial pH <7.1, base excess ≤10mmol/L), consider using bicarbonate 50mmol (50mL of 8.4% sodium bicarbonate).
- Maintenance infusion of vasoconstrictor (e.g. adrenaline or noradrenaline) or inotrope (e.g. dobutamine), if required.

Other considerations

- **Adrenaline** 1:10 000 = 100 micrograms/mL; dilute in 10mL of 0.9% sodium chloride to get a 1:100 000 solution (10 micrograms/mL).
- Patients taking β-blockers may not demonstrate tachycardia, despite significant hypovolaemia.

Severe hypertension in theatre

Consider	Inadequate depth of anaesthesia/analgesia Measurement error Hypoxia/hypercapnia Iatrogenic drug error Pre-eclampsia Raised ICP Thyroid storm Phaeochromocytoma Carcinoid syndrome
Action	Stop surgery until controlled Confirm readings Increase depth of anaesthesia and analgesia Vasodilators, β-blockade, α-blockade, calcium channel blockers as required
Investigations	ECG, troponin, thyroid function tests, 24h urinary catecholamine metabolite excretion or plasma catecholamine metabolites

Risk factors

- Untreated or 'white coat' hypertension preoperatively (↑ lability).
- Aortic surgery (cross-clamp may ↑↑ SVR).
- Drugs: MAOIs (plus pethidine), ketamine, ergometrine.
- Family history of MEN (type 2) syndrome, medullary thyroid carcinoma, Conn's syndrome.
- Acute head injury.

Differential diagnosis

- Hypoxia/hypercapnia: go through ABC, and check for patient colour and SpO_2.
- Inadequate depth of anaesthesia: check volatile agent concentration; sniff test (smell gases); check TIVA pump, line and IV cannula.
- Inadequate analgesia: if in doubt, administer opioid (e.g. alfentanil 10–20 micrograms/kg) and observe effect. Consider remifentanil infusion.
- Measurement error: palpate the distal pulse manually, while repeating NIBP; check when pulsation returns against the monitor deflation figure. Invasive BP: check the transducer height.
- Iatrogenic drug response: cocaine, wrong drug (such as ephedrine and adrenaline), or wrong dilution (remember surgical drugs, e.g. adrenaline with LA, Moffett's solution, phenylephrine).
- Pre-eclampsia: if over 20w pregnant, check for proteinuria, platelet count ± clotting studies and LFTs.
- Thyroid storm causing elevated T_4 and T_3 levels.
- Phaeochromocytoma causing elevated plasma catecholamine levels.
- Cushing response: hypertension and reflex bradycardia (baroreceptor-mediated). This intracranially mediated response maintains cerebral perfusion in the presence of ↑ ICP (see pp. 559–60).

Immediate management

- **ABCDE approach**: identify and treat the underlying cause.
- **Keep calm** and aim for an acceptable safe BP value based on patient's age and comorbidities.
- Avoid alternating between severe hypertension and severe hypotension. The aim is to minimise myocardial stress leading to MI, arrhythmia or failure (Takotsubo cardiomyopathy), and to minimise the risk of hypertensive stroke and surgical bleeding.
- If difficult BP to control, insert an arterial line for continuous BP monitoring early.
- Consider the following drug options to decrease SVR and control HR. The choice of drug will depend on availability:
  - **Vasodilators** (may cause tachycardia): ↑ volatile concentration, but beware of increasing desflurane which may cause sympathetic activation at >1.5 MAC. Hydralazine 5mg slow IV every 15min. GTN (50mg/50mL; start at 3mL/h, and titrate to BP) or sodium nitroprusside (50mg/50mL and titrate with care for fine control). Magnesium sulfate 2–4g slow IV (8–16mmol) over 10min, followed by infusion of 1g/h.
  - **β-blockade** (particularly in the presence of ↑ HR or dysrhythmias): esmolol 25–50mg bolus doses, then 50–200 micrograms/kg/min. (Note that esmolol is supplied as 10mg/mL and 250mg/mL solutions.) Labetalol 5–10mg IV PRN (1–2mL increments from a 100mg/20mL ampoule). β:α block ratio = 7:1.
  - **α-blockade** (particularly in the presence of normal or ↓ HR): phentolamine 1–2mg IV PRN (10mg ampoule made up to 10mL).
  - **Calcium channel blockade** with IV nicardipine in 2.5mg increments.

Subsequent management

- Phaeochromocytoma crisis, or thyroid storm, may require prolonged treatment and ICU admission. Liaise with endocrinologist to ensure the correct samples are taken for investigation, and further treatments (e.g. antithyroid drugs, iodine, steroids for thyroid storm).
- Check for evidence of MI and myocardial injury (troponin, 12-lead ECG, echocardiography).
- Patient may require longer-term medication for BP. For phaeochromocytoma, this includes an α-blocker (phenoxybenzamine or doxazosin), and if they have tachycardia, a β-blocker.

Severe hypoxia in theatre

Equipment problems	Incorrect flow meter settings Machine or O_2 failure Disconnection Inadequate peripheral perfusion
Failure to ventilate	Hypoventilation Misplaced ETT Airway obstruction ↑ airway resistance ↓ FRC Atelectasis
Shunt	Airway secretions ↓ hypoxic pulmonary vasoconstriction Pulmonary oedema Aspiration of gastric contents/blood Pre-existing pathology
Poor O_2 delivery	Systemic hypoperfusion Embolus
Action	100% O_2 Call for help Check airway and ventilation; lungs to machine Check for cardiovascular cause
Investigations	SpO_2, capnography, temperature, CXR, ABG, FOB

Risk factors

- Reduced FRC (obesity, intestinal obstruction, pregnancy) reduces O_2 reserves.
- Failure to preoxygenate exacerbates airway difficulties at induction.
- Laryngospasm can result in negative-pressure pulmonary oedema.
- Head and neck surgery (shared access to the airway) increases the risk of undetected disconnection.
- History of CHD or detection of a heart murmur (left-to-right communication).
- Chronic lung disease.
- Methaemoglobinaemia (interpreted as deoxyhaemoglobin by pulse oximeters).
- Systemic absorption of patent blue dye.

Differential diagnosis

- Inappropriate FiO_2: use an O_2 analyser at all times.
- Signal error: reduced peripheral perfusion in hypovolaemic, hypotensive, hypothermic patients or AF, Raynaud's. Does the patient appear cyanotic?
- Obstruction of airway equipment: check for kinks (e.g. during positioning of gag during tonsillectomy), foreign bodies in filter/ETT, water in circuit.
- Ventilation problem: is ETT same depth as it was? Has it been dislodged? Look for chest movement with auscultation over the stomach and in both axillae. Is the patient hypoventilating? Look at minute ventilation

(common after opioids or insufflation of abdomen during laparoscopy—take note of the insufflation pressure). Has the capnograph waveform changed, indicating obstruction, restriction or hypoperfusion? Has the peak ventilatory pressure changed? Common in Trendelenburg position, laparoscopy or endobronchial placement of ETT.

- ↑ airway resistance (laryngospasm, bronchospasm).
- Aspiration/airway secretions: aspirate with tracheal suction catheter.
- Tension pneumothorax: suspect, particularly following CVC insertion and accompanied by cardiovascular signs.
- Hypovolaemia: HR >100bpm, RR >20 breaths/min, capillary return >2s, cool peripheries, a narrow and peaked arterial line trace or marked respiratory swing to either CVP or arterial line trace (quantifiable using PPV; >10% suggests hypovolaemia).
- Cardiac failure: HR >100bpm, RR >20 breaths/min, engorged central veins, capillary return >2s, pulmonary oedema or ↓ SpO_2 with fluid.
- Air or gas embolus: pre-existing low CVP and open venous bed. Signs include sudden ↓ $ETCO_2$, ↓ SpO_2, loss of palpable pulse, PEA and subsequent rise in CVP.
- Other embolic events (thrombus/cement/fat/amniotic fluid) during at-risk surgery (intramedullary surgery, cementing, obstetrics).
- Cardiac shunt (e.g. VSD, ASD plus ↓ SVR with reversal of flow).
- MH: especially if accompanied by ↑ $ETCO_2$, ↑ RR, ↑ HR and ↑ ectopics.
- Anaphylaxis: CVS collapse, bronchospasm, angio-oedema, erythema, rash, urticaria.
- Second gas effect: especially in recovery after using N_2O.

Immediate management

Administer 100% O_2.

ABC

Manual ventilation gives tactile feedback. Perform recruitment manoeuvre, e.g. 30cmH_2O for 40s (be alert for cardiovascular effects, e.g. hypotension during recruitment). Expose the chest, breathing circuit and airway connections. If steep head-down, consider supine position until hypoxia resolved.

Bronchospasm

Treat by increasing the volatile agent concentration and IV salbutamol (250 micrograms) (see pp. 1078–9). Sometimes no wheeze will be heard as bronchospasm severely limiting air entry; eliminate ETT obstruction by gently passing a suction catheter through, then treat as above.

Misplaced ETT

Confirm ETT position at teeth, and look for rise and fall of the chest with auscultation over the stomach and in both axillae and the capnograph trace. Direct or videolaryngoscopy to ensure ETT through cords.

Ruling out equipment problems

Switching to self-inflating bag with alternative O_2 supply immediately rules out machine and circuit problems, but ensure means of keeping patient anaesthetised without inhalational agent. This does not rule out problem with ETT or filter/HME. If in doubt about the inspired O_2 concentration, use a separate cylinder supply (as a last resort, use room air via a self-inflating bag = 21% O_2). The source of a leak or obstruction is not as important as oxygenation of the patient. Make the patient safe first, then use a systematic approach.

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Hypotension/hypovolaemia

Use fluid/blood/vasopressors as needed. During laparoscopy, check insufflation pressures and ask surgeon if they can be reduced if needed.

Severe right-to-left shunt

Severe hypoxia occurs when blood starts flowing through a congenital heart defect in the presence of low SVR, thus bypassing the pulmonary circulation. The resultant hypoxaemia exacerbates the problem by causing hypoxic pulmonary vasoconstriction which increases PVR and the tendency for blood to shunt across the cardiac defect. Treatment is therefore twofold: (1) to increase SVR by lifting the legs/Trendelenburg and giving adrenaline plus IV fluid, especially in sepsis; and (2) to minimise PVR by removing PEEP, avoiding high intrathoracic pressure and maximising FiO_2.

Other considerations

- In chronic bronchitis, the bronchial circulation can shunt up to 10% of the CO.
- The foramen ovale remains patent in 20–30% of patients but is normally kept closed, because the left atrial pressure is usually higher than the right atrial pressure. IPPV, PEEP, breath-holding, CCF, thoracic surgery and PE can reverse the pressure gradient and result in shunt.
- Always check the SpO_2 probe is well positioned and has a good trace. However, this should not be presumed to be the offending problem until other causes have been excluded!

Severe laryngospasm

Condition	Acute glottic closure by the vocal cords
Presentation	Crowing or absent inspiratory sounds and marked tracheal tug
Immediate action	100% O_2, CPAP Larson's manoeuvre (vigorous jaw thrust) Remove irritants from the airway Deepen anaesthesia
Follow-up action	Muscle relaxation if intractable
Also consider	Bronchospasm Laryngeal trauma/airway oedema Recurrent laryngeal nerve damage Tracheomalacia Inhaled foreign body Epiglottitis; croup

Risk factors

- Light anaesthesia, especially in anxious patients.
- Intense surgical stimulation: anal stretch, cervical dilation, etc.
- Extubation of a soiled airway.
- Thyroid surgery.
- Hypocalcaemia (neuromuscular irritability).
- Multiple crowns, poor dentition (inhaled foreign body).

Immediate management

(See ➲ p. 370.)

- Remove the stimulus that precipitated the laryngospasm.
- Check that the airway is clear of obstruction or potential irritants, including airway adjuncts in the light patient.
- Administer 100% O_2 and CPAP.
- If unsuccessful, deepen anaesthesia with propofol or sevoflurane.
- If this fails, muscle relaxant is required. Suxamethonium 0.25–0.5mg/kg IV or 3mg intralingual/submental or 4mg/kg IM thigh.
- Monitor for pulmonary oedema. Decompress stomach with an OGT.

Other considerations

- Risk may be reduced by co-induction with IV opioids, IV lidocaine, or by topical lidocaine spray prior to laryngoscopy.
- Unilateral recurrent laryngeal nerve trauma results in paralysis of one vocal cord and causes hoarseness, ineffective cough and the potential to aspirate. Bilateral vocal cord paralysis is more serious, leading to stridor on extubation. This may mimic laryngospasm but does not get better with standard airway manoeuvres. The patient will require reintubation, and possibly tracheostomy.
- Tracheomalacia is likely to cause more stridor with marked negative inspiratory pressure, so treat initially with CPAP.

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Air/gas embolism

(See also ➲ pp. 584–5.)

Condition	Venous gas produces an airlock in RV and obstructs pulmonary capillaries
Presentation	↓ $ETCO_2$ ↓ SpO_2 Hypotension and loss of palpable pulse ↑ CVP, then ↓ CVP PEA
Immediate action	Stop insufflating gas Ensure open wound lower than heart Flood wound and compress drainage veins
Follow-up action	↑ venous pressure; turn off N_2O; left lateral head-down tilt; CVS support
Investigations	Auscultation; Doppler; ECG; CXR
Also consider	Breathing circuit disconnection Other causes of PEA (4Hs and 4Ts) Cement reaction Pulmonary thromboembolism AFE

It is claimed that symptoms/signs of an air embolus appear following 0.5mL/kg/min of intravascular gas.

Risk factors

- Patient: spontaneously ventilating (negative CVP), patent foramen ovale (risk of paradoxical emboli).
- Anaesthesia: hypovolaemia, any open vascular access point, operation site higher than the heart, pressurised infusions.
- Orthopaedic surgery: multiple trauma, long bone surgery (especially intramedullary nailing), hip surgery, shoulder surgery in beach chair.
- General surgery: laparoscopic procedures, hysterectomy, neck surgery, vascular surgery, middle ear procedures.
- Neurosurgery: posterior fossa operations in the sitting position (almost historical).

Diagnosis

- High index of suspicion is needed in the 'at-risk' patient.
- Respiratory: dramatic fall/loss of the $ETCO_2$ trace and fall in SpO_2.
- CVS: severe chest pain if awake, tachycardia, arrhythmia, sudden ↑ CVP due to fall in CO and rise in PVR.
- PEA arrest may occur. ECG may show signs of acute ischaemia, e.g. ST-segment depression of >1mm.
- Neurological: CVE-like symptoms, failure to wake up.
- Classically, a 'millwheel' murmur can supposedly be heard.
- Doppler ultrasound can detect 0.25mL of air.

Immediate management

ABC

- Eliminate possibility of breathing circuit disconnection; give 100% O_2; check the ECG trace and pulse.
- Increase venous pressure with rapid IV fluids ± vasopressors.
- If PEA arrest occurs, commence the ALS protocol for non-VF/VT cardiac arrest.

Prevent further gas/air entrainment

- Surgeon to apply compression to major drainage vessels, flood the surgical field with irrigation fluid or cover with damp pack, stop reaming, etc.
- Decompress any gas-pressurised system/cavity, e.g. the abdomen during laparoscopy.
- Lower the operation site to below the heart level.

Turn off N2O

- N_2O will expand any intravascular gas volume.

Central venous line

- Classic teaching is to tip the patient head-down in the left lateral position, to keep the bubble of gas in the right atrium or apex of the RV. It can then be aspirated via a central line advanced into the right atrium or will eventually dissolve. In practice, even if a CVP line is *in situ*, aspiration is likely to be difficult.

Moderate CPAP

- Advocated as a means of rapidly increasing the intrathoracic pressure, and therefore CVP, in the event of a gas embolus. While this manoeuvre may limit the extent and progress of an air embolus, it must be borne in mind that 10% of patients may have a patent foramen ovale. Sustained rise in right atrial pressure may then lead to a right-to-left shunt and paradoxical air embolism to the cerebral circulation.

Subsequent management

- Ask the surgeon to apply bone wax to exposed bone edges.
- Correct any pre-existing hypovolaemia.
- Perform a 12-lead ECG to look for ischaemia. Air in coronary arteries is suggestive of paradoxical air embolism.
- Consider hyperbaric therapy, if available. ↑ ambient pressure (3–6 bar) will decrease the volume of gas emboli.

Other considerations

CO_2 is the safest gas to use for laparoscopic insufflation. It is non-flammable and more soluble than other agents. Should a gas embolus occur, it will dissolve over time. The priority of management should therefore be to limit the extent and central progress of the gas 'bubble', thereby minimising its systemic CVS effect.

Aspiration

Condition	Chemical pneumonitis; foreign body obstruction and atelectasis
Presentation	↓ SpO_2, ↑ RR, ↑ HR, ↓ lung compliance
Immediate action	Minimise further aspiration Secure the airway Suction
Follow-up action	100% O_2 and consider CPAP Empty the stomach
Investigations	CXR; bronchoscopy
Also consider	Pulmonary oedema Embolus ARDS

Risk factors

- Full stomach/delayed emptying (many causes, including opioids, recent trauma, DM, CKD).
- Known reflux.
- Raised intragastric pressure (intestinal obstruction, pregnancy, laparoscopic surgery, high BMI).
- Anaesthesia—topically anaesthetised airway, LMA (especially 1st generation), light anaesthesia.
- Oral/nasal surgery where 'coroner's clot' may be aspirated from nasopharynx on extubation.
- Steep Trendelenburg position.

Diagnosis

- Clinical: auscultation may reveal wheeze and crepitations.
- CXR: diffuse infiltrative pattern, especially in the right lower lobe distribution (but often not acutely).

Immediate management

- Avoid a GA in high-risk situations, if possible.
- Use of an RSI when appropriate.
- Administer 100% O_2, and minimise the risk of further aspirate contaminating the airway.
- If light anaesthesia: suction and put into recovery position.
- If under GA, suction; consider securing the airway with ETT ± cricoid pressure (controversial—avoid if actively vomiting, as risk of oesophageal rupture or when it distorts the view on laryngoscopy).

Subsequent management

- Empty the stomach with a large-bore NGT prior to extubation.
- Monitor respiratory function and arrange a CXR. Look for evidence of oedema, collapse or consolidation.
- If SpO_2 remains <90%, despite 100% O_2, there may be solid food material obstructing part of the bronchial tree. Consider performing a bronchoscopy.

- If maintaining SpO_2 with minimal requirements, extubate. May require post-extubation CPAP and chest physiotherapy.
- Refer to ICU if requiring support or concern regarding solid material in airway.

Other considerations

- The NAP4 audit found that aspiration was the leading cause of airway-related mortality. The risk was ↑ greatly for higher ASA status and emergency surgery. Thus, thorough preoperative assessment and appropriate planning are pertinent to reduce the risk.
- Corticosteroids may modify the inflammatory response early after aspiration, but do not alter the outcome, except by potentially interfering with the normal immune response.
- Prophylactic antibiotics are not generally given routinely (unless infected material aspirated) but may be required to treat subsequent 2° infections.
- If the gastric aspirate has been buffered to pH 7, the resulting aspiration pneumonitis is less severe, volume for volume, than if it is highly acidic. However, solid food material can produce prolonged inflammation, even if the overall pH is neutral.
- Consider aspiration of a blood clot in patients undergoing intraoral procedures with difficult ventilation and lack of capnograph trace. This is potentially life-threatening. Bronchial suction, exchange of airway, reintubation and bronchoscopy may be required to remove the clot.

Further reading

Cook T, Woodall N, Frerk C, et al. (2011). Aspiration of gastric contents and of blood. In: *4th National Audit Project of The Royal College of Anaesthetists and the Difficult Airway Society. Major complications of airway management in the United Kingdom. Report and findings*. Chapter 19, pp. 155–64.

Severe bronchospasm

Presentation	↑ airway pressure Sloping expiratory capnograph trace Wheeze or silent chest ↑ HR
Immediate action	100% O_2 Salbutamol 250 micrograms IV/2.5mg neb; aminophylline 250mg slow IV Magnesium sulfate 2g IV
Follow-up action	Hydrocortisone 200mg
Investigations	CXR; ABG
Also consider	Breathing circuit obstruction Kinked ETT/cuff herniation Endobronchial intubation/tube migration Foreign body in airway Anaphylaxis Pneumothorax

Risk factors

- Asthma; particularly with previous acute admissions, especially to ICU, and/or systemic steroid dependence.
- Paediatric: prematurity and LBW.
- Trigger exposure: smoke, allergens.
- Intercurrent respiratory tract infection.
- Carinal irritation by ETT.

Diagnosis

- ↑ airway pressure, ↑ expiratory phase of capnography waveform.
- Central trachea, with bilaterally hyperexpanded and resonant lung fields ± expiratory wheeze (absent if severe).
- Severe bronchospasm is a diagnosis of exclusion. The quickest method of ascertaining the source of ↑ airway resistance is to connect a self-inflating bag directly to the ETT (not HME) and manually ventilate. If the inflation pressure still feels too high, the problem is due to airway/ETT obstruction or reduced compliance.
- Eliminate ETT obstruction by passing a suction catheter. Perform gently to prevent further bronchospasm via carinal irritation.

Immediate management

ABC

- 100% O_2.

Increase the volatile agent concentration

- Sevoflurane is the least irritant and is less likely to precipitate dysrhythmias in the presence of hypercapnia. However, this may be insufficient in severe bronchospasm.

Specific treatment

- Salbutamol and aminophylline (see Table 39.1).

Table 39.1 Drug doses in bronchospasm

	Adult	Paediatric
Salbutamol: IV (slow)	250micrograms	15micrograms/kg
Salbutamol: nebulised	2.5–5mg every 15min	2.5–5mg every 15min
Salbutamol: puffs	Ten puffs	Ten puffs
Ipratropium bromide (nebulised)	0.25–0.5mg 4–6h	0.25mg 4–6h
Aminophylline (IV)	250mg slowly	5mg/kg slowly
Ketamine (IV)	2mg/kg	0.5–1mg/kg
Magnesium (IV)	2g slowly	40mg/kg slowly
Adrenaline (IV)	10micrograms	1microgram/kg
Hydrocortisone (IV)	200mg	4mg/kg

Subsequent management

- If immediate treatment fails, consider ipratropium bromide, adrenaline IV boluses, ketamine or magnesium (Table 39.1).
- Give hydrocortisone.
- Check for drug allergies to agents already administered.
- Arrange CXR—check for pneumothorax and ETT tip position (withdraw if carinal).
- Check ABGs and electrolytes (prolonged use of β2-agonists causes hypokalaemia).
- Refer to ICU.

Other considerations

- Gas trapping: ↑ mean intrathoracic pressure may result from IPPV in the presence ↑ expiratory time. If pulse pressure falls and neck veins appear distended, consider obstructed venous return and a dependent fall in CO. Disconnect the ETT from the circuit and push on chest; preload should be restored as intrathoracic pressure is ↓, and pulse pressure and V_T should improve.
- Ventilator settings in bronchospasm: 100% O_2, low RR and prolonged expiration (I:E ratio 1:3 or 1:4); accept ↑ $ETCO_2$, provided SpO_2 is adequate (permissive hypercapnia).

Further reading

British Thoracic Society, Scottish Intercollegiate Guidelines Network (2008). *British guideline on the management of asthma. A national clinical guideline*. Revised July 2019. https://www.brit-thoracic.org.uk/quality-improvement/guidelines/asthma/

Pulmonary oedema

Condition	↑ hydrostatic pressure ↑ vascular permeability ↓ plasma colloid osmotic pressure Negative interstitial pressure Obstructed lymphatic drainage
Presentation	Pink frothy sputum, ↑ HR, ↑ RR, ↓ SpO_2, ↑ CVP, ↑ PAOP
Immediate action	100% O_2, sit up/reverse Trendelenburg. Opioids, diuretics and vasodilators
Investigations	CXR; ECG; ABG; echocardiography
Also consider	Asthma MI ARDS Aspiration

Risk factors

- Cardiogenic: MI, severe hypertension, CCF.
- Non-cardiogenic: airway obstruction (negative pressure), neurogenic, sepsis, aspiration, pre-existing lung disease, impairment of lymphatic drainage and rapid lung expansion.

Diagnosis

- Clinical: wheeze; pink, frothy sputum; fine crackles; quiet bases; gallop rhythm; ↑ JVP; liver engorgement.
- ↑ HR; ↑ RR; ↓ SpO_2; ↑ airway pressure; ↑ CVP; ↓ PAOP.
- CXR: basal shadowing; upper lobe diversion; hilar haze; bronchial cuffing; pleural effusions; septal/interlobar fluid lines.
- ECG: evidence of right heart strain; evidence of MI.

Immediate management

ABC

- If awake and breathing spontaneously: sit up to offload the pulmonary vasculature and improve FRC; 100% O_2 via mask/HFNO/CPAP 5–10mmHg.
- If anaesthetised and intubated: commence IPPV with PEEP (5–10cmH_2O), 15° head-up position to reduce atelectasis and improve FRC; aspirate free fluid from the trachea intermittently.

Medication

- Furosemide 50mg IV.
- Diamorphine 5mg IV.
- Vasodilator if hypertensive (e.g. GTN 0.5–1.5mg sublingually or 10mg transcutaneous patch. IV GTN only if arterial line *in situ*).

Anaphylaxis

Condition	Immunoglobulin E-mediated type 1 hypersensitivity reaction to an antigen, resulting in histamine and serotonin release from mast cells and basophils
Presentation	↑ HR, ↓ BP, rash, wheeze, oedema
Immediate action	Remove trigger 100% O_2 Elevate legs and fluid resuscitation IV adrenaline 20–50 micrograms
Follow-up action	Chlorphenamine 10–20mg Hydrocortisone 100–300mg
Investigations	Plasma tryptase; directed allergy testing, ABG
Also consider	Airway obstruction Asthma Tension pneumothorax Anaphylactoid reaction Sepsis Histamine release (atracurium) Cardiogenic shock

Risk factors

- IV administration of the antigen.
- Note that cross-sensitivities with NSAIDs and muscle relaxants mean that previous exposure is not always necessary.
- True penicillin allergy is a reaction to the basic common structure present in most penicillins (the β-lactam ring).
- Antibiotics (46%), muscle relaxants (33%), chlorhexidine (10%) and patent blue are the most frequent triggers.

Diagnosis

- CVS collapse (51%)
- Erythema (45%)
- Bronchospasm (40%)
- Rash (13%)
- Angio-oedema (12%)
- Urticaria (8.5%).

Immediate management

ABC

- Stop any potential triggers, particularly IV agents. Be aware of chlorhexidine-coated central lines.
- Call for help.
- 100% O_2; maintain the airway and consider intubation.
- Lay the patient flat, with the legs elevated/Trendelenburg position.
- Give adrenaline in IV increments every 1–2min. Alternatively, give adrenaline IM, repeated after 5min, if necessary (Table 39.2).
- Give IV crystalloid fluids 20mL/kg.

Table 39.2 Drug doses in anaphylaxis

	<6mo	6mo to 6y	6–12y	>12y
Adrenaline (IM)	150 micrograms	150 micrograms	300 micrograms	500 micrograms
Adrenaline (IV)	1 microgram/kg	1 microgram/kg	1 microgram/kg	20–50 micrograms
Hydrocortisone (IV)	25mg	50mg	100mg	200mg
Chlorphenamine (IV/IM)	250 micrograms/kg	2.5mg	5mg	10mg

Subsequent management

- Adrenaline infusion (1st line): 0.05–0.1 micrograms/kg/min or noradrenaline 0.05–0.1 micrograms/kg/min (see ➲ p. 1209).
- Resistant hypotension: consider glucagon in those on a β-blocker (1–2mg IV every 5min); vasopressin (IV bolus 1–2 units, then 2 units/h).
- Antihistamines: consider PO non-sedating antihistamine when awake. Alternatively, IV/IM chlorphenamine; however, evidence to support their use is weak.
- Corticosteroids may help prevent or shorten protracted reactions.
- Consider bronchodilators (see ➲ p. 1071) for persistent bronchospasm.
- Pregnancy: consider left uterine displacement or CS within 4min if arrest or periarrest.
- Check for the presence of airway oedema by letting down the ETT cuff and confirming a leak prior to extubating.

Other considerations

- NMBAs are responsible for 60–70% of serious anaesthetic adverse drug reactions (AADRs), frequently on 1st contact.
- The quaternary ammonium group found in neuromuscular-blocking drugs is present in other drugs, foods, cosmetics and hair care products. Previous sensitisation is possible, predominantly in ♀ .
- Clinically, anaphylactic reactions may be indistinguishable from anaphylactoid responses. Isolated cutaneous erythema is commonly seen following IV thiopental or atracurium. If there are no further histaminoid manifestations, investigation is unwarranted.
- Timing is important. Onset is usually rapid following an IV drug bolus. Slower onset is expected if, for example, gelatin infusion, latex sensitivity or diclofenac suppository is responsible.

Further reading

Royal College of Anaesthetists (2018). *Anaesthesia, surgery and life-threatening allergic reactions. Report and findings of the Royal College of Anaesthetists' 6th National Audit Project: perioperative anaphylaxis*. https://www.nationalauditprojects.org.uk/NAP6Report#pt

Australian and New Zealand College of Anaesthetists (ANZCA) and Australian and New Zealand Anaesthetic Allergy Group (ANZAAG) (2016). *Perioperative anaphylaxis management guidelines*. https://www.anzca.edu.au/getattachment/ce1ac7fb-45c3-434c-9da9-7a61f14916b8/Anaphylaxis-management-guidelines

Resuscitation Council UK (2008). *Emergency treatment of anaphylactic reactions*. Revised 2016. https://www.resus.org.uk/anaphylaxis/emergency-treatment-of-anaphylactic-reactions/

Anaphylaxis follow-up

Investigation of reactions

Referral to anaesthetic allergy clinic

Referral to an allergy clinic is the responsibility of the anaesthetist and should include:

- Anaesthetic chart
- Drug chart
- Timings of all administered substances
- Tryptase results and timings.

A list of specialist clinics is available on the British Society for Allergy and Clinical Immunology website (https://www.bsaci.org).

Serum tryptase evaluation

Tryptase is a neutral protease released from secretory granules of mast cells during degranulation. *In vivo* half-life is 3h (compared with 3min for histamine), and it is stable in isolated plasma or serum.

- Take three venous blood samples—immediately after resuscitation, at 1–2h (not later than 6h) and baseline levels at 24h. Serum should be separated and stored at –20°C and sent to an appropriate laboratory.
- Basal plasma tryptase concentration is usually <11 nanograms/mL. Levels of up to 15 nanograms/mL are seen in pseudoallergy, e.g. anaphylactoid reactions. In patients with low baseline tryptase levels, even if levels remain within normal limits, a level of **>2 nanograms/mL + 1.2 × baseline** is indicative of anaphylaxis.

Radioallergosorbent/CAP tests

- Radioallergosorbent tests (RASTs) for antigen-specific immunoglobulin E antibodies have now been largely superseded by the CAP system (Phadia®).
- Currently, only helpful in confirming penicillin, suxamethonium, chlorhexidine and latex allergy. Sensitivity is low, and a negative result still requires skin testing.

Skin testing

- Diagnosing an AADR depends on skin prick test or intradermal testing. In proven neuromuscular-blocking drug anaphylaxis, no *in vitro* test has been shown to have comparable specificity and sensitivity.
- Tests should take place at 4–6w post-event to allow the regeneration of immunoglobulin E.
- Antihistamines should not have been given within the last 5d.
- Testing is required to all drugs given before the event. Remember antibiotics, latex, chlorhexidine and lidocaine, if mixed with propofol. Suspected LA allergy is best tested by challenge.
- Negative control is with 0.9% sodium chloride (to exclude dermographia). Positive control is with a commercially available histamine solution. The latter demonstrates a normal skin response. Wheal and flare give a reference for reactions to test drugs.
- Wheal >2mm wider than the 0.9% sodium chloride control is interpreted as positive. Positive test with undiluted drug is repeated with 1:10 dilution to reduce the chance of a false positive.

- Following a positive result, other drugs in the same pharmacological group are tested. In neuromuscular-blocking drug allergy, up to 60% of people may be sensitive to other relaxants.
- If there is a strong history, but negative skin prick test, diluted drugs can be tested by intradermal testing.

After testing

- The patient should be informed and given a letter describing the reaction and potential triggering agents used until testing is completed.
- An allergy alert should be added to the case notes, and treating GP, anaesthetist and proceduralist/surgeon informed.
- Patients may consider wearing a medical alert bracelet.
- Report the reaction to Medicines and Healthcare products Regulatory Agency (MHRA) ('yellow card system'). AADRs are currently under-reported.

Future anaesthesia

- Avoid all untested drugs related to the original culprit.
- Do not use IV 'test' doses; this is unsafe if a true allergy exists.

Further reading

Resources regarding follow-up of UK suspected anaphylactic reactions can be located at: https://www.nationalauditprojects.org.uk/NAP6-Resources?newsid=1256#pt

Laxenaire MC, Mertes PM (2001). Anaphylaxis during anaesthesia. Results of a two year study in France. *Br J Anaesth*, **87**, 549–58.

Fisher MM, Bowey CJ (1997). Alleged allergy to local anaesthetics. *Anaesth Intensive Care*, **25**, 611–14.

Latex allergy

Latex is derived from the sap of *Hevea brasiliensis* (rubber tree). Hev b proteins within latex act as the major allergens (there are 14 types—Hev b1–14). Latex, although previously commonly found in much anaesthetic and surgical equipment, has significantly lessened with the awareness of the prevalence of latex allergy. However, this should not result in complacency in preventing such reactions.

Classification of reaction

Irritant contact dermatitis

Non-allergic, irritant contact dermatitis, presenting over minutes to hours. Damage to skin due to exogenous substance causing irritation.

Contact dermatitis

Type 4 (delayed) hypersensitivity reaction, based on allergic sensitisation mediated by T-lymphocytes. Presents over 48–72h with an eczematous eruption. This can progress to lichenification and scaling on chronic exposure.

Type 1 hypersensitivity

Development of latex sensitivity is dependent on previous exposure. Immunoglobulin E-mediated type 1 hypersensitivity has been attributed to Hev b proteins in latex. The three main presentations are:

- Contact urticaria: particularly of health care workers, typically 10–15min following, and usually at the site of, exposure. This may develop into a more severe reaction
- Asthma and rhinitis: characterised with bronchospasm and secretions. Inhalation of airborne latex particles from powdered gloves has been implicated
- Anaphylaxis: this is more commonly encountered intraoperatively. IV and membrane inoculation are the commonest triggers; however, donning of gloves and indirect contact have also been described.

High-risk individuals

Eight per cent of the population are sensitised to latex; however, 1.4% of the population exhibit a latex allergy. Latex anaphylaxis appears to be more common in ♀. There are certain groups at particular risk of developing latex sensitivity.

Multiple surgical procedures

Patients with repeated exposure to latex have an ↑ risk.

Neural tube defects, including spina bifida

Incidence of latex sensitivity due to recurrent bladder catheterisation is 20–65%.

Associated medical conditions

Patients with atopy, asthma, rhinitis and severe dermatitis have an ↑ incidence of sensitivity.

Health care workers

Prevalence of sensitivity can be between 3% and 12%.

Occupation

Rubber industry workers, occupations involving the use of protective equipment (policemen, hairdressers, service food workers).

Fruit allergens

Patients allergic to fruit have an 11% risk of a latex reaction. Crossreactivity has been demonstrated with certain fruit allergens (banana, chestnut, avocado, passion fruit, tomato, grape, celery, peach, watermelon, cherry and kiwi fruit).

Prevention of latex anaphylaxis

- All team members need to be alerted if a patient has a latex allergy.
- The operating theatre should be prepared the night before as this reduces the number of latex particles in the air. The patient should be scheduled 1st on the list.
- 'Latex allergy' notices should be placed on theatre doors.
- Only use latex-free equipment.
- Remove non-essential equipment from the vicinity of the patient.
- Limit staff traffic during surgery.
- Resuscitation equipment must be latex-free.
- There is no evidence to support prophylactic use of antihistamines and corticosteroids.

Clinical features of latex anaphylaxis

Onset is normally 20–60min following exposure and progressively worsens over 5–10min. Treat as for anaphylaxis (see ➔ pp. 1081–3).

Further reading

Lieberman P, Nicklas RA, Oppenheimer J, *et al*. (2010). The diagnosis and management of anaphylaxis practice parameter: 2010 update. *J Allergy Clin Immunol*, **126**, 477–80.

Bousquet J, Flahault A, Vandenplas O, *et al*. (2006). Natural rubber latex allergy among health care workers: a systematic review of the evidence. *J Allergy Clin Immunol*, **118**, 447–54.

Suli C, Lorini M, Mistrello G, Tedeschi A (2006). Diagnosis of latex hypersensitivity: comparison of different methods. *Eur Ann Allergy Clin Immunol*, **38**, 24–30.

Cullinan P, Brown R, Field A, *et al*.; British Society of Allergy and Clinical Immunology (2003). Latex allergy. A position paper of the British Society of Allergy and Clinical Immunology. *Clin Exp Allergy*, **33**, 1484–99.

Hepner DL, Castells MC (2003). Latex allergy: an update. *Anesth Analg*, **96**, 1219–29.

ALGRAWANY

Intra-arterial injection

Condition	Unintentional intra-arterial injection of medication can cause: paraesthesiae, pain, motor dysfunction, compartment syndrome, gangrene and limb loss
Presentation	Intense burning pain on injection; distal blanching; blistering
Immediate action	Stop injection, label cannula, flush with 0.9% sodium chloride and maintain tissue perfusion
Follow-up action	Anticoagulation and specialist referral
Investigations	APTT
Also consider	Extravasation Dilution error of drug administered

Historically, barbiturates and benzodiazepines have been implicated in many of the inadvertent intra-arterial injections, but many drugs, including antihistamines and antibiotics, have resulted in serious harm.

Risk factors

- Arterial line *in situ* for BP monitoring.
- Multiple ports for IV and arterial lines.
- Cannulae placed under difficult circumstances (hypotensive, obese patients, emergent situation) and not flushed. If in doubt, take a blood gas from the line.
- Proximity of the brachial artery makes antecubital fossa cannulae more prone, but also aberrant ulnar or radial arteries can be cannulated at the wrist.
- Stronger drug concentration (e.g. 5% thiopental).

Pathophysiology

- Not well defined and may depend on medication but results in tissue ischaemia distal to injection site.
- One proposed mechanism: chemical endarteritis characterised by arterial vasospasm, local release of noradrenaline, crystal deposition within the distal arteries (thiopental), subsequent thrombosis and distal ischaemic necrosis.

Diagnosis

- Intense burning pain on injection that may last for several hours.
- Blanching of the skin and blistering.
- Within 2h: oedema, hyperaesthesia, motor weakness.
- Later: signs of arterial thrombosis ± gangrene, necrosis.

Immediate management

Empirical, aimed at maintaining distal perfusion and symptomatic relief.

- Stop injecting and keep the cannula *in situ*, labelled clearly, and place an IV cannula.
- If the drug administered was highly irritant, flush the vessel with 0.9% sodium chloride or heparinised 0.9% sodium chloride and then maintain cannula patency with slow infusion.
- Initiate anticoagulation with heparin (APTT 1.5–2) and discuss with vascular surgeon.
- Administer LA via the cannula to reduce vasospasm and pain.
- Give analgesia.
- Administer a vasodilator (e.g. papaverine 40mg) through cannula or consider prostacycline analogue (iloprost) IV.
- Once the immediate reaction has subsided, if the hand is well perfused and pink, remove the cannula and apply sufficient pressure to the puncture site to minimise local haematoma formation.
- Inform the patient of what has happened and document all actions clearly.

Subsequent management

- To reduce pain, consider a sympathetic blockade (via stellate ganglion or brachial plexus) or guanethidine block (consult a chronic pain specialist).
- Anticoagulation with heparin to reduce the risk of late arterial thrombosis.
- Re-establishment of blood flow to extremity if necessary.
- Treat sequelae of tissue hypoperfusion (fasciotomy/amputation/skin graft).
- Rehabilitation and follow-up.

Incomplete reversal of neuromuscular blockade

(See also ➲ pp. 420–2 and ➲ p. 425.)

Condition	Residual competitive antagonism at nicotinic acetylcholine receptor of neuromuscular junction
Presentation	Uncoordinated, jerky movements during the recovery phase. Inability to maintain an airway/inadequate minute ventilation
Action	Maintain and protect airway, and provide adequate ventilation and reassurance Maintain/re-establish anaesthesia, if appropriate Reverse aminosteroids with sugammadex
Investigations	PNS: TOF/DBS/post-tetanic count
Also consider	High volatile agent concentration Hyperventilation (ETCO2 <4kPa) CO_2 narcosis (ETCO2 >9kPa) Intracerebral event/injury Hypoglycaemia

NMBAs are routinely monitored prior to extubation. Reversal of NMBAs with neostigmine 50 micrograms/kg, accompanied by glycopyrronium bromide, is commonplace. A TOFR >0.9 indicates a return of neuromuscular activity acceptable for extubation. Residual NMB in recovery, despite reversal with neostigmine, can occur and generates extreme anxiety in patients.

Risk factors

- Recent dose of relaxant or drug administration error.
- Renal and hepatic impairment causing delayed elimination of NMBA (except atracurium).
- Perioperative administration of magnesium (>1.25–2.5mmol/L).
- Hypothermia.
- Acidosis and electrolyte imbalance.
- Co-administration of aminoglycoside antibiotics.
- Myasthenia gravis (reduced number of receptors).
- Competition with drugs also metabolised by plasma cholinesterase (etomidate, ester LAs and methotrexate).
- Low levels of plasma cholinesterase (pregnancy, renal and liver disorders, hypothyroidism).
- Abnormal plasma cholinesterase (suxamethonium apnoea).

Diagnosis

- Uncoordinated, jerky patient movements are suggestive of an inadequate reversal of NMB. Sustained head lift off the pillow for 5s is a good clinical indicator of adequate reversal.
- TOF is classically measured as adductor pollicis twitches in response to supramaximal stimulation via two electrodes placed over the ulnar nerve (see ➲ p. 423).

- DBS may be useful to appreciate small degrees of residual block vs the TOFR (see ➲ p. 423).
- Post-tetanic count is used to monitor deep relaxation when the TOF will not show any twitches. A 50Hz tetanic stimulus is applied for 5s, followed by single stimuli at 1Hz. Post-tetanic facilitation in the presence of non-depolarising blockade allows a number of twitches to be seen. Reversal with an anticholinesterase should be possible with a count of >10.
- If no twitches, establish that the PNS is working.

Immediate management

- ABC.
- If patient still has an ETT, assess depth of anaesthesia—if contributing to hypoventilation, reduce; if patient awake and anxious, deepen. If $ETCO_2$ is not normal, correct by adjusting ventilatory settings (consider pressure support ventilation).
- If the patient has been extubated, reassure them; consider a sedative. Open the airway, remove secretions and support ventilation with careful bag–mask assistance.
- If neostigmine has been given, ensure it was the correct dose (50 micrograms/kg) and the cannula was flushed.
- If you have used an aminosteroid muscle relaxant (rocuronium, vecuronium and pancuronium), administer sugammadex at a dose of 2–4mg/kg if available.
- Hypothermia, electrolyte imbalance and acidosis will impair reversal and should be corrected.
- Aminoglycoside or Mg^{2+} can interfere with reversal which may improve with calcium gluconate (10mL of 10% IV) titrated to effect.

Subsequent management

- Wait patiently—this is not an emergency!
- Explain to the patient what has happened; apologise and arrange counselling, if required. Awareness is difficult to predict and the use of the Brice interview questionnaire may help in this scenario.

Other considerations

- A dual (phase II) blockade occurs when large amounts of suxamethonium are used.
- Suspected myasthenia gravis should be confirmed postoperatively with an edrophonium test.

Further reading

Lenz A, Hill G, White PF (2007). Emergency use of sugammadex after failure of standard reversal drugs. *Anesth Analg*, **104**, 585–6.

Local anaesthetic toxicity

Condition	Excessive LA in systemic circulation
Presentation	Excitatory CVS and neurological symptoms followed by CVS and neurological depression
Immediate action	Stop LA administration Resuscitate and support Treat seizures Lipid emulsion therapy
Follow-up action	ICU if CVS collapse, awareness of pancreatitis as a potential complication of lipid emulsion
Investigations	ABG, ECG, amylase or lipase
Also consider	Anaphylaxis CVE/migraine Other causes of cardiac arrest (4Hs + 4Ts)

Risk factors

- Type of LA:
  - Short- vs long-acting
  - Ester vs amide (prilocaine is an ester which is rapidly metabolised by the liver with less risk of LA toxicity)
  - Intrinsic vasoconstrictive effects (reduces systemic absorption and thus improves safety, e.g. ropivacaine)
  - Formulation (bupivacaine is a racemic mixture with the R-enantiomer binding more firmly and released more slowly from the myocardium).
- Site of block: intercostal > central neuraxial > brachial plexus > SC.
- Single vs infusion/multiple dosing (↑ overall dose).
- Conduct of block: not visualising needle tip with ultrasound scanning, overly firm pressure with probe causing compression of veins preventing their identification, failure to aspirate causing inadvertent IV administration; not using incremental dosing or test dose; performance on anaesthetised patient rather than awake.
- Comorbidities: cardiac failure (susceptible to myocardial depressant), liver disease (reduced metabolism), renal disease (reduced clearance).
- High-risk groups: elderly, paediatric and pregnant.

Presentation

- Light-headedness, dizziness, drowsiness, tingling around the lips and fingers, or more generalised, metallic taste, tinnitus, blurred vision.
- Confusion, restlessness, incoherent speech, tremors or twitching, leading to convulsions, with loss of consciousness and coma.
- Bradycardia, hypotension, CVS collapse and respiratory arrest.
- ECG changes (prolongation of QRS and PR interval, AV block and/or changes in T-wave amplitude).

Immediate management

- Discontinue injection.
- Call for help.
- ABC and administer 100% O_2.
- Intubation may be required to prevent hypoxic CVS collapse. Hyperventilation may help by ↑ pH in metabolic acidosis.
- CPR if pulseless—commence the ALS protocol (see ➔ pp. 1055–7).
- Treat convulsions with IV:
  - Midazolam 3–10mg
  - Diazepam 5–15mg
  - Lorazepam 0.1mg/kg
  - Propofol 20–60mg
  - Thiopental 50–150mg.

Lipid emulsion therapy

- Give an IV bolus injection of 20% lipid emulsion, e.g. Intralipid® 20%, 1.5mL/kg over 1 min (100mL for a 70kg patient).
- Start an IVI of 20% lipid emulsion at 15mL/kg/h (1000mL over 1h for a 70kg patient).
- Repeat the initial bolus twice at 5min intervals if an adequate circulation has not been restored.
- After 5min, double the infusion rate if an adequate circulation has not been restored.
- Continue CPR, the ALS protocol and lipid infusion until a stable circulation has been restored.
- The mechanism of action is thought to be through extraction of lipophilic LAs from aqueous plasma and tissues or by counteracting LA inhibition of myocardial fatty acid oxidation, although other theories exist.

Other considerations

- Propofol is **not** a suitable alternative to lipid emulsion.
- Resuscitation attempts can be prolonged due to prolonged protein binding of the offending LA.
- Methaemoglobinaemia may occur with high doses of prilocaine (>600mg in an adult) 2° to the metabolite O-toluidine. Treat with methylthioninium chloride (methylene blue 1–2mg/kg).
- Allergic reactions to LAs are extremely rare. The ester groups are more prone to exhibit allergic reactions than amides, because they are metabolised to para-aminobenzoic acid (PABA), which acts as a hapten. There is also a cross-sensitivity of ester-type agents with sulfonamides. Allergic reactions range from simple local irritation with rash or urticaria to laryngeal oedema or anaphylaxis.

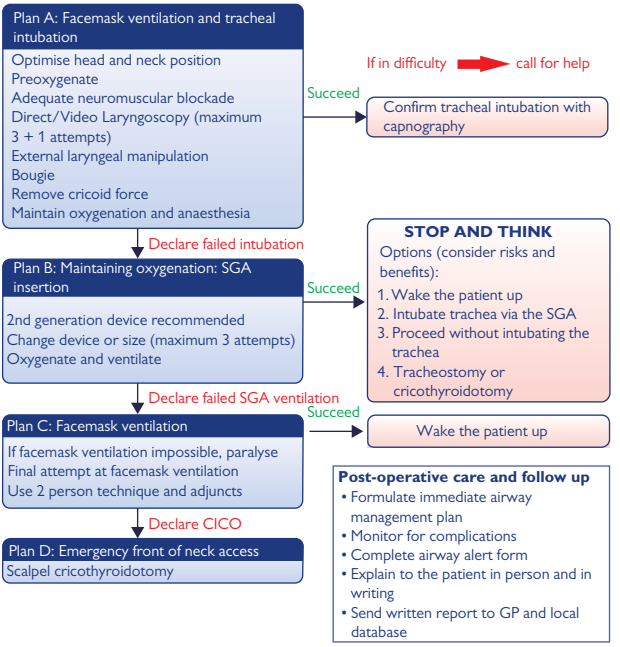
Further reading

The Association of Anaesthetists of Great Britain and Ireland (2010). *AAGBI safety guideline. Management of severe local anaesthetic toxicity*. https://anaesthetists.org/Portals/0/PDFs/Guidelines%20PDFs/Guideline_management_severe_local_anaesthetic_toxicity_v2_2010_final.pdf?ver=2018-07-11-163755-240&ver=2018-07-11-163755-240

Association of Anaesthetists. *Management of severe local anaesthetic toxicity*. https://anaesthetists.org/Home/Resources-publications/Guidelines/Management-of-severe-local-anaesthetic-toxicity

Unanticipated difficult intubation

(See Fig. 39.5; see pp. 368–9.)



This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

Fig. 39.5 Unanticipated difficult tracheal intubation. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6): 827–48 (2015) doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

Can't intubate, can't oxygenate

(See Fig. 39.6; see p. 381.)

CALL FOR HELP

↓ Continue 100% O_2
Declare CICO

Plan D: Emergency front of neck airway

Continue to give oxygen via upper airway
Ensure neuromuscular blockade
Position patient to extend neck

Scalpel cricothyroidotomy

Equipment: 1. Scalpel (number 10 blade)
2. Bougie
3. Tube (cuffed 6.0mm ID)

Laryngeal handshake to identify cricothyroid membrane

Palpable cricothyroid membrane

Transverse stab incision through cricothyroid membrane
Turn blade through 90° (sharp edge caudally)
Slide coudé tip of bougie along blade into trachea
Railroad lubricated 6.0mm cuffed tracheal tube into trachea
Ventilate, inflate cuff and confirm position with capnography
Secure tube

Impalpable cricothyroid membrane

Make an 8–10cm vertical skin incision, caudad to cephalad
Use blunt dissection with fingers of both hands to separate tissues
Identify and stabilise the larynx
Proceed with technique for palpable cricothyroid membrane as above

Postoperative care and follow up

- Postpone surgery unless immediately life threatening
- Urgent surgical review of cricothyroidotomy site
- Document and follow up as in main flow chart

This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

Fig. 39.6 Can't intubate, can't oxygenate. Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. Difficult Airway Society intubation guidelines working group, *BJA*, **115**(6): 827–48 (2015) doi:10.1093/bja/aev371. Permission for the use of these algorithms for commercial purposes must be sought directly from Difficult Airway Society as they hold the copyrights.

Malignant hyperthermia

(See also ➲ pp. 322–3.)

Condition	Life-threatening hypermetabolism in genetically susceptible patients triggered by volatile agents and suxamethonium
Presentation	↓ SpO_2, ↑ $ETCO_2$, ↑ HR, ↑ core temperature, muscle rigidity, dysrhythmias, DIC
Immediate action	Stop volatile agents Hyperventilate with high-flow 100% O_2 Dantrolene 2.5mg/kg Cool
Follow-up action	Correct acidosis, ↑ K^+ and coagulopathy Promote diuresis Monitor for compartment syndrome
Investigations	ABGs, K^+; clotting studies; CK
Also consider	Rebreathing CO_2 absorption from pneumoperitoneum Extended tourniquet time Phaeochromocytoma Neuroleptic malignant syndrome Recreational drug toxicity, e.g. Ecstasy Thyroid storm

Risk factors

- Family history.
- Exposure to suxamethonium or volatile agents (even if previous exposures were uneventful).
- Heat stroke, exercise-induced rhabdomyolysis, central core disease.
- Some neuromuscular diseases have idiosyncratic MH-like reactions.

Diagnosis

- Sustained jaw rigidity after suxamethonium (masseter spasm).
- Unexplained ↑ $ETCO_2$ (IPPV) or ↑ minute ventilation (SV), plus unexplained ↑ HR; accompanied, or followed, by an increasing core temperature.
- Falling SpO_2, despite ↑ FiO_2.
- CVS instability, dysrhythmias, especially multiple ventricular ectopics, peaked T-waves on ECG.
- Generalised muscle rigidity.

Immediate management

Turn off volatile agent, stop giving suxamethonium

ABC

- Declare an emergency. Ask for help and dantrolene immediately.
- Hyperventilate with 100% O_2, preferably not from the anaesthetic machine containing volatile agent. Prepare an alternative method of keeping the patient anaesthetised (e.g. propofol infusion).
- Ask the surgical team to conclude surgery as quickly as possible.

Dantrolene

- Give dantrolene 2.5mg/kg. Select dose based on actual body weight to maximum 300mg per dose.
- Repeat dose every 10min until $PaCO_2$ <6kPa and decreasing body temperature. No cumulative ceiling dose. (Mortality associated with insufficient dosing.)

Activated charcoal filters

- If available, place on inspiratory and expiratory limbs of the circle breathing system.

Temperature

- Reduce temperature by exposing the patient, ice to the groin and axillae, cold fluids and extracorporeal heat exchange if available.

Monitoring

- Check ABGs and K+, and correct acidosis/hyperkalaemia.

Subsequent management

- Place invasive monitoring (arterial line and CVP line).
- Send a clotting screen for DIC and serum CK assay.
- Promote diuresis with fluids and mannitol. Monitor for AKI.
- Avoid calcium channel blockers; treat arrhythmias with magnesium or amiodarone or metoprolol.
- Will require HDU/ICU admission for at least 24h after resolution of metabolic derangement.
- Monitor for recrudescence; likelihood increases with increasing metabolic derangement during initial episode.
- Monitor for rhabdomyolysis and compartment syndrome.
- Refer to the MH investigation unit for follow-up.

Other considerations

- There are two formulations of dantrolene: Dantrium® 20mg (mixed with 60mL of sterile water) and Ryanodex® 250mg (mixed with 5mL of sterile water). Ryanodex® is currently unavailable in Europe. A 100kg adult requires 12 ampoules of Dantrium® per dose.
- The use of visual aids for task prioritisation, such as prepared task cards allocating roles, improves team performance during an MH crisis (see http://malignanthyperthermia.org.au/mh-task-cards/)
- Follow-up involves DNA testing for ryanodine receptor *RYR1* mutations. If DNA negative, a muscle biopsy will be offered with *in vitro* halothane and caffeine contracture tests.
- Beware of using bicarbonate to correct acidosis if the acidosis is principally respiratory, since the reaction with H^+ produces an ↑ CO_2 load.

Anaesthesia for known or suspected MH-susceptible patients

- MH patients should not be denied surgery solely because of MH.
- Preoperative questioning about personal and family anaesthetic history is essential to identify potential MH patients.
- It is not essential to test suspected cases prior to surgery, provided individual assessment has been made of the risks involved.

- An MH-'safe' technique (i.e. avoid suxamethonium and volatile agents), such as propofol TIVA or regional anaesthesia, is appropriate (all other drugs are MH 'safe').
- Dantrolene is not required prophylactically. It is unpleasant for the patient and prolongs the action of NDMRs. It should be available.
- Standard monitoring, i.e. ECG, NIBP, SpO_2, $ETCO_2$. A baseline core temperature should be established before the procedure and monitored for 4h postoperatively.
- The anaesthetic machine must be purged and flushed to remove traces of volatile agents. In modern machines with complex internal silicon components, this can take at least 70min, and then high FGF must be maintained throughout the case.
- A cost-effective alternative is the use of activated charcoal filters. These reduce machine preparation time to 2min, e.g.Vapor-Clean® activated charcoal filters.

Instructions for use of activated charcoal filters

- Remove vaporisers from the anaesthetic machine.
- Flush circuit for 90s with O_2 or air at 10L/min using the ventilator with a 2L test lung attached.
- Change the breathing circuit and soda lime while maintaining flushing at 10L/min. Use new ETT and face masks.
- Insert activated charcoal filters on both the inspiratory and expiratory ports of the breathing system. Two activated charcoal filters are used to remove the risk of incorrectly placing a single filter on the expiratory limb which is ineffective.
- Maintain FGF of 10L/min for 90min from the commencement of the anaesthetic.
- After 90min, it is safe to reduce FGF to 3L/min.
- Activated charcoal filters are single-use items and can be used at 3L/min for a total of 12h.

Further reading

Hopkins PM, Girard T, Dalay S, *et al*. (2021). Malignant hyperthermia 2020. Guideline from the Association of Anaesthetists. *Anaesthesia*, **76**, 655–64.

UK Malignant Hyperthermia Registry. https://www.ukmhr.ac.uk

European Malignant Hyperthermia Group. https://www.emhg.org

Useful contact

UK MH Investigation Unit, Academic Unit of Anaesthesia, Clinical Sciences Building, St James's University Hospital, Leeds LS9 7TF. Emergency telephone number 07947 609601.

Chapter 40

Regional anaesthesia

Mark Fairley

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Fundamentals of safe practice

Patient consent

In terms of regional anaesthesia, this will include:

- An explanation of how the block will be performed and if any sedation or GA will be used in addition to the regional block.
- An explanation of the risks and benefits of the regional anaesthesia technique and any alternatives. Generic risks relevant to most regional techniques include failure, local anaesthetic systemic toxicity (LAST) and vessel and nerve damage. Generic benefits usually include less pain, less opioids and their side effects (e.g. less nausea and vomiting) and earlier mobilisation. Technique-specific risks and benefits should also be mentioned, and the consent process documented.

Preparation

- Regional anaesthesia should ideally be performed in a quiet, calm environment, in an unhurried manner. Specific 'block rooms' are becoming popular for this purpose.
- Appropriate aseptic precautions should be taken.
- Equipment necessary for the regional anaesthetic should be prepared, and drugs clearly labelled.
- The equipment and drugs necessary for resuscitation and administration of GA should be immediately available and checked.
- Monitoring should include the continuous presence of an anaesthetist and the provision of ECG, NIBP and SpO_2.
- Trained assistance is as necessary for the safe and effective conduct of regional anaesthesia as it is for GA.

Documentation

Documentation of the procedure should include the following:

- Whether the patient is anaesthetised, sedated or awake
- Block performed; needle used
- Nerve location technique used (ultrasound, PNS, loss of resistance)
- Volume and concentration of LA used, along with any adjuncts
- If using a PNS, the stimulus duration, current threshold and whether the start of LA injection was associated with 'twitch abolition'
- If using ultrasound, consideration of recording images, as per local facility capability and policies
- Whether the injection was easy without resistance or if any injection pressure monitoring was used
- Whether any pain or paraesthesiae was experienced on injection
- Any complications that occurred
- The block effect and any supplementation required.

Training and supervision

The acquisition of a detailed knowledge of the anatomy and pharmacology should be followed by a study of the block to be performed. The trainee should then discuss the block with an appropriately experienced teacher and should observe the performance of the block. The teacher should closely supervise the trainee in the performance of the block for as many times as is necessary, to be confident that the trainee is both competent and safe.

All trainees should focus on learning a small number of basic blocks that cover the vast majority of surgical procedures. A proposed list of these high-value basic blocks that all general trainees should learn has recently been published[1] (Table 40.1).

Regional anaesthesia fellowships and diplomas are formal training pathways added on to general training for those wanting advanced skills in regional anaesthesia.

Table 40.1 Proposed high-value basic ultrasound-guided regional anaesthetic techniques

Anatomical location	Plan A (basic blocks)	Plan B/C/D (advanced blocks)
Shoulder	Interscalene brachial plexus block	Superior trunk block, combined axillary and suprascapular nerve blocks
Below shoulder	Axillary brachial plexus block	Infraclavicular block, supraclavicular block
Hip	Femoral nerve block	Fascia iliaca block, lumbar plexus block
Knee	Adductor canal block	Femoral nerve block ± iPACK block
Foot and ankle	Popliteal sciatic block	Ankle blocks, proximal sciatic nerve block
Chest wall	Erector spinae plane block	Paravertebral, superficial serratus anterior plane, PSP and IPP blocks
Abdominal midline	Rectus sheath block	Quadratus lumborum blocks

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Note some names of blocks have been changed from the original article, in line with 2021 international consensus on nomenculture.

Personal audit

Regional anaesthetists should keep a record of the blocks they perform. Difficulties encountered, success rates and complications should be recorded and should be both compared with available published data and discussed in an appraisal process. Lower success rates or higher complication rates than are currently accepted, or any serious complications, should be discussed with an appropriate colleague.

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Local anaesthetics and adjuncts

Local anaesthetics

Properties of common LAs are listed in Table 40.2.

Table 40.2 Properties of common local anaesthetics

Local anaesthetic	pKa	Onset	Protein binding (%)	Duration of action	Maximum dose (mg/kg)
Bupivacaine	8.1	Medium	95	Long	2
Levobupivacaine	8.1	Medium	95	Long	2
Ropivacaine	8.1	Medium	94	Long	3
Prilocaine	7.7	Fast	55	Medium	6 (8 with adrenaline)
Lidocaine	7.7	Fast	65	Medium	3 (7 with adrenaline)

LAST occurs when an excessive amount of LA enters the circulation. Risk can be reduced by observing maximum doses, injecting slowly in small boluses, interspersed with frequent gentle aspirations to exclude accidental intravascular needle placement, and use of ultrasound.[2,3] Older, sicker patients, single-site large doses and more vascular injection sites can all increase risks.

Levobupivacaine and ropivacaine are less toxic than bupivacaine. The higher toxicity of bupivacaine is related to the R-enantiomer which binds more firmly and is released more slowly from the myocardium.

Toxicity from prilocaine is less likely because of its rapid metabolism, primarily by the liver. Methaemoglobinaemia may occur with high doses (>600mg in an adult) and should be treated with methylthioninium chloride (methylene blue 1–2mg/kg).

Allergic reactions to LAs are extremely rare. The ester groups are more prone to exhibiting allergic reactions than amides. (See ➲ pp. 1092–3 for presentation and management of LAST.)

Adjuncts

- Table 40.3 provides a list of adjuncts commonly added to LAs to improve their effects.
- None of the currently used adjuncts are approved for perineural administration. Optimal doses are not clearly established.
- Ensure any adjunct preparation is preservative-free.

Table 40.3 Commonly used adjuncts to local anaesthetics

Drug	Characteristics
Dexamethasone	Prolongs long-acting LA block by 4–8h.[4] Also has similar effects when given IV. Possibly incompatible with ropivacaine as crystallisation demonstrated *in vitro*. Can increase blood sugar level. Dose 4mg
Clonidine	Prolongs block by 1.5–2.5h.[4] Effective in epidural, caudal, spinal and peripheral nerve blocks. Use is limited by hypotension, bradycardia and sedation. Dose 150 micrograms
Dexmedetomidine	Prolongs block by 3–4h.[4] Longer than clonidine, but not as long as dexamethasone. ↑ risk of sedation, bradycardia and hypotension. Suggestion that it may cause more differential block (sensory greater than motor) and may have some neuroprotective effects. Dose 50–60 micrograms
Bicarbonate	Added to reduce pain from SC injection and to increase speed of onset by increasing pH of solution, and therefore fraction of unionised LA. Add 1mL of 8.4% to every 10mL of lidocaine or 20mL of bupivacaine. Discard LA if precipitate forms
Opioids	Proven synergism with intrathecal and epidural LA. Beware delayed respiratory depression with intrathecal morphine. All opioids have been used. Of doubtful benefit in peripheral blocks. Intrathecal remifentanil is contraindicated due to the presence of glycine
Glucose	Used to increase baricity of LA for intrathecal use. Hyperbaric bupivacaine contains 80mg/mL of glucose. Allows more consistent spread of block and provides the opportunity to control spread by altering patient position
Hyaluronidase	Used in eye blocks to enhance LA spread. Dose 15 units/mL

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Finding nerves

Anatomical landmark techniques

The safe practice of regional anaesthesia is based on a detailed knowledge of anatomy and its variations. Although most techniques based solely on surface anatomy and the palpation of deeper structures have now been superseded by PNS and ultrasound, several purely anatomical landmark-based blocks are still effective and practical, e.g. spinal. Ultrasound and PNS techniques still require good anatomical knowledge for their safe and effective use.

Clicks, pops and loss of resistance

Some fascial plane techniques, e.g. fascia iliaca block, have relied upon the sensation of a blunt or blunted needle passing through a fascial plane to identify the correct anatomical location for injection. These 'clicks' and 'pops' take experience to appreciate and have a significant failure rate. With ultrasound availability, most of these techniques have been superseded. However, they still have a role where ultrasound is not available or not practical. Loss of resistance has been used successfully for many decades to identify the epidural space correctly and remains the technique of choice.

Paraesthesia

Direct contact between needle and nerve may result in an unpleasant 'electric shock' sensation that is felt in the distribution of the target nerve. This is termed 'paraesthesia' and, before the introduction of nerve stimulation, was often sought as confirmation of needle proximity to a nerve. With the availability of PNS and ultrasound machines, paraesthesia is now seen as an unnecessary needle–nerve contact that could potentially increase the risk of nerve damage. However, in experienced hands and in the absence of nerve stimulators and ultrasound machines, this technique may still be effectively used.

Nerve stimulation

The use of nerve stimulators dominated the art of nerve location in the final 20y of the last century and is still widely practised. The production of evoked muscle contractions at low current levels (0.2–0.5mA) is thought to confirm the placement of a needle near a nerve, while the production of evoked contractions at very low current thresholds (<0.2mA) is thought to indicate possible intraneural needle tip placement. If the target nerve contains sensory fibres, the sensation may be unpleasant. The use of nerve stimulators remains popular, either on their own, if no ultrasound machine is available, or in combination with ultrasound. When used with ultrasound, they can provide the anaesthetist with reassurance that the structure on the screen is the target nerve, demonstrate twitches and functional anatomy to educate trainees and, as an additional monitor, can help exclude intraneural needle tip placement.

Using a peripheral nerve stimulator

- Connect the stimulating needle to the negative lead (black) and the ground electrode and the ECG pad to the positive lead (red): 'negative to needle, positive to patient'.
- Keep the ECG electrode at least 20cm from the site of injection. Start with a current of 1.0–2.0mA and a frequency of 2Hz. In theory, a stimulus duration of 0.1ms will preferentially stimulate motor nerves, rather than sensory nerves, and may be less unpleasant for the patient.
- Insert the insulated needle. At all times, move the needle slowly and gently, watching for signs of nerve stimulation. Aim to move the needle in small, steady steps, no more than 1–2mm at a time.
- When the desired muscle contractions are evoked, try to optimise the position of the needle to obtain a good motor response in the muscles supplied by the target nerve while reducing the stimulating current to 0.2–0.5mA.
- If twitches continue to occur at <0.2mA, withdraw the needle slightly as this may indicate intraneural needle placement.
- Aspirate to exclude intravascular needle placement and inject 1mL of LA solution. The motor response should disappear because the nerve is displaced by fluid ('twitch abolition'). If the motor response does not disappear with the initial 1mL, suspect that the needle may be intraneural. Withdraw the needle slightly before further injection.
- Inject the full volume in small boluses, interspersed by careful aspirations. If there is any pain or significant resistance to injection, stop immediately and withdraw the needle slightly as this may also indicate intraneural placement.

Ultrasound

Ultrasound has become increasingly available in the last 10–20y and has allowed the direct vision of needle tip placement and LA spread relative to the target nerve and surrounding structures.

Long-axis and short-axis imaging

Cylindrical structures like nerves and vessels can be imaged in long axis, meaning the structure is parallel to the long axis of the probe, giving a long rectangular view of the structure across the screen. Short-axis imaging means the structure is perpendicular to the long axis of the probe, resulting in a short circular view of the structure in the middle of the screen. Short-axis imaging is the most popular for nerve blocks as it is easier to keep the target nerve in the imaging plane and the relative positions of any surrounding neurovascular structures are more easily seen.

In-plane and out-of-plane needling

When using an in-plane (IP) needling technique, the needle is inserted from one end of the probe and passed along the long axis of the probe, so the image is of the entire length of the needle. This has the advantage that you can keep the tip of the needle visible as you advance it. With the out-of-plane (OOP) needling technique, the needle is inserted at right angles to the long axis of the probe and only a cross-section of the needle is visible at any one time as a small white dot. The probe must be moved to continually

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reimage the needle tip as it is advanced. OOP needling is popular for vascular access as catheters need to be advanced along the long axis of the vessel. IP needling is popular for most nerve blocks as the needle tip can be imaged as it is advanced. Inexperienced ultrasound users are probably safer using the IP needling technique. Experienced operators can use either IP or OOP safely.

Developing skills

Manipulating the ultrasound probe and needle in 3D, while looking at the 2D image on the screen, can be a difficult skill to acquire. Some tips that can accelerate your passage from novice to expert include:

- Get taught properly by experienced ultrasound users.
- Learn from all available media: online videos, mobile apps, ultrasound and cadaver courses and phantoms, as well as books.
- Do not only practise when performing a block. Practise scanning easily accessible aspects of yourself, colleagues and consented patients whenever you can to become familiar with the knobology, probe manipulation and identification of the sonoanatomy. Practise needle imaging with commercially available or easily made phantoms or meat models.

Using ultrasound

- Plan the ergonomics. Position yourself, your hands and the screen in a line, so you can easily look from your needle and probe to the display. This usually means the machine is on the opposite side of the patient. Make sure you know which end of the probe corresponds to the left and right side of the display.
- Make small, slow movements of the probe. Like a torch beam, a small change in angle sweeps the beam through a large arc.
- Perform a prescan to identify the sonoanatomy; tilt the probe to make the image of the nerve bright (anisotropy), then maintain that probe tilt by holding the probe low and stabilising your hand on the patient. Look at your hands and insert the needle from the near end of the probe, making sure the needle trajectory is aiming along the centre of the ultrasound beam. Once the needle has advanced under the probe, look at the screen and slide (do not change the tilt) the probe slightly as required to bring the needle into view.
- The needle needs to be in the 1mm-wide ultrasound beam to be seen. Echogenic needles and needle imaging software can make a needle image brighter but will not help if the needle is not in the ultrasound beam!
- You should not advance the needle or inject if you cannot see its tip.
- Beware of obliquely imaged needles which will give a false tip appearance. Ensure you can recognise an obliquely imaged needle.
- You should see the LA distending the tissues as soon as you inject the first mL. If you do not, stop injecting as either you are not imaging your needle tip or you may be in a compressed vessel.

- Never inject against high resistance. Consider use of an injection pressure monitoring device and/or perform injection yourself rather than have an assistant inject.
- Swelling of the nerve with separation of its fascicles indicates intraneural injection. Stop injecting and withdraw slightly.
- Following injection, perform a postscan of the sonoanatomy to confirm correct target and check spread of LA.

Evidence for ultrasound

The use of ultrasound has revolutionised regional anaesthesia practice in the last 20y. Its use has been shown to decrease the time taken to perform nerve blocks, accelerate block onset, increase block success rate, decrease the dose of LA used and decrease the incidence of certain complications such as vascular puncture and LAST.[2,5] Its use has become standard of care in most countries.

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Needle design

Many types of needle design are commonly used for regional anaesthesia, whether PNBs or central neuraxial blocks.

- **Long-bevelled needles** (usually 10–15°) are sharp and pass readily through tissues, without giving the operator a clear sensation of passage of the needle through tissue planes and fascial layers. These are more likely to enter nerves than short-bevelled needles; hence they are rarely used.
- **Short-bevelled needles** (18–45°) are less likely to enter individual nerves, making them safer. They also provide the operator with more feel of the passage of the needle through fascial layers. They are currently the most popular tips for PNBs.
- **Pencil-point needles** are popular for spinal anaesthesia, as they are thought to separate the fibres of the dura mater, rather than cut them as would a bevelled needle. They are associated with a lower incidence of PDPH than the use of the bevelled alternative (often called the Quincke-tip needle). Pencil-point needles are rarely used for PNBs as they are too difficult to pass through intact skin and fascial planes.
- **Tuohy needles** were originally designed to allow epidural catheterisation, but the tip design has also been adapted for use in placing peripheral nerve catheters. They also provide a lot of resistance to passing through fascial planes, enhancing the feel of 'pops' used in some landmark techniques.
- **Insulated needles** are coated with a non-conducting material that allows current flow only from a small area at the tip of the needle. This can improve accuracy of nerve location with PNS.
- **Echogenic needles** have been designed to more effectively reflect ultrasound waves, via geometric indentations on their shaft, enhancing visibility, especially at steeper angles of insertion.

Continuous regional anaesthesia

- Catheters can be placed near nerves to provide prolonged neural blockade. Studies have shown that continuous regional anaesthesia (CRA) provides superior analgesia to systemic techniques, while accelerating early mobilisation and improving rehabilitation. In addition, the side effects of opioids are minimised or avoided.[6]
- Surveillance during CRA is important. The patient needs to be taught how to care for an insensate limb, and both patient and carers need to be taught to identify the signs and symptoms of LA toxicity and catheter sepsis. CRA carries with it all the complications of single-shot blocks, along with additional risks of LA toxicity, catheter misplacement or movement and bacterial colonisation and sepsis.
- Nerve catheter placement is an advanced technique for experienced practitioners.

Regional anaesthesia and coagulation disorders

- Blood vessels are frequently encountered and damaged during the performance of regional anaesthesia. These may be large vessels, such as the axillary, subclavian and femoral arteries, or small blood vessels such as epidural veins. Fortunately, the small holes made by regional needles in patients with normal coagulation very rarely lead to significant morbidity. Those with abnormalities of coagulation, however, can suffer significantly.[7]
- The use of anticoagulant drugs to prevent or treat VTE is increasing rapidly, along with the number and potency of the drugs employed for such purposes.
- Haemorrhage can be significant in non-compressible locations. Haematomas can compress nerves and other anatomical structures. The presence of even a relatively small haematoma in the non-distensible spinal canal can cause permanent spinal cord injury.
- Different blocks have different relative risks of bleeding complications:
- Neuraxial
  - > deep block, e.g. quadratus lumborum, paravertebral
  - > perivascular blocks, e.g. supraclavicular, femoral
  - > fascial plane blocks, e.g. fascia iliaca, erector spinae plane
  - > superficial blocks, e.g. ankle blocks, forearm blocks.[8]
- Catheter insertion involves a greater risk than single-shot blocks.
- Ultrasound techniques allow vessel imaging and have been shown to reduce the risk of vessel puncture relative to non-ultrasound techniques.[2]
- Coagulopathy is therefore a relative contraindication to the performance of regional anaesthesia techniques. The degree of contraindication will depend upon the extent of the coagulation defect, the specific block planned, the block technique and the likely benefit of the regional anaesthesia technique, compared to the alternatives. These decisions should be made by experienced clinicians, familiar with current guidelines, in consultation with the patient.
- Several guidelines for the management of patients with abnormalities of coagulation exist:
  - AoA (2013)[9] https://anaesthetists.org/Home/Resources-publications/Guide lines/Regional-anaesthesia-and-patients-with-abnormalities-of-coagulation
  - American Society of Regional Anaesthesia (2018)[7] https://rapm.bmj.com/content/rapm/43/3/263.full.pdf
  - Canadian Anesthesiologists Society (2019)[8] https://rdcu.be/b0PUx

Table 40.4 is drawn from the AoA guidelines[9] (see also p. 276.)

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Table 40.4 Recommendations related to drugs used to modify coagulation

Drug	Acceptable time after drug for block performance	Administration of drug while spinal or epidural catheter in place	Acceptable time after block performance or catheter removal for next drug dose
UFH SC prophylaxis	4h or normal activated partial thromboplastin time ratio	Caution	1h
UFH IV treatment	4h or normal activated partial thromboplastin time ratio	Caution	4h
LMWH SC prophylaxis	12h	Caution	4h
LMWH SC treatment	24h	Not recommended	4h
Danaparoid prophylaxis	Avoid (consider anti-Xa levels)	Not recommended	6h
Danaparoid treatment	Avoid (consider anti-Xa levels)	Not recommended	6h
Bivalirudin	10h or normal APTTR	Not recommended	6h
Argatroban	4h or normal APTTR	Not recommended	6h
Fondaparinux prophylaxis*	36–42h (consider anti-Xa levels)	Not recommended	6–12h
Fondaparinux treatment*	Avoid (consider anti-Xa levels)	Not recommended	12h
NSAIDs	No additional precautions	No additional precautions	No additional precautions
Aspirin	No additional precautions	No additional precautions	No additional precautions
Clopidogrel	7d	Not recommended	6h
Prasugrel	7d	Not recommended	6h

Drug	Acceptable time after drug for block performance	Administration of drug while spinal or epidural catheter in place	Acceptable time after block performance or catheter removal for next drug dose
Ticagrelor	5d	Not recommended	6h
Tirofiban	8h	Not recommended	6h
Eptifibatide	8h	Not recommended	6h
Abciximab	48h	Not recommended	6h
Dipyridamole	No additional precautions	No additional precautions	6h
Warfarin	INR ≤1.4	Not recommended	After catheter removal
Rivaroxaban prophylaxis* (CC >30mL/min)	18h	Not recommended	6h
Rivaroxaban treatment* (CC >30mL/min)	48h	Not recommended	6h
Dabigatran prophylaxis or treatment** (CC >80mL/min)	48h	Not recommended	6h
(CC 50–80mL/min)	72h	Not recommended	6h
(CC 30–50mL/min)	96h	Not recommended	6h
Apixaban prophylaxis	24–48h	Not recommended	6h
(Alteplase, anistreplase, reteplase, streptokinase)			10d Not recommended 10d

* Manufacturer recommends caution with use of neuraxial catheters.

** Manufacturer recommends that neuraxial catheters are not used.

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Regional anaesthesia and nerve injury

- Perioperative peripheral nerve dysfunction is due to many causes and may even be pre-existing and only noted for the 1st time in the perioperative period. Intraoperative causes include ischaemia due to hypotension and vascular occlusion, tourniquets, pressure from haematomas, thermal injury from electrocautery, poor patient positioning, retraction or direct surgical trauma. Postoperative causes include oedema, inflammatory neuropathy, compression from immobilising devices and compartment syndrome. Identifying the cause with certainty is usually not possible.
- Estimates of the incidence of postoperative peripheral nerve dysfunction associated with regional anaesthesia vary widely, depending on type of block, time since block, patient factors and symptoms reported. Minor sensory-only abnormalities during the first few days after proximal brachial plexus blocks can be as high as 1:10,[3] while significant sensory and/or motor abnormalities persisting for 6–12mo can occur up to 1:2000.[2,3,5] The likelihood of postoperative nerve dysfunction depends upon many factors, including which nerve block is performed, how the nerve block is performed, the age of the patient and their comorbidities. Mechanisms include direct needle trauma, injectate neurotoxicity and ischaemia from vasoconstriction or compression. Most perioperative peripheral nerve symptoms are not related to regional anaesthesia and most fortunately resolve with the passage of days or weeks.
- Visualisation of the nerves and needle tip with ultrasound allows the anaesthetist to achieve a very low incidence of needle–nerve contact, thereby minimising the chances of needle–nerve trauma.
- Intraneural injection (direct injection inside the epineurium of a nerve) increases the risks of direct needle trauma and injectate neurotoxicity. It can also be associated with high injection pressures leading to ischaemic injury. The hallmarks of intraneural injection include low current thresholds when using a PNS, high injection pressures, pain on injection, failure of the evoked contractions to disappear at the start of LA injection and swelling of the nerve with separation of fascicles when visualised with ultrasound. If any of these are encountered, injection should cease immediately and the needle repositioned.
- Publications suggest the interscalene brachial plexus block is the PNB with the highest incidence of postoperative nerve dysfunction symptoms.[3] Great caution should be exercised when placing a needle anywhere near the upper reaches of the brachial plexus.
- It appears that children only very rarely suffer nerve injury from regional anaesthesia and pregnant women undergoing spinal and/or epidural anaesthesia also have a very low incidence. Conversely, it appears that the elderly, obese patients, patients with DM and those with pre-existing neurological conditions are at a higher risk of nerve damage.
- The management of perioperative peripheral nerve dysfunction involves its early recognition, discussion with surgeons regarding possible causes, and if minor sensory changes only, reassurance that most of these will spontaneously resolve is usually all that is required. If significant deficits, motor deficits or non-resolving symptoms, referral

to a neurologist, nerve conduction studies, MRI and EMG can all assist in the identification of the severity and location of the injury. There is little that can be done to hasten the recovery of nerve function or to minimise the extent of the nerve injury once harmed. If there is acute compression from a haematoma, especially in the spinal canal, urgent surgical decompression is indicated. If there is neuropathic pain, appropriate antineuropathic pain medications should be prescribed.
- Recovery of neurological function is mercifully the norm. More than 90% of cases of neuropathy thought to have resulted from regional anaesthesia recover within 3mo, and >99% within a year.[10]

Awake or asleep?

- PNBs are currently performed in awake, sedated or asleep patients. Controversy exists as to whether performing blocks on anaesthetised patients places the patient at ↑ risk.
- In theory, awake patients may get symptoms of needle–nerve contact and warn the anaesthetist before further needle advancement or injection is performed, possibly averting nerve injury. Awake patients may also complain of early symptoms of LA toxicity from inadvertent intravascular injection, warning the anaesthetist to cease further injection and possibly averting a major LAST event.
- However, needle–nerve contact and intraneural injection are known to not always cause symptoms. Patient symptoms cannot therefore be relied upon and other monitors of needle–nerve contact and intraneural injection should be used. These are ultrasound visualisation of intraneural needle tip placement, LA injection causing distension of the nerve with separation of fascicles, low-threshold currents if using a PNS, failure of evoked contraction disappearance on injection of the LA and difficulty with injection/high injection pressure. Paediatric anaesthetists argue that for most of their patients, 'awake' is not an option; they are perhaps fortunate that the incidence of nerve damage associated with PNBs in children is very low.
- The performance of PNBs on the non-anaesthetised patient need not be unpleasant for the patient. Many anaesthetists successfully use sedation with small doses of a benzodiazepine (e.g. midazolam) and/or an opioid (e.g. fentanyl) or infusions of small amounts of propofol or remifentanil. The increasing use of ultrasound for nerve location is known to improve patient comfort if the evoked contractions produced by nerve stimulators are avoided. It is therefore relatively easy to perform ultrasound-guided blocks on patients who are lightly sedated.
- While there is currently no hard evidence to support either 'awake' or 'asleep' PNBs, it seems prudent to perform them in responsive patients when possible.

(See also ➲ p. 927.)

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Nerve blocks: central neuraxial blocks

Spinal and epidural anatomy

- The spinal cord terminates at ~L1 in adults and at ~L3 in infants.
- The line joining the iliac crests (intercristine or Tuffier's line) is at ~L4 level.
- C7 is the most prominent spinous process; T7 is at the inferior angle of the scapula level.
- The subarachnoid space ends at ~S2 in adults, and is lower in children.
- The subarachnoid space extends laterally along the nerve roots to the dorsal root ganglia.
- The subdural space is a potential space between the dura and the arachnoid.
- The epidural (extradural) space lies between the walls of the vertebral canal and the spinal dura mater. It is a low-pressure space, occupied by areolar tissue, loose fat and the internal vertebral venous plexus.
- The ligamentum flavum is thin in the cervical region, reaching maximal thickness in the lumbar region (2–5mm).

Spinal block

Indications Lower abdominal surgery (CS, inguinal hernia), lower limb surgery, perineal surgery.

Landmarks Spinous processes of the lumbar vertebrae and the line joining the iliac crests (Tuffier's line).

Technique The patient should be sitting or lying on their side. Mark a line joining the iliac crests. Identify the spinous process at the level of this line. The nearest interspace at this level is L3/4 (there is significant variation). Spinal blocks should always be carried out caudal to this space to avoid trauma to the tail end of the spinal cord (the conus).

Midline At the level of the interspace, insert a needle in the midline. With a 15° cephalad angulation, advance until a click or pop is felt (the dura is pierced) at an approximate depth of 4–6cm. After confirming free flow of CSF, carefully connect the syringe containing the LA. Aspirate gently before and after injection to confirm correct placement in CSF throughout injection. Inject slowly.

Paramedian One to 2cm lateral to the upper border of the spinous process, insert a needle perpendicular to the skin to contact the lamina of the vertebra. Withdraw slightly, reinserting the needle 15° medially and 30° cephalad to pass over the lamina through the interlaminar space. Advance until a click or pop is felt. Aspirate and inject as above.

Relative contraindications AS or mitral valve stenosis (hypotension and inability to compensate by increasing CO). Hypovolaemia (hypotension). Previous back surgery (technical difficulty). Neurological disease (↑ risk of neurological complications). Systemic sepsis (↑ incidence of epidural abscess, meningitis).

Absolute contraindications

- Local sepsis
- Patient refusal
- Anticoagulation (see ➲ pp. 269–75).

Local anaesthetic drugs and doses for spinal anaesthesia

- Dosing of LA in adults depends upon age and pregnancy. The older the patient, the less drug will be needed. This is thought to be due to degenerative changes of the vertebral column and ↑ sensitivity of neural tissue to LA. Pregnant patients need less than their non-pregnant counterparts, due to epidural vein engorgement compressing the thecal sac.
- Bupivacaine 0.5% is usually used. 'Heavy' is hyperbaric and contains 8% glucose. 'Plain' is isobaric at body temperature.
- Due to spread to the dependent part of the thoracic kyphosis in the supine position, hyperbaric solutions can be used to achieve a higher block and provide an ability to influence height with position changes of the patient.
- A volume of 2.5–3.0mL of a hyperbaric solution of LA will reach T6–T10 in most non-pregnant young adults placed in the recumbent position shortly after spinal injection.
- Pregnant patients require less, often 1.8–2.2mL (see ➲ p. 857).
- The volume of plain LA needed tends to be a little higher.
- Lidocaine is associated with a risk of cauda equina syndrome, transient radicular irritation and transient neurological symptoms.
- Ropivacaine can be used but does not come in a 'heavy' preparation and has a shorter duration of action than bupivacaine.
- Opioids are often added to improve the quality of the block. (See ➲ p. 1166; Table 41.9.)
- Ensure any drug preparations intended for intrathecal use do not contain preservatives.

Clinical tips

- If there is difficulty at L3/4, ideally go down, not up, as the level of termination of the conus is variable. Surface identification of the L3/4 interspace is inaccurate; 70% of clinicians mark it as a higher space. Ultrasound can be used to improve accuracy.
- A sitting position increases CSF pressure, and hence improves CSF flow with fine needles. It is also easier to find the midline in obese patients in this position.
- A lateral position offers patient comfort and the possibility of sedation.
- Problems are often due to the lumbar spine not flexed enough, or a short introducer not inserted enough and a too flexible needle. When difficulty is encountered in an elderly, osteophytic patient, consider a 22G Quincke-tip needle. PDPH is rare in this patient group.
- When hitting bone at a superficial level, this can only be the top of spinous processes. This is a good sign that you are midline and need to keep walking the needle cephalad or caudad.

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- When hitting bone at a deep level, this is more likely to be lamina. Ask the patient to identify on which side you are. If they can identify one side, you are out of the midline and need to redirect to the opposite side. If they state 'middle', you could be deep on a spinous process and could continue walking cephalad or caudad, recheck landmarks or try another level.

Complications

- Hypotension (due to sympathetic block), bradycardia (if block extends to the mid-thoracic region): can progress to cardiac arrest.
- High block (compromising breathing, may extend to 'total spinal' with loss of consciousness, apnoea and cardiorespiratory arrest).
- Urinary retention.
- Nerve damage (see ➔ pp. 1112–13).
- PDPH (see ➔ pp. 848–51).
- Infection: abscess, meningitis.
- Spinal canal haematoma: more likely in patients with disorders of coagulation. Can cause spinal cord compression and permanent paraplegia if not urgently decompressed with laminectomy.
- The serious complications of spinal and epidural anaesthesia have been the subject of a nationwide audit in the UK—The RCoA's 3rd National Audit Project (NAP3) 2009. The incidence of permanent injury due to neuraxial blocks was 1:25 000–1:50 000, with an incidence of death or paraplegia of 1:50 000–1:140 000. The incidence of complications in children, obstetric patients and those undergoing chronic pain procedures was very low. There was an excess incidence of serious complications in elderly patients with epidurals used during and after surgery, and in patients undergoing CSEs, a finding supported by other large studies (http://www.rcoa.ac.uk/nap3).

Ultrasound for central neuraxial block

- Ultrasound for CNB is usually used to prescan the anatomy and identify landmarks before performing the procedure. Ultrasound allows identification of the midline, the accurate spinal level, an estimation of the depth to the ligamentum flavum, the angle of insertion needed to reach the space and the level with the widest interlaminar space for needle passage. It can increase success and decrease technical difficulty, the number of needle reinsertions and redirections and the risk of traumatic procedures, and may therefore improve safety.[11]
- While this technique may not be of benefit to all patients, it may be particularly useful in those with abnormal anatomy or in those whose bony landmarks are not palpable. It is, however, a difficult skill in the difficult patient (e.g. morbid obesity) where it is most useful. Practice is required to become competent.
- Ultrasound can also be used as a teaching aid as it allows demonstration of the anatomy by the instructor and confirmation to the student that they have identified the correct insertion point and maximum depth to insert the needle (Fig. 40.1).[12]

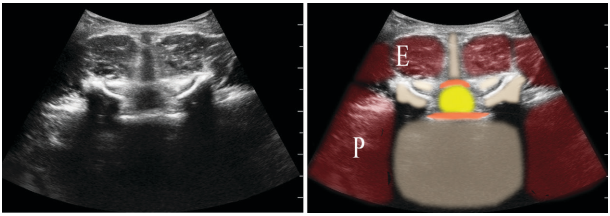

Fig. 40.1 Low lumbar transverse interlaminar sonoanatomy. Erector spinae (E), psoas (P), interspinous ligament, facet joints and vertebral body (tan), posterior and anterior complexes (orange), spinal canal (yellow). Courtesy of Mark Fairley.

A suggested technique for pre-procedural ultrasound

If landmarks cannot be confidently palpated Using a low-frequency curvilinear probe placed transversely over the sacrum, slide the probe cephalad over the lumbar spine, marking the midline. With the probe then in a parasagittal plane just off the midline (parasagittal oblique interlaminar view), identify the sawtooth appearance of the lamina. Starting from the continuous white line of the sacrum, slide cephalad over the sawtooth pattern of the laminae, marking spinal levels to accurately identify levels, including L3/4. At this level, rotate the probe to a transverse orientation (transverse interlaminar view) and identify the interspinous ligament between spinous processes. Small movements of the probe are required. Rock the probe to ensure the interspinous ligament shadow is vertical on the screen—this reveals any rotation of the spine. Tilt the probe to identify the anterior complex (anterior dura and vertebral body) and posterior complex (ligamentum flavum and dura). Note the depth to the posterior complex as this indicates the depth to the dura. Take account of indentation of the skin from probe pressure. When the best view of the anterior complex is achieved (this indicates a bony window to the spinal canal), the direction of the ultrasound beam is the direction the needle should be inserted. Look at the probe, note any tilt or angulation, mark the position on the patient by marking the centres of each side of the probe, remove the probe and draw your crosshairs, marking the needle insertion site. Perform the procedure knowing, at what level you are, that there is a bony window to the spinal canal, the direction required and the depth to space.

If landmarks can be confidently palpated The first half of this procedure can be omitted, and the level selected by palpation. This space can then be quickly checked with one probe position (transverse interlaminar) for: a bony window (anterior complex visible), any rotation of the spine (L or R rocking of probe required to make the interspinous ligament shadow vertical on the screen), the angle of insertion between the spinous processes (cephalad or caudad tilt of the probe) and the depth to space (depth to the posterior complex). Put the probe down and perform the procedure with this extra information.

Epidural block

(See ➲ pp. 841–4; ➲ pp. 855–6; ➲ p. 1103; ➲ pp. 1114; ➲ p. 1157; ➲ pp. 1164–6.)

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Nerve blocks: neck

Cervical plexus block

Indications Analgesia or anaesthesia for carotid surgery and other superficial neck procedures.

Positioning Lateral, or supine with the patient's head turned to contralateral side.

Ultrasound 'Intermediate' block of superficial cervical plexus nerves usually performed. Place probe transverse on lateral neck, starting at about cricoid level. Identify C7 (absent anterior tubercle) and C6 (wide foramen, prominent anterior tubercle) transverse processes. Slide cephalad, identifying C5 and C4 (carotid often divides at C4). Block usually performed at C4 level. Identify lateral border of sternocleidomastoid muscle (SCM) superficially. Target is plane between deep border of SCM and deep cervical fascia (prevertebral fascia) overlying deep muscles. Branches of the cervical plexus may be seen travelling in this plane from anteromedially, as they leave the transverse processes, to posterolaterally where they wrap around the posterior boarder of the SCM. Insert needle IP from posterior, infiltrating this plane and surrounding any nerve-like structures (Fig. 40.2).[12]

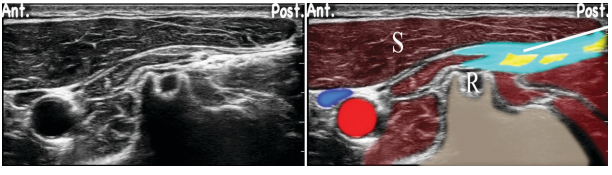

Fig. 40.2 Cervical plexus sonoanatomy. Cervical plexus (yellow), carotid artery (red), internal jugular vein (blue), C5 transverse process (tan), muscles (burgundy), C5 nerve root (R), SCM (S), needle path (white line), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique 'Superficial' block (SC infiltration) usually performed. The point of injection is at the midpoint of the posterior border of the SCM. Injection fanned SC cephalad and caudad from this point along the posterior border of the SCM.

Volume 10–15mL.

Side effects Nil with SC 'superficial' block. Deeper needle insertion during 'intermediate' block may result in phrenic, recurrent laryngeal and brachial plexus nerve block.

Complications Nerve injury, haematoma.

Tips 'Deep' cervical plexus block at nerve root depth, at higher cervical levels, has greater risks and side effects and is rarely indicated.

Caution in severe respiratory compromise or contralateral phrenic palsy due to risk of phrenic block.

Nerve blocks: upper limb

(See Figs. 40.32, 40.33, 40.34 and 40.36.)

Interscalene brachial plexus block

Indications Analgesia for shoulder surgery.

Positioning Lateral, or supine with head turned to contralateral side.

Ultrasound The brachial plexus roots can be visualised lateral to the carotid and internal jugular between the scalene muscles. The classic 'traffic lights' pattern of nerve roots in the interscalene groove usually represents C5 and C6 divided, alternatively C5, C6 and C7. A muscle bridge commonly exists between C7 and C8, impairing imaging and LA spread to C8 and T1. C7 transverse process is usually deeper than those above and has an absent anterior tubercle which allows imaging of the vertebral artery and vein. C6 transverse process is often wide with a prominent anterior tubercle (Chassaignac's tubercle). The posterior IP approach is the most popular, although anterior and OOP approaches are also used. Position the probe in the transverse plane with the carotid vessels on the edge of the image and the interscalene groove in the centre. Identify C5, C6 and C7 roots (Fig. 40.3).[12]

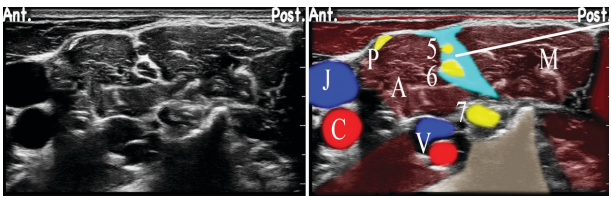

Fig. 40.3 Interscalene brachial plexus sonoanatomy. C5 (5), C6 (6), C7 (7) roots (yellow), carotid artery (C), internal jugular vein (J), vertebral artery and vein (V), C7 transverse process (tan); muscles (burgundy), scalenus anterior (A), scalenus medius (M), phrenic nerve (P), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique Insertion point is at the level of the cricoid cartilage (C6), lateral to the lateral border of the SCM, in the palpable 'groove' between the scalenus anterior and the scalenus medius. Direct the needle towards the contralateral elbow. The nerves are very superficial, a depth of no greater than 2.5cm.

PNS twitches C5 deltoid, C6 biceps. Triceps or pectoralis major contractions may be acceptable if deltoid or biceps contractions are not found. Phrenic nerve stimulation means the needle is too anterior. Levator scapulae stimulation indicates the needle is too posterior (on the dorsal scapular nerve).

Volume 10mL with ultrasound technique. Traditionally ~20mL with landmark technique.

Side effects Phrenic nerve block (up to 100%), subjective dyspnoea, Horner's syndrome, recurrent laryngeal nerve block causing a hoarse voice.

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Complications Minor paraesthesiae lasting days or weeks are more common with this block than others; vessel puncture, intravascular injection, intrathecal or epidural injection, pneumothorax, damage to nerve roots, damage to dorsal scapular or long thoracic nerve as they pass through middle scalene.

Tips This block should ideally be performed on awake or lightly sedated patients by experienced clinicians due to risks of serious complications. Avoid in patients with severe respiratory disease and contralateral phrenic nerve palsy because of phrenic nerve block.

Supraclavicular brachial plexus block

Indications Analgesia or anaesthesia for distal humerus, elbow, forearm, wrist or hand surgery.

Positioning Supine or semi-reclined with head turned to contralateral side.

Ultrasound The brachial plexus is superficial and easily visible as it passes over the 1st rib, lateral to the subclavian artery. The probe is placed parallel and cephalad to the clavicle, in the supraclavicular fossa. An IP technique with lateral needle entry is most popular. The brachial plexus appears as a 'triangular-shaped bunch of grapes' immediately lateral to the subclavian artery. If the probe is angled caudad slightly under the clavicle, it is usually possible to image the plexus and subclavian artery on top of the 1st rib, with the pleura deep on both sides of the rib shadow. Performing the procedure with this image of the plexus on the rib, rather than on the pleura, reduces the pneumothorax risk. The dorsal scapular artery may often be seen passing through the plexus at this level—check with colour Doppler (Fig. 40.4).[12]

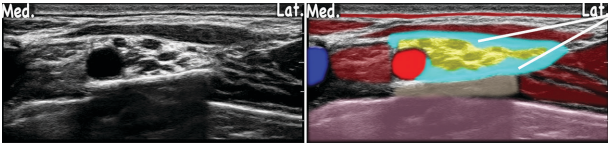

Fig. 40.4 Supraclavicular brachial plexus sonoanatomy. Brachial plexus (yellow), subclavian artery (red), subclavian vein (blue), pleura and lung (pink), 1st rib (tan), muscles (burgundy), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Volume 20–30mL as necessary to surround the plexus.

PNS twitches Finger flexion or extension.

Side effects Horner's syndrome, phrenic nerve block.

Complications Pneumothorax, artery puncture, intravascular injection. Paraesthesia is common from needle–nerve contact.

Tips Colour-flow Doppler is advised to identify all vessels close to, or passing through, the plexus. Avoid in patients with severe respiratory disease and contralateral phrenic nerve palsy because of risk of phrenic nerve block. Injection should include the angle between the artery and the 1st rib (the 'corner pocket') to increase success rate of blocking the lower trunk. The suprascapular nerve (needed for shoulder coverage) has often just left the plexus at this level and may be seen travelling posterolaterally under the omohyoid.

Infraclavicular paracoricoid brachial plexus block

Indications Analgesia or anaesthesia for elbow, wrist or hand.

Positioning Supine with the arm adducted and resting by the side.

Ultrasound The ultrasound probe should be positioned in a parasagittal plane just medial to the coracoid process. Deep to the pectoralis muscles, the axillary artery is seen cephalad to the vein, with the lateral, medial and posterior cords around the axillary artery. The cords of the brachial plexus can be deep and difficult to visualise. Using an IP technique from cephalad, the needle tip should be advanced until it lies posterior to the artery (Fig. 40.5).[12]

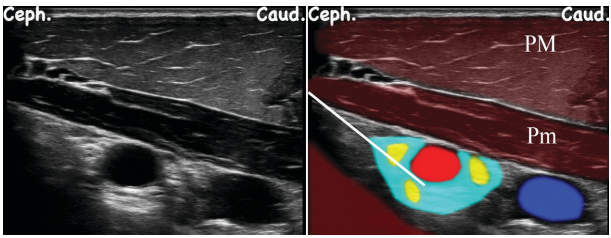

Fig. 40.5 Infraclavicular paracoricoid brachial plexus sonoanatomy. Brachial plexus cords (yellow), axillary artery and vein (red, blue), pectoralis major (PM), pectoralis minor (Pm), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique The coracoid process of the scapula may be palpated inferior to the lateral 3rd of the clavicle. It must be differentiated from the acromion, which can be palpated as a bony continuation of the distal clavicle. The point of insertion is 1.5cm medial and 1.5cm caudal to the most anterior point of the coracoid. The depth can vary between 3cm and 9cm, depending on body mass.

PNS twitches Pectoralis major: expected at 1–2cm depth (needle too superficial); lateral cord: elbow flexion (do not accept; too lateral); medial cord: wrist flexion (acceptable); posterior cord: wrist or finger extension (optimal).

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Volume 20–30mL.

Side effects Nil.

Complications Vascular puncture, intravascular injection, pneumothorax.

Tips Never angle the needle medially as this can increase the risk of pneumothorax.

Axillary brachial plexus block

Indications Anaesthesia for hand surgery; analgesia for forearm, wrist or hand surgery.

Positioning Supine with the arm abducted to 90°.

Ultrasound The ultrasound probe should be positioned high in the axilla along the axillary crease, imaging the axillary artery in short axis. Three nerves sit adjacent to the artery. The median nerve is usually superficial and anterior, the ulnar nerve superficial and posterior, the radial nerve deep and posterior. The musculocutaneous nerve is more anterior, most commonly in the plane between the biceps and the coracobrachialis, or in the body of the coracobrachialis (Fig. 40.6).[12]

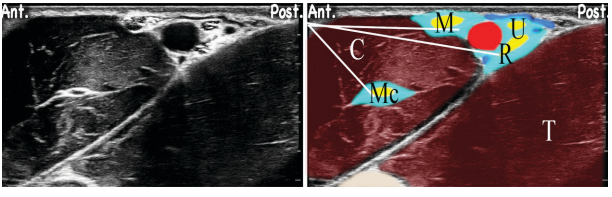

Fig. 40.6 Axillary brachial plexus sonoanatomy. Median nerve (M), ulnar nerve (U), radial nerve (R), musculocutaneous nerve (Mc), axillary artery and compressed veins (red, blue), humerus (tan), coracobrachialis (C), triceps (T), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

PNS twitches Radial: thumb, wrist or finger extension; ulnar: adduction of the thumb, little finger flexion; median: finger and wrist flexion and pronation of the wrist; musculocutaneous: biceps and brachialis contraction.

Volume 20mL.

Complications Artery puncture (compress for 5min if it occurs), intravascular injection.

Tips Highly variable anatomy exists. Following the nerves up from their location in the arm may help with identification in the axilla. There may be multiple veins, usually compressed by probe pressure. Great care should be exercised to avoid IV injection of LA. Aspirate and ensure LA spread in tissues is observed with injection.

Suprascapular nerve block, posterior approach

Indications Analgesia of shoulder, as part of multimodal analgesia for arthroscopic shoulder surgery or when interscalene block contraindicated. Often combined with other nerve blocks, e.g. axillary nerve.

Positioning Sitting or lateral.

Ultrasound Place the probe parallel and immediately cephalad to the spine of the scapula. Tilt probe caudad to image floor of the suprascapular fossa, deep to the trapezius and supraspinatus. Slide probe laterally until 'hockey stick' appearance of lateral end of the suprascapular fossa. The nerve and artery run in a fascial compartment deep to the supraspinatus. Artery sometimes seen; nerve rarely seen (Fig. 40.7).[12]

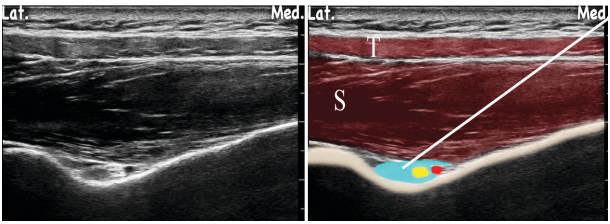

Fig. 40.7 Suprascapular nerve sonoanatomy. Suprascapular nerve (yellow), suprascapular artery (red), suprascapular fossa (tan), trapezius (T), supraspinatus (S), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

PNS twitches Supraspinatus and infraspinatus.

Volume 10mL.

Complications Arterial puncture.

Tips Nerve often not seen, but fascial compartment deep to the supraspinatus easily seen and filled with LA. Can be deep in muscular patients, impairing imaging. Bone backstop aids safety. If the probe is tilted cephalad, the suprascapular notch may be imaged (break in continuous white line of suprascapular fossa bone). Do not perform block in notch as this increases the risk of pneumothorax.

Axillary nerve block

Indications Analgesia of shoulder. Innervates anterior joint, deltoid and overlying skin. Usually combined with suprascapular nerve block for arthroscopic shoulder surgery or when interscalene contraindicated.

Positioning Sitting or lateral.

PNS twitches Anterior deltoid.

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Ultrasound The probe is placed in a parasagittal plane over posterolateral humeral neck. The posterior circumflex humeral vessels are identified coursing around the neck of the humerus. The nerve is often not seen but runs in proximity to (usually cephalad to) the vessels, deep to the deltoid, distal to teres minor insertion, proximal to triceps insertion (Fig. 40.8).[12]

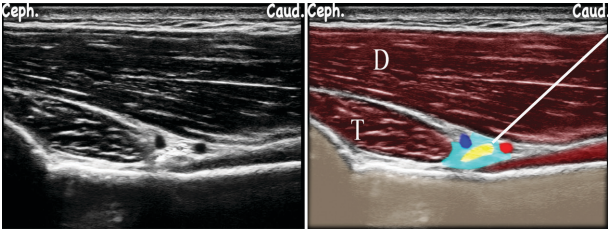

Fig. 40.8 Axillary nerve sonoanatomy. Axillary nerve (yellow), posterior circumflex humeral vessels (red, blue), humerus (tan); deltoid (D), teres minor (T), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Volume 10mL.

Complications Vessel puncture.

Tips Needle can be inserted from cephalad or caudad end of probe.

Intercostobrachial nerve block

Indications Anaesthesia of skin to upper medial arm. Commonly added to brachial plexus blocks for vascular surgery at ACF that may extend proximally to medial aspect of arm or for tourniquet tolerance.

Positioning Arm abducted 90°.

Ultrasound Place probe transversely in axillary crease to image axillary vessels. Slide probe posteriorly, placing vessels on anterior edge of image. Insert needle IP from anterior to infiltrate from vessels posteriorly and superficial to deep fascia of arm (superficial to muscles). Nerve not easily identified. Infiltrate plane and surround any nerve-like structures.

Landmark technique Palpate axillary artery in axillary crease. Perform SC infiltration from artery posteriorly.

Complications Vessel puncture.

Tips Intercostobrachial nerve block can also be achieved via a high superficial serratus anterior plane block; however, more volume will be required.

Median, ulnar and radial nerve blocks in the forearm

Indications Anaesthesia or analgesia of hand.

Positioning Supine with arm abducted 90°.

Ultrasound Allows these nerves to be blocked anywhere along their course from the axilla to the wrist.

Median nerve Commonly traced proximally from carpel tunnel and blocked mid forearm between superficial and deep flexor digitorums. Alternatively, in the ACF, it sits medial and slightly deep to the brachial artery between the brachialis and pronator teres. In the medial aspect of the upper arm, it is superficial, adjacent to the brachial artery (Fig. 40.9).[12]

Fig. 40.9 Median nerve mid-forearm sonoanatomy. Median nerve (yellow), ulnar and radial arteries (red), ulnar and radius (tan), deep flexor digitorum (D), superficial flexor digitorum (S), flexor carpi radialis (F), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Radial nerve Commonly blocked in lateral aspect of distal upper arm where it leaves the spiral groove of the humerus and travels in the plane between the brachialis and brachioradialis before dividing into superficial and deep branches around the level of the lateral epicondyle of the humerus (Fig. 40.10).[12]

Fig. 40.10 Radial nerve ACF sonoanatomy. Radial nerve (yellow), brachial artery (red), cephalic and cubital veins (blue), brachioradialis (Br), brachialis (B), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Ulnar nerve Commonly traced up from the medial wrist where it is found adjacent to the ulnar artery. The nerve and artery separate from each other two-thirds of the way up the forearm, which is a commonly blocked location. Alternatively, in the medial aspect of the upper arm, it is superficial and posterior to the brachial artery and median nerve where it courses with the ulnar collateral vessels before passing deep towards the cubital tunnel of the elbow (Fig. 40.11).[12]

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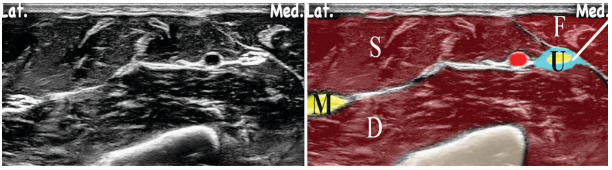

Fig. 40.11 Ulnar nerve mid-forearm sonoanatomy. Ulnar nerve (U), ulnar artery (red), ulnar (tan); deep flexor digitorum (D), superficial flexor digitorum (S), flexor carpi ulnaris (F), median nerve (M), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique

Median nerve Position the patient with their arm slightly abducted, elbow slightly flexed and forearm supinated. Feel the brachial artery at the antecubital fossa crease. The median nerve lies medial and deep to the artery. A pop may be felt on passing through the fascial plane to reach the nerve. It usually lies at 1–2cm depth.

Radial nerve Position as above. Radial nerve lies between the insertion of the biceps and brachioradialis, proximal to the flexor crease in the ACF. It is slightly deeper than the median nerve at 2–4cm depth.

Ulnar nerve The elbow should be slightly flexed, with the arm abducted at the shoulder and externally rotated to expose the ulnar groove at the elbow, or with the hand on the contralateral shoulder and arm across the chest. The ulnar nerve lies in the groove between the medial epicondyle of the humerus and the olecranon process. Pressure neurapraxia may develop from blocking at the groove, so the point of injection is often 2–3cm proximal to this, at a depth of 1–3cm.

PNS twitches

Radial Thumb, wrist or finger extension.

Ulnar Adduction of the thumb, little finger flexion.

Median Finger and wrist flexion and pronation of the wrist.

Volume 5mL per nerve.

Complications Artery puncture.

Wrist block

Indications Analgesia or anaesthesia for hand surgery.

Landmark technique

Median nerve Passes between the palmaris longus (look for the tendon in the middle of the wrist when clenching the fist and flexing the wrist) and the flexor carpi radialis. Inject 2–3cm proximal to the wrist creases, at ~1cm depth

Ulnar nerve Runs between the ulnar artery and flexor carpi ulnaris, deep to both. Inject 1–2cm proximal from the wrist creases from the ulnar side of the wrist towards the radius underneath the flexor carpi ulnaris, 1cm depth.

Radial nerve Becomes SC 3–5cm proximal to wrist joint. Can be blocked by infiltrating SC ~2–3cm proximal to the anatomical snuffbox at the base of the thumb over the dorsum of the radius.

Tips Avoid median nerve block and wrist block in patients with carpal tunnel syndrome.

Digital (ring) block

Indications Distal finger or toe surgery.

Landmark technique The nerves run on either side of the phalanges, two on the palmar side and two on the dorsal side of each finger.

- Insert a 25G needle just distal to the metacarpophalangeal joint from the dorsal side (less painful), past the proximal phalanx on either side of the finger to be blocked.
- Inject 2–4mL of LA (non-adrenaline-containing) on either side, while withdrawing the needle.

Complications Vascular puncture, digital ischaemia.

Intravenous regional anaesthesia—Bier's block

Indications Anaesthesia for superficial arm surgery or fracture reduction. Maximum operation length limited to about 30min. Can be used for lower limb.

Technique Measure the patient's BP. Insert one IV cannula into the limb requiring surgery, and one into another limb. Apply a double- or single-cuff tourniquet to the upper arm. Reliable arterial compression cannot be obtained over the forearm as vessels will be held open between the radius and ulna. The limb should be exsanguinated with a compression bandage or by elevation if fractured. The cuff should then be inflated to 50–100mmHg above the patient's systolic BP. A non-adrenaline-containing LA with low systemic toxicity, such as prilocaine 0.5%, should be used. Inject slowly: 40mL for small, 50mL for medium and 60mL for a large arm. Alternatively, lidocaine 0.5% can be used, maximum dose 250mg. Other LAs are not appropriate. The patient should be warned that the arm will begin to feel warm and appear mottled. Surgery can start within a few minutes. On no account should the tourniquet cuff be deflated before 15min for prilocaine and 20min for lidocaine. Potentially devastating systemic toxicity can occur if large volumes of LA are released before it becomes bound or metabolised. If tourniquet pain is experienced during the procedure and a double cuff is used, the distal cuff can be inflated before deflating the proximal cuff. The tissue under the distal cuff should be anaesthetised at this stage. The technique is contraindicated if pre-existing circulatory difficulties, e.g. crush injury, homozygous SCD, peripheral vascular disease. A reliable tourniquet and resuscitation equipment are essential.

Complications LAST.

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Nerve blocks: trunk

(See Fig. 40.36.)

Anatomy of the nerve supply to the thorax and abdomen

The muscles and skin of the chest and abdomen are supplied by the spinal nerves T2–T12, with a contribution from L1 in the inguinal region. These mixed spinal nerves emerge from the intervertebral foramen into the paravertebral space, dividing into the dorsal and ventral rami. The dorsal rami supply the deep muscles and skin over the dorsum of the trunk. The ventral rami form the intercostal nerves, which pass into the neurovascular plane between the internal and innermost intercostal muscles. A lateral cutaneous branch is given off before the costal angle, piercing the intercostal and overlying muscles in the mid-axillary line. The intercostal nerves end as an anterior cutaneous nerve.

Thoracic paravertebral block

Indications Analgesia for breast surgery, thoracic surgery, open cholecystectomy, renal surgery or fractured ribs.

Positioning Sitting or lateral.

Ultrasound Imaging the paravertebral space is difficult due to depth and predominance of bone shadows and is recommended for experienced practitioners.

- With the probe in the sagittal plane over midline spinous processes, slide laterally to identify transverse processes. Too far laterally and curved ribs with the pleura between will be easily imaged. Slide back medially and the pleura and ribs disappear deep and the more superficial, squared-off appearance of the transverse processes come into view. Tilting the probe laterally will help to image the pleura as it dives deep under the transverse processes (Fig. 40.12).[12]

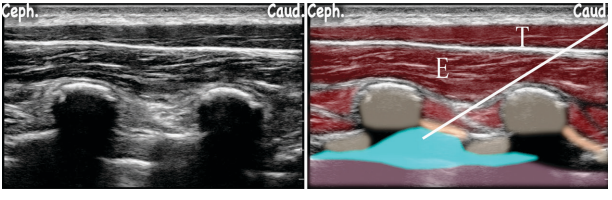

Fig. 40.12 Thoracic paravertebral sagittal view. Pleura and lung (pink), superior costotransverse ligament (orange), transverse processes (superficial, tan) rib necks (deep, tan), trapezius (T), erector spinae (E), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

- Alternatively, with the probe in the transverse orientation, the spinous process can be placed on the edge of the image and the contour of the lamina rising to the transverse process can be imaged with the bone shadow deep to it. Lateral to the transverse process, the pleura or rib are imaged. Slide or tilt the probe off the rib to image the pleura

disappearing under the transverse process. The innermost intercostal membrane can be imaged as it extends from adjacent to the pleura to the transverse process (Fig. 40.13).[12]

Fig. 40.13 Thoracic paravertebral transverse view. Pleura and lung (pink), innermost intercostal membrane (orange), spinous process and transverse process (tan), trapezius (T), erector spinae (Es), external intercostal muscle (E), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

- Following identification of relevant sagittal or transverse sonoanatomy as above, an IP or OOP needle approach can be used. Needle imaging is difficult due to the steep angle of insertion. Use bony landmarks and hydrolocation to identify the needle tip. Depression of the pleura with injection is a positive sign of correct needle tip placement.
- Alternatively, the transverse process location and depth can be marked with ultrasound and then the landmark technique used.

Landmark technique Palpate the spinous processes. The needle insertion point is 2.5cm lateral to the cephalad aspect of the spinous process at the desired block level. The needle should be inserted to contact the transverse process usually at a depth of 2–4cm; note the depth at which this occurs. After contact with bone, withdraw the needle slightly and change the angle such that the needle will pass cephalad to the transverse process. At this point, a loss of resistance technique can be used, or the needle can be simply inserted 1cm further than the depth at which the transverse process was first encountered. Aspirate to exclude blood or air before injecting the LA.

Volume 5mL per level or 20mL at a single level.

Side effects Epidural spread, sympathetic block.

Complications Pneumothorax, LA toxicity, intravascular injection.

Tips To reduce the risk of pneumothorax, try to keep the needle tip in the ultrasound image at all times. If imaging or block performance is too difficult to ensure safe practice, an alternative and more superficial block, e.g. erector spinae plane or retrolaminar, may be chosen.

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Erector spinae plane block

Indications Still being clarified by clinical trials. Consider for chest wall analgesia, e.g. rib fractures, thoracotomy, breast surgery, when paravertebral risk considered too great, or too difficult to perform.

Positioning Sitting or lateral.

Ultrasound With the probe in the sagittal plane over midline spinous processes, slide laterally to identify the transverse processes. Too far laterally and the curved surface of the ribs with the pleura between will be easily imaged. Slide back medially and the pleura and ribs disappear deep and the more superficial, squared-off appearance of the transverse processes come into view. The muscles immediately superficial to the transverse processes are the erector spinae. Insert the needle IP from the caudad end of the probe. Aim for the cephalad side of the transverse process. Hit bone and pull back a touch or advance past the cephalad edge of the transverse process, injecting a small amount to identify the opening of the correct fascial plane between the erector spinae and the transverse process. The LA should open an elliptical fascial plane space in the cephalad–caudad direction when correctly positioned (Fig. 40.14).[12]

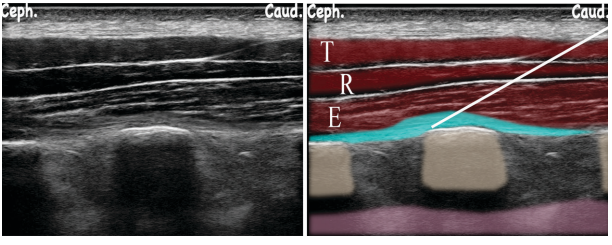

Fig. 40.14 Erector spinae sonoanatomy. Pleura and lung (pink), transverse processes (tan), trapezius (T), rhomboids (R), erector spinae (E), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Volume 20–30mL.

Complications Pneumothorax, LA toxicity, sympathetic block, epidural spread.

Tips Can be performed bilaterally and at multiple levels.
A useful option if paravertebral considered too difficult.

Pectoserratus plane (PSP) and interpectoral plane (IPP) blocks

Indications Analgesia to breast, axilla, pectoral muscles and anterolateral chest wall.

Positioning Supine with arm abducted 45–90°.

Ultrasound Place probe in a parasagittal orientation in deltopectoral groove just below clavicle. Identify pectoralis major and minor. Rotate distal end of probe laterally towards axilla 30–45°. Slide probe inferolaterally along the direction it is pointing towards the axilla. Identify serratus anterior deep to pectoralis minor and immediately superficial to ribs. The target planes are between the two pectoral muscles and between pectoralis minor and serratus anterior. Insert needle from cephalad end of probe to deposit LA in these planes (Fig. 40.15).[12]

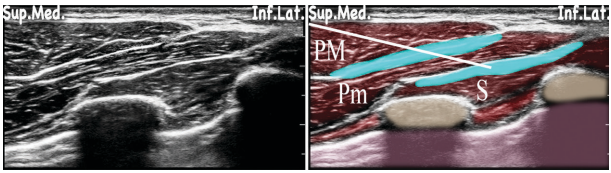

Fig. 40.15 PSP and IPP sonoanatomy. Pleura and lung (pink), ribs (tan), pectoralis major (PM), pectoralis minor (Pm), serratus anterior (S), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Volume 20–30mL.
Side effects Some surgeons have objected to PSP and IPP for breast cancer surgery as LA tracks to the axilla, distorting fascial planes and there is a risk of needle trauma to malignant nodes.
Complications Vessel puncture, pneumothorax, LAST.
Tips IPP block involves placing LA between the pectoralis major and minor only. This only blocks the medial and lateral pectoral nerves innervating the pectoral muscles. Useful for breast implants or pacemaker insertions that are deep to pectoralis major. PSP and IPP also blocks the lateral cutaneous branches of the intercostal nerves, providing analgesia to the anterolateral chest wall, the intercostobrachial nerve innervating the axilla and the long thoracic nerve innervating the serratus anterior.

Ilioinguinal and iliohypogastric block

Indications Analgesia for inguinal hernia, orchidopexy or hydrocele surgery.

Positioning Supine.

Ultrasound The ilioinguinal and iliohypogastric nerves are branches of the lumbar plexus. The nerves run initially in the TAP before piercing first the internal oblique (IO) and then the external oblique (EO) muscles to provide sensory innervation over the lower abdomen and upper thigh. Ultrasound can be used to identify the correct planes, or it may be possible to locate specific nerves. Place the probe between the ASIS and the umbilicus, and scan caudally. Blood vessels may lie with the nerves and aid in identification. Use an IP technique with the needle from either end of the probe. The nerves most commonly lie between the transversus abdominis (TA) and IO, but variations are common (Fig. 40.16).[12]

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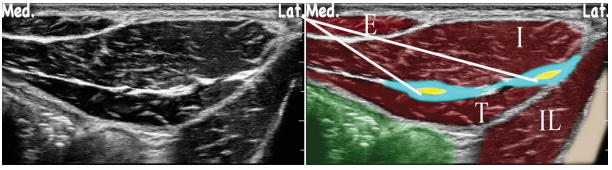

Fig. 40.16 Ilioinguinal and iliohypogastric nerves sonoanatomy. External oblique (E), internal oblique (I), transversus abdominis (T), iliacus (IL), ilium (tan), peritoneum (green), ilioinguinal and iliohypogastric nerves (yellow), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique The classical technique relies on performing a plane block between the TA and IO and between the IO and EO to block the ilioinguinal and iliohypogastric nerves, respectively. The point of needle insertion is 2cm medial to the ASIS. Inject after the 1st pop; insert the needle deeper and inject after the 2nd pop.

LA dose 5–10mL in each plane.

Side effects Femoral nerve block.

Complications Puncture of bowel, intravascular injection.

Tips Nerves often close to ilium at ASIS level; they will travel medially as you scan caudally.

Penile block

Indications Circumcision.

Landmarks Palpate the symphysis pubis above the root of the penis. Insert a 25G needle at the lateral base of the shaft of the penis to just touch the inferior border of the pubis. When contact is made, withdraw slightly; change the needle angle to pass just beneath the pubis—a pop may be felt at 1–2cm when Buck's fascia is pierced. If an assistant pulls down slightly on the end of the penis, the passage through the fascia will become more apparent. Inject 5mL (or 0.1mL/kg of bupivacaine 0.5% in children); repeat on the other side. Performing an additional infiltration SC around the root of the penis onto the scrotum blocks input from the ilioinguinal and genitofemoral nerves but increases the risk of bleeding.

Complications Haematoma.

Tips Never use adrenaline-containing solutions. A caudal block may be easier and a more appropriate analgesia for circumcision in infants. If stimulation is noted during the surgery, ask the surgeon to supplement the block (most commonly around the frenulum).

Midaxillary transversus abdominis plane (TAP) block

Indications Analgesia for incisions to the lower abdominal wall.

Ultrasound Place the ultrasound probe on the lateral abdominal wall between the costal margin and the iliac crest to image in the transverse plane. Identify the three muscle layers (EO, IO and TA). The TA is usually the thinnest and the most hypoechoic layer. Deep to the TA, intraperitoneal structures will be seen moving. An IP technique with the needle coming from the anterior is used. The target plane is between the IO and the TA (Fig. 40.17).[12]

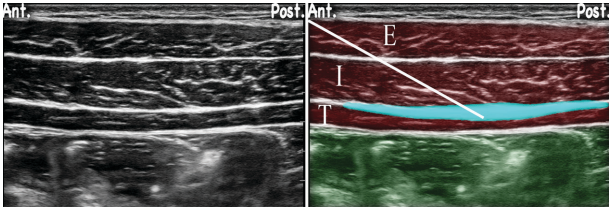

Fig. 40.17 Midaxillary TAP sonoanatomy. Peritoneum and intraperitoneal (green), external oblique (E), internal oblique (I), transversus abdominis (T), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique Classically, the block is performed in the triangle of Petit, which is located above the iliac crest, with the EO muscle forming the anterior border and the latissimus dorsi the posterior border. The EO is still present as fascia in this area, so two pops should be felt as the needle passes perpendicular to the skin, just above the iliac crest, before injection of the LA.

Volume 20–30mL per side.

Complications Puncture of bowel, intrahepatic or intrasplenic injection, LAST.

Tips Be careful with LA dose when performing this bilateral large-volume block. A subcostal TAP block can also be performed to cover incisions from T8 to T10.

Rectus sheath block

Indications Analgesia for upper midline abdominal incisions.

Ultrasound Place the probe in a transverse orientation on the anterior abdominal wall at or above the umbilicus level, depending on incision location. Identify the three lateral muscle layers, the linear semilunaris, the rectus muscle and the peritoneum. The target plane is immediately superficial to the posterior rectus sheath. Use an IP technique from lateral to inject between the rectus muscle and the posterior rectus sheath (Fig. 40.18).[12]

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Fig. 40.18 Rectus sheath block sonoanatomy. Peritoneum and intraperitoneal (green), linear semilunaris (orange), rectus abdominis (R), external oblique (E), internal oblique (I), transversus abdominis (T), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique To perform the block, four points should be marked at 5cm cephalad/5cm lateral and 5cm caudad/5cm lateral on each side of the umbilicus. A short bevelled or blunted 22G needle should be inserted through the skin and SC tissue. The 1st fascial plane is the anterior rectus sheath. A scratch technique of wiggling the needle against the resistance (and feeling a 'scratching' sensation) may make it more apparent before 'popping' through the plane. The needle should then be inserted until a 2nd resistance is felt, but no further, and 10–15mL injected. The technique should be repeated at the other three locations.

Volume 20–30mL per side.

Complications Bowel puncture, LAST.

Tips Beware of superior epigastric vessels running in this plane, often seen with ultrasound. Perform bilaterally for midline incisions. Not possible on lower abdomen below arcuate line due to absence of posterior rectus sheath.

Nerve blocks: lower limb

(See Figs. 40.35 and 40.36.)

Fascia iliaca block

Indications Popular analgesia for patients suffering from a fractured neck of the femur, analgesia for hip surgery, femur and anterior and lateral thigh.

Positioning Supine. Flatten the bed as much as possible to open any flexing of the hip. Tilt the whole bed head-up if the patient requires some head-up positioning rather than flexing in the middle.

Ultrasound Two techniques described.

Infrainguinal As per femoral nerve block (see ➲ p. 1136), with the aim of identifying the fascia iliaca and placing the LA under the fascia iliaca a few cm lateral to the neurovascular bundle.

Suprainguinal The probe is placed over the ASIS in a parasagittal orientation. Slide the probe inferomedially along the inguinal ligament towards the pubic symphysis. After a few cm, the anterior inferior iliac spine should be seen as a peaked hyperechoic shape. Overlying this is the iliacus muscle with the EO muscle superficial and cephalad and the sartorius superficial and caudad, together making a 'bow-tie' appearance. The fascia iliaca is the hyperechoic fascial layer covering the superficial surface of the iliacus. The deep circumflex iliac vessels are usually seen superficial to the fascia iliaca in the pelvis. Insert the needle from the caudad end of the probe, aiming for LA to spread under the fascia iliaca, deep to deep circumflex iliac vessels, in a cephalad direction (Fig. 40.19).[12]

Landmark technique Find the junction of the middle and lateral thirds of the inguinal ligament, joining the ASIS and the pubic tubercle. Move 2cm distal and 2cm lateral. The aim is to be lateral to the neurovascular bundle. The needle needs to pass through the fascia lata (1st pop) and fascia iliaca (2nd pop). A large blunt needle, e.g. Tuohy, will facilitate feeling the pops.

Volume 30–40mL.

Complications Vascular puncture, nerve injury, LAST intraperitoneal injection.

Tips Reliably blocks the femoral nerve and usually the lateral femoral cutaneous nerve.

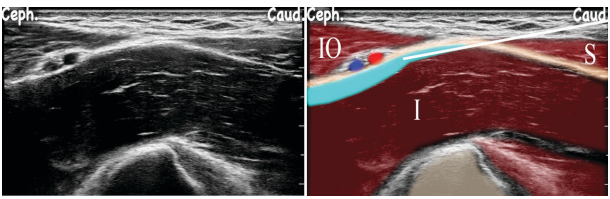

Fig. 40.19 Suprainguinal fascia iliaca sonoanatomy. Fascia iliaca (orange), deep circumflex iliac vessels (red, blue), anterior inferior iliac spine (tan), internal oblique (IO), sartorius (S), iliacus (I), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

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Femoral nerve block

Indications Analgesia for hip, knee or femoral shaft surgery. Combined with sciatic nerve block to produce anaesthesia or analgesia for surgery to the lower leg.

Positioning Supine. Flatten the bed as much as possible to open any flexing of the hip. Tilt the whole bed head-up if the patient requires some head-up positioning rather than flexing in the middle.

Ultrasound Place the probe in a transverse orientation in the inguinal crease over the femoral vessels. Identify the femoral vein and artery. Stay as proximal as possible. If the femoral artery has divided, you are usually too distal. Immediately deep and lateral to the artery, you will see a large hypoechoic muscle—the iliopsoas. On the superficial surface of the iliopsoas is a bright hyperechoic fascia, the fascia iliaca, that runs medially deep under the artery. A more superficial fascia that runs superficial to the artery is the fascia lata. The femoral nerve is usually seen as a flattened, hyperechoic honeycomb structure between the iliacus and the fascia iliaca lateral to the artery. Tilting the probe is often required due to anisotropy of the nerve. Advance the needle from the lateral end of the probe to penetrate the fascia iliaca at the lateral edge of the nerve. LA can be placed above and/or below the nerve or just below the fascia iliaca lateral to the artery if nerve not seen (Fig. 40.20).[12]

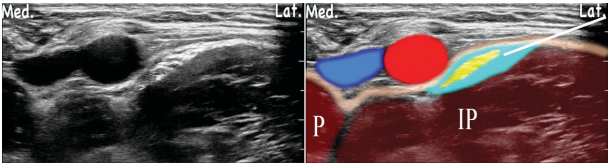

Fig. 40.20 Femoral nerve sonoanatomy. Femoral nerve (yellow), femoral artery and vein (red, blue), fascia iliaca (orange), iliopsoas (IP), pectineus (P), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique Palpate the femoral artery beneath the inguinal ligament; ~1–1.5cm lateral to this is the femoral nerve. Insert the needle 1cm distal to the ligament; two 'pops' may be felt as the needle passes the fascia lata, then the fascia iliaca. Depth to nerve of 2–4cm.

PNS twitches Patella dance.

Volume 10–20mL.

Complications Arterial puncture, intravascular injection, nerve injury.

Tips Imaging difficulty often due to scanning too distally. The femoral nerve quickly flattens and divides a few cm distal to the inguinal ligament. In obese patients, an assistant may be required to retract abdominal pannus.

PENG block

Indications Analgesia of hip joint. This is a new block still needing clinical trials to clarify indications.

Ultrasound Place transverse probe over ASIS. Scan caudally and medially a few cm to image the anterior inferior iliac spine. Rotate medial end of probe 45° and tilt caudally to image the ilium from the anterior inferior iliac spine to the iliopubic eminence as a continuous hyperechoic line. Too distal and the femoral head and joint space will be imaged. Identify femoral vessels and femoral nerve. Psoas tendon is often seen as a hyperechoic area deep in psoas muscle. Insert needle from lateral end of probe. Target is plane deep to psoas and psoas tendon immediately anterior to bone between anterior inferior iliac spine and iliopubic eminence (Fig. 40.21).[12]

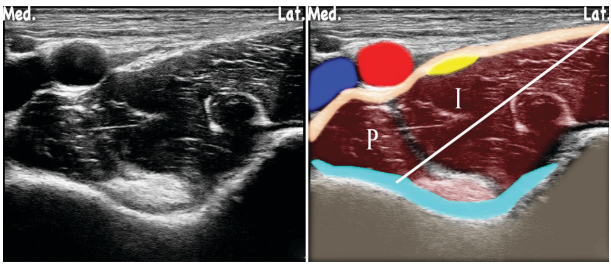

Fig. 40.21 PENG block sonoanatomy. Femoral artery (red), femoral vein (blue), femoral nerve (yellow), psoas muscle and tendon (P), iliacus (I), ilium from anterior inferior iliac spine to iliopubic eminence (tan), needle path (white), LA (cyan). Courtesy of Mark Fairley.

Volume 10–20mL.

Complications Vessel puncture, femoral nerve injury, bladder or ureter injury.

Tips If the femoral head is imaged, you are too distal and need to slide the probe proximally to image the ilium from anterior inferior iliac spine to iliopubic eminence.

Lateral femoral cutaneous nerve

Indications Analgesia for incisions on lateral thigh.

Ultrasound Place the probe transversely just distal to the ASIS and slide a few cm caudally. Identify the sartorius medially as the most superficial, more echogenic, boat-shaped muscle. Laterally is the tensor fascia lata. Superficially between the two is often a small, triangular, fat-filled compartment containing the small hyperechoic nerve. The nerve often divides as it is traced distally and usually runs superficial to the sartorius as it is traced proximally. The needle can be passed IP from the lateral end of the probe or OOP (Fig. 40.22).[12]

Landmark technique The nerve runs under the inguinal ligament, just medial to the ASIS and superficially over the sartorius muscle. The nerve can be blocked 2cm medial and 2cm caudal to the ASIS. Insert the needle perpendicular to the skin to a depth of 1–3cm, until you feel the needle pass through the fascia lata, and inject here.

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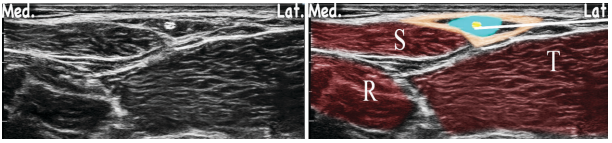

Fig. 40.22 Lateral femoral cutaneous nerve sonoanatomy. Sartorius (S), tensor fascia lata (T), rectus femoris (R), lateral femoral cutaneous nerve (yellow), fat-filled triangular fascial compartment (orange), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Volume 5–10mL.

Complications Nerve injury.

Tips If you are unable to visualise the nerve, you can infiltrate the plane superficial to the sartorius, just distal to the ASIS where the nerve usually runs.

Subgluteal sciatic nerve block

Indications Rarely indicated as more distal block usually preferred. Analgesia for ankle or foot surgery, or for lower limb amputation. Combined with femoral nerve block for analgesia of the leg.

Ultrasound Place a curvilinear probe midway between the greater trochanter and ischial tuberosity in an oblique transverse orientation. Identify the gluteus maximus superficially, the greater trochanter laterally, the ischial tuberosity medially and the quadratus femoris deep. The sciatic nerve is usually a flattened hyperechoic structure in the fascial plane joining these two bony landmarks deep to the gluteus maximus, superficial to the quadratus femoris. Nerve stimulation is helpful in confirming nerve location. IP needle from lateral side (Fig. 40.23).[12]

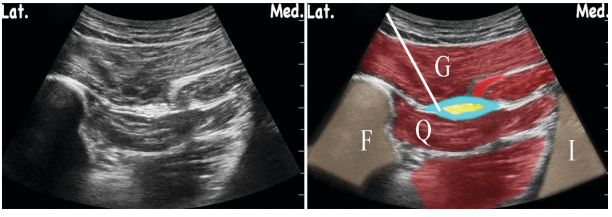

Fig. 40.23 Subgluteal sciatic sonoanatomy. Sciatic nerve (yellow), gluteus maximus (G), quadratus femoris (Q), femur (F), ischium (I), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique Position the patient in the Sims' position/recovery position, with the operative leg uppermost. Identify and mark the posterior superior iliac spine (PSIS), the greater trochanter of the femur and the sacral hiatus. Draw a line between the PSIS and greater trochanter, and between the greater trochanter and sacral hiatus. Draw a 3rd line perpendicular from the midpoint between the PSIS and greater trochanter to intersect the 2nd line. This is the point of needle insertion perpendicular to the skin to a depth of 5–10cm.

PNS twitches Tibial component—plantar flexion of the foot (optimal); common peroneal component—eversion and dorsiflexion of the foot (withdraw the needle and aim more medially); gluteal muscles—direct stimulation, needle too shallow.

Volume 15–20mL.

Complications Intravascular injection, nerve injury.

Tips Nerve can be difficult to see due to depth, anisotropy and sometimes a very flattened nerve in the fascial plane. Care with LA maximum doses if performing with femoral or other blocks.

Popliteal sciatic nerve block

Indications Very popular for foot, ankle and lower leg surgery. Covers everything below the knee, except skin on the medial leg, including medial malleolus periosteum (saphenous nerve).

Positioning Lateral preferred, but can be performed supine or prone.

Ultrasound Place the probe transversely in the popliteal fossa. Identify the popliteal vessels and the tibial nerve superficial to the vessels. Trace the tibial nerve proximally to identify the smaller common peroneal nerve coming from the lateral side to join the tibial nerve usually about 10cm (highly variable) above the popliteal crease. Block usually performed where the two nerves are first enclosed in a common sheath (Fig. 40.24).[12]

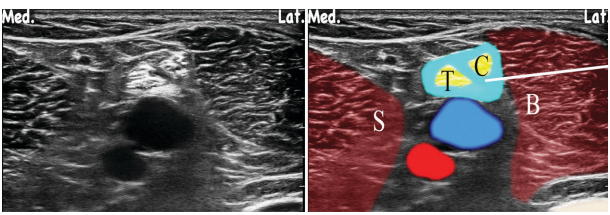

Fig. 40.24 Popliteal sciatic sonoanatomy. Tibial nerve (T), common peroneal nerve (C), popliteal artery and vein (red, blue), biceps femoris (B), semimembranosus (S), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

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Landmark technique Lateral approach, supine, with the knee slightly flexed, mark the groove between the vastus lateralis (above) and biceps femoris (below). Draw a line down from the superior border of the patella where it crosses this groove. Insert a 22G 100mm needle, directed posteriorly 25–30° and slightly caudally. The needle passes through the biceps femoris into the popliteal fossa, initially encountering the common peroneal nerve, then the tibial nerve.

PNS twitches Common peroneal nerve, foot dorsiflexion and eversion; tibial nerve, foot plantarflexion and inversion.

Volume 20mL.

Tips If this junction location is too deep and imaging poor (due to obesity), the nerves can be blocked more distally where they are more superficial and easily imaged as two separate nerves. Branches to the sural nerve may have come off and be missed if too distal. Anisotropy is prominent with these nerves; tilt probe to maximise brightness of nerve.

Saphenous nerve block

Indications Provides analgesia for the medial lower leg and ankle.

Usually combined with popliteal sciatic to complete coverage of lower leg and ankle.

Ultrasound The saphenous nerve is small and difficult to image in the leg. The adductor canal block is therefore usually performed.

Landmark technique The patient should be supine, with the leg externally rotated. Identify the tibial tuberosity and inject 10–15mL SC from the tibial tuberosity towards the medial tibial condyle.

Adductor canal and distal femoral triangle blocks

Indication Usually performed to achieve saphenous nerve and nerve to vastus medialis block for total knee replacement or combined with popliteal sciatic to complete lower leg coverage.

Positioning Supine with hip slightly flexed and externally rotated.

Ultrasound Place probe transversely over anteromedial aspect of mid thigh. Identify the femoral artery with the sartorius muscle superficial to it. The sartorius is usually a small, boat-shaped muscle slightly more echogenic than other muscles. The target is the area lateral to the artery deep to the vasoadductor membrane (bright fascial plane between the sartorius and the artery). The saphenous nerve is sometimes able to be visualised in this location. Pass the needle IP from the lateral end of the probe (Fig. 40.25).[12]

Volume 10mL.

Complications Artery puncture.

Tips Ensure LA spreads deep to vasoadductor membrane lateral to artery. Usually performed at mid thigh level which is usually a distal femoral triangle block. Sliding more distally becomes an adductor canal block which blocks saphenous nerve only.

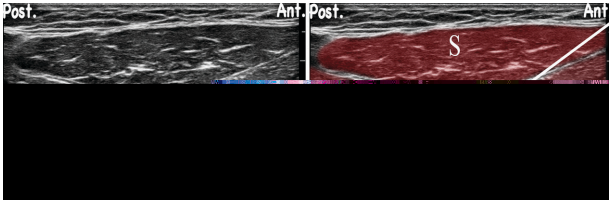

Fig. 40.25 Adductor canal sonoanatomy. Saphenous nerve (yellow), superficial femoral artery and vein (red, blue), sartorius (S), vastus medialis (V), adductor magnus (A), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

iPACK block

Indication Posterior capsule analgesia for total knee replacement, often combined with distal femoral triangle or adductor canal block for motor-sparing analgesia.

Positioning Supine, hip flexed and externally rotated, knee slightly flexed.

Ultrasound Place curvilinear probe over medial aspect of distal thigh just above level of proximal patella. Identify femur and popliteal artery. Target plane is between artery and femur. Slide probe posteriorly into a posteromedial location, so needle trajectory is parallel to posterior surface of femur. Insert needle from medial end of probe, and advance between femur and popliteal artery until 2cm lateral to artery. Inject a few mL here and remainder while withdrawing to infiltrate plane between artery and femur (Fig. 40.26).[12]

Fig. 40.26 iPACK block sonoanatomy. Femur (tan), popliteal artery and vein (red, blue), sartorius (S), semimembranosus (Sm), biceps femoris (B), vastus medialis (V), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Volume 10–20mL.

Complications Artery puncture, tibial or common peroneal nerve block.

Tips Easily combined with distal femoral triangle or adductor canal block.

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Ankle block

Indications Analgesia or anaesthesia to the foot.

Positioning Depending on the nerve to be blocked, position as convenient in figure 4 position, supine or lateral.

Ultrasound

- The **tibial nerve** is blocked first due to slowest onset. Place probe proximal to medial malleolus between tibia and Achilles tendon. Identify posterior tibial artery and veins. The tibial nerve usually lies posterior to vessels lying on the flexor hallucis longus muscle and sheath. The flexor hallucis longus tendon within the muscle should not be confused with the nerve. Trace these structures proximal and distal to confirm their identity. Pass needle IP from posterior end of probe (Fig. 40.27).[12]

Fig. 40.27 Tibial nerve ankle sonoanatomy. Tibial nerve (yellow), posterior tibial artery and veins (red, blue), great saphenous vein (S), tibialis posterior tendon (T), flexor digitorum longus (D), flexor hallucis longus (H), tibia medial malleolus (tan), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

- The **saphenous nerve** is usually the most difficult to image. Place the probe transversely over the anteromedial ankle with light pressure (or use a tourniquet) to identify the greater saphenous vein. Slide the probe proximally and distally on the vein to identify the saphenous nerve running in close proximity. It may be divided around the vein. Pass the needle from the posterior end of the probe to surround the vein if nerve not identified (Fig. 40.28).[12]

Fig. 40.28 Saphenous nerve ankle sonoanatomy. Tibia medial malleolus (tan), posterior tibial vessels and tibial nerve (P), great saphenous vein (S), saphenous nerve (yellow), flexor digitorum longus (D), tibialis posterior (T), flexor hallucis longus (H), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

- The **deep peroneal nerve** is identified with the probe transversely positioned over the anterior ankle joint. Identify the anterior tibial artery. Trace the artery proximally and back distally to identify the deep peroneal nerve as a small hypoechoic circle or a string of circles passing over the artery from medial to lateral. Pass the needle IP from medial or lateral, depending on the position of the nerve relative to the artery (Fig. 40.29).[12]

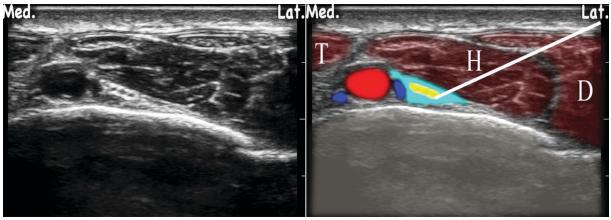

Fig. 40.29 Deep peroneal nerve ankle sonoanatomy. Tibia (tan), deep peroneal nerve (yellow), anterior tibial artery and veins (red and blue), tibialis anterior (T), extensor hallucis longus (H), extensor digitorum longus (D), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

- The **superficial peroneal nerve** is found with the probe transverse on the lateral aspect of the fibula. Scan from middle third to distal third, looking for peaking of the bony contour of the fibula pointing to the intermuscular fascial plane between the peroneus brevis posteriorly and the extensor digitorum longus anteriorly. The superficial peroneal nerve is usually seen exiting this intermuscular plane to continue distally in the SC plane. Pass the needle IP from anterior or posterior (Fig. 40.30).[12]

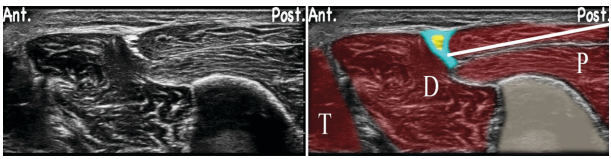

Fig. 40.30 Superficial peroneal nerve sonoanatomy. Fibula (tan), tibialis anterior (T), extensor digitorum longus (D), peroneus longus and brevis (P), superficial peroneal nerve (yellow), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

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- The **sural nerve** can be blocked by placing the probe transversely over the posterolateral leg just proximal to the lateral malleolus. Identify the peroneus brevis anteriorly, the Achilles tendon posteriorly and the lessor saphenous vein in the fascial plane between them. Use light probe pressure to avoid squashing the vein. The nerve is difficult to see but is usually near the vein. The nerve can be traced from mid-posterior calf where it exits the deeper planes between the medial and lateral heads of the gastrocnemius and then travels laterally in the SC tissues towards the lateral malleolus. Pass the needle IP from anterior or posterior, surrounding the vein if nerve not seen (Fig. 40.31).[12]

Fig. 40.31 Sural nerve ankle sonoanatomy. Fibula (tan), peroneus brevis (P), flexor hallucis longus (H), soleus (S), lesser saphenous vein (blue), sural nerve (yellow), needle path (white), LA spread (cyan). Courtesy of Mark Fairley.

Landmark technique Palpate the tibial artery posterior to the medial malleolus. To block the tibial nerve, inject just behind the tibial artery. The saphenous nerve is usually blocked by infiltrating a ring of 5mL of LA from the medial malleolus anteriorly to the tibial ridge. To block the deep peroneal nerve, palpate the dorsalis pedis artery and insert the needle just lateral to the artery. When contact is made with bone, withdraw the needle slightly and inject. To block the sural nerve, raise an SC wheal of LA from the lateral malleolus inferiorly to the Achilles tendon. The superficial peroneal nerve can be blocked by infiltrating 10mL SC medially and laterally over the dorsum of the foot, 2–3cm distal to the intermalleolar line.

Volume With ultrasound identification of nerves, 2–5mL each is usually enough. With the landmark technique and SC infiltration, 5–10mL each is often required.

Side effects Five injections can be painful in awake patients. Use a fine needle.

Complications Arterial puncture, nerve injury.

Tips A proximal tourniquet during ultrasound-guided block helps with vein filling and identification. For forefoot surgery, saphenous nerve block not required.

Resources

Illustrations

- Sensory innervation (Figs. 40.32–40.35)
- Cutaneous dermatomes (Fig. 40.36).

Websites

- American Society of Regional Anesthesia and Pain Medicine (ASRA). https://www.asra.com/
- European Society of Regional Anaesthesia and Pain Therapy (ESRA). https://esraeurope.org/
- New York School of Regional Anesthesia (NYSORA). https://www.nysora.com/
- Regional Anaesthesia United Kingdom (RA-UK). https://www.ra-uk.org/
- Ultrasound for Regional Anesthesia. http://www.usra.ca

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YouTube channels

- Ki-Jinn Chin. https://www.youtube.com/channel/UC8oN4Iw8d7uGzhARRrGZjiQ
- Vicente Roqués Escolar. https://www.youtube.com/channel/UCd6XscTpV0qK4DDEvfM3saQ
- LSORA Videos. https://www.youtube.com/channel/UCV8d6B_W6KmPoL_bWXeYiqQ

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Declaration of interest

The author is the producer of the *AnSo Anaesthesia Sonoanatomy* mobile application.

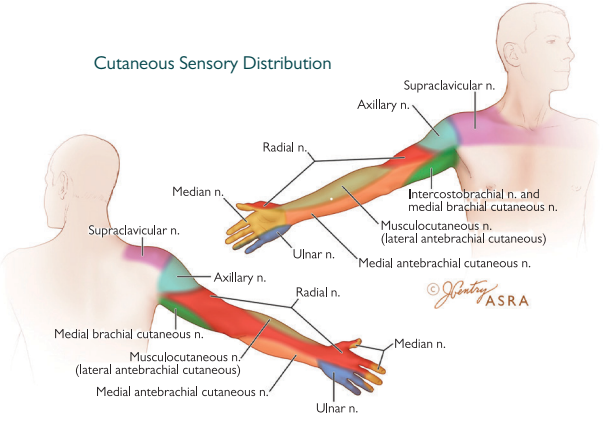

Fig. 40.32 Arm cutaneous sensory innervation. Copyright American Society of Regional Anesthesia and Pain Medicine. Used with permission. All rights reserved.

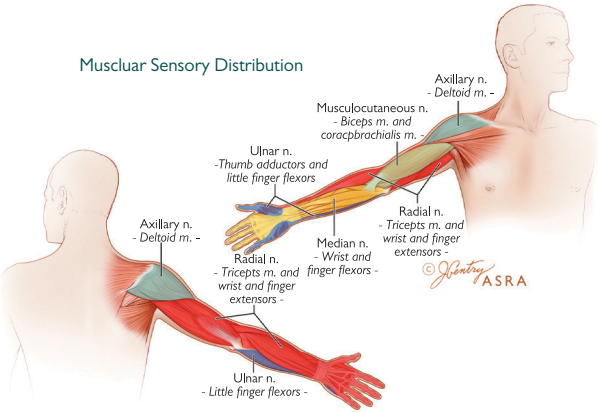

Fig. 40.33 Arm muscular sensory innervation. Copyright American Society of Regional Anesthesia and Pain Medicine. Used with permission. All rights reserved.

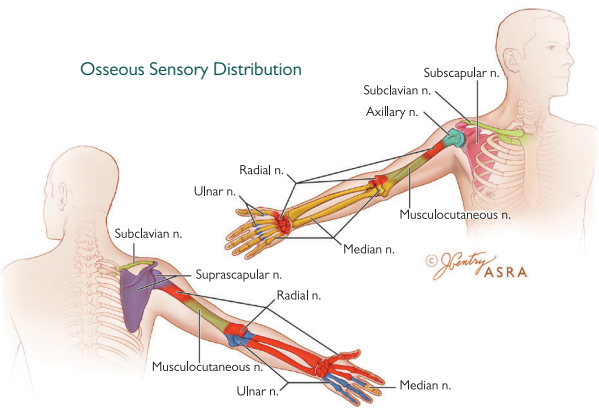

Fig. 40.34 Arm osseous sensory innervation. Copyright American Society of Regional Anesthesia and Pain Medicine. Used with permission. All rights reserved.

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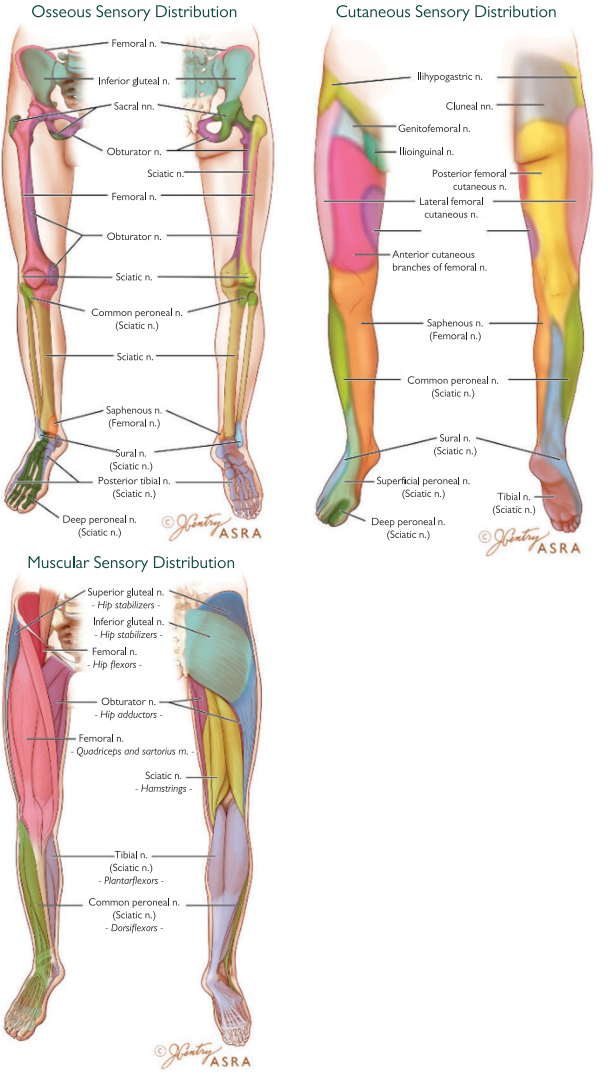

Fig. 40.35 Leg sensory innervation. Copyright American Society of Regional Anesthesia and Pain Medicine. Used with permission. All rights reserved.

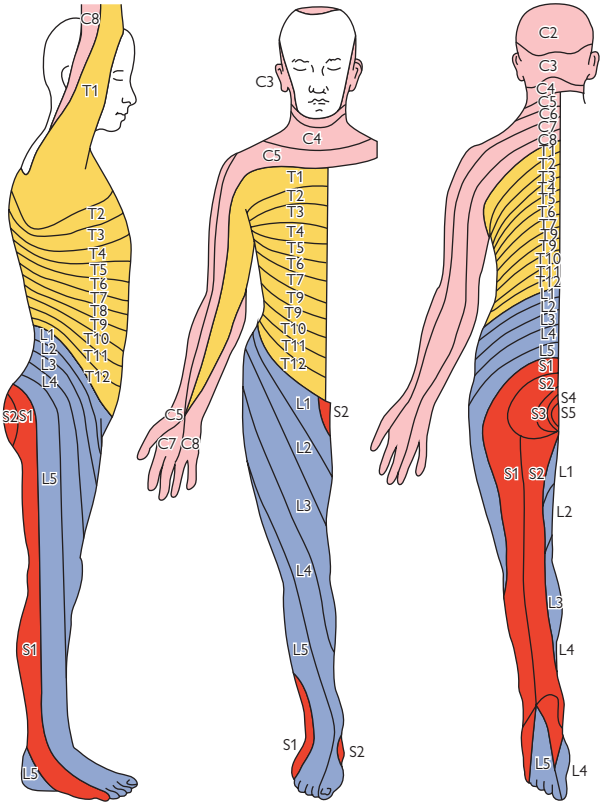

Fig. 40.36 Cutaneous dermatomes. Reproduced with permission of Oxford Publishing Limited through PLSclear from McLeod G, *et al*. (2012) *Principles and Practice of Regional Anaesthesia*, with permission from Oxford University Press. Copyright © Oxford University Press, 2012.

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Chapter 41

Acute pain

Adrian Dashfield

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Introduction

Problems with inadequate acute pain management

Kehlet[1] identified the aim of postoperative pain relief was firstly to provide subjective comfort, secondly to reduce the magnitude of the surgical stress response and thirdly: 'to enhance restoration of function by allowing the patient to breathe, cough and move easily'. Severe postoperative pain and the stress response to surgery cause ↑ morbidity and mortality.

- CVS: tachycardia, hypertension and ↑ peripheral vascular resistance cause ↑ myocardial O_2 consumption/demand and myocardial ischaemia. Altered regional blood flow (sympathetic stimulation), reduced mobility, venous stasis and ↑ clotting cause venous thrombosis.
- Respiratory system: abdominal/thoracic pain results in diaphragmatic splinting and weakened cough. Reduction in lung volumes, atelectasis and sputum retention cause chest infections and hypoxaemia.
- GI: delayed gastric emptying and reduced intestinal motility. This can be a direct effect of pain or as a side effect of opioids and surgery.
- Genitourinary: urinary retention.
- Metabolic/endocrine: release of vasopressin, aldosterone, renin, angiotensin, cortisol, glucagon, growth hormone and catecholamines and reduction in insulin and testosterone lead to ↑ protein breakdown, impairment of wound healing/immune function, Na^+ and water retention, ↑ fibrinogen and platelet activation and ↑ metabolic rate.
- Chronic pain: there is increasing evidence that patients who suffer from acute pain are more likely to develop chronic pain.
- Psychological: poor acute pain management can lead to patient anxiety, sleeplessness, fatigue and distress well into the postoperative period.
- Cancer survival: surgical stress produces an environment that favours tumour growth and metastasis.
- Despite these insights, there remains compelling evidence that pain after surgery is often poorly managed, and up to 40% of patients report severe pain that negatively impacts on their recovery.[2]

Measurement of pain

Verbal rating scales Stratify pain intensity according to commonly used adjectives such as 'mild', 'moderate' and 'severe'. They are widely applied and easy for patients to use. The semiquantitative nature makes them less suitable for research purposes.

Numerical rating scales Take the two extremes of the pain experience and have a numerical scale in between 'no pain' and 'worst imaginable', for example. These scales are robust, reproducible and easy for patients to understand. A disadvantage is that a digital scale reduces the capacity to detect subtle changes, as the digits act as anchoring points.

Visual analogue scales Similar to numerical rating scales, with two extremes of the pain experience on either end of the scale. The patient is asked to mark across a line of standard length (usually 100mm). The distance along this line is used. The continuous data generated make analysis easier than with verbal or numerical rating scales.

Currently, pain assessment tends to be linked to the delivery of analgesic drugs with the aim of reducing subjective pain scores. Routine use of pain scores that promote restoration of function has not been widely adopted and no scoring tools have been validated. An alternative, simplified 3-point functional activity score has been developed by Scott and McDonald[3] and is:

- A: no limitation of (relevant) activity because of pain
- B: mild limitation of activity because of pain
- C: unable to complete activity because of pain.

This simple score utilises functional ability to determine pain relief.

World Health Organization analgesic ladder

In 1986, WHO proposed the analgesic ladder (Fig. 41.1). It was intended to be logical, safe and applicable to many different types of pain, and consists of drugs which are easily available in most countries.

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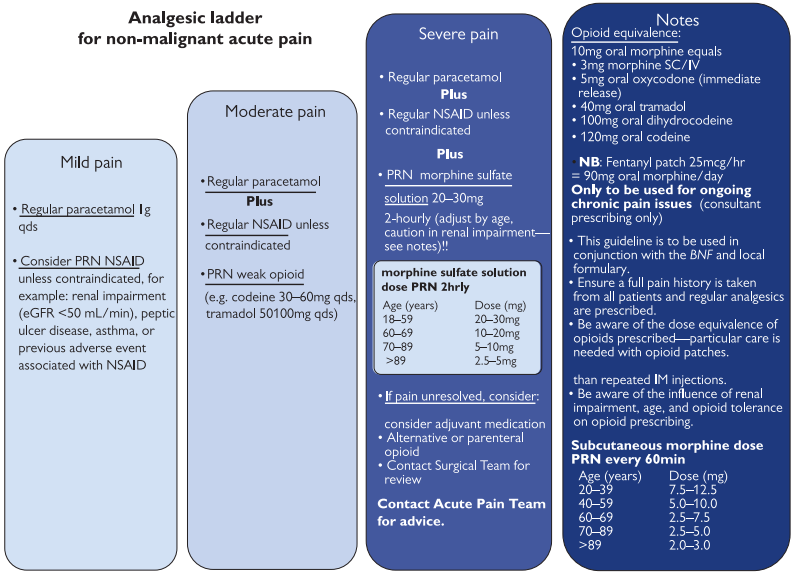

Fig. 41.1 The WHO analgesic ladder. Source: data from https://apps.who.int/iris/rest/bitstreams/1173681/retrieve

Analgesic drugs

Paracetamol

The action of paracetamol is via a number of central mechanisms, including effects on prostaglandin production and serotonergic, opioid, NO and cannabinoid pathways. Analgesic and antipyretic, without anti-inflammatory activity. Excreted renally after glucuronide and sulphate conjugation in the liver. A hepatotoxic metabolite *N*-acetyl-*p*-benzoquinoneimine is normally inactivated by conjugation with hepatic glutathione. In paracetamol overdose, this pathway is overwhelmed, leading to hepatic cell necrosis. Toxicity may occur in certain patients, even within the recommended dose range, because of altered metabolism.

- Usually given PO or PR, but available as an IV preparation. Particularly effective when administered IV.
- Recommended dose 4g/d in adults. Most effective when prescribed regularly, rather than PRN. The MHRA licensed dose of paracetamol is the same for all routes of administration in adults over 50kg. In July 2010, the MHRA issued a Drug Safety Update for dosing IV paracetamol in neonates, infants and children, following a number of cases of accidental overdose. The dose in children weighing ≤10kg is now 7.5mg/kg (>10kg: 15mg/kg).

Non-steroidal anti-inflammatory drugs

Analgesic, anti-inflammatory, antiplatelet and antipyretic actions are due to inhibition of the enzyme COX, and consequently the synthesis of prostaglandins, prostacyclins and thromboxane A_2 from arachidonic acid.

- Two types of COX: COX-1 is normally present in the kidney, GI mucosa and platelets where prostaglandin contributes to normal organ function. COX-2 is associated with inflammatory mediators following tissue damage. COX-2 inhibitors may be associated with fewer adverse effects than COX-1.
- NSAIDs have some central, as well as peripheral, activity. Absorption from the upper GI tract is rapid. Metabolised in the liver, excreted in the kidney.
- Opioid-sparing effect of between 20% and 40%. May be used as the sole analgesic for mild to moderate pain. Side effects with NSAIDs are relatively common.
- The VIGOR study,[4] in which patients on low-dose aspirin were excluded, found an ↑ risk of MI for patients given rofecoxib, compared to naproxen. Rofecoxib and some other COX-2 inhibitors have been withdrawn from clinical practice because of further concerns about the risks of CVS events, including MI and CVE. Celecoxib and etoricoxib remain available in the UK for the relief of pain in osteoarthritis, RA and ankylosing spondylitis. IV parecoxib remains a useful analgesic drug in the perioperative period.
- In January 2010, the MHRA assessed the ↑ thrombotic risk at three additional events per 1000 patient-years. The ↑ risk relates mainly to MI and includes CVE and peripheral events in some studies. For the majority of patients, the potential increase in the thrombotic risk is small. In patients with pre-existing risk factors for, or a history of, cerebrovascular disease, the risk may be higher (Table 41.1).

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Table 41.1 NSAID prescribing

Prescribe with caution in patients with	Contraindications to COX-2 inhibitors	Contraindications to NSAIDs
History of GI ulceration or high risk of adverse GI events	IHD	Active or recurrent (≥2) GI bleeding or ulceration
Cerebrovascular disease	Inflammatory bowel disease	History of NSAID-induced GI bleed or ulceration
Coagulation disorders	Peripheral arterial disease	Known hypersensitivity to NSAIDs, including aspirin
Hypertension	Cerebrovascular disease	Severe hepatic impairment (albumin <25g/L)
DM	CCF (NYHA II–IV)	Severe heart failure
Smokers		Severe renal impairment (eGFR <30mL/min/1.73m²)
Connective tissue disorders		
IHD		
Peripheral arterial disease		
Hepatic, renal and cardiac impairment		
Women trying to conceive		
Elderly		

NICE Guidelines 2019. https://cks.nice.org.uk/nsaids-prescribing-issues#!scenario

- The effect of conventional NSAIDs and coxibs on bone healing is unclear. After a fracture, COX-2 results in local release of prostaglandins as part of the acute inflammatory response, which plays a role in the induction of osteoblasts to promote bone healing. Ketorolac has been linked to higher non-union rates after spinal fusion surgery, but studies are often of poor quality and design. There is no robust scientific evidence to discard the use of NSAIDs or coxibs in patients suffering from a fracture, especially if prescribed for a short period of time, to treat acute pain.

Inhalational analgesia

Ideal for procedures of short duration such as dressing changes, removal of drains, labour and application of traction.

- **Entonox®** (50% N_2O, 50% O_2) is a quick-acting, potent analgesic of short duration which relies on self-administration. Side effects include drowsiness, nausea, excitability and augmentation of respiratory depressant drugs. Rapid diffusion increases volume of gas-containing cavities. Contraindications thus include pneumothorax, decompression sickness, intoxication, bowel obstruction, bullous emphysema and head injury.
- **Isonox®** (isoflurane 0.2–0.75% in Entonox®). Lower concentrations of isoflurane produce less drowsiness.[5]
- **Penthrox®** (methoxyflurane) is a volatile anaesthetic which is analgesic at low concentrations and supplied in a handheld, single-use inhaler device containing 3mL of liquid agent. Often used in prehospital care and EDs. Caution in renal impairment. Avoid daily use due to nephrotoxicity of fluoride ions.

Opioids

Opioid drugs act as agonists at opioid receptors, found mainly in the brain and spinal cord, but also peripherally. The opioid system comprises four types of receptor: mu-, delta- and kappa-opioid and nociceptin. Opioid receptors all have selective endogenous peptides. Analgesia elicited by clinically applied opioids act predominantly via the mu-opioid receptor. Tolerance to mu-opioid receptor analgesics may be attenuated by both nociceptin and delta-opioid receptor antagonism.

Morphine Remains the gold standard against which all new analgesics are compared. It is the least lipid-soluble opioid in common use. Metabolised in the liver, with only 10% excreted unchanged by the kidney. The metabolite morphine-6-glucuronide is more potent than morphine. The other main metabolite is morphine-3-glucuronide which has no analgesic activity. Both metabolites are excreted in the kidney. Accumulation can occur after prolonged use in patients with impaired renal function. Dose ranges and dose intervals vary according to the route of administration.

Diamorphine A prodrug (diacetylmorphine) rapidly hydrolysed to 6-monoacetylmorphine and then morphine. Diamorphine is much more lipid-soluble than morphine and thus has a more rapid onset of action than morphine when given by epidural or IV route.

Fentanyl Highly lipid-soluble synthetic opioid with a short duration of action because of rapid tissue uptake. The high lipid solubility makes it suitable for transdermal administration. Metabolites of fentanyl are inactive. Fentanyl is commonly administered IV, epidurally, intrathecally, buccally or via the nasal mucosa as a spray.

Pethidine Analgesic with anticholinergic and some LA activity. Primarily metabolised in the liver, with metabolites excreted in the kidney. One of the main metabolites is norpethidine with a half-life of 15–20h. Norpethidine is a potent analgesic. High blood concentrations can lead to CNS excitation. Patients with impaired renal function are at risk. Pethidine can be used to treat postoperative shivering associated with volatile anaesthetic agents and epidural and spinal anaesthesia.

Codeine A prodrug for morphine. Usually administered for the treatment of mild to moderate pain. About 10% of the dose is converted to morphine. Metabolism to morphine requires an enzyme (CYP2D6) which is part of the cytochrome P450 system; 8–10% of Caucasians lack this enzyme, obtaining little or no benefit. This variable metabolism results in an unpredictable analgesic efficacy, a high NNT and a concurrent risk of inadvertent overdose. Subsequently, codeine is not recommended for use in children, pregnancy or breastfeeding mothers.

Tramadol Synthetic, centrally acting opioid-like drug. Less than half of its analgesic activity is at the mu-opioid receptor. It inhibits noradrenaline and serotonin uptake at nerve terminals. Lower tolerance and abuse potential, less respiratory depression and constipation reported, compared to other opioids. Metabolised in the liver and excreted in the kidney. The main metabolite of tramadol is O-desmethyltramadol (M1) which is more potent. Formation of M1 also depends on the presence of CYP2D6 within the cytochrome P450 system.

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All opioids are equianalgesic if adjustments are made for the dose and route of administration. Allowance should be made for long-term opioid therapy, incomplete cross-tolerance between opioids, differing half-lives and interpatient variability (Table 41.2).

Opioids have a similar spectrum of side effects. There is considerable interpatient variability, and some patients may suffer from more side effects with one particular drug compared to another.

Side effects include respiratory depression (↓ RR and V_T and irregular respiratory rhythm), sedation, euphoria, dysphoria, nausea and vomiting, muscle rigidity, miosis, bradycardia, myocardial depression, vasodilation, delayed gastric emptying, constipation and pruritus.

Table 41.2 Equianalgesic dosages

Opioid	IM/IV (mg)	PO (mg)
Morphine	10	25
Diamorphine	5	–
Fentanyl	0.15–0.2	–
Pethidine	100	250
Codeine	–	175
Tramadol	100	100

Opioid antagonists Act at all opioid receptors. Naloxone is the most commonly used. By titrating the dose of naloxone administered, it is possible to reverse side effects such as respiratory depression, nausea and vomiting and sedation, without antagonising the analgesic effects. It must be remembered that naloxone is effective for about 60min.

Routes of administration

Oral

Oral bioavailability of most opioids is limited due to 1st-pass metabolism. The slower onset and longer duration of controlled-release formulations make rapid titration impossible. Immediate-release PO opioids (e.g. morphine syrup, oxycodone) are preferred for early management of acute pain. PO fentanyl should be restricted to treating breakthrough pain in patients receiving opioid therapy for chronic cancer pain.

Intermittent SC or IM opioids Traditional route of administration ordered 4-hourly PRN. A reluctance to give opioids more frequently than 4-hourly often leads to failure of regimens. Blood levels of an opioid need to reach a minimum effective analgesic concentration before any relief of pain is perceived. This requires an adequate initial dose. The only way to achieve good pain relief is to titrate the dose of opioid for each patient.

Intermittent IV opioid To achieve sustained pain relief without excessive drowsiness and respiratory depression, small doses of opioids should be given often. This technique of opioid administration is suitable for recovery wards, but not for routine maintenance of analgesia by untrained staff. Commonly used regimens are 1–3mg of morphine or 20–60 micrograms of fentanyl every 5min, until the patient is comfortable. Morphine can take up to 15min to exhibit its full effect.

Continuous IV infusion To avoid peaks and troughs in blood opioid concentrations associated with intermittent administration, continuous opioid infusions are sometimes used. Close observation and monitoring of the patient is essential. Patients are best made comfortable with IV boluses to 'load' the patient.

Intrathecal opioids Intrathecal opioids are administered at the same time as the intrathecal LA during spinal anaesthesia. Fentanyl 10–30 micrograms has a rapid onset (10–20min) and a short duration of action (4–6h). After a single administration, it can be used in day case arthroscopic surgery to enhance analgesia, without prolonging hospital stay. Diamorphine 0.3–0.4mg is used for analgesia after an elective CS. Doses of up to 1mg of diamorphine have been used. Intrathecal morphine 0.1–0.2mg has been shown to give good postoperative pain relief following hip arthroplasty; 0.3–0.5mg of morphine similarly provides good postoperative relief following knee arthroplasty.

Intranasal diamorphine Very effective in children (>1y) needing acute analgesia. A suitable dosing regime is 0.1mg/kg in 0.2mL of 0.9% sodium chloride (0.1mL to each nostril). To prepare the solution, add 10mg of diamorphine to 20/weight (kg) of 0.9% sodium chloride (mL), and draw up 0.2mL. Fentanyl nasal spray is available as a 50-microgram or 100-microgram metered spray. Use should be restricted to treating breakthrough pain in patients receiving opioid therapy for chronic cancer pain.

Transmucosal administration Fentanyl lollipops (PO transmucosal fentanyl citrate) allow absorption from the oral mucosa. More frequently used for anaesthetic premedication in children. Can be used for breakthrough analgesia in opioid-tolerant patients with cancer.

Transdermal administration Very lipid-soluble opioids are absorbed through the skin. Fentanyl patches are available in five sizes (12–100 micrograms/h), and patches are replaced every 72h. Buprenorphine patches are available as low-dose 7d-release patches or in higher-dose patches replaced every 72h. Steady plasma concentrations occur, on average, 12h after application of the transdermal patch. Dangerously high plasma concentrations can occur if patients are actively warmed while wearing a transdermal patch. Although not suitable for acute pain management, the recommended dose in chronic pain, based on the daily parenteral morphine dose, is shown in Tables 41.3 and 41.4.

The Oxford league table of analgesic efficacy is a helpful synthesis of all the available evidence about the relative efficacy of commonly used analgesics. An extract of the league table is provided in Table 41.5.

Acute opioid toxicity results in opioid-induced ventilatory impairment. Comorbidity and co-administration of other sedating drugs (e.g. gabapentinoids, benzodiazepines, other opioids) increases the risk of harm. In 2017, the UK MHRA issued a safety alert on the dangers of concomitant use of gabapentin with opioids and mandated that these patients should be carefully observed for signs of respiratory depression. As a result, sedation scoring should now be mandatory for all patients receiving opioids and the concomitant use of other sedative agents avoided where possible.

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Table 41.3 Transdermal fentanyl vs morphine

Transdermal fentanyl dose (micrograms/h)	PO morphine dose in 24h (mg)	Parenteral morphine dose in 24h (mg)
12	30	4–11
25	60	8–22
50	120	23–37
75	180	38–52
100	240	53–67
125	450	68–82
150	540	83–97

Table 41.4 Transdermal buprenorphine vs morphine

Transdermal buprenorphine dose (micrograms/h)	PO morphine dose in 24h (mg)	Parenteral morphine dose in 24h (mg)
5	12	1–3
10	24	2–5
20	48	4–9
35	84	6–15
52.5	126	8–22
70	168	14–22

There is universal acceptance that a global prescribed opioid crisis exists and it is timely for clinicians to reflect on their role in potentially facilitating this crisis. There is some evidence that reducing the duration of discharge medication prescriptions may reduce the incidence of subsequent opioid dependence.[6] Modified-release opioid preparations have been identified as one of the main causes of the prescribed opioid crisis and ideally should be avoided in the management of post-surgical pain in the opioid-naïve surgical patient. If an institution does decide to use modified-release preparations in perioperative pain management, they should be strictly dose- and time-limited and must not be included in discharge medication.

Table 41.5 The Oxford league table of analgesic efficacy

Analgesic	NNT	Lower CI	Higher CI
Diclofenac 100mg	1.8	1.6	2.1
Paracetamol 1000mg + codeine 60mg	2.2	1.7	2.9
Parecoxib 40mg (IV)	2.2	1.8	2.7
Diclofenac 50mg	2.7	2.4	3.1
Ibuprofen 600mg	1.7	1.4	2.3
Ibuprofen 400mg	2.5	2.4	2.7
Ketorolac 10mg	2.6	2.3	3.1
Paracetamol 650mg + tramadol 75mg	2.6	2.3	3.0
Ibuprofen 200mg	2.7	2.5	2.9
Diclofenac 25mg	2.6	2.2	3.3
Pethidine 100mg (IM)	2.9	2.3	3.9
Morphine 10mg (IM)	2.9	2.6	3.6
Parecoxib 20mg (IV)	3.0	2.3	4.1
Ketorolac 30mg (IM)	3.4	2.5	4.9
Paracetamol 500mg	3.5	2.2	13.3
Paracetamol 1000mg	3.8	3.4	4.4
Paracetamol 600/650mg + codeine 60mg	4.2	3.4	5.3
Aspirin 600/650mg	4.4	4.0	4.9
Tramadol 100mg	4.8	3.8	6.1
Tramadol 75mg	5.3	3.9	8.2
Paracetamol 300mg + codeine 30mg	5.7	4.0	9.8
Tramadol 50mg	8.3	6.0	13.0
Codeine 60mg	16.7	11.0	48.0

CI, confidence interval; NNT, number needed to treat.

Source: data from http://www.bandolier.org.uk/booth/painpag/Acutrev/Analgesics/lftab.htm

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Patient-controlled analgesia

PCA refers to self-administration of IV opioids and helps overcome the marked variability in response to postoperative opioids. Patients titrate their plasma opioid concentration to remain in the analgesic window (above the minimum effective analgesic concentration and below the minimum toxic concentration). The inherent safety of PCA lies in the fact that excessive doses of opioid will not be delivered, should the patient become sedated. No one but the patient is allowed to operate the PCA demand button.

Patient-controlled analgesia regimens

- The most commonly used opioid is morphine. Fentanyl, pethidine, tramadol and other opioids have also been used. No opioid is noticeably superior to any other, although a greater incidence of pruritus may be seen with morphine; on an individual basis, one opioid may be better tolerated than another.
- The optimal bolus dose consistently results in analgesia without side effects. Initial values for PCA variables are given in Table 41.6.
- For paediatric use of PCA, see pp. 926–7.

Table 41.6 PCA regimes

PCA variable	Drug and dose	Comments
Loading dose	0mg	Patients should be comfortable before starting PCA
Bolus dose	Morphine 1mg Pethidine 10mg Fentanyl 20 micrograms Diamorphine 0.5mg Tramadol 10mg	Patients over the age of 70y may require half this amount
Concentration	Varies, depending on pumps used and hospital protocols	Should be standardised in hospital protocols for each drug
Lockout interval	5min is usual	
Background infusion	0mg/h	If used, the background infusion rate (mg/h) usually should not exceed the bolus dose (mg)
Dose limit	30mg morphine or equivalent in 4h	No clear opinion on how this facility should be used. Often no dose limit is set

Complications

- Equipment malfunction is rare. Interference in pump operation has been reported following current surges and static electricity. Modern PCA pumps have a number of fail-safe design features where the program defaults to the lowest setting possible for a bolus dose. Most machines have a battery backup lasting up to 8h. Failure of antireflux valves has led to cases of respiratory depression.

- Operator error is much more common. Programming errors, use of the wrong drug or incorrect drug concentrations and incorrect background infusions have all been reported and have led to fatalities due to respiratory depression.

Troubleshooting

- Nausea and vomiting:
  - Add an antiemetic to the PCA (ondansetron 4mg, cyclizine 50–100mg, haloperidol 2mg)
  - Prescribe an antiemetic on a regular basis
  - Change the opioid.
- Breakthrough pain: add regular NSAID and paracetamol, if not contraindicated. Increase the bolus dose, or consider a background infusion if severe.
- Respiratory depression: this is caused by the direct action of opioids on the respiratory centre. All opioids, given in equianalgesic doses, have the same potential for respiratory depression. This is a relatively uncommon side effect, and if doses are properly titrated, the risk is small. The best early clinical indicator of respiratory depression is increasing sedation. Opioid doses are adjusted, so that the sedation score remains below 2 (Table 41.7). Respiratory depression (RR <8/min) is reversed with IV naloxone (100–400 micrograms).

Table 41.7 Sedation scores

Sedation score	
0	Patient wide awake
1	Mild drowsiness. Easy to rouse
2	Moderate drowsiness. Easy to rouse
3	Severe drowsiness. Difficult to rouse
4	Asleep but easy to rouse

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Epidural analgesia

Regional anaesthesia reduces acute pain and chronic pain after some surgical procedures and can reduce length of hospital stay.[7] Other benefits of epidural analgesia are:

- The incidence of postoperative atelectasis and pulmonary infection is reduced, improving oxygenation. Effective pain relief allows the patient to cough, breathe deeply and cooperate with physiotherapy. Epidural analgesia combined with GA reduces pulmonary complications in thoracic, abdominal and lower limb procedures, and is of proven benefit in patients with pulmonary disease.[8,9]
- The hypercoagulable response to surgery is attenuated, and fibrinolytic function is improved by attenuation of the stress response. This has been shown to be of benefit for graft survival in patients undergoing lower limb revascularisation.
- Epidural analgesia reduces pain and opioid consumption, and has been shown to reduce the duration of both ileus and time to 1st flatus. There has been concern that epidural analgesia increases the risk of anastomotic leakage perhaps caused either by gut hypoperfusion or by an increase in peristalsis 2° to associated sympathectomy, but this has not been consistently demonstrated.
- Reduction in surgical site infections.[9]
- There is, however, no survival benefit in high-risk patients, despite being beneficial in terms of pain relief and respiratory function.[10]

Contraindications

- Patient refusal, staff untrained in epidural care on wards and contraindications to catheter or needle placement (local or general sepsis, hypovolaemia, coagulation disorders, concurrent treatment with anticoagulant drugs and some central neurological diseases).

Troubleshooting

Breakthrough pain

Consider:

- Adding regular PO/PR/IV NSAID and paracetamol, if not contraindicated.
- Bolus dose (3–5mL), followed by ↑ infusion rate.
- Check all connections and insertion site.
- Check the block level (with ice or touch). If block patchy or unilateral, withdraw the catheter to 2cm in space.
- Bolus dose of opioid only (fentanyl 50–100 micrograms, diamorphine 2–3mg).
- Pruritus; give naloxone (50–100 micrograms), and consider adding 300 micrograms to infusion fluids or removing the opioid from the epidural infusion. Antihistamines may give some relief.
- Hypotension: check fluid status of the patient who is probably relatively hypovolaemic. Check block height. Consider reducing the infusion rate. If acute/severe, raise the legs; give fluid bolus and vasopressor.
- Motor block: reduce the infusion rate. Consider reducing LA concentration.
- Complications of epidural analgesia are summarised in Table 41.8 (see also ➲ p. 844).

Table 41.8 Complications of epidural analgesia

Complication	Incidence (%)	Management
Dural puncture	0.16–1.3	Bed rest, analgesia, hydration, blood patch (see ➔ pp. 848–51)
Headache (benign, not PDPH)	16–86	Bed rest, analgesia, hydration; suspect dural puncture
Nerve or spinal cord injury	0.016–0.56	Immediate neurological assessment
Catheter migration	0.15–0.18	Remove catheter, and resite if appropriate
Epidural abscess and haematoma	0.01–0.05 0.0004–0.03	MRI or CT scan. Immediate neurosurgical assessment. Antibiotics (see also ➔ p. 847)
Respiratory depression	0.13–0.4	Decrease in opioid concentration may be required
Hypotension	3–30	IV fluids ± vasopressors. Temporarily reduce or stop infusion
Pruritus	10	Naloxone IV (50–100 micrograms) ± antihistamine
Urinary retention	10–30 (in ♂)	Catheterisation
Motor block	3	Check for catheter migration. Temporarily cease infusion. Consider epidural haematoma (see ➔ p. 847)

Schug, S.A., Palmer, G.M., Scott, D.A., *et al. Acute pain management: scientific evidence*. 4th edn. Australian and New Zealand College of Anaesthetists and Faculty of Pain Medicine, Melbourne; 2015.

Drugs used for epidural analgesia

To minimise the side effects of each class of drug and provide optimal analgesia, a combination of an opioid and a low concentration of LA solution is given by continuous infusion. Commonly used mixtures are:

- Bupivacaine 0.125% with 5 micrograms/mL of fentanyl or 100–125 micrograms/mL of diamorphine
- Bupivacaine 0.1% with 5 micrograms/mL of fentanyl or 100 micrograms/mL of diamorphine
- Bupivacaine 0.0625% with 2 micrograms/mL of fentanyl or 50 micrograms/mL of diamorphine.

There is no universally accepted optimal combination of drugs. Infusion rates vary, according to the concentration, surgical site and dermatomal level of the epidural catheter placement. Usual infusion rates for the above solutions are 8–15mL/h for adult patients, and reduced rates of 4–8mL/h in patients over 70y of age. Some anaesthetists reduce or avoid epidural opioids in the very elderly and use LA solutions only.

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Intrathecal opioids

Opioids can be administered intrathecally, in combination with LA, during spinal anaesthesia. The opioid is delivered directly into the CSF, so avoiding distribution into epidural fat and blood vessels. Consequently, the doses used are much smaller, compared to epidural or parenteral routes (Table 41.9).

- The more lipid-soluble the drug, the more rapid the onset and the shorter the duration of action.
- Pethidine has LA, as well as opioid, properties. It can be used as the sole drug for spinal anaesthesia (requires higher doses).
- Delayed or late respiratory depression can occur with the less lipid-soluble drugs (particularly morphine). Increasing patient age, high doses of opioid administered intrathecally, concurrent use of sedatives and systemic opioids are associated with ↑ risk of respiratory depression.
- Diamorphine (if available) offers the best combination of duration of analgesia with fewest side effects.

Table 41.9 Comparison of intrathecal opioids

Opioid	Intrathecal dose	Onset (min)	Duration (h)	Epidural dose
Morphine (preservative-free)	0.1–0.2mg	15–30	8–24	2–3mg
Pethidine (preservative-free)	10–25mg	<5	1–2	10–50mg
Fentanyl	10–25 micrograms	<10	1–4	50–100 micrograms
Diamorphine	0.25–0.5mg	<10	10–20	2.5–5mg

Spinal infection

Extreme vigilance is needed for all patients who have had epidural analgesia because of the risk of spinal infection.[11,12] Only 13% of patients with an epidural abscess present with the classical triad of fever, backache and neurological signs and symptoms. Back pain is the initial symptom in 75% of cases. Fever occurs in 66% of cases. Only two out of three patients have leucocytosis. A raised ESR (>30mm) is a consistent finding. If there is suspicion of infection, a full infection screen and blood cultures are mandatory. The epidural catheter should always be removed immediately and sent to the laboratory for microbiological investigation. Ninety per cent of spinal infections are bacterial, mainly *Staphylococcus aureus*. MRI with gadolinium is the investigation of choice. The whole spine should be scanned early, before neurological signs and symptoms occur. Once muscle weakness is present, only 20% of patients regain full function, even after spinal surgery. Poor recovery is predicted by patient age (older patients do worse), extent of cord compression and duration of neurological symptoms (<36h has better prognosis). Mortality from an epidural abscess is 10%. Treatment is based on surgical or percutaneous abscess drainage and antibiotics. Steroids are contraindicated.

Continuous peripheral nerve blockade

There are many potential benefits of continuous PNB. Successful catheter placement relies on a high degree of skill in a practitioner who is already very familiar with single-shot PNB (see ➔ p. 1108).

Table 41.10 suggests typical bolus and infusion rates for PNB: 0.5% or 0.25% ropivacaine or levobupivacaine is commonly used for the initial bolus; 0.1–0.25% levobupivacaine or ropivacaine is used for the continuous infusion. Safe doses must be calculated on a per kg basis for every patient. Do not exceed 0.6mg/kg/h for either levobupivacaine or ropivacaine.

Absolute contraindications for this technique include: patient refusal, skin infection at or near the puncture site, systemic infection, pyrexia, bleeding diathesis (including systemic anticoagulation), peripheral neuropathy and compartment syndrome.

Table 41.10 Typical bolus and infusion rates for peripheral nerve blockade

Catheter site	Initial bolus (mL)	Basal rate (mL/h)	Patient-controlled bolus (mL)
Interscalene	25–35	3–5	3–5
Axillary	30	5–10	5
Femoral/fascia iliaca	30	4–6	5–10
Sciatic	15–20	2–4	2–4

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Abdominal wall blocks

Abdominal wall blocks are performed on the principle of high-volume LA deposition within a fascial plane[13] (see ➲ pp. 1128–34). Abdominal wall blocks avoid the risks and adverse effects of CNB and provide a relatively safe alternative to thoracic epidural anaesthesia. These blocks may therefore be warranted where epidural anaesthesia has not been possible either for technical reasons or due to patient wishes, where the risk of complications is ↑ (e.g. sepsis, coagulopathy or pre-existing neurological conditions) or in circumstances such as unexpected conversion to an open surgical procedure.

- The duration of abdominal wall blockade can also be prolonged by catheter placement, especially where enhanced recovery protocols are used.
- There are a number of ultrasound-guided techniques for abdominal wall blocks for intra-abdominal surgery: TAP, quadratus lumborum, erector spinae plane and transversalis fascia plane blocks.
- Infusion of LA by catheter into the posterior rectus sheath can provide effective postoperative analgesia and reduce opioid requirements. Rectus sheath catheter (RSC) insertion can be performed by the anaesthetist under ultrasound guidance or by the surgeon at laparotomy.
- RSC LA dosing regimens reported in the literature vary.[14] Manual bolus regimens include: 20mL of ropivacaine 0.2% or 20mL of bupivacaine 0.25% or 20mL of levobupivacaine 0.25% 6-hourly to each catheter. Pump-delivered LA bolus regimens via a Y-connector to both RSCs include: 40mL of ropivacaine 0.2% 4-hourly and 18mL of ropivacaine 0.5% 4-hourly. Continuous LA infusion of ropivacaine 0.2% at 8mL/h via an elastomeric pump connected to each RSC has been described.

Stimulation-produced analgesia: transcutaneous electrical nerve stimulation and acupuncture

Stimulation techniques activate the body's pain modulation systems. The gate control theory of pain by Melzack and Wall in 1965 provided a model to explain this phenomenon.

- Incoming noxious pain signals are reduced by presynaptic and postsynaptic inhibition in laminae 1–5 in the dorsal horn of the spinal cord. Modulatory input arrives via the descending pathways and lateral branches from myelinated afferent A-fibres. A-fibres arise in low-threshold mechanoreceptors activated by TENS, and in high-threshold mechanoreceptors activated by needles used in acupuncture techniques.
- TENS: A-fibres are recruited at 50–200Hz and respond to low-intensity stimulation, increasing levels of the inhibitory neurotransmitters dynorphin A and B in the dorsal horn. Pain relief occurs immediately but lasts only as long as stimulation continues.
- Acupuncture: A-fibres are recruited at 2–4Hz, responding to high-intensity stimulation by increasing inhibitory neurotransmitter met-enkephalin levels in the dorsal horn. Pain relief takes 20–30min but lasts hours or days.

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Non-opioid adjuvant analgesic drugs

The occurrence of persistent postsurgical pain following surgery is becoming increasingly recognised. Acute postoperative neuropathic pain does not usually occur in isolation; there will also be nociceptive pain as a result of tissue damage/inflammation.

- About 14% of patients presenting to the pain clinic attribute their pain to surgery, with the pain beginning acutely.
- Patients may complain of an unusual type of pain different from the usual postoperative nociceptive pain. Patients often describe the pain as burning or shooting in nature. Pain may extend beyond the territory of a single peripheral nerve.
- The pain is often poorly responsive to opioid analgesia, despite high doses being administered and may be:
  - **Allodynia**: pain following a normal innocuous stimulation
  - **Hyperalgesia**: pain disproportionate to a noxious stimulus
  - **Dysaesthesias**: spontaneous unpleasant abnormal sensations.
- The presence of a neurological deficit, such as brachial plexus avulsion or spinal cord injury, makes the presence of acute neuropathic pain more likely.
- Risk factors for persistent postsurgical pain include young adult age group, genetic predisposition, psychosocial factors, severity of perioperative pain, neuropathic pain, general chronic pain states and type of surgery.[15,16] These patients may benefit from an 'individualised' pain management plan.

Treatment

Currently, there is some evidence to support the use of the following interventions in preventing persistent postsurgical pain.[7]

Mechanisms of neuropathic pain involve CNS changes and ↑ peripheral nerve excitability. Drug therapy focuses on reducing neuronal hyperexcitability and reducing activity of the *N*-methyl-*D*-aspartate (NMDA) receptor in an attempt to reverse neuronal changes.

Ketamine NMDA receptor antagonist in the CNS and peripheral nervous system. NMDA receptor activation has a key role in the development of central sensitisation, wind-up and pain memory, resulting in chronic postsurgical pain (CPSP). Ketamine has been used as an adjuvant analgesic in a variety of settings, with a reported reduction of up to 25% in pain intensity and 30–50% in analgesic consumption up to 48h after surgery. The analgesic effect of ketamine is independent of the type of opioid used, timing of ketamine administration and dose of ketamine.[17] Major side effects are uncommon. Psychomimetic side effects are more common in patients undergoing awake procedures, compared to GA.[18]. At the present time, there is insufficient evidence to recommend ketamine as a routine perioperative analgesic. More concrete evidence is required to ascertain the role of ketamine in modulating the development of CPSP.

Gabapentinoids Gabapentin and pregabalin are increasingly used as adjuvants for perioperative analgesia. They act on presynaptic Ca^{2+} channels and inhibit neuronal Ca^{2+} influx. They may prevent central sensitisation and subsequent hyperalgesia and allodynia. Gabapentinoids have been used to contribute to better postoperative pain management, enhance opioid analgesia, prevent opioid tolerance, reduce the incidence of PONV, pruritus and urinary retention and prevent persistent postsurgical pain, as well as having useful anxiolytic and sleep-modulating properties. However, a recent meta-analysis has thrown doubt on the routine use of these drugs perioperatively.[19] They are not likely to be sufficiently effective if used as sole agents for the management of acute postoperative pain.[8,20] Common side effects are drowsiness, dizziness and gait disturbance.

Lidocaine Lidocaine has analgesic, anti-inflammatory and antihyperalgesic properties. Level 1 evidence from GI surgery demonstrates ↓ pain scores, opioid analgesic consumption and side effects.[21] IV lidocaine can be a useful acute pain adjunct to achieve enhanced recovery after surgery. Patients may show particular benefit when they have acute hyperalgesia and opioids are not effective in treating acute pain. IV lidocaine is contraindicated when other regional anaesthesia techniques are concurrently used, especially where boluses or large doses of any LA are used. Examples include epidural, plexus blocks and TAP blocks. IV lidocaine infusion can be administered 4–8h after the last epidural, regional catheter bolus or TAP block and is best initiated without giving a bolus dose first. Having appropriate monitoring and nursing policies in place is vital before considering a lidocaine infusion for acute pain. There is no clear consensus on the dose, but many studies used a bolus of 100mg or 1.5–2mg/kg at least 30min prior to incision, followed by an infusion of 1.5–2mg/kg/h intraoperatively and up to 24h postoperatively. No major complications were reported. IV lidocaine may prevent the development of CPSP by inhibition of NMDA receptors and polymorphonuclear leucocyte priming.

α2-adrenoceptor agonists This group of drugs are a useful adjuvant in perioperative care because of several extra-analgesic properties, such as sedation, anxiolysis, prevention of postoperative shivering and PONV, mitigation of the stress response, anaesthetic-sparing effect and supplementing neuraxial and peripheral nerve blocks. They act at supraspinal, spinal and peripheral sites, causing membrane hyperpolarisation and reduced Ca^{2+} conductance into cells. Clonidine and dexmedetomidine are the two commonly used drugs in this class. Dexmedetomidine is ~8 times more specific at the receptor, but analgesic efficacy seems comparable.[22] A recent review showed that systemic clonidine and dexmedetomidine were associated with a moderate decrease in pain intensity, opioid consumption and early postoperative nausea. Clonidine was associated with ↑ intraoperative and postoperative hypotension, and dexmedetomidine with ↑ incidence of bradycardia. The best dose, timing and route of administration required to produce maximum benefit and minimum harm are unknown. Currently, there is no evidence to suggest that perioperative use of α2-agonists has a preventive analgesic effect or reduces the incidence of CPSP.

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Post-amputation pain Pre-emptive regional analgesia (regional analgesia commenced before surgical incision with the aim of being more effective than the same treatment started after surgery) has conferred little benefit for preventing CPSP. In contrast, preventative regional analgesia has demonstrated some promising results (although data are limited). Perineural blockade provides excellent analgesia for surgical stump pain following limb amputation, and in doing so can attenuate peripheral and central sensitisation that may either prevent or at least minimise the impact of phantom pain. Many studies looking at the use of regional anaesthesia in the prevention of phantom limb pain discontinue neural blockade within 48h of surgery. Borghi *et al.*[23] demonstrated a very significant reduction in the incidence of phantom limb pain with the use of prolonged perineural blockade. They reported only a 2% incidence of phantom pain by continuing neural blockade for up to 80d after amputation. Prolonging perineural blockade to this extent is unlikely to be feasible in most hospitals, but enhancing existing practice is possible.

Continuous perineural blockade is commenced perioperatively with 400mL of ropivacaine 0.2% infused via an elastomeric pump at 10mL/h if a single catheter is used or 5mL/h per catheter if two infusions are required for above-knee amputations. Perineural blockade is continued for a minimum of 80h after amputation to get a patient beyond the crucial 1st days of maximal pain in order to minimise sensitisation. Local experience indicates this is probably the single most important technique for acute pain management after amputation and is crucial in decreasing the likelihood of developing significant phantom limb pain.

Persistent pain patients in acute pain

Basic principles

Patients with persistent pain presenting for surgery present an additional challenge to the anaesthetist striving for effective postoperative pain control. The concept of total pain is very useful. Pain is a combination of physical, spiritual, emotional and social injury, not simply tissue damage. Most of these patients have some form of psychiatric illness such as depression, anxiety, emotional dysregulation or personality or addictive disorder. These will impact on their perception of pain, and therefore recovery. Clearly it is not the role of the anaesthesthetist to address these issues, but an understanding of, and empathy with, them is hugely beneficial for the anaesthetist, the patient and the nursing staff.

Preoperative assessment

The underlying principle is to fully optimise the patient. Ideally this group of patients will have seen a pain physician and have a plan for acute pain, but this is often not the case. An acute pain plan for the patient with persistent pain is about mood, as well as drug management.

It is very useful to establish baseline pain levels. For all its faults, the numerical 0–10 scale is very useful, easy to use and well understood. (A mild–moderate–severe scale can also be used.) This is important because if the baseline pain is 7/10, there is little point aiming for a pain score of 3/10 after surgery. There is a move away from describing pain as the '5th vital sign' and from aiming to completely abolish pain. The current thinking is to focus on optimisation and function. Pain is 2° to function and although important, elimination of pain should not be the 1° endpoint. This concept is sometimes difficult to accept for patients, their relatives and those looking after them.

- Patients are often already on a number of medications with the potential for interactions. For simplicity, there are two major groups:
  - Respiratory depressants: opioids, benzodiazepines and gabapentinoids
  - Potential to cause serotonin syndrome: SSRIs, serotonin noradrenergic reuptake inhibitors (SNARIs), TCAs, lithium, 5-HT_3 inhibitors, ketamine, pethidine and, to a lesser degree, fentanyl. Tramadol is very potent in this regard. In this group of patients, it is not always possible to avoid co-prescribing these medications. Therefore, the concept of 'serotonin load' is useful; as the load increases, so does the risk.
- The psychosocial history is probably the most important aspect of care in this group but, with modern day-of-surgery admission, arguably the most difficult to address.

Drug management

- Use the simple analgesic ladder.
- Use regional nerve or plexus catheters where possible.
- Opioids (see ➲ pp. 1176–8 for long-term opioid use and the opioid-dependent patient). Consider PCA in this group.
- Ketamine.[24] There are a number of effective dosing regimes for either perioperative or postoperative use. A conservative and safe maximum is 0.1mg/kg/h. Infuse at this rate for 24–48h only. If the patient requires

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higher doses or a longer duration for pain control, then discuss with a pain specialist.

- Lidocaine infusions:[14] 1–2mg/kg/h or combined with ketamine, e.g. 25mg of ketamine added to 20mL of 2% lidocaine running at 0.1mL/kg/h.
- Antineuropathic medications. These include TCAs (amitriptyline 10mg nocte, NNT of 3–4) and gabapentinoids (e.g. gabapentin 300mg tds, NNT of roughly 7).
- Psychotropic drugs. This group can be particularly useful for management of distress. Haloperidol 2.5–5mg IV or PO is probably most familiar to anaesthetists, but the atypical antipsychotics quetiapine (12.5–25mg) or risperidone (0.5–1.0mg) are also helpful.
- Benzodiazepines are also useful to manage distress. Consider lorazepam (1–2mg), temazepam or diazepam.

Non-drug management

On occasion, despite best management, no relief from pain can be obtained. It is important to eliminate ongoing tissue damage and request a surgical review if necessary. Once this has been ruled out, then it is useful to bear in mind the concept of total pain and explore these avenues. This can be very rewarding if you are comfortable in this area, but equally it may be appropriate to ask for psychiatric consultation.

Predictability

Sometimes a patient in this group has less pain than predicted and follows a relatively normal clinical pathway, but most commonly pain is a major problem. As a simple rule of thumb, this group will need three times the analgesia for three times as long and be three times as sore, no matter what you do.

Distress management

Distress and anxiety compound pain are often manifest as out-of-control pain, even in those in whom you would not expect such a response. Reassurance or sometimes specialist pain or psychiatric input may be required. Refer to the flow chart in Fig. 41.2.

Plan and progress

It is important that all members of the treating team are consistent in their approach to analgesic management. A well-documented plan is key. It is acceptable to change the plan, but communicate with all members of the team and the patient. Sometimes patients can play on the lack of consistency and split the team. Remember to start discharge planning well in advance.

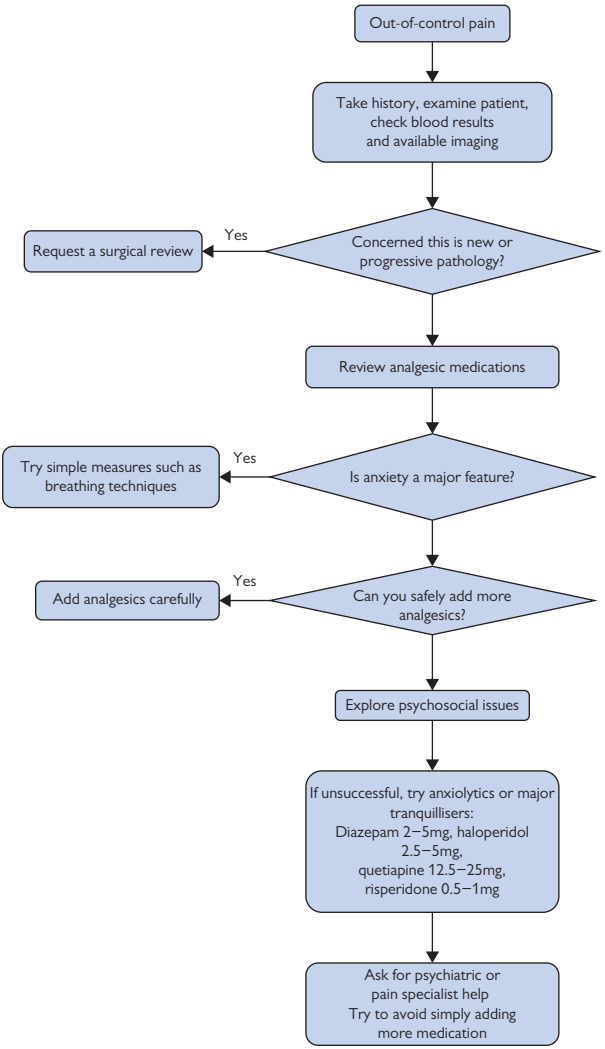

Fig. 41.2 Aide memoire for approaching persistent pain patients with acute perioperative pain.

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Long-term opioid use and the opioid-dependent patient

The number of patients who are receiving long-term opioid therapy has ↑ dramatically, and the provision of analgesia in the perioperative period can be challenging. There is evidence to suggest that two-thirds of patients receiving a dose of >120mg/d PO morphine equivalent, or those treated for a duration of >3mo, are likely to continue their medication for years. The aim is to bring acute pain under control.

- Opioid-dependent patients normally fall into one of three groups: opioid addicts, chronic non-cancer pain and cancer pain. The principles of management are the same for each group.
- Tolerance is a decrease in sensitivity to opioids, resulting in less effect from the same dose. Opioid-tolerant patients report higher pain scores and lower incidence of opioid-induced nausea and vomiting.
- Physical dependence is a physiological phenomenon characterised by a withdrawal reaction when the drug is withdrawn or an antagonist is administered.
- Addiction is a pattern of drug abuse characterised by compulsive use despite evidence of harm.
- Pseudoaddiction is an iatrogenic drug-seeking behaviour, normally due to undertreatment of acute pain by the physician, and perhaps 'undertreated patient' would be a more appropriate term for this group.
- Withdrawal: symptoms and signs of withdrawal include yawning, sweating, anxiety, rhinorrhoea, lacrimation, tachycardia, hypertension, diarrhoea, nausea, vomiting, abdominal pain and cramps. On average, these symptoms peak at 36–72h after the last dose. Aims of treatment are provision of analgesia, prevention of opioid withdrawal and management of abnormal drug-taking behaviour. Non-opioid analgesics, such as paracetamol and NSAIDs, should be prescribed regularly if possible. Opioid-tolerant patients are at risk of opioid withdrawal if non-opioid analgesic regimens or tramadol are used. PO or SC clonidine (50 micrograms 8-hourly) can be used to treat symptoms of opioid withdrawal.
- An objective assessment of function, e.g. the ability to cough, may be a better guide to opioid requirements than pain scores.
- Opioid requirements will, in general, be much higher than in non-opioid-dependent patients. The initial dose prescribed should take the patient's current opioid requirement into account. It may be difficult to judge current opioid use when illicit drugs have been taken. The GP, local pharmacist or drug rehabilitation centre may provide helpful information.
- Whenever possible, regional analgesic techniques should be used.
- PCA with larger-than-average bolus doses is the preferred means of administrating opioids.[25] PCA settings may need to include a background infusion to replace the usual opioid dose, and a higher bolus dose. Total dose should be ↑ until acceptable analgesia is achieved or until side effects prohibit any further dose increase. Opioid rotation may be of use, particularly with an agent of higher intrinsic opioid agonist activity.[7]

- The aim is to eventually discharge the patient on no more opioid than was used before admission. Normally, dose reductions of 20–25% every day towards the preadmission opioid intake will avoid symptoms of withdrawal.
- Patients in drug-free recovery may be concerned about the risk of relapse into active SAD if given opioids for acute pain management. Use of multimodal analgesic strategies, reassurance that the risk of reversion is small and information that ineffective analgesia can paradoxically lead to relapses in recovering patients help avoid undertreatment.

Opioid-induced hyperalgesia

Opioid-induced hyperalgesia (OIH) is a paradoxical phenomenon whereby an opioid can induce a pronociceptive state in the CNS. OIH was first observed in patients taking methadone for opioid addiction. The underlying mechanism is complex but likely to involve a combination of glial cell activation, NMDA receptor activation, glutaminergic activation and alterations in opioid intracellular signalling. As a result, the treatment of OIH necessitates an opioid dose reduction. This can be a difficult concept to understand for a patient who is in pain, as well as for their doctor. Nevertheless, it is often difficult to clinically distinguish between opioid tolerance, OIH and progression of the underlying disease process. Importantly, patients with OIH may report more diffuse and widespread pain and 'sensitivity' which does not respond to an increase in the dose of opioid. It was previously thought that OIH only occurred in patients on long-term opioids, but both tolerance and OIH have been associated with short-term use of high-potency opioids, e.g. remifentanil.

Converting from IV to PO opioid

Patients will need to be converted back to PO medication as their clinical situation allows. A typical strategy for returning to PO medication is to identify the IV opioid consumption in the previous 24h and convert this to an equivalent PO dose. Fifty per cent of this PO equivalent dose is then given in a sustained-release form, and one-sixth of the equivalent dose is prescribed as an immediate-release preparation every 4h.

Opioid maintenance programmes

Opioid maintenance programmes often use a single daily dose to prevent withdrawal and not to treat pain. Splitting the dose into two or three divided doses can increase the analgesic effect.

Methadone

A long-acting mu-agonist, NMDA antagonist and monoamine reuptake inhibitor. Patients who are receiving methadone maintenance therapy should be managed as any other opioid-tolerant patient. In the acute pain setting, methadone should be continued at the same dose. If the patient is unable to take methadone PO, substitution with parenteral methadone or other opioids may be required in the short term. Additional opioids should be used to manage acute pain if appropriate.

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Naltrexone

A long-acting competitive opioid antagonist with a duration of action of ~48–72h. Chronic use results in ↑ sensitivity to morphine-induced analgesia and a doubling of brain mu- and delta-opioid receptors, which may return to baseline in ~6d post-treatment. It is recommended to stop naltrexone 72h prior to surgery, and it is important to note that although patients may be resistant to opioids while taking naltrexone, they may then become extremely opioid-sensitive once stopping it. It is therefore imperative to maximise opioid-sparing strategies (e.g. regional anaesthesia) and consider managing the patient in a high dependency setting in the postoperative period, to ensure appropriate monitoring of treatment. Usual maintenance dose is 25–50mg daily.

Buprenorphine

This partial opioid agonist is used in the treatment of opioid addiction. Commonly prescribed doses are 8–32mg. The high receptor affinity and long half-life of buprenorphine result in difficulty obtaining effective analgesia by simply adding further opioid. At high doses of Suboxone® (buprenorphine and naloxone), e.g. 22–32mg, there is a theoretical concern that buprenorphine will antagonise the effect of a full agonist. There are very few reports describing perioperative pain management of patients administered high-dose buprenorphine. Strategies in this situation have included continuing the usual dose of buprenorphine and either (1) providing additional full opioid agonists or (2) prescribing supplemental doses of buprenorphine. An alternative strategy involves rotating to a full agonist before surgery such as methadone. The author recommends continuing the patient's usual dose of buprenorphine, maximising the use of opioid-sparing adjuncts, and using conventional full opioid agonists to treat acute pain. The doses of opioid required for analgesia may be appreciably higher than one would expect for other opioid-tolerant patients and in view of this, it may be advisable to monitor the patient in a high dependency environment.

Specific patient groups

Substance abuse disorder

A substance abuse disorder (SAD) exists when the extent and pattern of substance use interfere with the psychological and sociocultural integrity of the person. Patients with SAD may be abusing CNS-depressant drugs (alcohol, benzodiazepines, opioids) or CNS-stimulant drugs (cocaine, amphetamines, ecstasy, cannabinoids).

Effective management of acute pain in patients with SAD may be complicated by:

- Psychological and behavioural characteristics
- Presence of the drug of abuse and morbidities related to it
- Physical dependence (physiological phenomenon characterised by a withdrawal reaction when the drug is withdrawn or an antagonist is administered) and the risk of withdrawal
- Medications used to assist with drug withdrawal or rehabilitation
- Ethical dilemmas arising as a result of the need to balance concerns of undermedication against anxieties about safety and possible abuse or diversion of the drugs
- Management of pain in patients with SAD which should focus on prevention of withdrawal-effective analgesia and symptomatic treatment of affective and behavioural problems.

Personality disorders

Personality disorder is common among those with persistent pain and by definition, this is a difficult group to manage. There is no magic approach. Stick to basic principles and the outline above. Revisit the concept of total pain. Communication is the key—to the patient, to the team and to the GP.

Complex regional pain syndrome (CRPS)

There is evidence that those who have previously had CRPS are more likely to develop CRPS from a subsequent injury or surgery (up to 1000 times). There is no magic preventative approach. Appropriate consent is therefore very important. The evidence around use of high-dose vitamin C (500–1500mg/d for 50d) is controversial and of low quality as it is extrapolated from older ♀ wrist fractures.[26] Consider regional techniques and/or intraoperative ketamine, although this is empirical.

Fibromyalgia

This is a difficult disease to understand, but even more difficult to endure. This is the archetypal example of central sensitisation where non-noxious stimuli are interpreted as noxious. In a perioperative patient, sorting out what is noxious vs non-noxious is extraordinarily difficult. These patients are very sensitive to even minor stimuli and often have multiple drug sensitivities.

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Chapter 42

Drug formulary

Andrew Bradley

Drug formulary *1182*
Infusion regimes *1209*

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Drug formulary

(See Table 42.1.)

Table 42.1 Drug formulary

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Adenosine	Endogenous nucleoside with antiarrhythmic activity. Slows conduction through AV node. Treatment of acute paroxysmal SVT (including WPW) or differentiation of SVT from VT. Duration 10s	2nd- or 3rd-degree heart block. Long QT. Asthma/COPD. Reduce dose in heart transplant or dipyridamole treatment	Flushing, dyspnoea, headache, AV block, transient angina	1mo to 1y: 0.1mg/kg fast IV bolus, increasing by 0.05–0.1mg/kg every 1–2min to max 0.5mg/kg (max 12mg). >12y: as adult	6mg fast IV bolus, followed by 12mg at 1–2min, then further 12mg at 1–2min, as necessary. Reduce to quarter of dose if giving with dipyridamole

Adrenaline	Endogenous catecholamine with α and β action: 1. Treatment of anaphylaxis 2. Bronchodilator 3. Positive inotrope 4. Given by nebuliser for croup 5. Prolongation of LA action. 1:1000 contains 1mg/mL, 1:10 000 contains 100 micrograms/mL, 1:200 000 contains 5 micrograms/mL 6. Cardiac arrest	Arrhythmias, especially with halothane. Caution in elderly. Via central catheter whenever possible	Hypertension, tachycardia, anxiety, hyperglycaemia, arrhythmias. Reduces uterine blood flow	1. Refer to paediatric anaphylaxis emergency (see ➲ pp. 1081–3) 2. ETT 0.1mL/kg of 1:1000 (100 micrograms/kg) 3. Infusion 0.05–1 micrograms/kg/min 4. Nebuliser 0.5mL/kg (up to 5mL) of 1:1000 5. Maximum dose for infiltration 2 micrograms/kg 6. 10 micrograms/kg, refer to cardiac arrest (see ➲ pp. 1052–4)	1–3. IV/IM/ETT 1mL aliquots of 1:10 000 up to 5–10mL (0.5–1mg). Infusion 2–20 micrograms/min (0.04–0.4 micrograms/kg/min) 4. Nebulisation 5mL of 1:1000 (max 5mg) 5. Max dose for infiltration 2 micrograms/kg 6. 1mg (10mL of 1:10 000), every 3–5min
Alfentanil	Short-acting, potent opioid analgesic Duration 10min Sedation in ICU	Use IBW	Respiratory depression, bradycardia, hypotension. Prolonged half-life in neonates	Injection: 5–20 micrograms/kg, then 10 micrograms/kg boluses Infusion: 10–100 micrograms/kg over 10min, then 0.5–1 micrograms/kg/min	250–750 micrograms (5–10 micrograms/kg). Attenuation of CVS response to intubation: 10–20 micrograms/kg Sedation: infusion 2mg/h

(Continued)

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Amiodarone	Mainly class III antiarrhythmic, useful in treatment of supraventricular and ventricular arrhythmias	Via central catheter. Sinoatrial heart block, thyroid dysfunction, pregnancy, porphyria, iodine sensitivity. Dilute in glucose 5%, not 0.9% sodium chloride	Commonly causes thyroid dysfunction and reversible corneal deposits	>1y IV: 5mg/kg over 20–120min. Infusion: 5 micrograms/kg/min, max 1.2g/24h. 5mg/kg slow IV bolus for defib-resistant VF/VT	5mg/kg over 20–120min, followed by infusion if required, max 1.2g in 24h. 300mg slow IV bolus for defib-resistant VF/VT
Atenolol	Long-acting, cardioselective β-blocker	Asthma, heart failure, AV block, verapamil treatment	Bradycardia, hypotension, ↓ contractility	0.05mg/kg every 5min, max four doses	5–10mg over 10min
Atracurium	Benzylisoquinolinium NDMR. Undergoes Hofmann elimination plus non-specific enzymatic ester hydrolysis. Useful in severe renal or hepatic disease. Duration 20–35min	Potentiated by aminoglycosides, loop diuretics, Mg^{2+}, lithium, ↓ temp, ↓ K^+, ↓ pH, prior use of suxamethonium, volatile agents. Store at 2–8°C	Mild histamine release and rash common with higher doses. Flush with 0.9% sodium chloride before and after	Intubation: 0.3–0.6mg/kg Maintenance: 0.1–0.2mg/kg Infusion: 0.3–0.6mg/kg/h, monitor blockade	Use IBW Intubation: 0.3–0.6mg/kg Maintenance: 0.1–0.2mg/kg Infusion: 0.3–0.6mg/kg/h, monitor NMB
Atropine	Muscarinic acetylcholine antagonist. Vagal blockade at AV and sinus node increases HR (transient decrease at low doses due to weak agonist effect). Tertiary amine, therefore crosses blood–brain barrier	Obstructive uropathy and CVS disease. Glaucoma, myasthenia gravis	Decreases secretions and lower oesophageal sphincter tone, relaxes bronchial smooth muscle. Confusion in elderly	IV: 10–20 micrograms/kg. Control of muscarinic effects of neostigmine: 20 micrograms/kg. IM/SC: 10–30 micrograms/kg. PO: 40 micrograms/kg	300–600 micrograms. Prevention of muscarinic effects of neostigmine: 600–1200 micrograms

Bicarbonate (sodium)	Alkaline salt used for correction of acidosis and to enhance onset of action of LAs. 8.4%, 1000mmol/L. Dose (mmol) in acidosis: weight (kg) × base deficit × 0.3	Precipitation with calcium-containing solutions, ↑ CO_2 production, necrosis on extravasation. Via central catheter if possible	Alkalosis, hypokalaemia, hypernatraemia, hypocalcaemia	Dependent on degree of acidosis. 1mL/kg of 8.4% solution (1mmol/kg)	Dependent on degree of acidosis. Resuscitation: 50mL of 8.4%, then recheck blood gases. Bicarbonation of LA: 1mL of 8.4% to 20mL of bupivacaine. 1mL of 8.4% to 10mL of lidocaine/prilocaine
Bupivacaine	Amide-type LA used for infiltration and epidural and spinal anaesthesia. Slower onset than lidocaine. Duration 3–6h (slightly prolonged by adrenaline), pKa 8.1	Greater cardiotoxicity than other LAs. Do not use for IVRA. Adrenaline-containing solutions contain preservative and do not prolong action	Toxicity: tongue/circumoral numbness, restlessness, tinnitus, seizures, cardiac arrest	Infiltration/epidural: max dose dependent upon injection site; 2mg/kg/4h recommended	0.25–0.75% solution. Infiltration/epidural: max dose dependent upon injection site; 2mg/kg/4h (2mg/kg with adrenaline). 0.75% solution contraindicated in pregnancy
Buprenorphine	Opioid with both agonist and antagonist actions. Duration 6h	May precipitate withdrawal in opioid-dependent patients. Only partially reversed by naloxone	Nausea, respiratory depression, constipation	6mo to 12y: IV 3–6 micrograms/kg tds (max 9 micrograms/kg) 12–18y: IV 300–600 micrograms tds	Slow IV/IM: 300–600 micrograms qds. Sublingual: 200–400 micrograms qds
Calcium chloride	Electrolyte replacement, positive inotrope, hyperkalaemia, hypermagnesaemia. Calcium chloride 10% contains Ca^{2+} 680 micromoles/mL	Necrosis on extravasation. Incompatible with bicarbonate	Arrhythmias, hypertension, hypercalcaemia	0.1mL/kg of 10% solution, slow IV	2–10mL of 10% solution (10mg/kg, 0.07mmol/kg)

(Continued)

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Calcium gluconate	As calcium chloride. Calcium gluconate 10% contains Ca^{2+} 225 micromoles/mL	Less phlebitis than calcium chloride	As calcium chloride	0.3–0.5mL/kg of 10% solution (max 20mL)	6–15mL of 10% solution (30mg/kg, 0.07mmol/kg)
Carboprost	Synthetic prostaglandin F2α analogue used to treat severe postpartum haemorrhage due to uterine atony (after ergometrine and oxytocin failed)	Asthma, DM, epilepsy, jaundice, anaemia, glaucoma. Large doses may cause uterine rupture	Fever, bronchospasm. Nausea, vomiting, flushing. May cause CVS collapse		Never give IV. 250 micrograms deep IM or directly into myometrium. Repeat, if needed, after at least 15min. Max dose 2mg/24h
Chloral-hydrate	Formerly a popular hypnotic in children	Avoid prolonged use. Caution in elderly, gastritis and porphyria	Gastric irritation, ataxia	PO: 30–50mg/kg as single dose for sedation (up to 1g)	
Chlor-phenamine	Sedative antihistamine. Relief of allergy, urticaria, anaphylaxis (see ➔ pp. 1081–3)	Prostatic hypertrophy, urinary retention, glaucoma, porphyria	Drowsiness, dry mouth	PO 0.1mg/kg, up to 4mg qds	Slow IV/IM: 10mg qds. PO: 4mg qds
Cisatra-curium	Single isomer of atracurium with greater potency, longer duration of action and less histamine release. Duration 55min	As for atracurium	Enhanced effect in myasthenia gravis, effects antagonised by anticholinesterases. Monitor response with PNS	Intubation: (>1mo) 150 micrograms/kg. Maintenance (>2y) 30 micrograms/kg every 20min. Infusion: (>2y) 0.06–0.18mg/kg/h	Intubation: 150 micrograms/kg. Maintenance: 30 micrograms/kg every 20–30min. Infusion: 0.06–0.18mg/kg/h

Clonidine	Centrally acting α2-agonist. Reduces requirement for opioids and volatile anaesthetics. Enhances epidural analgesia	Rebound hypertension on acute withdrawal of chronic therapy	Hypotension, sedation	Over 6mo: 1–3 micrograms/kg slowly. PO premed: 4 micrograms/kg. Caudal: 1 microgram/kg	150–300 micrograms over 5min. Epidural: 75–150 micrograms in 10mL of 0.9% sodium chloride
Cyclizine	Antihistamine, antimuscarinic, antiemetic agent	Caution in severe heart failure	Drowsiness, dry mouth, blurred vision, tachycardia	IV/IM/PO: 1mg/kg, up to 50mg tds	IV/IM/PO: 50mg tds
Dantrolene	Direct-acting skeletal muscle relaxant used in treatment of MH and neuroleptic malignant syndrome. 20mg/vial. Reconstitute in 60mL of warm water, and give via blood giving set	Avoid combination with calcium channel blockers (verapamil), as may cause hyperkalaemia and CVS collapse. Crosses placenta	Skeletal muscle weakness (22%), phlebitis (10%)	1mg/kg, repeated every 5min to max of 10mg/kg	1mg/kg, repeated every 5min to max of 10mg/kg. Usually 2.5mg/kg
Desmo pressin	Synthetic analogue of vasopressin (ADH) with longer duration of action and reduced pressor effect. Used for neurogenic diabetes insipidus and haemophilia (enhances factor VIII activity)	Caution in hypertension and CVS disease	Hypertension, angina, abdominal pain, flushing, hyponatraemia	Diabetes insipidus: IV/IM/SC 0.5–2 micrograms/d (not per kg). Haemophilia: 0.3 micrograms/kg (in 50mL of 0.9% sodium chloride over 30min IV)	Diabetes insipidus: IV/IM/SC 0.5–2 micrograms/d (not per kg). Haemophilia: 0.3 micrograms/kg (in 50mL of 0.9% sodium chloride over 30min IV)

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Dexamethasone	Prednisolone derivative corticosteroid. Less Na^+ retention than hydrocortisone. Cerebral oedema, oedema prevention, antiemetic	Interacts with anticholinesterase agents to increase weakness in myasthenia gravis. Dexamethasone 0.75mg, prednisolone 5mg Different formulations available (dexamethasone sodium phosphate, dexamethasone phosphate, dexamethasone sodium phosphate). Dosing refers to dexamethasone base	See Prednisolone	IV/IM/SC: 83–333 micrograms/kg, 1–2 divided doses (max 20mg/d). Cerebral oedema: see *BNF for Children*. Croup: 150 micrograms/kg ± repeat at 12h. Antiemetic: 150 micrograms/kg (max 8mg)	IV/IM/SC: 3.3–6.6mg. Cerebral oedema: 8–16mg initially, then 5mg qds (use 3.8mg/mL preparation) Antiemetic: 3.3–6.6mg
Diamorphine	Potent opioid analgesic	Spinal/epidural use associated with risk of respiratory depression, pruritus, nausea	Histamine release, hypotension, bronchospasm, nausea, vomiting, pruritus, dysphoria	IV/SC: 20–100 micrograms/kg, then 15 micrograms/kg/h. Epidural: 2.5mg in 60mL of 0.125% bupivacaine at 0.1–0.4mL/kg/h. Intranasal: 100 micrograms/kg in 0.2mL of 0.9% sodium chloride	IV/IM/SC: 2.5–5mg 4-hourly. Epidural: 2.5mg diluted in 10mL of LA/0.9% sodium chloride, then 0.1–0.5mg/h. Spinal: 0.25–1mg* * Caution in patients >80y

Diazepam	Long-acting benzodiazepine. Sedation or termination of status epilepticus. Alcohol withdrawal	Thrombophlebitis: emulsion (Diazemuls®) less irritant to veins. Reduce in elderly	Sedation, circulatory depression	0.2–0.3mg/kg. PR: 0.5mg/kg as Stesolid®, or may use IV preparation	IV/IM/PO: 2–10mg, repeat if required (max tds)
Diclofenac sodium	Potent NSAID analgesic for mild to moderate pain	Hypersensitivity to aspirin, asthma, severe renal impairment, peptic ulceration, proctitis	GI upset or bleeding, bronchospasm, tinnitus, fluid retention, platelet inhibition, thrombotic events	>1y: PO/PR: 1mg/kg tds. Max 150mg/d. PR: NR <6mo	PO/PR: 25–50mg tds (or 100mg 18-hourly). Max 150mg/d
Digoxin	Cardiac glycoside. Weak inotrope and control of ventricular response in supraventricular arrhythmia. Therapeutic levels 0.8–2 micrograms/L (1.2–2.6nmol/L)	Reduce dose in elderly. Enhanced effect/toxicity in hypokalaemia. Avoid cardioversion in toxicity	Anorexia, nausea, fatigue, arrhythmias, blurred/yellow vision	Rapid IV/PO loading: 20–35 micrograms/kg stat	Rapid IV loading: 250–500 micrograms over 30min. Maximum 1mg/24h. PO loading: 1–1.5mg in divided doses over 24h. PO maintenance: 125–250 micrograms/d
Doxapram	Respiratory stimulant acting through carotid chemoreceptors and medulla. Duration 12min	Epilepsy, airway obstruction, acute asthma, severe CVS disease	Risk of arrhythmia. Hypertension	1mg/kg slowly. Infusion: 0.5–1mg/kg/h for 1h. NR <12y	1–1.5mg/kg over >30s. Infusion: 2–4mg/min
Droperidol	Butyrophenone related to haloperidol. Neuroleptic anaesthesia and potent antiemetic. Duration 4h	α-adrenergic blocker. PD	Vasodilation, hypotension. Dystonic reactions	Antiemetic: 25–50 micrograms/kg (max dose 1.25mg qds)	Antiemetic: 0.5–2.5mg

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Ephedrine	Direct and indirect sympathomimetic (α-and β-adrenergic action). Vasopressor, safe in pregnancy. Duration 10–60min	Caution in elderly, hypertension and CVS disease. Tachyphylaxis. Avoid with MAOI	Tachycardia, hypertension		3–6mg repeated (dilute 30mg in 10mL of 0.9% sodium chloride, 1mL increments). IM: 30mg
Ergometrine	Ergot alkaloid used to control uterine hypotony or bleeding. Syntometrine® = ergometrine 500 micrograms/mL and oxytocin 5 units/mL	Severe cardiac disease and hypertension	Vasoconstriction, hypertension, vomiting		IM: 1mL as Syntometrine®. Careful **slow** IV: 250–500 micrograms, with antiemetic cover recommended
Esmolol	Short-acting cardioselective β-blocker. Metabolised by red cell esterases. Treatment of SVT or intraoperative hypertension. Duration 10min	Asthma, heart failure, AV block, verapamil treatment	Hypotension, bradycardia. May prolong action of suxamethonium	SVT: 0.5mg/kg over 1min, then 50–200 micrograms/kg/min	SVT: 0.5mg/kg over 1min, then 50–200 micrograms/kg/min. Hypertension: 25–100mg, then 50–300 micrograms/kg/min
Fentanyl	Synthetic phenylpiperidine derivative opioid analgesic. High lipid solubility and cardiostability. Duration 30–60min	Reduce dose in elderly. Delayed respiratory depression and pruritus if epidural/spinal	Circulatory and ventilatory depression. High doses may produce muscle rigidity	1–5 micrograms/kg, up to 50 micrograms/kg if ventilating postoperatively. Infusion: 2–4 micrograms/kg/h	1–5 micrograms/kg (up to 50 micrograms/kg). Epidural: 50–100 micrograms (diluted in 10mL of 0.9% sodium chloride/LA) Spinal: 5–20 micrograms

Flumazenil	Benzodiazepine receptor antagonist. Duration 45–90min	Benzodiazepine dependence (acute withdrawal), resedation if long-acting benzodiazepine	Arrhythmia, seizures	10micrograms/kg (max 200 micrograms), repeat if required (max 50 micrograms/kg). Infusion: 2–10 micrograms/kg/h	200micrograms, then 100 micrograms at 60s intervals (up to max 1mg). Infusion: 100–400 micrograms/h
Furosemide (frusemide)	Loop diuretic used in treatment of hypertension, CCF, renal failure, fluid overload		Hypotension, tinnitus, ototoxicity, hypokalaemia, hyperglycaemia	0.5–1.5mg/kg bd	10–40mg slowly
Gabapentin	Structural analogue γ-aminobutyric acid. Indications: postherpetic neuralgia, neuropathic pain, focal seizures	Avoid abrupt withdrawal, elderly, renal impairment	Nausea, vomiting, abdominal pain, malaise	Seizures: d1 10mg/kg (max 300mg) od, then bd, then tds, max 70mg/kg	Pain: d1 300 mg od, d2 300mg bd, then 300mg tds to max 3.6g/d
Glucagon	Polypeptide hormone used in treatment of hypoglycaemia and overdose of β-blocker. Hyperglycaemic action lasts 10–30min. 1 unit = 1mg	Glucose must be administered as soon as possible. Phaeochromocytoma	Hypertension, hypotension, nausea, vomiting	<25kg: 0.5 units (0.5mg). >25kg: 1U (1mg)	SC/IM/IV: 1 unit (1mg). β-blocker overdose unresponsive to atropine: 2–10mg (max 10mg) in glucose 5%
Glucose	Treatment of hypoglycaemia in unconscious patient	50% solution irritant, therefore flush after administration into large vein. <20% peripherally		0.5mL/kg of 50% solution; use more dilute solutions: bolus 5mL/kg 10%, repeat PRN	25–50g (50–100mL of 50% solution). Can use more dilute solutions

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Glyceryl trinitrate	Organic nitrate vasodilator. Controlled hypotension, angina, CCF	Remove patches before defibrillation to avoid electrical arcing	Tachycardia, hypotension, headache, nausea, flushing, methaemoglobinaemia	10–30 micrograms/kg/h, starting dose up to 300 micrograms/kg/h. Max 600 micrograms/kg/h	Infusion: 0.5–10mg/h. Sublingual tabs: 0.3–1mg PRN. Sublingual spray: 400 micrograms PRN. Patch: 5–10mg/24h
Glyco-pyrronium bromide (glyco-pyrrolate)	Quaternary ammonium anticholinergic agent. Bradycardia, blockade of muscarinic effects of anticholinesterases, antisialogogue	Caution in glaucoma, CVS disease. Unlike atropine, does not cross blood–brain barrier	Paradoxical bradycardia in small doses. Reduces lower oesophageal sphincter tone	4–10 micrograms/kg	200–400 micrograms. Control of muscarinic effects of neostigmine: 200 micrograms for each 1mg neostigmine
Haloperidol	Butyrophenone derivative antipsychotic. Useful antiemetic	Neuroleptic malignant syndrome. Half dose in elderly	Extrapyramidal reactions	NR	IM/IV: 2–10mg 4- to 8-hourly (max 18mg/d). Antiemetic: 0.5–2mg IV. PO: 0.5–3mg
Heparin (unfraction-ated)	Endogenous mucopolysaccharide used for anticoagulation. Half-life 1–3h. 100 units, 1mg	Monitor APTT. Reversed with protamine	Haemorrhage, thrombocytopenia, hyperkalaemia	Low dose: IV 50–75 units/kg, then 10–15 units/kg/h. Full dose: IV 200 units/kg, then 15–30 units/kg/h. Anticoagulation for bypass: IV 300–400 units/kg	Low dose SC: 5000 units bd. Full dose IV: 5000 units, then 18 units/kg/h infusion. Anticoagulation for bypass: IV 300–400 units/kg

Human prothrombin complex (Beriplex®, Octaplex®)	Dried prothrombin complex, prepared from human plasma. Rapid reversal of warfarin anticoagulation		Risk of thrombotic events	Discuss with haematologist	Discuss with haematologist
Hydralazine	Direct-acting arteriolar vasodilator used to control arterial pressure. Duration 2–4h	Higher doses required in rapid acetylators. SLE	↑ HR, CO, stroke volume	0.1–0.5mg/kg 4- to 6-hourly	5mg every 5min to max of 20mg
Hydrocortisone (cortisol)	Endogenous steroid with anti-inflammatory and potent mineralocorticoid action (steroid of choice in replacement therapy, active form of cortisone). Treatment of allergy	Hydrocortisone 20mg, prednisolone 5mg	Hyperglycaemia, hypertension, psychiatric reactions, muscle weakness, fluid retention	4mg/kg, then 2–4mg/kg qds	IV/IM: 50–200mg qds. Adrenal suppression and surgery: 25mg at induction, then 25mg qds. PO: 10–30mg/d
Hyoscine butylbromide	Antimuscarinic agent used as an antispasmodic (racemic hyoscine)	See Atropine	See Atropine	2–6y: IV/IM 5mg. 6–12y: IV/IM 5–10mg. >12y: adult dose	IV/IM: 20mg slowly, repeated if necessary
Hyoscine hydrobromide	Antimuscarinic sedative, antiemetic agent used as premedication (L-isomer of hyoscine)	See Atropine. Avoid in elderly due to delirium	See Atropine	IM/SC: 15 micrograms/kg (max 600 micrograms)	IV/IM/SC: 200–600 micrograms. PO: 300 micrograms tds

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Insulin (soluble)	Human soluble pancreatic hormone facilitating intracellular transport of glucose and anabolism. DM, ketoacidosis and hyperkalaemia	Monitor blood glucose and serum K^+. Store at 2–8°C	Hypoglycaemia, hypokalaemia	Ketoacidosis: 0.1–0.2 units/kg (max 20 units), then 0.1 units/kg/h (max 5–10 units/h)	Ketoacidosis: 10–20 units, then 5–10 units/h. Sliding scale (see ➔ pp. 218–19). Hyperkalaemia (see ➔ pp. 240–1)
Intralipid®	20% emulsion used in treatment of severe LA toxicity	See ➔ pp. 1092–3 for LA toxicity guidelines		1.5mL/kg bolus, followed by 15mL/kg/h	1.5mL/kg bolus, followed by 15mL/kg/h
Ketamine	Phencyclidine derivative producing dissociative anaesthesia. Induction/maintenance of anaesthesia in high-risk and hypovolaemic patients	Emergence delirium reduced by benzodiazepines. Caution in hypertension. Control excess salivation with antimuscarinic agent	Bronchodilation. ↑, BP, uterine tone, salivation. Respiratory depression if given rapidly. Weak evidence for transient ↑ ICP, more likely lowers. Small ↑ in CBF. Safe in TBI (recent evidence)	Induction: IV 0.5–2mg/kg, IM 5–10mg/kg. Infusion: 10–45 micrograms/kg/min. Caudal: 0.5mg/kg (preservative-free only)	Induction: IV 1–2mg/kg, IM 5–10mg/kg. Infusion: 1–3mg/kg/h (analgesia only 0.25mg/kg/h)
Labetalol	Combined α- (mild) and β-adrenergic receptor antagonist. BP control without reflex tachycardia. Duration 2–4h	Asthma, heart failure, AV block, verapamil treatment	Hypotension, bradycardia, bronchospasm, liver damage	0.2mg/kg boluses up to 0.5mg/kg (max 20mg <12y). Infusion: 0.5–3mg/kg/h	5mg increments up to 100mg. Infusion: 20–160mg/h (in glucose)
Levobupivacaine	Levorotatory (S) enantiomer of bupivacaine with reduced cardiotoxicity	Use IBW See Bupivacaine)	See Bupivacaine)	See Bupivacaine. Max dose: 2mg/kg	See Bupivacaine). Max dose: 2mg/kg

Lidocaine	Amide-type LA: 1. Treatment of ventricular arrhythmias 2. Reduction of pressor response to intubation 3. LA, rapid onset, duration 30–90min (prolonged by adrenaline), pKa 7.7 4. Perioperative systemic analgesia allowing reduction of opioid requirement	Adrenaline-containing solutions contain preservative. Max dose dependent upon injection site: 3mg/kg/4h (6mg/kg with adrenaline)	Toxicity: tongue/circumoral numbness, restlessness, tinnitus, seizures, cardiac arrest. Prolongs action of neuromuscular blockers. Use IBW	1. Antiarrhythmic: 0.5–1mg/kg, then 10–50 micrograms/kg/min 2. Attenuation of pressor response: 1.5mg/kg 3. LA: 0.5–2% solution 4. Systemic analgesia: NR without expert guidance	1. Antiarrhythmic: 1mg/kg, then 1–4mg/min 2. Attenuation of pressor response: 1.5mg/kg 3. LA: 0.5–2% solution 4. Systemic analgesia: slow IV bolus dose of 1–1.5mg/kg over 2-4min (max. 20min), then infusion 0.5–2mg/kg/h (usual starting rate 1mg/kg/h)
Lorazepam	Benzodiazepine: 1. Sedation or premedication 2. Status epilepticus Duration 6–10h	↓ requirement for anaesthetic agents. Half in elderly	Respiratory depression in combination with opioids. Amnesia	Status 0.1mg/kg; max 4mg	1. PO: 1–4mg 1–2h preoperatively. IV/IM: 1.5–2.5mg 2. Status: 4mg IV, repeat after 10min if required

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Magnesium sulfate	Essential mineral used to treat: 1. Hypomagnesaemia 2. Arrhythmias 3. Eclamptic seizures 4. Severe asthma Magnesium sulfate 50%, 500mg/mL, 2mmol Mg^{2+}/mL. Normal plasma level Mg^{2+} 0.75–1.05mmol/L. Therapeutic level 2–4mmol/L	Potentiates muscle relaxants. Monitoring of serum level essential during treatment. Myasthenia and muscular dystrophy. Heart block. Magnesium sulfate 1g = Mg^{2+} 4mmol	CNS depression, hypotension, muscle weakness	1. Hypomagnesaemia: 0.2–0.4mmol/kg (max 20mmol/d). Check levels 2. Arrhythmias: 25–50mg/kg over 10min (max dose 2g), repeat once if necessary	1. Hypomagnesaemia: 0.5–1mmol/kg (max 160mmol/5d), check levels 2. Arrhythmias/asthma: 2g (8mmol) over 10min, repeat once if necessary 3. Eclampsia: 4g (16mmol) over 10min, then 1g/h for 24h (see pp. 872–6)
Mannitol	Osmotic diuretic used for renal protection and reduction of ICP. 20% solution, 20g/100mL	Extracellular volume expansion, caution in severe renal and CVS disease	Diuresis, AKI, hypertonicity	0.25–1.5g/kg	0.25–2g/kg (typically 0.5g/kg of 20% solution)
Metaraminol	Potent direct-/indirect-acting α-adrenergic sympathomimetic. Treatment of hypotension. Duration 20–60min	MAOIs, pregnancy. Caution in elderly and hypertensives. Extravasation can cause necrosis	Hypertension, reflex bradycardia, arrhythmias, ↓ renal and placental perfusion	10micrograms/kg, then 0.1–1 micrograms/kg/min, >12y	0.5–2mg. Dilute 10mg in 20mL of 0.9% sodium chloride, and give 0.5–1mL increments (increase dilution in elderly)

Methyl-thioninium chloride (methylene blue)	1. Treatment of methaemoglobinaemia 2. Ureteric identification during surgery (renally excreted) 3. Identification of parathyroid glands during surgery 4. Identification of sentinel node during cancer surgery	G6PD deficiency. Blue coloration causes acute changes in SpO_2 readings	Tachycardia, nausea, stains skin, allergy reported	1mg/kg slow IV (max 7mg/kg)	1mg/kg slow IV (max 7mg/kg)
Meto-clopramide	Dopaminergic antiemetic which increases gastric emptying and lower oesophageal sphincter tone	Hypertension in phaeochromocytoma. Inhibits plasma cholinesterase. Increases IOP	Extrapyramidal/ dystonic reactions (treat with benztropine or procyclidine)	PO/IM/IV: 0.15mg/kg, up to 5mg tds (>60kg, up to 10mg tds)	PO/IM/IV: 10mg tds
Metoprolol	Cardioselective β-blocker	Asthma, heart failure, AV block, verapamil treatment	Causes bradycardia, hypotension and ↓ cardiac contractility	0.1mg/kg up to 5mg over 10min	1–5mg over 10min, repeat if required (max 15mg)
Mivacurium	Short-acting NDMR. Metabolised by plasma cholinesterase. Duration 6–16min (often variable). Enhanced duration if low plasma cholinesterase. Antagonised by neostigmine, but avoid giving too early to avoid inhibiting drug metabolism	See Cisatracurium. Avoid in asthma	See Cisatracurium. Some histamine release	Intubation: 0.15–0.2mg/kg. Maintenance: 0.1mg/kg. Infusion: 8–10 micrograms/kg/min	Intubation: 0.07–0.25mg/kg (doses of 0.07, 0.15, 0.2 and 0.25mg/kg produce block for 13, 16, 20 and 23min, respectively). Maintenance: 0.1mg/kg. Infusion: 0.4–0.6mg/kg/h

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Morphine	Opioid analgesic. Half-life 2–4h	Prolonged risk of respiratory depression, pruritus, nausea when used via spinal/epidural	Histamine release, hypotension, bronchospasm, nausea, vomiting, pruritus, dysphoria	PO: 0.05–0.3mg/kg 4-hourly. IV boluses: 50–100 micrograms/kg. For PCA, NCA and infusion, see p. 926	IV: 2.5–10mg. IM/SC: 5–10mg 4-hourly. PO: 10–30mg 4-hourly. PCA: 1mg/5min lockout. Infusion: 1–3.5mg/h. Epidural: 2–5mg preservative-free. Spinal: 0.1–1mg preservative-free
Naloxone	Pure opioid antagonist. Can be used in low doses to reverse pruritus associated with epidural opioids and as depot IM injection in newborn of mothers given opioids	Beware renarcotisation if reversing long-acting opioid. Caution in opioid-dependent patients, may precipitate acute withdrawal. Duration of action 30min	Common: Arrhythmias, dizziness, headache, hypertension, hypotension, nausea, vomiting	5–10 micrograms/kg. Infusion: 5–20 micrograms/kg/h. IM depot in newborn: 200 micrograms. Pruritus: 0.5 micrograms/kg	200–400 micrograms, titrated to desired effect. Treatment of opioid/epidural pruritus: infusion rate 0.25–1 micrograms/kg/h ± bolus of 40–100 micrograms
Neostigmine	Anticholinesterase used for: 1. Reversal of NDMR 2. Treatment of myasthenia gravis Duration 60min IV (2–4h PO)	Administer with antimuscarinic agent	Bradycardia, nausea, excessive salivation (muscarinic effects)	50micrograms/kg with atropine 20 micrograms/kg or glycopyrronium 10 micrograms/kg	1. 50–70 micrograms/kg (max 5mg) with atropine 10–20 micrograms/kg or glycopyrronium 10–15 micrograms/kg 2. PO: 15–30mg at suitable intervals

Neostigmine and glyco-pyrronium	Combination of neostigmine metilsulfate (2.5mg) and glycopyrronium (500 micrograms) per 1mL	See Neostigmine	See Neostigmine	0.02mL/kg (dilute 1mL with 4mL of 0.9% sodium chloride, give 0.1mL/kg). Max 2mL	1–2mL over 30s
Omeprazole	PPI. Reduction in gastric acid secretion	Liver disease, max 20mg od	Headache, diarrhoea, prolonged QT	PO: 0.7–1.4mg/kg up to 40mg od. IV: 0.5mg/kg/d	PO/slow IV: 20–40mg/d. Premedication PO: 40mg. Bleeding peptic ulcer: 80mg bolus, then 8mg/h for 3d
Octreotide	Somatostatin analogue used in treatment of carcinoid, acromegaly and variceal bleeding (unlicensed use)	Pituitary tumour expansion, reduced need for antidiabetic treatments	GI disturbance, gallstones, hyper- and hypoglycaemia	SC: 1–5 micrograms/kg 6- to 8-hourly	SC: 50 micrograms od/bd, ↑ up to 200 micrograms tds. IV: 50 micrograms diluted in 0.9% sodium chloride (ECG monitoring)
Ondanse-tron	Serotonin (5-HT_3) receptor antagonist antiemetic	QT interval prolongation	Hypotension, headache, flushing	>1y: slow IV 100 micrograms/kg (max 4mg) qds	Slow IV/IM/PO: 4–8mg tds
Oxycodone	Opioid used for moderate pain, often in palliative care. IV preparation available: dose 1–10mg 4-hourly	Porphyria, acute abdomen	Nausea, vomiting, dysphoria, drowsiness	PO: Oxynorm® >1mo: initially 200 micrograms/kg (max 5mg) 4- to 6-hourly. >12y: adult doses	PO: Oxynorm® 5mg 4- to 6-hourly, ↑ as required. Oxycontin® 10mg bd, ↑ as required

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Oxytocin	Nonapeptide hormone which stimulates uterine contraction. Induction of labour and prevention of postpartum haemorrhage	Avoid rapid administration. Fetal distress	Vasodilation, hypotension, flushing, tachycardia		Postpartum slow IV: 5 units, followed, if required, by infusion 10 units/h (40 units in 40mL of 0.9% sodium chloride)
Pancuro-nium	Long-acting aminosteroid NMDR. Little histamine release. Duration 45–65min	See Cisatracurium	See Cisatracurium. ↑ HR and BP due to vagolysis and sympathetic stimulation	Intubation: 0.1mg/kg. Maintenance: 0.02mg/kg, PRN	Intubation: 0.1mg/kg. Maintenance: 0.02mg/kg, PRN
Pantoprazole	PPI used to inhibit gastric acid secretion	Liver disease, pregnancy. Renal disease	Headache, pruritus, bronchospasm	NR	PO/slow IV: 40mg od (max 80mg)
Paracetamol	Mild to moderate analgesic and antipyretic	Neonates: PO 10–15mg/kg 6-hourly (5mg/kg if jaundiced). Max 60mg/kg/d. <10kg: IV 7.5mg/kg 6-hourly. Max 30mg/kg/d	Liver damage in overdose	Slow IV: 15mg/kg qds (max 60mg/kg/d) (10–50kg, max 60mg/kg; >50kg, max 4g/d). PO/PR: 20mg/kg for 1st dose, then 10–15mg/kg qds (max 75mg/kg/d, up to 4g/d). PR loading dose: 30–40mg/kg (>44w post-conception)	Slow IV: >50kg, 1g qds; <50kg, 15mg/kg qds. PO: 0.5–1g qds

Paraldehyde	Status epilepticus	Dilute neat solution with equal volume of olive oil before PR administration	Rash	Deep IM: 0.2mL/kg. PR: 0.3mL/kg	Deep IM: 5–10mL. PR: 10–20mL
Parecoxib	Prodrug of valdecoxib. COX-2 inhibitor. Licensed for acute pain	Severe renal impairment, peptic ulceration, IHD and inflammatory bowel disease. Hypersensitivity to sulphonamides and aspirin. Reconstitute with 0.9% sodium chloride	GI upset, thrombotic events	Not evaluated for use in <18s	IV/IM: 40mg, then 20–40mg 6- to 12-hourly (max 80mg/d)
Pethidine	Synthetic opioid: 1. Analgesia 2. Postoperative shivering	Seizures possible in high dosage: max daily dose 1g/d (20mg/kg/d). MAOI	Respiratory depression, hypotension, dysphoria	>12y: IV/IM/SC: 0.5–1 mg/kg (max 100mg). Infusion: 5mg/kg in 50mL of 5% glucose at 1–3mL/h (100–300 micrograms/kg/h)	IM/SC: 25–100mg 3-hourly. IV: 25–50mg. PCA: 10mg/5min lockout. Shivering: 10–25mg
Phentol-amine	α1- and α2-adrenergic antagonist. Peripheral vasodilation and controlled hypotension. Treatment of extravasation. Duration 10min	Treat excessive hypotension with noradrenaline or methoxamine (not adrenaline/ephedrine due to β effects)	Hypotension, tachycardia, flushing	0.1mg/kg, then 5–50 micrograms/kg/min	2–5mg (10mg in 10mL of 0.9% sodium chloride, 1mL aliquots)

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Phenyl-ephrine	Selective direct-acting α-adrenergic agonist. Peripheral vasoconstriction and treatment of hypotension. Duration 20min	Caution in elderly and CVS disease. Hyperthyroidism	Reflex bradycardia, arrhythmias	2–10 micrograms/kg, then 0.1–0.5 micrograms/kg/min	20–100 microgram increments (10mg in 500mL of 0.9% sodium chloride, 1mL aliquots). IM: 2–5mg. Infusion: 30–60 micrograms/min (5mg in 50mL of 0.9% sodium chloride at 0–30mL/h)
Phenytoin	Anticonvulsant and treatment of digoxin toxicity. Serum levels 10–20mg/L (40–80 micromoles/L)	Avoid in AV heart block, pregnancy and porphyria. Monitor ECG/BP on IV administration	Hypotension, AV conduction defects, ataxia. Enzyme induction	IV loading dose: 20mg/kg over 1h	20mg/kg (max 2g) over 1h (dilute to 10mg/mL in 0.9% sodium chloride), then 100mg tds. Arrhythmia: 3.5–5mg/kg (rate <50mg/min)
Potassium chloride	Electrolyte replacement (see ➲ pp. 238–9)	Dilute solution before administration	Rapid infusion can cause cardiac arrest. High concentration causes phlebitis	0.5mmol/kg over 1h. Maintenance: 1–2mmol/kg/d	10–20mmol/h (max concentration 40mmol/L peripherally). With ECG monitoring: up to 20–40mmol/h via central line (max 200mmol/d)

Pregabalin	Binds to voltage-dependent calcium channels and decreases release of neurotransmitters. Adjunct for focal seizures	Avoid abrupt withdrawal, severe CCF, renal impairment	Dry mouth, constipation, oedema, dizziness		Pain: 150mg 2–3 divided doses with slow increase. Epilepsy: 25mg bd increasing
Prednisolone	Orally active corticosteroid. Less mineralocorticoid action than hydrocortisone	Adrenal suppression, severe systemic infections	Dyspepsia and ulceration, osteoporosis, myopathy, psychosis, impaired healing, DM	PO: 1–2mg/kg od. Croup: 4mg/kg, then 1mg/kg tds	PO: initially 20–60mg od, reduced to 2.5–15mg od for maintenance
Prilocaine	Amide-type LA. Less toxic than lidocaine. Used for infiltration and IVRA. Rapid onset. Duration 30–90min (prolonged by adrenaline), pKa 7.9	Adrenaline-containing solutions contain preservative. Significant methaemoglobinaemia if dose >600mg. Use IBW	Toxicity: tongue/ circumoral numbness, restlessness, tinnitus, seizures, cardiac arrest	NR <6mo	LA: 0.5–2% solution. 6mg/kg/4h (max dose dependent upon injection site); 9mg/kg with adrenaline
Prochlorperazine	Phenothiazine antiemetic	Hypotension on rapid IV administration. Neuroleptic malignant syndrome	Tardive dyskinesia and extrapyramidal symptoms	>10kg: PO: 0.25mg/kg tds. IM: 0.1–0.2mg/kg tds	IM: 12.5mg tds. PO: 20mg, then 5–10mg tds
Procyclidine	Antimuscarinic used in acute treatment of drug-induced dystonic reactions (except tardive dyskinesia)	Glaucoma, GI obstruction. Lower dose in elderly	Urinary retention, dry mouth, blurred vision	<2y: 0.5–2mg. 2–10y: 2–5mg. >10y: adult dose	IV/IM: 5–10mg, repeat after 20min if needed

(Continued)

Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Propofol	IV induction agent. Rapid recovery and little nausea. 1° drug in TIVA	Reduce dose in elderly or if haemodynamically unstable. Caution in severe allergy to peanuts, soya and soybean oil	Apnoea, hypotension, pain on injection. Myoclonic spasms, rarely convulsions	Induction: 2–4mg/kg. Infusion: 4–15mg/kg/h. NR induction <1mo. NR maintenance <3y	Induction: 2–3mg/kg. Infusion: 6–10mg/kg/h. TCI: initially 4–8 micrograms/mL, then 3–6 micrograms/mL (reduce in elderly)
Propranolol	Non-selective β-adrenergic antagonist. Controlled hypotension, symptomatic treatment of thyrotoxicosis	Asthma, heart failure, AV block, verapamil treatment	Bradycardia, hypotension, AV block, bronchospasm	0.1mg/kg over 5min	1mg increments, up to 5–10mg
Protamine	Basic protein produced from salmon sperm. Heparin antagonist	Weakly anticoagulant and marked histamine release. Risk of allergy	Severe hypotension, pulmonary hypertension, bronchospasm, flushing	Slow IV: 1mg per 1mg heparin (100 units) to be reversed	Slow IV: 1mg per 1mg heparin (100 units) to be reversed
Ranitidine	Histamine (H_2) receptor antagonist. Reduction in gastric acid secretion	Porphyria	Tachycardia	IV: 1mg/kg slowly tds (max 50mg). PO: 2–4mg/kg bd	IV: 50mg (diluted in 20mL of 0.9% sodium chloride, given over 2min) qds. IM: 50mg qds. PO: 150mg bd or 300mg od

Rocuronium	Rapidly acting aminosteroid NDMR. RSI (avoiding suxamethonium). Duration 10–40min (variable). Intubating conditions within 1min	See Cisatracurium	Mild tachycardia. See Cisatracurium	Intubation: 0.6–1mg/kg. Maintenance: 0.1–0.15 mg/kg. Infusion: 0.3–0.6mg/kg/h	Intubation: 0.6–1mg/kg. Maintenance: 0.1–0.15 mg/kg. Infusion: 0.3–0.6mg/kg/h
Ropivacaine	Amide-type LA agent. Possibly less motor block than other agents. Duration similar to bupivacaine, but lower toxicity. pKa 8.1	Acute porphyrias, cardiovascular disease, complete heart block, epilepsy, myasthenia gravis	Toxicity: tongue/circumoral numbness, restlessness, tinnitus, seizures, cardiac arrest	0.2–1% solution. Maximum dose dependent upon injection site, 3–4mg/kg/4h	0.2–1% solution. Infiltration/epidural: max dose dependent upon injection site, 3–4mg/kg/4h
Salbutamol	β2 receptor agonist. Treatment of bronchospasm. Larger doses now suggested in paediatrics or IV. Tocolytic agent in obstetric anaesthesia	Monitor K^+ concentration with higher doses	Tremor, vasodilation, tachycardia, hypokalaemia	Slow IV: 1mo to 2y, 5 micrograms/kg; >2y, 15 micrograms/kg (max 250 micrograms). Infusion: 1–5 micrograms/kg/min. Nebuliser: <5y, 2.5mg; >5y, 2.5–5mg	250micrograms slow IV, then 5 micrograms/min (up to 20 micrograms/min). Nebuliser: 2.5–5mg PRN
Sufentanil	More potent thiamylal analogue of fentanyl (five times potency). Analgesia. Duration 20–45min	See Fentanyl	See Fentanyl	Analgesia: 10–30 micrograms (0.2–0.6 micrograms/kg). Anaesthesia 0.6–8 micrograms/kg	Analgesia: 10–30 micrograms (0.2–0.6 micrograms/kg). Anaesthesia 0.6–8 micrograms/kg

(Continued)

Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Sugammadex	Specific cyclodextrin reversal agent for rocuronium and vecuronium	Wait 24h after use before using rocuronium/ vecuronium in patient; fusidic acid or flucloxacillin may displace relaxant from sugammadex within 6h	Binds with contraceptive pill	If two twitches present from TOF, 2mg/kg. Full reversal NR at present	If two twitches present from TOF, give 2mg/kg; 4mg/kg if neuromuscular blockade reoccurs; and 16mg/kg to reverse full intubating dose of rocuronium immediately
Suxa-methonium	Depolarising muscle relaxant. Rapid short-acting muscle paralysis. Phase II block develops with repeated doses (>8mg/kg). Store at 2–8°C	Prolonged block in plasma cholinesterase deficiency, hypokalaemia, hypocalcaemia. MH, neuromuscular disorders. ↑ serum K^+ (normally 0.5mmol/L, greater in burns, trauma, upper motor neurone injury)	↑ IOP. Bradycardia with 2nd dose	IV: 1–2mg/kg. IM: 3–4mg/kg	1–1.5mg/kg. Infusion: 0.5–10mg/min
Tapentadol	Moderate to severe pain managed only by opioids	Reduce in hepatic impairment	Diarrhoea, dyspepsia, weight loss	Consult tertiary consultant	>18y PO: 50mg 4- to 6-hourly, max 700mg
Temazepam	Benzodiazepine.Sedation or premedication. Duration 1–2h	↓ requirement for anaesthetic agents	Respiratory depression in combination with opioids. Amnesia	PO: 0.3mg/kg preoperatively. NR <12y	PO: 10–40mg 1h preoperatively (elderly 10–20mg)

Thiopental	Short-acting thiobarbiturate. Induction of anaesthesia, anticonvulsant, cerebral protection. Recovery due to redistribution	Accumulation with repeated doses. Caution in hypovolaemia and elderly. Porphyria	Hypotension.Necrosis if intra-arterial	Induction: neonate 2–4 mg/kg, child 5–6mg/kg. Status: 2–4mg/kg, then 8mg/kg/h	Induction/cerebral protection: 3–5mg/kg. Anticonvulsant: 0.5–2 mg/kg PRN
Tramadol	Analgesic thought to have less respiratory depression, constipation, euphoria and abuse potential than other opioids. Has opioid and non-opioid mechanisms of action	Only 30% antagonised by naloxone. Caution in epilepsy. Previously not recommended for intraoperative use. MAOI	Nausea, dizziness, dry mouth. ↑ side effects in conjunction with other opioids	>12y: adult dose	PO: 50–100mg 4-hourly. Slow IV/IM: 50–100mg 4-hourly (100mg initially, then 50mg increments to max 250mg). Max 600mg/d
Tranexamic acid	Inhibits plasminogen activation, reducing fibrin dissolution by plasmin. Reduced haemorrhage in major trauma, prostatectomy and dental extraction. Can be used for surgical bleeding in obstetric haemorrhage and prophylaxis in arthroplasty	Avoid in thromboembolic disease, renal impairment and pregnancy	Dizziness, nausea	Slow IV: 10–15mg/kg tds. PO: 10–25mg/kg tds	Slow IV: 0.5–1g tds. PO: 15–25mg/kg tds
Vasopressin	Synthetic ADH used in treatment of diabetes insipidus, resistant vasodilatory shock, variceal bleeding	Extreme caution in coronary vascular disease	Pallor, coronary vasoconstriction, water intoxication	Diabetes insipidus SC/IM: <12y, 0.1–0.4 micrograms/d; >12y, 1–4 micrograms/d. See *BNF for Children* for specific indications	Diabetes insipidus SC/IM: 5–20 units 4-hourly. Septic shock infusion: 1–4 units/h. Variceal bleed: 20 units over 15min

(Continued)

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Table 42.1 *(Contd.)*

Drug	Description and perioperative indications	Cautions and contraindications	Side effects	Dose (paediatric)	Dose (adult)
Vecuronium	Aminosteroid NDMR. Cardiostable and no histamine release. Duration 30–45min	See Cisatracurium	See Cisatracurium	Intubation: 80–100 micrograms/kg. Maintenance: 20–30 micrograms/kg. Infusion: 0.8–1.4 micrograms/kg/min	Intubation: 80–100 micrograms/kg. Maintenance: 20–30 micrograms/kg. Infusion: 0.8–1.4 micrograms/kg/min

bd, twice daily; ET, endotracheal; IM, intramuscular; IV, intravenous; NR, not recommended; od, once daily; PO, *per os* (oral); qds, four times daily; SC, subcutaneous; SL, sublingual; tds, three times daily. Doses are IV and dilutions in 0.9% sodium chloride, unless otherwise stated.

Infusion regimes

(See Table 42.2.)

Table 42.2 Infusion regimes

Drug	Indication	Diluent	Dose	Suggested regime (60kg adult)	Infusion range	Initial rate (adult)	Comments
Adrenaline	Treatment of hypotension	0.9% sodium chloride, 5% glucose	2–20 micrograms/min (0.04–0.4 micrograms/kg/min)	5mg/50mL (100 micrograms/mL)	1.2–12+ mL/h	5mL/h	Via central catheter. Suggest 1mg/50mL for initial intraoperative use (or 1mg/500mL if no central access)
Alfentanil	Analgesia	0.9% sodium chloride, 5% glucose	0.5–1 micrograms/kg/min	Undiluted (500 micrograms/mL)	0–8 mL/h	4mL/h	1–2mg can be added to 50mL of propofol for infusion
Aminophylline	Bronchodilation	0.9% sodium chloride, 5% glucose	0.5mg/kg/h	250mg/50mL (5mg/mL)	0–6mL/h	6mL/h	First 5mg/kg can be given over 20min if theophylline-naïve Caution in patients already receiving theophyllines (serum level 10–20mg/L); 0.6mg/kg of aminophylline should increase serum level by 1mg/L Side effects include tachydysrhythmias, tachypnoea, seizures and nausea

(Continued)

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Table 42.2 *(Contd.)*

Drug	Indication	Diluent	Dose	Suggested regime (60kg adult)	Infusion range	Initial rate (adult)	Comments
Amiodarone	Treatment of arrhythmias	5% glucose only	Loading infusion 5mg/kg over 20–120min, then 900mg over 24h	300mg/50mL (6mg/mL)	25–50 mL/h, then 6mL/h	25mL/h	Via central line (peripherally 'in extremis'). Max 1.2g in 24h. Adjust to therapeutic levels
Atracurium	Muscle relaxant	0.9% sodium chloride, 5% glucose	0.3–0.6mg/kg/h	Undiluted (10mg/mL)	1.5–4 mL/h	3mL/h	Assess rate with nerve stimulator
Digoxin	Rapid control of ventricular rate	0.9% sodium chloride, 5% glucose	250–500 micrograms over 30–60min; 0.75–1mg over 2h	250–500 micrograms/50mL	0–100 mL/h	50mL/h	ECG monitoring suggested
Dobutamine	β1-adrenergic agonist, positive inotrope and chronotrope. Cardiac failure	0.9% sodium chloride, 5% glucose	2.5–10 micrograms/kg/min	250mg/50mL (5mg/mL)	2–7mL/h	2mL/h	ECG monitoring advised. Hypertension and arrhythmias
Dopamine	Naturally occurring catecholamine with α, β1 and dopaminergic activity. Inotropic agent	0.9% sodium chloride, 5% glucose	2–10 micrograms/kg/min	200mg/50mL (4mg/mL)	2–9mL/h	2mL/h	ECG monitoring advised. Via central line. Avoid in phaeochromocytoma (due to noradrenaline release)

Esmolol	β-blocker	0.9% sodium chloride, 5% glucose	50–200 micrograms/kg/min	2.5g/50mL (50mg/mL)	3–15 mL/h	3mL/h	ECG monitoring
Glyceryl trinitrate	Controlled hypotension	0.9% sodium chloride, 5% glucose	0.5–12mg/h	50mg/50mL (1mg/mL)	0.5–12 mL/h	5mL/h	
Heparin	Anticoagulation	0.9% sodium chloride, 5% glucose	24 000–48 000 units/24h	50 000 units/50mL (1000 units/mL)	1–2mL/h	2mL/h	Check APTT after 12h. See local guidelines
Insulin (soluble)	DM	0.9% sodium chloride	Sliding scale	50units/50mL (1 unit/mL)	Sliding scale	Sliding scale	
Isoprenaline	Synthetic catecholamine with β1-adrenergic agonist activity. Treatment of heart block and bradycardia and β-blocker overdose	0.9% sodium chloride, 5% glucose	0.5–10 micrograms/min	1mg/50mL (20 micrograms/mL)	0.5–30 mL/h	7mL/h	MHRA special order request required. Caution with IHD, hyperthyroidism, DM . Paediatric dose; bolus 5 micrograms/kg. Infusion: 0.02–1 micrograms/kg/min

(Continued)

Table 42.2 *(Contd.)*

Drug	Indication	Diluent	Dose	Suggested regime (60kg adult)	Infusion range	Initial rate (adult)	Comments
Ketamine	GA	0.9% sodium chloride, 5% glucose	1–3mg/kg/h	500mg/50mL (10mg/mL)	6–18 mL/h	10mL/h	Induction 0.5–2mg/kg
Ketamine	Analgesia	0.9% sodium chloride, 5% glucose	0.2mg/kg/h	200mg/50mL (4mg/mL)	0–6mL/h	3mL/h	With midazolam 2–5mg/h
Ketamine	'Trauma' mixture	0.9% sodium chloride	0.5mL/kg/h	50mL mixture (4mg/mL ketamine)	15–45 mL/h	30mL/h	200mg ketamine + 10mg midazolam+ 10mg vecuronium in 50mL
Lidocaine (lignocaine)	Ventricular arrhythmias	0.9% sodium chloride	4mg/min for 30min, 2mg/min for 2h, then 1mg/min for 24h	500mg/50mL (10mg/mL, 1%)	6–24 mL/h	24mL/h	After 50–100mg, slow IV bolus. ECG monitoring
Lidocaine (lignocaine)	Postoperative analgesia	0.9% sodium chloride	Loading dose 1.5mg/kg over 2-4min (max. 20min), followed by 0.5–2mg/kg/h	2% lidocaine neat (20mg/mL)	1.5–6 mL/h	3mL/h	Use IBW. ECG monitoring throughout. Avoid in hypotension, hypovolaemia, heart block, concurrent regional technique with LA. Caution with renal, hepatic and cardiac dysfunction. Monitor for signs of toxicity and sedation

Morphine	Analgesia	0.9% sodium chloride	0–3.5mg/h	50mg/50mL (1mg/mL)	0–3.5 mL/h	2mL/h	Monitor respiration and sedation hourly. Administer O_2
Naloxone	Opioid antagonist	0.9% sodium chloride, 5% glucose	>1 microgram/kg/h	2mg/500mL (4 micrograms/mL)		100mL/h	Rate adjusted according to response
Noradrenaline	Catecholamine α-adrenergic agonist. Treatment of hypotension	5% glucose	2–25 micrograms/min (0.04–0.5 micrograms/kg/min, up to 1 microgram/kg/min in extremis, weaned as soon as possible)	4mg/40mL (100 micrograms/mL)	1.2–12+ mL/h	5mL/h	Via central line. Potentiated by MAOIs and TCAs If infusion >0.5micrograms/kg/min consider adding a second ionotropic agent
Octreotide	Somatostatin analogue	0.9% sodium chloride	25–50 micrograms/h	500micrograms/50mL (10 micrograms/mL)	2–5mL/h	5mL/h	Use in variceal bleeding unlicensed
Oxytocin	Prevention of uterine atony	0.9% sodium chloride, 5% glucose	0.02–0.125 units/min (10 units/h)	30units in 500mL (0.06 units/mL)	30–125 mL/h	125mL/h	Individual unit protocols vary
Phenylephrine	Treatment of hypotension	0.9% sodium chloride, 5% glucose	30–60 micrograms/min	5mg in 50mL (100 micrograms/mL)	18–36 mL/h	30mL/h	Gaining popularity for regional Caesarean

(Continued)

Table 42.2 *(Contd.)*

Drug	Indication	Diluent	Dose	Suggested regime (60kg adult)	Infusion range	Initial rate (adult)	Comments
Phenytoin	Anticonvulsant prophylaxis	0.9% sodium chloride	20mg/kg	900mg/90mL (administer via 0.22–0.5 micrometre filter)	Up to 50 mg/min	180mL/h	ECG and BP monitoring. Complete within 1h of preparation
Propofol	Anaesthesia		6–10mg/kg/h	Undiluted (10mg/mL)	36–60 mL/h		TCI: initially 4–8 micrograms/mL, then 3–6 micrograms/mL
Propofol	Sedation		0–3mg/kg/h	Undiluted (10mg/mL)	0–20 mL/h		TCI: 0–2.5 micrograms/mL
Remifentanil	Analgesia during GA or labour as PCA	0.9% sodium chloride, 5% glucose	0.1–1.0 micrograms/kg/min PCA in labour: 25–75 micrograms bolus, 3min lockout (0.5–1.5mL of 50 micrograms/mL)	2mg/40mL (50 micrograms/mL)	5–40 mL/h	8mL/h (2mL/h if spontaneous breathing)	Metabolised by non-specific esterases, duration 5–10min. May be mixed with propofol; SV: 125 micrograms/50mL; IPPV: 250–500 micrograms/50mL
Salbutamol	Bronchospasm	5% glucose	5–20 micrograms/min	1mg/50mL (20 micrograms/mL)	15–60 mL/h	30mL/h	After 250 micrograms, slow IV bolus

Sodium bicarbonate	Acidosis		(Weight (kg) × base deficit × 0.3) mmol	Undiluted (8.4% solution)			8.4%, 1000mmol/L. Via central line, if possible
Vasopressin	Vasodilatory shock	5% glucose	0.01–0.04 units/min	Argipressin (synthetic vasopressin) 20 units (1mL) in 50mL	1.5–6 mL/h	3mL/h	Side effects become more common with higher doses and with little clinical benefit. Central access only

Helpful hints when calculating infusion rates.

3mg/kg/50mL, then 1mL/h = 1 microgram/kg/min; 3mg/50mL, then 1mL/h = 1 microgram/min.

Rate (mL/h) = 60 × rate (micrograms/kg/min) × weight (kg)/concentration (micrograms/mL).

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