THE INDISPENSABLE SURGICAL HANDBOOK
- A CUT ABOVE THE REST

OXFORD HANDBOOK OF CLINICAL SURGERY

SENIOR EDITORS Greg McLatchie | Neil Borley

Associate editors: Anil Agarwal, Santhini Jeyarajah, Rhiannon Harris, Ruwan Weerakkody

Comprehensively updated to include latest guidelines, management algorithms, and evidence-based practice

Concise, quick-reference style to provide essential surgical information and common operative techniques with practical hints and procedures

Includes brand new chapters on emergency surgery, day case surgery, and surgery in remote and rural environments

FIFTH EDITION

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FIFTH EDITION

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Clinical Surgery

FIFTH EDITION

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Foreword

This popular little book has stood the test of time. Having first been published just over three decades ago it has now reached this, the fifth edition. In keeping with the Oxford handbook series, it offers systematic bite-sized chapters of salient information on the broader aspects of clinical surgery. It is both easily read and speedily referenced.

Surgery and surgical training have changed so much over the last thirty years with a greater emphasis on the timely management of emergencies and the professionalism of the surgeon. The expansion of editors offers reassuring credibility to a book that has evolved with the ever-changing vista. I was delighted to see that two of the 'bright young things' (associate editors) have recently been my own trainees.

The pocket size and layout of this book lend it to being a working guide for the medical student embarking on a surgical attachment. The helpful bullet points serve as a crib book for medical student finals examinations and as an aide memoire for the budding MRCS candidate.

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Vice President, Royal College of Surgeons of England

Preface to the fifth edition

These woods are lovely, dark and deep
But I have promises to keep
And miles to go before I sleep
And miles to go before I sleep
'Stopping by Woods on a Snowy Evening'
Robert Frost

The poem is about a man riding his horse home who stops to watch snow fall in a forest and reflects on the beauty of the scene. Frost in his simplicity of expression adds layers of meaning about a journey through Life or indeed, Surgery.

The OHCS was first conceived in the mid-eighties and refused acceptance by several publishers but accepted by OUP if I, the author, would consider modelling it on the first Oxford Handbook, the OHCM, which had been successfully received. I was asked to submit 5 sample chapters i.e. 5 pages. These included: The no-lose philosophy in Surgery.
It is possible to die of a broken heart
and
Beware the Patient You Do Not Like

Such chapters are not the usual fare of traditional surgical texts but it was accepted. The label, BYT (Bright Young Thing) was appended to my file by the senior medical publisher and the book was written by me with the assistance of my surgical registrar, Mr Para. In the text some of these chapters are included albeit in abbreviated form.

The book has become a world wide bestseller and continues to evolve. In particular, we have recruited 4 new BYTs. to the editorial team and are sure they will continue to modernize the text.

The horse is no longer our method of transport but general surgical training is still essential in many countries of the world.

Surgery is a personal experience second to none involving the knowledge of the physician with the expertise of the surgeon and is 'lovely, dark and deep'. There has been no diminution in skill following Specialist Training and reduced hours.

However, our clinical independence is increasingly being threatened by burgeoning bureaucracy with erosion of our freedom to treat patients

In my thirty plus years as a surgeon I have witnessed enormous changes in surgical outcomes and a welcome involvement of our female colleagues. Sir Lancelot Spratt stated in the 'Doctor in the House' films:
'In the good surgeon:
The eye of the eagle
The heart of the lion
And
The hands of a woman!'

The remaining miles to go belong to all of us and I am sure that our new young surgeons will fulfil this before they sleep … before they sleep!

Greg Mclatchie
December 2020

Preface to the fourth edition

Sometimes we have to look backward to look forward. Since 1990, surgery has witnessed cataclysmic changes. In our Trust, the first laparoscopic cholecystectomy was performed in 1992, and has now become the procedure of choice for most gall bladder disease and many other surgical operations in the western world. With the expansion of laparoscopic surgery, we have encountered a whole new range of complications with an escalation in the demise of general surgery as the result of hyperspecialization. There are many surgical trainees who have scant experience of open surgery and who have, due to European directives, limited time exposure to surgical procedures. In fact, most technical training is now obtained from emergency on call such that a new speciality of emergency surgery is developing. A recent *British Medical Journal* (BMJ) article recommended a training programme for surgeons wishing to work in remote and rural surgery—not only in the Developing World, but in remote and isolated communities in the United Kingdom! General surgery may largely have gone, but it should not be forgotten. Most countries in the world do not have access to these recent innovations and there is still a case in the developed world for experience in open and general surgery to be incorporated in the formal training programmes of junior surgeons.

Greg McLatchie
Hartlepool, September 2012

Preface to the first edition

The idea of this book was first suggested by Mr Gordon McBain, consultant surgeon at the Southern General Hospital, Glasgow. We have received considerable support from the staff of Oxford University Press, and are also indebted to Mr J. Rhind and Dr J. Daniel for their contributions and our surgical teachers, especially Mr J. S. F. Hutchison, Mr M. K. Browne, Mr J. Neilson, Mr D. Young, Mr A. Young, and the late Mr I. McLennan whose practical advice and anecdotes pepper the pages …

Greg McLatchie
S. Parameswaran 1990

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Symbols and abbreviations

↓	decreased
↑	increased
↔	normal
→	leading to
▶	important
▶▶	don't dawdle
	controversial topic
	warning
⚠	warning
▶	important
▶▶	don't dawdle
	website
	cross reference
♀	female
♂	male
<	less than
>	more than
≥	equal to or greater than
≤	equal to or less than
±	plus or minus
%	per cent
~	approximately
≈	approximately equal to
α	alpha
β	beta
γ	gamma
°	degree
1°	primary
2°	secondary
®	registered
™	trademark
2WW	2-week wait
2D	two-dimensional
3D	three-dimensional
4D	four-dimensional
AAA	abdominal aortic aneurysm
AAGBI	Association of Anaesthetists of Great Britain and Ireland
ABG	arterial blood gas
A&E	Accident and Emergency
Ab	antibody
ABG	arterial blood gas
ABPI	ankle–brachial pressure index
ACC	adrenocortical carcinoma
ACE	angiotensin-converting enzyme
ACh	acetylcholine
ACJ	acromioclavicular joint
ACL	anterior cruciate ligament
ACT	activated clotting time
ACTH	adrenocorticotrophic hormone
AD	autosomal dominant
ADH	antidiuretic hormone
ADHD	attention-deficit/hyperactivity disorder
ADP	adenosine diphosphate
AED	automated external defibrillator
AF	atrial fibrillation
AFP	alpha-fetoprotein
Ag	antigen
AIDS	acquired immune deficiency syndrome
AIN	anal intraepithelial neoplasia
AJCC	American Joint Committee on Cancer
AKA	above-knee amputation
AKI	acute kidney injury
AKIN	Acute Kidney Injury Network
ALCL	anaplastic large cell lymphoma
ALL	acute lymphoblastic leukaemia
ALP	alkaline phosphatase
ALS	Advanced Life Support
ALT	alanine aminotransferase*or* anterolateral thigh
a.m.	*ante meridiem*
AMES	Age, Metastases, Extent, Size
AML	acute myeloid leukaemia
AMPLE	Allergy, Medication, Past medical history, Last meal, Events of the incident
AMTS	Abbreviated Mental Test Score
ANA	antinuclear antibodies
ANC	axillary node clearance
ANDI	aberrations in normal development and involution (of breast)
AP	anteroposterior

APACHE	Acute Physiology and Chronic Health Evaluation
APC	antigen-presenting cell *or* argon plasma coagulation
APER	abdominoperineal resection
APTT	activated partial thromboplastin time
AR	aortic regurgitation
ARDS	acute respiratory distress syndrome
ARR	absolute risk reduction *or* aldosterone:renin ratio
ASA	American Society of Anesthesiologists
5-ASA	5-aminosalicyclic acid
ASC	adipose-derived stem cell
ASIS	anterior superior iliac spine
ASO	anti-streptolysin O
AST	asparate aminotransferase
ATC	anaplastic thyroid cancer
ATD	antithyroid drug
ATG	anti-thymocyte globulin
ATH	atherosclerosis
ATLS	advanced trauma life support
ATP	adenosine triphosphate
AUR	acute urinary retention
AV	arteriovenous *or* atrioventricular
AVM	arteriovenous malformation
AVN	avascular necrosis
AVPU	Alert, responds to Vocal stimuli, responds only to Painful stimuli, Unresponsive to all stimuli
AXR	abdominal X-ray
BADS	British Association of Day Surgery
BAL	bronchoalveolar lavage
BAPRAS	British Association of Plastic, Reconstructive and Aesthetic Surgeons
BB	borderline borderline (leprosy)
BCC	basal cell carcinoma
BCG	bacille Calmette–Guérin
BCR	B-cell receptor
bd	twice daily
BDNF	brain-derived neurotrophic factor
BE	base excess
BIA-ALCL	breast implant associated-anaplastic large cell lymphoma
BiPAP	bilevel positive airway pressure
BKA	below-knee amputation
BMI	body mass index
BNF	*British National Formulary*
BOA	British Orthopaedic Association
BOO	bladder outflow obstruction
BP	blood pressure
BPH	benign prostatic hyperplasia
bpm	beats per minute
BS	blood sugar
BSA	body surface area
BXO	balanitis xerotica obliterans
Ca^{2+}	calcium
CABG	coronary artery bypass graft
CAD	coronary artery disease
cAMP	cyclic adenosine monophosphate
CAPD	continuous ambulatory peritoneal dialysis
CAST	childhood accidental spiral tibial
CBD	common bile duct
CCF	congestive cardiac failure
CDT	*Clostridium difficile* toxin
CEA	carcinoembryonic antigen *or* carotid endarterectomy
CEPOD	Confidential Enquiry into Patient Outcome and Death
CF	cystic fibrosis
CFA	common femoral artery
CFU	colony-forming unit
CI	confidence interval
CJD	Creutzfeldt–Jakob disease
Cl^-	chloride
CIN	cervical intra-epithelial neoplasia
CLI	critical limb ischaemia
CLL	chronic lymphocytic leukaemia
cm	centimetre
CMF	cyclophosphamide, methotrexate, and 5-fluorouracil
cmH_2O	centimetres of water
CMI	cell-mediated immune (reaction)
CMV	cytomegalovirus *or* controlled mechanical ventilation
CN	cranial nerve
CNI	calcineurin inhibitor
CNS	central nervous system

CO	cardiac output
CO_2	carbon dioxide
COAD	chronic obstructive airways disease
COMT	catechol-O-methyltransferase
COPD	chronic obstructive pulmonary disease
CPAP	continuous positive airway pressure
CPB	cardiopulmonary bypass
CPD	continuing professional development
CPET	cardiopulmonary exercise testing
CPEX	cardiopulmonary exercise
C,P,&O	cysts, parasites, and ova
CPR	cardiopulmonary resuscitation
CQC	Care Quality Commission
Cr	creatinine
CRCa	colorectal cancer
CRP	C-reactive protein
CSF	cerebrospinal fluid
C-spine	cervical spine
CT	computerized tomography
CTA	CT angiography
CTEV	congenital talipes equinovarus
CTPA	computerized tomography pulmonary angiography
Cu	copper
CUSUM	cumulative sum
CV	central venous
CVA	cerebrovascular accident
CVC	central venous cannulation *or* central venous catheter
CVP	central venous pressure
CVVH	continuous veno-venous haemofiltration
CVVHDF	continuous veno-venous haemodialysis with filtration
Cx	circumflex
CXR	chest X-ray
DA	dopamine
DALM	dysplasia-associated lesion or mass
DBD	donor after brainstem death
DC	direct current
DCA	deoxycholate citrate agar
DCD	donor after circulatory death
DCIA	deep circumflex iliac artery
DCIS	ductal carcinoma *in situ*
DDH	developmental dysplasia of the hip
DHS	dynamic hip screw
DHT	dihydrotestosterone
DIC	disseminated intravascular coagulation
DIEP	deep inferior epigastric artery perforator (flap)
DIPJ	distal interphalangeal joint
DIT	diiodotyrosine
dL	decilitre
DM	diabetes mellitus
DMSA	dimercaptosuccinate
DNA	deoxyribonucleic acid *or* did not attend
DNR	do not resuscitate
DP	distal phalanx
DPL	diagnostic peritoneal lavage
DRE	digital rectal examination
DRUJ	distal radioulnar joint
DSA	digital subtraction angiography
DSD	disorder of sex development
dsDNA	double-stranded deoxyribonucleic acid
DSU	day surgical unit
DTC	differentiated thyroid cancer
DVT	deep vein thrombosis
EBV	Epstein–Barr virus
ECD	expanded criteria donor
ECF	extracellular fluid
ECG	electrocardiogram
ECMO	extracorporeal membrane oxygenation
ED	erectile dysfunction
EGF	epidermal growth factor
eGFR	estimated glomerular filtration rate
EHEC	enterohaemorrhagic *Escherichia coli*
EHIC	enteroinvasive *Escherichia coli*
EMD	electromechanical delay
EMG	electromyography
EMR	endoscopic mucosal resection
ENT	ear, nose, and throat
EPEC	enteropathogenic *Escherichia coli*
EPL	extensor pollicis longus
EPO	erythropoietin
ER	(o)estrogen receptor

ERAS	enhanced recovery after surgery
ERCP	endoscopic retrograde cholangiopancreatography
ES	endoscopic sphincterotomy
ESD	endoscopic submucosal dissection
ESR	erythrocyte sedimentation rate *or* endoscopic submucosal resection
ESWL	extracorporeal shock wave lithotripsy
ET	endotracheal tube
$ETCO_2$	end-tidal carbon dioxide
ETEC	enterotoxigenic *Escherichia coli*
ETT	endotracheal tube
EU	European Union
EUA	examination under anaesthesia
EuroSCORE	European System for Cardiac Operative Risk Evaluation
EUS	endoscopic ultrasound
EVAR	endovascular aneurysm repair
FAP	familial adenomatous polyposis
FARO	fixed-angle removable orthotic
FAST	focused abdominal sonography for trauma
FBC	full blood count
FDP	flexor digitorum profundus
FDS	flexor digitorum superficialis
FEV_1	forced expiratory volume in 1 second
FFP	fresh frozen plasma
FGF	fibroblast growth factor
FHH	familial hypocalciuric hypercalcaemia
FiO_2	fraction of oxygen in inspired air
FIT	faecal immunochemical test
FLR	future liver remnant
FMTC	familial medullary thyroid cancer
FNA	fine needle aspiration
FNAB	fine needle aspiration biopsy
FNAC	fine needle aspiration cytology
FNH	focal nodular hyperplasia
FOB	faecal occult blood
FPL	flexor pollicis longus
FSH	follicle-stimulating hormone
FTC	follicular thyroid cancer
5-FU	5-fluorouracil
g	gram
G	gauge
GA	general anaesthesia
GANT	gastrointestinal autonomic nerve tumour
GAVE	gastric antral vascular ectasia
GCS	Glasgow Coma Scale
gFOBT	guaiac-based faecal occult blood test
GFR	glomerular filtration rate
GGT	gamma glutamyl transferase
GH	growth hormone
GI	gastrointestinal
GIP	gastric inhibitory polypeptide
GIST	gastrointestinal stromal tumour
GMC	General Medical Council
GORD	gastro-oesophageal reflux disease
GP	general practitioner
GpIIb/IIIa	glycoprotein IIb/IIIa
G6PD	glucose-6-phosphate dehydrogenase
GTN	glyceryl trinitrate
GU	genitourinary
Gy	gray
h	hour
H^+	hydrogen ion
HAI	health care-associated infection
HALO	haemorrhoidal artery ligation operation
HAT	hepatic artery thrombosis
Hb	haemoglobin
HbA1c	glycosylated haemoglobin
HBc	hepatitis B core
HBsAg	hepatitis B surface antigen
HBV	hepatitis B virus
HCC	hepatocellular carcinoma
HCG	human chorionic gonadotrophin
HCO_3^-	bicarbonate
HCV	hepatitis C virus
HDU	high dependency unit
HDV	hepatitis D virus
HER2	human epidermal growth factor receptor 2
HFNO	high-flow nasal oxygen
HGV	heavy goods vehicle
HHD	handheld Doppler
HHV-8	human herpesvirus 8

HIDA	hepatobiliary iminodiacetic acid
HIT	heparin-induced thrombocytopenia
HITT	heparin-induced thrombocytopenia and thrombosis
HIV	human immunodeficiency virus
HLA	human leucocyte antigen
H_2O	water
HPV	human papillomavirus
HR	heart rate
HRT	hormone replacement therapy
HSV	herpes simplex virus
HTLV	human T-cell lymphocytotropic virus
HTN	hypertension
HVA	homovanillic acid
I^-	iodide
IABP	intra-aortic balloon pump
IC	intermittent claudication
ICA	internal carotid artery
ICAM-1	Intercellular adhesion molecule 1
ICD	intracardiac defibrillator
ICP	intracranial pressure
ICS	incident command system
ICU	intensive care unit
IF	intrinsic factor
IFAT	indirect fluorescent antibody test
IFN	interferon
IgA	immunoglobulin A
IgE	immunoglobulin E
IGF	insulin growth factor
IgG	immunoglobulin G
IgG4	immunoglobulin G4
IgM	immunoglobulin M
IHD	ischaemic heart disease
IJV	internal jugular vein
IL	interleukin
IM	intramuscular
IMA	inferior mesenteric artery
IMCA	independent mental capacity advocate
IMHS	intramedullary hip screw
in	inch
INR	international normalized ratio
IO	intraosseous
ioPTH	intraoperative parathyroid hormone (assay)
IPJ	interphalangeal joint
IPL	intense pulsed light
IPPV	intermittent positive pressure ventilation
IPSS	International Prostate Symptom Score
IT	information technology
ITA	internal thoracic artery
ITU	intensive treatment unit
IU	international unit
IV	intravenously
IVC	inferior vena cava
IVRA	intravenous regional anaesthesia
IVU	intravenous urogram
J	joule
JVP	jugular venous pressure
K	Kirschner (wire)
K^+	potassium
kcal	kilocalorie
KCl	potassium chloride
KDIGO	Kidney Disease: Improving Global Outcomes
kg	kilogram
kPa	kilopascal
KUB	kidneys/ureters/bladder
L	litre
LA	local anaesthetic *or* left atrium/atrial
LAD	left anterior descending (artery)
LAGB	laparoscopic adjustable gastric banding
lb	pound
LC	laparoscopic cholecystectomy
LD	latissimus dorsi
LDH	lactate dehydrogenase
LDL	low density lipid
LESS	laparoscopic and endoscopic single-site surgery
LFA1	lymphocyte function-associated antigen 1
LFT	liver function test
LGI	lower gastrointestinal
LH	luteinizing hormone
LHRH	luteinizing hormone-releasing hormone

Li	lithium
LIF	left iliac fossa
LIMA	left internal mammary artery
LITA	left internal thoracic artery
LL	lepromatous leprosy
LLQ	left lower quadrant
LMA	laryngeal mask airway
LMS	left main stem
LMWH	low-molecular weight heparin
LOS	lower oesophageal sphincter
LPA	Lasting Power of Attorney
LRINEC	Laboratory Risk Indicator for Necrotizing Fasciitis
LSV	long saphenous vein
LTB4	leukotriene B4
LUQ	left upper quadrant
LUTS	lower urinary tract symptoms
LV	left ventricle *or* left ventricular
LVEDD	left ventricular end-diastolic dimension
LVEDP	left ventricular end-diastolic pressure
LVEDV	left ventricular end-diastolic volume
LVEF	left ventricular ejection fraction
LVESD	left ventricular end-systolic dimension
LVF	left ventricular failure
m	metre
MACIS	Metastases, Age, Completeness of resection, Invasion, Size
MALToma	tumour of mucosa-associated lymphoid tissue
MAC	membrane attack complex
MAG3	^{99m}Tc-mercaptoacetyltriglycine
MALT	mucosa-associated lymphoid tissue
MAO	monoamine oxidase
MAP	mean arterial pressure
MASS	Multicentre Aneurysm Screening Study
MCA	Mental Capacity Act
MCH	mean cell haemoglobin
MCPJ	metacarpophalangeal joint
MCRP	magnetic resonance cholangiopancreatography
M,C,&S	microscopy, culture, and sensitivities
MCV	mean cell volume
MDRTB	multidrug-resistant tuberculosis
MDT	multidisciplinary team
MEN	multiple endocrine neoplasia
mEq	milliequivalent
mg	milligram
Mg^{2+}	magnesium
MHC	major histocompatibility complex
MHz	megahertz
MI	myocardial infarction
MIBG	metaiodobenzylguanidine
min	minute
MIST	mechanism of injury, injuries identified, symptoms and signs at the scene, and treatment initiated
MIT	monoiodotyrosine
mL	millilitre
mmHg	millimetre mercury
MHz	megahertz
mmol	millimole
MMR	mismatch repair (genes)
Mn	manganese
MNG	multinodular goitre
MODS	multiple organ dysfunction syndrome
MR	mitral regurgitation
MRA	magnetic resonance angiography
MRC	Medical Research Council (scale)
MRCP	magnetic resonance cholangiopancreatography
MRCS	Member of the Royal College of Surgeons of England
MRI	magnetic resonance imaging
MRSA	methicillin (or multiply)-resistant *Staphylococcus aureus* ms millisecondMSU midstream urine
MTC	medullary thyroid cancer
mTOR	mammalian target of rapamycin
MTP	mid-thigh perforator
MTPJ	metatarsophalangeal joint
MUA	manipulation under anaesthesia
MUGA	multigated acquisition
MV	mitral valve
Na^+	sodium

NA	noradrenaline (norepinephrine)
NAAT	nucleic acid amplification test
NaCl	sodium chloride
NAFLD	non-alcoholic fatty liver disease
NAI	non-accidental injury
NASCET	North American Symptomatic Carotid Endarterectomy Trial
NASH	non-alcoholic steatohepatitis
NBM	nil by mouth
NCEPOD	National Confidential Enquiry into Patient Outcome and Death
NEC	necrotizing enterocolitis
NEWS	National Early Warning Score
ng	nanogram
NG	nasogastric
NGT	nasogastric tube
NH_3	ammonia
NHS	National Health Service
NHSBSP	NHS Breast Screening Programme
NHSBT	NHS Blood and Transplant
NICE	National Institute for Health and Care Excellence
NIV	non-invasive ventilation
NJ	nasojejunal
NJT	nasojejunal tube
NK	natural killer (cell)
NNT	number needed to treat
N_2O	nitrous oxide
NOAC	non-vitamin K antagonist oral anticoagulant
NoSPG	North of Scotland Planning Group
NOTSS	non-technical skills for surgeons
NPP	Northern Periphery Programme
NRL	nasal restraining loop
NSAID	non-steroidal anti-inflammatory drug
NSCLC	non-small cell lung cancer
NSGCT	non-seminomatous germ cell tumour
NSPCC	National Society for the Prevention of Cruelty to Children
NSTEMI	non-ST segment elevation myocardial infarction
NVB	neurovascular bundle
nvCJD	new-variant Creutzfeldt–Jakob disease
NVH	non-visible haematuria
NYHA	New York Heart Association
O_2	oxygen
OCP	oral contraceptive pill
od	*omne in die* (once a day)
ODP	operating department practitioner
OGD	oesophago-gastro-duodenoscopy
OM	obtuse marginal
OPAT	outpatient parenteral antibiotic therapy
OPT	orthopantomogram
OR	odds ratio
ORIF	open reduction with internal fixation
PA	posterior–anterior
PAC	plasma aldosterone concentration *or* pulmonary artery catheter
$PaCO_2$	arterial carbon dioxide tension (partial pressure of carbon dioxide in arterial blood)
PAF	platelet-activating factor
PAIR	puncture, aspiration, injection, reaspiration
PAK	pancreas after kidney
PALS	Patient Advice and Liaison Service
PaO_2	arterial oxygen tension (partial pressure of oxygen in arterial blood)
PAP	pulmonary artery pressure
PAU	preoperative assessment unit
PAWP	pulmonary artery wedge pressure
PCA	patient-controlled analgesia
PCI	percutaneous coronary intervention
PCNL	percutaneous nephrolithotomy
pCO_2	carbon dioxide tension
PCR	polymerase chain reaction
PCV	pressure control ventilation
PDA	posterior descending artery
PDGF	platelet-derived growth factor
PDT	percutaneous dilatational tracheostomy
PE	pulmonary embolism

PECAM-1	platelet endothelial cell adhesion molecule-1
PEEP	positive end-expiratory pressure
PEFR	peak expiratory flow rate
PEG	percutaneous endoscopic gastrostomy
PEJ	percutaneous endoscopic jejunostomy
PEP	post-exposure prophylaxis
PET	positron emission tomography
PF	profunda femoris
pg	picogram
PG	parathyroid gland
pHPT	primary hyperparathyroidism
PICC	peripherally inserted central venous catheter
PID	pelvic inflammatory disease
PIPJ	proximal interphalangeal joint
PLL	posterior longitudinal ligament
PMN	polymorphonuclear neutrophil
pmol	picomole
PO	orally (*per os*)
PO_2	oxygen tension
PO4	phosphate
POEM	per-oral endoscopic myotomy
PONV	post-operative nausea and vomiting
POSSUM	Physiologic and Operative Severity Score for the enUmeration of Mortality and morbidity
PPD	purified protein derivative
PPH	procedure for prolapse and haemorrhoids
PPI	proton pump inhibitor
PPN	peripheral parenteral nutrition
P-POSSUM	Portsmouth-POSSUM
PPPD	pylorus-preserving pancreaticoduodenectomy
PR	per rectum *or* progesterone receptor
PRC	packed red cell
PSA	prostate-specific antigen
PSARP	posterior sagittal anorectoplasty
PSV	pressure support ventilation
PT	prothrombin time
PTA	pancreas transplant alone
PTC	percutaneous transhepatic cholangiogram *or* papillary thyroid cancer
PTE	pulmonary thromboembolism
PTH	parathyroid hormone
PTLD	post-transplant lymphoproliferative disorder
PTT	partial prothrombin time
PUJ	pelviureteric junction
PV	per vagina
PVD	peripheral vascular disease
PVR	pulmonary vascular resistance
qds	*quater die sumandus* (four times a day)
qFIT	quantitative faecal immunochemical test
qSOFA	quick SOFA
RA	right atrial *or* rheumatoid arthritis
RAI	radioactive iodine
RAP	right atrial pressure
RCA	right coronary artery
RCT	randomized controlled trial
RDT	rapid diagnostic test
REVAR	EVAR for ruptured abdominal aortic aneurysm
RFA	radiofrequency ablation
Rh	rhesus
RIF	right iliac fossa
RIFLE	risk, injury, failure, loss, end-stage
RIG	radiologically inserted gastrostomy
RLN	recurrent laryngeal nerve
RLQ	right lower quadrant
ROTEM	rotational thromboelastometry
RR	relative risk *or* risk ratio
RRIG	(Scottish) Remote and Rural Implementation Group
RRR	relative risk reduction
RRT	renal replacement therapy
RSTL	relaxed skin tension line
RTA	road traffic accident
RUQ	right upper quadrant
RV	right ventricle *or* right ventricular
RVP	right ventricular pressure
RYGB	Roux-en-Y gastric bypass
s	second
SA	sinoatrial (node)
SAAG	serum ascites albumin gradient
SBE	subacute bacterial endocarditis
SBO	small bowel obstruction

SBP	spontaneous bacterial peritonitis
SC	subcutaneous
SCC	squamous cell carcinoma
SCI	spinal cord injury
SCM	sternocleidomastoid
ScotSTAR	Scottish Specialist Transport and Retrieval
SD	standard deviation
SEMS	self-expanding metal stenting
SFA	superficial femoral artery
SFJ	saphenofemoral junction
SG	sleeve gastrectomy
SI	serious incident
SILS	single-incision laparoscopic surgery
SIMV	synchronized intermittent mandatory ventilation
SIRS	systemic inflammatory response syndrome
SIRT	selective internal radiotherapy
SL	sublingual
SLE	systemic lupus erythematosus
SLNB	sentinel lymph node biopsy
SMA	superior mesenteric artery
SOFA	Sequential Organ Failure Assessment (score)
SPC	suprapubic catheterization/ catheter
SPECT	single-photon emission computerized tomography
SPJ	saphenopopliteal junction
SPK	simultaneous pancreas and kidney
spp.	species
SPT	superficial partial thickness
SSC	Surviving Sepsis Campaign
SSI	surgical site infection
ssRNA	single-stranded ribonucleic acid
SSTI	severe soft tissue infection
STD	sexually transmitted disease
STD	sodium tetradecyl sulfate
STEMI	ST segment elevation myocardial infarction
STI	sexually transmitted infection
SUFE	slipped upper femoral epiphysis
SV	stroke volume *or* specific gravity
SVC	superior vena cava
SVR	systemic vascular resistance
SVT	supraventricular tachycardia
T_3	triiodothyronine
T_4	tetraiodothyronine (thyroxine)
TA	transabdominal
TACE	transarterial chemoembolization
TAMIS	transanal minimally invasive surgery
TAP	transversus abdominis plane
TAPP	transabdominal pre-peritoneal
TAVI	transcatheter aortic valve implantation
TB	tuberculosis
TBSA	total body surface area
TCC	transitional cell carcinoma
TCR	T-cell receptor
tds	*ter die sumendus* (three times a day)
TEDS	thromboembolic deterrent stockings
TEG	thromboelastography
TEMS	transanal endoscopic microsurgery
TEP	totally extraperitoneal
TF	tissue factor
TFCC	triangular fibrocartilage complex
TFL	tensor fascia lata
TFT	thyroid function test
Tg	thyroglobulin
TGF	transforming growth factor
Th1	T helper 1
Th2	T helper 2
THR	total hip replacement
TIA	transient ischaemic attack
TKA	through-knee amputation
TKR	total knee replacement
TLSO	thoracolumbar spine orthosis
TMT	tarsometatarsal
TNF	tumour necrosis factor
TNM	tumour nodes metastasis (cancer staging)
tPA	tissue plasminogen activator
TPG	transpulmonary pressure gradient
TPN	total parenteral nutrition
TPO	thyroid peroxidase antibodies
TRAb	thyroid receptor antibody

TRAM	transverse rectus abdominis myocutaneous (flap)
TRUS	transrectal ultrasound
TSAT	transferrin saturation
TSE	transmissible spongiform encephalopathy
TSH	thyroid-stimulating hormone
TSST	toxic shock syndrome toxin
TT	thrombin time *or* total thyroidectomy *or* tuberculoid (leprosy)
TTE	transthoracic echocardiogram
TURB	transurethral resection of bladder
TURBT	transurethral resection of bladder tumour
TURP	transurethral resection of the prostate
TV	transvaginal
TVF	transversalis fascia
TXA2	thromboxane A2
U	unit
UADT	upper aerodigestive tract
U&Es	urea and electrolytes
UC	ulcerative colitis
UCL	ulnar collateral ligament
UFH	unfractionated heparin
UGI	upper gastrointestinal
UOS	upper oesophageal sphincter
USMLE	United States Medical Licensing Examination
USS	ultrasound scan
UTI	urinary tract infection
UV	ultraviolet
V	volt
VACTERL	vertebral defects/anorectal atresia/cardiac defects/ tracheo-oesophageal fistula ± (o)esophageal atresia/renal anomalies/limb defects
VAD	ventricular assist device
VAS	visual analogue scale
VATS	video-assisted thoracoscopic surgery
VCE	video capsule endoscopy
vCJD	variant Creutzfeldt–Jakob disease
VEGF	vascular endothelial growth factor
VF	ventricular fibrillation
VH	visible haematuria
VHL	von Hippel–Lindau (disease)
VIP	vasoactive intestinal polypeptide
VLAD	variable life-adjusted display
VMA	vanillylmandelic acid
V/Q	ventilation/perfusion (scan)
VRAM	vertical rectus abdominis musculocutaneous
VRE	vancomycin-resistant enterococci
VRS	verbal response score
VT	ventricular tachycardia
VTE	venous thromboembolism
vWF	von Willebrand factor
WBRT	whole breast radiotherapy
WCC	white cell count
WHO	World Health Organization
WLE	wide local excision
XDRTB	extensively drug-resistant tuberculosis
y	year
Zn	zinc

Chapter 1

Good surgical practice

Pearls of wisdom

Surgical strategy and the military metaphor

Surgeons, consciously and unconsciously, adopt a philosophy or strategy that governs how they make decisions and manage their patient.

A wise surgeon should have a clear strategy to achieve success before embarking on surgery. Schein (2016)[1] likened the surgeon to an army officer in a battle. The rules by which an officer goes can be very similar to the principles of management that a good surgeon will follow. A competent army officer will destroy the enemy before the enemy destroys him (save lives), spare his own men (reduce morbidity), save ammunition (use resources rationally), know his enemy (understand disease severity), know his men (appreciate the benefit–risk ratio of therapy), attack at 'soft' points (tailor management to disease and patient), not call air force support in a hand-to-hand battle (use of mind and hands first and avoidance of useless gimmicks or investigations), seek advice from comrades (consult colleagues and other specialties), avoid friendly fire (reduce iatrogenesis), and maintain high morale among his troops (show leadership and teamwork).

Choose well, cut well, get well

The adage 'choose well, cut well, get well'[2] encapsulates the requirements for successful surgery.

'**Choose well**' involves a thoughtful decision-making process. The American surgical teacher Frank Spencer stated that good surgery is 20–25% manual dexterity and 70–75% decision-making. A critical and reflective judgement incorporates anticipation and timely response to change throughout. Clinical decisions should be based on knowledge acquired from critical reading of the literature, learning from personal and others' experience, benefiting from multidisciplinary team (MDT) discussion, and customizing therapies on an individual patient.

'**Cut well**'—good surgeons need to know how to operate. It is a losing battle if the surgeon chooses well, but the operation is imperfectly or inappropriately performed. The patient must be prepared carefully and a safety checklist employed (➲ WHO checklist, p. 100). Cutting well is achieved if the surgeon continuously learns and develops his skills through watching assiduously his trainers and copying them, utilizing courses and simulation, attending courses, watching experts operating, respecting the planes and knowing the anatomy, and always reflecting on his practice and modifying it accordingly.

'**Get well**'—your commitment towards patients does not end with skin closure. Sir Berkeley Moynihan (1865–1936) said, '*The operation itself is but one incident, no doubt the most dramatic, yet still only one in the long series of events which must stretch between illness and recovery.*' Always document your operative findings and give clear post-operative instructions. Post-operative recovery will be enhanced by early mobilization, fluid and electrolyte balance, and meeting nutritional requirements.

No-lose philosophy

Pascal's 'no-lose philosophy'[3] originally related to belief in God. 'Pascal's wager' argues that if God does exist and we live our lives believing in Him, then everything is gained; if God does not exist, then at least nothing is lost.

Clinical implications

The 'no-lose philosophy' is occasionally applied in practice. When faced with a problem, a doctor may err on the side of 'no-lose' in his approach, either in management (ordering investigations) or in giving a prognosis to relatives.

Dangers of 'no-lose'

Application of the philosophy to all areas of medical practice carries considerable risks, especially when investigating patients (in order not to lose, a 'diagnosis' must be made). Such an approach can potentially increase patient anxiety and waste limited resources. (Note if you do multiple tests, one will be 'positive'.)

Advice: no decision is a decision

Beware of (invasive) tests which may be expensive, dangerous, or unnecessary; doing unnecessary tests where action is required can be dangerous, e.g. computerized tomography (CT) chest in a patient with penetrating chest trauma and hypotension. Are the risks of the procedure worth taking? Would you carry out the same investigation on yourself? The 'no-lose philosophy' has considerable potential for loss and it may adversely influence decision-making and prevent the sensible resolution of clinical and ethical problems. Note that it is not uncommon to have no diagnosis or an uncertain outcome; in these instances, effective, collaborative communication with the patient may be a better alternative to the no-lose approach.

Beware the patient with a label

Once a patient has been labelled with a diagnosis (particularly a prognostically less 'serious' one, e.g. irritable bowel syndrome), it becomes increasingly difficult to review the situation and think outside the box—a phenomenon knows as 'labelling'. New symptoms tend to be attributed to the diagnostic label and risk missing important clues that could lead to another (serious) pathology.

Example

A 56-year-old woman had a 'known' diverticular disease. She lived with this condition for 10 years and suffered mild to moderate flare-ups over the years. She went to her general practitioner (GP) because of increasing loose stool. The GP, who knows her well, reassured her and reiterated his previous advice about diverticular disease and the importance of having a high-fibre diet. Six months later, she presents again with fatiguability and weight loss. She is proven to have iron deficiency anaemia on blood tests. Urgent colonoscopy and CT imaging now identify a locally advanced bowel cancer with liver metastasis, deemed suitable only for palliative treatment.

Morals

'Red flag' symptoms in a patient with a label—if the doctor had a higher index of suspicion for the 'red flags' (prolonged change in bowel habit in a middle-aged person), the tumour may have been detected earlier and been potentially amenable to surgical resection and better prognosis.

Healthy cynicism—beware of chronic diseases, as they can cloud the clinical picture. Although previous diagnoses often provide a very useful clinical context, it is intellectual laziness to attribute everything to the 'label'. Clinicians should think laterally and adopt an attitude of healthy cynicism. This will be very rewarding for the clinician and their patient alike.

Beware the patient you do not like (unconscious bias)

When excessive bias is introduced, logical decision-making becomes almost impossible. One human trait is to moralize on the actions of the patient or to patronize them because of their station in life, speech, physical characteristics, etc. Lack of sleep and fatigue (a common junior surgeon's problem) may also introduce some bias or misjudgement.

Examples

1. It is 4 a.m. and your pager goes off. You are the surgical registrar on call. It has been a busy night and you need to present a long list of cases to Mr Grump in the morning handover. You see the call has come from the Accident and Emergency (A&E) department—your senior house officer (SHO) wishes you to see a young man with epigastric pain. Your room is miles from the A&E department and, what's more, it is raining. Even before you have got out of bed, you have decided that you do not want to admit this patient.
2. It is the New Year's Day post-take ward round. You are the registrar on call over the festive period. Your consultant has 'popped out' to visit friends. Although you have missed out on Christmas cheer, the ward is full of young men suffering from hangovers and alcoholic gastritis. On the post-take round, you do not devote your full attention to assessing the patients. You are astonished when one is readmitted later, collapsed. At laparotomy, you have to resect a large segment of ischaemic bowel as the result of a mesenteric infarction. The SHO reminds you his lactate was 4.

Morals

When on call, train yourself to become resigned to the time commitment. Establish a checklist for clinical examination; review all relevant results, and discuss them with your colleagues if possible. Devote your full attention to each patient with a 'fresh pair of eyes'. Resist at all times the temptation to self-pity and to moralize. That will detract from your clinical competence. Professionalism is a learnt skill which combines good surgical practice with unbiased decision-making. If you genuinely cannot cope because of fatigue, report it to your colleagues and your seniors. The system will not collapse without you, and most consultants are sympathetic.

The busy on-call and cognitive overload (too much on your plate)

Cognitive overload is a concept from learning theory. This is where the 'student' (in this case, you the doctor) gets too much information or too many tasks simultaneously, thereby leaving gaps in management or missing important patient developments. This is a common problem with shift-working patterns where multiple patient tasks may be handed over from the previous shift, whilst also receiving multiple new tasks/referrals from across the hospital. The key is to keep a clear record of tasks and referrals, and to review, prioritize, and delegate effectively. Take a few minutes 'time out' to make a list of jobs and prioritize your plan of action—consider which patients need to be reviewed most urgently and which jobs can be delegated safely or deferred. ❗ Ensure that you clearly communicate patient plans to your colleagues, especially nursing staff (they are the ones who will ensure

your plans are executed). Do not leave important tasks undone—once you start seeing a patient, ensure you see the whole treatment for that patient through to completion.

References

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2 Kirk RM (2013). Choose well, cut well, get well. In: Novell R, Baker DM, Goddard N (Eds). *Kirk's General Surgical Operations*, 6th edn. Elsevier Health Sciences, Oxford; pp. 1–6.
3 Galbraith S (1978). The 'no lose' philosophy in medicine. *J Med Ethics* **4**: 61–3.

Duties of a doctor

The duties of a doctor are outlined in the General Medical Council (GMC)'s *Good Medical Practice* (see Box 1.1). These have been adapted by the Royal College of Surgeons of England for surgical practice.[1]

Box 1.1 The four domains of Good Medical Practice

Knowledge, skills, and performance

- Make the care of your patient your first concern.
- Provide a good standard of practice and care:
  - Keep your professional knowledge and skills up-to-date.
  - Recognize and work within the limits of your competence.

Safety and quality

- Take prompt action if you think patient safety, dignity, or comfort is being compromised.
- Protect and promote the health of patients and the public.

Communication, partnership, and teamwork

- Treat patients as individuals and respect their dignity.
- Work in partnership with patients.
- Work with colleagues in ways that best serve patients' interests.

Maintaining trust

- Be honest and open, and act with integrity.
- Never discriminate unfairly against patients or colleagues.
- Never abuse your patients' trust in you or the public's trust in the profession.

Knowledge, skills and performance

Develop and maintain professional performance

Keep up-to-date with current clinical guidelines, undertaking 'continuing professional development (CPD)' and educational activities, including simulation training for new surgical procedures. Maintain a portfolio of procedures undertaken and clinical activity; participate in annual appraisal.

Apply knowledge and experience to practice

Good standards of clinical practice

Treat patients according to priority of clinical need and take full responsibility for patient management, including preoperative workup and postoperative care (➲ Principles of surgery, pp. 51–173).

Provide adequate time for consent (➲ Consent, pp. 14–7).

Carry out operations in a timely, safe, and competent manner, following clinical guidelines in the field.

Work within the limits of your competence; use the MDT and other clinicians (especially in complex cases). Ensure patients are cared for in an environment with the correct resources. Ensure regular patient review (ideally once every 24h by a consultant).

Emergency surgery

Take responsibility for patients admitted under your care from the emergency department and ensure you are able to respond promptly to emergency patients when on-call.

Ensure the risk of mortality and complications of emergency surgery is assessed (➲ Risk scoring, p. 150) and effectively communicated to the patient and their relatives. When this risk is deemed to be high, the consultant should be closely involved.

Emergency patients should be reviewed by a consultant ideally every 24h—more often if the patient is high risk.

Research and new techniques

Participate in, and understand the relevance of, research; be able to critically appraise published research and apply it to practice (➲ Critical appraisal, pp. 32–3).

When undertaking research, carry this out in partnership with patients, respecting their dignity and clinical circumstances. Ensure appropriate ethical approval, training, and licences are obtained (see Box 1.2).

Be open and transparent about sources of funding and conflicts of interest, when undertaking or publishing research findings.

New techniques should be in keeping with local clinical governance (➲ Clinical governance, pp. 24–5) and be carried out in the best interests of the patient.

Box 1.2 Key legislation in research

- **World Medical Association Declaration of Helsinki 1964/2013 and Good Clinical Practice (GCP):** outline the ethical framework for carrying out research on humans—in most institutions, participation in clinical research requires mandatory GCP training.
- **Research Ethics Committee (REC) or ethical review board:** all research requires assessment and approval to ensure any potential ethical issues have been considered.
- **Human Tissue Act 2004:** outlines the laws surrounding the 'removal, storage and use' of human tissue. Proper consent is paramount.
- **Animals (Scientific Procedures) Act 1986:** regulates the use of animals in scientific experiments and testing.

Record your work clearly, accurately, and legibly

Be fully versed in the use of electronic health records in your institution. Take part in mandatory training in information governance in your hospital and be compliant with the Data Protection Act 2018[2] regarding patient-identifiable information. Ensure all medical records are accurate, clear, legible, comprehensive, and contemporaneous and have the patient's identification details on every page. These include inpatient medical entries (see Box 1.3), operative notes (see Box 1.4), and discharge summaries.

Box 1.3 Medical entries—always include the following

- **Date and time** of entry (if writing retrospectively, this must be mentioned, including the date and time of the specific event).
- Name/grade of the **most senior surgeon** seeing the patient.
- **Nature of entry**, e.g. 'routine review', 'ward round', 'asked to see patient re: … '.
- **Signature/name, grade, contact number ± GMC number** of person making the entry.

Box 1.4 Operative notes

- Date and time.
- Elective or emergency procedure.
- Name of operating surgeon and assistant(s).
- Name of the anaesthetist.
- Name of operative procedure carried out.
- General anaesthesia (GA)/local anaesthesia (LA); patient position; antibiotic prophylaxis(if relevant).
- Incision.
- Operative diagnosis and findings.
- Problems/complications.
- Any extra procedure carried out and the reason it was performed.
- Identification of any prostheses or implanted materials used, including serial numbers.
- Details of closure technique.
- Estimated blood loss.
- Detailed post-operative instructions (including oral intake, antibiotics, and specific parameters to observe and record).
- Details of tissue removed, added, or altered.
- Signature and contact number, GMC number.

Safety and quality

Contribute to, and comply with, systems to protect patients

Comply with national and local standard guidelines for patient safety, particularly the 'Five Steps to Safer Surgery' (which incorporates the WHO surgical safety checklist, team brief, and correct site marking (➲ Principles of surgery, pp. 96–7, 100) and surgical site infection prevention.[3] Engage in, and undertake, quality improvement (➲ Quality improvement and audit, pp. 22–3).

Respond to risks to safety

Your primary accountability is to your patient—support a culture of openness, honesty, and objectivity. Raise concerns at the earliest opportunity when you believe that patient safety may be put in jeopardy for any reason, including fitness of a colleague (start with immediate superiors and then escalate if you are still not satisfied).

Protect patients and colleagues from any risk posed by your health

Do not work in any state that might impair your judgement or jeopardize patient safety, including precautions against transmission of blood-borne viruses and working under undue fatigue or under the influence of drugs or alcohol.

Communication, partnership, and teamwork

Communicate effectively

(➲ Communication skills, pp. 12–3.)

Work collaboratively with colleagues to maintain and improve patient care

Individual behaviour

Be accessible and approachable; support colleagues in trouble, but also challenge counterproductive behaviour constructively. Encourage and seek feedback from colleagues about your performance and be willing to reflect on this feedback. Participate in appraisal.

Teamworking Work effectively and amicably with colleagues in the multidisciplinary team (MDT); be on time and share decision-making. Ensure that members of the team understand each other's roles and responsibilities. Always respond to calls for help from colleagues.

Teaching, training, supporting, and assessing

As a student or trainee Take responsibility for your training and be proactive in seeking opportunities that will help you meet the requirements of your syllabus. Maintain accurate and up-to-date records relating to your training. Recognize your limitations and communicate with your seniors when dealing with a situation with which you are unfamiliar.

As a trainer Support those under your supervision to carry out learning and development activities, and ensure that you provide appropriate supervision for junior colleagues.

Continuity and coordination of care

Ensure that the patient knows the name of the consultant responsible for their care. Ensure that there is a formal handover for the patients under your care and that sufficient time is set aside for handover. Take responsibility for patients under the care of an absent colleague, even if formal arrangements have not been made. Continue to see your patients when they are in the intensive care (ICU) or high dependency unit (HDU).

Establish and maintain partnership with patients

Consent and preoperative checks

(➲ Consent, pp. 14–7; ➲ Getting the patient to theatre, pp. 96–7.)

Take time to discuss and establish the patient's mental capacity and the views held by patients (and relatives) about their care, particularly about blood transfusion and, where relevant, 'ceilings of care', i.e. instructions to withhold or withdraw treatment such as cardiopulmonary resuscitation (CPR). These should be carefully documented and readily visible in the patients' care record.

Patient feedback and duty of candour

Proactively seek and reflect on feedback from patients and use this to improve your practice and that of your team. Treat complaints from patients or their supporters with courtesy and respect, and respond to complaints promptly, openly, and honestly. When any significant harm or error occurs, ensure the patient is made aware of the problem, has the opportunity to discuss, and is informed of ongoing investigations/outcomes; report all such incidents through the relevant incident reporting mechanism in your hospital.

Maintaining trust

Show respect for patients

Treat patients as individuals, considering their wishes. Support and assist with any request for a second opinion. Ensure that a patient's dignity is respected at all times and obtain verbal consent before carrying out any clinical examination.

Treat patients and colleagues fairly and without discrimination

Ensure that decisions about patient treatment are based on clinical need and not on social, managerial, or financial factors.

Act with honesty and integrity

Surgeons must demonstrate probity in all aspects of practice, ensuring they provide information about their knowledge or expertise truthfully and when asked to provide references on a colleague's performance. If working in the private sector, ensure transparency in dealings with patients and ensure you have the correct indemnity/insurance cover. Declare any commercial involvement/incentives or conflict of interest, and make sure these do not influence treatment options for patients.

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Communication skills

Communicating with patients and relatives

When

- During any patient contact, i.e. on ward rounds, during clinical examinations and procedures, and in outpatient clinics; when the results of treatments are known and management changes.

Where

► Maintain the patient's privacy. This is particularly important on an open ward. Knock on doors and close them after you. Draw the curtains around the bed. Ask a nurse to accompany you, particularly if you are explaining something complex or breaking bad news—they will have to answer the patients' and relatives' questions when you have left the ward or clinic room.

How

- Preparation—get your facts right. Are you giving the right diagnosis to the right patient? Are you equipped to consent for the procedure?
- Rapport—sit at the same level as the person to whom you are talking; maintain appropriate eye contact, and introduce yourself.
- Find out what the patient knows and what they are expecting.
- Listen. The patient's own knowledge, state of mind, and ability to grasp concepts will dictate both how and how much you explain.
- Tell the truth. Know your facts; be sensitive to what the patient may not want to know at this stage and do not lie.
- Avoid jargon. 'Chronic' may simply mean 'long-standing' to you; to the patient, it may mean 'severe'.
- Avoid vague terms. Try to describe risk quantitatively, 'a one in a hundred chance', rather than qualitatively, 'a small risk'.
- Check that the patient understands. Do not assume that they do.
- Help the patient to remember—use information sheets; draw diagrams, and write instructions down.
- Maintain a professional relationship—never allow your personal likes, dislikes, and prejudices to hamper your clinical skills.

Breaking bad news

- Is there a relative or friend whom the patient might wish to have with them who may be a source of emotional support, as well as being better able to retain information? Know what options, if any, are available—if a cancer is inoperable, is chemotherapy planned? If an operation is cancelled, when is the next date? Do not be afraid to stop to allow the patient time to gather their thoughts and emotions and recommence at a later time. Do not mistake numbness for calm acceptance, and try not to take anger personally unless the bad news is actually your fault.

Communicating with nurses

- Introduce yourself on arrival to the staff nurse in charge. Establish early on which nurses are experienced. The help you get from them will be different from the questions you get from others.
- In theatre, scrub nurses are not the enemy. Your inexperience is. Try to remember all their names as they will remember yours.

- Do ward work efficiently. Recognize how important it is for the smooth running of the ward that your ward rounds, note-keeping, prescriptions, and discharge letters are timely and accurate.
- Let the nurses know when you are going for lunch, teaching, or sleep—if they can discuss problems now, it will save you being paged later.
- Do an evening ward round to check on problem patients and drug requirements—your sleep is less likely to be interrupted.

Communication with hospital doctors

- Do not refer without first discussing with your consultant or registrar.
- When making requests for clinical consultations, write a concise, but clear, letter in the notes to the appropriate clinician.
- When asked to see a patient, go the same day; write your opinion in the case notes, stating clearly what you recommend, and always discuss it with the seniors on your own firm.
- If a preoperative patient is complex or has significant comorbidity, contact the appropriate anaesthetist. They will help you ensure that the patient is adequately prepared for surgery.

Communication with general practitioners

The GP has usually looked after your patient for years, and however inspired your diagnostic or operating skills, they will be there to sort out all the complications that are hidden from you once the patient is discharged. They often know your consultant well. So think!

- *Telephone the GP*: in the case of the death of a patient, if you unexpectedly admit a patient, or to help with a difficult discharge.
- *Write useful, clear discharge summaries*. Include what you would want to know if you were going to have to wait up to a few weeks for the typed discharge letter to arrive—at an absolute minimum, the date and name of the operation, post-operative complications, and plan.
- *Keep clinic letters clear and concise*.

Radiology and laboratory colleagues

- Know exactly which *specific* questions you wish to answer and how the investigation will change your management. If there is doubt about the correct investigation, discuss this first. Complete request forms correctly, and include as much relevant clinical data (this will improve reporting and interpretation of findings). Always include your contact details.

Administration

- Introduce yourself to your consultant's secretary/rota coordinator early; find out how they like things run, and run things their way—they will usually have more than typing-input on your reference!
- Produce General Medical Council (GMC), defence union, occupational health, holiday, and study leave paperwork with good grace. They are mostly legal requirements and being rude will not change that.

Consent

Informed consent for a surgical procedure is not merely the signing of a form; it is the process of providing information to enable the patient to make an informed decision.[1, 2]

Legal aspects

The patient's right to autonomy must be respected, even if their decision results in harm or death. This right is protected by law.

- A doctor performing a procedure on a patient without their consent can be found guilty of *battery*.
- A doctor who has failed to give the patient adequate information can be found guilty of *negligence* (adequate information means that which would cause a reasonable patient to decline surgery—in practice, this includes common or serious risks; see below Informed consent).[2]
- No adult in the UK can legally consent to surgery on behalf of another adult who has capacity.
- Where a patient lacks capacity to consent, act in their *best interests*, taking into account their likely wishes—it is important to identify if they have an Advance Directive (aka 'living will') or appointed a *Lasting Power of Attorney (LPA)*, i.e. a person who is legally appointed to make decisions about the patient's care. Involve relatives and others to determine the likely wishes of the patient (but note that the wishes conveyed by relatives are not legally binding without a valid LPA).

Modes of consent

Implied consent The patient is presumed to consent to minor procedures, e.g. X-rays, phlebotomy, by cooperating with ward procedures.

Express written consent This should be obtained for all patients undergoing procedures involving an anaesthetic, complex treatments with significant risks and side effects, or as part of research.

► *Written consent is not legal proof that adequate consent was obtained.*

Express verbal consent This should be obtained when it is not possible to get written consent or for simple procedures with minimal risk, witnessed by an independent health care professional and documented in the notes accordingly.

Informed consent

There are five aspects that the patient must understand to give informed consent. ► You, the doctor obtaining consent, must be familiar with each of these; if not, ask a senior before seeking consent.

- *The reason for carrying out the procedure*. The patient needs to understand the nature of their illness and its prognosis.
- *What the procedure involves*. For example, where and how long is the scar? What is being removed or what prosthesis implanted? Will there be drains?
- The risks of the procedure. Specific risks, e.g. stoma, limb dysfunction, and general risks, e.g. anaesthetic risks, deep vein thrombosis (DVT).

- The benefits of the procedure. Improvement in symptoms or prognosis or purely diagnostic.
- Alternatives. Including conservative treatment, with their advantages and disadvantages.

Obtaining consent—the process

1. First, establish mental capacity (see Box 1.5 and Figure 1.1). And if necessary, seek the help of your hospital's independent mental capacity advocate (IMCA), discuss with relatives, etc. (note that an LPA must have proven documentation). Involve your consultant and document communications carefully.
2. Allow time—ideally this should happen prior to the actual date of surgery and giving the patient sufficient time to reflect.
3. Informed consent—all five aspects of informed consent (➲ Informed consent, pp. 14–5) should be covered. In addition, tell the patient who

Box 1.5 Mental Capacity Act (MCA) 2005

Five principles of mental capacity

Mental capacity refers to a person's ability to make their own decisions. Always start from the **assumption that the person has the capacity** to make the decision in question (*Principle 1*) and make every effort to encourage and **support the person to make the decision themselves** (*Principle 2*) (e.g. for the patient with learning disability, involve the learning disability nurse; for the patient with delirium or fluctuating capacity, involve their next-of-kin and choose a time of day where they are known to be lucid). An **unwise or eccentric decision** does not necessarily indicate a lack of capacity (*Principle 3*). Once you are clear that there is a lack of capacity, always act in the **best interests** of the patient (this requires trying to understand what their likely wishes might be) (*Principle 4*) and in doing so, choose the treatment option that would be **least restrictive** to the patient's rights/freedoms and likely wishes or consider if there is a need to act at all (*Principle 5*).

Assessing mental capacity—a two-stage functional test

- Stage 1. Is there an impairment or disturbance in the functioning of a person's mind or brain? If so:
- Stage 2. Is the impairment or disturbance sufficient that the person lacks the capacity to make a specific decision? (▶mental capacity is decision- and time-specific—it can change; therefore, do everything you can to assist capacity when making the decision).

The MCA says that a person is unable to make the decision if they cannot do one or more of the following four things:

- **Understand** information given to them.
- **Retain** that information long enough to be able to make the decision.
- **Weigh up** the information available to make the decision.
- **Communicate their decision**—this could be by talking, using sign language, or even simple muscle movements such as blinking an eye or squeezing a hand.

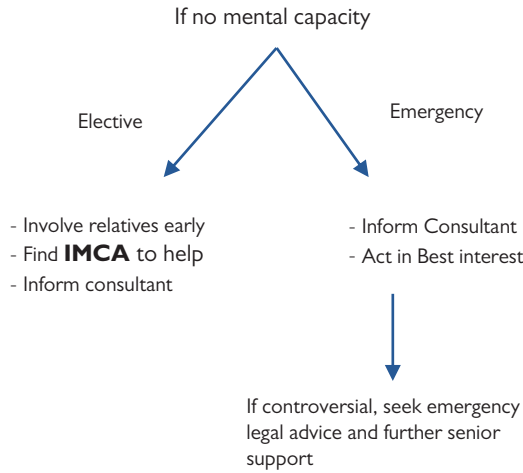

Fig. 1.1 Mental capacity.

will be doing the procedure and the post-operative/follow-up plan. For more complex procedures, use diagrams to explain and provide a written information sheet and advice on where they can obtain further information. Refer to national guidelines, e.g. National Institute for Health and Care Excellence (NICE), especially if your treatment is not in keeping with them.

4. Sign the consent form, allowing the patient to take a copy for reference and reflection, and record the discussion in the patient notes.
5. Consent must be obtained for taking photographs for teaching or publication and taking samples for research (these usually require separate consent) and it is good practice to gain agreement for students to attend the operation (if applicable).

Special considerations

Emergencies When consent cannot be obtained, you may provide emergency medical treatment, provided it is limited to what is needed to preserve life and in the best interests of the patient. However, you must respect any valid advance statements that you know about or that are drawn to your attention.

Advance statements/living wills Advance statements made by patients before losing the capacity of informed consent must be respected, provided the decision is applicable to the present circumstances and there is no reason to believe that they may have changed their mind. The known wishes of the patient should be taken into consideration if an advance statement is unavailable.

Mental capacity (see Box 1.5). Controversial and non-therapeutic treatments (e.g. sterilizations) require court approval if there is doubt about the validity of the LPA/advance decisions or expressed opposition to the proposed procedure.

Children

- *Giving consent*—over 16s are regarded as young adults and have capacity to decide; under 16s may give their consent for a procedure if they are judged to understand what is involved ('Gillick competence').
- *Refusing consent*—if a person <18y refuses a treatment that is deemed in their best interests, a person with parental responsibility (except in Scotland) or a court may authorize treatment. If the parents refuse treatment, you may seek a ruling from the court.
- *Emergency treatment*—may be instigated without consent, as for adults, if in the patient's best interests.

Pregnancy The right to autonomy applies equally to pregnant women, who have the right to refuse treatment intended to benefit the unborn child.

Cosmetic surgery Follow the requirements for consent set out by the Cosmetic Surgical Practice Working Party in Professional Standards for Cosmetic Practice (2013). For invasive cosmetic procedures, this includes a two-stage process, with a period of at least 2 weeks between the stages to allow the patient to reflect on the decision. It is important to identify the psychologically vulnerable patient and ensure that there is rapid and easy access to mental health services where relevant.

Language and communication barriers Use an interpreter where required (using the patient's relative can introduce incorrect information or bias).

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Death

Confirming death

There is no legal definition of death in the UK. It is generally regarded as the cessation of circulation and respiration. Clinically, there is:

- No respiratory effort, denoted by the absence of breath sounds on auscultation over 1min.
- Absence of a palpable pulse and heart sounds over 1min.
- No response to painful stimuli, e.g. sternal or supraorbital rub.
- Fixed, dilated pupils (beware drugs such as atropine).
- If there is doubt, perform an electrocardiogram (ECG).
- Hypothermia (core temperature <34°C) must have been corrected.

Brain death/brainstem death

The concept of brain death has arisen from advances in intensive therapy and the ability to maintain cardiac and respiratory function artificially in patients who have sustained severe, irreversible brain damage. Brain death is defined as the 'irreversible cessation of all functions of the entire brain, including the brainstem'.[1] This, alongside the traditional definition, is taken to equate to death in the UK, USA, Australia, and many other countries. In order to diagnose brain death, a number of strict criteria must be met:

- An identifiable cause for the brain death must be established (e.g. severe head injury/intracerebral bleed) and
- Other causes, including central nervous system (CNS) depressants, hypothermia, and metabolic and endocrine disturbances, need to be excluded, and
- The patient is unable to breathe spontaneously despite adequate arterial carbon dioxide tension ($PaCO_2$) >6.7kPa, and
- The following brainstem reflex tests, performed by the consultant in charge (or deputy of 5y registration) and another suitably experienced doctor, on two separate occasions, usually 24h apart:
  - Both pupils are fixed and unresponsive to light (oculomotor nerve).
  - Corneal reflexes are absent (trigeminal nerve).
  - Vestibulo-ocular reflexes are absent—absent eye movements when 20mL of ice-cold water is injected into each ear, with the tympanic membranes visualized beforehand (vestibulo-cochlear nerve).
  - Absent motor responses to painful stimuli in the distribution of the cranial nerves, in the absence of neuromuscular blockade—spinal cord injury (SCI) may ablate peripheral motor responses.
  - Absent of respiratory effort when disconnected from the ventilator despite $PaCO_2$ >6.7kPa—↑ in chronic obstructive pulmonary disease (COPD).
  - Absent gag and cough reflexes upon pharyngeal and endotracheal stimulation.

Coroners

It is always wise to discuss with the consultants involved if there may be reason to refer to the coroner's office. In-hospital deaths must be discussed with the coroner's officer if:

- Death has occurred during an operation.
- Death occurred before recovery from anaesthesia.

- More than 14 days have elapsed since the patient last saw a doctor.
- There is doubt about the cause of death.
- Death is thought to be suspicious (e.g. caused by overdoses of prescribed substances, medical error, suicide).

Certifying death

Documenting in the medical notes

If you are asked to 'certify' a patient, first confirm death (as described above):

- Document the date and time that death was pronounced.
- Document your examination.
- Document the causes of death as they will appear on the death certificate *if* these have been decided. ▶ If in doubt, always speak to the consultant.

The death certificate

This can be issued by anyone with full medical qualifications who looked after the patient during their last illness, or where referral to the coroner has been made and permission to issue the certificate has been granted.

- Write legibly. The record is retained by relatives—illegible or incomplete certificates may be rejected by the funeral director.
- Be specific—general terms, e.g. 'sepsis', may not be accepted.

Cremation forms

These forms vary slightly between regions, but certain rules always apply. There are two parts. The first is filled in by a doctor who attended the patient during the illness leading up to death, and the second by an independent clinician who has been fully registered for at least 5y.

- They should not be issued if the cause of death is not established.
- The issuing doctor must ensure that they have seen and identified the person after death, and that there are no radioactive implants or pacemakers present.

Post-mortems

- A coroner's post-mortem is required for suspicious deaths but is most commonly performed where the coroner's office has taken up a case where the cause of death is uncertain or may be related to a procedure. The consent of relatives is not necessary to proceed.
- A hospital post-mortem may be carried out with the consent of relatives to investigate other deaths. In 60% of post-mortems in one series, new diagnoses that would have substantially changed management were found—they are a vital part of audit.

Reference

1 A Code of Practice for the Diagnosis and Confirmation of Death. Academy of Medical Royal Colleges (UK) 2008.

End-of-life issues

Do not resuscitate (DNR) orders

A DNR order should be considered when the frailty, comorbidity (e.g. inoperable or disseminated malignancy, multiple organ failure), maximal medical treatment, and/or advanced age of a patient mean that any attempt at CPR in the event of a cardiac or respiratory arrest will be futile. DNR decisions should be reached on a case-by-case basis—a blanket 'do not resuscitate' policy based on a specific patient group is unacceptable. An 84y-old patient who was an appropriate candidate for cardiac surgery is an appropriate candidate for CPR post-operatively, whereas a 72y-old patient undergoing palliative care for end-stage hepatorenal failure is probably not.

- Never make a DNR decision without discussing it with a consultant.
- Patients and, where appropriate, their relatives must be involved.
- Document the clinical reasons for the DNR order and state explicitly whether 'full active medical management' is to be continued and other 'ceilings of care' (e.g. ward-based care only, not for invasive ventilation—ask the intensive care team for an opinion if necessary).
- Complete the appropriate documentation and review process.
- Make sure the nursing staff are fully informed.

Euthanasia

Euthanasia is the painless termination of life at the request of the patient concerned. In the UK, it is illegal to administer any drug to accelerate death, irrespective of how compassionate the motive may be. Withdrawing futile treatment is not euthanasia. Terminally ill people and the parents of terminally ill or severely disabled children may have several reasons for requesting euthanasia. Effective palliative care, counselling, and multidisciplinary support should be able to address most of these reasons, which include: pain, disability, disfigurement, depression, fear of being a burden, and being unable to cope.

Palliative care

Palliative care is surgical, medical, and nursing care aimed specifically to relieve the symptoms associated with terminal conditions when curative treatment is not feasible. ▶ Refer early—palliative care specialists will advise and provide support for: control of physical symptoms (pain, anorexia, nausea and vomiting, confusion, dysphagia, dyspnoea, incontinence); the psychological aspects of terminal illness (fear, stress, depression); and bereavement and support for relatives. They will also help coordinate the transfer or discharge of the patient to an appropriate setting.

Suicide

The suicide rate in the UK is currently ~10 per 100 000.

Patients at risk

- The recently bereaved; cancer patients (5× ↑ risk); men over 55y with oral cancer and a history of alcohol abuse; women of any age suffering from gynaecological or breast cancer (in both of these latter groups, the treatment of the disease involves *disfigurement and a change of body image*.)

Action

- *Preoperatively*: patients about to undergo disfiguring surgery for any reason should be counselled carefully in the period after confirmation of the diagnosis. Doctors should discuss all treatment options and implications clearly. The support of a **mastectomy counsellor** or **stoma therapist** is invaluable.
- *Post-operatively*: look for symptoms of depression; do not discontinue antidepressant medication; ensure that arrangements for discharge include community nursing support and that the GP is aware of the patient's mental state.

Organ donation

- When brain death is established, organ donation should be considered for all patients who are under 75y of age with no history of malignant disease or major untreated sepsis.
- All donors should be tested for human immunodeficiency virus (HIV), hepatitis B and C, herpes simplex virus (HSV), and cytomegalovirus (CMV).
- Organ donation is usually coordinated by regional transplant teams.
- The body should be identified and the next-of-kin contacted. If, despite reasonable attempts, the identity of the corpse or next-of-kin remains unknown, the body becomes the property of the health authority.
- If a donor card is present, it is reasonable to assume that the deceased wished to donate their organs and the transplant team can proceed. If relatives are identified and do not wish organ donation to proceed, even though there is a donor card, their wishes must be respected. Relatives should be asked to act as agents in expressing what they believe to be the wishes of the patient.
- The person seeking permission may be the consultant in charge, but on occasion, a senior staff nurse, chaplain (or other religious figure), or the family GP may be more appropriate.
- In the case of accidental deaths, the coroner's permission should be sought before proceeding.

Quality improvement and audit

What is quality improvement?

Quality improvement is the wider process of improving health care provision (see Box 1.6). 'Quality' has been defined along different lines, e.g. by the UK National Health Service (NHS) as 'Safe, Effective, Caring, Responsive and Well-led' care and by the Institute for Healthcare Improvement (USA) as 'Safe, Effective, Patient-centred, Timely, Efficient and Equitable' health care.[1]

Box 1.6 Quality improvement activities

- **Audit/peer review** to recognize 'outliers' in care.
- **Morbidity and mortality** meetings to discuss individual cases.
- **Incident reporting** to highlight errors in care.
- Compare with, and contribute activity data to, **national databases**, e.g. National Confidential Enquiry into Patient Outcome and Death (NCEPOD), National Emergency Laparotomy Database (NELA), National Vascular Database (NVD).

What is audit?

Audit is one quality improvement process that systematically reviews specific aspects of care against explicit criteria. Where indicated, changes are implemented and further monitoring is used to confirm improvement in health care delivery.[2]

Why do it?

Clinical audit is currently seen as the most effective way of assessing routine health care delivery and the basis of improving outcomes. All hospital doctors are required to fully participate in clinical audit.[1]

How to do it

Audit of outcome or process can be divided into five stages, which form the 'audit loop':[2]

1. *Preparation* Choose a topic and define the purpose of the audit. Keep the scope limited at first—'solve the solvable'. One option is to identify a potential problem for which there is good evidence to inform standards and that may be amenable to change. NICE stresses the importance of identifying skills and resources to carry out the audit.
2. *Select criteria* Audit can assess process or outcome. Define the patients to be included. Criteria to assess performance should be derived from available evidence, e.g. trials, systematic reviews, society guidelines. Benchmarking prevents unrealistically high or low targets.
3. *Measure performanc*e This is about collecting data. Identify patients or episodes from several sources to avoid missing cases. Electronic information systems and training dedicated audit personnel can improve data collection.
4. *Make improvements* Identify local barriers to change, and develop a practical implementation plan, which should involve several interventions (practice guidelines, education, and training).
5. *Sustain improvements (re-audit)* Repeating the audit to assess improvements is also called closing the audit loop. Alternatives such as critical incident review may be effective.

Measuring surgical performance and published surgeon outcomes

Rationale Surgical outcomes for cardiac surgery were first published in 2004. This followed the Kennedy inquiry into paediatric cardiac deaths at the Bristol Royal Infirmary. Hospital and consultants' outcome data are available online for the public to access. It should be noted that mortality data alone may not be a true reflection of the surgeon's quality and other factors should be considered.

Risk stratification Outcome measures should incorporate a system that accounts for differences in case mix, i.e. risk stratification, so that surgeons who operate on sicker or more complicated patients are not unfairly penalized, e.g. *EuroSCORE* and *Parsonnet* scores for predicting operative mortality in cardiac surgical patients, *POSSUM* (➲ Risk scoring, p. 150).

Presenting results The aim of presenting performance data is to distinguish between normal variation between surgeons or institutions and significant divergence. There are three main ways of doing this:

- Average outcome over a given time frame. *League tables* of surgical mortality or other complications—the data may be crude or risk-stratified; *survival plots* which may also be crude or risk-stratified; *standardised mortality ratio* plots.
- Volume and outcome control charts. *Funnel plots* (see Fig. 1.2); *spectrum plots*.
- Performance trends over time. Cumulative sum (*CUSUM*) charts; variable life-adjusted display (*VLAD*) charts; risk-adjusted CUSUM charts.

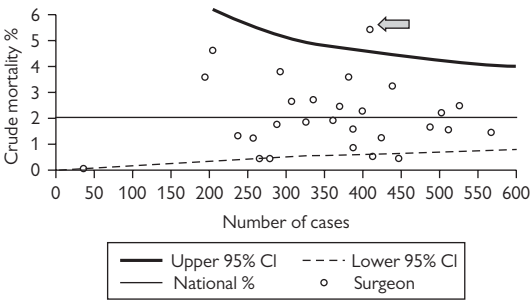

Fig. 1.2 Funnel plot of mortality data for 50 cardiac surgeons. The arrow marks an outlier with mortality outside 95% confidence interval (CI).

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2 Benjamin A (2008). Audit: how to do it in practice. *BMJ* **336**: 1241–5.

Clinical governance

Clinical governance is a quality assurance process through which a health service is accountable for maintaining and improving the quality of care.[1] Doctors have always been accountable for maintaining high quality care—clinical governance formalizes this. In practice, it involves setting standards, performance monitoring, and reporting errors and is commonly said to be held up by 'seven pillars' (see Box 1.7).

Box 1.7 The seven pillars of clinical governance

Clinical effectiveness
Audit
Risk management
Education and training
Patient and public involvement
Using information and information technology (IT)
Staffing and staff management

Setting standards

In addition to conventional clinical evidence and guidelines, the following organizations have a responsibility for setting standards in health care.

National Institute for Health and Care Excellence

The government organization responsible for publishing evidence-based guidance for the treatment of conditions and for evaluating the effectiveness of surgical interventions. Local authorities are obliged to fund interventions recommended by NICE, but NICE guidance does not overrule individual clinical decision-making.

General Medical Council

The body responsible for licensing, regulation and training of doctors, with overall responsibility for doctors' fitness to practise.

Performance monitoring

Care Quality Commission (CQC)

The independent regulator of all health and social care services in England. This body monitors, inspects and regulates care in hospitals, dentists, ambulances and the community.

National Confidential Enquiry into Patient Outcome and Death (NCEPOD)

An independent organization[2] which first studied the causes of all perioperative mortality, publishing its first report in 1990. This led to significant changes in surgical practice, such as the current practice of restricting night-time operating to only lifesaving and limb-saving operations and closer involvement of consultants in emergency operating. It now collects data on all areas of medical practice, by means of similar confidential questionnaires, auditing the outcomes of patients from multiple hospitals across the country.

Audit (➲ Quality improvement and audit, pp. 22–3.)

Revalidation The purpose of revalidation is to monitor individual doctors' performance and create public confidence that all licensed doctors are up-to-date and fit to practise. All doctors are required to undergo regular appraisal.

Medical error and reporting systems

Inspired by the airline industry, it has been shown that systems to detect and report errors in health care are crucial; organizations with high levels of incident reporting are more likely to have better governance (fewer medical errors and medical negligence claims). It is estimated that ~20 000 deaths in the UK and over 100 000 deaths in the USA annually are attributable to preventable medical errors. This has led to a greater focus on systems to improve patient safety.[3]

Critical incident reporting, serious incidents, and never events Health care workers are obliged to report incidents perceived to have exposed patients or staff to actual or potential risk, using a standard reporting system.

These include *serious incidents (SIs)* defined as incidents leading to permanent harm to patients or others. SIs should be discussed within the department and changes made to prevent them from recurring. *Never events* are SIs that should never occur if a health care system is following standard safety processes (e.g. wrong site surgery, wrong prosthesis implanted, retained foreign body post-procedure).[4]

Complaints (➲ Complaints, p. 26.)

Whistle-blowing Hospitals are required to enable individual staff members to report problems, and mechanisms should exist to investigate and act on such claims and to protect the whistle-blower.

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4 NHS Improvement (2018). *Never Events List 2018*. Available at: https://www.england.nhs.uk/publication/never-events/

The patient as a 'client'

There is increasing emphasis in many health care systems on the concept of the 'client' and patient experience. Clinicians do not always embrace this metaphor, which is felt by some to float the nature of the doctor–patient relationship from caring to commodification. Proponents for this believe that the term 'patient' connotes passivity, whereas a 'client' is closer to a customer, with more rights and powers, and demands good 'value for money'. In the context of a patient-centred approach, good customer care goes hand in hand with quality clinical care. Several quality indicators have evolved to assess patient care (surveys, online feedback, and complaints) and be more transparent (published outcomes for hospitals and consultants).

Surveys and feedback

Patient feedback is used to improve the quality of service provided. It is a tool to improve responsiveness to the expressed patient needs. Patients predominantly develop their impressions of the quality of care based on subjective judgements, rather than on clinical outcomes.[1] Real-time patient feedback must be a normal part of patient care in the NHS (e.g. the 'iWantGreatCare' website). From 2013, NHS Trusts in England were obliged to conduct surveys to gather views from patients within 48h of discharge.

Complaints

The Patient Advice and Liaison Service (PALS) is a front-line function to resolve patient concerns before they escalate, and acts to liaise with clinicians on behalf of patients. It has to be stressed that dealing with complaints is not merely a 'customer service' exercise like that of other industries. The issue cannot be settled by a refund or replacing faulty goods, and a complaint should never be ignored. Most complaints result from miscommunication. Acknowledgement and an apology does not mean necessarily admitting to liability and can prevent escalation. A response to a complaint should include what happened and what actions will be taken to prevent this from happening again. Unresolved complaints are referred to the Health Ombudsman.

Published hospital and consultants' outcomes

(➲ Quality improvement and audit, pp. 22–3.) It is widely held that every patient should know that their surgeon's outcomes are within acceptable limits. The purpose of the published survival rates is to aim for greater transparency and informed patient decision-making. The corollary to this is that published data can lead to a more risk-averse approach among surgeons, with a tendency to avoid treating patients deemed higher risk.

Reference

1 Lee F (2004). *If Disney Ran Your Hospital: 9½ Things You Would Do Differently*. Second River Healthcare Press, Bozeman, MT.

Evidence-based surgery

Summarizing simple data

This pattern of results (see Table 1.1) is called a normal or Gaussian distribution—the curve is a symmetrical, bell-shaped curve. Height, weight, age, serum sodium, and blood pressure (BP) are other examples of normally distributed data.

Table 1.1 Preoperative Hb in 100 patients

Hb (g/dL)	Number of patients	Hb (g/dL)	Number of patients
7–7.9	1	11–11.9	36
8–8.9	3	12–12.9	9
9–9.9	9	13–13.9	4
10–10.9	37	14–14.9	2

- The mean is the same as the 'average'—add up every result and divide by the number of results. The mean haemoglobin (Hb) here is 11.1g/dL.
- The standard deviation (SD) is a measure of how spread out the values are (result – mean = its deviation).
- SD = √[(sum of deviations2/(sample size – 1)]. Here SD = 1.6g/dL. With normally distributed data, the mean ± 1 SD includes 68% of observations; ± 2 SD includes 95%; and ± 3 SD includes 99%.

This pattern of results (see Table 1.2) is called a skewed distribution. Post-operative blood loss, length of stay, and survival all show skewed distributions. ▶ Do not use the mean and SD to summarize skewed data.

- The mean blood requirement, which is skewed to 8U of blood because of one outlier (*), is useful for planning budgets.
- The best summary statistic for skewed data is the median (2U of blood), which is the value exactly halfway through the sample.
- The interquartile range is what the middle 50% of observations were (1–2U here) and should be used, instead of SD, when summarizing skewed data.

Table 1.2 Post-operative blood transfusions in 100 patients

Units of blood	Number of patients	Units of blood	Number of patients
0	1	4	5
1	34	5–10	1
2	41	10–20	0
3	17	20–30	1*

* Outlier.

Tests

Table 1.3 Sensitivity

	Disease present	No disease
Test is positive	a	b
Test is negative	c	d

Sensitivity [a/(a + c)] A measure of how good the test is at positively identifying a condition (>98% is very sensitive). If a very sensitive test is negative, it rules the condition out ("*Sn.out*").

Specificity [d/(b + d)] A measure of how good the test is at correctly identifying a negative result (>98% is very specific). If a very specific test is negative, it rules the condition in ("*Sp.in*").

Likelihood ratio This is the chance that a person testing positive has the disease divided by the chance that a person testing positive does not have the disease, or sensitivity/(1 – specificity). A likelihood ratio >10 is large and represents an almost conclusive increase in the likelihood of disease; <0.1 is an almost conclusive decrease, and 1 signifies no change.

Treatments and hazards

Table 1.4 Risk assessment

	Outcome event	No outcome event
Exposed	a	b
Unexposed (control)	c	d

Absolute risk reduction (ARR) and relative risk reduction (RRR) Difference in event rate between the exposed group and the control group.

ARR [a/(a + b) – c/(c + d)] This is the raw difference in the event rate between the exposed group and the control group.

RRR [ARR/(c/c + d)] ARR divided by the control event rate—a measure of the difference in the event rate as a proportion of the control.

Number needed to treat (NNT) (1/ARR) The number of people who must be treated to prevent one event.

Risk ratio or relative risk (RR) [a/(a + b)/c/(c + d)] Event rate in the exposed group expressed as a proportion of that in the control group (i.e. event rate in the exposed group divided by event rate in the control group). Used in randomized controlled trials (RCTs) and cohort studies. It is not affected by the prevalence of a disease.

Odds ratio (OR) [(a/c)/(b/d)]
Looks at the association between two variables—in this case, exposure versus outcome event. An OR of 1 is neutral and means exposure does not affect the likelihood of an outcome event; an OR >1 means the exposed group is more likely to have an outcome event; an OR <1 means an exposed group is less likely to have an outcome event. ORs are less intuitive than RRs, but they are used because they are usually larger and mathematically versatile; they are always used in case-control studies and appear in meta-analyses of case-control studies; they are the basis of logistic regression analysis.

Statistical significance

- Studies are designed to disprove the null hypothesis that findings are due to chance.
- The p-value is the probability of a study rejecting the null hypothesis if it were true (a type I error), i.e. finding a difference where none exists.
- Statistical significance is commonly taken as a <1 in 20 chance of this happening, i.e. p <0.05.
- Power is the probability of detecting an association if one exists. Underpowered trials contain too few patients and may make type II errors, accepting the null hypothesis when it is false, i.e. finding no difference where one does exist.
- 95% confidence interval *(CI)*, calculated from the sample mean and SD, gives a predicted range in which the actual population mean is expected to lie with 95% confidence (i.e. if one were to repeat the same experiment an infinite number of times, 95% of the mean values would lie in this range).

Other useful terms

Censored data Essentially incomplete data, usually due to variable lengths of follow-up. Common in surgical studies because: (1) some patients will have been lost to follow-up; and (2) patients will have shorter follow-up where they had operations more recently.

Actuarial and Kaplan–Meier survival Two methods used to calculate the percentage of study patients who survive a specified time after an operation when a study provides censored data.

Survival curves Usually not curves. A linear graph, with percentage survival (or freedom from a complication) on the y-axis and time on the -axis, which drops as each study patient dies (or gets the complication). If there are thousands of patients in the study, the curve is smooth. If there are very few, it is possible to see individual deaths/events as steps in the graph. Ideally, these graphs should have *CIs*.

Confidence intervals These reflect the precision of the study results. Narrow CIs are better than wide ones because the CI provides a range of values for the mean or other value that has a specified probability (usually 95%) of containing the true value for the entire population from which the study patients were recruited. Always look for CIs; they give you a 'best case and worst case' snapshot.

Regression analysis Essentially looking back from a group of patients with a known outcome (e.g. dead/alive) to see whether there were any predictors, e.g. age, recent myocardial infarction (MI). Univariate analysis looks at single variables, in turn. Multivariate analysis looks at a group of variables together; it is used to identify independent risk factors for an outcome. For example, age may be found to be a risk factor for post-operative death in univariate analysis, but that is because elderly patients are more likely to have other risk factors for post-operative death (e.g. recent MI). If age is not found to be an independent risk factor in multivariate analysis, it suggests that elderly people without other risk factors (e.g. recent MI) are not at higher risk of post-operative death.

Critical appraisal

Types of study

Studies appraising treatments can take several forms.

Randomized controlled trial Prospective study in which participants are allocated to control or treatment groups on a random basis. Gold standard for assessing treatment efficacy, but time-consuming and expensive to run.

Cohort study Prospective study in which two cohorts of patients are identified, one of which was exposed to the treatment (or other factor) and one is the control group. They are followed over time to see the outcome. Logistically easier than RCT, but still requires long follow-up and large numbers (expensive) for meaningful results. Good for looking at prognosis, but prone to bias or false associations if inadequately powered.

Case control study Retrospective study in which patients with the outcome of interest are identified and paired with patients without the outcome of interest, and the exposure rates are compared. Cheapest and quickest way of looking for causation. More prone to bias most commonly when patients are misclassified as cases or controls.

Case series A collection of anecdotes or case reports (do not provide objective data).

Systematic review Differs from the traditional literature review by applying systematic criteria and reproducible methods to retrieve and appraise the literature to answer a specific question. Large amounts of data are summarized and conclusions are more accurate.

Meta-analysis A mathematical synthesis of the results of two or more 1° studies, increasing the statistical significance of positive overall results. However, it loses the demonstration of local effects.

Levels of evidence

Studies of treatment/hazard can be arranged in order of decreasing statistical robustness.

- Level 1a. Systematic review of >1 RCT.
- Level 1b. High-quality RCT with narrow CIs.
- Level 1c. All-or-none case series (either all patients died before treatment became available, but some now survive with the treatment or some used to die, but now with treatment, all survive).
- Level 2a. Systematic review with homogeneity of cohort studies.
- Level 2b. Cohort study or low-quality RCT.
- Level 2c. 'Outcomes' research.
- Level 3a. Systematic review with homogeneity of case control studies.
- Level 3b. Individual case control study.
- Level 4. Case series and poor-quality cohort and case control studies.
- Level 5. Expert opinion without explicit critical appraisal or based on physiology, bench research, or first principles.

How to appraise a paper

Answer these questions systematically. This information should all be stated explicitly within the manuscript.

How relevant is the paper?

- Does the paper address a clearly focused, important, and answerable clinical question that is relevant to my patients?

How valid are the findings?

- Was the paper published in an independent peer-reviewed journal?
- Does the paper define the condition to be treated, the patients to be included, the interventions, and the outcomes?
- Was a power calculation performed and is the power adequate?
- Were all clinically relevant outcomes reported?
- Was follow-up adequate?
- Were all patients accounted for at the end of the study?
- Were the appropriate study type and design appropriate?
- Were the statistical methods described and were they appropriate?
- Were the sources of error discussed?

Systematic reviews

- Is the clinical question clearly defined and an acceptable basis for including or excluding papers?
- Was the literature search thorough and were other potentially important sources explored?
- Were trials appropriately included and excluded?
- Was the methodological quality assessed and trials appropriately weighted?

Randomized controlled trials

- Were patients properly randomized?
- Were patients treated equally, apart from the intervention being studied?
- Was analysis on an intention-to-treat basis?
- Are CIs narrow and not overlapping?

Case control studies

- Were patients correctly classified as case or control?
- Were all patients accounted for at the end of the study?

How important are the results?

- Were the results statistically significant?
- Were the results expressed in terms of NNT and are they clinically important?

How applicable are the findings?

- Were the study patients similar to mine?
- Is the treatment feasible within my practice? Is information on safety, tolerability, efficacy, and price presented?

Non-technical skills for surgeons

It is now widely recognized that non-technical skills are as important for patient outcome as clinical expertise and technical skills. About two-thirds of serious medical errors are associated with human factors such as miscommunication.

There are four skill categories and 12 elements that make up the skills taxonomy (see Box 1.8). A non-technical skills for surgeons (NOTSS) rating scale is used to assess these skills. It utilizes structured observations, ratings, and feedback in the operating theatre. This allows one to assess and develop these skills within the training environment.

Box 1.8 NOTSS skills

Situation awareness	Gathering information Understanding information Projecting and anticipating future state
Decision-making	Considering options Selecting and communicating options Implementing and reviewing decisions
Communication and teamwork	Exchanging information Establishing a shared understanding Coordinating team activities
Leadership	Setting and maintaining standards Supporting others Coping with pressure

Table 1.5 shows exemplar behaviours (good and poor), generated by consultant surgeons.[1]

Reference

1 Flin R, Youngson GG, Yule S (Eds) (2015). *Enhancing Surgical Performance: A Primer in Non-Technical Skills*. CRC Press, Boca Raton, FL.

Table 1.5 Examples of good and poor behaviours in NOTSS

	Good behaviour	Poor behaviour
Situation awareness	• Carries out preoperative checks of patient notes, including investigations and consent • Acts according to information gathered from previous investigation and operative findings • Plans operating list, taking into account potential delays due to surgical or anaesthetic challenges	• Arrives in theatre late or has to be called repeatedly • Does not ask for results until the last minute or not at all • Overlooks or ignores important results • Carries out overconfident manoeuvres without regard for what may go wrong • Does not discuss potential problems
Decision-making	• Recognizes and articulates problems • Initiates balanced discussion of options • Reaches a decision and clearly communicates it • Realizes if 'plan A' is not working and changes to 'plan B'	• No discussion of options • Does not solicit views of other team members • Fails to inform team of surgical plan • Continues with 'plan A' in face of predictably poor outcome or when there is evidence of a better alternative
Communication and teamwork	• Talks about the progress of the operation • Listens to concerns • Provides briefing and clarifies objectives and goals before commencing operation • Stops operating when asked to by anaesthetist or scrub nurse	• Fails to communicate concerns with others • Attempts to resolve problems alone • Makes no attempt to discuss problems and successes at end of operation • Does not ask anaesthetist if it is OK to start operation
Leadership	• Introduces self to new or unfamiliar members of theatre team • Modifies behaviour according to trainee needs • Remains calm under pressure	• Breaks theatre protocol • Engages in 'tunnel vision' approach to technical aspects of operation • 'Freezes' and displays inability to make decisions

Lessons from the COVID-19 pandemic

Coronaviruses are zoonoses (originating from animals) that cause primarily respiratory symptoms in humans. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)/coronavirus disease 2019 (COVID-19) emerged as a global pandemic when the World Health Organization (WHO) declared a public health emergency on 20 January 2020. This surpassed previous coronavirus epidemics [SARS-CoV in 2002; Middle East respiratory syndrome coronavirus (MERS-CoV) in 2012]. The majority of COVID-19 patients develop mild symptoms; however, more severe symptoms include pulmonary oedema, severe pneumonia, acute respiratory distress syndrome (ARDS), and sepsis. At-risk groups include the elderly, immunocompromised patients, and those with a body mass index (BMI) >40. It is estimated that the median incubation period is 5.1 days, with further symptomatic infections unlikely after 14 days of exposure without symptoms.[1] Serious complications and the scale of the disease put major strain on health services worldwide and significant compromises to surgical services occurred in the first wave. Some surgical services reported poorer outcomes in patients who presented with, or developed, COVID-19 in the perioperative period.[2,3]

The gold standard for COVID-19 screening is a pharyngeal swab reverse transcriptase polymerase chain reaction (RT-PCR), with a sensitivity of 71–98% and typically taking >24h for a result.[4] Rapid testing with lateral flow devices provides a faster alternative, typically within 30min, with high sensitivity and specificity.

A large number of vaccines have been developed, with the first approved for use in the UK in late 2020 being the Pfizer BioNTech mRNA vaccine and the Oxford AstraZeneca chimpanzee adenovirus-vectored vaccine.

Clinical prioritization

Due to the widespread disruption and pressure on beds, initially all elective surgery ceased in the National Health Service (NHS). The Federation of Surgical Specialty Associations (FSSA) classified cases according to novel COVID-19 pandemic criteria (Table 1.6). This stratification is useful for day-to-day surgical practice, especially with ongoing pressures on health services, and, as a junior doctor, may be useful to keep in mind.

The effect of delays on mortality—why targets matter

The COVID-19 pandemic has highlighted the adverse impact of delays to treatment in patients normally treated on an urgent pathway. Normally, cancer patients in the UK NHS are expected to be seen within 2 weeks of referral—called the '2-week wait (2WW) pathway'—and treatment initiated within 2 months. Other urgent conditions (e.g. critical limb ischaemia) have similar treatment targets.

As a result of the health care crisis from COVID-19, many operations for urgent life-limiting conditions were delayed. In some cases, private hospitals or designated 'COVID-19-free' areas were used to continue to provide surgical or cancer services. There was a balance of mitigating the risk of suffering morbidity or mortality from COVID-19 against the risk from the underlying disease. It was known that older patients (>80y) and those in high-risk groups had a high perioperative risk and poor outcome when treated in areas of high COVID-19 transmission. Having COVID-19 was associated with higher in-hospital mortality (25–36%) and higher risk of thrombotic events, including end-organ ischaemia in surgical patients.[2,3]

Table 1.6 FSSA prioritization system

Level	Category[a]	Examples
Priority 1a	Emergency—operation needed within 24h	Ruptured abdominal aortic aneurysm (AAA), laparotomy for peritonitis or trauma Ear, nose, and throat (ENT) bleeding or airway compromise Open fracture with neurovascular compromise
Priority 1b	Urgent—operation needed within 72h	Laparotomy for bowel obstruction
Priority 2	Within 4 weeks	Cancer surgery
Priority 3	Within 3 months	Colectomy—inflammatory bowel disease (IBD)
Priority 4	Can be delayed for >3 months	Elective total knee/hip replacements

[a] The timings were a short-term expedient during the pandemic only, not for long-term use.

Note that any unstable or actively bleeding patient is an immediate priority.

On the other hand, early evidence showed that delays in treatment for younger and medically fitter patients also conferred a worse outcome. For patients with cancer (breast, colorectal, lung, and oesophageal), it is estimated that delays to diagnosis and treatment as a result of pandemic restrictions will confer an excess 5y mortality of 6–15%.[5] For patients with a large aortic aneurysm of >7.0cm, delay to treatment of >3 months is associated with a predicted ↑ mortality of ~6%, and 1.5–2% for AAAs of >6cm.[6] This highlights the importance of early investigation and treatment in urgent conditions. The use of specified 'pathways', such as the 2WW system, allows such cases to be prioritized.

Virtual consultation

To reduce the risk of transmission, most outpatient consultations were conducted over the telephone or by video call, with remote triage prior to consultation. This has allowed much of the outpatient work to continue and the prioritization of urgent referrals. This new approach to consultation is likely to continue, post-COVID-19. Research needs to be done on the validity of examination by video assessment, and as such, some face-to-face clinic capacity is still required.

According to the Royal College of Surgeons England, virtual consultations should not be used:

- For patients with high-risk conditions, which may need a physical examination or close visual examination of an area.
- When an internal examination is required.
- When the patient's mental state is unsuitable for a virtual consultation (e.g. dementia).

- For patients unable to use remote technology to communicate and they cannot be supported to do so by a carer.
- When there are safeguarding concerns.

In these cases, clinic areas with good pathways, including personal protective equipment (PPE) and screening for symptoms, should be in place to be sure both patients and providers are safe.

Infection control measures: hand hygiene and personal protective equipment

The key principles of infection control have been highlighted during the COVID-19 pandemic but also apply to other nosocomial infections, e.g. methicillin-resistant *Staphylococcus aureus* (MRSA), *Clostridium difficile*, resistant enterobacteria [vancomycin-resistant *Enterococcus* (VRE), carbapenem-resistant *Enterobacteriaceae* (CPE)].

Handwashing

COVID-19 can survive on surfaces for up to 9 days. Therefore, good hygiene and surface disinfectants are essential in lowering transmission. Alcoholic concentrations of 60–95% are deemed acceptable for use, though handwashing is the first preference, where possible. The transmission of COVID-19 and many infectious diseases, such as MRSA and *C. difficile*, can be reduced by up to 50% with effective handwashing, according to WHO.

Isolation

Patients who test positive or are suspected or confirmed to have COVID-19 are isolated in side-rooms on the ward and isolated when undergoing investigations or surgical procedures. During surgery, special measures are taken to minimize contamination of theatre equipment and staff; following surgery, the theatre requires special cleaning procedures. This should be borne in mind when putting together a theatre list, and positive patients deferred to the end of the list if possible. Only essential personnel should be in the operating theatre for induction of patients suspected of having COVID-19 and those in whom their COVID status is unknown.

Personal protective equipment

As COVID-19 is transmitted as a respiratory fomite (aerosol), the rigorous use of PPE in all clinical areas has come to the forefront of medical practice. Special PPE is advised in aerosol-generating procedures (airway intubation, and laparoscopic and certain orthopaedic procedures; see Table 1.7).

Surgical training and simulation

Though there is no substitute for hands-on training in theatre, adaptations and redeployment during the pandemic have highlighted some vital complementary aspects of surgical practice, including intensive therapy unit (ITU) management and communication with patients and relatives. In addition, there has been a focus on the use of simulation training and online teaching, which has proved very valuable in consolidating surgical training and will continue to grow in future.[7,8]

Table 1.7 PPE types and their indications

Gloves and apron	Contact with blood or body fluids in any setting
Eye protection—mask and goggles or face shield	Risk of splashes or sprays (generally within 1m of patient)
Standard face mask **Respirator mask (FFP-3, N95)**	Any clinical area dealing with respiratory fomites (e.g. COVID-19, tuberculosis) Any surgical procedure High-risk area or aerosol-generating procedures (e.g. operating theatre, intensive therapy unit) for respiratory fomites
Full-length gown and surgical hat	Any invasive procedure High-risk area for respiratory fomites

'*Donning and doffing*' is the concept of putting on or removing PPE in a particular manner, so as not to contaminate oneself in the process—removal of PPE should generally be in the order of: gloves > face shield/goggles > gown > mask.

References

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3 Kahlberg A, Mascia D, Bellosta R *et al*. (2020). Vascular surgery during COVID-19 emergency in hub hospitals of Lombardy: experience of 305 patients. *Eur J Vasc Endovasc Surg* S1078–5884(20)30935-7.

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Principles of good prescribing

Good practice in prescribing

The UK GMC[1] lists the principles of good practice in prescribing (➔ Duties of a doctor, pp. 6–10):

- Keep up-to-date with the law and other regulations.
- Work within the limits of your competence—prescribe treatment only when you have adequate knowledge of the patient's health.
- Provide effective treatment based on the best available evidence.
- Check that the care or treatment you provide is compatible with any other treatments the patient is receiving,
- Make good use of the resources available to you, e.g. *British National Formulary* (*BNF*) app/book, hospital-wide policies (e.g. antibiotic prescribing policy), the ward pharmacist, ward intravenous (IV) drug guide.

Teamworking

In the hospital, there are many different health care professionals to whom it is now routine practice to refer for specialist prescribing advice. Specific areas that are important in surgery include prescribing:

- *Analgesics*:
  - **Acute pain team**—useful for complicated pain management needs post-operatively.
  - **Anaesthetists**—have expertise in patient-controlled analgesia (PCA), epidural management, and perioperative symptom control.
  - **Palliative care team**—for end-of-life symptoms.
- *Antiemetics* (as above).
- *Antibiotics*: for infection management, the first line is the hospital antibiotic guidelines; for complicated infections, discuss with the **microbiologist** or **infectious diseases teams**.
- *Anticoagulants*—**haematologist** for advice on bleeding patients and management of perioperative anticoagulation; **local anticoagulation service** for long-term anticoagulation advice.
- *IV medications*—**ward nurses** (or ICU nurses).
- *General advice*, e.g. medicines reconciliation, advice on interactions, etc.—**ward pharmacist**.
- *Other specialty-specific advice*—senior colleagues, advanced nurse practitioners.

Reference

1 General Medical Council (2013). *Good Medical Practice*.[updated April 2019] General Medical Council, London.

Analgesia

Managing the patient's pain in the perioperative period is an important core responsibility of a surgical trainee. The benefits of treating acute pain include:

- Wound healing.
- ↑ mobility.
- Patient satisfaction.
- Earlier hospital discharge.
- Reduction in risk of thromboembolic events.

In acute pain caused by trauma, surgery, childbirth, or a medical condition, it is important to identify the location, temporal aspects, and the intensity of the pain. Several methods exist for assessing the patient's pain (see Table 1.8).

Table 1.8 Methods for assessing acute pain

Score/method	Aspect assessed
Visual analogue scale (VAS)	Scored between 'no pain' and 'pain as bad as it can be'
Verbal response score (VRS)	Pain scored as a number, e.g. 4 out of 10, or verbally, e.g. mild, severe, excruciating
Autonomic response	Sweating, tachycardia, hypertension (HTN)
Dynamic pain scores	Pain on movement; ability to take a deep breath; ability to cough

The goal of post-operative analgesia is to minimize the dose of the analgesic medications (hence minimizing side effects), whilst providing adequate and effective analgesia.

The WHO analgesic ladder

This was originally developed for the management of chronic pain in cancer. The ladder is now widely used to treat various types of pain. The strategy is to prescribe analgesics promptly at the onset of pain and adapt the regimen until the patient is pain-free, using the ladder system (see Fig. 1.3).
► **Immediately after surgery, patients will require effective and strong analgesia.** Wherever possible, the **oral route is preferred**. However, in the acute post-operative period or if the patient is vomiting, the IV route may be required. The intramuscular (IM) route is painful and exhibits variable absorption and should be avoided if possible. The analgesic regime can be stepped down using the World Health Organization (WHO) ladder, as the patient recovers from surgery in the days following the operation. To ensure patients have consistent and prolonged analgesia, these should be administered regularly, rather than 'on demand'. Adjuvant medications are those that enhance the effect of an analgesic (e.g. anticonvulsants such as gabapentin used for neuropathic pain).

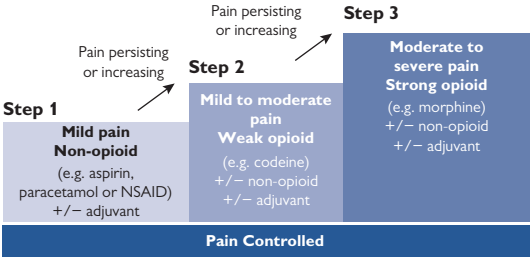

Fig. 1.3 The WHO analgesic ladder.

Reproduced from World Health Organization (2009). WHO's Pain Relief Ladder. www.who.int/cancer/palliative/painladder/en/

Post-operative nausea and vomiting

Post-operative nausea and vomiting (PONV) is a difficult and unpleasant side effect of anaesthesia and surgery, which can prolong recovery. The incidence of PONV depends on the type of surgery and case mix. It is estimated that with GA using inhalational agents, the incidence of PONV is 30% without prophylaxis. PONV is also a side effect of post-operative opioids and patient-specific factors—particularly a prior history of PONV contributes (see Box 1.9).[1]

Box 1.9 Apfel system for predicting PONV

- Post-operative opioids (1 point)
- Non-smoker (1 point)
- ♀ gender (1 point)
- History of PONV/motion sickness (1 point)

Several strategies have been proven to reduce the baseline risk of PONV.[2] These include:

- Avoidance of nitrous oxide (N_2O).
- Avoidance of volatile anaesthetics.
- Minimization of intraoperative and post-operative opioids.
- Adequate hydration.

Patients at risk of PONV should receive two antiemetics with different mechanisms of action, including ondansetron (unless contraindicated). Preoperative antiemetics are usually given orally (PO) but can be given IV.

Preoperative antiemetics that can be given as a single dose are:

- Ondansetron 8–16mg PO.
- Cyclizine 50mg PO.
- Prochlorperazine 3–6 mg buccal or, 5mg tablets PO.

Intraoperative antiemetics can be given IV at the end of the procedure, except dexamethasone which is given at induction:

- Dexamethasone 3.3–6.6mg IV (at induction).
- Ondansetron 4mg.
- Cyclizine 25–50mg.
- Droperidol 625–1250 micrograms.

Post-operative antiemetics—assess patients regularly to detect PONV. In patients who are actively vomiting, regular IV antiemetics should be administered. Oral and buccal antiemetics are suitable only for mild to moderate nausea, e.g. ondansetron 4mg 6-hourly, cyclizine 50mg 8-hourly, buccal prochlorperazine 12-hourly.

References

1 Apfel CC, Korttila K, Abdalla M, *et al.* (2004). A factorial trial of six interventions for the prevention of postoperative nausea and vomiting. *N Engl J Med* **350**: 2441–51.

2 Gan TJ, Diemunsch P, Habib AS, *et al.* (2014). Consensus guidelines for the management of postoperative nausea and vomiting. *Anesth Analg* **118**: 85–113.

Antibiotic prescribing in surgery

The principles of good antibiotic prescribing

In the last 40y, antibiotic resistance has risen dramatically. There are few new antibiotics being developed and there is an urgent need to conserve the efficacy of existing antibiotics. The UK Department of Health has issued a 'Start Smart – then Focus' stepwise approach to ensure antibiotics are used effectively and optimally.[1]

Start smart

- Do not start antibiotics unless there is clear evidence of infection.
- Take a thorough drug allergy history.
- Look at local antibiotic prescribing guidelines.
- Initiate prompt effective antibiotic treatment within 1h of diagnosis (or as soon as possible) in patients with severe sepsis or life-threatening infections. Avoid inappropriate use of broad-spectrum antibiotics (➲ Sepsis, pp. 172–3).
- Document clinical indication (± disease severity), drug name, dose, and route on the drug chart and in clinical notes.
- Include a review/stop date or the duration.
- Obtain cultures prior to commencing therapy where possible (but do not delay therapy).
- **Prescribe single-dose antibiotics for surgical prophylaxis where antibiotics have been shown to be effective.**
- Document the exact indication on the drug chart for clinical prophylaxis (rather than stating long-term prophylaxis).

Then focus

- Reviewing the clinical diagnosis and the continuing need for antibiotics at 48–72h and documenting a clear plan of action—the 'antibiotic prescribing decision'. All IV antibiotics to be reviewed at 48h.
- The five 'antibiotic prescribing decision' options are:

1. **Stop** antibiotics if there is no evidence of infection.
2. **Switch** antibiotics from IV to PO.
3. **Change** antibiotics, ideally to a narrower spectrum, or broader if required.
4. **Continue** and document the next review and stop date.
5. **Outpatient parenteral antibiotic therapy (OPAT)**—providing parenteral antibiotic therapy to patients in a non-inpatient setting.

Antibiotic prophylaxis in surgery

- Antibiotic prophylaxis is an effective intervention for preventing surgical site infections (SSI) in *certain* surgical procedures.
- Many other *risk factors* also affect the incidence of SSIs. Table 1.9[2] summarizes the main patient and surgical operation factors which influence the incidence of SSIs.

In deciding to prescribe antibiotics for surgical prophylaxis, the risks and benefits of treatment should be balanced.

Table 1.9 Factors influencing the incidence of SSIs

Patient	Extremes of age
	Poor nutritional status
	Obesity (>20% of ideal body weight)
	Diabetes mellitus (DM)
	Smoking
	Coexisting infections at other sites
	Bacterial colonization (e.g. nasal colonization with *Staphylococcus aureus*)
	Immunosuppression
	Prolonged post-operative stay
Operation	Length of surgical scrub
	Skin antisepsis
	Preoperative skin preparation
	Length of operation
	Antibiotic prophylaxis
	Operating theatre ventilation
	Foreign material in surgical site
	Surgical technique
	Post-operative hypothermia

Benefits of antibiotic prophylaxis

The value of antibiotic prophylaxis in surgery is related to the incidence and severity of SSIs. SSIs can increase the risk of patient morbidity and hospital stay. The length of stay associated with SSIs is dependent on the type of surgery the patient has undergone. Evidence indicates that prevention of wound infections is associated with shorter lengths of stay and faster patient recovery.

Risks of prophylaxis (and antibiotic use in general)

It is important to optimize surgical antibiotic prophylaxis to minimize the unintended consequences of antibiotic use. Important risks associated with antibiotic prescribing include: penicillin allergy, *Clostridium difficile* infection (CDI), and antibiotic resistance.

- ***Penicillin allergy***—penicillins and cephalosporin antibiotics are the most commonly used classes of antibiotics. If patients are wrongly labelled as having a penicillin allergy, it can compromise their antibiotic therapy. Taking a detailed medical history, including the nature of the reaction a patient recalls to penicillins or other antibiotics, is an important step in establishing the allergy status of the patient. Patients with a true penicillin allergy should not be prescribed β-lactam antibiotics due to cross-sensitivity with penicillins. Table 1.8 summarizes the list of the most commonly used antibiotics that should be avoided, those that should be used with caution, and those that can be used in penicillin allergy.

Table 1.10 Examples of antibiotics in penicillin allergy

Antibiotics that must be avoided in serious penicillin allergy	Amoxicillin (e.g. co-amoxiclav/Augmentin®, HeliClear®) Ampicillin (co-fluampicil/Magnapen®) Benzylpenicillin/penicillin G Flucloxacillin (co-fluampicil/Magnapen®) Phenoxymethylpenicillin/penicillin V Piperacillin-tazobactam (in Tazocin®) Pivmecillinam Ticarcillin (in Timentin®)
Antibiotics to be avoided in serious penicillin allergy *and* used with caution in non-severe penicillin allergy (e.g. minor rash only)	Cephalosporins: cefaclor, cefadroxil, cefalexin, cefixime, cefotaxime, cefpirome, cefpodoxime, cefprozil, cefradine, ceftazidime, ceftriaxone, cefuroxime Other β-lactam antibiotics: aztreonam, imipenem, meropenem, ertapenem
Antibiotics considered safe in penicillin allergy	Amikacin, ciprofloxacin, clarithromycin, clindamycin, colistin, co-trimoxazole, doxycycline, erythromycin, gentamicin, linezolid, metronidazole, nitrofurantoin, minocycline, rifampicin, sodium fusidate, teicoplanin, tetracycline, tobramycin, trimethoprim, vancomycin

- ***CDI***—the risk of acquiring *C. difficile* infection is ↑ in patients who:
  - Have had recent gastrointestinal (GI) surgery.
  - Have had a prolonged stay in hospital.
  - Are receiving proton pump inhibitors (PPIs).
  - Are elderly.
  - Are receiving or have recently received antibiotics—patients treated with broad-spectrum antibiotics are at greater risk of CDI.
- ***Antibiotic resistance***—the prevalence of antibiotic resistance is increasing globally, especially in health care settings. The emergence and spread of antibiotic resistance in any population is linked to the exposure to antibiotics across the population. Inappropriate and excessive antibiotic use can lead to antibiotic resistance.

Not all types of surgery require antibiotic prophylaxis. NICE guidelines recommend that antibiotic prophylaxis should *not* be used routinely for **clean non-prosthetic uncomplicated surgery**.[3]

Surgery that *does* require prophylaxis includes:
- Clean surgery involving the placement of a prosthesis or implant.
- Clean-contaminated surgery.
- Surgery on a dirty or infected wound (requires antibiotic treatment, in addition to prophylaxis).[3]

Choice of antibiotic for prophylaxis

A wide range of microorganisms can cause infections in patients. Choosing the appropriate antibiotic will depend on patient factors, as well as on the likely site of infection and the local susceptibility patterns. ► **First, refer to your local/hospital antibiotic prescribing guidelines.** These policies are produced by MDTs and are based on the best available evidence and clinical expertise.

Timing of antibiotic administration for surgical prophylaxis

The route, dose, and pharmacokinetic profile of antibiotics decide the time it takes for an antibiotic to reach effective concentrations in specific human tissues. Administering prophylactic antibiotic doses too late or too early can reduce their efficacy and increase the risk of SSIs.

> '*Intravenous prophylactic antibiotics, in surgical patients should be given within 60 minutes before the skin is incised, and as close to time of incision as is possible.*'[3]

Vancomycin, when indicated, should be given by IV infusion starting 90min prior to skin incision.[3] In most circumstances, a single prophylactic dose of antibiotic is sufficient—always check the local policy to determine the dose and frequency of the antibiotic to be administered.

References

1 Public Health England (2015). *Start Smart—then Focus. Antimicrobial Stewardship Toolkit for English Hospitals*. Department of Health, London.
2 Berrios-Torres SI, Umscheid CA, Bratzler DW, *et al*. (2017). Centres for Disease Control and Prevention guideline for the prevention of surgical site infection. *JAMA Surg* **152**: 784–91.
3 National Institute for Health and Care Excellence (2017). *Surgical Site Infections: Prevention and Treatment*. National Institute for Health and Care Excellence, London.

Thromboprophylaxis in surgery

➔ Chapter 2, Prophylaxis pp. 98–9.

Key summary: prescribing in surgery

- ***Safe prescribing***

Always prescribe within your competence. Ensure you follow local policy and guidelines at all times. Make use of the expertise within the MDTs in the hospital to guide your prescribing decisions.

- ***Analgesia***

Ensure patients are prescribed adequate, effective analgesia post-operatively. Taper the analgesic needs of the patient through assessment and the WHO ladder.

- ***Post-operative nausea and vomiting***

Assess patients for risk of PONV and initiate appropriate therapy pre- and perioperatively. In the post-operative period, assess patients regularly for PONV and treat it promptly.

- ***Antibiotic prescribing***

Adopt the *Start Smart—then Focus* steps to ensure appropriate antibiotic prescribing.

Make use of the expertise provided by microbiology and infectious diseases doctors and pharmacists.

- ***Anticoagulation***

All surgical patients must be assessed upon admission, and regularly thereafter, for the risk of venous thromboembolism (VTE). Patients identified as being at risk of VTE must be initiated on appropriate thromboprohylaxis.

Chapter 2

Principles of surgery

Terminology in surgery

How to describe an operation

The terminology used to describe all operations is a composite of basic Latin or Greek terms.

First, describe the organ/area to be operated on

- lapar-, abdomen (laparus = flank).
- nephro-, kidney.
- pyelo-, renal pelvis.
- cysto-, bladder.
- chole-, bile/biliary system.
- ileo-, small bowel (distal).
- col(on)-, large bowel.
- hystero-, uterus.
- thoraco-, chest.
- rhino-, nose.
- masto/mammo-, breast.

Second, describe any other parts involved

- docho-, duct.
- angio-, vessel (blood- or bile-carrying).
- litho-, stone.

Third, describe what is to be done

- -otomy, to cut open.
- -ectomy, to remove (cut out).
- -plasty, to change shape or size.
- -pexy, to change position.
- -raphy, to sew together.
- -oscopy, to look into.
- -ostomy, to create an opening in (stoma = mouth).
- -paxy, to crush.
- -graphy/gram, image (of).

Lastly, add any terms to qualify how or where the procedure is done

- percutaneous, via the skin.
- trans-, across.
- antegrade, forward.
- retrograde, backward.

Examples of terms

- Choledochoduodenostomy. An opening between the bile duct and the duodenum.
- Rhinoplasty. Nose reshaping.
- Pyelolithopaxy. Destruction of pelvicalyceal stones.
- Bilateral mastopexy. Breast lifts.
- Percutaneous arteriogram. Arterial tree imaging by direct puncture injection.
- Loop ileostomy. External opening in the small bowel with two sides.
- Flexible cystourethroscopy. Internal bladder and urethral inspection.

History taking and case presentation

Basics

- Start with name, age, occupation, and method of presentation, e.g. Accident and Emergency (A&E)/GP referral/admission from clinic.
- Cover all the principal areas of the complete medical history, as follows.

Presenting complaint

This is a one- or two-word summary of the patient's main symptoms, e.g. "right iliac fossa (RIF) pain", "abdominal pain and vomiting", "bleeding per rectum (PR)".

- In emergency admissions, do not write a diagnosis here (e.g. ischaemic leg). The diagnosis of referral may well turn out to be wrong.
- In elective admissions, it is reasonable to write e.g. 'elective admission for anterior resection for rectal adenocarcinoma'.

History of presenting complaint

- This is a detailed description or exploration of the main symptom(s) and should include the relevant systems enquiry.
- Start with any relevant background history to set the context for the presenting complaint.
- Try to put the important positives first, e.g. "right-sided lower abdominal pain, worse with moving and coughing, anorexia.
- Include the relevant negatives, e.g. "no vomiting, no PR bleeding.
- Be very clear about the chronology of events.
- In a complicated history or with multiple symptoms, use headings, e.g. 'Previous episodes/operations for this problem', 'Current episode', 'Results of investigations'.

Summarize the results of previous investigations systematically: blood tests, microbiology, histopathology, radiology, and specialized tests.

Past medical history

- List specific medical diagnoses.
- And relevant negatives (it is good practice to ask about cardiorespiratory and renal conditions, which impact on the patient's operative/anaesthetic risk, e.g. ischaemic heart disease (IHD), heart failure, COPD, renal impairment, as well as those specific to the presenting complaint, e.g. neurological diagnoses in neurosurgery/ear, nose, and throat (ENT), risk factors for atherosclerosis in vascular surgery.
- List and date all previous operations.
- Ask about previous problems with an anaesthetic.

Systematic enquiry

This is very important, though often neglected—it can be highly relevant to rule out other diagnoses (e.g. gynaecological cause for lower abdominal pain) and to assess the patient's surgical/anaesthetic risk.

- Cardiovascular. Chest pain, effort dyspnoea, orthopnoea, nocturnal dyspnoea, palpitations, swollen ankles, strokes, transient ischaemic attacks (TIAs), claudication.
- Respiratory. Dyspnoea, cough, sputum, wheeze, haemoptysis.

- GI. Anorexia, change in appetite, weight loss (quantify how much over how long).
- Genitourinary (GU). Sexual activity, dyspareunia (pain on intercourse), abnormal discharge, last menstrual period (all ♀ patients).
- Neurological. Three Fs: fits; faints; funny turns.
- Nil by mouth (NBM) time: what time did they last eat or drink? (for emergency admissions)

Social history

- Ask about who will look after the patient. Do they need help to mobilize and/or with activities of daily living?
- Smoking and alcohol history.
- Occupation.

▶ Tips for case presentation

- Practise. Every case is a possible presentation to someone!

Always 'set the scene' properly. Start with name, age, occupation (if elderly, general fitness/independence), relevant background history, mode of referral, and presenting complaint, e.g. '*78-year-old man, normally fit and well, with a prior history of open anterior resection for rectal adenocarcinoma, presents with a 3-day history of abdominal pain and vomiting*'.

- Be chronological. Start at the beginning of any relevant prodrome or associated symptoms; they are likely to be an important part of the presenting history, e.g. '*He was well until … when he started experiencing … the current symptoms started …* '
- Be concise with the past medical history. Only expand on things that you really feel may be relevant either to the diagnosis or to the management.
- Always summarize the general appearance and vital signs first.
- Describe the most significant findings first, but be systematic, e.g. '*on inspection … , palpation … , percussion … , auscultation*'.
- *Briefly summarize other systemic findings.* Expand on them if they are directly relevant to the diagnosis or management.
- Finally, summarize and synthesize. Try to group symptoms and signs into clinical patterns that lead to the proposed diagnoses or differential list.
- Be prepared to discuss what diagnostic or further evaluation tests might be necessary.

Common surgical symptoms

Pain

Pain anywhere should have the same features elicited. These can be summarized by the acronym SOCRATES:

- **S**ite. Where is the pain? Is it localized, in a region, or generalized?
- **O**nset. Gradual, rapid, or sudden? Intermittent or constant?
- **C**haracter. Sharp, stabbing, dull, aching, tight, sore?
- **R**adiation. Does it spread to other areas? (From loin to groin in ureteric pain; to shoulder tip in diaphragmatic irritation; to back in retroperitoneal pain; to jaw and neck in myocardial pain.)
- **A**ssociated symptoms. Nausea, vomiting, dysuria, jaundice?
- **T**iming. Does it occur at any particular time?
- **E**xacerbating or relieving factors. Worse with breathing, moving, or coughing suggests peritoneal/pleural irritation; relief with hot water bottles suggests deep inflammatory or infiltrative pain.
- **S**urgical history. Does the pain relate to surgical interventions?

Dyspepsia (epigastric discomfort or pain, usually after eating). What is the frequency? Is it precipitated by food or is it spontaneous in onset? Is there any relief with milky drinks or food? Is it positional?

Dysphagia (difficulty during swallowing). Is the symptom new or long-standing? Is it rapidly worsening or relatively constant? Is it worse with solids or fluids? (Worse with fluids suggests a motility problem, rather than a stenosis.) Can it be relieved by anything, e.g. warm drinks? Can the patient point to a 'level' of hold-up? This often accurately relates to the level of an obstructing lesion. Is it associated with 'spluttering' (suggests a tracheo-oesophageal fistula or inhalation of food/fluid).

Oesophageal reflux (bitter or acidic tasting fluid in the pharynx or mouth). How frequent? What colour? (Green suggests bile, whereas white suggests only stomach contents.) When does it occur (lying only, on bending, spontaneously when standing)? Is it associated with coughing?

Haematemesis (the presence of blood in vomit). What colour is the blood? (Dark red-brown 'coffee grounds' is old or small-volume stomach bleeding; dark red may be venous from the oesophagus; bright red is arterial and often from major gastric or duodenal arterial bleeding.) What volume has occurred over what period? Did the blood appear with the initial vomits or only after a period of prolonged vomiting? Suggests a traumatic oesophageal cause.

Abdominal distension Symmetrical distension suggests one of the '5 Fs' (*fluid* ascites, *flatus* due to ileus or obstruction, *fetus* of pregnancy, *fat*, or a '*flipping big mass*'). Asymmetrical distension suggests a localized mass. What is the time course? Does it vary? Is it changed by vomiting? Passing stool/flatus?

Change in bowel habit May be change in frequency or consistency (↑ frequency and looser stools are more likely to be due to a pathological cause). Is it persistent or transient? Persistent change in bowel habit >6 weeks requires further investigation.

Frequency and urgency of defecation New urgency of defecation is almost always pathological. What is the degree of urgency—how long can the patient delay? Is there associated discomfort? What is passed—is the stool normal?

Bleeding per rectum What colour is the blood? Pink-red and only on the paper when wiping or splashing in the pan suggests a cause from the anal canal. Bright red on the surface of the stool suggests a lower rectal cause. Darker blood with clots or marbled into the stools suggests a colonic cause. Blood fully mixed with the stool or altered suggests a proximal colonic cause.

Tenesmus (desire to pass stools with either no result or a feeling of incomplete defecation). Suggests rectal pathology.

Jaundice (yellow discoloration of skin/sclera/uvula due to hyperbilirubinaemia; ➲ Jaundice, pp. 406–10) How quickly did the jaundice develop? Is there associated pruritus? Are there any symptoms of pain, fever, or malaise? (Suggests infection.)

Haemoptysis (the presence of blood in expectorate). What colour is the blood? (Light pink froth suggests pulmonary oedema.) Are there clots or dark blood (infection or endobronchial lesion)? How much blood? Moderate bleeds quickly threaten airways—get help quickly.

Dyspnoea (difficulty in, or ↑ awareness of, breathing) When does the dyspnoea occur? (Quantify the amount of effort.) Is it positional?

- **Orthopnoea**. Difficulty in breathing that occurs on lying flat; quantify it by asking how many pillows the patient needs at night to remain symptom-free.
- **Paroxysmal nocturnal dyspnoea**. Intermittent breathlessness at night. Both orthopnoea and paroxysmal nocturnal dyspnoea suggest cardiac failure.

Claudication (pain in the muscles of the calf, thigh, or buttock precipitated by exercise and relieved by rest). After what degree of exercise does the pain occur (both distance on the flat and gradients)? How quickly is the pain relieved by rest?

Rest pain (pain in a limb at rest without significant exercise). How long has the pain been present? Is it intermittent? Does it occur mainly at night? Is it relieved by dependency of the limb involved?

Dysuria (pain on passing urine). When does the pain occur (beginning, end, or throughout the stream)? Is it felt in the penis or suprapubically? Is it associated with frequency? Is the urine discoloured or does it contain debris?

Haematuria (blood in the urine). Does the blood occur at the start (suggests bladder origin), during, or at the end (suggests prostatic or penile origin) of the stream? Is there associated pain (suggests infection or stone disease)?

Evaluation of breast disease

The key principle of breast evaluation is *Triple Assessment:* This comprises physical examination, followed by radiological imaging and biopsy (The fine needle or core biopsy)

Physical examination

Positioning and inspection

Breasts are best examined semi-recumbent and then sitting upright. Initially, the arms are by the side, semi-recumbent. Then they should be inspected sitting upright, with hands on hips (initially relaxed and then with forced pressure on the hips to tense the pectoral muscles), and finally abducted slowly above the head.

Inspection is critical and should concentrate on the following.

- Overall symmetry and position. Are the breasts the same size? Is there deformity due to underlying disease? Is the position normal?
- Skin appearance. Is the skin erythematous or oedematous? Is there fixed lymphoedema of the skin ('peau d'orange')? Are there scars from previous surgery?
- Skin tethering. Does the skin move freely as the arms are raised? (Tethering is suggestive of underlying intraparenchymal scarring or tumour.)
- Nipples. Are the nipples indrawn, deviated, or ulcerated (suggestive of retroareolar tumour or infection)? Is there any discharge?

Palpation

For palpation, the hands should return to the hips and the patient may lie back semi-recumbent again. Use the flat of the fingers and use all four fingers at once. Palpate the 'normal' breast first. Be methodical and do not 'knead' the breast. A common routine is: upper outer quadrant; lower outer; lower inner; upper inner; central (retroareolar); supraclavicular fossa; and axilla. Features to look for include:

- Palpable mass. Is it hard, irregular, and tethered (cancer) or smooth, rounded, and mobile (cysts or fibroadenoma)?
- Diffuse nodularity. Typical of benign disease.
- Nipple discharge on palpation of the central area. Blood suggests tumour; pus suggests infection; serous or milky may not be relevant.
- Axillary and supraclavicular lymphadenopathy. Is it multiple and tethered (cancer)?

Imaging

Ultrasound

- Easy to perform and painless—often done in breast outpatient clinic.
- Avoids radiation dose in young women.
- Highly sensitive for differentiating between solid tumours and cysts.

Mammography

- Used both for population screening and diagnostic testing.
- Uncomfortable for most women and involves a low radiation dose.
- Able to identify impalpable lesions.
- Able to identify premalignant lesions, e.g. ductal carcinoma *in situ* (DCIS).
- Mammographic features of malignancy include: spiculated microcalcification; irregularity; and stellate outline.

Biopsy

Aspiration cytology

- Well tolerated, easy to perform, and quick to report on—often done in one half-day during breast outpatient clinic.
- Does not provide histology; provides only cellular information and relies upon cellular atypia for a diagnosis of malignancy.
- Does not differentiate between invasive and *in situ* carcinoma.
- Occasionally therapeutic for cysts.
- Good sensitivity and specificity.

Guided core biopsy

- Performed under ultrasound or mammographic guidance using a Trucut® needle or similar device.
- Can be done under GA or LA.
- Provides actual histology information—allows cancers to be graded.
- Able to differentiate between invasive and carcinoma *in situ*.
- Highly sensitive and specific.

Other (less commonly used)

Computerized tomography (CT) scanning

- Useful for assessment of extensive local invasion and regional and systemic staging.

CT positron emission tomography (PET) scanning

Useful for investigating indeterminate lesions and to identify unsuspected metastatic disease.

Magnetic resonance imaging (MRI) scanning

Occasionally used for diagnosis e.g. women with breast implants.

Key revision points—anatomy of the breast

- The breast comprises epithelial ductal tissue, epithelial secretory lobules, fat, and connective tissue.
- It is divided into four 'quadrants' and a peri-/retroareolar central zone for clinical description of abnormalities.
- The arterial supply is from segmental perforators from the internal thoracic artery (ITA).
- Lymphatic drainage—important in breast cancer management:
  - Non-pathological lymph drainage is almost entirely to the axillary nodes.
  - Medial half can occasionally drain to internal mammary nodes.
  - Axillary nodes are divided into three levels (1, below; 2, behind; 3, above the pectoralis minor).

Evaluation of the neck

Positioning and inspection

- Sit the patient upright at rest, with the head looking straight ahead.
- Inspect the neck from the front, side, and, if necessary, behind.
- Observe the neck at rest and during swallowing (a glass of water). If necessary, inspect rotation left and right.
- Observe the neck, whilst asking the patient to protrude the tongue.

Inspection (what to look for)

- *Overall symmetry and lumps*. Are there obvious lumps? Are they single or multiple? Is the lump lying in, or close to, the midline? Does the lump move with swallowing (suggests thyroid-related lesion)?
- *Skin abnormalities*. Are there any ulcers or sinuses (suggests chronic infection such as tuberculosis)?
- *Associated structures*. Is there evidence of venous engorgement or visible collateral vessels?

Palpation

Be systematic; palpate the regions of the neck in order. Use both hands with the flat of the fingers to compare each side, but move only one hand at once to prevent 'cross-palpation'. A typical sequence of palpation is: anterior triangle (bottom to top); submental area; submandibular area; posterior triangle (top to bottom); supraclavicular fossae; and parotid, preauricular, and post-auricular areas. Repalpate the neck, with the patient swallowing a mouthful of water—particularly the anterior triangle (see Fig. 2.1). Lastly, feel specifically for the carotid arteries.

- *Lump*. Is it single or multiple? (Multiple strongly suggests lymphadenopathy.) Is it strictly in the midline (likely to be related to the thyroid)? Does it move with swallowing (almost always thyroid-related)? What are the general features?
- *Thyroid lump*. Is it unilateral or bilateral? Does it move with tongue protrusion?
- *Carotid arteries*. Are they normal, ectatic, or aneurysmal?
- *Supraclavicular fossae*. Is there associated lymphadenopathy (suggests malignancy)?

Auscultation

Listen to the carotid arteries and any large masses for bruits, suggesting a hypervascular local circulation or stenosis.

Investigations

Ultrasound

- Easy to perform and painless.
- Avoids radiation dose.
- Highly sensitive for differentiation between solid tumours and cysts.

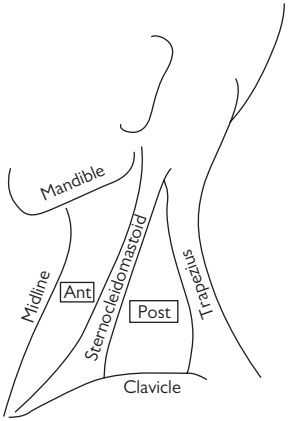

Fig. 2.1 Key revision points—triangle of the neck.

Aspiration cytology
- Easy to perform and quick to report on.
- Usually well tolerated in outpatients.
- Provides only cellular information and relies upon cellular atypia for a diagnosis of malignancy.
- Does not provide histological information.
- Occasionally therapeutic for cysts.
- Good sensitivity and specificity.
- Contraindicated where there is suspicion of a vascular lesion.

CT scanning
- Useful for assessment of extensive local invasion and regional and systemic staging of tumours.
- Allows evaluation of the thorax in some thyroid tumours.

CT PET scanning
- Occasionally used to assess indeterminate lesions identified on plain CT and identify unsuspected metastatic disease.

MRI scanning
- Useful for detailed assessment of local invasion of tumours.

Evaluation of the abdomen

Positioning

- Lie the patient supine with adequate support for the head (e.g. one pillow) to ensure the abdominal muscles are relaxed.
- Arms should be by the sides to relax the lateral abdominal muscles.
- Stand the patient up to examine the groin if a groin hernia is suspected; most hernias and groin pathology can be assessed in the supine position.

Inspection

- General appearance of the patient. Is there evidence of jaundice or signs of anaemia? Does the patient look malnourished or cachectic?
- First, ask the patient to cough and raise their head during inspection; this may reveal hernias—inspect during normal and deep respiration.
- Overall appearance. Is it symmetrical? Is there evidence of global distension (e.g. ascites, distended bowel)? Is there evidence of local distortion (e.g. a local mass or organomegaly)? Does the abdomen move well and symmetrically with deep respiration (reduced in peritoneal irritation)? Is there any discoloration (periumbilical bruising—Cullen's sign, or flank bruising—Grey Turner's sign) suggestive of retroperitoneal haemorrhage or major inflammation?
- Scars. Where are they? How old do they appear? Is there evidence of herniation on coughing?
- Is there a stoma? What type? Does it look healthy or abnormal? What is the content in the stoma appliance?
- Umbilicus. Is it herniated? Is there discharge or ulceration suggestive of infection or a malignant deposit?
- Pulsation. Is there visible pulsation?
- Peristalsis. Is there visible peristalsis? (This may take several minutes to observe.)

Palpation and percussion

Be methodical. Use the flat of one hand (usually the right). It is usual to examine and describe the abdomen in areas. It can be divided into nine regions or five 'quadrants' (see Fig. 2.2). Examine the areas lightly at first in a set order. Identify any masses or areas of tenderness. Repeat the examination with deeper palpation. Go back to any identified masses and try to ascertain their key features.

- Signs of peritoneal irritation (localized or generalized). Are there signs of visceral peritoneal irritation (tenderness on palpation)? Are there signs of parietal peritoneal irritation (guarding, rebound tenderness, percussion tenderness, or, if marked, rigidity).
- Masses. Assess their surface, edge, consistency, movement with respiration, and overall mobility.
- Organs.
  - Liver. Palpate from right lower quadrant (RLQ) into right upper quadrant (RUQ). Assess the edge and any palpable surface—are they smooth/nodular/craggy?
  - Spleen. Palpate from RLQ into left upper quadrant (LUQ), as for the liver.
  - Kidneys. Palpate bimanually in each loin.

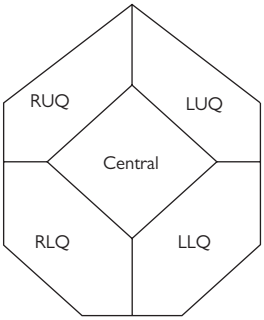

Fig. 2.2 The five quadrants: RUQ, right upper quadrant; LUQ, left upper quadrant; LLQ, left lower quadrant; and RLQ, right lower quadrant.

- Percussion to assess for gas or fluid.
- Gas. Hyperresonance—is it generalized or localized? Is there evidence of loss of dullness over the liver, suggestive of copious free intraperitoneal gas?
- Fluid (ascites): identified as 'shifting dullness'—dullness in the flanks which moves medially in the lateral position.

Auscultation

To fully assess bowel sounds, it is necessary to listen for at least 1min. Bowel sounds should broadly be divided into: absent, normal, active, or obstructive (characterized by high-pitched, frequent sounds, often with crescendos of activity, e.g. 'tinkling', 'bouncing marbles').

Rectal examination and external genitalia

Abdominal assessment should include a rectal examination in most adults; in children, this is very rarely useful and should usually be avoided. In ♂, one should examine the external genitalia (to exclude it as a differential diagnosis).

Abdominal investigations

Faecal occult blood testing

- May be chemical or immunological/quantitative—quantitative faecal immunochemical test (qFIT).

Commonest use is as the 1° community test for colorectal carcinoma (➲ Colorectal cancer, pp. 500–2), as part of the National Bowel Cancer Screening Programme.

Rigid proctoscopy and sigmoidoscopy

(See next section ➲ Evaluation of pelvic disease pp. 66–7 and ➲ Chapter 4 Rigid sigmoidoscopy, p. 284.)

Flexible sigmoidoscopy

- Very low risk (perforation <1 in 10 000) usually performed without sedation.
- Should visualize up to the descending colon.
- Allows minor therapeutic procedures (polypectomy, biopsy, injection).
- Typically used for: diagnosis and assessment of colitis and colonic neoplasia, investigation of anorectal bleeding.

Colonoscopy

- Low risk (perforation 1 in 1000); performed with or without IV sedation or Entonox®; requires bowel preparation.
- Should visualize the entire colon (>95% of the time).
- Allows minor therapeutic procedures—polypectomy, including endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD), injection, marking by tattoo, and biopsy.
- Where full colonic assessment is indicated or a colonic and/or terminal ileal pathology is suspected.

Transabdominal ultrasound

- Easy, safe, and non-invasive, and avoids radiation dose.
- Typical uses include:
  - 1° investigation of the biliary tree for gallstones, bile duct size, and liver parenchymal texture.
  - Identification of ovarian disease, e.g. in suspected appendicitis.
  - Assessment of the liver/splenic parenchyma.
  - Identifying free fluid in abdominal trauma.

CT scanning

- Easy, non-invasive; requires significant radiation exposure and IV/PO contrast.
- Typical uses include:
  - 1° assessment of all intra-abdominal masses.
  - Staging of intra-abdominal and pelvic malignancy.
  - Investigation of acute abdominal pain (increasingly used).
  - Investigation of suspected intestinal obstruction.
  - Investigation of suspected post-operative complications.

MRI scanning

- Avoids radiation dose.
- May be performed with specialized 'contrast' agents
- Typically used for:
  - Investigation of suspected bile duct disease—magnetic resonance cholangiopancreatography (MRCP).
  - Assessment of liver disease/possible metastases.
  - Assessment of the pancreas.
  - Assessment of pelvic and retroperitoneal soft tissue disease, e.g. pelvic cancers, complex perianal sepsis.
  - Assessment of small bowel where radiation exposure is best avoided (magnetic resonance enterography)

Plain abdominal radiograph

- May identify intestinal obstruction, urinary tract stones, free intra-abdominal air, and intra-abdominal fluid.

Barium/Gastrografin® enema (double contrast, single contrast)

- May be single contrast (contrast material filling the colon—used to identify strictures and obstructions) or double contrast (dilute contrast and air to coat the mucosal surface of the colon—now rarely used).

CT colonography

- Requires full bowel preparation, rectal catheter insertion; significant radiation exposure with IV/PO contrast.
- Typically used in place of colonoscopy where colonoscopy is contraindicated or impossible, e.g. age, frail patient, known stricture, failed colonoscopy.

Intestinal transit studies

- Serial abdominal X-rays (AXR) to identify the progress of ingested radio-opaque markers.
- Used to assess intestinal motility and transit time.

PET scanning

- Injection of radioactive metabolic substrate to identify metabolically active tissue (malignancy or inflammation / infection)
- Combined with high-resolution CT scanning to co-locate 'hot spots'.
- Typically used to identify unsuspected metastatic tumour deposits or differentiate fibrosis from residual tumour post-surgery.

Physiological testing

- Manometry testing of the oesophagus, including lower oesophageal sphincter and the anal canal.
- Pressure sensitivities of the oesophagus and anal canal.
- pH testing of the contents of the oesophagus (isolated or continuously for 24h).
- Used to assess anorectal function, oesophageal motility and function, and gastro-oesophageal reflux.

Evaluation of pelvic disease

Positioning and inspection

Examination is performed in up to three positions: supine (for transabdominal palpation of the 'false' pelvis); supine with hips flexed and abducted (for vaginal and bimanual palpation which may be performed to help assess rectal disease); and left lateral position with hips flexed (for rectal palpation and rigid endoscopy). Any intimate examination should always have a chaperone present and particularly so for pelvic examinations.

- *Inspection of the perineum*. Is the anus deformed? Is there evidence of mucosal or rectal prolapse? Does the vaginal introitus look normal? Is there vaginal prolapse or evidence of a cystocele? Are there scars from previous surgery, sinuses, or evidence of sepsis? Look for additional or abnormal tissue. Are there skin tags, external haemorrhoids, warts, or abnormal areas of skin—such as anal intraepithelial neoplasia (AIN)? Is there an external punctum (as may be seen in a fistula) or the outer limit of a fissure visible?

Palpation

- Palpate the lower abdominal quadrants.
- *Rectal examination*. Is anal tone normal and the sphincter symmetrical? Is the prostate normal size with a normal central sulcus? Does the rectal mucosa feel normal? Is there any mass or tenderness anterior to the upper rectum (pouch of Douglas)? The latter may be due to sigmoid disease, small bowel in the pelvis, a pelvic appendix, or ovarian disease.
- *Vaginal examination* (often omitted unless there is a clear indication that valuable information may be gained from it). Is the cervix present and normal? Is the vagina of normal calibre and feel? Is there tenderness in either vaginal fornix?

Investigations

Rigid proctoscopy ('anoscopy')

- Performed in outpatients without sedation.
- Visualizes the very lowermost rectum and anal canal useful for assessing haemorrhoids.
- May be combined with therapy (banding, injection, or cryotherapy).

Rigid sigmoidoscopy (best considered as 'proctoscopy')

- Performed in outpatients without sedation.
- Aims to visualize the rectum to the recto-sigmoid junction. The sigmoid colon is NOT adequately seen with this. Views may not be good if done without enema preparation.

Transabdominal/transvaginal ultrasound

- Easy and safe; and avoids radiation dose.
- Good for identification of ovarian disease (e.g. in RIF pain).

Endoanal/transrectal ultrasound

- A 360° scanning endoanal/endorectal probe without sedation.
- Endoanal scans, for assessment of anal sphincter integrity.
- Transrectal scans, for assessment of some rectal tumours, prostatic disease (including biopsy), and pre-sacral lesions.

CT scanning

- Investigation of choice for undiagnosed pelvic symptoms and post-operative complications.

MRI scanning

Usually via conventional body scanner with external coils, though occasionally performed with endorectal coil).

- Investigation of choice for the assessment of advanced rectal, gynaecological, and urological cancers, or complex pelvic and anal sepsis.

Key revision points—pelvic anatomy

- The true pelvis lies between the pelvic inlet (sacral promontory, iliopectineal lines, symphysis pubis) and outlet (coccyx, ischial tuberosities, pubic arch). The 'false pelvis' lies above the pelvic inlet and can be palpated on abdominal examination.
- Pelvic floor muscles (such as levator ani) support, and are integral to, the function of the anorectum, vagina, and bladder. They are innervated by the anterior 1° rami of S2, 3, 4.
- Anterior relations of the rectum (palpable during PR exam) are (from below up):
  - Women—vagina, cervix, pouch of Douglas.
  - Men—prostate, seminal vesicles, recto-vesical pouch.

Evaluation of peripheral vascular disease

Positioning and inspection

The patient should be examined in a warm environment at rest. Remember to take the pulse and BP, examine the abdomen (aneurysm, scars) and complete with a general cardiovascular examination (all peripheral pulses, heart sounds, carotid auscultation). Inspect the limb in the supine position, then elevated (passively), and finally dependent. Expose the entire limb, including the foot or hand, to allow a thorough inspection and remove any dressings. For venous disease, the patient should also be examined standing.

During supine inspection, look for the following:

- *Appearance*. Are there any areas of established skin necrosis (dry gangrene, e.g. apex of digits, between digits, heel of the foot)? Are there changes of chronic venous stasis (flare veins, venous eczema, lipodermatosclerosis, leg ulceration)? Colour. Waxy white suggests critical or acute ischaemia; blue and mottled suggests potentially irreversible acute ischaemia; dark red/purple suggests chronic ischaemia.
- *Colour changes during position*. Note the angle at which the skin of the limb blanches when passively elevated (Buerger's test). Normal limbs may not blanch at all. An angle of 15° or less suggests severe ischaemia. Note the presence of, and delay in, change in colour when the limb is dependent. Ischaemic limbs slowly turn deep scarlet (reactive hyperaemia).
- *Ulcers*. What is the location (digital or foot suggests arterial disease)? Be sure to inspect between the toes/fingers and on the plantar surface of the foot (especially for diabetic disease).
- *Venous inspection*. Stand the patient up. Inspect for varicose veins. Are they in the long saphenous or short saphenous distribution?

Palpation

- *Temperature*. Does the skin feel cold or warm? Is there a transition level?
- *Skin capillary compression and refill*. Normal is 2s or less. A delay of >5s suggests significantly reduced perfusion.
- *Peripheral pulses*. Start with the most proximal (major) vessels and work distally. Record if the pulse is normal (++), reduced (+), or absent (–). Record if there are any thrills palpable.
- In venous disease, tests of venous competence may be performed (➲ Varicose veins, pp. 800–1).
- *Surgical grafts*. Palpate the course of any surgical grafts and record the presence or absence of pulses.

Auscultation

Listen for bruits. Are there bruits in the proximal vessels (suggestive of stenosis)?

Investigations

Handheld Doppler (at the bedside) followed by Duplex USS or CT angiography are the main modalities for investigating vascular disease.

Handheld Doppler ultrasound

- Straightforward and portable "bedside" test for initial evaluation.
- Used to:
  - Confirm the presence of flow in a vessel or graft.
  - Evaluate the ankle–brachial pressure index (ABPI).
  - Evaluate the presence of reflux in veins.

Duplex Ulrasound (USS)

- Combined two-dimensional (2D) ultrasound image with Doppler-derived flow represented using colour, superimposed in real time.
- Arterial duplex: used for assessment of stenoses/occlusions.
- Venous duplex: used for assessment of reflux or thrombosis in deep and superficial veins.

CT angiography

- Requires multi-slice rapid acquisition ('helical'/'spiral') scanner.
- Images acquired in arterial and/or venous phase after IV injection of contrast.
- Three-dimensional (3D) reconstruction allows 'virtual angiogram' images to be produced.
- Fast, non-invasive and relatively safe
- Requires iodinated contrast which is nephrotoxic (relatively contraindicated in renal disease) and carries a small risk of allergy
- Presence of vessel wall calcification can obscure visualization of the lumen.

Digital subtraction angiography (DSA)

- X-ray following contrast injection into the vessel of interest, which removes background image (bone, etc.) to show the vessel lumen in black.
- Mainly used where endovascular intervention is required at the same sitting or previous investigations inconclusive.
- Invasive, requiring arterial puncture with the associated risks (e.g. false aneurysm or retroperitoneal haemorrhage with groin puncture).
- Requires iodinated contrast (so caution in renal disease and allergy).

Magnetic resonance angiography

- Provides images of an arterial tree based on the presence of arterial flow during scanning.
- Safe and non-invasive: requires no iodinated contrast, commonly gadolinium used to highlight flowing blood.
- Tends to overestimate degree of stenosis due to very low flow being underrepresented.

Evaluation of the skin and subcutaneous tissue

Assessment and description of a lump

Key features in the history include the following:

- Speed of development. Rapid increase in size is suspicious of malignancy (1° or 2°).
- Recent change in size. Suggests malignant change or infection in a previously benign lesion.
- Associated symptoms. Paraesthesiae or weakness suggests involvement of nerves; reduced movement suggests involvement of muscle.
- History of local trauma. May indicate a cause, although a previously undiagnosed underlying lump should always be suspected.

Consider the following when examining a lump

Basic facts

- Position.
- Size.
- Shape.

Features of infection or inflammation

- Temperature, 'calor'.
- Tenderness, 'dolor'.
- Colour, 'rubor'.

Features of malignancy

- Surface (e.g. craggy).
- Edge (e.g. irregular).
- Consistency (e.g. hard).

Features of fluid or vascular lesions

- Fluctuance and/or transilluminance (fluid-filled).
- Presence of a thrill (fluid-filled, connected to the vascular tree).
- Pulsatile (arterial) ± expansile (indicative of an arterial aneurysm).
- Audible bruit (arterial lesion).
- Compressibility (e.g. venous lesion or arteriovenous malformation).

Features of locoregional invasion

- Tethering to surrounding structures.
- Involvement of surrounding structures (e.g. nerves).
- Regional lymphadenopathy.

Lumps in detail

- Neck lumps (➔ Head and Neck surgery, pp. 293–309).
- Abdominal lumps and herniae (➔ Adominal Wall, pp. 431–47).
- Scrotal lumps (➔ Scrotal swellings, pp. 466–7).

Assessment and description of an ulcer

Key features in the history include the following:

- *Is it painful?* (diabetic, and neuropathic ulcers are often painless.)
- *Did it start as an ulcer or did a lump become ulcerated?* (suggests a malignancy in/of the skin)
- *Is there a history of underlying infection*, e.g. of bone?

Describe the basic morphology of the ulcer

- *Location*.
  - Over pressure points and bony prominences suggests pressure sore.
  - Medial shin suggests venous ulcer.
  - Lateral shin, dorsum of foot, and toes suggest arterial ulcer.
- *Edge*.
  - Sloping edge suggests a conventional ulcer (many aetiologies).
  - Rolled edge is typical of basal cell (BCC) or squamous carcinomas.
  - Everted edge suggests squamous or metastatic carcinomas.
  - Vertical edge (punched out) suggests arterial ulcer or chronic infection.
- *Base*.
  - Friable, red, and bleeding suggest venous or traumatic.
  - Slough suggests infection.
  - Black hard eschar suggests chronic ischaemia.
- *Discharge*. May suggest an underlying cause, e.g. intestinal fistula with enteric content.
- *Surrounding tissue*. Erythema and swelling suggest 2° infection.

Ulcers in detail

- Ulcers overview (➔ Ulcers, pp. 188–9).
- Diabetic ulcers (➔ The diabetic foot, pp. 794–5)
- Cutaneous malignancy (➔ Skin cancer, pp. 734–5).

Surgery at the extremes of age

Surgery is increasingly used in older and older patients, and the range of procedures available to surgeons for both the very elderly and the very young and neonates is increasing. Minimally invasive surgery is increasingly being offered to older patients at risk from open surgery. Both these groups need particular attention and have specific potential problems.

Surgery and the elderly

Common misconceptions corrected

- Elderly patients benefit just as much from potentially curative cancer surgery as younger patients. Cancers demonstrate the same range of behaviours in all ages and are neither more 'benign' nor less responsive to treatment in the elderly.
- Minimally invasive procedures in the elderly can offer all the benefits available to younger patients.
- 'Palliative' procedures for benign disease (e.g. cholecystectomy, joint surgery, eye surgery) are just as important in the elderly as they may allow preservation of independence and offer just as much improvement in quality of life as in the young.

Common problems in the elderly

- Multiple comorbidities and polypharmacy increase the scope for potential complications and drug interactions.
- Comorbidities can be 'silent', either due to atypical presentation or underreporting of symptoms (e.g. angina may not be manifest due to reduced mobility).
- Social, family, nursing, and medical support structures are often complex and easily lost during a hospital admission.
- Reduced or acutely impaired mental faculties may make history taking and consent taking difficult.
- Reduced or abnormal immune responses may reduce or impair some physical signs (e.g. clinically peritonism may be undetectable).
- The elderly are particularly prone to chronic malnutrition, increasing general complication rates, and the risk of pressure sores, etc.

Strategies for the management of the elderly

- Involve all the necessary specialties as soon as possible prior to admission for elective surgery, e.g. elderly care, anaesthetists, physicians.
- Consider pre-optimization in critical care (HDU), especially for urgent or emergency surgery.
- Start to plan for discharge on the day of admission and liaise with the GP and family, if necessary.
- Consider nutrition as soon as possible after surgery. Is hyper-alimentation necessary?

Surgery and the young

Although most surgery undertaken in neonates and very young children is done so by specialist paediatric surgical and nursing teams, most surgeons will care for young children at some time and the principles of care used in paediatric surgery can be usefully applied to older children.

Common problems in children

- Young children may not be able to accurately report symptoms, and illness behaviour is often non-specific.
- Cardiovascular responses in the young are excellent. Therefore, Tachycardia and particularly hypotension are (very) late signs of hypovolaemia.

► Tips for managing children

- Take the history from the parents or carers and the child.
- Remember infections are common and often present with non-specific signs.
- Consider non-surgical diagnoses at all times, e.g. meningitis, urinary sepsis, systemic viral infections – examine all systems.
- Involve the parents during examination/phlebotomy, e.g. examine or take blood while they are sitting on a parent's lap.
- Use LA cream on phlebotomy sites 30min in advance.
- Note children are less likely to cooperate with procedures under LA and will require GA for relatively trivial procedures.
- Make sure all prescriptions for drugs and fluids are written according to weight to avoid inadvertent adult dosing—if in doubt, ask.
- Fluid balance may be critical since small volume changes are highly significant in small children. Pay close attention to fluid resuscitation.

Paediatric surgery

- Conversion tables (➲ Principles of managing paediatric surgical cases, p. 522).
- Paediatric surgery (➲ Paediatric surgery, p. 522).
- Consent and children (➲ Consent, pp. 15–6).

Surgery in pregnancy

Pregnancy testing

- Urinary dipstick β-human chorionic gonadotrophin (HCG) is 91% sensitive (even lower for women self-testing). Specificity ranges from 61% to 100% if tested from the first day of the first missed period (2 weeks after ovulation).
- Blood β-HCG is almost 100% sensitive and specific and able to detect pregnancy 6–8 days after ovulation.
- False negatives/positives are most commonly due to user error.

Changes in anatomy and physiology

Pregnancy results in several changes relevant to surgery.

First trimester

- Drugs may have teratogenic effects (see Box 2.1).
- Reduced lower oesophageal sphincter (LOS) tone, increasing the risk of gastro-oesophageal reflux and aspiration when supine.

Second trimester

- Drugs may have adverse effects in fetal development or metabolism without causing gross malformation.
- ↑ susceptibility to urinary tract infections (UTIs), particularly ascending renal infections and pyelonephritis.
- ↑ risk of VTE rises in the second trimester and remains constantly raised in the third.
- ↑ susceptibility to superficial infections.

Third trimester

- Drugs may induce labour.
- Displacement of the mobile abdominal viscera superiorly and behind the enlarging uterus. In particular, the appendix comes to lie higher, in the RUQ.
- Risk of hypotension in the supine position due to inferior vena caval compression by the gravid uterus—this can be avoided by positioning the sedated or unconscious patient in slight lateral decubitus.

Risks of miscarriage

The risk of miscarriage related to surgical pathology and surgery varies according to trimester. It is highest in the first. The risk of viable premature labour rises in the third trimester. The risk of miscarriage induced by GA is always balanced against the risk induced by sepsis from untreated surgical pathology, particularly acute appendicitis. It is a common dilemma in surgical practice. Ultrasound imaging may be less useful due to poor views, and CT scanning is contraindicated due to radiation dose. MRI is often used (after first trimester). Diagnostic laparoscopy is contraindicated due to the effects of pneumoperitoneum on the pregnancy. The only way to a diagnosis may be surgery once important differential diagnoses have been excluded.

Common differential diagnoses of appendicitis in pregnancy

- Ectopic pregnancy complications.
- Pyelonephritis.
- Threatened miscarriage/placental abruption.

Box 2.1 Prescribing drugs in pregnancy

It is clearly unethical to screen drugs for harmful effects on the human fetus; many new and commonly used drugs have therefore never been used in pregnancy. Some older drugs have been used in pregnancy and are regarded as 'safe' in the absence of any reports of fetal harm. There is an important balance to maintain between treating serious illness in the mother and potentially harming the fetus.

Generally:

- Avoid prescribing drugs, if possible.
- Know the stage of the pregnancy; many drugs are only approved in particular trimesters.

▶ Check every drug that you prescribe in the BNF (or equivalent)

- If in doubt, seek specialist advice.
- Important teratogens include:
  - Thalidomide (an antiemetic).
  - Carbamazepine and sodium valproate.
  - Isotretinoin.
  - Tetracycline.
  - Warfarin.
  - Angiotensin-converting enzyme (ACE) inhibitors.
  - Lithium.
  - Methotrexate, cyclophosphamide.

Surgery and the contraceptive pill

(O)estrogen-containing contraceptive pills (OCPs) increase the risk of thromboembolic disease in women taking them prior to surgery. Progesterone-only contraceptives appear to pose little or no additional risk and may be continued during surgery. The increase in risk is related to the size of the operative procedure and the existing comorbidity; the advice is adjusted accordingly.

- Low-risk procedures, e.g. dental, day case, minor laparoscopic.
  - OCP may be continued.
- Medium risk, e.g. abdominal, orthopaedic, major breast surgery.
  - OCP should be discontinued at least 1 month prior to elective surgery.
  - Urgent or emergency surgery should be conducted with full thromboprophylaxis (➲ Prophylaxis—antibiotics and thromboprophylaxis, pp. 98–9).
- High risk, e.g. pelvic, lower limb orthopaedic surgery, cancer.
  - OCP should be discontinued at least 1 month prior to elective surgery.
  - Urgent or emergency surgery should be conducted with extended thromboprophylaxis (➲ Prophylaxis—antibiotics and thromboprophylaxis, pp. 98–9).

Surgery in endocrine disease

Diabetes

Specific perioperative risks

- Hypoglycaemia, hyperglycaemia, or ketoacidosis.
- Underlying DM-related comorbidity is often unrecognized (e.g. mild renal impairment, small-vessel coronary and cerebrovascular disease, mild autonomic neuropathy with associated reduced cardiovascular homeostatic responses).
- ↑ susceptibility to infection, poor wound healing.
- ↑ susceptibility to skin pressure necrosis.

Management of the diabetic patient

- Inform the anaesthetist, the diabetologist, and any specialists involved in the patient's ongoing care, e.g. nephrologists.
- Clarify if the patient is oral-controlled, insulin-dependent (low or high requirement), or brittle insulin-dependent since the risk of perioperative problems increases with each group.
- Diabetics should be first on operating lists to ensure timings can be as predictable as possible for blood sugar (BS) management.
- Check preoperative investigations for signs of other comorbidity.
- Ketoacidosis in the perioperative period is associated with very high morbidity and mortality and should be avoided at all costs.

Minor surgery

- Oral-controlled—give normal regimen.
- Insulin-controlled—omit preoperative insulin on day of surgery; monitor BS every 4h; restart normal insulin once oral diet is established.

Major surgery

- Oral-controlled—omit long-acting hypoglycaemics preoperatively. Monitor BS every 4h. If BS exceeds 15mmol/L, start IV insulin regimen.
- Insulin-controlled—commence on IV insulin sliding scale preoperatively once NBM and continue until normal diet is re-established. Check BS every 4h. Restart normal insulin regimen (initially at half dose) once oral diet is established.

Emergency surgery

- Check for existing ketoacidosis. If present, use medical treatment algorithm to control BS and postpone surgery until BS <20mmol/L unless the condition is life-threatening.
- Use IV insulin sliding scale for all patients to optimize BS control. A typical IV sliding scale (soluble insulin with 5% glucose) is:
  - BS <4mmol/L: infusion 0.5U/h + consider medical review.
  - BS 4–15mmol/L: infusion 2.0U/h.
  - BS 15–20mmol/L: infusion 4.0U/h.
  - BS >20mmol/L: infusion 4.0U/h + consult diabetology team and consider treatment as for ketoacidosis.

Steroids

Specific perioperative risks

Oral steroids are used to treat a number of common illnesses, including rheumatoid arthritis (RA), severe asthma, and COPD. Steroids reduce neutrophil and fibroblast function and immune response and lead to irreversible changes in connective tissue. Long-term use of systemic steroids results in adrenal suppression. The following problems are associated with chronic steroid use.

- Addisonian (hypoadrenal) crisis (see Box 2.2).
- ↑ susceptibility to infection.
- Poor wound healing, including anastomotic leaks.
- Osteoporosis.
- Patients on long-term inhaled steroids, e.g. for asthma and COPD, are not high risk as there is minimal systemic absorption.

Management of the patient on steroids

- If the steroid dose can be weaned preoperatively, this should be done.
- Prescribe IV hydrocortisone 25–100mg qds (roughly corresponding to 2.5–20mg once a day (od) of prednisolone) to start on the morning of surgery and continuing until the patient is able to go back to their oral steroids.

Thyroid disease

(➲ Goitre, pp. 330–1; Thyrotoxicosis, pp. 332–3.)

Box 2.2 Addisonian (hypoadrenal) crisis

Stresses, such as surgery and sepsis, require ↑ adrenal secretion of corticosteroids; failure to mount this response can result in an Addisonian (hypoadrenal) crisis. The following groups of patients are at high risk:

- Any patient currently taking >5mg prednisolone for >2 weeks.
- Any patient who reduced long-term steroids within 2–4 weeks.
- Patients who have undergone adrenalectomy.

Clinical features

- Lethargy and malaise.
- Abdominal pain, often poorly localized (may present as an acute abdomen).
- Nausea and vomiting.
- Hyponatraemia, hypotension, hypoglycaemia.
- Coma, death.

Management

- Treat with IV hydrocortisone 100mg four times a day (qds) or 400mg infusion over 24h, while the patient is NBM.
- Fluid resuscitation with normal saline.
- IV 50% glucose to treat hypoglycaemia (titrate against BS).

Surgery and heart disease

Ischaemic heart disease

Risk factors include (♂ >45y; ♀ >55y), family history of early MI, current or treated ↑ BP, smoking, DM, and ↑ cholesterol. Assess severity—quantify exercise tolerance; enquire about palpitations, orthopnoea, use of anti-anginals, previous MI, percutaneous coronary intervention (PCI), or coronary artery bypass graft (CABG). The ECG is the most important routine screening test, but it is normal in about one-third of patients with proven ischaemia. Symptomatic patients undergoing major surgery should be discussed with a cardiologist, with a view to optimizing medications.

Myocardial infarction

The risk of a perioperative MI relates to past history and risk factors.

- Overall population incidence after abdominal surgery: 0.5%.
- Incidence with pre-existing cardiovascular symptoms: 2%.
- Incidence with previous MI (old): 5–10%.
- Incidence after recent MI: 25% (70% will die with re-infarction).

Strategies to reduce risk

- Non-urgent surgery should be delayed for at least 6 months following acute MI and possibly acute ischaemia. Cancer surgery may be undertaken if the risk of disease progression is felt to outweigh the ↑ perioperative mortality rate.
- Ensure all normal cardiovascular medication is continued up to and through surgery. Control any new symptoms of angina if surgery is urgent.
- Continue antiplatelet medication if not contraindicated.
- Consider involving the critical care services (HDU) for the perioperative period.

Valvular heart disease

Cardiac murmurs are common. Request a transthoracic echocardiogram (TTE) to evaluate the lesion and discuss abnormalities with a cardiologist.

- *Severe aortic stenosis* carries a high risk of mortality - elective surgery should be postponed; high-gradient aortic stenosis carries an associated mortality of 10% with non-cardiac surgery.
- *Severe mitral stenosis* can lead to pulmonary oedema and heart failure—major elective surgery should be postponed until lesion is corrected.
- *Aortic regurgitation (AR)* requires attention to fluid and rate control. Antibiotic prophylaxis should be given, but surgery can go ahead.
- *Mitral regurgitation (MR)* should be managed with diuretics and vasodilators. Left ventricular (LV) function is frequently overestimated in MR.
- *Prosthetic valves* have several associated issues.
  - *Mechanical valves require anticoagulation*. Stop warfarin 5 days preoperatively and bridge with heparin once the international normalized ratio (INR) becomes subtherapeutic.
  - Stop IV heparin 2-6h pre-surgery and resume as soon as surgical bleeding is no longer a problem until INR is therapeutic. Thrombosis is most likely in mechanical valves, poor left ventricle (LV), previous thromboembolic disease, and less so in rate-controlled atrial fibrillation (AF).

- In surgery for life-threatening bleeding, e.g. bleeding peptic ulcer, intracranial haemorrhage, it may be necessary to reverse anticoagulation for several days. Liaise closely with cardiology.
  ▶ Prosthetic valves no longer require antibiotic prophylaxis for procedures that cause bacteraemias;[1] if in doubt, discuss with cardiology.

Arterial hypertension

Control of BP preoperatively may reduce the tendency to perioperative ischaemia. If severe (>180mmHg), surgery should be delayed until control is obtained.

- Review existing antihypertensive management or start treatment: discuss with the anaesthetist.
- Look for evidence of end-organ damage (renal, neurological) and associated heart disease.
- Look for rare, but important, causes: phaeochromocytoma, hyperaldosteronism, coarctation of the aorta, renal artery stenosis.

Congestive cardiac failure

Heart failure is associated with a poorer outcome in non-cardiac surgery. Risk factors include ischaemic and valvular heart disease.

- Look for: S3, pedal oedema, raised jugular venous pressure (JVP), bibasal crepitations. Order chest X-ray (CXR) if suspected.

Cardiac arrhythmias

Arrhythmias and conduction defects are common. Asymptomatic arrhythmias are not associated with an increase in cardiac complications, but look for underlying pathologies, e.g. IHD, drug toxicity, metabolic derangements.

- *High-grade conduction abnormalities*, e.g. complete heart block, should be discussed with a cardiologist. Pacing may be indicated.
- *Patients with known AF*, particularly with history of embolic stroke or structural cardiac defect normally take warfarin. Request a cardiology review preoperatively if rate control is poor.
- *Permanent pacemakers or intracardiac defibrillators (ICDs)*. Diathermy may cause the pacemaker to reset or completely inhibit pacing and trigger ICD discharge. Pacemakers and ICDs should be evaluated by a cardiac technician pre- and post-operatively. Pacemakers should be changed to fixed-rate pacing for surgery and then reprogrammed after surgery. ICDs should be switched off to prevent discharge, and external fibrillator pads positioned on the patient. If defibrillation or synchronized cardioversion is required, place the paddles as far from the pacemaker or ICD as possible. The type of diathermy used should be considered. Monopolar is not absolutely contraindicated, but bipolar may be preferable.

Reference

1 National Institute for Health and Care Excellence (2016). *Prophylaxis against infective endocarditis*. Available at: https://www.nice.org.uk/guidance/cg64

Surgery and respiratory disease

Surgery and smoking

Smoking tobacco increases the risks of anaesthesia and many of the risks of surgery. There is a 6-fold increase in post-operative respiratory complications among patients smoking in excess of ten cigarettes per day.

Effects of smoking

- Reduction in general and specific immune function via reduced neutrophil chemotaxis and reduced natural killer (NK) cell efficacy.
- ↑ platelet aggregation—probably explaining the ↑ risk of perioperative acute MI and cerebrovascular accident (CVA) in smokers.
- Reduced oxygen (O_2)-carrying capacity of blood per unit volume due to the presence of carboxyhaemoglobin increasing the risk of tissue hypoxia in susceptible organs.
- ↑ upper aerodigestive mucosal secretions. This worsens initially after stopping smoking until the chronic effects on the mucosa wear off.
- Reduced mucociliary escalator function.
- Reduced lung compliance and ↑ 'closing volume' of the small airways, increasing the risk of air trapping, especially whilst supine in the post-operative period.

Stopping smoking

- Within 48h: carboxyhaemoglobin is cleared from the blood; platelet aggregation begins to return to normal.
- Within 7 days: neutrophil, macrophage, and NK cell function improves. Mucus production temporarily increases, but mucociliary escalator function takes up to 6 weeks to recover, leading to a 'rebound' effect.
- Within 6 weeks: upper aerodigestive function returns to underlying level; lung dynamics improve to 'normal' levels (depending on the extent of fixed parenchymal disease).

The optimal time for stopping smoking is at least 6 weeks prior to surgery, but a minimum of 7 days is required to reduce the 'rebound' effects of stopping on upper aerodigestive tract function.

▶ *Mitigating the effects of smoking in the post-operative period*

Active and recently stopped smokers should receive extra attention to prevent the risks associated with smoking and surgery.

- Ensure patients remain well hydrated until oral intake is restored.
- Use thromboembolic prophylaxis in most cases.
- Use preoperative chest physiotherapy and education on breathing and coughing techniques.
- Mobilize as soon as possible post-operatively.
- Consider the use of epidural anaesthesia to improve compliance with post-operative physiotherapy.
- Use preoperative and post-operative nebulized saline (5mL qds).
- Ensure post-operative analgesia is effective.

Respiratory conditions

Respiratory tract infection

An active respiratory tract infection may be sufficient reason to cancel elective patients, so ask about cough, fevers, and sputum, but minor colds and nasal discharge may not prevent GA.

- If you suspect the patient has a respiratory tract infection, check vital signs, inflammatory markers, and CXR.
- Elective patients should be cancelled and asked to return in 2 weeks if their symptoms are better.
- Reserve antibiotics for patients with suspected bacterial infections; most acute respiratory tract infections are viral.

Asthma

- Assess severity of asthma by asking about hospital admissions, inhalers, nebulizers, peak expiratory flow rates (PEFRs), and home O_2.
- Elective surgery should ideally coincide with remission of symptoms.
- Identify patients on long-term steroid therapy.
  - Sometimes it is possible to time surgery to coincide with a reduction in steroids, but this requires several weeks' notice.
  - Any patient taking >5mg daily prednisolone and undergoing inpatient surgery or presenting with sepsis should be started on an equivalent dose of IV hydrocortisone; adrenal suppression may otherwise result in an Addisonian crisis (➲ Steroids, p. 79).
- Patients receiving GA generally experience deterioration in their lung function (➲ Respiratory complications, pp. 134–5). Prophylactically increase their normal therapy by converting inhalers to nebulizers and increasing frequency.

Chronic obstructive pulmonary disease

- If dyspnoea is the prominent symptom and the patient has COPD, get lung function tests, including blood gases.
- Admitting these patients a few days early for physiotherapy, education, and nebulizers can reduce the length of hospital stay.
- Patients receiving GA generally experience deterioration in their lung function (➲ Respiratory complications, pp. 134–5). Prophylactically increase their normal therapy by converting inhalers to nebulizers and increasing the frequency.
- Prescribe 6-hourly 5mL of nebulized saline and give humidified O_2 wherever possible (to prevent mucus plugging).
- Ensure the patient gets twice-daily chest physiotherapy.
- Ensure the patient is on their usual inhalers and consider converting these to nebulizers for major surgery (➲ Respiratory complications, pp. 134–5).

Surgery in renal and hepatic disease

Renal impairment

Renal impairment covers a spectrum, ranging from patients with subclinical dysfunction (normal serum creatinine (Cr) and urea, but borderline Cr clearance) to patients with end-stage renal failure. It is helpful to consider these patients in two main groups: patients with chronic renal impairment and dialysis-dependent patients. Post-operative management of renal impairment is discussed later (➲ Renal complications, p. 136); dialysis is discussed under ➲ Renal support, pp. 164–5.

Chronic renal impairment

Surgery may precipitate acute renal failure in patients with chronic renal impairment.

- Avoid hypovolaemia and hypotension. Ensure these patients receive adequate IV hydration if they are to be NBM for any length of time.
- Avoid nephrotoxic drugs wherever possible, including non-steroidal anti-inflammatory drugs (NSAIDs), aminoglycosides, ACE inhibitors, and radiological contrast.
- Reduce doses of drugs with renal elimination, e.g. morphine, low-molecular weight heparin (LMWH), digoxin, and request appropriate levels frequently.

Patients with established renal failure, on dialysis

- For patients undergoing major surgery, discuss their post-operative management with the anaesthetist and ICU as early as possible. Dialysis should be performed the day before surgery.
- Patients must have full blood count (FBC) and urea and electrolytes (U&Es) on admission and pre- and post-dialysis, and twice-daily U&Es post-major surgery until the patient is stabilized on their normal dialysis regime.
- Reduce doses of drugs with renal elimination, e.g. morphine, LMWH, digoxin, and request appropriate levels frequently.
- If the patient is normally anuric, there is little point in inserting a urinary catheter, which exposes them to unnecessary infection risk.
- Note the sites of arteriovenous fistulae. Never use them for phlebotomy or cannulation and avoid using BP cuffs on that side.
- These patients are prone to several problems:
  - ► Hyperkalaemia, acidosis, and pulmonary oedema are potential life-threatening emergencies (➲ Renal complications, pp. 136–8).
  - Infection.
  - Anaemia and coagulopathy.
  - Fluid and electrolyte disturbances.
  - Metabolic acidosis.
  - Systemic HTN, pericarditis.

Hepatic impairment

The risk posed by liver disease to patients undergoing general surgery was graded by Child and Turcotte (see Box 2.3). Child grade C is associated with high perioperative mortality.

Liver failure causes the following problems: hypoglycaemia; hepatic encephalopathy; coagulopathy (↑ INR); ascites; and infection.

Box 2.3 Child's classification of surgical risk in hepatic dysfunction

A (minimal risk)	B (moderate risk)	C (advanced risk)
• Serum bilirubin <20mg/L	• Serum bilirubin 20–30mg/L	• Serum bilirubin >30mg/L
• Serum albumin >35g/L	• Serum albumin 30–35g/L	• Serum albumin <30g/L
• No ascites	• Controlled ascites	• Uncontrolled ascites
• No focal neurology	• Minimal neurological dysfunction	• Coma
• Excellent nutrition	• Good nutrition	• Cachexia

- Several factors may cause acute decompensation of mild hepatic impairment and should be avoided or treated aggressively in this group:
  - Infection, especially bacterial peritonitis; sedation; diuretics; constipation; electrolyte imbalance; dehydration; and hypotension.
- Preoperatively: check hepatitis serology, request liver ultrasound in newly diagnosed hepatic impairment; discuss requesting additional blood products with haematology; discuss doses of standard medication with a specialist.

Jaundice

Patients with obstructive jaundice are at risk of developing renal failure post-operatively (hepatorenal syndrome). This is thought to be due to the nephrotoxic effect of toxins normally eliminated by the liver, as well as circulatory changes.

- Ensure adequate hydration. When the patient is NBM, prescribe IV normal saline 1L over 6–8h.
- Insert a urinary catheter and start an hourly fluid balance chart.
- Measure U&E and liver function tests (LFTs) daily.
- Coagulopathy in long-standing cholestatic jaundice may be improved with vitamin K 1mg IV—discuss with haematology.
- Avoid or reduce the doses of hepatotoxic drugs and drugs with hepatic elimination.

Surgery in neurological disease

Cerebrovascular accidents (stroke)

- Ischaemic events are associated with a risk of re-infarction or extension of the infarct due to interference with cerebrovascular autoregulation by anaesthetic agents. Autoregulation is re-established in around 6 weeks.
- Haemorrhagic infarcts are associated with a small ↑ risk of further bleeding, especially if the patient is given thromboprophylaxis.

Strategies to reduce risk

- Delay all non-essential surgery for 6 weeks following infarcts, especially ischaemic ones.
- Consider omitting thromboprophylaxis in patients with a recent haemorrhagic event.
- Ensure BP is well controlled (the prevention of both hypotension and HTN) in the perioperative period to reduce fluctuations in cerebral blood flow.
- Avoid positioning the patient head down on the operating table as this increases cerebral venous pressure.

Epilepsy

Paroxysmal neuronal discharge from various areas of the brain causes a range of disturbances that may affect consciousness (seizures or absences), movement, or sensory perception. In addition to epilepsy, cerebral space-occupying lesions, uraemia, cerebral oedema, drug toxicity, and hypercalcaemia may cause the same problems. In patients with known epilepsy, the following measures are advised.

- Try to establish the normal frequency and severity of seizures, what form they take, and features, if any, of the prodrome.
- Ensure that normal anticonvulsant medication is continued whilst the patient is NBM preoperatively and immediately post-operatively.
- If this is not possible, discuss the most appropriate bridging regime with the anaesthetist or neurologist
- ❗Phenytoin interacts with a number of drugs used perioperatively.
  - ↑ elimination of prednisolone, warfarin, lidocaine. oral absorption of phenytoin ↑ by amiodarone, fluconazole, omeprazole, and paroxetine; ↓ by antacids containing Mg/Ca/Al and enteral feeds.

Myasthenia gravis

An antibody-mediated autoimmune disease with insufficient muscle acetylcholine (ACh) receptors, leading to muscle weakness. Usually found in young adults, the disease presents with extraocular (ptosis, diplopia), bulbar (weak voice), neck, limb girdle, distal limb, and finally trunk weakness. Patients may present for thymectomy as a specific treatment or for incidental surgery.

Management includes the following:

- Continue normal medication.
- Consider if elective post-operative ventilation is required—indicated in major thoracic or upper abdominal surgery or if the patient's vital capacity is <2L. Discuss with an anaesthetist and ICU. (If ventilation is prolonged post-operatively, a tracheostomy may be required. This should be discussed with the patient at the time of consent.)
- Observe for post-operative respiratory failure which may occur as a result of muscle weakness. Precipitants include: hypokalaemia, infection, over- or undertreatment, and emotion/exertion.

Fluid optimization

Identifying patients in need of fluid optimization

Any patient may be in need of preoperative fluid resuscitation, but several groups are typically affected. Remember to think of less obvious cases of fluid depletion—there are typically more patients who would benefit from fluid optimization than receive it.

- Acute presentations with vomiting or diarrhoea, including intestinal obstruction, biliary colic, and gastroenteritis.
- Acute presentations where the patient has been immobile or debilitated for a period before presentation, causing reduced fluid intake, e.g. pancreatitis, chest infections, acute-on-chronic vascular insufficiency, prolonged sepsis with pyrexia.
- Elderly patients in whom reduced renal reserve makes fluid balance control less effective.
- Drugs that impair renal responses to fluid changes, e.g. diuretics.
- Patients with low body weight with overall lower total body fluid volume in whom similar losses have a greater effect.
- Children are more susceptible to fluid depletion and may not show such obvious physical signs.

Fluids used for optimization

The most important aspect of fluid optimization is using the correct volumes at the correct rate. Other than exceptional circumstances, isotonic crystalloids are the fluid of choice to correct imbalances.

- Isotonic ('normal') 0.9% saline—the most widely used fluid. Provided there is adequate renal function, isotonic saline prevents rapid cellular fluid shifts during rehydration and excess sodium (Na^+) is excreted via the kidneys. Potassium (K^+) should be added only if ↓ K^+ is present or likely (e.g. prolonged vomiting, pancreatic or small bowel fistula). Other crystalloids include glucose (4%), Hartmann's solution, and Ringer's lactate solution (technically, the closest fluid to serum composition, although theoretical advantages are of limited practical value).

Fluids that should only be used in very specific circumstances include hypertonic (1.8%) or hypotonic (0.45%) saline since they risk causing significant fluid shifts in and out of cells, which can cause cellular injury, particularly to neurons. If there is a significant disorder of Na^+ balance that may require non-isotonic fluid optimization, the patient is likely to require optimization in HDU.

How to give the fluids

Before giving fluid, it is important to assess the volume of depletion. It is rarely possible to use estimates of losses due to vomiting or diarrhoea as these are wholly inaccurate. Useful calculations include the following:

- Body weight on admission (provided a recent accurate body weight during normal health is known) since acute weight loss is mostly water.
- Haematocrit on admission (provided a recent haematocrit during normal health is known) since the degree of haemoconcentration is due to fluid depletion. An approximate calculation is given by:

  Fluid depletion (L) = (PCV1 – PCV2)/PCV1) × 0.7 × weight (kg)
  (PCV1 = normal haematocrit; PCV2 = current haematocrit)

- Serum urea is raised disproportionately more than serum Cr in dehydration, renal disease, GI bleeds, and acute proteolysis.
- Signs of extracellular fluid (ECF) depletion (lax skin tone, reduced sweating, dry mucosae) are often misleading and can be affected by age and underlying diseases, including pyrexia and tachypnoea.
- Signs of intravascular volume depletion (hypotension, tachycardia) may be unreliable and usually only occur with loss of 10–15% of body water.

Once the volume of fluid required is assessed, it can be given. There are some broad rules on how to give fluid resuscitation.

- Young, fit patients with normal renal and cardiac function can usually be given up to 15% of body fluid volume by rapid infusion. A typical regimen might include: 1000mL of 0.9% saline over 2h, further 1000mL infusions of 0.9% saline over 4h each until corrected.
- Elderly patients and patients with renal or cardiac impairment should have infusions more slowly to prevent acute intravascular volume overload. A typical regimen might include: 1000mL of 0.9% saline over 4h, 500mL infusion of 0.9% saline over 3–4h, with regular review of vital signs, including chest auscultation. Complex patients or those not responding to initial treatment should be discussed with seniors and/or other specialists as they may require monitoring in critical care.

Monitoring fluid optimization

Methods of assessing the progress of fluid optimization include the following:

- Skin turgor and mucosal hydration change slowly after optimization and are unreliable guides.
- One-hourly urine output measurement is a good guide to renal blood flow, which indirectly relates to intravascular fluid volume and cardiac output (CO). It is an easy and reliable indicator of adequate blood volume repletion. It is not a good indicator of total body water and there may be significant intra- and extracellular depletion in the presence of an acceptable urine output. A commonly used minimum is 0.5mL/kg/h.
- Monitoring of serum urea is an approximate guide, provided renal function is adequate and there is no acute GI bleeding or proteolysis.

In the emergency situation, particularly where patients require urgent surgery and fluid optimization prior to anaesthesia, more rapid fluid infusions may be required and it may be appropriate for this to be monitored on HDU.

Preoperative management of anaemia

Definition

Hb <13g/dL for both sexes.[1]

Classification

Traditionally classified based on mean cell volume (MCV) and mean cell haemoglobin (MCH) to identify potential causes (see Table 2.1).

Table 2.1 Common causes of pre-operative anemia

MCV	Cause	MCH
Microcytic	Iron deficiency	Hypochromic
	Thalassaemia	Hypochromic
	Chronic disease	
Normocytic	Acute blood loss	Normochromic
	Chronic disease	
Macrocytic	Alcohol dependence	Normochromic
	B12/folate deficiency	
	Hypothyroidism	Normochromic
	Pregnancy	
	Haemolytic	

Current guidance on diagnosis

Iron/haematinic deficiency and blood loss are common and correctable factors and should be investigated appropriately. Major elective surgery (expected blood loss >500mL) should be postponed until correctable causes of anaemia are investigated and addressed. Current guidelines[1] suggest serum ferritin level should also be used, and if <30 micrograms/L, it is the most sensitive and specific test to determine *absolute iron deficiency anaemia*. In the presence of inflammation (CRP >5 ± transferrin saturation (TSAT) <20%), a serum ferritin of <100 micrograms/L indicates iron deficiency anaemia.

Prevalence and outcomes of perioperative anaemia

Recent large multicentre studies and international registry databases have demonstrated that not only is perioperative anaemia common in surgical patients (30–35% prevalence), but it is also associated with ↑ morbidity and mortality with *any* severity of anaemia. Additionally, if combined with other perioperative risk factors, outcomes are worse than with either one alone by a factor of 3.5–7.

Assessment and management of anaemia

There are reliable data to suggest that patients will wait up to 2 months on average for elective surgery, and, as such, ample time to check and correct anaemia.

- If there is a >10% risk of transfusion and/or >500mL of blood loss is expected, lab investigations must be done immediately to assess haematinics.
- If Hb <13g/dL, look for causes and correctable factors. It is important to investigate signs and symptoms of overt and occult blood loss. Some patients will require further investigation such as radiographic imaging and/or endoscopic examination.
- If ferritin <20 micrograms/L with no inflammation (CRP/TSAT normal) OR ferritin <100 micrograms/L with CRP >5 and/or TSAT <20%, commence preoperative iron replacement therapy.
- If iron replacement therapy is commenced, non-urgent surgery should be postponed until target Hb is achieved.

Treatment options for correction of anaemia

Preoperative anaemia

- ~12% of patients will present for surgery with *B12 and/or folate* deficiency and this can be corrected with IM or PO supplementation for 2–3 weeks.
- Iron deficiency anaemia can often coexist. The choice of PO versus IM supplementation will be governed by severity, predicted malabsorption enterally, and ease of administration for the individual patient.[2]
  - Absolute iron deficiency anaemia can be corrected by administration of PO ferrous sulfate 400–600mg/day for 6–8 weeks.
  - If surgery is more urgent, IV iron therapy has been shown to be highly efficacious, with no difference in adverse effects compared to PO therapy or placebo. It may be administered in 1000–1500mg dose IV over an hour, with a peak rise in Hb seen over 1–3 weeks.
  - For patients who have had iron deficiency treated or excluded (ferritin >100 micrograms/L) and are still anaemic, subcutaneous (SC) erythropoietin (EPO) can be administered at a dose of 300–600mg once weekly for 4 weeks.

► Many hospitals have perioperative clinics jointly managed by surgical, anaesthetic, and critical care teams, and have local perioperative anaemia pathways that are an invaluable resource.

Post-operative anaemia

- It is good practice to check all patients' post-operative Hb by HemoCue® either in recovery or when returned to the ward. Formal FBC can usually be done on the first day after surgery.
- If there has been moderate to large volume blood loss intraoperatively and/or the patient is expected to bleed post-operatively due to known coagulopathy or for surgical reasons, the patient should be monitored in an appropriate setting, as judged by a senior clinician. Use of the National Early Warning Score (NEWS) to detect clinical deterioration *early* is critical to the management of these patients.[3]

- NICE guidelines recommend that for the majority of patients, the transfusion threshold for post-operative anaemia should be restrictive (defined as <7g/dL), with an Hb target of 7–9g/dL post-transfusion, except for:
  - Massive haemorrhage.
  - Acute coronary syndrome (target 8–10g/dL).
  - Chronic anaemia requiring regular transfusions.
- Any patient experiencing ongoing post-operative blood loss must be assessed regularly and expeditiously. The senior surgeon must be informed. Critical care outreach teams can be a valuable source of help.

References

1 Muñoz M, Acheson AG, Auerbach M, *et al*. (2017). International consensus statement on the peri-operative management of anaemia and iron deficiency. *Anaesthesia* **72**: 233–47.

2 Devalia V, Hamilton MS, Molloy AM; the British Committee for Standards in Haematology (2014). Guidelines for the diagnosis and treatment of cobalamin and folate disorders. *Br J Haematol* **166**: 496–513.

3 Royal College of Physicians (2017). *National Early Warning Score (NEWS) 2*. Available at: https://www.rcplondon.ac.uk/projects/outputs/national-early-warning-score-news-2

Nutrition in surgical patients

Nutrition is critical to the well-being of surgical patients. Timely nutritional support helps reduce acute catabolism and resultant skeletal muscle weakness due to ↑ metabolic demands. It is a common factor that influences the outcome of surgical patients.

Incidence of pre-existing malnutrition is substantial and rises with age. Anticipate those patients with higher-than-normal nutritional requirements (e.g. severe burns, severe sepsis, intestinal fistulae, advanced malignancy, immunosuppression); even if they are well nourished prior to illness, they may need nutritional support to prevent excessive acute catabolism due to ↑ metabolic demands.

Assessment of nutritional status

All patients should be considered for nutritional assessment. Many methods can be used:

- Body mass index (BMI) (weight/height^2 in kg/m^2). Relatively insensitive for all but major malnutrition. A BMI of 18–25 is normal; <18 is underweight; >30 is obese.
- Triceps skinfold thickness. Easy to perform and a good measure of body fat as a marker of chronic nutritional status.
- Grip strength. Easy repeatable index of lean skeletal muscle.
- Serum albumin. Poor indicator of acute nutritional status. Responds slowly to nutritional supplementation and is affected by many other factors.
- Serum transferrin. Accurate, responsive indicator of acute status and response to treatment. Not commonly used.

Effects of protein–calorie malnutrition

- Reduced neutrophil and lymphocyte function.
- Impaired albumin production.
- Impaired wound healing and collagen deposition.
- Skeletal muscle weakness ('critical illness myopathy'), with resultant increase in respiratory and abdominal complications.
- Micronutrient deficiencies may cause specific clinical syndromes.

Types of nutritional support

- Oral supplementation. High-calorie, high-protein nutritional supplements (e.g. Fortisip®, Calshakes®, Ensures®/Enlives®). The oral route is always to be preferred in nutritional supplementation. It promotes the normal health of the GI flora and has been shown to reduce the risk of complications after GI surgery.
- Nasogastric (NG)/nasojejunal feeding. Often used in addition to oral supplementation. Sometimes given overnight to reduce the impact on appetite suppression during the day.
- Feeding gastrostomy/jejunostomy (via a surgically implanted tube). Not routinely used. Reserved for patients where the GI tract is functioning but unable to take via the oropharyngeal route.
- Parenteral nutrition. May be central or peripheral.

(➲ Nutritional support, p. 166.)

Enhanced recovery after surgery

Enhanced recovery after surgery (ERAS) is one of the many approaches to patient recovery, aiming to minimize perioperative physiological derangement/stress response to surgery, optimize speed of recovery, and reduce complication rates. It is usually applied to otherwise healthy individuals who do not need particular preoperative correction. The major areas of consideration are as follows.

Nutrition

- Preoperative carbohydrate loading, often given as oral solutions over the 24h prior to surgery, including up to 4h prior to anaesthesia (thought to reduce the early catabolic response to major surgery).
- Early re-introduction of full nutrition: carbohydrate-rich fluids, from 6h post-surgery, nutritional supplements and 'light diet' components from 48h post-op to encourage 'immediate' return of GI tract function.

Anaesthetic technique

- Avoiding opiate use, e.g. morphine, including in PCA and epidurals, to prevent reduction of GI tract motility and nausea associated with opiates.
- Avoiding the use of epidurals, to improve early mobilization and reduce cardiovascular and GI effects of autonomic spinal blockade.
- Use of regional LA-based techniques (e.g. transversus abdominis plane (TAP) block, regional LA infiltration, or infusional catheters) to minimize central nociceptor input, which may enhance the systemic stress response to surgery.

Surgical technique

- Laparoscopic or other minimally invasive techniques aim to reduce the metabolic response to surgery, aid early mobilization, and reduce GI tract exposure in abdominal surgery.
- Avoiding bowel preparation for abdominal surgery reduces the risk of fluid and electrolyte imbalances and disruption to GI tract flora, and is associated with fewer GI complications (e.g. anastomotic leakage).

Physiotherapy

- Early mobilization, including specific exercises; sitting out within 12h of surgery; walking within 48h of surgery. Perioperative respiratory exercises.

Nursing

- Intensive patient preparation: preoperative education on what to expect. Intensive perioperative and post-operative nursing input: encouraging early re-establishment of diet, mobilization, and self-care.

Although intensive and demanding, ERAS-type protocols are as effective in the elderly as in the young. They are not suitable for: some insulin-dependent diabetics, patients with pre-existing significant nutritional compromise, and patients with cognitive impairment.

Getting the patient to theatre

Preparing the patient for a surgical procedure is all about organization and routine. If the preparation is not good, much can go wrong, with major consequences for the patient.

Background paperwork

Ensure the theatre or endoscopy list is correctly filled in and available well in advance. The list should specify the patient's name, hospital number, location, and operation details, as well as the operating surgeon and anaesthetist.

Patient paperwork

- Make sure the **medical notes** are available and contain the most up-to-date history and examination for this admission.
- Check the **blood results** are up-to-date and pay attention to specific blood results, e.g. K^+ especially in patients with renal failure, clotting function in anticoagulated patients, calcium (Ca^{2+}) in (para-) thyroidectomy.
- Ensure that if the patient requires any **blood or blood products**, these are available or requested from the transfusion department. (Most hospitals have protocols for the correct number of units of blood.)
- Ensure relevant **imaging results** are available.
- Check that the **consent form** has been completed and is in the medical notes.
- The **drug chart** should be completed.

Patient preparation

- Check that the operation side/site is marked on the patient (if relevant). This must be done with the patient awake and verified by the nursing staff. Also check that the patient has been marked by any relevant specialists (stoma care if a stoma is possible; prosthetist for amputees).
- Find out well in advance if any specific preparation is required, e.g. bowel preparation.

Bowel preparation

Used to empty the large bowel, for operations on or around the large bowel. Types of preparation include the following:

- Stimulant mechanical bowel preparation, e.g. sodium picosulfate, two sachets taken with plenty of fluid at least 8h prior to surgery. Should be avoided whenever there is a risk of obstruction.
- Osmotic mechanical bowel preparation, e.g. magnesium citrate, Klean Prep®, 2–4 sachets taken dissolved in water up to 8h prior to surgery. Used for most colonic operations where bowel preparation is required, colonoscopy, and CT colonography.
- Stimulant left colon preparation, e.g. phosphate enema. Used for operations on the rectum/anus or for flexible sigmoidoscopy.
- Mechanical bowel preparation is less commonly used than previously. There is evidence that it may actually increase the septic complication rates following bowel surgery and has well recognized side effects, including: electrolyte imbalances and hypovolaemia, especially in the elderly; and nausea and vomiting (particularly with large-volume osmotic preparations).

Anaesthetic premedication

Used to relax the patient to reduce anxiety during anaesthetic preparation and decreases the amount of anaesthetic agent required for induction of anaesthesia.

Agents used include: benzodiazepines, often given 1–2h preoperatively on the ward (e.g. diazepam PO) or shortly before some procedures (e.g. midazolam 5mg IV). Hyoscine butylbromide is rarely given, but sometimes used to reduce upper aerodigestive tract secretions.

Prophylaxis—antibiotics and thromboprophylaxis

Antibiotic prophylaxis

- Prophylactic antibiotics are used to reduce the risk of SSI and are usually of very short course (1–3 doses).
- Active antibiotic treatment for established infections encountered during surgery may be for 5 or more days.
- Most prophylaxis is directed at prevention of infection of the surgical wound or to counter the effect of potential spillage of organisms from colonized organs such as the bowel once it has been opened.

(➲ Antibiotic prescribing in surgery, p. 46.)

Thromboprophylaxis

VTE is a common *preventable* cause of death. All patients are 'at risk' of developing DVT, just as is the general population.

Assessing the risk of venous thromboembolism

National requirements for VTE prophylaxis require all patients to be assessed for risk factors on admission and reassessed after 24h in hospital.[1] Risk is judged according to:

- *Procedure factors*. Prolonged anaesthetic time, lower limb or pelvic surgery.
- *Patient factors*. Immobility, malignancy, age, inflammatory pathologies, etc. (see Box 2.4).

Balanced against:

- *Bleeding risks*. Active bleeding, stroke, invasive procedures, bleeding disorders (liver disease, thrombocytopenia, inherited disorders).

The risks should be recorded on the patient's drug chart or VTE documentation and mechanical, e.g. thromboembolic deterrent stockings (TEDS), and/or chemical, e.g. LMWH, thromboprophylaxis prescribed as per local policy. Patients who are already fully anticoagulated do not need VTE prophylaxis. Certain patient groups require extended VTE prophylaxis following surgery (e.g. lower limb joint replacement, pelvic surgery).

Surgical and trauma patients should be regarded as being at ↑ risk of VTE if they meet one of the following criteria:

- Surgical procedure with a total anaesthetic and surgical time of >90min, or 60min if the surgery involves the pelvis or lower limb.
- Acute surgical admission with inflammatory or intra-abdominal condition.
- Expected significant reduction in mobility.
- One or more of the risk factors shown in Box 2.4.

Treatment: mechanical devices

- *TEDS*. Reduces stasis in infrapopliteal veins by continuous direct compression. Not suitable for patients with peripheral vascular disease (PVD) or broken skin.
- *Pneumatic compression boots*. Reduce stasis in infrapopliteal veins by intermittent compression emptying of foot and lower leg veins, promoting venous flow.

Box 2.4 Risk factors for VTE

- Active cancer or cancer treatment.
- Age over 60 years (↑ DVT is almost linear with advancing age).
- Critical care admission.
- Dehydration.
- Known thrombophilias and polycythaemia.
- Obesity (BMI >30kg/m²).
- One or more significant medical comorbidities (e.g. heart disease, DM, other metabolic, endocrine, or respiratory pathologies; acute infectious diseases; inflammatory conditions).
- Personal, or first-degree relative, history of VTE.
- Use of hormone replacement therapy.
- Use of oestrogen-containing contraceptive therapy.
- Varicose veins with phlebitis.

▶ For women who are pregnant or have given birth in previous 6 weeks, seek advice from specialists

Treatment: pharmacological

- LMWH—activates antithrombin III.
  - Given SC. Longer half-life, compared to unfractionated heparin (UFH).
  - Examples include enoxaparin, dalteparin, and tinzaparin.
  - In renal failure, reduce dose or use UFH; alternatively, titrate doses with anti-Xa monitoring.
- *UFH*—activates antithrombin III.
  - Given SC. Short half-life, reversible with protamine.
- *Fondaparinux*—catalyses factor Xa inhibition by antithrombin III.
  - Minimal risk of heparin-induced thrombocytopenia (HIT). May have lower bleeding risk, compared to LMWH. Caution in renal failure.
- *Non-vitamin K antagonist oral anticoagulants (NOACs)*:
  - Benefit of daily PO dosing.
  - Examples include dabigatran, rivaroxaban, and apixaban.
  - Difficult to measure effect using conventional clotting assays.
  - Most do not have a direct antidote.

Reference

1 National Institute for Health and Care Excellence (2019). Venous thromboembolism in over 16s: reducing the risk of hospital-acquired deep vein thrombosis or pulmonary embolism. Available at: https://www.nice.org.uk/guidance/ng89

In-theatre preparation

Theatre is a potentially dangerous place for patients; many of these dangers arise directly as a result of poor preparation and checking, e.g. wrong side surgery (e.g. healthy kidney removed); wrong site surgery (e.g. inguinal, not femoral, hernia repaired); allergic reaction to medication; vital material not available (e.g. blood not cross-matched); vital equipment not available (e.g. image intensifier, specialist joint replacement jig); retained swabs or instruments.

Experience from other high-risk industries (e.g. the airline industry) has shown that strict adherence to a checklist can minimize the risk of these 'never events' happening. The WHO recommends a standardized checklist approach.

WHO checklist

The WHO checklist[1] is a basic template, which can be modified or adapted for different organizations, but has four key checkpoints.

Before commencement of the operating list

- Confirm surgical, anaesthetic, and nursing teams present and identified.
- Confirm patients on the list, the order of the procedures to be performed, and any specific concerns.
- Check anaesthetic requirements are correct and functioning (machine, medication, monitoring).
- Confirm vital imaging/equipment required for the list.

Before induction of anaesthesia

- Check patient identity and consent valid.
- Check site and side marked, if appropriate.
- Check anaesthetic requirements are correct and functioning.
- Check allergies and anticipated blood loss.

Before skin incision

- Check all team members present and known.
- Check the procedure to be performed.
- Confirm any surgical/anaesthetic/nursing concerns.
- Confirm vital imaging/equipment available.

Before the patient leaves the theatre

- Check the correct name for the procedure actually performed is known and recorded.
- Check the swab and instrument counts are correct.
- Confirm any surgical specimens collected and labelled correctly.
- Confirm any specific post-operative instructions.

Reference

1 World Health Organization (2008). *WHO surgical safety checklist and implementation manual*. Available at: https://www.who.int/patientsafety/safesurgery/ss_checklist/en/

Positioning the patient

Getting the patient onto the operating table

- The surgical team is partly responsible for the safety of the patient all the way onto and off the operating table. Be sure the basic rules of safety are being observed. (See Fig. 2.3 for some typical patient positions in surgery.)
- The anaesthetist is responsible for the patient's airway and should co-ordinate all moves of the patient to ensure it is maintained.
- Be sure not to dislodge IV cannulae, epidural sites, or existing drains.
- Use approved manual handling techniques (e.g. a 'Patslide' or similar device, rather than lifting the patient).
- Be aware if extra care needs to be taken, e.g. prosthetic joints that may dislocate once protective muscle tone is lost during relaxation, unstable fractures, potentially unstable joints due to RA, existing ulcers or skin lesions.

Once in position

- Ensure that no points on the patient are in contact with the metal of the operating table to prevent diathermy exit point burns.
- Make sure bony prominences and areas of thin skin are well padded, e.g. the neck of the fibula in leg stirrups.
- Ensure any diathermy pad is correctly applied and not liable to be affected by skin preparation.
- Ensure there are appropriate supports for the patient to secure the position, particularly if the table may be moved, tilted, or rotated during the procedure (e.g. arm, thoracic, and abdominal supports for lateral positions; shoulder bolsters if the patient will be head down).
- For procedures requiring access to the perineum, be sure that the pelvis is properly supported, but that the perineum is exposed over the end of the operating table.
- Consider the positioning of ancillary equipment. For example, where will video stacks be positioned? Is more than one energy source required and where will the generators be located? Is there access of mobile imaging equipment or on-table radiography? All equipment positioning needs to allow enough access to the patient for the surgical team.

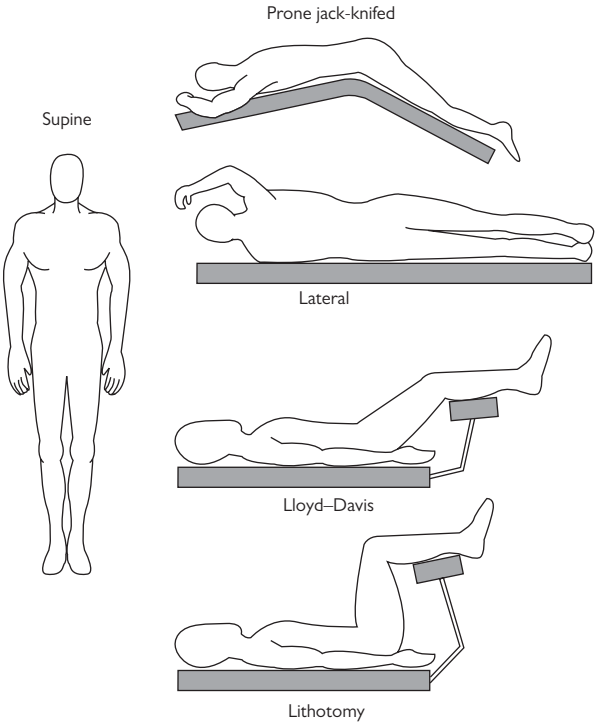

Fig. 2.3 Typical positions for surgery. Supine (most abdominal surgery); prone jack-knifed (some rectal or vaginal procedures); lateral (thoracotomy); Lloyd–Davis (pelvic surgery); and lithotomy (most perineal procedures).

Sterilization, disinfection, and antisepsis

Definitions

- **Sterilization** is removal of all viable microorganisms, vegetative, organisms and spores.
- **Disinfection** is removal of actively dividing vegetative microorganisms.
- **Antisepsis** is the process whereby the risk of medical cross-infection by microorganisms is reduced.

Sterilization

Heat

- Dry heat (e.g. incineration, flaming to red hot) is effective, but rarely useful. Dry heat requires temperatures of 160°C for at least 60min.
- Moist heat (e.g. autoclave heating using pressurized steam at 121°C at 15lb/in² for 15min) is effective and useful, especially in operating theatres.

Irradiation

- Gamma radiation. Effective for inorganic materials.

Filtration

- Air or fluids can be sterilized by ultrafine membrane filters but are rarely useful in hospital practice.

Disinfection

- Acids/alkalis, e.g. bleach. Effective for non-human contact use.
- Alcohols/phenols, e.g. ethyl alcohol—skin swabs; alcohol solutions—hand disinfection; carbolic chloroxylenols; phenol (Clearsol®).
- Oxidizers, e.g. povidone–iodine—skin disinfection/surgical scrubbing; hydrogen peroxide (H_2O_2)—superficial wound cleansing; aldehydes—surgical instruments such as endoscopes.
- Cationic solutions, e.g. chlorhexidine—antiseptic washes.
- Organic dyes, e.g. Proflavine.

Antisepsis

The principles of antisepsis include the following:

- Always remove gross contamination with simple soap first.
- Use high-potency acid/alkali disinfection on inert surfaces.
- Use less corrosive oxidizers on delicate inert materials.
- Use weak alcohols and oxidizers for skin cleansing.

Scrubbing up

Scrubbing up is designed to reduce the risk of infection from the surgeon to the patient. A thorough clean with bactericidal soaps reduces the number of organisms that can be cultured from skin swabs, but the skin (particularly sweat glands and hair follicles) cannot be sterilized. Moisture and heat occurring under surgical gloves quickly raise the bacterial count again and despite modern cleaning agents, significant growth can be achieved within 2h. Common bactericidal soaps include chlorhexidine and povidone–iodine.

Protocol

The first time you come to theatre, ensure that the senior scrub nurses know who you are. It is polite and safe. Everybody should know who the people in theatre are and what they are there for.

How to scrub

- Wet your hands and arms first.
- Lather well with disinfectant soap and wash off.
- 'Scrub' under the nails and heavily soiled areas with a sterile brush and more disinfectant soap. Do not scrub too vigorously, as this causes irritation without any ↑ bactericidal effect.
- Wash again with soap, being sure to cover the commonly missed areas—between the fingers, back of the hands, under fingernails, and base of the thumbs.
- Rinse thoroughly to remove all soap to reduce the chance of skin irritation.
- Rinse off, trying to ensure the water runs off the arms at the elbows.
- Dry the hands completely first and do not go back to them once drying the arms.

How to gown and glove

- Be sure to open the gown without touching the outer 'face'.
- Do not push your hands through the cuffs.

Pick up the right glove, with your right hand still in the cuff of the gown—the glove palm side down, with the fingers pointing up towards your forearm.

- With your left hand, fold the other side of the edge of the glove 'over' your right hand.
- Slide your right hand into the glove.
- Once on, pick up the left glove, holding it by the edge and pull it over the cuff of the left hand.
- Slide your left hand into the glove and adjust glove positions.

It is becoming common practice to wear eye protection and two pairs of gloves to reduce the risk of exposure to infectious agents. (➲ Infection control measures: Hand hygiene, Personal protective equipment (PPE) p. 38.

Surgical instruments

(See Fig. 2.4.)

'Sharps'

- Scalpels. Two sizes of handle (4 and 6). Types of blade and uses include: no. 11 (used for stab incisions); no. 10 (most skin incisions); no. 15 (fine incisions); and no. 22 (adhesiolysis).
- Scissors. May be dissecting or stitch cutting. Dissecting scissors may be straight (e.g. Mayo) or curved (e.g. curved Mayo, McIndoe, Metzenbaum, Nelson's).

Forceps

- Non-toothed. Fine, non-toothed (e.g. DeBakey, Adson's forceps) used for handling delicate tissues such as vessels and bowel. Heavy, non-toothed used for general handling, including specimens and sutures.
- Toothed. Fine-toothed (e.g. Gillies', McIndoe's forceps) used for handling skin and fine fascia, and occasionally for precise holds on delicate tissues. Heavy-toothed (e.g. Lane's forceps) used for holding heavy tissues such as fascia and scar tissue.
- Ring-tipped and micro-forceps. Used in vascular anastomoses.

Clips and clamps

- Artery clips, e.g. Spencer–Wells, Robert's (large); Dunhill's, Mosquito (small), have serrated jaws. Used for vascular clamps and tissue/suture holding.
- Tissue clamps, e.g.:
  - Lahey clamp. Clamp with curved tip, often used to clamp/dissect around vessels.
  - Doyen bowel clamp. Non-crushing atraumatic.
  - Babcock/Duval clamp. Non-toothed, semi-atraumatic tissue-holding clamp (often used for holding bowel).
  - Lane's/Allis's/'Littlewood' clamp. Heavy-toothed traumatic tissue-holding clamps.

Retractors

- Self-retaining retractors:
  - Large (e.g. Goligher retractor for abdominal incisions; Finichetto retractor for thoracic incisions).
  - Small (e.g. Travers (superficial), Norfolk and Norwich (deeper) retractors for smaller skin and abdominal incisions).
- Handheld retractors:
- Large (e.g. Deaver, Kelly, Morris).
- Small (e.g. Langenbeck, Kilner/'Catspaw').

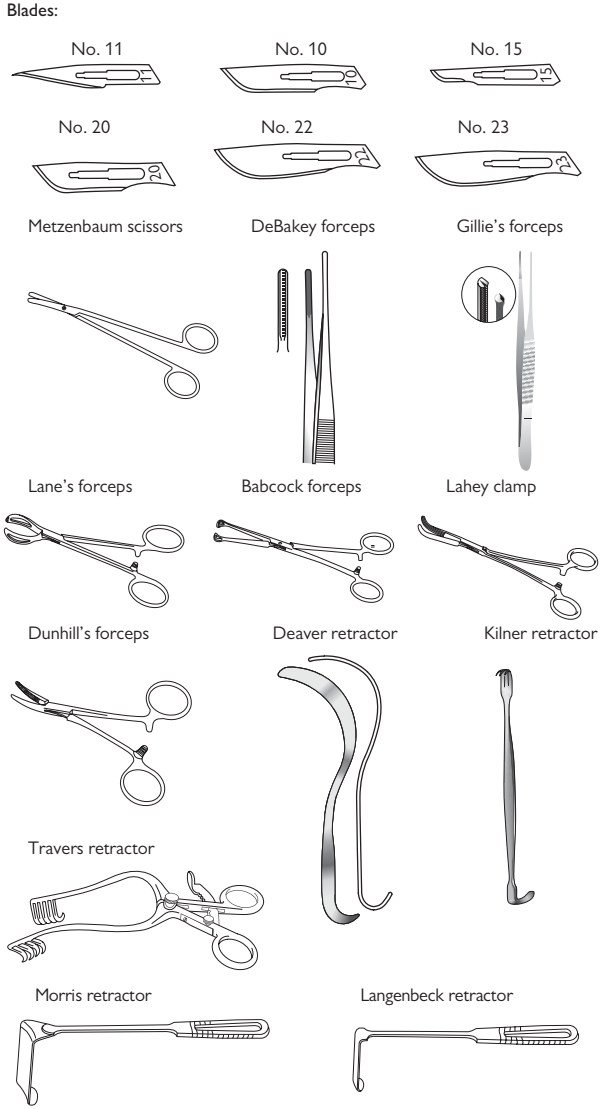

Fig. 2.4 Commonly used surgical instruments.

Incisions and closures

Body cavity incisions

General terms are applied to incisions accessing each body cavity.

- *Laparotomy*. Any incision accessing the peritoneal cavity or retroperitoneal space. Separate types of laparotomy are described according to their location in the abdomen, tissues that are crossed, or occasionally the individual who described them (see Fig. 2.5).
- *Thoracotomy*. Accessing the chest cavity, typically the pleural space or posterior mediastinum. Median sternotomy is a particular type of thoracotomy for access to the anterior and middle mediastinum.
- *Craniotomy*. Accessing the compartments of the skull.

Incision closures

Incisions are closed according to some basic principles.

- Fascial layers offer the best tissue to bear the strength of apposition and form the main closure in the abdomen. Closure is usually made with heavy, non-permanent sutures.
- Bony defects, such as in a craniotomy, should be apposed to allow minimal movement.
- Defects in fascial or bony tissues should be replaced either with transposed tissues (e.g. skin, fascial, muscle flaps) or with inserted tissues (e.g. synthetic products such as polypropylene mesh).
- Large cavities and potential spaces between tissues should be avoided to reduce the risk of fluid collections which run the risk of becoming infected.

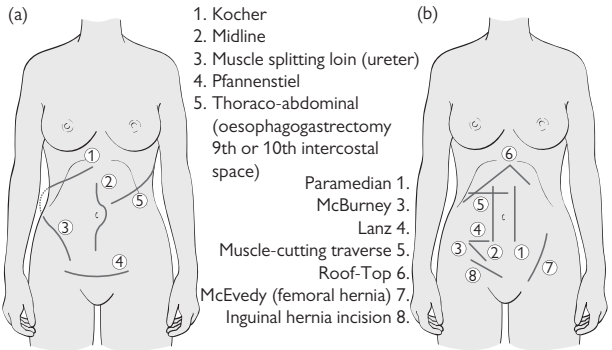

Fig. 2.5 Incisions.

Reproduced with permission from Longmore, M. et al. (2007). Oxford Handbook of Clinical Medicine, 7th edn. Oxford University Press, Oxford.

Stomas

Terminology and types

The term stoma is usually applied to an external opening (temporary or permanent) in a lumenated organ.

- **Ileostomy**. Formed from any part of the mid- or distal small bowel. May be loop (often to 'rest' the distal bowel) or end (usually as a result of surgical removal of distal bowel).
- **Colostomy**. Formed from any part of the large bowel. May be loop (to rest distally) or end (because of surgical resection).
- **Urostomy**. Formed from a short length of disconnected ileum into which one or both ureters are diverted (usually after radical lower urinary tract surgery).
- **Gastrostomy**. Either a surgically created or endoscopically formed connection between anterior stomach and anterior abdominal wall. Often for stomach drainage or direct feeding.
- **Jejunostomy**. Either a surgically created or endoscopically formed connection between proximal jejunum and anterior abdominal wall. Often for direct feeding.

Identifying stomas

Any stoma may have many different appearances. Typical features that help in identifying stomas are the following:

- **Ileostomies** (loop or end) are usually spouted, have prominent mucosal folds, tend to be dark pink/red in colour, and are commonest in the right side of the abdomen.
- **Colostomies** (loop or end) are usually flush, have flat mucosal folds, tend to be light pink in colour, and are commonest in the left side of the abdomen.
- **Urostomies** (end) are indistinguishable from end-ileostomies unless the output can be seen.
- **Gastrostomies** and **jejunostomies** are usually narrow calibre and flush with little visible mucosa, and are commonest in the left upper quadrant of the abdomen. They are usually fitted with indwelling tubes or access devices.

Knots and sutures

Types of suture

(See Fig. 2.6 and Table 2.2.)
In general, sutures are classified along two lines:

- Non-absorbable versus absorbable.
- Braided versus monofilament.

Non-absorbable sutures tend to be used for prolonged integrity where any loss of strength might compromise the future integrity of the tissues being joined, e.g. vascular anastomoses, hernia mesh fixation, tendon repairs, sternal wiring.

Absorbable sutures tend to be used where the persistence of foreign material would cause unnecessary tissue reaction or ↑ risk of infection, e.g. bowel anastomoses, skin and subcutaneous tissues.

Monofilaments have the advantage of smooth tissue passage and minimal tissue reaction but tend to have a crystalline structure that increases the 'memory' effect of the suture, making knotting less secure and increasing the risk of suture 'fracture'.

Braided polyfilaments exert more tissue friction during the passage through but are intrinsically more flexible and knot more securely.

Table 2.2 Types of suture material

	Natural fibre	Synthetic fibre
Non-absorbable monofilament	Silver wire Steel wire	Polypropylene nylon
Non-absorbable braided polyfilament	Silk (eventually does decay)	Ethilon®
Absorbable monofilament		Monocryl® PDS®
Absorbable braided polyfilament	Catgut (the original absorbable suture, no longer used)	Vicryl® Dexon®

Sizes of suture

Size is denoted by imperial sizes from 10/0 (smallest—invisible to the naked eye) through 7/0–5/0 (typical size for small to medium vascular anastomoses), 3/0–2/0 (typical size for bowel anastomoses, closure of most subcutaneous fascial layers, ligation of vascular pedicles), and 1 (typical size for abdominal closure), to 4 (largest available—size of sternal wires).

Types of needle

- **Curved** (from shallow-curved to half-round) or **straight**.
- **Round-bodied**. Blunt (relatively safe as it has low tissue penetrance; used for closing major incisions) or sharp (sharp point, but round cross-sectional profile). 'Pushes' tissue apart, so often used for delicate tissues such as bowel, blood vessels, etc.

- **Cutting point/reverse cutting point** (sharp point, with triangular cross-section giving a cutting edge). Slices through tissues, so often used for dense structures such as fascia and tendons.

See also ➲ Table 17.1 –Common stitch materials and their uses

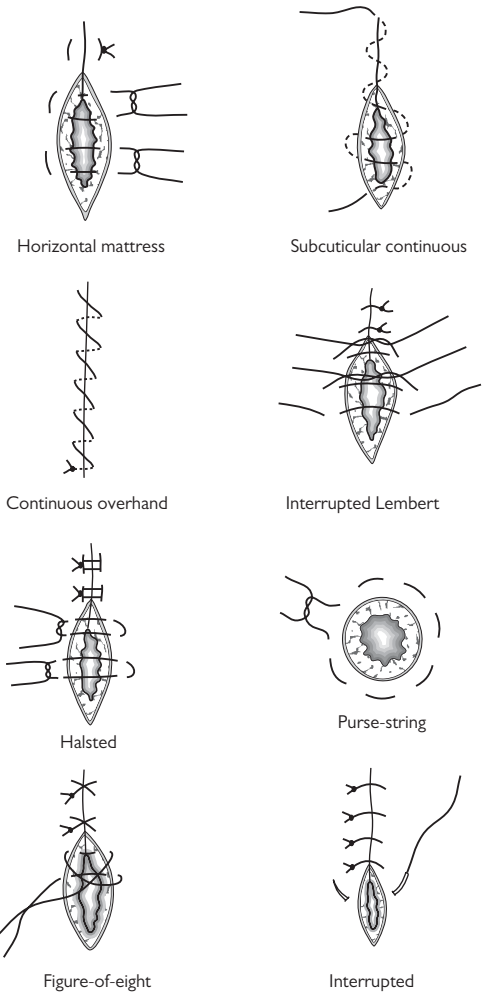

Fig. 2.6 Types of suturing used (illustrated as skin sutures).

Post-operative management

Routine tests

Protocols vary widely according to the complexity of surgery and the age of the patient; this is a rough guide to management of the older patient following major abdominal, cardiac, or reconstructive surgery.

Blood tests

FBC and U&Es on days 1, 2, and 5. Look for the following:

- Anaemia: consider haemodilution or slow surgical bleeding.
- Raised white cell count (WCC): look for other signs of sepsis (➲ Wound emergencies, pp. 128–9; ➲ Sepsis, pp. 172–3).
- Monitor INR/clotting daily if the patient is anticoagulated and before insertion of drains or central lines.
- Check Na^+ and K^+ to guide choice of crystalloid (➲ Fluid management, pp. 116–17).
- Monitor urea and Cr, especially in preoperative renal dysfunction, cardiac and aortic surgery, and nephrotoxic drugs (e.g. NSAIDs, ACE inhibitors, vancomycin/gentamicin, fluid restriction).

ECG

Very rarely used routinely outside post-cardiac surgery. Look for: rhythm disturbances such as AF or evidence of ischaemia.

CXR

Request daily if chest drains are present on suction, after drain removal, and to check position of newly placed central lines. Look for:

- Position of indwelling lines.
- Consolidation, pneumothorax (on side of central line), pulmonary oedema, and pleural effusion.

Ward rounds

See patients once a day (twice if unwell, not progressing as expected, or undergoing investigations or treatment). In the evening, review the blood results and other investigations from that day. For formal ward rounds, do the following:

- Make sure you have a nurse with you and/or handover to nursing staff.
- Make sure someone writes a summary in the patient's notes.
- Check the obs chart for 'ABCDE' (O_2 saturations, pulse and BP trends, temperature), as well as fluid balance and drainage if monitored.
- Ask the patient if they are experiencing any problems—establish whether they have adequate pain control and are mobilizing appropriately, eating and drinking, passing urine, and opening bowels.
- Look at exposed wounds for evidence of infection or seromas (covered wounds only require routine inspection if there is suspicion of infection or to assess tissue viability).
- Check diabetic charts for BS control.
- Review the drug chart. Restart regular oral medication as soon as possible. Convert IV to PO where appropriate. Look actively for drugs that can be discontinued to minimize polypharmacy; specifically review antimicrobials daily (➲ Antibiotic prescribing in surgery, p. 46)
- Review the most recent blood results.
- Review the nutritional status of the patient—what is the best route?
- Make a clear problem list and a clear plan.

Special cases

Surgery for malignancy

Liaise with specialist nurses; they will know the overall plan from the MDT. Do not discuss results without consulting them first. Breaking bad news is discussed under ➲ Communication skills, p. 12.

Plastic and reconstructive surgery

- Check perfusion of flaps daily.
- Check take of split-skin grafts on day 5.
- Book post-operative medical illustration for photos prior to discharge.

Orthopaedic surgery

- Check X-rays of prosthesis to assess position and fraction reduction.

Vascular surgery

- Endovascular: check groins/other access sites for haematoma.
- Arterial bypass surgery: check distal (and graft) pulses (use handheld Doppler if not palpable) and capillary refill.
- Post-carotid endarterectomy: perform focused neurological assessment (especially contralateral limbs, cranial nerves (CN) XII (tongue movement), (IX/X larynx & swallowing), and sensorimotor function in ipsilateral neck/face).
- Amputees: arrange amputee pathway with physio- and occupational therapists.

Cardiac surgery

- Check sternal stability daily (ask the patient to cough whilst you feel for abnormal sternal movement).
- Request TTE for days 4–5 post-operatively in valve repair patients, and auscultate daily.

Also see ➲ Principles of cardiac surgery > Post-operative management, *p. 750*

Discharge

- Plan discharge from the day of admission. Use the hospital discharge team and identify patients requiring special facilities, such as rehabilitation, as soon as possible.
- Make sure the patient understands what operation they have had.
- Tell them how to look out for common problems like wound infections, what is normal, and who to contact if they are worried.
- Tell them when they will be seen in clinic.
- Tell them when they can expect to go back to work.
- Ensure an informative, but concise, discharge summary for the GP (➲ Communication with general practitioners, p. 13).

Rehabilitation

The average times for patients to be fit to go back to work are as follows:

- Two to six weeks after abdominal or thoracic surgery, depending on size and approach (e.g. laparoscopic or open).
- Weight-bearing takes up to 2 months after lower limb arthroplasties and 3 months after lower limb fractures.
- Patients can usually drive once they are fully mobile as long as they have not experienced blackouts or fits.
- For those who have had limb surgery or if there is any doubt about their ability to drive, the patient should be asked to inform the DVLA (or equivalent).

Drains and drain management

Uses and complications

Drains may be used for several reasons:

- To remove existing abnormal collections of fluid, blood, pus, and air (e.g. drainage of a subphrenic abscess, removal of a pneumothorax).
- To prevent the build-up of normal bodily fluids (e.g. bile after surgery to the bile duct) or potential abnormal fluids or air (e.g. bloody fluid in the pelvis after rectal surgery).
- Occasionally used to prevent or 'warn' of potentially serious complications (e.g. bleeding—neck drains after thyroid surgery, chest drains after chest trauma).

Potential complications should be balanced against drain use:

- Damage to structures during insertion, even if under CT or ultrasound guidance (e.g. risk of injury to organs in image-guided drains or haemorrhage from vessels from operative drains).
- Drains provide a potential route for the introduction of infection, especially external drains that remain for longer than a few days.
- Damage to structures close to the drain, e.g. pressure injury to bowel if subjected to high-pressure suction drainage.
- Drains do not always drain the substance expected and may give a false 'sense of security', e.g. failure to drain bleeding after thyroid surgery or failure to drain faecal fluid after anastomotic leakage.

There is no place for routine use of drains after surgery unless there is a clear indication—'Better no drainage than ignorant use of it' (Halstead). Materials used include latex (e.g. T tubes), silastic rubber (e.g. long-term urinary catheters), polypropylene (e.g. abdominal drains), and polyurethane (e.g. nasogastric tube (NGT)).

Box 2.5 Types of drains

Open passive drains

These provide a conduit around which secretions may flow.

- Yates corrugated drain (after subcutaneous abscess drainage).
- Penrose tube drain.
- Drainage setons placed in anal fistulae.

Closed passive drains

These drain fluid by gravity ('siphon effect') or by capillary flow.

- Robinson tube drain (after intra-abdominal abscess drainage).
- NGT.
- Ventriculoperitoneal shunt.
- Chest drain (tube thoracostomy).

Closed active drains

These generate active suction (low or high pressure).

- Exudrains®, Redivac drains®, Minivac®, Jackson Pratt drains (after pelvic or breast surgery); or chest drain connected to wall suction.

Chest drains

Indications and the method for chest drain insertion are described under ➔ Chest drain insertion, pp. 262–3. Management of chest trauma is described under ➔ Thoracic injuries, pp. 586–7. Principles of chest drains are as follows:

- Drainage should always be into an underwater sealed container.
- Swinging of the air–fluid meniscus with respiration demonstrates continuity with the intrapleural space (i.e. drain working).
- Bubbling in the underwater seal, either continuously or when the patient coughs, indicates an air leak from the lung parenchyma (i.e. air is being removed by the drain).
- The safest mode is an unclamped drain connected to an underwater seal that is kept below the level of the patient at all times.

Management of chest drains inserted for pneumothorax

- Put the drain on low-pressure, high-volume wall suction (–3–5kPa) initially (not the high-pressure wall suction used for tracheal toilet).
- Request and review CXRs daily.
- ❗ Do not remove the drain when there is an air leak; otherwise a pneumothorax will rapidly re-form.
- As the lung repairs and re-expands, the bubbling (air leak) will eventually stop. When the air leak stops, take the drain off suction for 12h and repeat the CXR; if the lung is fully expanded, the drain can be removed. Repeat CXR after drain removal to check for a pneumothorax.

Management of chest drains inserted to drain collections

- There is no evidence that suction improves outcome.
- Measure the drainage volume hourly in the post-operative patient, and every 24h in longer-term drains. ❗ A haemodynamically unstable post-operative patient, *or one who is draining >200mL of blood per hour*, should be discussed urgently with the thoracic surgeons.
- Post-operative drains are normally removed when they drain nothing for 2 consecutive hours unless there is an air leak.
- Drains for pleural effusions can be removed when they drain <250mL in 24h.
- Drains for empyemas can be removed when they stop draining.
- Always request and review a CXR after drain removal to check for pneumothorax.

Management of abdominal drains

- Daily review of the contents and drain site.
- Drain output and contents should be recorded at least 12-hourly.
- Timing of abdominal drain removal is patient-specific—always consult with senior colleagues before removing.

Clamping chest drains

Should only be done under specialist supervision.

- Clamping a thoracic drain in a patient with an air leak may rapidly result in a tension pneumothorax.
- Clamping a mediastinal drain in a patient who is bleeding may rapidly result in cardiac tamponade.

NB If you connect the drain to wall suction, with the wall suction off or if you press in the one-way valve on the top of the underwater seal too tightly, this effectively clamps the drain. If you and the nursing staff do not know what you are doing, ask for help.

Fluid management

Fluid management is aimed at making sure the patient is neither fluid-depleted nor fluid-overloaded. The principle is to replace whatever is lost. Success can make the difference between a short, uncomplicated post-operative course and the patient ending up on ICU.

Maintenance crystalloid

- The principle is to replace Na^+, K^+, and water loss from urine, vomiting, diarrhoea, high-output fistulae and stomas, and fluid 'third-spaced' (e.g. ascites, tissue oedema). For fluid management in burns, ➲ Burns: assessment, p. 718; ➲ Burns: management, pp. 720–2.

Approximate average daily loss of fluid and electrolytes

- Water loss: 2500mL/day (insensible loss from skin and respiratory and GI tracts, and in urine). ↑ loss in sepsis, ventilation, diarrhoea/vomiting, high-output fistulae, and polyuric renal failure.
- Na^+ loss: 1–2mmol/kg/day (in urine). ↑ loss in pyrexia, diarrhoea, vomiting, and high-output fistulae. Urine concentration is less effective in elderly patients.
- K^+ loss: 0.7–1mmol/kg/day (in urine). ↑ loss in pyrexia, diarrhoea, vomiting, and high-output fistulae.

Common crystalloids

- *Hartmann's solution* contains 130mmol/L of Na^+, 103mmol/L of chloride (Cl^-), 28mmol/L of lactate, 4mmol/L of K^+, and 1.5mmol/L of Ca^{2+}. It is iso-osmolar and isotonic; lactate is metabolized to glucose in the liver.
- *0.9% normal saline (NaCl)* contains 154mmol/L of Na^+ and 154mmol/L Cl^-. It is almost iso-osmolar and isotonic, although infusion of large amounts can cause hyperchloraemic acidosis. Also manufactured with either 20mmol/L or 40mmol/L of K^+.
- *5% glucose* contains no electrolytes but is iso-osmolar (due to glucose). Initially isotonic, but then becomes hypotonic as glucose is metabolized. Should not be used for volume replacement, as it is not retained intravascularly and large amounts cause hyponatraemia.

Example regime

A simple regime for adults may consist of 3L of Hartmann's solution/24h, although this does not consider extraneous losses. Substitute Hartmann's for normal saline, with K^+ if supplementation is required.

Colloids

Colloids (especially blood) produce a more lasting expansion of intravascular volume than crystalloid, as the latter rapidly enters the interstitial tissues.

- *Gelofusine®* is succinylated gelatin (bovine collagen) which has a half-life of about 2h in plasma and is associated with ↑ bleeding times in post-operative patients.
- *Albumin* is a naturally occurring plasma protein, sterilized by ultrafiltration—5% albumin is isotonic; 20% albumin is hypertonic. Indications for use of albumin as a volume expander are very limited.
- *Blood*, *platelets*, *fresh frozen plasma (FFP)*, and *cryoprecipitate* (➲ Blood products and procoagulants, pp. 120–1).

Assessing volume status

This is usually straightforward, but in the HDU patient 24h post-complex surgery, you need information from several sources.

History and examination

- The dry patient. May have been NBM several days preoperatively and feels thirsty, complains of a dry mouth, may be dehydrated because of diarrhoea or vomiting, and has a low JVP, dry mucous membranes, and reduced skin turgor.
- The overfilled patient. Usually does not feel thirsty, has a raised JVP and normal skin turgor, and may have dependent oedema and evidence of pulmonary oedema on auscultation.

Observations chart

- The dry patient. May have falling BP, rising pulse rate, and low central venous pressure (CVP) that rises transiently with fluid challenges; weight is several kilograms below preoperative weight.
- The overfilled patient. Is not usually tachycardic and has a high CVP that rises and plateaus with fluid challenges. BP may fall with fluid challenges; weight is several kilograms above preoperative weight.

Fluid balance chart

In sick patients, start an hourly fluid balance chart. Add up all fluid loss (urine output, wound, stoma, and fistula drainage) and subtract from all IV, NG, and PO fluids given.

- The dry patient. Will usually be in several litres of negative fluid balance, possibly over a few days, with a urine output <1mL/kg/h.
- The overfilled patient. Will be in several litres of positive fluid balance, possibly over a few days. Urine output may be low because of heart failure or renal dysfunction.

Blood results

- The dry patient has high Na^+, K^+, Cr, and urea, with urea often disproportionately raised.
- The overfilled patient may have low Na^+.

CXR

- The dry patient has no evidence of pulmonary oedema or effusions.
- The overfilled patient may have both.

Resuscitation fluids

Isotonic crystalloids (e.g. Hartmann's, normal saline) or colloid solutions can be used to replenish a deplete intravascular compartment. If used to replace blood loss, 3–4 times the volume lost must be replaced as only one-third to a quarter remains intravascularly. In a patient with massive haemorrhage (e.g. trauma, peripartum), resuscitate with blood products.

Acid–base balance

The pH of arterial blood is maintained at 7.35–7.45. Normal functioning of the body's complex enzyme systems depends on this stability, which is regulated by the lungs and kidneys. Derangements may be primarily due to respiratory or metabolic dysfunction. Compensatory mechanisms (if present) are performed by the non-diseased organ, i.e. a 1° respiratory acidosis/alkalosis is compensated by a metabolic alkalosis/acidosis, and vice versa.

Remember:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

Normal values

- pH 7.35–7.45.
- $PaCO_2$ 4.7–6.0kPa (an ↑ drives an acidosis; a ↓ an alkalosis).
- Bicarbonate (HCO_3^-) 22–26mmol/L (a ↓ drives an acidosis; an ↑ an alkalosis).
- Base excess (BE) –2 to +2mEq/L (a ↓ drives an acidosis; an ↑ an alkalosis).

Diagnosis of acid–base abnormalities

The clinical picture can often give you clues as to what acid–base abnormalities to expect. The Flenley nomogram (see Fig. 2.7) is also a useful diagnostic aid.

- **Look at the pH. Is there an acidaemia (<7.35) or an alkalaemia (>7.45), or is it normal?**
- **Look at $PaCO_2$. Is the change in keeping with pH derangement?**
  - If pH <7.35 and $PaCO_2$ >6.0kPa: 1° respiratory acidosis.
  - If pH >7.45 and $PaCO_2$ <4.7kPa: 1° respiratory alkalosis.
- **Look at HCO_3^-. Is the change in keeping with pH derangement?**
  - If pH <7.35 and HCO_3^- <22mmol/L: 1° metabolic acidosis.
  - If pH >7.45 and HCO_3^- >26mmol/L: 1° metabolic alkalosis.
- **Is there any compensation? Changes not in keeping with pH derangement suggest the presence of compensation.**

Bicarbonate, base excess, and anion gap

These are derived numbers, calculated by blood gas analysers. Because they depend on several assumptions, they do not always reflect the true acid–base balance. **BE** is defined as the mEq/L of strong acid that would be required to titrate the blood pH back to 7.4 if the $PaCO_2$ were normal. The **anion gap** is the difference between measured cations and measured anions (= $[K^+ + Na^+] - [Cl^- + HCO_3^-]$). This is made up of metabolic acids: ketones, lactate, and phosphates. The anion gap is normally 8–16mmol/L; an increase in anion gap indicates a metabolic acidosis.

Metabolic acidosis

- *Uncompensated*: ↓ pH, ↔ pCO_2, ↓ HCO_3^-.
- *Compensated*: ↔ pH, ↓ pCO_2, ↓ HCO_3^-.

Metabolic acidosis due to increased metabolic acids (increased anion gap)

- Lactic acid (global and/or regional hypoperfusion, hypoxia, sepsis, hepatic failure as the liver normally metabolizes lactate).
- Uric acid (renal failure).

- Ketones (diabetic ketoacidosis, starvation ketoacidosis).
- Drugs/toxins (salicylates, sodium nitroprusside overdose).

Due to loss of bicarbonate or hyperchloraemia (normal anion gap)

- Renal tubular acidosis (loss of HCO_3^-).
- Diarrhoea, high-output ileostomy (loss of HCO_3^-).
- Pancreatic fistulae (loss of HCO_3^-).
- Hyperchloraemic acidosis (excessive saline administration).

Metabolic alkalosis

- *Uncompensated*: ↑ pH, ↔ pCO_2, ↑ HCO_3^-.
- *Compensated*: ↔ pH, ↑ pCO_2, ↑ HCO_3^-.
- Loss of hydrogen ions (H^+) from gut (vomiting).
- Renal loss of H^+ (diuretics), ↑ reabsorption of HCO_3^- (hypochloraemia).
- Administration of base ($NaHCO_3$, citrate in blood transfusions).

Respiratory acidosis

- *Uncompensated*: ↓ pH, ↑ pCO_2, ↔ HCO_3^-.
- *Compensated*: ↔ pH, ↑ pCO_2, ↑ HCO_3^-.
- Any cause of respiratory failure or hypoventilation.
- ↑ production of CO_2, e.g. sepsis, malignant hyperpyrexia.
- Rebreathing CO_2 (low gas flows, soda lime exhaustion).
- Compensation occurs over hours to days.

Respiratory alkalosis

- *Uncompensated*: ↑ pH, ↓ pCO_2, ↔ HCO_3^-.
- *Compensated*: ↔ pH, ↓ pCO_2, ↓ HCO_3^-.
- Hyperventilation: deliberate, inadvertent, or in non-ventilated patients caused by stroke, anxiety, pulmonary embolism (PE), pneumonia, asthma, and pulmonary oedema.

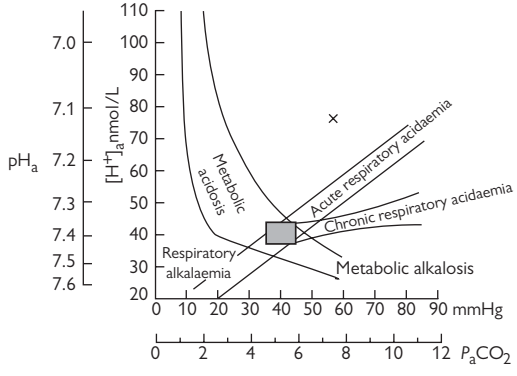

Fig. 2.7 Flenley nonogram.

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Blood products and procoagulants

Bleeding and coagulation pp. 232–4

Indications for blood products

- Young, fit patients tolerate haemodilution much better than elderly patients with multiple comorbidities (particularly cardiovascular and respiratory disease).
- There is a higher threshold for giving blood products in the patient who is not actively bleeding or about to undergo a procedure.
- Packed red cells (PRCs). Aim to maintain Hb >70g/L; in older patients and those with cardiorespiratory disease, maintain Hb >90g/L.
- FFP. Major haemorrhage *or* to correct clotting abnormalities (INR >1.5, activated partial thromboplastin time (APTT) ratio >1.5) with active bleeding.
- Platelets. Major haemorrhage *or* if platelets $<50 \times 10^9$/L *or* platelets $<100 \times 10^9$/L with active bleeding (lower threshold if patient was on aspirin or clopidogrel within 5 days and is actively bleeding).
- Cryoprecipitate. Major haemorrhage or fibrinogen <1g/L with active bleeding.

▶ Blood products are a limited resource. If in doubt, discuss the use of blood products with the haematology service.

Packed red cells

- One unit of PRCs has a volume of 300mL and increases Hb by about 10g/L in a 70kg adult.
- Cross-matched blood can normally be provided within 20min; it contains blood from a single donor.
- In dire emergencies, O rhesus (Rh)-negative PRCs can be given to recipients of any ABO Rh group without incompatibility reaction.
- Autologous blood transfusion may be used with up to 4U of blood withdrawn from patients preoperatively over the course of a month.
- Cell salvage reduces the need for allogeneic blood. Shed blood is collected intraoperatively, heparinized, spun with normal saline to remove all material, including residual heparin, platelets, and clotting products, and repackaged as red blood cells suspended in saline for transfusion.
- Simple measures to reduce the need for blood transfusion include:
  - Treating anaemia (with PO/IV iron) and coagulopathies preoperatively.
  - Stopping warfarin, heparin, aspirin, and clopidogrel appropriately.

Platelets

- One pool of platelets (~200–300mL) increases platelet count by 20×10^9/L in a 70kg adult.
- Platelets are usually pooled from 4–6 donors but can be apheresed from a single donor.
- Platelets do not need to be cross-matched, but they should be ABO-compatible (and Rh-matched in ♀ of childbearing age).
- They are stored at 22°C and have a shelf-life of 5 days.

Fresh frozen plasma

- One unit of FFP contains all the coagulation factors, except platelets.
- One unit of FFP ~250mL and 10–15mL/kg is normally given.
- One unit of FFP usually contains product from a single donor but may be pooled from several donors.
- FFP does not need to be cross-matched but should be ABO-compatible (and Rh-matched in ♀ of childbearing age).
- FFP is stored at –30°C for up to a year. It must be thawed, usually over 20min, before giving and discarded if not used within 2h.

Cryoprecipitate

- Produced by thawing FFP to 4°C, then removing the precipitate layer.
- One unit of 'cryo' contains ~140mg of fibrinogen, factors VIII and XIII, and von Willebrand factor (vWF).
- One unit of 'cryo' raises the fibrinogen by 0.6–0.7mg/L in a 70kg adult.
- ABO and Rh compatibility are not essential, but preferred.

Prothrombin complex concentrates (e.g. Beriplex®)

- Concentrates of factors II, VII, IX, and X.
- Used to reverse the effects of warfarin.
- Dose depends on the degree of reversal required and weight.
- Has a relatively short half-life (6–8h), so vitamin K needs to be given concomitantly.

Antifibrinolytics (e.g. aprotinin and tranexamic acid)

- Action. Inhibit plasminogen and plasmin; reduce active fibrinolysis.
- Indications. Prophylaxis against bleeding in cardiovascular surgery, especially high risk (e.g. redo surgery). Emerging evidence of the role of tranexamic acid (1g tds) in reducing bleeding in trauma and obstetric haemorrhage.

Key revision points—physiology of blood groups

- ABO antigens pre-exist on red cells. ABO antibodies pre-exist in the circulation and will cause immediate reaction if incompatible.
- Anti-Rh (D/E) antibodies only develop following exposure to the RhD antigen, e.g. during blood transfusion or delivery.
- O Rh-negative are universal donors (red cells carry no ABO/Rh antigens).
- AB Rh-positive are universal recipients (serum contains no A, B, or RhD antibodies).
- Blood grouping involves adding A, B, and RhD agglutinins to donated blood to determine the blood type; it takes <5min.
- Cross-matching involves mixing donated blood with the intended recipient serum; assessing compatibility takes about 20min.

Transfusion reactions

▶▶Acute haemolytic reaction

ABO incompatibility as a result of clerical, bedside, sampling, or laboratory error is the commonest cause. It may also be caused by incompatibility within other antigen systems (Duffy/Kidd). Donor erythrocytes carrying either A and/or B erythrocyte antigens bind to the recipient's anti-A or anti-B antibodies, resulting in complement formation, membrane attack complex, and immediate haemolysis. Cytokine and chemokine release mediates sympathetic inflammatory response characterized by sudden onset of hypotension, tachycardia, pyrexia, breathlessness, tachypnoea, and back pain. Bilirubinaemia, anaemia, and haemoglobinuria as a result of haemolysis ensue.

- Stop the transfusion immediately and give basic life support if required.
- Keep the bag and giving set for analysis; inform haematology.
- Give crystalloid and furosemide to encourage diuresis.
- Dialysis may be required.

▶▶Anaphylaxis and allergic reactions

Normally, immunoglobulin E (IgE)-mediated histamine release reactions to plasma, platelets, and red blood cells. Mild allergic reactions are relatively common and are characterized by erythematous papular rashes, wheals, pruritus, and pyrexia. These are treated by stopping the transfusion and administering chlorphenamine (10mg IV). Anaphylaxis characterized by hypotension, bronchospasm, and angio-oedema occasionally occurs.

- Stop the transfusion immediately and disconnect connection tubing.
- Basic life support may be required.
- Treat bronchospasm and angio-oedema with adrenaline (0.5mL of 1:1000 IM), chlorphenamine (10mg IV), and hydrocortisone (100mg IV).

Non-haemolytic febrile reaction

These common and normally mild reactions are caused by recipient antibodies directed against donor human leucocyte antigen (HLA) and leucocyte-specific antigen on leucocytes and platelets. Cytokine release mediates mild pyrexia, typically over an hour after transfusion is started. Antipyretics such as paracetamol 1g po/IV limit pyrexia, but antihistamines are not helpful. Severe reactions feature high-grade fever, rigors, nausea, and vomiting. The severity of symptoms is proportional to the number of leucocytes in the transfused blood and the rate of transfusion. Leucocyte-depleted blood helps prevent these reactions.

Delayed extravascular haemolytic reaction

Although pre-transfusion antibody testing is negative (a satisfactory cross-match), these patients experience accelerated destruction of transfused red blood cells 7–10 days following transfusion. This is an antibody-mediated reaction, usually by a patient's antibody (commonly Rh E, Kell, Duffy, and Kidd), present in levels too low to be detected clinically until produced in larger amounts on exposure to circulating antigen. As haemolysis is extravascular, haemoglobinaemia and haemoglobinuria are uncommon; it is characterized by an unexpected fall in haematocrit a few days post-transfusion, hyperbilirubinaemia, and positive Coombs' test.

Transfusion-related acute lung injury

Non-cardiogenic pulmonary oedema, typically within 6h of transfusion. Thought to be mediated by recipient antibodies against donor HLA. Activated recipient leucocytes migrate to the lung, releasing proteolytic enzymes that cause a localized capillary leak syndrome and pulmonary oedema.

Infection

Bacterial

Serious bacterial contamination of stored blood may occur and is difficult to detect. Platelets—usually stored at room temperature—are at greater risk. Common organisms include *Staphylococcus* spp., *Enterobacter*, *Yersinia*, and *Pseudomonas* spp. The recipient becomes pyrexial at >40°C and hypotensive. This may occur during or hours after the transfusion and, unlike febrile transfusion reactions, is not self-limiting.

- Volume resuscitation.
- Culture the patient, and send bag and giving sets to microbiology.
- Start empirical broad-spectrum antibiotics.

Non-bacterial

Pre-transfusion testing includes screening for hepatitis B (HBsAg, anti-HBc), hepatitis C (anti-HCV), HIV (anti-HIV-1/2, HIV-1 p24 antigen), human T-cell lymphocytotropic virus (HTLV) (anti-TLC-1/2), and syphilis. HIV and hepatitis C virus (HCV) can be transmitted by an infective, but seronegative, donor for 15–20 days after infection. CMV is common in the donor population (40–60%), and immunocompromised donors must receive leucocyte-depleted or CMV-negative blood. Malaria may be transmitted by blood transfusion, as may new-variant Creutzfeldt–Jakob disease (nvCJD).

Transfusion-related circulatory overload

Characterized by acute dyspnoea, high CVP, and hypoxia.

- Stop the transfusion.
- Give high-flow O_2 and loop diuretics (40mg furosemide IV).

Massive transfusion

- Replacement of the patient's whole circulating volume within 24h.
- Large volumes of this blood lead to a blood volume that has poor O_2-carrying capacity, ↑ K^+, hypothermic (if not warmed), and coagulopathic due to Ca^{2+} sequestration. Loss of such a large amount of blood also leads to depletion of clotting factors.
- Reduce the effect of massive transfusion by the following:
  - Use infusion warmers and a warming blanket.
  - Monitor central circulation and respiratory function closely.
  - Consider giving Ca^{2+} supplements (with care!).
  - Check platelets, APTT, and fibrinogen—replace if needed.
  - Check K^+ regularly.

Shock

Definition

Inadequate end-organ perfusion and tissue oxygenation. 'Cellular shock' is a term describing failure of normal cellular processes, including O_2 processing.

▶▶ *Recognizing shock*
- ↓ BP, ↑ pulse, and usually cold, clammy, pale, and sweating.
- Confused—may be agitated or drowsy.
- Young patients will compensate, with the only signs being ↓ pulse pressure, tachycardia, and ↓ urine output.

Emergency management

▶▶ Seek help early. There is often more to do in a short period of time than can be managed by one person.
- Assess the airway. If patent, give high-flow O_2 by non-rebreathing mask.
- Check carotid or femoral pulse.
- Secure IV access and start giving 500mL of crystalloid rapidly.
- Take a rapid history, and examine the patient to differentiate between the following types of shock.

Hypovolaemic shock
- *Causes*. Trauma, ruptured abdominal aortic aneurysm (AAA), ruptured ectopic, post-operative haemorrhage, profound dehydration, burns, pancreatitis.
- *Clinical features*. As above, with history of trauma/surgery/illness.
- *Treatment*.
  - Lie patient flat; high-flow O_2; lift legs to autotransfuse if no IV access.
  - Repeat fluid infusion 500mL IV rapidly; you should see rise in BP. Perform further fluid challenges as necessary.
  - Take blood and send for FBC, U&Es, clotting, and cross-match.
  - Take arterial blood gas (ABG); estimate Hb and K^+, as well as ABG.
  - If no rapid improvement in BP, look for other causes.

Anaphylactic shock
- *Causes*. Drug allergy, blood product reaction, latex allergy.
- *Clinical features*. History of sudden onset after administration of drug. Stridor or bronchospasm, angio-oedema, urticaria, pruritus, rash.
- *Treatment*.
  - Sit patient up; give high-flow O_2; call anaesthetist if stridor.
  - ▶▶ Give 0.5mL of 1:1000 adrenaline IM, repeating every 5min if no improvement.
  - Give 100mg hydrocortisone and 10mg chlorphenamine IV.
  - If wheezy, give 5mL of nebulized salbutamol.

Septic shock

- *Cause*. Overwhelming sepsis (➔ Sepsis, pp. 172–3).
- *Clinical features*. May be the same as hypovolaemic shock or, if established, with circulatory collapse. Earlier in the evolution, the patient may look 'septic'—pyrexial, flushed, bounding pulses.
- *Treatment*.
  - As for hypovolaemic shock.
  - May require addition of vasopressors if fluid-replete.
  - Take blood cultures; then give empirical broad-spectrum antibiotics.

Cardiogenic shock

- *Rapidly reversible causes.* Cardiac tamponade (trauma, post-cardiac surgery), arrhythmias, tension pneumothorax.
- *Other causes.* Fluid overload and congestive cardiac failure (CCF), MI, PE, subacute bacterial endocarditis (SBE), aortic dissection, decompensated valvular heart disease.
- *Clinical features.* History of recent surgery/trauma, chest pain, dyspnoea, palpitations, new cardiac murmurs.
- *Treatment.*
  - Give high-flow O_2.
  - Give 2.5mg morphine IV (anxiolytic, venodilator, analgesic, anti-arrhythmic).
  - Put patient on cardiac and sats monitors; request 12-lead ECG.
  - Treat arrhythmias (➔ Advanced Life Support (ALS) algorithm on inside back cover).
  - Treat MI with 300mg aspirin, 300mg clopidogrel, and LMWH/fondaparinux if no contraindications.
  - Auscultate heart sounds and lung fields.
  - Treat tension pneumothorax (➔ Pneumothorax, pp. 768–70) and cardiac tamponade (➔ Mediastinal disease, p. 772).
  - Discuss with intensive treatment unit (ITU).
  - Consider central venous and peripheral arterial monitoring.
  - Send blood for ABGs, FBC, U&Es, clotting, and troponin.
  - Catheterize the patient.
  - Request CXR—look for pulmonary oedema.
  - Treat fluid overload with diuretics—furosemide 40mg IV.
  - Consider TTE to exclude pericardial effusion and valvular lesions, and to assess LV function.

Post-operative haemorrhage

Post-operative haemorrhage may be arterial or venous. Significant arterial haemorrhage is rare and usually occurs from vascular anastomoses. Very rarely, it arises from solid organ injury or loosening of arterial ties. It is rapid, bright red in colour, and often pulsatile. Venous bleeding is a commoner cause of post-operative haemorrhage and is usually due to the opening up of unsecured venous channels or from damage to the liver or spleen at surgery. Although it is non-pulsatile, low pressure, and dark in colour, it can be very large volume and is every bit as life-threatening as arterial bleeding. Most post-operative bleeding is not overt and is contained within body cavities. Drains, even correctly placed, are an unreliable sign of bleeding. Rely on your clinical instincts, even if the drains are empty.

Causes and features

- **1° haemorrhage.** Occurs immediately after surgery or as a continuation of intraoperative bleeding. Usually due to unsecured blood vessels (e.g. liver bleeding following trauma).
- **Reactionary haemorrhage.** Occurs within the first 24h. Usually due to venous bleeding and is commonly thought to be due to improved post-operative circulation and fluid volume, exposing unsecured vessels that bleed (e.g. delayed splenic bleeding following minor trauma at laparotomy).
- **2° haemorrhage.** Occurs up to 10 days post-operatively. Usually due to infection of operative wounds or raw surfaces, causing clot disintegration and bleeding from exposed tissue.

Symptoms

- Confusion and agitation (due to cerebral hypoxia 2° to hypotension).

Signs

- Soaked dressings, acute wound swelling, blood in drains.
- Pallor, sweaty, tachypnoea, tachycardia, hypotension (a late sign in children and young adults).

Emergency management

Resuscitation

- Establish 2× large-calibre IV access. Give crystalloid fluid up to 1000mL bolus if tachycardic or hypotensive. Do not waste time trying to insert a central venous (CV) catheter—they are too long and too fine to be of use for rapid volume resuscitation.
- Attempt to control superficial bleeding with direct compression. Do not use tourniquets on limb wounds.
- Take blood for emergency cross-match if not already available. Detail an assistant to telephone blood transfusion for emergency cross-match of a minimum of 2U of blood.
- Consider tranexamic acid 1g IV (except post-vascular surgery).
- Inform senior help immediately if significant blood loss. Consider alerting theatres and/or ITU.
- Catheterize and place on a fluid balance chart if hypotensive, but stable.

Establish a diagnosis

The cause may be obvious from the bleeding or the operation.

- Read the operation notes. Is there any potential cause mentioned?
- If the bleeding is severe, the only way to establish a diagnosis may be at re-operation.
- If the patient is stable and re-operation is undesirable, consider imaging. CT scanning may reveal intra-abdominal or intrathoracic blood.
- Angiography may reveal active bleeding sites and may be therapeutic (coil embolization).

Definitive management

Most post-operative bleeding does not require re-operation, but if it does, it should always be done by a senior surgeon. If this is not the surgeon who performed the original surgery, it may be wise to try to contact them in case they can give useful information about the original procedure.

If re-operation is highly undesirable, e.g. rebleeding after solid organ trauma, then definitive conservative management might include:

- Radiologically guided embolization; correction of deranged clotting (➲ Blood products and procoagulants, pp. 120–1); controlled, permissive hypotension; and monitoring on ITU.

Wound haematoma

A localized collection of blood beneath the wound or at the site of surgery, usually characterized by swelling and discoloration.

- ► If this occurs after vascular surgery, flap surgery, or procedures on the limbs or neck, get senior help as urgent surgical exploration and evacuation may be indicated to avoid ischaemia, compartment syndrome, airway obstruction, flap failure, or ongoing haemorrhage.
- Apply firm pressure, followed by a pressure dressing.
- Check clotting and FBC, and treat appropriately (➲ Blood products and procoagulants, pp. 120–1).
- Withhold heparin/anticoagulants.
- Surgical management is the same as for haemorrhage.

Wound emergencies

Infection

Causes

Most wound infections are acquired from the patient's own flora. The majority are skin organisms (e.g. *Staphylococcus aureus*, *Staphylococcus epidermidis*), although the second commonest cause is contamination from opened viscera during surgery (e.g. *Escherichia coli* from the GI tract, *Pseudomonas* from the biliary tree).

Symptoms

- Pain and discharge in the wound.
- Malaise, anorexia, and fever (systemic inflammatory features).

Signs

- Fever, tachycardia.
- Red, swollen, tender wound (may be discharging pus or fluctuant due to contained pus).

Complications

- Bacteraemia is common, but rarely significant.
- Septicaemia is rare unless the organism is resistant or the patient is immunosuppressed.

Emergency management

Resuscitation

- Ensure there is IV access. Give crystalloid fluid up to 1000mL if tachycardic or hypotensive.

Establish a diagnosis

- Send any discharging pus for microscopy, culture, and sensitivities (M, C, & S).
- Send blood for FBC (Hb, WCC) and blood cultures.

Early treatment

- Give IV antibiotics if there are any systemic features. If there is no pre-existing infection, use anti-staphylococcals: flucloxacillin 1g + 500mg qds. If the patient is immunosuppressed or very unwell, add in broad-spectrum cover to include anaerobic cover: metronidazole 500mg IV tds and cefuroxime 1.5g IV + 500mg IV tds (consult with microbiologist if in doubt).
- If there is real concern about meticillin-resistant *Staphylococcus aureus* (MRSA) infection, consult microbiology and consider adding IV vancomycin 500mg. If so, monitor drug plasma concentration.
- Open or aspirate the wound if there is contained pus. Wash the wound and review daily.

Dehiscence

Wound dehiscence may be superficial (including skin and subcutaneous tissue) or full thickness/deep (involving fascial closures or bony closures). Full-thickness dehiscence may expose deep structures. In the abdomen, this includes the viscera which may protrude through the wound (evisceration).

Causes

Most wound dehiscences are 2° to wound infection. Contributory factors include immunosuppression, malnutrition, steroid use, poor surgical technique, and previous surgery or procedures. Occasionally, the dehiscence is due to intracavity pathology causing wound breakdown from within (e.g. anastomotic leakage causing enteric fistulation).

Symptoms

- Usually painless.

Signs

- Open wound and visible fat and fascia if superficial; visible viscera if full thickness. Occasionally associated organ dysfunction if involved by the accompanying wound infection (e.g. pericarditis/anterior mediastinitis in sternal dehiscence).

Emergency management

Resuscitation

- Ensure there is IV access.
- Calm the patient, particularly if there is any degree of evisceration.

Early treatment

- If there are exposed viscera, cover these with saline-soaked dressings.
- Give IV antibiotics if there are features of wound infection (as above). If the dehiscence is superficial, ensure the wound is open and any pus is fully drained. Lightly pack the wound with absorbent dressing (e.g. Sorbsan®).

Definitive management

Superficial

- Continue regular wound lavage and dressings.
- For large defects, consider vacuum-assisted closure.

Full thickness

- Resuturing/closure of the defect in theatre may be appropriate.
- For some deep defects, re-closure may be inappropriate (e.g. presence of infection, intestinal contents/fistulae, severe immunocompromise, physiologically unstable, intracavity pathology causing the dehiscence). In these cases, the wound should be allowed to form a chronic wound and close by 2° intention (e.g. called a laparostomy in the abdomen). This may be assisted by vacuum closure devices.

Bleeding

(➲ Post-operative haemorrhage, pp. 126–7.)

Cardiac complications

Arrhythmias

FOR ARRHYTHMIAS IN THE CARDIOTHORACIC PATIENT ➔ Common cardiac emergencies pp. 760–1.

Arrhythmias such as AF are common post-operatively, particularly if there is underlying IHD. The Resuscitation Council (UK) ALS guidelines provide a useful framework to manage the acutely unwell patient with a tachy-/bradyarrhythmia.

▶▶ Patients who exhibit the *adverse features* below are unstable and require *immediate* management:

- Chest pain.
- Pulmonary oedema/shortness of breath.
- Hypotension (systolic BP <90mmHg).
- Collapse/loss of consciousness.

Diagnosis

Aim to elucidate the underlying rhythm and cause for the arrhythmia.

- Take a careful history and examine the patient.
- Do a 12-lead ECG; if this is not readily available, use the rhythm strip on a cardiac monitor/defibrillator.
- Monitor BP, ECG, and saturations.
- Do bloods: FBC, U&Es, including magnesium (Mg^{2+}), CRP, and cardiac troponins.

Causes

- Hypovolaemia.
- Pain.
- Sepsis.
- Electrolyte abnormalities (e.g. K^+, Mg^{2+}).
- Myocardial ischaemia/MI.

Management

Aim to restore a normal rate (and/or rhythm if appropriate) and to treat the underlying cause. Keep in mind that PO agents are unsuitable in patients who have an ileus. ▶▶ If in doubt, seek expert help.

- Give O_2 if hypoxic; establish IV access.
- Obtain ECG monitoring; ensure access to defibrillation trolley.
- Seek advice from the medical registrar/cardiology.
- Anaesthetic/ICU support may be required.
- Treat underlying cause.

Tachyarrhythmias

- With adverse features: synchronized direct current (DC) cardioversion.
- Fast AF: β-blockers, diltiazem, digoxin (with cardiac monitoring).
- Supraventricular tachycardia (SVT): vagal manoeuvres, adenosine, amiodarone.
- Pulsed ventricular tachycardia (VT): amiodarone.

Bradyarrhythmias
- With adverse features: atropine 500-microgram bolus up to 3mg, transcutaneous pacing, isoprenaline or adrenaline infusion.
- Risk of asystole if recent asystole, Mobitz type II atrioventricular (AV) block, complete heart block, ventricular pauses >3s.
- Otherwise conservative management may suffice.

Chest pain

Taking a careful pain history should help differentiate between the causes of chest discomfort listed in Box 2.6 below.

Box 2.6 Causes of post-operative chest pain

Dull, central ache
- Myocardial ischaemia/MI.
- Gastric distension.

Central pain radiating through to back
- Thoracic aneurysm or dissection.
- Peptic ulcer disease, oesophagitis, rarely pancreatitis.

Pain on movement
- Musculoskeletal pain.
- Chest drains.

Pleuritic pain
- Chest infection.
- Pneumothorax.
- Haemothorax, pleural effusion, empyema.
- Chest drain *in situ*.
- PE.

Diagnosis
- Take a careful history and examine the patient.
- A CXR will demonstrate most lung pathology.
- A 12-lead ECG should help identify myocardial ischaemia/MI.
- Recent WCC and CRP help identify sepsis.
- Review previous medical history for peptic ulcer disease and the drug chart for NSAID use.

Myocardial ischaemia

Patients, particularly in vascular surgery, may have pre-existing IHD. Surgery can precipitate ischaemia through:
- Stress response to major surgery (endogenous catecholamine release triggered by anxiety and pain).
- Fluid overload post-operatively.
- Profound hypotension.
- Failing to restart anti-anginal medication post-operatively.

Perioperative myocardial infarction

Perioperative MI may be difficult to diagnose because the patient may be unable to give a good history or to distinguish between chest and upper abdominal pain (Box 2.7).

- The presentation is similar to that of myocardial ischaemia, but the duration is longer (>20min) and may be associated with haemodynamic instability, nausea, vomiting, confusion, and distress.
- The patient will often be cold and clammy, and may be hypoxic.

Box 2.7 Diagnostic criteria for MI

In the setting of symptoms suggestive of acute coronary syndrome:

- ECG shows ST segment elevation—ST segment elevation MI (STEMI).
- No ST elevation, but inversed T waves or troponin T/I positive—non-ST segment elevation MI (NSTEMI).

Management

- Attach an ECG monitor and get a 12-lead ECG.
- Make sure the defibrillator trolley is close at hand.
- Measure O_2 saturation and give high-flow O_2 if hypoxic.
- Obtain IV access.
- Titrate morphine IV up to 10mg and metoclopramide 10mg IV.
- Give aspirin 300mg po/PR and glyceryl trinitrate (GTN) 0.5mg SL.
- Contact cardiologists urgently for consideration of acute intervention.
- Further anticoagulation may be required (e.g. with clopidogrel, fondaparinux or LMWH) but depends on risk of bleeding.

Key revision points—physiology of coronary blood flow

- Myocardial cells extract up to 70% of O_2 from blood.
- Coronary blood flow occurs during diastole.
- Tachycardia reduces diastolic interval and increases O_2 demand, which may reveal occult ischaemia.
- Coronary vasodilatation is mediated by adenosine, K^+, hypoxia, and β2 stimulants and the N_2O pathway.

Respiratory complications

These are common after surgery as a result of the effect of GA, post-operative pain, and immobility.

Respiratory failure

Definitions of respiratory failure

- **Hypoxaemia.** PaO_2 <10.5kPa.
- **Hypercapnia.** $PaCO_2$ >6.0kPa. **Hypocapnia.** $PaCO_2$ <4.5kPa.
- **Type I respiratory failure.** PaO_2 <8.0kPa on air.
- **Type II respiratory failure.** PaO_2 <8.0kPa and $PaCO_2$ >6.0kPa.

Basic assessment and management

- Sit the patient up and give high-flow O_2 through a tight-fitting mask.
- Assess the airway for patency. Is the trachea central?
- Examine the chest. Is chest expansion symmetrical? Auscultate for bilateral breath sounds, poor air entry, wheeze, bronchial breathing, and crepitations.
- Assess circulation and treat shock, which causes hypoxaemia (➲ Shock, pp. 124–5).
- Treat bronchospasm with nebulized salbutamol 5mg.
- Get a CXR. Look for consolidation, oedema, effusions, and pneumothoraces.
- Obtain an ABG.

Chest infection

Diagnosis

- Cough with purulent sputum.
- Pyrexia.
- Bronchial breath sounds and reduced air entry on auscultation.
- Leucocyte neutrophilia, raised CRP.
- Consolidation on CXR.
- Culture of sputum may yield sensitivities of causative organisms.
- In the dyspnoeic, hypoxic patient, perform ABGs to guide immediate management.

Prevention

There is no good evidence that prophylactic physiotherapy helps to prevent chest infection after surgery. The single most important intervention is to prevent patients with active chest infections from undergoing surgery. Any elective patient with a current cough (dry or productive), temperature, clinical signs of chest infection, neutrophilia, or suspicious CXR should be deferred for a fortnight and then reassessed. Other risk factors include active smokers or those who have stopped smoking within the last 6 weeks; patients with COPD or obesity; patients requiring prolonged ventilation post-operatively; and patients who aspirate.

Management

- Physiotherapy helps the patient with a cough to expectorate sputum and prevent mucus plugging.
- Effective analgesia is important to allow patients to cough.
- Definitive treatment is antibiotics—refer to local antibiotic policy for hospital acquired pneumonia.
- If the patient requires O_2 (PaO_2 <8.0kPa on room air), humidifying it reduces the risk of mucus plugs and makes secretions easier to shift.
- Continuous positive airway pressure (CPAP) can be used to improve basal collapse.
- The hypoxic, tachypnoeic, tiring patient on respiratory support should be reviewed urgently by the critical care team.

Exacerbation of COPD

The incidence of moderate to severe COPD in surgical patients is 5%.

- Most studies show that moderate COPD is not associated with an increase in post-operative complications, mortality, or length of stay.
- Severe COPD and preoperative steroid use are associated with ↑ morbidity and mortality after surgery.
- Ensure that all patients on preoperative inhalers continue these during the perioperative period. If the inhaler technique is inadequate (e.g. due to pain), commence short-acting nebulized alternatives (e.g. salbutamol, ipratropium).
- In hypoxic patients with COPD, give O_2 via a Venturi mask and titrate against $PaCO_2$ and PO_2; do not restrict O_2 empirically.

Key revision points—monitoring/measuring lung function

- Pulse oximetry estimates the percentage of saturated Hb present in capillary blood by the change in wavelength ratios of absorbed red and infrared light. It is inaccurate in carbon monoxide poisoning, cold peripheries, low flow states, and tachydysrhythmias.
- PaO_2 can be approximately estimated from the SaO_2.
  - 95%, >12kPa.
  - 85%, ~8kPa.
  - 75%, <6kPa.
- Capnography works on similar principles; different gases (e.g. CO_2) absorb different amounts of infrared light.

Renal complications

Acute kidney injury

- Increase in serum Cr by ≤26.5 micromol/l within 48h; or
- Increase in serum Cr to ≤1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- Urine volume <0.5mL/kg/h for 6h.

Renal complications are not uncommon post-operatively. Whilst there is no consensus definition of acute renal failure, the term acute kidney injury (AKI) has been proposed to encompass the entire spectrum of disease, from mild injury to that requiring renal replacement therapy (RRT). Even mild renal injury has been associated with significantly ↑ morbidity and mortality.

Staging of AKI

Numerous definitions exist; commonly used are the RIFLE (risk, injury, failure, loss, end-stage) and AKIN (Acute Kidney Injury Network) criteria, both of which use serum Cr or urine output to determine severity. The KDIGO (Kidney Disease: Improving Global Outcomes) definition combines both of these and grades AKI into three stages. ↑ AKI stage requires more invasive monitoring and diagnostic workup, culminating in the need for RRT and ICU admission.

Risk factors

- *Preoperative risk factors.* Age >75; chronic kidney disease; LV dysfunction; HTN; DM; peripheral vascular disease; dehydration; sepsis; nephrotoxic drugs; obstructive uropathy; trauma; burns.
- *Intraoperative risk factors.* Cardiac surgery, aortic surgery, interventional radiology procedures, major non-cardiac surgery.
- *Post-operative risk factors.*
  - *Pre-renal.* Shock, hypovolaemia, sepsis.
  - *Renal.* Sepsis, hypoxia, drugs (e.g. NSAIDs, aminoglycosides, glycopeptides), rhabdomyolysis, autoimmune.
  - *Post-renal.* Obstructive uropathy, e.g. obstructed Foley catheter, prostatic hypertrophy.

Reducing risk of AKI

There are a number of perioperative measures that may reduce the risk of renal dysfunction.

- Ensure adequate hydration, particularly before undergoing procedures involving contrast.
- Identify and eliminate nephrotoxic medications where possible, particularly NSAIDs and ACE inhibitors.
- Avoid intraoperative hypotension.
- Consider invasive monitoring ± CO monitoring.
- Post-operatively maintain satisfactory BP and optimize intravascular filling.

Investigations/diagnosis

Aim to quantify the degree of renal injury; determine the cause of AKI, and identify complications thereof.

- Take a careful history and examine the patient.
- Take bloods for FBC, U&Es, and CRP; cultures if sepsis suspected.
- Obtain an ABG.
- Ultrasound kidneys/ureters/bladder (KUB).
- ECG if electrolyte abnormalities.
- Measure urine output and send urine chemistry if indicated.

Management of AKI

Involves avoiding the potentially lethal complications of renal failure (hyperkalaemia, acidosis, pulmonary and cerebral oedema, severe uraemia, and drug toxicity) and exacerbating the renal insult, with concurrent treatment of the underlying cause. Invasive monitoring/ICU support may be required, particularly if complications arise or RRT is required.

- Aim for mean arterial pressure (MAP) of >60mmHg by optimizing intravascular filling and adding vasopressors as required.
- Avoid 'chasing' urine output if adequate BP and well filled.
- Give O_2 if hypoxic.
- Once fluid-resuscitated, aim for neutral daily fluid balance to avoid pulmonary oedema.
- Monitor electrolytes at least daily, with K^+ and acid–base balance more frequently if required.
- Avoid K^+ supplements and medication that increase K^+ levels (ACE-inhibitors, K^+-sparing diuretics).
- Stop nephrotoxic drugs (e.g. aminoglycosides, NSAIDs, ACE-inhibitors. diuretics).
- Ensure adequate enteral or parenteral nutrition.
- Diuretics may be useful in the management of fluid overload whilst awaiting RRT but have no role in the treatment of AKI.
- Relieve obstructions, e.g. urinary retention, blocked catheters.
- Failure of conservative measures necessitate RRT.

Complications of AKI

Hyperkalaemia

Hyperkalaemia (K^+ >5.0mmol/L) is seen in the setting of renal failure, tissue necrosis, massive blood transfusion, and K^+-sparing diuretics and supplements. Severe hyperkalaemia (K^+ >6.0mmol/L) can cause life-threatening ventricular arrhythmias. ECG changes that herald myocardial dysfunction are flattened P waves, wide QRS complexes, tenting of T waves, and, in cardiac arrest, ventricular fibrillation (VF) or asystole.

- ▶▶ Treat the patient with ECG changes as an emergency.
- If K^+ >6.0mmol/L or if ECG changes present, give *10mL of calcium gluconate 10% IV over 2min*.
- Give 50mL of 50% glucose containing 10U of soluble insulin as an IV infusion over 10–20min, repeating as necessary, monitoring BS after each infusion.
- Salbutamol 5mg nebulized may also be used in addition.
- Refractory hyperkalaemia is an indication for RRT.

Pulmonary oedema

- Sit patient up and give high-flow O_2.
- Start CPAP.
- If oliguric, try furosemide 20–40mg IV as a temporizing measure.
- Pulmonary oedema is an indication for RRT.

Drug monitoring

AKI can rapidly increase serum drug concentrations to toxic levels, particularly if it depends on renal clearance. Measure drug levels and decrease doses accordingly, or use an alternative. Common examples include:

- Aminoglycosides, e.g. gentamicin, amikacin.
- Glycopeptides, e.g. vancomycin, teicoplanin.
- LMWH—measure anti-Xa levels.
- Digoxin.

Gastrointestinal complications

Paralytic ileus

This is the cessation of GI tract motility.

Causes

- Prolonged surgery, exposure and handling of the bowel.
- Peritonitis and abdominal trauma.
- Electrolyte disturbances (most can affect GI function!!).
- Anticholinergics or opiates.
- Prolonged hypotension or hypoxia.
- Immobilization.

Clinical features

- Nausea, vomiting, and hiccoughs.
- Abdominal distension, tympanic or dull on percussion.
- Air-/fluid-filled loops of small and/or large bowel on AXR.

Prognosis

- Intestinal ileus usually settles with appropriate treatment.

Treatment

- Pass an NGT to empty the stomach of fluid and gas if the patient is nauseated or vomiting.
- Ensure adequate hydration by iv infusion ('drip and suck').
- Maintain the electrolyte balance.
- Reduce opiate analgesia and encourage the patient to mobilize.
- Consider other causes (e.g. occult intra-abdominal sepsis) and consider nutritional status.

Post-operative mechanical small bowel obstruction

It is important to distinguish between mechanical obstruction and ileus since management may be different.

Causes

- Early adhesions (usually self-limiting).
- Internal, external, parastomal, or wound herniation.
- Intra-abdominal sepsis (usually slightly later presentation).

Clinical features

- Nausea and vomiting.
- Colicky abdominal pain.
- Abdominal distension, tympanic on percussion.
- Examine hernial orifices and stoma, if any, for incarcerated hernias.
- High-pitched 'tinkling' bowel sounds MAY be present.
- Dilated loops of small bowel (relative paucity of gas in colon).

Treatment

- As for paralytic ileus, with strict bowel rest.
- Consider CT scan to define diagnosis and level of the obstruction.

Prognosis

Surgery is rarely indicated (for suspected herniation or complications or, very occasionally, adhesional obstruction that fails to resolve).

Nausea and vomiting

This affects up to 75% of patients. It predisposes to ↑ bleeding, incisional hernias, aspiration pneumonia, ↓ absorption of oral medication, poor nutrition, and ↓ K^+.

Causes include: Prolonged surgery; anaesthetic agents, e.g. etomidate, ketamine, N_2O, opioids; spinal anaesthesia; gastric dilatation from CPAP; post-operative ileus; bowel obstruction; constipation; gastric reflux; peptic ulceration or bleeding; medications, including many antibiotics, NSAIDs, opiates, statins; pancreatitis; sepsis; and hyponatraemia.

Classification of antiemetics

Combining two different types of antiemetic increases efficiency.

Antiserotonergics

- Lowest side effect profile of all antiemetics, e.g. ondansetron 4–8mg PO/IV tds.

Antihistamines

- Sedation, tachycardias with IV injection. Painful with IV/IM administration, e.g. cyclizine 50mg IM/IV/PO tds.

Antidopaminergic agents

- Good against opioid nausea and vomiting, sedative, and extrapyramidal side effects, e.g. prochlorperazine 12.5mg IM, metoclopramide 10mg IV/IM/PO tds.

Anticholinergics

- Active against emetic effect of opioids, sedation, confusion, and dry mouth, e.g. hyoscine (scopolamine) 0.3–0.6mg IM.

Constipation

Failure to pass stool is common. Caused by lack of privacy, immobility, pain from wounds or anal fissures, dehydration, poor nutrition, ↓ dietary fibre, opiates, iron supplements, and spinal anaesthesia.

Treat with: bulking agents (e.g. ispaghula husk one sachet po bd); stool softeners, e.g. sodium docusate up to 500mg daily in divided doses; osmotic agents, e.g. lactulose 15mL bd; stimulants, e.g. senna one tablet bd po, bisacodyl 5–20mg nocte po.

Diarrhoea

Common causes in post-operative patients are: resolving ileus or obstruction; related to underlying disease or surgery (e.g. ileal pouch or Crohn's); antibiotic-related diarrhoea (send for M,C,&S); *Clostridium difficile* diarrhoea (send stool for *C. difficile* toxin) and pseudomembranous colitis (➲ Other forms of colitis, pp. 496–7).

Anastomotic leakage

(➲ Post-operative anastomotic leakage, pp. 518–19.)

Neurological complications

Delirium

Delirium is common post-operatively and is associated with ↑ morbidity and mortality. It is obvious in a disoriented, uncooperative, or hallucinating patient (hyperactive delirium). Frequently, however, it is more subtle, consisting of inactivity, quietness, slowed thinking, and labile mood, and it is only spotted by relatives or nursing staff (hypoactive delirium). Actively assess whether the patient is oriented in time, person, and place. Perform a quick Mini-Mental State Examination if you are still unsure.

Common causes of delirium

- Medication (e.g. benzodiazepines, opiates, anticonvulsants).
- Stroke.
- Hypoxia, hypercapnia.
- Shock.
- Sepsis.
- Alcohol withdrawal.
- Metabolic disturbances (↓ glucose, Na^+, pH; ↑ Ca^{2+}, Cr, urea, bilirubin).
- Post-ictal.
- Preoperative dementia.

Management

- Conservative measures include re-orientating the patient to time and place in a calm, quiet environment with natural light. Do not forget glasses and hearing aids.
- If the patient's behaviour poses a physical danger to themselves or others, it may be necessary to sedate as first-line management. Haloperidol 2.5mg may be given up to a total of 10mg in 24h po, im, or iv, but if the patient remains disturbed, a low dose of an atypical antipsychotic (e.g. quetiapine, olanzapine) may be used.
- Assess and treat hypoxia and hypotension.
- Reassess the drug chart—stop opiates and benzodiazepines.
- Correct metabolic abnormalities, e.g. ↓ glucose, ↓ Na^+.
- Alcohol withdrawal is diagnosed from a history of chronically high alcohol consumption, often with raised gamma glutamyl transferase (GGT), combined with psychomotor agitation post-operatively. It can be treated with the long-acting benzodiazepine chlordiazepoxide, alongside B vitamins (Pabrinex®) and thiamine replacement.
- Perform a neurological examination to look for focal neurological deficits, and consider head CT to exclude an infarct or haemorrhage.
- Reassure the patient and relatives—confusion is common, almost always reversible, and it is not a sign that the patient is 'going mad'.

Stroke

Stroke is commonest in vascular and cardiac surgical patients (2%), but elderly patients undergoing other major surgery are at risk.

Risk factors for stroke

- Increasing age (>80y risk of CVA 5–10%).
- DM.
- Previous history of stroke or TIA (increases risk 3-fold).
- Carotid artery atherosclerosis.
- Perioperative hypotension.
- Left-sided mural thrombus.
- Mechanical heart valve.
- Post-operative AF.

Aetiology

- *Embolic.* Carotid stenosis/atheroma, thrombus from AF.
- *Haemorrhagic.* Post-operative warfarinization, HTN.
- *Cerebral hypoperfusion.* Profound hypotension, raised intracranial pressure (ICP).
- *Hypoxia.*

Clinical features

Any neurological deficit resolving within 24h is called a TIA. Clinical features of perioperative stroke include:

- Failure to regain consciousness once sedation has been weaned.
- Hemiplegia (middle cerebral artery or total carotid artery occlusion).
- Initial areflexia becoming hyperreflexia and rigidity after a few days.
- Aphasia, dysarthria, ataxia (gait or truncal), inadequate gag reflex.
- Visual deficits, unilateral neglect, confusion, seizures.
- Persistent, marked HTN.
- Hypercapnia.

Diagnosis

The aims are to establish a definitive diagnosis, establish a cause to guide appropriate 2° prevention, and establish a baseline of function to help plan long-term rehabilitation or withdrawal of therapy.

- Carry out a full neurological examination (cognitive function, cranial nerves, and tone, power, reflexes, and sensation in all four limbs).
- Modern contrast head CT will show infarcts within 2h. You must distinguish between haemorrhagic and ischaemic CVAs (1 in 10 are haemorrhagic). MRI is necessary to image brainstem lesions.

Initial management

- Assess the airway, breathing, and circulation.
  - If the patient is unable to maintain their airway, insert a Guedel airway, bag and mask, ventilate with high-flow O_2, and call an anaesthetist.
  - Monitor O_2 saturations.
  - Monitor BP. It is unclear if BP should be lowered in ischaemic strokes (to minimize risk of haemorrhagic transformation). Aim for systolic BP <140mmHg in haemorrhagic strokes. Secure IV access and give a fluid challenge if indicated.
  - If the patient is able to maintain their own airway and is not haemodynamically compromised, explain what has happened and reassure them.
- Perform a full neurological examination.

- Put the patient NBM if there is no gag reflex; consider inserting a fine-bore NGT for feeding.
- Send FBC, U&Es, glucose, and clotting.
- Send blood cultures if there is any history of endocarditis and pyrexia.
- Request an urgent CT head.
- Request a carotid duplex ultrasound.
- Request an ECG and consider a TTE if an embolic stroke is suspected.

Thrombolysis

- Should be administered within 4.5h of onset of symptoms.
- Haemorrhagic stroke is an absolute contraindication.
- Recent surgery is a relative contraindication.
- Discuss all cases with the local acute stroke service.

Seizures

- Ensure IV access; check ABC's & DEFG ("Don't Ever Forget **GLUCOSE**)
- Check drug chart: have usual anticonvulsants been given? Withdrawal regime for EtOH (e.g. chlordiazepoxide) prescribed if appropriate

Management of status epilepticus (continuous seizure activity >5mins):

- Airway. Remove dentures; insert Guedel or nasophayngeal airway if unable to open jaw.
- Breathing. Give high-flow O_2; get Yankauer sucker and wall suction.
- Give lorazepam 4mg IV or diazepam 10mg IV over 2min (PR if no IV access); repeat once if seizures do not stop.
- Check BS and give 50mL of 50% glucose IV if BS <5mmol/L.
- If seizures persist, start phenytoin 18mg/kg IV at a maximum rate of 50mg/min in a separate line to the diazepam.

▶▶Seizures should resolve quickly. If they do not, get expert help immediately.

Haematological complications

Heparin-induced thrombocytopenia (HIT)

HIT occurs in about 5% of patients receiving heparin. It is characterized by the formation of complement-mediated heparin-dependent immunoglobulin G (IgG) platelet antibody. It occurs 5–10 days after initiation of heparin therapy or after the first dose of heparin in patients with previous exposure to heparin within the last 3 months.

Diagnosis

- Fall in platelet count by over 30% to $<150 \times 10^9$/L or by over 50%.
- *And* positive serology for HIT antibodies.
- Heparin-induced thrombocytopenia and thrombosis (HITT) occurs in about 20% of patients with HIT and is characterized by major thrombotic episodes. It has a mortality of about 30%.
- Patients may show tachyphylaxis to heparin, as well as bleeding complications.

Treatment

- Discontinue all heparin therapy, including heparinized saline flushes.
- If at all possible, delay any surgery requiring bypass until HIT antibodies are undetectable, and then follow standard heparinization, but do not use heparin in the post-operative period.
- If it is impossible to delay bypass surgery, then danaparoid and iloprost are alternatives to heparin, with the major disadvantages that they cannot be reversed after bypass and require specialized assays.
- Hirudin, iloprost, danaparoid, and warfarin are alternative anticoagulants to heparin in the post-operative period.
- Discuss with the haematologist.

Disseminated intravascular coagulation (DIC)

DIC may occur as a complication of sepsis, transfusion reaction, drug reaction, transplant rejection, and aortic aneurysm surgery. It is characterized by widespread activation of coagulation, resulting in the formation of intravascular fibrin, fibrin degradation products, consumption of platelets, and clotting factors, and ultimately, thrombotic occlusion of vessels. Patients may present with bleeding from indwelling venous lines, wounds, and minor abrasions.

Diagnosis

There is no single diagnostic test. The following findings suggest DIC:

- Sudden fall in platelet count to $<100 \times 10^9$/L.
- Bleeding and/or thrombotic complications.
- ↑ APTT, prothrombin time (PT), INR.
- ↑ fibrin degradation products.
- ↓ fibrinogen in severe DIC.

Management

The key is to treat the underlying derangements. Bleeding patients should receive FFP, platelets, blood, and cryoprecipitate, as indicated by coagulation screens. Patients with thrombosis should be heparinized.

Excessive warfarinization

- Warfarin inhibits carboxylation of vitamin K, inhibiting the synthesis of vitamin K-dependent factors (II, VII, IX, and X; proteins C, S, and Z) ➔ Bleeding and coagulation pp. 232–4 Management depends on whether the patient is bleeding, why they are warfarinized, and if urgent/emergency surgery is indicated. As a guide:
- If INR is >5 and <8 and the patient is not bleeding, omit warfarin and recheck INR daily, restarting warfarin once INR is <5.
- If INR is >8.0, omit warfarin and give 2mg vitamin K po.
- If INR is elevated and emergency reversal to normal clotting is required (e.g. for high-risk surgery), or if the patient is bleeding, IV Beriplex® (mixed factors II/VII/IX/X, protein C/S), in conjunction with PO/IV vitamin K, may be used.
- If the patient has a mechanical valve, the risk of thromboembolic events when anticoagulation is reversed is <0.1% per day. Anticoagulation with heparin or warfarin should be recommenced as soon as possible once any bleeding complications have resolved.
- ▶ Notify or Consult with senior surgeon and/or local haematology services

Deep vein thrombosis and pulmonary embolism

DVT is commonest in patients over 40y of age who undergo major surgery. A post-operative increase in platelets coupled with venous endothelial trauma and stasis all contribute (Virchow's triad). If no prophylaxis is given, 30% of these patients will develop DVT and 0.1–0.2% will die from pulmonary thromboembolism (pte).

High-risk groups

(➲ Prophylaxis—antibiotics and thromboprophylaxis, p. 98.)

DVT

Clinical features

- Pain, erythema, swelling of the leg, a rise in local skin temperature.
- May present with embolism (pulmonary).
- The DVT Wells score is useful in risk-stratifying patients.

Investigations

D-dimers are often raised post-operatively and are of little value in this context. DVT is mainly diagnosed using Duplex ultrasound. CT–or MR-venography are more sensitive but only used in special cases e.g. suspected iliac vein or IVC thrombus and planning for endovenous intervention eg thrombolysis.

Treatment

- Therapeutic LMWH or fondaparinux is the initial treatment of choice.
- In severe renal failure (estimated glomerular filtration rate (eGFR) <30mL/min/1.73m^2) use unfractionated heparin (UFH) infusion targeting APTT, or titrate LMWH doses according to anti-Xa levels.
- In patients with an ↑ bleeding risk, consider UFH infusion.
- Catheter-directed thrombolysis may be considered in selected patients with iliofemoral DVTs.
- If anticoagulation is contraindicated, a temporary inferior vena cava (IVC) filter may be inserted to prevent PE, but its use is controversial.
- Unless contraindicated, patients should be started on a vitamin K antagonist (warfarin) or a NOAC (e.g. apixaban) for at least 3–6 months.
- Refer patient to the local anticoagulation clinic.

Prevention

(➲ Prophylaxis—antibiotics and thromboprophylaxis, p. 98.)

Pulmonary embolism

Diagnosis of pulmonary embolism

PE is incorrectly diagnosed in almost 75% of patients. The differential diagnosis includes acute MI, aortic dissection, septic shock, chest infection, haemothorax, and pneumothorax.

Massive PE is PE resulting in haemodynamic compromise or where >30% of the pulmonary vasculature is compromised.

Clinical features of PE

- *Symptoms*. Dyspnoea, pleuritic or dull chest pain.
- *Signs*. Tachypnoea, tachycardia, hypotension, elevated JVP.
- *Risk factors* for, or clinical evidence of, DVT.
- *ECG* shows right ventricular (RV) strain pattern (S1, Q3, T3), but this is neither a specific nor a sensitive test.
- ↓ PaO_2; $PaCO_2$ may also be low.
- *CXR* classically shows normal lung fields.
- *Echo* may show RV dilatation, tricuspid regurgitation, and right atrial (RA) or RV thrombus.

Investigations

- PE may be diagnosed using:
  - Computerized tomography pulmonary angiography (CTPA) (gold standard, first-line test).
  - Ventilation/perfusion (V/Q) lung scan (if CTPA contraindicated eg in pregnancy).
- Consider TTE to assess RV function.

Outcome

The 30-day mortality of acute massive PE is about 50% (40% within the first 2h)

- The mortality of surgical intervention is up to 70% for patients requiring CPR or mechanical circulatory support preoperatively.
- The operative mortality of stable patients is about 30%.

Management

- If the patient is haemodynamically unstable, thrombolysis is the definitive management, provided there are no contraindications (e.g. surgery within 30 days).
- Otherwise do the following:
  - Sit the patient up and give 100% O_2.
  - Patient may require intubation.
  - Anticoagulate with LMWH/UFH/fondaparinux, as per treatment of DVT (see above).
  - Begin warfarinization or alternative PO anticoagulant.
  - Look for causes of PE.

Risk scoring

Scoring systems attempt to quantify the severity of illness so that:

- Different interventions, clinicians, or centres can be compared, adjusting for differences in case mix.
- Clinicians can predict prognosis more accurately and scarce resources can be allocated more appropriately.

Examples of risk scoring systems

- Predicting risk of dying in hospital (*APACHE III*).
- Quantifying morbidity (*ASA* score, *Apgar* score).
- Quantifying symptoms (*NYHA* cardiac classification).
- Predicting operative mortality (*POSSUM*; *EuroSCORE* or *Parsonnet* score for cardiac surgery).
- Predicting risk of dying for specific illnesses (*Glasgow* and *Ranson* criteria in pancreatitis).

POSSUM (Physiologic and Operative Severity Score for the enUmeration of Mortality and morbidity)

This is a weighted additive score. Twelve physiological and six operative variables are combined to produce a physiological score and an operative score, which, in turn, are combined to produce an estimate of the percentage risk of defined morbidity and mortality. Variants exist for vascular, oesophagogastric, and colorectal surgery, as well as the updated Portsmouth version (P-POSSUM).

EuroSCORE (European System for Cardiac Operative Risk Evaluation)

This is a weighted additive score, based on a European sample of cardiac surgical patients. Variables such as age, renal function, and comorbidity are given points that add up to an approximate percentage of predicted perioperative mortality. Scoring systems like this are useful when consenting patients for surgery and in risk-stratifying operative outcomes, so that surgeons and hospitals can be compared with one another.

APACHE (Acute Physiology and Chronic Health Evaluation) scoring system

This predicts an individual's risk of dying following admission to ICU. Several patient variables (physiological variables such as core temperature, heart rate (HR), BP, Cr, age, and chronic illness variables) are entered into a programme that gives a score which can be compared against previous performance to give a risk of dying in hospital. There are several variants, the most popular being APACHE II. APACHE III and IV are more accurate but have proprietary algorithms.

Critical care

Recognizing the critically ill surgical patient

(See Box 2.8.) It may be obvious that a patient needs a critical care bed, e.g. the patient needing ventilation, inotropes, or dialysis. But anticipating, and maybe avoiding, this is more difficult. The first step is recognizing compensated critical illness (e.g. shock compensated by tachycardia and peripheral shutdown or respiratory failure compensated by unsustainable respiratory effort).

Box 2.8 Signs that should ring alarm bells

- *History.* 'I feel like I'm dying.' Timor mortis (fear of dying) may accompany MI, hypovolaemic shock, and respiratory failure. Never ignore the patient who thinks they are dying; they are often right.
- *Nurses.* 'Mr Smith just doesn't look right.' Experienced nurses quickly recognize the patterns of critical illness—listen to them.
- *General.* Hypothermia or hyperpyrexia, sweating.
- *Cardiovascular.* ↓ BP, ↑ pulse, arrhythmias, peripheral shutdown.
- *Respiratory.* Tachypnoea, difficulty getting full sentences out.
- *Renal.* Oliguria <0.5mL/kg/h.
- *GI.* New anorexia, nausea, and vomiting.
- *Neurological.* Confusion, agitation or drowsiness, fits.

Immediate management

▶▶ First identify and treat potentially life-threatening conditions (resuscitate). Then quantify the problem (important for referring patients to other clinicians and for establishing a baseline by which to guide treatment and monitor progress). Finally start looking for the underlying problem. Some of these tasks overlap. Keep reassessing the patient and adjust your management.

Assess airway, breathing, and circulation. ALS algorithms are printed on the inside back cover; management of shock is described under ➲ Shock, pp. 124–5; management of haemorrhage is described under ➲ Post-operative haemorrhage, pp. 126–7.

- Sit patient up and give high-flow O_2.
- Secure IV access and take blood for FBC, U&Es, amylase, glucose, LFTs, cardiac enzymes, clotting, group and save ± blood cultures.
- Take an ABG—good O_2 saturations do not rule out respiratory failure and ABGs will also show acidosis and electrolyte abnormalities.
- Give a fluid challenge if the patient is not obviously overloaded.
- Request a 12-lead ECG.
- Review drug, diabetic, and fluid balance charts.
- Perform a focused history and examination—ask about symptoms that have changed recently and focus your examination on that.
- Review recent bloods and X-rays, and request appropriate radiology.
- If the patient might need HDU or ITU, talk to your seniors early.

High dependency unit

HDU allows a level of care between ICU and the general ward. Invasive monitoring and inotropic support are routine, but invasive ventilation and renal support are not. Nurse:patient ratio is 1:2. Patients with single organ failure requiring basic respiratory support, including non-invasive mask ventilation (NIV), should be admitted to HDU.

Guidelines for admission to HDU

- Need for a monitored bed.
- Need for invasive monitoring ± vasopressors/inotropes.
- Need for NIV or other respiratory support (e.g. high-flow nasal oxygen (HFNO)).
- Need for 1:2 nursing.
- Elective post-operative admission following major surgery.

Intensive care unit

The ICU offers advanced ventilatory and inotropic support, renal replacement therapy (RRT), full invasive monitoring, and 1:1 nursing care.

Guidelines for admission to ITU

- Need for mechanical ventilation.
- Failure of two or more organ systems.
- Need for advanced monitoring, e.g. pulmonary artery catheter (PAC).
- Need for escalating or additional inotropes.
- 1° pathology should be reversible.
- Consultant involvement from both surgery and ITU is essential.
- Patient's stated or written preference against intensive care should be taken into account and documented.

How to use critical care

The surgical team should consider using critical care services for both elective and emergency surgical patients. Some guides follow:

- Is the elective patient in need of intensive infusion treatment prior to surgery (e.g. IV anticoagulation, IV clotting factors)? → HDU.
- Has the patient undergone major surgery with significant transfusion requirements that might lead to haemodynamic and clotting abnormalities (e.g. elective extensive pelvic surgery, aortic surgery, extensive burns surgery)? → HDU.
- Is the patient over 80, having had major abdominal, thoracic, or limb surgery? → HDU.
- Does the patient have known significant respiratory disease, making invasive respiratory therapy likely? → ITU.
- Does the post-op patient need inotropic support, RRT, or invasive monitoring? → ITU/HDU.

► **The golden rule is, 'If in doubt, ask for the advice of the critical care team—more patients can benefit than do.'**

Invasive monitoring

Invasive monitoring is used on ITU and HDU because it provides accurate and sensitive real-time measurements, routes for sampling blood, and routes for administration of drugs. (*Also see* ➔ Table 18.1 Key haemodynamic formulae and normal values for key haemodynamic formulae).

Arterial monitoring

Insertion technique and complications are described under ➔ Arterial puncture and lines, p. 260–1.

Indications

- Continuous measurement of BP when inotropic or mechanical support is used.
- Frequent sampling of ABGs.

Contraindications

- *Absolute.* Infection at the site of insertion, distal limb ischaemia, AV FISTULA.
- *Relative.* Coagulopathy, proximal obstruction, surgical considerations.

Central venous pressure lines

Insertion technique and complications are described under ➔ Insertion of central venous catheter, p. 256–7.

Indications

- Continual RA pressure (RAP) measurements in patients requiring circulatory support.
- Infusion port for drugs given centrally (including total parenteral nutrition (TPN)).
- Insertion of PACs or transvenous pacing wires.

Contraindications

- *Absolute.* Superior vena cava (SVC) syndrome, infection at the site of insertion.
- *Relative.* Coagulopathy, undrained contralateral pneumothorax, uncooperative patient. DVT of the head and neck vessels may make insertion impossible. Patients with septal defects are at risk of CVA from air emboli caused by poor technique.

Pulmonary artery catheter

Complications are as for CVP lines, additionally arrhythmias and pulmonary artery infarction or perforation. The catheter has four lumens: a proximal lumen 25cm from the tip that sits in the RA; a distal lumen connected to a pressure transducer sitting in the pulmonary artery; a balloon lumen allowing balloon inflation; and a thermistor lumen. The tip of the catheter is 'floated' into a pulmonary artery where it is then 'wedged'.

Indications

These are indicated in patients with hypoperfusion states refractory to first- and second-line inotropic support.

- Pressure monitoring: RAP, RV pressure (RVP), pulmonary artery pressure (PAP), and pulmonary artery wedge pressure (PAWP).
- Flow monitoring: CO measurement using thermodilution.
- Mixed venous O_2 saturations.
- Derived parameters: systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), stroke volume (SV), all of which can be indexed to body surface area.

Contraindications

As for CV catheters. Those specific to pulmonary artery catheterization include tricuspid or pulmonic valvular stenosis, RA and RV masses that may embolize, tetralogy of Fallot, severe arrhythmias, and coagulopathy.

Minimally invasive cardiac output monitoring

Avoids the morbidity associated with PACs but still requires an arterial line at the very least. Various systems are available, the most popular relying on derivation of flow data from the arterial waveform. Accuracy is ↑ by calibrating the system at set intervals (central line required) using a lithium dilution method (e.g. LiDCO) or thermodilution method (e.g. PiCCO).

Waveforms

(See Fig. 2.8.)

The arterial waveform

The arterial waveform has a fast upstroke and a slower downstroke, with a notch that represents aortic valve closure.

The CVP waveform

The waveform is composed of three upstrokes (the 'a', 'c', and 'v' waves) and two descents (the 'x' and 'y' descent). The 'a' wave—atrial systole; 'v' wave—venous return filling the RA; 'c' wave—bulging of the closed tricuspid valve cusps into the RA. The 'x' descent occurs in atrial diastole. The 'y' descent occurs in ventricular diastole.

The pulmonary artery pressure waveform

Waveform progression during correct insertion of the PAC shows a sudden increase in systolic pressure as the catheter enters the RV. As the catheter enters the pulmonary artery, the diastolic pressure increases. There is a decrease in mean pressure as the catheter enters the wedge position.

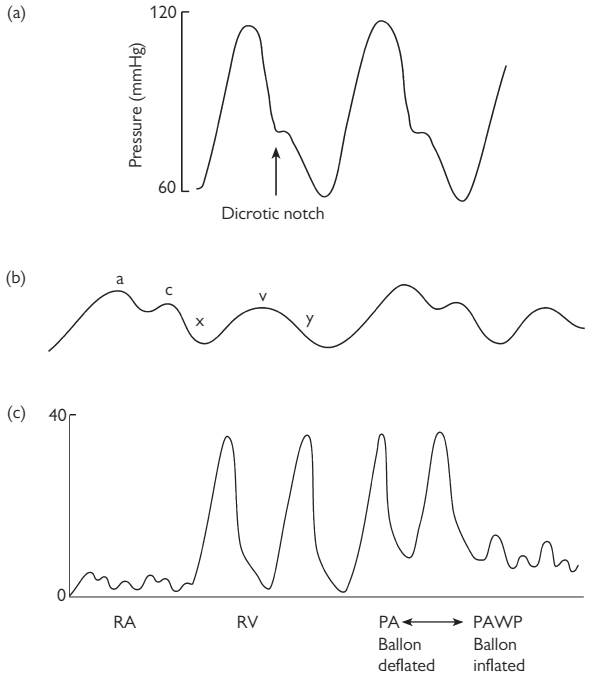

Fig. 2.8 (a) Arterial waveform. (b) Central venous pressure waveform. (c) Pulmonary artery pressure waveform.

Ventilation and respiratory support

The aims of respiratory support are to maintain adequate oxygenation and ventilation, while minimizing barotrauma, volutrauma, and atelectrauma.

- **Oxygenation**, the amount of O_2 in arterial blood, is described in terms of the partial pressure of O_2 in arterial blood (PaO_2).
- **Ventilation**, the movement of air in and out of the lungs, is described in terms of minute volume, and assessed by measuring the partial pressure of carbon dioxide in arterial blood ($PaCO_2$). $PaCO_2$ is inversely related to minute volume. Oxygenation is independent of minute volumes until they are very low.

Respiratory support may be achieved via **invasive** (e.g. tracheal intubation) or **non-invasive** (e.g. tight-fitting mask) methods, utilizing a variety of ventilation modes (some unique to certain ventilators). In its simplest form, a pre-determined volume is delivered a set number of times a minute. Parameters that can be altered include the pressures at which ventilation is delivered (in place of volume), the level of positive end-expiratory pressure (PEEP), and ventilator behaviour when a spontaneous breath is detected.

Common ventilatory modes

Intermittent positive pressure ventilation (IPPV); controlled mechanical ventilation (CMV); pressure control ventilation (PCV)

Commonly used during routine surgery. A pre-determined volume or pressure is delivered a set number of times a minute, regardless of patient effort. If not set appropriately, the *pressure* required to deliver the preset volume or the *volume* achieved with the preset pressures may be too high, causing barotrauma or volutrauma, respectively. This mode is poorly tolerated by the awake patient and is therefore used primarily with invasive ventilation.

Synchronized intermittent mandatory ventilation (SIMV)

A variation of IPPV where positive airway pressure is synchronized with patient-initiated breaths. Mandatory (machine-initiated) breaths are given if no spontaneous breaths occur in a preset time. Mandatory breaths do not occur at the same time as patient-initiated breaths. This can be either volume- or pressure-controlled. This mode can be used to wean patients from respiratory support, but any patient may safely be ventilated on SIMV.

Pressure support ventilation (PSV)

Patient-initiated breaths are supported with a preset positive airway pressure. The ventilator detects the change in flow as the patient begins inspiration and assists air inflow with positive airway pressure. Used as a weaning mode in invasively ventilated patients or can be delivered in a non-invasive manner via a tight-fitting mask in order to avoid invasive ventilation (e.g. exacerbation of COPD).

PEEP

A small positive airway pressure (5–10cmH_2O) maintained throughout expiration prevents the collapse of small airways and alveoli that occurs at the end of expiration.

- Functional residual capacity, intrapulmonary shunts, lung compliance, and PaO_2 are improved and the work of breathing is reduced.
- High levels of PEEP (>15cmH_2O) may be necessary, but increase intrathoracic pressure, reducing venous return and CO.
- Physiological PEEP is provided by an intact glottis—patients with COPD purse their lips in expiration to increase physiological PEEP.

Continuous positive airway pressure

A standing pressure applied throughout all phases of respiration. It is more commonly administered non-invasively via a tight-fitting face mask in order to improve *oxygenation* (e.g. type I respiratory failure, pulmonary oedema), but can also be used as part of weaning strategy in invasively ventilated patients. Does not aid *ventilation*.

Oxygen delivery methods

- **Nasal prongs** (variable performance). Nasal prongs deliver low flows of O_2 (2–4L/min) in a less obtrusive way, allowing the patient to expectorate and eat. They increase fraction of O_2 in inspired air (FiO_2) to barely more than room air levels, particularly if the patient breathes through their mouth.
- **Hudson face mask** (variable performance). A loose-fitting mask which allows delivery of O_2 flows at rates higher than nasal prongs (2–15L/min). It is diluted by air drawn into the mask, which depends on the patient's minute volumes, ranging 5–30L/min. The FiO_2 achieved depends primarily on the patient and the delivery system should not be used when accurate control of FiO_2 is required. The maximum FiO_2 that can be reliably delivered is about 30%. Use of a non-rebreathing mask and reservoir bag increases FiO_2 to up to 60%—the reservoir bag must be filled with O_2 before the patient uses it.
- **Venturi face mask** (fixed performance). The Venturi valve draws in a fixed amount of air through calibrated inlets, which is mixed with O_2 flowing into the valve before entering the mask. The FiO_2 is set by the choice of valve, not by the patient's breathing pattern (hence the term fixed performance). The maximum FiO_2 that can be delivered by a Venturi mask is about 60%. There is a minimum flow rate of O_2 for each Venturi (this is usually indicated on the mask).
- **HFNO** (fixed performance). Humidified O_2 is delivered at rates of up to 60L/min via specially designed nasal prongs, matching the patient's maximal peak inspiratory flow. This allows for FiO_2 close to 100% O_2 to be inhaled. Humidification allows for such high flows to be tolerated comfortably. It is thought that a small degree of PEEP is also created.

Circulatory support

Principles

Improving cardiac function and end-organ perfusion involves:
- Careful fluid balance to optimize preload or 'filling'.
- Using vasoconstrictors and vasodilators to optimize afterload;
- Using inotropes and chronotropes to improve CO.
- Mechanical support in selected cases.

No one vasopressor or inotrope has been demonstrated to be superior to another. Use is largely determined by local practice and experience. ▶ Vasopressors and inotropes are not treatments for hypovolaemia. Ensure adequate 'filling' prior to commencing.

Inotropes

Inotropes increase myocardial contractility

- α1-adrenergic receptor stimulation leads to ↑ SVR and ↑ PVR.
- β1 stimulation leads to ↑ contractility, HR, and conduction.
- β2 stimulation causes vasodilatation and bronchodilation.
- Dopamine (DA) receptor stimulation causes coronary, renal, and mesenteric vasodilatation.

Adrenaline (epinephrine) 0.03–0.5 micrograms/kg/min or bolus

Catecholamine produced by the adrenal medulla.
- *Action*. Direct agonist at α1-, β1-, and β2-adrenergic receptors.
- *Pharmacodynamics*. Instant onset, half-life 2min. Metabolized by MAO.
- *Indications*. Cardiac arrest (1mg IV bolus); anaphylaxis (0.5mg IM); low CO states (infusion).

Dopamine 1–5 micrograms/kg/min (low dose), 5–15 micrograms/kg/min (medium dose), 20–50 micrograms/kg/min (high dose)

An endogenous catecholamine, precursor to noradrenaline (NA) and adrenaline.
- *Action*. α1, β1, β2 and DA1 agonist, and release of stored neuronal NA. At low doses, thought to improve renal perfusion via DA effects. At medium doses, β effects predominate. At >10 micrograms/kg/min, α effects predominate.
- *Pharmacodynamics*. Fast onset, slow offset. Metabolized by MAO.
- *Indications*. Low CO states.

Dobutamine 5–15 micrograms/kg/min

A synthetic catecholamine.
- *Action*. β1-adrenergic receptor antagonist.
- *Pharmacodynamics*. Half-life 5min. Metabolized by catechol-O-methyltransferase (COMT).
- *Indication*. Low CO state in the setting of ↑ SVR.

Milrinone 0.3–0.8 micrograms/kg/min
Bipyridine derivative that inhibits phosphodiesterase.
- *Action*. Potent inotropic and vasodilating effects by inhibiting the hydrolysis of cyclic adenosine monophosphate (cAMP) by phosphodiesterase III.
- *Pharmacodynamics*. Half-life 2.3h; 10% hepatic metabolism, 85% renal excretion.
- *Indication*. Low CO state.

Vasopressors

Vasopressors cause vasoconstriction
- α1-adrenergic receptor stimulation leads to ↑ SVR and ↑ PVR.

Noradrenaline (norepinephrine) 0.03–1.0 micrograms/kg/min
Catecholamine produced by the adrenal medulla and post-ganglionic neurons of the sympathetic nervous system.
- *Action*. Direct α1 agonist: potent vasoconstriction + β1 effect (at higher doses) = ↑ BP.
- *Pharmacodynamics*. Immediate onset; half-life 2min.
- *Indication*. Hypotension because of low SVR in normal to high CO states.

Metaraminol 0.5mg IV bolus, 1–5mg/h IV infusion
Sympathomimetic amine, can be given peripherally.
- *Action*. Direct α1 agonist.
- *Pharmacodynamics*. Immediate onset; half-life up to 20min.
- *Indication*. Hypotension because of low SVR, e.g. sepsis.

Chronotropes

Chronotropes increase heart rate
- β1 stimulation leads to ↑ contractility, HR, and conduction.
- Muscarinic cholinergic activity is predominantly parasympathetic.

Atropine (0.3–0.6mg IV bolus)
Atropine is a belladonna alkaloid.
- *Action*. A competitive antagonist at muscarinic cholinergic receptors, reducing parasympathetic tone to 'reveal' underlying sympathetic tone.
- *Pharmacodynamics*. Almost instant onset. When given iv, offset is 15–30min. When given im, sc, or po, 4h. Renal elimination.
- *Indication*. Bradydysrhythmias, reduction of oral secretions.

Isoprenaline (0.01–0.3 micrograms/kg/min)
Synthetic catecholamine.
- *Action*. Direct β1 (↑ contractility, conductivity, and HR) and β2 (↑ vasodilatation and bronchodilation) effect. No α activity.
- *Pharmacodynamics*. Plasma half-life 2min. Hepatic metabolism by MAO, 40% conjugated, 60% excreted unchanged.
- *Indication*. Bradycardia unresponsive to atropine.

Mechanical support

Intra-aortic balloon counterpulsation

- *Mechanism*. The intra-aortic balloon pump (IABP) is a polyethylene balloon filled with helium, ranging from 2 to 50cm³ in size, that sits in the descending aorta just distal to the left subclavian artery. The balloon inflates in early diastole, improving coronary perfusion, and deflates just before systole, reducing afterload. The myocardial O_2 supply:demand ratio is improved and CO may be ↑ by up to 40%.
- *Indications*. Weaning from cardiopulmonary bypass, refractory myocardial ischaemia, cardiogenic shock.
- *Contraindications*. AR (balloon inflation during diastole will worsen AR). Aortic dissection.

Ventricular assist devices (VADs)

These are pumps that are anastomosed to the great vessels or cardiac chambers to support failing RV, LV, or both ventricles. Only used in specialized units, typically as a bridge to transplantation.

Renal support

The main aims of treatment of renal failure are to:

- Maintain renal perfusion.
- Optimize fluid balance.
- Correct hyperkalaemia and acidosis.
- Provide adequate enteral support.
- Target therapy to underlying pathology.
- Identify and treat sepsis.
- Provide RRT.

Renal replacement therapy

Indications for renal replacement therapy

- Refractory hyperkalaemia (➲ Hyperkalaemia, p. 137).
- Refractory acidosis.
- Pulmonary oedema.
- Drug toxicity.
- Progressive uraemia or uraemia associated with pericarditis, encephalopathy, seizures, or coagulopathy.

In the acute setting, RRT can be delivered in a continuous or intermittent manner, via the techniques of haemofiltration and haemodialysis. Most ICUs utilize continuous haemofiltration, with or without the addition of a dialysis circuit, in contrast to intermittent dialysis utilized by renal units. In both methods, access to the circulation is required and blood passes through an extracorporeal circuit that includes either a haemofilter or a dialyser.

- In **haemofiltration**, blood under pressure passes down one side of a highly permeable membrane to the other side of which is a static crystalloid solution.
  In **haemodialysis**, blood flows along one side of a semi-permeable membrane as a solution of crystalloids is pumped against the other side of the membrane, against the direction of blood flow.
- In **haemofiltration**, removal of small and medium-sized molecules depends on convective flow as in glomerular filtration—all molecules and virtually all ions are removed at a similar rate.
  In **haemodialysis**, removal of solutes depends on diffusion—molecules move from high to low concentration and smaller molecules move faster, so the amount of solute removed depends on its concentration in the dialysis fluid and on the size of the molecule.
- In **haemofiltration**, large molecules are so effectively removed that drugs such as heparin, insulin, and vancomycin may need to be replaced, and reduction in circulating inflammatory mediators and pyrogens leads to a reduction in pyrexia and systemic inflammation.
  In **haemodialysis**, large molecules are not efficiently removed.
- In **haemofiltration**, large amounts of salt and water are removed and must be replaced by an infusion of an appropriate amount of physiological crystalloid into the distal port of the haemofilter.
  In **haemodialysis**, controlled amounts of Na^+ and water are removed by creation of a transmembrane pressure gradient.

Types of haemofiltration

Haemofiltration has become the standard form of RRT for acute renal failure post-operatively. This is because the continuous nature of the process avoids the big swings in fluid balance and electrolytes that characterize dialysis. There are several variants.

- *Continuous veno-venous haemofiltration (CVVH)*. The most commonly used system in ICUs. Blood is drained and returned to a central vein. A peristaltic pump drives blood through a haemofilter, allowing control of blood flow and filtration rate.
- *Continuous veno-venous haemodialysis with filtration (CVVHDF)*. Some systems add a dialyser to the CVVH circuit allowing for more efficient effluent removal.
- *Continuous arteriovenous haemofiltration (CAVH)*. This is the original and simplest form of filtration, now rarely used. The femoral artery and vein are cannulated and blood passes through the haemofilter under arterial pressure alone. It is therefore less appropriate for patients with low CO states. Prolonged arterial cannulation carries complications.

Management of the haemofiltered patient

The aims of haemofiltration are to maintain acid–base and electrolyte homeostasis and achieve the desired fluid balance.

- *Vascular access*. A large-bore double-lumen catheter inserted into a central vein, e.g. VasCath, Permacath.
  ▶ Pre-existing AV fistulae should not be used.
- *Dose*. Theoretically, this is the volume of blood 'purified' per unit time but is commonly described in terms of 'effluent rate' (= ultrafiltrate rate + dialysis rate), usually 20–25mL/kg/h.
- *Fluid balance and haemodynamics*. Expect a degree of hypotension when commencing haemofiltration which may require vasopressor support. Fluid removal rate = effluent rate – (replacement fluid rate + additional infusions). Replacement fluids vary in composition but are generally physiological with a HCO_3^- or lactate buffer.
- *Anticoagulation*. Used to prolong circuit life. Can be limited to either the circuit or the patient, or none at all (at the expense of circuit life). Usually achieved via an infusion of heparin, prostacyclin, or citrate.

Nutritional support

Neglecting the patient's nutritional needs increases morbidity and mortality. (Assessment of nutritional status, p. 94.)

Routine management

- Patients may be started on oral intake directly after most forms of surgery unless contraindicated. There is good evidence of reduced pulmonary, septic, and anastomotic complications in patients fed early.
- Patients with a history of dyspepsia or peptic ulceration should be commenced electively on an iv H_2-receptor antagonist (e.g. ranitidine) or a PPI.
- Analgesia should be downscaled from IV and PO opiates to non-opiate analgesia such as paracetamol 1g qds po as soon as possible to avoid ileus, constipation, anorexia, nausea, and vomiting.
- Avoid NSAIDs in patients at risk of peptic ulceration.
- Give regular iv antiemetics to treat persistent nausea and vomiting.

Enteral nutrition

This is nutrition using the GI tract. It is superior to, safer, and cheaper than TPN.

Routes/methods for supplemented enteral nutrition

- Sip feeds.
- Fine-bore NGT with the use of an infusion pump.
- Nasojejunal tube (NJT) (needs endoscopic placement).
- Percutaneous endoscopic gastrostomy (PEG), radiologically inserted gastrostomy (RIG), and percutaneous endoscopic jejunostomy (PEJ).
- Open surgical gastrostomy or jejunostomy.

Examples of enteral feeds

- Standard feeds—Osmolite®, Nutrison® standard.
- Fibre enriched—Jevity®, Nutrison® Multi-Fibre.
- High energy—Nutrison® Energy, Ensure® Plus.

Enteral tube feeds come in sterile, ready-to-hang 500mL and 1000mL packs. Standard feeds are nutritionally complete, based on whole protein and provide 1kcal/mL. Fibre-enriched feeds also provide 1kcal/mL but contain soy polysaccharides that are soluble. These remain undigested, passing to the colon where they are fermented by bacteria to produce short-chain fatty acids which promote absorption of Na^+ and water, reducing diarrhoea. High-energy feeds provide 1.5kcal/mL and are used in patients requiring reduced fluid input.

- Renal failure patients require high-energy, low-volume, and electrolyte feeds. Nepro® provides 2kcal/mL and high protein content.
- Drugs can be given by the NGT used for enteral feeding. Liquid preparations designed for PO administration should ideally be used, as crushed tablets may block the tube. Avoid crushing and administering prolonged-release tablets via NGTs. The feed should be stopped and the NGT flushed with saline before and after administration of drugs.

▶ Interactions: The therapeutic effect of warfarin is reduced by the vitamin K content in feeds; it is often necessary to increase the dose of warfarin. Phenytoin interacts with enteral feeds, which should be stopped for 2h before and after phenytoin administration.

Indications for supplementary enteral feeding

- Inadequate oral intake due to anorexia, practical difficulties with feeding, being on ITU/HDU, drug-induced nausea, poor oral health.
- Hypercatabolism exceeding normal intake (e.g. sepsis, malignancy, trauma, burns).
- Gut absorption impaired with excessive losses: chronic diarrhoea, high-output stoma or fistula (usually low-residue or elemental feeds to maximize absorption).

Complications of supplementary enteral feeding

- Complications of **feeding tube**. Malposition, blockage, wound infection around PEG/PEJ tubes.
- Complications of **administration**. Regurgitation and pulmonary aspiration, diarrhoea, bloating, nausea, cramps.
- Complications of **contents**. Vitamin and trace mineral deficiencies, electrolyte imbalances, drug interactions.
- **Diarrhoea** is the commonest complication. If feed-related, reducing the osmotic load is effective (using half-strength feed or slowing the infusion). Omeprazole, co-phenotrope, and erythromycin may be tried.

▶ It is mandatory to exclude other causes of diarrhoea such as subacute obstruction and infectious causes (particularly *Clostridium difficile*).

Total parenteral nutrition

TPN is commonly encountered in surgical wards. It is a major advance in the treatment of surgical malnutrition but has serious side effects and both long- and short-term potential complications.

Routes of administration for TPN

- *Peripheral parenteral nutrition (PPN).* Given via a medium-calibre cannula in a peripheral vein. Maximum calorie input limited by the maximum osmolarity of the solution. Avoids the risks of CV cannulation. Usually used for short-term supplementation.
- *Central (TPN).* Given into a central vein (SVC or brachiocephalic). May be via a dedicated tunnelled line (e.g. Hickman line), a conventional CV cannula, or a peripherally inserted central venous catheter (PICC line). Maximum calorie input only limited by volume of fluid that can be infused. Carries risks of CV catheterization.

General risks of TPN/PPN

- Hyperosmolarity.
- Lack of glycaemic control.
- Micronutrient deficiencies.
- Liver cell dysfunction, cholestasis, and pancreatic atrophy.
- Fluid volume overload.

Specific catheter-related risks of TPN

- Complications of insertion (air embolism, pneumothorax, vascular injury, dysrhythmias).
- Catheter thrombosis and thromboembolism.
- Central line infection, infective endocarditis, and bacteraemia.

Care of TPN patients

Patients on TPN require regular review and monitoring, including:

- U&Es (initially daily, then twice weekly once established).
- Glucose (initially od, then twice weekly unless signs of abnormal glucose levels).
- LFTs (twice weekly).
- Micronutrients, including Mg^{2+}, phosphate (PO_4), manganese (Mn), and copper (Cu) (weekly).

CV catheters require specific attention. They should not be used for non-TPN infusions and/or phlebotomy unless in exceptional circumstances, as this increases the risk of catheter sepsis dramatically. Dressings should be dated, changed regularly, and the catheter entry site kept clean.

Common indications for TPN

Short term

- Prolonged post-operative 'ileus' (unresolved GI tract dysfunction).
- Acute abdominal sepsis, with ITU likely (possibly slow PO route).

Long term

- Inability for GI tract to absorb adequate nutrition (e.g. extensive resection, extensive radiotherapy damage, extensive disease such as Crohn's).

Peripheral parenteral nutrition

TPN consists of specially formulated feed given IV. Because TPN solutions have high osmolality, they cause thrombophlebitis if infused into peripheral veins. They are given via a dedicated lumen in central lines (subclavian or internal jugular) which may be tunnelled for long-term use. PPN solutions have a reduced osmolality. When given through a fine-bore cannula and large peripheral vein, it minimizes the risk of thrombophlebitis. PPN should not be used as a permanent substitute for TPN, as the reduced osmolality limits the amount of nutrition that can be provided.

Indications

- Failure of bowel to absorb food, e.g. radiation damage, severe acute enteritis, or malabsorption syndromes.
- Inadequate length of bowel for absorption (short bowel syndrome due to Crohn's disease or after massive intestinal resection).
- GI tract not accessible for enteral route, e.g. oesophagogastric surgery, or disease where tube feeding not possible.
- Failure of enteral feeding to accomplish nutritional targets (rare).

Complications

- Complications of CV catheters, commonly line sepsis.
- Late complications: line migration, erosion, DVT, occlusion.
- Metabolic complications: ↑ glucose, refeeding syndrome.
- Hepatic complications: transient rise in LFTs often seen; prolonged rise due to hepatic steatosis, cholestasis, or cholelithiasis.

Refeeding syndrome

A syndrome seen after the reinstitution of nutrition in patients who are severely malnourished or metabolically stressed due to critical illness. Intracellular movement of already depleted stores of electrolytes, in particular PO_4, K^+, and Mg^{2+}, leads to life-threatening falls in serum concentrations. Manifests as confusion, seizures, coma, and arrhythmias. Can occur with both enteral and parenteral nutrition.

Diagnosis

- High index of suspicion in at-risk patients (prolonged period of little or no nutrition, low BMI, low levels of serum electrolytes prior to feeding). ↓ PO_4^{3-}, ↓ K^+, and ↓ Mg^{2+} after commencement of nutritional support.

Treatment

- Gradual increase in nutritional support, with the aim of achieving calorific requirements by day 7.
- Judicious replacement of electrolytes, with careful monitoring of biochemistry.

Acute respiratory distress syndrome (ARDS)

ARDS is a type of acute diffuse, inflammatory lung injury, leading to ↑ pulmonary vascular permeability, ↑ lung weight, and loss of aerated lung tissue.[1] An updated consensus definition was proposed in 2012 (Berlin definition). The mild, moderate, and severe categories of ARDS correspond to increasing morbidity and mortality (up to 45% in the severe ARDS).

Berlin definition

- *Timing*. Within 1 week of a known clinical insult, or new or worsening respiratory symptoms.
- *Chest imaging*. Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules.
- *Origin of oedema*. Respiratory failure not fully explained by cardiac failure or fluid overload. Need objective assessment (e.g. echocardiography) to exclude hydrostatic oedema if no risk factor present.

Oxygenation

- *Mild*: 200mmHg < PaO_2/FiO_2 ≥300mmHg, with PEEP/CPAP ≤5cmH_2O.
- *Moderate*: 100mmHg < PaO_2/FiO_2 ≥200 mmHg, with PEEP ≤5cmH_2O.
- *Severe*: PaO_2/FiO_2 ≥100mmHg, with PEEP ≤5cmH_2O.

Aetiology

ARDS can be seen following direct injury to lung tissue, e.g. pneumonia, or via indirect mechanisms, e.g. sepsis, major surgery, major trauma, burns.

Management

Management remains supportive, with optimization of ventilator strategies, targeted antimicrobial therapy, negative fluid balance, and additional organ support as necessary. Advanced ventilation strategies include the use of muscle paralysis, inverse ratio, and prone ventilation. Extracorporeal membrane oxygenation (ECMO) may be employed for patients refractory to conventional treatment.

Reference

1 ARDS Definition Task Force, Ranieri VM, Rubenfeld GD, Thompson T, *et al.* (2012). Acute respiratory distress syndrome: the Berlin definition. *JAMA* **307**: 2526–33.

Sepsis

The 2016 Sepsis-3 definition defines sepsis as 'life-threatening organ dysfunction caused by a dysregulated host response to infection'.[1] It is one of the leading causes of death worldwide. Patients may present with sepsis as a complication of an acute surgical disease or may develop sepsis following an elective or emergent operative procedure.

▶▶ Early recognition and timely intervention remain the cornerstone of sepsis management.

Screening for and diagnosing sepsis

Quick SOFA (qSOFA)

Patients with *suspected* infection and any two of the following:

- Altered mentation.
- Respiratory rate ≤22/min.
- Systolic BP ≥100mmHg.

Should be investigated for signs of organ dysfunction.

Identifying organ dysfunction

An acute change in the Sequential Organ Failure Assessment (SOFA) score of ≤2 in patients who have suspected or documented infection *have sepsis*. This is associated with an overall mortality risk of 10% in a general hospital population. Clinical and biochemical elements of SOFA include:

- PaO_2/FiO_2.
- Glasgow Coma Scale (GCS).
- Bilirubin.
- Cardiovascular—MAP ± vasopressor/inotrope use.
- Serum Cr and urine output.
- Platelets.

Septic shock

This is defined as sepsis and vasopressor therapy required to elevate MAP ≤65mmHg and lactate >2mmol/L despite adequate fluid resuscitation. Septic shock has an associated mortality of >40%.

Management

Both the Sepsis Six and the Surviving Sepsis Campaign (SSC) care bundles have been shown to improve outcomes in sepsis.

▶▶ Involve critical care early if there is no response to initial management.

Sepsis Six

Perform the following **within the first hour** of presentation.

1. Give O_2 to target sats >94% (or 88–92% if at risk of hypercapnic respiratory failure).
2. Take blood cultures before administering antibiotics.
3. Administer broad-spectrum IV antibiotics.
4. Start IV fluid resuscitation.
5. Measure urine output.
6. Measure serum lactate.

SSC bundle

To be completed **within 3h**:

- Measure serum lactate.
- Take blood cultures.
- Give broad-spectrum IV antibiotics.
- Give 30mL/kg of crystalloid for hypotension or lactate ≤4mmol/L.

To be completed **within 6h**:

- Vasopressors to maintain MAP ≤65mmHg if no response to fluid.
- Reassess volume status and tissue perfusion.
- Reassess lactate if initial lactate elevated.

Source control

- Source control for infections arising from specific anatomical diagnoses (e.g. necrotizing skin infections, peritonitis, bowel infarction) should be effected within 12h.
- Use the most minimally invasive, but effective, method available (e.g. percutaneous versus open).
- Intravascular lines which are suspected to be infected should be removed as soon as feasibly possible.

Reference

1 Singer M, Deutschman CS, Seymour CW, *et al*. (2016). The third international consensus definitions for sepsis and septic shock (Sepsis-3). *JAMA* **315**: 801–10.

Chapter 3

Surgical pathology

Introduction

Pathology forms the basis for understanding surgical diagnosis and management. This chapter aims to be a high-yield, clinically-oriented summary of pathology relevant to surgical practice. We focus on areas that are popular in undergraduate and postgraduate exams such as the Membership of the Royal College of Surgeons of England (MRCS) and the United States Medical Licensing Examination (USMLE). Key pathogenesis factors and clinical points are highlighted (►). For a more comprehensive review, you will need to refer to other texts (see the references at the end of this chapter for recommendations).

Cellular injury

Causes and mechanisms of injury

Cellular injury may be caused by a number of agents and via a number of cellular mechanisms.

Causes

- Trauma
- Thermal injury
- Chemicals, including drugs
- Microorganisms
- Ionizing radiation, including ultraviolet (UV) light

Mechanisms

- *Mechanical disruption*. Trauma, freezing, osmotic imbalance
- *Failure of membrane integrity*. Failure of ion pumps, cytolysis, trauma
- *Blockage of metabolic pathways*. Cellular respiration (e.g. cyanide), protein synthesis (e.g. streptomycin), deoxyribonucleic acid (DNA) damage or loss (e.g. X-rays)
- *Deficiency of essential metabolites*. Oxygen (ischaemia), glucose (diabetic ketoacidosis), hormones (↓ trophic hormones result in apoptosis)
- *Free radicals*. UV radiation (sunburn), toxins, ischaemia–reperfusion, intracellular killing of bacteria

Reversible versus non-reversible cellular injury

These may lead to reversible or irreversible cellular changes, which can lead to cell death by two different means: necrosis or apoptosis.

Reversible	Irreversible
Cellular swelling	Disruption of cell membrane (necrosis)
Clumping of nuclear chromatin	Lysosomal rupture
Ribosomal detachment	Ca^{2+} influx
↓ Adenosine triphosphate (ATP) synthesis	Nuclear pyknosis, karyolysis, karyorrhexis
Glycogen depletion	Mitochondrial permeability
Fatty change	

Necrosis

Necrosis is irreversible death of tissue or groups of cells due to lethal cell injury (cell membrane disruption) and release of intracellular components.

There are different patterns of necrosis. The three main types are: *coagulative*, *colliquative*, and *caseous*.

Coagulative e.g., heart (acute MI), kidney (acute tubular necrosis), liver.

Colliquative (liquefactive) e.g., brain, some bacterial infections. ▶ Release of powerful hydrolytic enzymes causes cystic/fluid areas.

Caseous necrosis e.g., tuberculosis (TB). Dead tissue lacks any structure and is characterized by a white, soft, 'cheesy' appearance.

Gangrenous e.g., limbs. Critical limb ischaemia or diabetes, GI tract—necrosis with dessication or putrefaction (➔ Gangrene and capillary ischaemia, p. 196).

Fibrinoid necrosis e.g., *blood vessels in malignant hypertension*. Necrosis of smooth muscle vessel walls allows seepage of plasma into the media and deposition of fibrin.

Fat necrosis ▶ ***e.g. acute pancreatitis. Fat is digested by pancreatic lipase to produce fatty acids which precipitate with Ca^{2+} ('saponification').***
Another example is direct trauma—the release of extracellular fat produces an inflammatory response, fibrosis, and, in some cases, a palpable mass.

Apoptosis

Apoptosis (programmed cell death) is the cell-mediated programmed elimination of individual cells.

Apoptosis is a physiologically driven process requiring energy. It is *initiated* by various extracellular and intracellular signals (e.g. **p53**, **bcl-2** family) and *executed* by endogenous **caspases** (cytoskeletal dismantling) and endonucleases (DNA breakdown). Finally, orderly piecemeal *disposal* of the cell occurs by **apoptotic bodies**, which bud off from the cell. Examples include:

- *Physiological*: all organ development during embryogenesis, hormone-mediated changes, e.g. endometrial lining during menstrual cycle, atrophy, etc.
- *Pathological, following cell injury*: ionizing radiation, hypoxia, toxins (e.g. chemotherapy), free radicals, viral infection.

Apoptosis	**Necrosis**
• Single or few cells	• Many cells/tissue
• Programmed, energy-dependent process (ATP required)	• Caused by injury, not energy-dependent,
• Cells shrink (cytoskeletal disassembly)	• Cells swell (water (H2O) influx)—cell membrane disrupted
• Nuclear shrinkage and basophilia (aka pyknosis), fragmentation (karyorrhexis), fading (karyolysis)	• Enzymatic digestion and protein denaturation from ruptured lysosomes cause haphazard destruction of cell components
• Orderly 'disposal system'—apoptotic bodies bud off and phagocytosed	• Debris stimulate inflammatory response
• No inflammatory response	

Inflammation

Inflammation is the local physiological response to tissue injury. It can be acute or chronic. Usually described with the suffix '-itis'.

Acute inflammation

This is usually the initial immunological reaction to a wide range of agents. Key features are:

- **Rapid in onset** (seconds to minutes) and lasts hours to days. **Vasodilatation** and ↑ **vascular permeability** → fluid exudation. **Leucocyte activation** (see Box 3.1).
- Main cell type is **neutrophils** (macrophages arrive later).

Box 3.1 Leucocyte activation

Neutrophils are recruited to a site of inflammation in four steps:

1. **Rolling**. E-selectin/P-selectin on vascular endothelium bind to Sialyl-Lewis X on leucocyte.
2. **Tight binding**. **Intercellular adhesion molecule 1 (ICAM-1)** on vascular endothelium binds to lymphocyte function-associated antigen 1 (LFA1—integrin) on the leucocyte.
3. **Diapedesis**. The leucocyte traverses between endothelial cells to exit the vessel lumen and enter the interstitial space (platelet endothelial cell adhesion molecule-1 (PECAM-1)).
4. **Migration (chemotaxis)**. Once in the interstitium, the leucocyte is guided by chemotactic signals (e.g. interleukin-8 (IL-8), C5a, leukotriene B4 (LTB4), kallikrein) to the site of injury/infection.

On arrival at the target: phagocytosis and killing mediated by **neutrophil NADPH oxidase → reactive oxygen species** production. This is aided by the complement system, which help with recruiting leukocytes ('opsonisation'), phagocytosis and attacking the pathogen cell-membrane (Fig. 3.1).

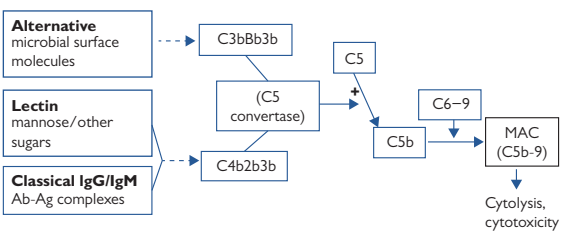

Fig. 3.1 The 'final common pathway' in complement activation is the conversion of C5 to C5b, which forms the membrane attack complex (MAC). This is catalysed by two enzyme complexes with C5 convertase activity: C3bBb3b (alternative) and C4b2b3b (lectin and classical). Dotted arrows signify multiple preceding steps.

Causes

- Infection (all microorganisms); physical/chemical injury, e.g. mechanical trauma; X-rays; acid, alkali; ischaemia; hypersensitivity.

Macroscopic appearance

Calor, rubor, tumor, dolor, and *functio laesa* (heat, redness, swelling, pain, and impaired function) are characteristic.

Specific types include: Serous—abundant exudates, e.g. peritonitis; Catarrhal—mucus hypersecretion, e.g. common cold; Haemorrhagic—e.g. pancreatitis; Suppurative/purulent—pus produced to form abscess or empyema; Fibrinous—exudate containing fibrin, e.g. pericarditis; Membranous—fibrin and epithelial cells, e.g. laryngitis; Pseudomembranous—superficial mucosal ulceration with slough, e.g. pseudomembranous colitis 2° to *Clostridium difficile*; Necrotizing (gangrenous) (➲ Gangrene and capillary ischaemia, p. 196).

Sequelae of acute inflammation

- Resolution. Restoration of tissue to normal (if minimal tissue damage, rapid destruction of causal agent, rapid removal of exudates by good vascular supply, and organs with restorative capacity, e.g. liver).
- Suppuration. Formation of pus.
- Organization. Replacement by granulation tissue.
- Chronic inflammation.

Exudate	**Transudate**
Hypercellular and protein-rich Specific gravity (SG) >1.020 Mechanisms: inflammation, malignancy, lymphatic obstruction	Hypocellular and protein-poor SG <1.0.12 Mechanisms: ↑ hydrostatic pressure (heart failure, Na^+ retention), ↓ oncotic pressure (cirrhosis, nephrotic syndrome)

also ➲ Pleural effusion pp. 766–7 Box 18.1 Lights criteria for differentiating an exudate from a transudate biochemically

Chronic inflammation

Key features:

- Macrophages, lymphocytes, and plasma cells predominate.
- Granulation tissue formation (proliferation of fibroblasts and blood vessels—part of healing ➲Wound healing, pp. 186–7). ❗ Not the same as a granuloma (see below).
- Persistent destruction and repair.
- Fibrosis.

Causes

- Resistance of infective agent to phagocytosis (TB, viral infections).
- Foreign body (endogenous, e.g. urate; or exogenous, e.g. asbestos).
- Autoimmune (e.g. contact hypersensitivity, RA).
- Unkown aetiology (e.g. ulcerative colitis (UC), sarcoidosis).

Macroscopic appearances

The commonest appearances are:

- Chronic ulcer, e.g. **peptic ulcer/chronic gastritis (*Helicobacter pylori*)**; chronic abscess cavity, e.g. empyema; thickening of the wall of a hollow viscus, e.g. **Crohn's disease**; fibrosis, e.g. **chronic cholecystitis**; granulomatous, e.g. **TB**.

Granuloma

A special type of chronic inflammation characterized by aggregates of modified macrophages (**epithelioid cells** and **giant cells**).

Causes

- Infections.
  - *Bacteria*: mycobacteria (**TB, leprosy**), tertiary syphilis, *Listeria*, *Bartonella* (cat scratch disease); *fungae*: some fungal pneumonias (e.g. **coccidioidomycosis**), histoplasmosis; *protozoa*: *Toxoplasma*, pneumocystis; *parasites*: schistosomiasis.
- *Foreign bodies*: silicosis, asbestosis, berylliosis, talc, sutures.
- *Drugs*. Sulfonamides, allopurinol.
- *Autoimmune*. RA, primary biliary cirrhosis, hypersensitivity pneumonitis (aka extrinsic allergic alveolitis).
- *Idiopathic*. **Crohn's**, **sarcoidosis**, **Wegener's**, **vasculitides** (Churg–Strauss, giant cell arteritis, Takayasu arteritis).

A granuloma is a nodular collection of epithelioid histiocytes and giant cells (macrophages). Initiated by interferon-gamma (IFN-γ).

Acute phase reactants

↑	↓
Ferritin	Albumin
CRP	Transferrin
Fibrinogen	
Serum amyloid A	
Hepcidin	

Important cytokines

IL-1	Macrophage	Fever, inflammation
IL-2	T-cells	T-cell growth and differentiation, and NK cell growth
IL-3	T-cells, NK	Bone marrow stem cells
IL-4	T helper 2 (Th2) cells	IgE and IgG; Th2 differentiation; B-cell growth
IL-5	Th2 cells	Immunoglobulin A (IgA) production; B-cell and eosinophil growth and differentiation
IL-6	Macrophage	Stimulates acute phase protein production
IL-7 and 9	(Various)	B- and T-cell growth factors
IL-8	Macrophage	Chemotactic factor for neutrophils
IL-12	T-cells	NK cell activation; Th1 differentiation
Tumour necrosis factor alpha (TNF-α)	Macrophage	Endothelial activation (leucocyte recruitment/↑ permeability); cachexia in malignancy; maintains granulomas in TB
IFN-γ	T helper 1 (Th1) cells	NK cell- and macrophage-mediated killing
IL-10	Th2 and Treg	Attenuate immune/inflammatory response. (as does transforming growth factor beta (TGF-β); inhibits Th1

Treg are regulatory T-cells. Th1/Th2 are the two main classes of helper T-cells. IL-10 and TGF-β tend to attenuate inflammation. Th2 cells secrete IL-4, 5, and 10; Th1 cells secrete IFN-γ.

Leucocytosis

Neutrophilia (aka 'left shift') Typically seen in acute bacterial infections (e.g. a good differentiator in acute appendicitis versus other differential diagnoses). Also seen in inflammation or stress (e.g. trauma, surgery, burns), drugs (steroids), disseminated malignancy, myeloproliferative disorders.

Eosinophilia ('worms, wheezes, and weird diseases') Mainly seen in **allergic** (atopic) diseases, including asthma; **parasitic** infections; skin disease (e.g. pemphigus, eczema, psoriasis); also acute myeloid leukaemia (AML), Hodgkin's lymphoma, some cancers, Churg–Strauss syndrome, Crohn's/UC, hypereosinophilic syndrome, and others.

Lymphocytosis Typically seen in acute **viral** infection; also in chronic infections (e.g. TB, hepatitis); haematological malignancy (acute lymphoblastic leukaemia (ALL), chronic lymphocytic leukaemia (CLL)) and autoimmune disease.

Hypersensitivity reactions

There are four types, which can be remembered using the mnemonic ABCD: I, **A**naphylactic/**A**topic; II, anti**B**ody-mediated; III, immune **C**omplex; IV, **D**elayed (T-cell-mediated).

Type I—anaphylactic and atopic
Mast cell-mediated

Two phases:

- Immediate (min): antigen (Ag) reacts with **IgE** on presensitized **mast cells** → degranulation of '**1° mediators**' (**histamine** and tryptase, chondroitin sulfate, serotonin).
- Late (hours): '2° mediators' (lipid-derived compounds and cytokines)—prostaglandins and leukotrienes (from arachidonic acid), platelet-activating factor (PAF), chemokines, and cytokines → inflammatory cell infiltrates.

e.g., anaphylaxis (food or drug allergy), hay fever, asthma, eczema.

Type II—antibody-mediated
Antibodies (Abs) bind to a cell surface antigen leading to:

- *Cellular destruction*—a cell that is coated by Abs ('opsonized') gets phagocytosed or NK cell killing.
  e.g., autoimmune haemolytic anaemia, transfusion reactions.
- *Activation of inflammation* via the complement system.
  e.g., Goodpasture's, rheumatoid fever, hyperacute transplant reaction.
- *Dysregulation* of intracellular molecular pathways.
  e.g., Graves' disease, myasthenia gravis, pemphigus.

Type III—immune complex
Ag–Ab complex + complement

Serum sickness—Ab produced to foreign protein, 1–2 weeks later forms Ag–Ab complex → deposit in tissues → complement activation → inflammation and tissue damage.

Arthus reaction—injection of Ag into a presensitized individual → immune complex formation and localized inflammation in the skin.

e.g., systemic lupus erythematosus (SLE), polyarteritis nodosa, post-streptococcal glomerulonephritis.

Type IV (delayed) hypersensitivity
T-cell-mediated

(Does not involve Abs)

1. Direct cell cytotoxicity: CD8+ cytotoxic T-cells kill targeted cells.
2. Effector CD4+ T-cells recognize Ag and release cytokines → inflammation.

e.g., contact dermatitis (and patch test), graft-versus-host disease; TB skin test (PPD).

Wound healing

General principles

Tissue healing in any organ follows some basic principles:

- Cells may be *labile* (good capacity to regenerate, e.g. surface epithelial cells), *stable* (capacity to regenerate slowly, e.g. hepatocytes), or *permanent* (no capacity to regenerate, e.g. nerve and striated muscle cells).
- Tissue architecture is important—complex arrangements cannot be reconstructed if destroyed, e.g. renal glomeruli.
- *Complete restitution* occurs when part of a labile population of cells is damaged, e.g. a minor skin abrasion.
- *Granulation tissue* is the combination of new capillary proliferation and (myo)fibroblasts, secreting collagen.

Stages of wound healing

When specialized tissue is destroyed, it cannot be replaced and a stereotyped response called **repair** then follows in four stages:

- **Haemostasis** (*immediate*). In response to exposed collagen, *platelets* aggregate at the wound and degranulate, releasing inflammatory mediators, with activation of clotting and complement cascades.
- **Inflammation** (*0–3 days*). ↑ vascular permeability, *neutrophil* migration; *macrophages* clear debris.
- **Proliferation** (*3 days to 3 weeks*). *Fibroblasts*, *myofibroblasts*, *keratinocytes*, *macrophages*. **Granulation tissue** and type III **collagen** deposition, angiogenesis (mediated by fibroblast growth factor (FGF)), epithelial cell proliferation, dissolution of clot; *myofibroblasts* mediate wound contraction via actin—can reduce tissue defect by up to 80%, but this can also cause problems, e.g. burns, contractures.
- **Remodelling** (up to 1y). Fibroblasts. Type III collagen is replaced by type I collagen, mediated by collagenases, for which zinc is a co-factor (zinc deficiency is associated with delayed wound healing). Maximum tensile strength occurs at ~3 months (this is the plateau of collagen production).

Growth factors in wound repair

- **FGF**—main stimulant of angiogenesis, wound repair (cell migration, epithelialization); also in development and haematopoiesis.
- **Platelet-derived growth factor (PDGF)**—stimulates fibroblast growth → collagen production.
- **Epidermal growth factor (EGF)**—stimulates cell growth (and angiogenesis, to a degree).

Factors affecting wound healing

Local factors

- *Vascular*. Impaired arterial supply or venous/lymphatic drainage.
- *Infection* or presence of foreign body or necrotic tissue.
- *Mechanical*. Excessive movement, local distension.

Systemic factors

- *General factors*. ↑ age, smoking.
- *Malnutrition*. Obesity, recent weight loss, nutrient deficiency (Zn).
- *Immunosuppression*. Cancer, HIV, drugs (e.g. steroids).
- *Anticancer therapies*. Radiotherapy and chemotherapy.
- *Metabolic*. Diabetes, jaundice, uraemia, musculoskeletal disease.

Specific cases

Blunt trauma to skin/soft tissue (bruising and haemosiderin)

Localized haemorrhage following blunt trauma leads to localized excess of free Hb. Gradual conversion of *red Hb* → green biliverdin → *brown* ***haemosiderin*** by macrophages leads to the characteristic colour changes observed in a bruise.

Skin: first intention healing

This takes place where there is close apposition of clean wound edges. Coagulated blood forms a surface scab which seals the wound. Fibrin precipitates to form a weak scaffold between the two edges. Capillaries proliferate to bridge the gap. Fibroblasts secrete collagen into the fibrin network. Basal epidermal cells bridge the gap and are eventually resorbed. The elastic network in the dermis cannot be replaced.

Skin: second intention healing

This takes place in wounds where skin edges cannot be cleanly apposed. There is phagocytosis to remove debris. Granulation tissue fills the defect. Epithelial regeneration covers the surface.

Gastrointestinal tract

- Erosion. Partial-thickness loss of the mucosa. Adjacent epithelial cells proliferate to regenerate the mucosa. Can take place in a few hours.
- Ulceration. Full-thickness loss of the mucosa. Mucosa is replaced from the margins. The muscularis propria cannot be regenerated; it is replaced by a scar. Damaged blood vessels bleed; fibrin covers the raw surfaces. Macrophages migrate in and phagocytose dead tissue. Granulation tissue is produced in the base. If the cause persists, the ulcer becomes chronic. Fibrous scar tissue may result in contractions.

Scars

Occur when repair cannot be achieved by cell regeneration alone—non-regenerated cells are replaced by connective tissue (collagen).

- *Two types*:
  - **Hypertrophic**—organized (parallel arrangement of) type I collagen, confined to borders of original wound, infrequent recurrence.
  - **Keloid**—disorganized type I and III collagen, extends beyond original wound. Recurrence is frequent. Higher incidence in darker skin and typically occurs in face and upper limbs.

Ulcers

An ulcer is a breach in an epithelial surface.

Causes

Venous, arterial (small or large vessel, including vasculitis), diabetic, other neuropathic, lymphoedema, malignancy, trauma, decubitus (pressure), pyoderma gangrenosum, infection (TB, HSV, syphilis). *Trophic ulcers* are caused by impaired nutrition to the involved part (vascular or neuropathic).

Features to note on examination

- Site. Neck/groin/axilla (TB); legs and feet (vascular); anywhere (malignant).
- Surface. Usually depressed. Elevated in malignancy, vascular granulations.
- Size. Measure the ulcer. Is it large in relation to the length of history?
- Shape. Oval, circular, serpiginous, straight edges.
- Edge. Eroded (actively spreading), shelved (healing), punched out (arterial, syphilitic), rolled or everted (malignant).
- Base. Fixed to underlying structures? Mobile? Indurated? Penetrating?
- Discharge. Purulent (infection), watery (TB), bleeding (granulation or malignancy).
- Pain. Usually occurs during the extension phase of non-specific ulcers. In diabetic patients, ulcers tend to be relatively painless due to neuropathy.
- Number. Widespread locally (local infection such as cellulitis), widespread generally (constitutional upset).
- Progress. Short history (pyogenic), chronic (vascular or trophic, e.g. post-phlebitic syndrome, decubitus ulceration of paraplegia).
- Lymph nodes. In the region of an ulcer may indicate 2° infection or malignant change.

Natural history

- Extension. There is discharge, thickened base, and inflamed margin. Slough and exudates cover the surface.
- Transition. Slough separates and the base becomes clean. The discharge becomes scanty, and the margins less inflamed.
- Repair. Granulation becomes fibrous tissue and forms a scar after re-epithelialization.

Investigations

- History, biopsy and histology, serology, as indicated by presentation.

Leg ulcers

- Their aetiology is diverse but can usually be diagnosed clinically.

	Venous	Arterial	Diabetic
Pathology	Superficial and/or deep venous insufficiency	Chronic arterial insufficiency, i.e. ischaemia	Neuropathic 50% Neuro-ischaemic 50%
Clinical	Previous DVT ('post-phlebitic limb') Varicose veins, oedema Pigmentation Lipoder matosclerosis	Evidence of peripheral arterial disease, e.g. claudication, rest pain, gangrene Absence of pedal pulses. Pallor, ↓ Capillary refill tim.	Warm foot with pulses (pure neuropathic) OR with coexisting arterial insufficiency; tend to be more aggressive, often presenting with deep tissue infection/abscess ± osteomyelitis
Distribution	Gaiter area (distal calf)	Toes, pressure points	Arterial distribution, particularly pressure points
Investigation	Venous duplex*	Arterial duplex	HbA1c Arterial duplex/ABPI

* For venous ulcers, it is also advisable to check pulses and ABPI as the main treatment is compression bandaging, which requires an intact arterial supply.

(see also ➲ The diabetic foot, pp. 794–5)

Other causes

- **Neuropathic**: remember that any other cause of neuropathy will predispose to ulcers (e.g. spina bifida, tabes dorsalis, leprosy, peripheral neuropathy), as will a bedridden state or reduced mobility.
- **Marjolin ulcer**: a painless carcinoma that develops in chronic scar tissue (typically burns), with long latency (>25y) following exposure.
- **Infective**, e.g. HSV, TB.
- Traumatic and artefactual causes.

Leg ulcer clinics emphasize the value of a multidisciplinary approach.

Cysts, sinuses, and fistulae

Cysts

A cyst is a collection of fluid in a sac lined by endothelium or epithelium, which usually secretes the fluid.

- True cysts are lined by endo- or epithelium.
- False cysts are the result of exudation or degeneration, e.g. pseudocyst of pancreas, cystic degeneration in a tumour.

Classification

Congenital

- Sequestration dermoid. Due to displacement of epithelium along embryonic fissures during closure, e.g. skin. Sites include outer and inner borders of the orbit, midline of the body, and anterior triangle of the neck (branchial cyst; cf. implantation dermoid due to skin implantation from injury).
- Tubulo-dermoid/tubulo-embryonic. Abnormal budding of tubular structures, e.g. enteric cysts, post-anal dermoid, thyroglossal cyst.
- Dilatation of vestigial remnants. e.g., urachal, vitellointestinal, paradental and branchial cleft cysts, hydatid of Morgagni, Rathke's pouch.

Acquired

- Retention cysts. Due to blocking of a glandular or excretory duct, e.g. sebaceous cyst (sweat gland), ranula (salivary gland), cysts of the pancreas, gall bladder, parotid, breast, epididymis, Bartholin's glands, hydronephrosis, hydrosalpinx.
- Distension cysts. Due to distension of closed cavities as a result of exudation or secretion, e.g. thyroid or ovarian cysts, hygroma (lymphatic cysts), hydrocele, ganglia, bursae (false cysts).
- Cystic tumours, e.g. ovarian cystadenoma or cystadenocarcinoma.
- Parasitic cysts, e.g. hydatid cysts (*Taenia echinococcus*).
- Pseudocysts. Due to necrosis of haemorrhage with liquefaction and encapsulation (e.g. necrotic tumours, cerebral softening) or coalescence of inflammatory fluid collections (e.g. pancreatic pseudocyst).

Clinical features

Spherical and fluctuant when palpated in two planes, with the fingers at right angles to each other. If tense contents, may produce pain. If the fluid is clear, the swelling will transilluminate. Ultrasound ± aspiration of contents are methods of determining whether a given swelling is cystic and may differentiate a cyst from a lipoma. May compress surrounding tissues. They are also subject to infection, torsion if on a pedicle, haemorrhage, and calcification.

Treatment

- Not all need treatment. Rationale for treatment is if symptomatic, concern over diagnosis, e.g. malignant potential, or for cosmetic reasons.
- Surgical options are: *excision*; *marsupialization* (de-roofing and suture of the lining to skin—for chronic or infected cysts); *drainage* (deep site; not done if concern over malignancy).

Sinuses/fistulae

- A sinus is a blind epithelial track lined by granulation tissue extending from a free surface into the tissues, e.g. pilonidal sinus.
- A fistula is an abnormal communication between two epithelial surfaces. It is lined by granulation tissue and colonized by bacteria, e.g. fistula-in-ano, pancreaticocutaneous, colovesical.

Causes

- Specific disease, e.g. fistula-in-ano from Crohn's.
- Abscess formation and spontaneous drainage, e.g. diverticular abscess discharging into the vagina (colovaginal fistula).
- Iatrogenic, e.g. anastomotic leak discharging via wound.
- Neoplastic.

Persistence of a fistula/sinus is due to the following

- Presence of foreign material, e.g. suture/bone.
- Inefficient/non-dependent drainage (e.g. long, narrow, tortuous track).
- Distal obstruction of the viscus of origin.
- Persistent discharge (absence of rest), e.g. faecal matter in fistula-in-ano.
- Epithelialization of the track.
- Continuing active infection, e.g. TB, actinomycosis.
- Chronic inflammation (e.g. Crohn's) or fibrosis.
- Malignancy in the track.
- Irradiation, e.g. rectovaginal fistula after radiotherapy for cervical cancer.
- Systemic factors inhibiting healing, e.g. malnutrition, drugs, ischaemia.

Investigations

- MRI and/or sinography/fistulogram and/or examination under anaesthesia (EUA) to establish anatomy.

Treatment

Principles of sinus treatment

- Excise/lay open. Remove granulations, infected/non-viable tissue, and foreign bodies. Biopsy sinus wall if concern over underlying pathology.

Principles of fistula treatment

- Treat any sepsis and fluid imbalances, and optimize nutritional state.
- Ensure good drainage to prevent fistula extension.
- Characterize the anatomy.
- Biopsy the fistula if concern over underlying diagnosis.
- Definitive surgical treatment requires excision of the organ of origin or closure of the site of origin; removal of chronic fistula track and surrounding inflamed tissue; and closure of 'recipient' organ if internal or drainage of external site if to skin.

Atherosclerosis

- **Atherosclerosis (ATH)**—atheromatous plaque causing vessel narrowing, mainly involves the intima.
- **Arteriosclerosis**—general term for 'hardening of arteries', mainly a disease of the media (↑ wall thickness and ↓ elasticity) → HTN.
- **Arteriolosclerosis**—proliferative change affecting small arteries.

Risk factors

- *Irreversible*. ↑ age, ♂, family history, DM.
- *Reversible*. Smoking, hypercholesterolaemia, obesity, HTN.

The arteriopath

A 48y-old man with type 1 DM presents with blurred vision over a 3-month period. He also complains of exertional breathlessness, with cramping pain in his calf muscles after walking 100 yards.

On examination: ↑ BP, mild pulmonary oedema, systolic bruit in the neck. Bloods show ↑ urea and ↑ Cr.

He has numerous risk factors for **ATH**—long-standing DM significantly increases the risk of ATH, compared to a non-diabetic of the same age. Visual symptoms may suggest diabetic retinopathy. Coronary ATH will cause chronic myocardial ischaemia → progressive heart failure (pulmonary oedema and shortness of breath). Femoropopliteal ATH will lead to claudication (lower limb ischaemia). The neck bruit raises the suspicion of carotid ATH and the risk of thromboembolic stroke. **Hyaline arteriolosclerosis** of renal vessels will lead to glomerular damage (↑ Cr/urea) and ↑ BP (via the renin–angiotensin system).

Pathological features of atherosclerosis

Spectrum of changes

Fatty streaks, intimal cushion lesions, and atheromatous fibrolipid plaques. A plaque comprises a **fibrous cap** (smooth muscle and collagen), a **cellular layer** (smooth muscle, macrophages, lymphocytes), and a **necrotic (lipid) core** (lipid, cholesterol, foam cells).

Plaque stability

A **stable plaque** will have a *thick fibrous cap* and be smooth muscle-rich with a *concentric* architecture—these will either remain static or cause progressive stenosis → occlusion (the clinical picture will be stable symptoms or chronic progressive ischaemia).

Features of a potentially **unstable plaque** are: *eccentric* shape, *lipid- and macrophage*-rich, with ↑ *inflammation*, endothelial damage (IFN-γ), and thinning of the fibrous cap (metalloproteinases, collagenases). Plaque rupture → acute thrombosis ± embolism. Clinically presents with an acute ischaemic event (e.g. acute MI/sudden cardiac death, thromboembolic stroke, acute ischaemic leg), depending on the involved vessel.

Thromboembolic disease

Thrombus

A thrombus is a friable mass of blood constituents formed in flowing blood (Virchow's triad).
A **blood** clot (aka haemostasis) occurs in stagnant blood, extravascularly in response to endothelial injury (or intravascularly post-mortem).

Aetiology
Virchow's triad describes the three mechanisms contributing to thrombosis (see Box 3.2). Not all three are needed—any one of them may result in thrombus formation in arteries or veins. Arterial thrombus is most commonly associated with atherosclerosis (acute plaque rupture), and venous thrombus with stasis.

Box 3.2 Virchow's triad

Endothelial disruption:
- Atheromatous plaque rupture, e.g. acute MI.
- Thrombophlebitis, e.g. DVT.
- Trauma, e.g. from pressure, surgery, fractures.

Blood flow:
- Stasis, e.g. immobilization, surgery, low CO states.
- Turbulence, e.g. post-stenotic, atrial fibrillation.

Blood constituents:
- Malignancy.
- Age.
- Smoking.
- DIC, HITT.
- Pregnancy, OCP.

Thrombus formation
Wherever a thrombus forms, the principal mechanisms are similar.

The initial trigger is one or more of Virchow's triad → fibrin deposition on vessel wall and formation of platelet layer → red cells trapped in fibrin meshwork on top of platelet layer. Classically, in venous thrombus, alternating patterns of white platelets and red blood cells appear as lines of Zahn.

- Thrombophlebitis is inflammation of veins 2° to thrombus.
- Phlebothrombosis is thrombus formation 2° to phlebitis.
- Phlegmasia alba dolens (white painful leg) occurs after slow thrombosis formation in the iliofemoral veins and is a chronic condition.
- Phlegmasia cerulea dolens (blue painful leg) is due to acute massive iliofemoral venous thrombosis causing venous ischaemia (↑ venous pressure impedes capillary perfusion)— ❗it is a surgical emergency.
- Thrombophlebitis migrans are transient thromboses in previously healthy veins anywhere in the body, suggestive of visceral malignancy (called Trousseau's sign).

Embolism

An embolus is a mobile mass of material in the vascular system capable of blocking a vessel lumen. An arterial embolus will cause end-organ ischaemia (e.g. acute lower limb ischaemia, stroke, MI); a venous embolus will tend to cause PE.

Types of embolus

- *Thrombus—arterial*: mostly from the LV/LA (usually in the context of AF); also from ulcerated aortic plaques, aortic aneurysm, and cardiac valves. *Venous*: from DVT. ▶*For both*: consider underlying malignancy especially if unprovoked or no clear aetiology.
- *Non-thrombotic causes* include: *air embolus*; injection or entraining of air (e.g. coming off cardiopulmonary bypass), decompression sickness; *fat* from long bone fractures, severe burns, extensive soft tissue trauma; *amniotic fluid*; *septic emboli,* e.g. vegetations from endocarditis—tend to cause localized septic infarcts at site of embolization; *tumour*; *foreign bodies*; IV drug use, medically inserted catheters.

Most arterial emboli (>80%) arise from intracardiac thrombi (LV or LA), usually from AF. Most (70%) travel to the lower limbs.

Ischaemia and infarction

Ischaemia is tissue effect due to insufficient O_2 delivery.
Infarction is tissue death due to insufficient O_2 delivery.

Ischaemia results from oxygen supply–demand mismatch caused by

- Vascular narrowing (ATH, thrombus, embolus, spasm).
- Global hypoperfusion (shock, cardiopulmonary bypass).
- Hypoxaemia (anaemia, hypoxia).
- Vascular compression (ventricular distension, venous occlusion).
- ↑ O_2 demand (exercise, pregnancy, hyperthyroidism).

Pathological features of an infarct

The shape of the infarct depends on the territory and perfusion of the occluded vessel.

- Seconds. Change from aerobic to anaerobic metabolism.
- Minutes. ↓ contractility of muscle; cells and mitochondria swell.
- Hours. Myocyte death; coagulation necrosis; pale, oedematous muscle.
- Days. Inflammatory exudates with polymorphonuclear leucocytes; then fibroblast infiltration with scar formation; macroscopically, the infarcted area appears yellow and rubbery with a haemorrhagic border.
- Weeks. Neovascularization.
- Months. Scar maturation—tough, white contracted area.

Gangrene and capillary ischaemia

Gangrene is ischaemic tissue necrosis with dessication (dry gangrene) or putrefaction (wet gangrene).

Gangrene

Aetiology

- ATH or thromboembolic disease.
- Extrinsic vascular compression.

Clinical appearances

Dry gangrene

The affected limb, digit, or organ is black (because of breakdown of Hb), dry, and shrivelled. Dry gangrene shows little or no tendency to spread—a zone of demarcation appears between the dead and viable tissue and separation begins to take place by aseptic ulceration.

Wet gangrene (= infected ❗*)*

There is infection (putrefaction). The skin and superficial tissues become blistered. There is a broad zone of ulceration which separates it from normal tissue. Proximal spread is a feature, leading to septicaemia and death. ▶▶ Requires emergency admission, IV Abx and surgical debridement.

Gas gangrene

Gangrene complicated by infection with gas-producing anaerobic bacteria, e.g. *Clostridium perfringens* (➲ The Clostridia, p. 213; ➲ Necrotizing fasciitis p. 229).

Principles of treatment

- Systemic treatment: O_2, fluids, broad-spectrum IV antibiotics (according to microbiological advice), pain relief.
- For dry gangrene involving non-vital organs only: conservative treatment—allow affected areas to mummify and spontaneously separate.
- For wet gangrene in the septic patient: radical surgical excision of all non-viable tissue or appropriate amputation.
- Revascularization: consider revascularization options (the timing of this will depend on the state of the patient—in the case of wet gangrene with sepsis, surgical debridement is followed by revascularization, and further debridement/resection, as appropriate.
- For unresectable cases (e.g. retroperitoneal gangrene, very extensive intestinal gangrene) or the frail/unfit patient: palliative care.

Capillary ischaemia

This is ischaemia mediated by injury to capillaries.

- *Frostbite*. Exposure to cold with freezing results in fixed capillary contraction, ischaemia, and infarction.
- *Trenchfoot*. Exposure to cold without freezing results in capillary contraction followed by fixed dilation.
- *DIC* (➲ Disseminated intravascular coagulation (DIC), p. 146).
- Cryoglobulinaemia, sickle cell, parasites.

Neoplasia

- **Neoplasia**. Uncontrolled clonal proliferation of cells (can be benign or malignant).
- **Metaplasia**. Reversible transformation of one type of terminally differentiated cell into another cell type. It is an adaptive response to environmental stress, e.g. ciliated to squamous cells in the respiratory epithelium in smokers, change from oesophageal squamous to columnar cells in gastro-oesophageal reflux disease (GORD) or Barrett's oesophagus), myositis ossificans—bone formation in injured muscle post-trauma.
- **Dysplasia**. A potentially premalignant condition characterized by loss of uniformity of cell size and shape (pleomorphism); loss of tissue orientation; nuclear changes (↑ nuclear:cytoplasm ratio).
- **Carcinoma in situ (pre-invasive)**. Irreversible severe dysplasia involving the entire epithelial thickness, but not penetrating the basement membrane.
- **Invasive carcinoma**. Invasion of basement membrane (metalloproteinases, e.g. collagenase, hydrolase) and E-cadherin inactivation lead to loss of cell–cell contacts.
- **Metastasis**. Spread to distant organ.

Benign versus malignant tumour characteristics

Benign Slow-growing, usually encapsulated, do not metastasize. Do not recur if completely excised, rarely endanger life. Effects are due to size and site. Histology: well differentiated (resemble tissue of origin), low mitotic rate, no necrosis.

Malignant These expand and infiltrate locally. Encapsulation is rare. Metastasize to other organs via blood, lymphatics, or body spaces; endanger life if untreated. ↓ apoptosis, ↑ telomerase activity. Histology: varying degrees of differentiation from tissue of origin, pleomorphic (variable cell shapes), high mitotic rate.

►The hallmarks of malignant transformation on a molecular level are: evasion of apoptosis, growth signal self-sufficiency and insensitivity to anti-growth signals, Warburg effect (shift of glucose metabolism away from mitochondria towards glycolysis), sustained angiogenesis, and limitless replicative potential.

Tumour nomenclature

- Based on tissue of origin and whether benign or malignant. Carcinoma suggests epithelial origin; sarcoma suggests mesenchymal origin. (See Table 3.1.)

Table 3.1 Tumour nomenclature

Tissue of origin	Benign	Malignant
Epithelium	Adenoma (glandular) Papilloma	Adenocarcinoma Papillary carcinoma
Connective tissue (mesenchymal)		-sarcoma
Fibrous tissue	Fibroma	Fibrosarcoma
Bone	Osteoma	Osteosarcoma
Fat	Lipoma	Liposarcoma
Cartilage	Chondroma	Chondrosarcoma
Blood vessels	Haemangioma	Angiosarcoma
Smooth muscle	Leiomyoma	Leiomyosarcoma
Striated muscle	Rhabdomyoma	Rhabdomyosarcoma
Haemopoietic (blood cells)		Leukaemias, Hodgkin's disease, multiple myeloma, lymphosarcoma, reticulosarcoma
Melanocytes	Naevus/mole	Melanoma
Mixed origins	Fibroadenoma	Nephroblastoma*, teratoma (all three germ layers), choriocarcinoma
Developmental blastomas		Neuroblastoma (adrenal medulla), nephroblastoma* (kidney), retinoblastoma (eye)

* Nephroblastoma is also known as Wilms' tumour. ➲ Solid tumours of childhood p. 548

Grading and staging

- **Grading** is the process of assessing the degree of differentiation of a malignant tumour.
- **Staging** is the process of assessing the extent of local and systemic spread of a tumour.

- **Grading** is the process of assessing the degree of differentiation of a malignant tumour.
- **Staging** is the process of assessing the extent of local and systemic spread of a tumour.

The objectives of staging and grading a tumour are:

- To plan appropriate (treatment) for the individual patient.
- To give an estimate of the prognosis.
- To compare similar cases when assessing outcomes or designing clinical trials.

Grade

- Reflects the degree of cellular differentiation (how closely the tumour cells resemble the tissue of origin) and mitotic activity.
- Based on histology.
- Low-grade ('well-differentiated') lesions are usually less aggressive. High-grade ('poorly differentiated', 'undifferentiated', or 'anaplastic'—tissue bearing no resemblance to tissue of origin) lesions are usually more aggressive.

Stage

- Degree of spread. Generally has better prognostic value than grade.
- **TNM classification**. Each TNM factor has independent prognostic value. Prefixed by mode of assessment: p(athology), c(linical), r(adiological), e.g. pT3N2Mx. (The specific TNM system is different for each type of cancer; it is not used for leukaemia, lymphomas, or myeloma.) Other staging systems include the **AJCC** (American Joint Committee on Cancer) staging system (I–IV: I, confined to organ, no nodal involvement; II–III, extension outside organ ± nodal involvement; IV, metastasis) and organ-specific scoring systems (e.g. **Duke's** score for colorectal cancer, **Gleason** score for prostate cancer).

- **T** = **T**umour size/invasiveness (Tx: unknown; T0: 1° tumour not evident; T1–4: degree of local invasion).
- **N** = **N**odal involvement (Nx: unknown; N0: no nodes involved; N1–3: number of involved nodes).
- **M** = **M**etastases (Mx: unknown; M0: no mets; M1: confirmed mets)

Other indicators of stage

- Depth of invasion (e.g. Breslow thickness in malignant melanoma).
- Tumour type (e.g. small cell versus non-small cell lung cancer).

Carcinogenesis

Carcinogenesis is the process that results in malignant neoplasm formation. Usually more than one carcinogen is necessary to produce a tumour, a process which may occur in several steps—"**multistep hypothesis"**.

- *Initiators* produce a permanent change in the cells but do not themselves cause cancer, e.g. ionizing radiation—this change may be in the form of a genetic mutation.
- *Promoters* stimulate clonal proliferation of initiated cells, e.g. dietary factors and hormones—they are not mutagenic.
- *Latency* is the time between exposure to carcinogen and clinically apparent tumour.

Genetic abnormalities in tumours

Two genetic mechanisms of carcinogenesis are proposed:

- *Oncogenes*. Enhanced expression of stimulatory dominant genes. The proteins produced (oncoproteins) can be produced in abnormal quantities or be abnormally active forms (see Table 3.2).
- *Tumour suppressor genes*. Inactivation of recessive inhibitory genes (see Table 3.3).

Table 3.2 Oncogenes—gain-of-function mutation in proto-oncogene (normal) to oncogene → ↑ cancer risk

Gene (gene product)	Associated neoplasm
ALK (receptor tyrosine kinase)	Adenocarcinoma of the lung
BCR–ABL (tyrosine kinase)	CML, ALL
BCL-2 (anti-apoptotic molecule)	B-cell lymphomas (follicular and diffuse)
BRAF (Ser–Thr kinase)	Melanoma, non-Hodgkin's lymphoma, papillary thyroid cancer
c-KIT (cytokine receptor)	GI stromal tumour (GIST)
c-MYC (transcription factor)	Burkitt's lymphoma
HER2/neu (*c-erbB2*) (receptor tyrosine kinase)	Breast and gastric carcinomas
JAK2 (tyrosine kinase)	Chronic myeloproliferative disorders
KRAS (GTPase)	Colon, lung, pancreatic cancer
MYCL1 (transcription factor)	Lung cancer
N-myc (*MYCN*) (transcription factor)	Neuroblastoma
RET (receptor tyrosine kinase)	Multiple endocrine neoplasia type 2A (MEN2A) and 2B, papillary thyroid cancer

Table 3.3 Tumour suppressor genes—loss of function → ↑ cancer risk

Gene (gene product)	Associated neoplasm
APC (inhibitor/negative regulator of β-catenin/WNT pathway)	Colorectal cancer (familial adenomatous polyposis (FAP))
BRCA1/*BRCA2* (DNA repair protein)	Breast, ovarian, and pancreatic cancer
CDKN2A (p16, G1IS phase inhibitor)	Melanoma, pancreatic cancer
DCC (Deleted in Colon Cancer)	Colon cancer
SMAD4 (*DPC4*) (Deleted in Pancreatic Cancer)	Pancreatic cancer
MEN1 (menin)	MEN1 (phaeochromocytoma, parathyroid, medullary thyroid cancer)
NF1 (neurofibromin) (Ras GTPase-activating protein)	Neurofibromatosis type 1
NF2 (Merlin (schwannomin) protein))	Neurofibromatosis type 2
PTEN (inhibits P13k/AKT pathway)	Breast, prostate, endometrial cancer
Rb (inhibits E2F; blocks G1IS)	Retinoblastoma, osteosarcoma
TP53 (p53)	(Most human cancers) and Li Fraumeni syndrome (syndrome of multiple malignancies at a young age, aka 'SBLA' syndrome—Sarcoma, Breast, Leukaemia, Adrenal)
TSC1 (hamartin protein) *TSC2* (tuberin protein)	Tuberous sclerosis
VHL (inhibits hypoxia inducible factor 1a)	von Hippel–Lindau syndrome
WT1 (transcription factor that regulates urogenital development)	Wilms' tumour (nephroblastoma)

Table 3.4 Environmental carcinogens

Agent	Type of cancer
Chemicals	
Cigarette smoke	Lung cancer, laryngeal squamous cell carcinoma (SCC), oesophageal cancer, renal cell carcinoma, pancreatic adenocarcinoma, bladder transitional cell carcinoma (TCC), cervical SCC
Ethanol	Oesophageal SCC, hepatocellular cancer
Asbestos (old roofing, shipyards)	Lung cancer > mesothelioma
Aflatoxins (from Aspergillus and other fungi associated with stored grain and nuts)	Hepatocellular cancer
Polyaromatic hydrocarbons	Lung cancer, skin cancers
Aromatic amines (rubber and dyes, cigarettes)	Bladder cancer
Alkylating agents (chemotherapy)	Leukaemia
Nickel	Nasal and lung cancer
Microbes	
HIV	Kaposi's sarcoma, lymphoma (and others)
Epstein–Barr virus (EBV)	Burkitt's lymphoma, nasopharyngeal cancer
Hepatitis B/hepatitis C	Hepatocellular cancer
Human papillomavirus (HPV) types 16 and 18	Cervical cancer (also penile/anal cancer)
Helicobacter pylori	Gastric adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma
HTLV	Adult T-cell leukaemia/lymphoma
Liver fluke (*Clonorchis sinensis*)	Cholangiocarcinoma
Schistosoma haematobium	Bladder cancer (squamous cell)
Radiation	
UV light (UVB > UVA)	Malignant melanoma, BCC
Ionizing radiation	Breast, bone, papillary thyroid cancer
Biological agents	
Hormones, e.g. oestrogens	Breast and endometrial cancer
Transplacental exposure	
Diethylstilbestrol	Vaginal adenocarcinoma

Tumours

Commonest tumours epidemiologically

The commonest cancers overall are probably non-melanoma skin cancers (BCC and SCC), but exact numbers are not known. After this, the commonest cancer types are as follows.

♀		♂
Breast	>	Prostate
	Lung	
	Colorectal	

The second and third commonest cancers for both sexes are lung and colorectal cancer.

Common tumours in surgical practice

These are covered in other chapters as follows:

- Colorectal cancer and familial polyposis syndromes (➔ Colorectal cancer, pp. 500–2).
- Breast cancer (➔ Breast cancer, pp. 312–15).
- Thyroid cancer (➔ Endocrine surgery, pp. 327–63).
- Parotid tumours (➔ Salivary gland tumours, pp. 302–3).
- Pancreatic cancer (➔ Pancreatic cancer, pp. 420–1).
- Prostate cancer (➔ Adenocarcinoma of the prostate, pp. 478–9, pp. 478–9).
- Renal cell carcinoma (➔ Adenocarcinoma of the kidney, pp. 474–5).
- Testicular tumours (➔ Testicular tumours, pp. 482–3).
- Bone tumours (➔ Bone tumours, pp. 684–7).

Other tumours

Hamartoma A benign tumour comprising an abnormal amount and arrangement of normal tissue. Not malignant and does not metastasize. The commonest site is the lung where they comprise the majority of benign lung lesions. *Peutz–Jeghers syndrome* is an autosomal dominant (AD) inherited condition characterized by multiple GI tract hamartomas.

Carcinoid tumours Tumours of endocrine cell origin. Primarily found in the GI tract and lung. Of the GI tract carcinoids, the **appendix** is the commonest site (followed by the small bowel, colon, and stomach). They have a good prognosis if treated. They rarely present with carcinoid syndrome (1%, compared with 20% if metastatic).

GI lymphoma Forty per cent of lymphomas occur outside the lymphoreticular system. Of these, the commonest site is the GI tract (mainly small bowel). 1° GI lymphoma does not involve the reticuloendothelial system. The main risk factors are: chronic *H. pylori* gastritis, chronic sprue-like syndromes, Mediterranean, and immunosuppression (HIV, transplant).

Sarcoma Rare tumours of mesenchymal origin (fat, muscle, bone, cartilage, etc.). *Leiomyosarcoma*: the commonest, arising from smooth muscle (e.g. uterine, stomach, small bowel); early surgical resection is the treatment of choice, as they do not respond to chemotherapy/radiotherapy well. *Liposarcoma*: large tumours affecting adults in 40–60s, arising in retroperitoneum or proximal extremities; treated mainly with surgery. Others include *fibroblastic* sarcomas, *GISTs*, and *rhabdomyosarcoma* (from skeletal muscle); occur in all age groups; a significant proportion affect younger adults. Most high-grade bone sarcomas, including *Ewing's sarcoma* and *osteosarcoma* are commoner in children and young adults.

Metastases

- Most carcinomas spread via the lymphatics.
- Follicular thyroid carcinoma, choriocarcinoma, renal cell carcinoma, hepatocellular cancer, and most sarcomas spread haematogenously.
- The commonest sites for metastases are: lung, liver, brain, and bones.

Site of metastasis	Commonest associated primary tumours
Lung	Breast, colorectal, kidney, bladder, prostate Head and neck, sarcoma, Wilms'
Liver	Colorectal >> gastric > pancreatic
Bone	Prostate, breast > Kidney, thyroid, lung
Brain	Lung > Breast > Melanoma, colorectal, kidney

Tumour markers

Key facts

- Complex molecules, most produced by normal cells in small amounts, but which may be produced in ↑ amounts by tumour cells due to changes in cellular function.

Testing

Used for screening and diagnosis, monitoring response to treatment, or detecting recurrence. Changes in levels may occur due to extraneous factors, e.g. inflammation, trauma/surgery, renal or hepatic impairment.

Serum tumour markers

Marker	Associated neoplasm
Alkaline phosphatase (ALP)	Metastasis to bone or liver Paget's disease of the bone Seminoma (placental ALP)
α-fetoprotein	Hepatocellular cancer, endodermal sinus (yolk sac) tumour, mixed germ cell tumour, ataxia telangiectasia, neural tube defects (*mnemonic*: 'HE-MAN')
β-HCG	Hydatidiform mole, choriocarcinomas (gestational trophoblastic disease), testicular cancer, mixed germ cell tumour
CA15-3/CA 27-29	Breast cancer
CA 19-9	Pancreatic adenocarcinoma
CA 125	Ovarian cancer
Calcitonin	Medullary thyroid cancer (and in MEN2A/2B)
Carcinoembryonic antigen (CEA)	*Mainly*: colorectal and pancreatic cancer *Minor associations*: gastric, breast, medullary thyroid cancer (*very non-specific marker*)
Chromogranin	Neuroendocrine tumours
Lactate dehydrogenase (LDH)	Testicular germ cell tumours, ovarian dysgerminoma, other cancers
Prostate-specific antigen (PSA)	Prostate cancer—*also elevated in benign prostatic hyperplasia (BPH) and prostatitis (moderately); surveillance marker for recurrence after prostatectomy*

Screening

Screening is testing any population for a disease.

The aim is reduction in morbidity and mortality by early detection.

Requirements for successful screening

A screening test must be:

- Sensitive; specific; safe; inexpensive; acceptable.

The population screened must be:

- Easily identified and contactable; compliant.

The disease screened must be:

- Detectable in a treatable, premalignant form or earlier stage.
- Preventable or more amenable to successful or curative treatment; a sufficient burden on the population to justify the cost of screening; chronic or of suitable evolution for sporadic testing to detect it.

Disadvantages of screening

- Cost (time and resources).
- The benefit may be small.
- False positive tests may be physically or psychologically detrimental.

Examples of screening programmes

Colorectal cancer (16 000 deaths/y)

- *Currently screened population (UK)*: men and women 60–74y.
- *Methods*: guaiac-based faecal occult blood test (gFOBT) (<50% sensitivity, <90% specificity)—followed up by colonoscopy for positive results; flexible sigmoidoscopy offered in some regions at age 55y (sensitivity 80%, specificity near 100%).
- Other methods not routinely used for population screening are: faecal immunochemical test (FIT) (80% sensitivity, 95% specificity) or stool DNA testing (current assays less accurate than FIT). Colonoscopy is the gold standard test, used to follow up on 'positive' faecal occult blood (FOB) results and as a 1° diagnostic tool (sensitivity and specificity near 100%).
- *Evidence*: the lifetime risk of colorectal cancer is about 1 in 20. Several RCTs have shown that the incidence and mortality from colorectal cancer can be substantially reduced by screening. Colorectal cancer is suited to screening because it has a detectable premalignant phase and is detectable at an earlier and potentially highly treatable stage.

Breast cancer (14 000 deaths/y)

- *Currently screened population (UK)*: all women between 50 and 70y (covers peak ages of incidence and excludes low-risk younger women—prevents 'psychological morbidity of screening the well').
- *Method*: Two-view (lateral and oblique) mammography of both breasts. Suspicious or malignant-looking lesions invited for clinical assessment by standard triple assessment (➲ Evaluation of breast disease, p. 58).

- *Evidence*: a meta-analysis of 13 breast cancer screening trials concluded that screening mammography significantly reduced breast cancer mortality in women aged 50–74y. A *BMJ* analysis concluded that: for every 1000 women screened over 10y, around 200 are recalled because of an abnormal result and of these: around 60 will have at least one biopsy; about 15 will have invasive cancer, and five will have DCIS. Between 0.5 and 3 fewer deaths from breast cancer occur over 10y per 1000 women screened.

  Ten per cent of invasive carcinoma is not radiologically detectable.
  Risk of a false positive screen is ~25% over 10y.
  Studies suggest up to 30% reduction in mortality from screen-detected early breast cancer.

Abdominal aortic aneurysm (4000 deaths/y)

- *Currently screened population (UK)*: all ♂ >65y.
- *Method*: GP-/community-based ultrasound.
- *Evidence*: the UK Multicentre Aneurysm Screening Study (MASS) of 68 000 men aged >65y showed that the group offered screening had 42% lower aneurysm-related deaths. It was estimated that 216 men need to be screened to save one death over the following 13y.

Cervical cancer (1500 deaths/y)

- *Currently screened population (UK)*: all women 25–64y.
- *Method*: cervical smear test—1° HPV testing from 2019.
- *Evidence*: since the mid-1980s, the incidence of, and mortality from, cervical cancer in women <70y in England and Wales has fallen. Screening is thought to be the most likely explanation. A *BMJ* analysis concluded that: in the NHS cervical screening programme, 1000 women need to be screened for 35y to prevent one death; 55 have an abnormal biopsy result; two have carcinoma, and the rest have dysplasia. At least one woman dies within the 35y despite being screened.

Prostate cancer (9000 deaths/y)

- Currently no population-wide screening programme.
- A third of men over 50y have evidence of prostate cancer at post-mortem, but <1% of these have clinically active disease.
- Screening is controversial because:
  - PSA, rectal examination, and transrectal ultrasound (TRUS) have low specificity and sensitivity alone or in combination.
  - Treatment of prostate cancer is controversial
  - No randomized trial has shown a survival benefit in screened populations—screening may cause more harm than good.
  - Screening can be carried out on request despite the evidence above.

Surgically important bacteria

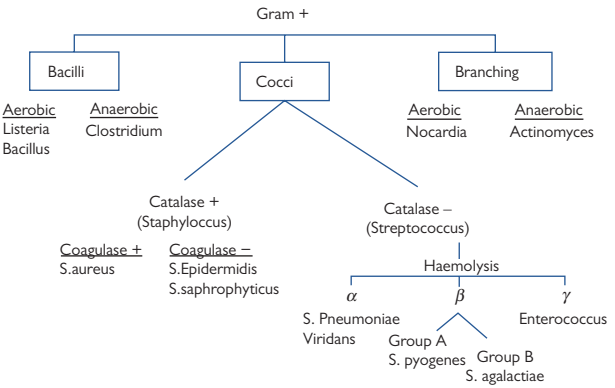

Fig. 3.2 Gram-positive bacteria.

Gram-positive bacteria

Staphylococcus aureus

Virulence factors: ▶ Protein A, exotoxins (toxic shock syndrome toxin (TSST), enterotoxin).

Diseases: (a) by inflammation— ▶commonest organism in soft tissue infections, abscesses, osteomyelitis, and septic arthritis, and in **SSIs** and **nosocomial infections** in general (⚠ consider MRSA associated with prolonged hospitalization). Pneumonia (often following 'flu' virus) and especially in hospital-acquired setting. Endocarditis. *(b)Toxin-mediated*—toxic shock syndrome (associated with vaginal tampon or nasal packing), scalded skin syndrome, *rapid-onset* food poisoning (enterotoxin → non-bloody diarrhoea within 1h of ingestion).

Staphylococcus epidermidis

Virulence factors: ▶adherent biofilm.

Diseases: IV line and prosthesis infections (especially in the immunocompromised), common blood culture contaminant.

Staphylococcus saprophyticus

Normal flora of ♀ genital tract/perineum.

Common cause of uncomplicated UTI in young women.

Streptococcus pneumoniae (Pneumococcus)

▶Encapsulated organism.

Diseases: Post-splenectomy sepsis (and sickle cell disease). 'MOPS'—Meningitis, Otitis media (children), Pneumonia Sinusitis.

Viridans streptococci

Oropharyngeal commensal.

Diseases: Streptococcus mitis/Streptococcus mutans cause dental caries; *Streptococcus sanguinis* causes SBE.

Streptococcus pyogenes (group A Streptococcus)

►*Virulence factors: Hyaluronic acid capsule and M-protein; erythrogenic toxin.*

Diseases:

Pyogenic (purulent): pharyngitis, cellulitis, impetigo.

Toxin-mediated: ► scarlet fever, toxic shock-like syndrome, necrotizing fasciitis.

Immune-mediated: rheumatic fever, glomerulonephritis.

Diagnosed by anti-streptolysin O (ASO) or anti-DNase B antibodies.

Streptococcus agalactiae (group B Streptococcus)

Diseases: ►Post-partum infection and neonatal sepsis (pregnant women screened at 35–37 weeks with rectal and vaginal swabs).

Streptococcus bovis

GI tract commensal.

Can cause bacteraemia → endocarditis; associated with colon cancer.

Enterococci (Enterococcus faecalis and Enterococcus faecium)

UTI, biliary sepsis, endocarditis (following GI/genitourinary (GU) procedures); **Vancomycin-resistant enterococci (VRE)** (vancomycin-resistant form) is an important cause of nosocomial infections.

Bacillus cereus

Acute non-bloody, watery diarrhoea and pain—associated with rice/pasta ('reheated rice syndrome').

The Clostridia (spore-forming anaerobes)

Clostridium tetani ► Tetanospasmin toxin causes tetanus (spastic paralysis, lockjaw, risus sardonicus, opisthotonus). Prevention: tetanus vaccine. Treatment: antitoxin, antibiotics, diazepam (for spasms).

Clostridium botulinum ►Botulinum toxin inhibits ACh release → flaccid paralysis. It is also used therapeutically.

Clostridium perfringens Spores survive in undercooked food or in soil. ► Common cause of food poisoning in the UK/USA via enterotoxin. ❗α-toxin → **gas gangrene** (myonecrosis—presents with crepitus and gas on X-ray/CT and rapid onset severe systemic sepsis).

Clostridium difficile ► Toxins A and B—both cause diarrhoea → **pseudomembranous colitis** (associated with antibiotic use, especially clindamycin, cephalosporins, and quinolones, e.g. ciprofloxacin). Treatment: Metronidazole or vancomycin.

Nocardia and actinomycosis

Both form branching filaments, like fungi. Nocardia is aerobic and acid-fast (can mimic TB, but purified protein derivative (PPD) negative). In the immunocompetent, causes post-traumatic skin infection; in the immunocompromised, causes pneumonia. Can involve the CNS.

Actinomyces is anaerobic and not acid-fast. Causes maxillofacial infections (oral/facial abscesses, etc., associated with maxillofacial trauma).

Mycobacteria Are all acid-fast organisms.

Mycobacterium tuberculosis

► **1° TB**: **Ghon complex** is hilar lymphadenopathy + Ghon focus (mid-/lower lobe lung lesion).

- In the immunocompromised (<10%) → progressive 1° TB with lung disease and haematogenous spread (→ miliary TB).
- In most cases (90%) → healing, calcification with later reactivation (→ 2° TB).

▶ 2° **TB**: characterized by cavitating lesion usually in upper lobe (caseating granulomas with central necrosis, Langerhans giant cells).

Gram-negative bacteria

(See Fig. 3.3.)

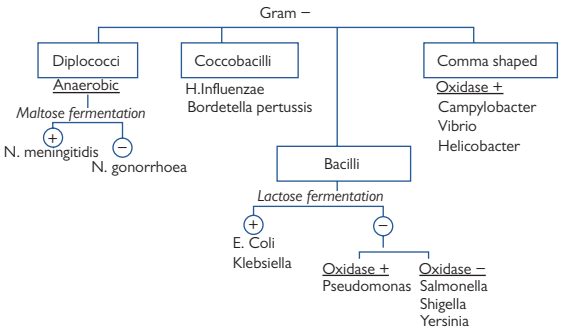

Fig. 3.3 Gram-negative bacteria.

Neisseria gonorrhoeae Sexual or perinatal transmission.

Diseases: Sexually transmitted disease (STD): gonorrhoea (urethritis), pelvic inflammatory disease (PID), Fitz-Hugh–Curtis syndrome, septic arthritis. Perinatal: neonatal conjunctivitis (cf. ➲ *Chlamydia* spp., below, p. 216).

Neisseria meningitidis (meningococcus) Respiratory or oral transmission.

Diseases: Meningitis, meningococcaemia (septicaemia with DIC—small vessel thrombosis and haemorrhages) → petechial rash, gangrene of extremities, Waterhouse–Friderichsen syndrome.

Rx: Ceftriaxone (also consider cover for *Chlamydia* in urethritis/PID).

Haemophilus influenzae Aerosol transmission.

*Diseases: E*piglottitis, meningitis, otitis media, pneumonia (note: it does not cause 'flu, which is caused by the influenza virus).

Pseudomonas aeruginosa

▶ **Blue-green** pigment.

Virulence factors: phospholipase C, endo- and exotoxins.

Diseases: pneumonia, UTI, osteomyelitis, otitis externa (swimmer's ear), skin and soft tissue infections (including wound infection in burns). Ecthyma gangrenosum (immune compromise). Associated with: immune-compromised states (e.g. DM and drug abuse) and nosocomial infections.

Salmonella and Shigella

▶ Both invade the GI tract via Peyer's patches (M-cells); produce endotoxins; *Salmonella* has flagella.

Shigella causes dysentery (bloody diarrhoea), non-typhi *Salmonella* cause gastroenteritis.

Salmonella typhi	***Other Salmonella spp.***
Comes from humans Causes **typhoid fever** (constipation with abdominal pain, rose spots on abdomen, fever); colonizes the gall bladder in carrier state	Commonly from **animals** (poultry and eggs, pets) Causes **gastroenteritis**, sometimes bloody

Yersinia enterocolitica. Acute diarrhoea, can mimic appendicitis (RIF pain from terminal ileitis or mesenteric adenitis).

Escherichia coli

▶ *Virulence factors*: fimbriae and P-pili (UTI and pyelonephritis); K-capsule (pneumonia, neonatal meningitis); lipopolysaccharide (LPS) endotoxin (septic shock).

E**I**EC (enteroinvasive)	Dysentery (similar to *Shigella*)
E**T**EC (enterotoxigenic)	**T**raveller's diarrhoea (watery), usually from developing countries
E**P**EC (enteropathogenic)	**P**aediatric'—common cause of infant diarrhoea, usually in developing world
E**H**EC (enterohaemorrhagic)	Common cause of food poisoning in the West (commonest serotype = O157:H7) **H**aemorrhagic (dysentery) **H**amburgers (often from undercooked meat or leafy vegetables) **H**aemolytic uraemic syndrome (caused by Shiga-like toxin)

Campylobacter

One of the commonest causes of bloody diarrhoea. From undercooked poultry or meat, unpasteurized milk, or animal contact. Antecedent to Guillain–Barré syndrome or reactive arthritis.

Vibrio cholerae From contaminated water or uncooked food (e.g. raw fish), developing countries. *Disease:* Profuse watery diarrhoea (enterotoxin).

Klebsiella pneumonia in the immune-compromised typically alcoholics and diabetics—'redcurrant jelly' sputum. Also causes: lung and liver abscess, nosocomial UTI.

Helicobacter pylori

▶ Colonizes gastric antrum; urease activity produces alkaline ammonia (NH_3) (allowing it to survive acidic environment) and is the basis for the urease breath test. *Diseases:* Gastritis and peptic ulcers (especially duodenal ulcers); ❗ risk factor for **gastric adenocarcinoma** and **MALT** lymphoma.Rx: 'Triple therapy': amoxicillin + clarithromycin + PPI.

Gardnerella vaginalis

▶ 'Clue cells' under the microscope. *Disease:* Bacterial vaginosis → Grey vaginal discharge with fishy smell.

Chlamydia spp. (including *C. trachomatis*, *C. pneumoniae*, *C. psittaci*) Obligate intracellular organisms.

Diseases: C. trachomatis: STI—urethritis/PID, neonatal conjunctivitis, Reiter's syndrome (reactive arthritis).

C. pneumoniae, *C. psittaci*: atypical pneumonia.

Rx: azithromycin or doxycycline (in urethritis/PID; also consider cover for *Gonococcus*).

Antimicrobials

Mechanism	Examples
Cell wall synthesis: Peptidoglycan synthesis Peptidoglycan cross-linking	Glycopeptides (e.g. vancomycin) Penicillins Cephalosporins Carbapenems Monobactams (e.g.) Antipseudomonals (e.g. piperacillin)
Membrane integrity	Daptomycin
Folic acid (needed for DNA synthesis)	Sulfonamides (sulfamethoxazole) Trimethoprim
DNA integrity	Metronidazole
mRNA synthesis	Rifampicin
DNA gyrase	Fluoroquinolones (e.g. ciprofloxacin) Quinolones
Protein synthesis (ribosome function): 50S subunit 30S subunit	Macrolides (e.g. erythromycin, clarithromycin), streptogramins, chloramphenicol, clindamycin, linezolid Aminoglycosides (gentamicin, neomycin, amikacin, streptomycin), tetracycline, glycylcyclines

Viruses

Viral hepatitis

- Commonest liver disease worldwide.
- May cause acute hepatitis (fever, jaundice with ↑ ALT and AST) → liver failure OR chronic active hepatitis. (Some, via a chronic carrier state, predispose to hepatocellular carcinoma (HCC) (hepatitis B, C, and D) or cirrhosis (hepatitis C).

Table 3.5 Hepatitis viruses

	Hepatitis A and E	**Hepatitis B**	**Hepatitis C**
Type	Single-stranded RNA (ssRNA)	Double-stranded DNA (dsDNA)	ssRNA
Transmission	Faeco-oral **A:** shellfish, travellers, day care (children) **E:** waterborne	Parenteral (blood), sexual, perinatal	Mainly blood (IV drug use/ transfusion)
Incubation	Short (weeks)	Long (months)	Long
Typical clinical presentation	**A:** asymptomatic (usually) or acute hepatitis (commonest cause of jaundice in children and young adults) **E:** fulminant hepatitis in pregnant women (high mortality)	Acute prodrome, like serum sickness Full resolution in 90% of adults (worse prognosis in babies); carrier state common; may progress to HCC.[1]	Chronic stable hepatitis (in most), carrier state very common; may progress to HCC.[1] or cirrhosis
Pathology	**A:** hepatocyte swelling with '*Councilman bodies*' **E:** patchy necrosis	Eosinophilic ground-glass appearance	Lymphoid aggregates with focal macrovesicular steatosis
Risk of HCC[1]	No	Yes (and hepatitis D)	Yes
Diagnosis/ serological markers	**A:** anti-HAV IgM—acute; IgG—prior infection or vaccination, protective	(See Box 3.3)	

[1] HCC, hepatocellular carcinoma.

Hepatitis D (ssRNA virus) has a similar transmission, clinical course, and pathological features to hepatitis B virus (HBV). It is called a 'defective virus' as it requires the HBsAg coat to gain entry into hepatocytes. This may occur by superinfection (hepatitis D virus (HDV) after HBV), associated with a shorter incubation and worse prognosis, or co-infection, which has a longer incubation time and better prognosis.

Box 3.3 Hepatitis B serological markers

Antigens and viral constituents

- *HBsAg*—found on virus surface—indicates presence of hepatitis B (early phase—during incubation and acute disease).
- *HBeAg* (also HBV DNA and HBV DNA polymerase)—found inside the virus, secreted by infected hepatocytes into circulation, and indicates active viral replication and high infectivity.

Antibodies

- *Anti-HBc IgM*: acute/recent infection—gradually replaced by *IgG, which* indicates prior exposure or chronic infection.
- *Anti-HBe*—early recovery phase (indicates low infectivity).
- *Anti-HBs*—indicates immunity (vaccination or full recovery from infection). Detected late, few months after acute disease.

Human immunodeficiency virus

Epidemiology

- HIV affects 37 million people worldwide, with highest prevalence in Africa (10% of all cases are in South Africa). New diagnoses in the UK/USA in 2017 were due to: ♂ homosexual transmission (50–70% of new diagnoses), heterosexual transmission (25–50%), injecting drug users (5–10%), and perinatal (<2%).

Pathogenesis

Double-stranded RNA retrovirus transmitted by passage of infected body fluids from one person to another.

- Virus components: RNA and ▶ **reverse transcriptase** (allows synthesis of dsDNA from viral RNA, which incorporates into host genome); **capsid** proteins (e.g. p24); **envelope** proteins (e.g. gp120).
- The virus infects immune cells—macrophages (CCR5 receptor) and CD4 T-cells (CXCR4 receptor)—eventually causing widespread immune dysfunction with fall in CD4+ cell count → acquired immune deficiency syndrome (AIDS).

Clinical course and diagnosis

- There are four stages of untreated HIV infection (see Table 3.6), with AIDS developing 5–10y from initial infection. Median survival with untreated AIDS is 2y; treated is >20y.
- Prior to detectable HIV antibodies (seroconversion), there is a 3- to 4-week window of high infectivity (diagnosed by p24 antigen immunoassay or HIV nucleic acid amplification test (NAAT)); after seroconversion, anti-gp120 antibodies are detectable. Viral load is used to monitor response to treatment.

Table 3.6 HIV infection—stages

Acute • (Few weeks from infection) • CD4+ >500	• HIV seroconversion illness—flu-like symptoms • Acute ↓ CD4+ count within normal range • HIV RNA levels peak • Anti-envelope (gp120) antibody levels increase and plateau after this
Latent period • (Few years) • CD4+ >500	• Asymptomatic • Virus replicates in lymph nodes
Symptomatic stage • Moderate immunocompromise • CD4+ <500	• Skin and mucous membrane infections, e.g. oral thrush, anal/cervical SCC
Systemic immunocompromise • CD4+ <200	• 'AIDS-defining illnesses', e.g. *Pneumocystis*

Common diseases in HIV and AIDS-defining illnesses

- Certain organisms/infections are characteristic of HIV/AIDS (see Table 3.7). In addition, falling CD4+ count may manifest with reactivation of latent infections (e.g. TB, shingles, HSV) or disseminated, more aggressive forms of common bacterial/fungal infections.

Table 3.7 HIV and AIDS-defining diseases

Moderate immunocompromise CD4+ <500	
• Skin and mucous membranes	• Oral thrush—*Candida albicans* • Oral hairy leukoplakia—EBV • Kaposi's sarcoma—human herpesvirus 8 (HHV-8) • Anal or cervical SCC—HPV
AIDS-defining illnesses CD4+ <200	
GI tract	• *C. albicans*—oesophagitis • *Cryptosporidium*—chronic diarrhoea
Respiratory	• *Pneumocystis jirovecii*—pneumonia • *Aspergillus fumigatus*—haemoptysis and pleuritic pain • *Histoplasma capsulatum—pulmonary or disseminated histoplasmosis*
CNS	• JC virus—progressive multifocal leukoencephalopathy—(demyelination) • *Cryptococcus neoformans*—meningitis • *Toxoplasma gondii*—brain abscesses • HIV—dementia
Lymph nodes	• EBV—B-cell lymphoma

Skin	• *Bartonella hensellae*—bacillary angiomatosis (can occur in other organs)
• Generalized or multisystem	• CMV—retinitis, pneumonitis, oesophagitis, colitis, encephalitis • *Mycobacterium avium* complex—disseminated infection

Modes of occupational infections of health workers

- Direct percutaneous inoculation of infected blood (e.g. needle-stick injury, scalpel wounds).
- Entry of infection through minute skin abrasions after contact with spilled bodily fluids (e.g. blood, saliva, semen, urine, faeces).
- Entry of infection via mucosal surfaces after exposure to contaminated infectious bodily fluids (e.g. eye splashes in theatre, faeco-oral route).
- Transfer of infection by fomites (e.g. via contaminated equipment—prions transferred by neurosurgical equipment).

Precautions and post-exposure prophylaxis (PEP)

HIV

- The infection risk from a contaminated hollow needle is 0.3%.
- The risk from splashes on broken skin or mucous membranes is 0.1%.
- **PEP** reduces the risk of seroconversion by over 80% if started within 1h of exposure; PEP is continued for 4 weeks. Side effects include diarrhoea and nausea/vomiting.

HBV

- Transmission from a contaminated sharps injury is 30%.
- Hospital staff are routinely offered vaccination for hepatitis B:
  - Infectious carriers may not perform exposure-prone procedures.
  - Vaccination against hepatitis B is not compulsory. The alternatives are frequent testing to check infectivity or limited clinical practice.

HCV

Transmission from a contaminated sharps injury is 2–3%. Surgeons must be tested for hepatitis C and may not carry out exposure-prone procedures if hepatitis C +ve.

Universal precautions

These are designed to protect workers from exposure to diseases spread by blood and body fluids. ▶All patients are assumed to be infectious for blood-borne diseases, including HIV.

- Infectious body fluids (for viral transmission) include: blood; amniotic, synovial, pleural, peritoneal, and pericardial fluids; semen; vaginal secretions; and cerebrospinal fluid (CSF) (not faeces, sputum, urine, vomit, or saliva).

- Universal precautions include:
  - Protective clothing, e.g. gloves, gowns, masks, eye-shields.
  - Removing hazards, e.g. sharps bins, ventilation.
  - Work practice, e.g. handwashing, handling of sharps, disposal and transport of soiled goods, reducing unnecessary procedures.
  - Single-use disposable injection equipment.
  - Hospital policy for all sharps injuries: squeeze, wash, and report.
- Barrier nursing for *C. difficile*.
- Specific treatment and sterilization of non-disposable equipment.
- Additional/specific precautions for theatre staff:
  - Wearing plastic aprons in procedures with expected soiling with urine/faeces/ascites and minimizing unnecessary spillage.
  - Double-gloving to reduce the risk of skin exposure when gloves tear or reinforced gloves for procedures with a high risk of penetrating injury (e.g. fragmented fractures).

Other important viruses in surgery and their associations

Virus	Common Associations
HPV	Types 16 and 18: cervical cancer and cervical intra-epithelial neoplasia (CIN) Type 16: anal SCC Types 1, 2, 6, 11: warts
HHV-8	Kaposi's sarcoma in the immunocompromised (AIDS/transplant patients). Vascular proliferations presenting as dark red/purple plaques or nodules
CMV (HHV-5)	Immunocompetent patients: mononucleosis Immunocompromised patients: pneumonia in organ transplant recipients; oesophagitis, AIDS retinitis Congenital CMV ► Infected cells have 'owl eye' intranuclear inclusions. Monospot test –ve
EBV (HHV-4)	Mononucleosis ('kissing disease') Splenic rupture Lymphomas (endemic Burkitt's lymphoma) Post-transplantation lymphoproliferative disease Nasopharyngeal carcinoma *Monospot test +ve*

CMV, cytomegalovirus; EBV, Epstein–Barr virus; HPV, human papillomavirus; HHV, human herpesvirus.

There are nine types of HHV: HHV-1 and 2 = herpes simplex viruses 1 and 2, respectively; HHV-3 = varicella-zoster; HHV-4 = EBV; HHV-5 = CMV; HHV-6/7 cause roseola.

Surgical site infections

Definition

A wound infection occuring after an invasive surgical procedure.

Key facts

- Up to 20% of all nosocomial (health care-acquired) infections.
- The majority of SSIs are preventable.
- Most are caused by microorganisms from the patient's own flora intraoperatively. Post-operative infection from external sources is less common.
- Effects: quality of life, morbidity (including additional surgical procedures), and extended hospital/ITU stay and higher mortality; ↑ health care costs.

- Nosocomial infections are acquired in hospital.
- Community-acquired infections are acquired outside hospital.

Incidence/epidemiology

Incidence is at least 0.5–9.5% of all surgical procedures in the European Union (EU) and USA. They account for ~2% of deaths due to health care-associated infections (HAIs).[1, 2]

Approximate mean incidence in Europe and the USA by operation type are as follows (figures from European Centre for Disease Prevention and Control and Centers for Disease Control and Prevention):[1, 2]

- CABG: 3%.
- Laparoscopic cholecystectomy: 1.5% (open biliary surgery: 4%).
- Colorectal surgery, laparoscopic: 7% (open: 11%).
- Caesarean section: 2%.
- Hip/knee replacements: <1%.
- Spinal surgery (laminectomy): 1%.

Classification of wounds

- ***Clean***. Non-traumatic wounds with no break in surgical technique, no septic focus, and no viscus opened (e.g. hernia repair, CABG).
- ***Clean-contaminated***. Non-traumatic wounds with entry into a viscus, but minimal spillage (e.g. cholecystectomy, elective colon resection).
- ***Contaminated***. Traumatic wound or significant spillage from a viscus or acute inflammation (e.g. appendicectomy).
- ***Dirty***. Includes traumatic wounds from a dirty source or when significant bacterial contamination or release of pus is encountered, e.g. perforated appendicectomy, incision and drainage of an abscess.

Sources of surgical site infection

- The main source is normal body flora (see Table 3.8). Of these, the commonest organism is *S. aureus*. Consider if the wound is 'clean' (in which skin flora would be the main cause), 'clean-contaminated', etc.

Table 3.8 Normal body flora

Organism	Site
S.aureus, streptococci	Skin, oropharynx, nasopharynx
Enterococci, Gram-negative rods	Large intestine
Anaerobes	Skin, oropharynx, large bowel and terminal ileum, GU tract

- Other sources include:
  - Indirect contact: hands of health care workers, other patients, visitors, or contaminated surfaces.
  - Direct inoculation: surgeon or environmental flora through failure of aseptic technique, including contaminated instruments or dressings; colonization of indwelling drains, catheters, and IV lines; contaminated field (e.g. spillage of bowel content in laparotomy).
  - Airborne: Skin and clothing of staff, patients, and visitors; air flow in operating theatre or ward.
  - Haematogenous: IV and intra-arterial lines, sepsis at other anatomical sites, contaminated infusions, etc.

Common organisms in SSI

Operation type	Likely organism
Operations involving grafts, implants, or prostheses	S. *aureus*, coagulase negative *(coag. –ve) Staphylococcus*
Cardiac, vascular, neurosurgery, breast	S. *aureus*, coag –ve *Staphylococcus*
Orthopaedic and trauma	S. *aureus*, coag –ve *Staphylococcus* Gram –ve bacilli
Diabetic foot sepsis	*Staphylococcus* spp. *(*including MRSA*)*, streptococci, Gram –ve bacilli (including *Pseudomonas*), anaerobes
Thoracic (non-cardiac)	S. *aureus*, coag –ve *Staphylococcus* S. *pneumoniae*, Gram –ve bacilli
Appendicectomy, colorectal, biliary tract	Gram –ve bacilli, anaerobes
Upper GI (gastroduodenal)	Gram –ve bacilli, streptococci, oropharyngeal anaerobes
Head and neck (involving oropharyngeal mucosa)	S. *aureus*, streptococci, oropharyngeal anaerobes
Obstetric and gynaecological	Gram –ve bacilli, enterococci, group B *Streptococcus*, anaerobes
Urology	Gram –ve bacilli

Risk factors and antibiotic prophylaxis

(➲ Antibiotic prescribing in surgery, p. 42

Procedures to minimize risk of SSI[3]

Preoperative

- Smoking cessation*
- Preop showering*
- Hair removal (use electric clippers, not razors)**,[1]
- Nasal decontamination**
- Mechanical bowel preparation**
- Antibiotic prophylaxis

Intraoperative

- Skin prep—povidone-iodine or chlorhexidine*
- Wound dressing*

Post-operative

- Use of aseptic, non-touch technique for dressing changes*
- Sterile saline for wound cleaning up to 48h post-op*
- Patient can shower safely after 48h*
- Topical antimicrobials—not indicated for wound healing by 1° intention**
- Involve tissue viability nurse for complex wound healing by 2° intention

* Advisable routinely.

** Not routinely beneficial but may be used.

[1] Hair removal with razors is thought to increase the rate of SSI.

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Soft tissue and bone infections

- **Cellulitis** is the presence of actively dividing infectious bacteria within the skin.
- **Abscess** is a liquid collection of pus lined by granulation tissue and fibrosis (if chronic).
- **Lymphangitis** is the presence of actively dividing infectious bacteria in the lymphatic vessels of an area of the body.
- **Fasciitis** is infection or inflammation of connective tissue.
- **Myositis** is infection or inflammation of muscle.

Cellulitis

Pathological features

- Skin entry by pathogenic bacteria (scratch, ulcer, hair follicle).
- Usually **Gram +ve cocci** (e.g. *S. pyogenes* or *S. aureus*).
- Usually heals by resolution if treated promptly.
- Spread may result in lymphangitis; suppuration results in a furuncle (skin gland), carbuncle (upper dermis), or abscess (deep skin tissue).

Clinical features

- Warm, red (usually blanches with pressure), oedema (often pitting), and painful. ❗ Rapidly progressing cellulitis in a systemically unstable patient should raise the suspicion of deeper infection, e.g. necrotizing fasciitis (➲ Necrotizing fasciitis, below p. 229).

Treatment

- IV benzylpenicillin (1g qds) and flucloxacillin (500mg qds).

Abscess

Pathological features

- Collection of pus—leucocytes (live and dead), bacteria (dead and viable), and liquefied tissue products—'walled off' in deeper layers. Almost always *S. aureus*. May rupture ('pointing'), discharge into another organ (forming a fistula), or open onto another epithelial surface (sinus)). Incomplete treatment due to resistant organisms (mycobacteria) or poor treatment may lead to chronic abscesses; complete elimination of the organisms in a chronic abscess without drainage can lead to a 'sterile abscess'.

Diagnosis

- Deep abscesses are characterized by swinging fever, rigors, and high WCC and CRP. Untreated, they lead to catabolism, weight loss, and falling serum albumin. Ultrasound, CT, MRI, or isotope studies may be necessary to confirm the diagnosis.

Treatment

- **Drainage**, e.g. surgical incision and drainage ± debridement (perianal abscess), CT-guided drainage (intra-abdominal/pelvic abscess), closed surgical drainage (chest empyema).
- **IV antibiotics**—may require a course of several weeks, indicating for PICC line insertion.

Necrotizing fasciitis

- Deep tissue infection involving subcutaneous tissue and fascial layers. Can spread to underlying muscle (myositis).
- Usually from **anaerobes** or ***S. pyogenes*** in immunocompromised.
- Specific types: *Fournier's* (perineum) or *Meleney's* (post-operative abdominal wound).
- ❗ Diagnosis requires a high index of suspicion—look for the following 'red flags': rapid progression, systemically unwell patient (hypotension, ↑ lactate), pain out of proportion to examination findings, crepitus or soft tissue gas on X-ray/CT; bullae/purple discoloration of skin (also ➲ Necrotizing soft tissue infections, under Plastic surgery p. 731).

Gas gangrene (and myositis)

The bacterial infection has penetrated and destroyed muscle bundles. Gas gangrene is caused by *C. perfringens*. There is muscle oedema and serosanguinous exudate; exposed muscle is swollen and ranges from light pink to grey to deep green/black in appearance. Crepitation may be present. Viability of muscle can be tested by contractility to electrical stimulation with diathermy. Treatment is by aggressive excision of non-viable muscle (may require limb amputation if widespread).

Tetanus

This is a rare infection in the UK, but common in many parts of the world, with a mortality rate of about 60%. Caused by C. tetani. Infection often follows a trivial puncture wound—a powerful neurotoxin (tetanospasmin) enters the spinal cord via peripheral nerves where it blocks inhibitory spinal reflexes. Treatment is with benzylpenicillin 1g IV every 6h, metronidazole, and human anti-tetanus immunoglobulin 30U/kg IM. If a wound is present, it is excised and left open to heal by 2° intention. Immunization with 10-yearly boosters is protective.

Osteomyelitis and septic arthritis

(➲ Acute haematogenous osteomyelitis pp. 668–9 ➲ Chronic osteomyelitis pp. 670–1; ➲ Septic arthritis pp. 672–3) The commonest organism is *S. aureus*.

Other organisms in specific cases of bone and joint infections are:

- Sexually transmitted infection (STI): *N. gonorrhoeae*.
- Sickle cell disease: *Salmonella* and *S. aureus*.
- Prosthetic joints: *S. aureus* and *S. epidermidis*.
- Vertebrae: *S. aureus* and TB (Pott disease).
- Cat and dog bites: *Pasteurella multocida*.
- IV drug use: *S. aureus*, *Pseudomonas*, *Candida*.

Diabetic foot infection

(➲ The diabetic foot, pp. 794–5)

Other infections in the surgical patient

Nosocomial infections in the surgical patient

- The commonest nosocomial (health care-acquired) infection in the surgical patient is SSI (➔ Surgical site infections, pp. 224–6).
- Other common nosocomial infections that mainly affect surgical patients are post-operative respiratory and urinary tract infections, bacteraemias (including MRSA and intravascular cannula infections), and antibiotic-related diarrhoeas, particularly *C. difficile* enteritis. Table 3.9 gives a summary.
- The commonest organisms overall are *S. aureus* and *E. coli*.

Table 3.9 Common nosocomial pathogens

Risk factor	Pathogen
Antibiotic use	*C. difficile*
IV lines and drains	*S. aureus* (consider MRSA) *S. epidermidis* *Enterobacter*
Urinary catheterization/UTI	*Proteus*, *E. coli*, *Klebsiella*
Post-endoscopic retrograde cholangiopancreatography (ERCP) biliary sepsis (cholangitis)	Enteric bacteria (e.g. *E. coli*, *Klebsiella* spp., *P. aeruginosa*, *Enterococcus*); also Gram +ve bacteria, e.g. α-haemolytic streptococci, *S. epidermidis*. Multidrug-resistant forms are increasing
Hospital-acquired pneumonia	*Pseudomonas*, MRSA, other enteric Gram –ve bacilli
Aspiration pneumonia	Polymicrobial, often anaerobes
Immunocompromise	(All of the above) + fungi, viruses, *Pneumocystis* (HIV)
Intubated patient, mechanical ventilation	*Pseudomonas*, *Klebsiella*, *Acinetobacter*, *S. aureus* (MRSA)
Burns	*Pseudomonas*

Other infections

Infective diarrhea

Bloody diarrhoea	Watery diarrhoea
Campylobacter	*C. difficile*
Entamoeba histolytica	*C. perfringens*
EHEC	ETEC
EIEC	Protozoa (*Giardia*, *Cryptosporidium*)
Salmonella	V. *cholerae* (rice water diarrhoea, often from seafood)
Shigella	Viruses—rotavirus, norovirus, enteric adenovirus
Y. *enterocolitica*	

UTI

The commonest cause in the community is *E. coli* (and other enteric bacteria). Ten times commoner in women (shorter urethra). Nosocomial/catheter-related UTIs are likely to require broader-spectrum cover.

Pelvic inflammatory disease

Common pathogens: *Chlamydia* (subacute, often undiagnosed) and *N. gonorrhoeae* (acute). Commonly presents with lower abdominal/pelvic pain (can mimic appendicitis). Sequelae include: salpingitis, endometritis, hydrosalpinx, and tubo-ovarian abscess. It is a risk factor for infertility, chronic pelvic pain, and adhesions (Fitz-Hugh–Curtis syndrome—perihepatitis with adhesions associated with *N. gonorrhoeae*).

The asplenic patient

Prone to infection with **encapsulated organisms**—*S. pneumoniae* >> *H. influenzae* > *N. meningitidis* (useful mnemonic: 'SHiN').

Parasites

A rarity in the West but should be considered in patients presenting with unusual surgical presentations or following foreign travel.

Presentation	Associated parasite
Biliary tract disease (+ cholangiocarcinoma)	*Fasciola* (common liver fluke)—associated with watercress or contaminated water *Clonorchis sinensis* (Chinese liver fluke, associated with raw fish ingestion, endemic in East Asia)
Liver (hydatid) cyst	*Echinococcus granulosus* (associated with sheep farming)
Perianal pruritus	*Enterobius*
Portal HTN	*Schistosoma mansoni*, *Schistosoma japonicum**
Haematuria, bladder SCC	*Schistosoma haematobium**
Generalized pruritus (scabies)	*Sarcoptes scabeii* (children, crowded populations)

* Schistosomiasis (aka bilharzia)—from freshwater snails via contaminated water (farmers, children in endemic areas, i.e. Africa, Asia)—can start with skin rash, 'swimmer's itch', commonly affects urinary or GI tract. Note it is not a common cause of portal HTN (the commonest being liver cirrhosis or portal vein thrombosis).

Bleeding and coagulation

Blood products are described under ➲ Blood products and procoagulants, pp. 120–1.

Haemostasis

This is the physiological process by which bleeding is controlled. It has four components:

- *Vessel wall response*, primarily vasoconstriction.
- *Platelet adherence* to exposed endothelial collagen, a process which requires vWF.
- *Platelet aggregation* promoted by release of ADP, arachidonic acid, prostaglandin, and thromboxane A2 (TXA2) ('1° haemostatic plug').
- *The coagulation cascade* (see Fig. 3.4) leads to the formation of thrombin which stimulates the production of an insoluble fibrin mesh from soluble fibrinogen molecules (stable haemostatic plug).

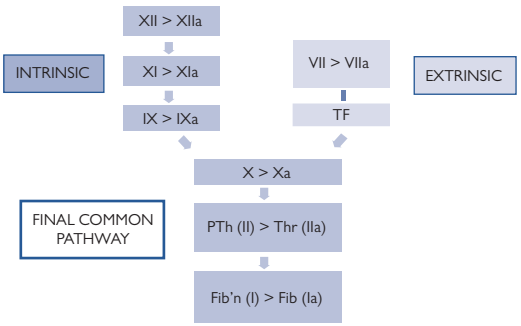

Fig. 3.4 The coagulation cascade. Fib, fibrin; Fib'n, fibrinogen; PTh, prothrombin; TF, tissue factor (aka thromboplastin or factor III); Thr, thrombin. The 'a' signifies the activated form of a factor. Factors VIIIa and Va are co-factors. Factor XIII (fibrin stabilizing factor) not shown.

Coagulation cascade

The coagulation factors are serine proteases—all, except VIII synthesized by the liver. The traditional coagulation cascade involves two linear pathways (intrinsic and extrinsic) leading to a final common pathway (see Fig. 3.4).

More recently, a **cell-based model** of coagulation has been proposed which involves three phases:

- *Initiation*: the first phase occurs on a tissue factor (TF)-bearing cell binding VIIa. Small amounts of IXa and Xa and thrombin are generated.
- *Amplification*: thrombin activates platelets and coagulation factors—via vWF–VIII, produces Va, VIIIa, and XIa.
- *Propagation*: finally, activated coagulation factors (particularly Va and Xa) accumulate on the surface of platelets, which results in the propagation of large amounts of thrombin.

Fibrinolysis

- The fibrinolytic system terminates thrombus propagation to maintain circulating blood in a fluid state; it depends on four proteins:
  - *Plasmin*, produced from plasminogen activated by agents such as tissue plasminogen activator (tPA) or urokinase—attack unstable bonds between fibrin molecules to generate fibrin degradation products.
  - *Antithrombin III* which deactivates thrombin and Xa.
  - *Proteins C and S* which prevent thrombin generation by binding factors Va and VIIIa. Tissue factor pathway inhibitor, produced by platelets, inhibits factor VIIa.

Drugs affecting haemostasis

Antiplatelets

- *Aspirin* and *clopidogrel* irreversibly inhibit platelets, lasting for the life of the platelet (7–10 days). *Aspirin* inhibits prostaglandin (TXA2) synthesis via cyclo-oxygenase; *clopidogrel* inhibits ADP binding.
- *Other antiplatelets*: *dipyridamole* (24h duration) reversibly increases cAMP levels in the platelet; *glycoprotein IIb/IIIa* (GpIIb/IIIa) inhibitors, such as abciximab (4- to 5-day duration), directly inhibit the platelet receptor used for aggregation.

Anticoagulants

- *Warfarin* inhibits the production of vitamin K-dependent clotting factors (II, VII, IX, X)—taking 3–4 days to take effect and to wear off. Reversed with vitamin K.
- *Heparin* potentiates antithrombin III, with immediate effect; protamine binds heparin, reversing its effect almost immediately.
- *NOACs (e.g. rivaroxaban, apixaban)* directly inhibit factor X or prothrombin (factor II)—onset is within hours and effect lasts 12–24h after cessation. Their advantage is a more consistent anticoagulant effect (the close therapeutic monitoring as for warfarin is not needed) and interactions with other drugs are fewer. Disadvantage is that there is no reliable reversal agent.

Thrombolytics

Whilst anticoagulants prevent clot propagation, these agents actively dissolve clots. Naturally occurring compounds (e.g. tPA, urokinase) or synthetic activators of plasmin (alteplase, reteplase) are used, mainly for acute thromboembolism.

Disorders of haemostasis

These can be thought of in terms of the four components below:

- *Vessel wall abnormalities*, e.g. Henoch–Schönlein purpura, Cushing's syndrome, steroid use, vitamin C deficiency (scurvy).
- *Platelets*:
  - *Thrombocytopenia* (<100 × 10^9/L)—caused by reduced production (bone marrow failure, radiotherapy, chemotherapy, infiltrative disease, e.g. neoplasia, leukaemia); faulty maturation (e.g. folate and B12 deficiencies); abnormal distribution (splenomegaly); ↑ destruction (autoimmune disorders, drugs, DIC, haemorrhage); and dilutional thrombocytopenia in massive banked blood transfusion.

- *Abnormal platelet function*, e.g. von Willebrand's disease, uraemia, idiopathic causes, drug effects, especially aspirin and clopidogrel.
- *Coagulation abnormalities*:
  - *Congenital*, e.g. haemophilia A (↓ factor VIII), haemophilia B or Christmas disease (↓ factor IX), von Willebrand's disease (↓ vWF).
  - *Acquired*, e.g. DIC (➲ Disseminated intravascular coagulation (DIC), p. 146); ↓ vitamin K (produced by gut flora) 2° to poor nutrition, antibiotic therapy, or obstructive jaundice; liver disease; exogenous anticoagulants.

Evaluation of haemostasis

History and examination

- Ask about bleeding problems, e.g. menorrhagia, bruising, family history of bleeding, and medication.
- Look for petechiae and purpura, jaundice, and evidence of hepatic or renal disease.

Laboratory investigations

- *Platelet count*. Normal range: 200–400 × 10^9/L; >70 × 10^9/L is needed for surgical haemostasis; <20 × 10^9/L results in spontaneous bleeding.
- *Blood film*. Estimate of platelet count and indicates morphology.
- *Bleeding time*. Normal bleeding time indicates normal platelet number and function and normal vascular response to injury, but it is uncommonly used as a screening test.
- *PT*. Reflects the extrinsic pathway (I, II, V, VII, X).
- *Partial thromboplastin time (PTT)*. Reflects the intrinsic pathway (all factors, except VII).
- *Individual clotting factor assays*.
- *Thrombin time (TT)*. Rate of conversion from fibrin to fibrinogen.
- *Fibrin degradation products*. These are released by the action of plasmin and are raised in DIC.
- *Factor Xa levels*. Used to monitor LMWH, particularly in patients with renal failure.
- *Novel point-of-care methods* such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM).

Anaemia and polycythaemia

Anaemia ➔ Preoperative management of Anaemia pp. 90–2

Defined as Hb <13g/dL in men and women.

Classified as follows:

- ↓ *red cell production*.
  - Haematinic deficiency (↓ Fe, B12, folic acid).
  - Bone marrow failure (congenital, chemo-/radiotherapy, infiltrative disease).
- *Abnormal red cell maturation*.
  - Myelodysplasia.
  - Sideroblastic anaemia.
- ↑ *red cell destruction (the haemolytic anaemias)*.
  - Inherited (e.g. sickle cell disease, thalassaemia).
  - Acquired (e.g. autoimmune, DIC ➔ Disseminated intravascular coagulation (DIC), p. 146).
- *Chronic disease* (common cause of anaemia in surgical patients).
  - Renal failure (↓ production of EPO).
  - Endocrine, liver disease.

Iron deficiency anaemia

Commonest cause of anaemia in surgical patients. Causes include:

- Menstruation (in 15% of ♀).
- GI losses (peptic ulcer, oesophagitis, gastric carcinoma, colorectal carcinoma).
- Reduced iron uptake (poor diet, coeliac disease, malabsorption).

Sickle cell anaemia

A single-base substitution gene defect causing an amino acid substitution in Hb, making HbS instead of HbA. Deoxygenated HbS polymerizes and causes red blood cells to sickle, resulting in occlusion of small blood vessels and infarction. Common in Africans. Homozygotes have high levels of HbS and are prone to crises. Heterozygotes ('sickle trait') are only symptomatic in hypoxic conditions, e.g. unpressurized aircraft, limb ischaemia.

- Most patients are diagnosed as screening is widespread.
- The patient typically has an Hb of 6–8g/dL; reticulocytes 10–20%.
- There are three types of sickle cell 'crisis':
  - *Thrombotic crises*. Precipitated by cold, dehydration, infection, and ischaemia; may mimic acute abdomen or pneumonia; priapism.
  - *Aplastic crises*. Due to parvoviruses and require urgent transfusion.
  - *Sequestration crises*. Spleen and liver enlarge rapidly from trapped erythrocytes, resulting in RUQ pain, ↑ INR, ↑ LFTs, and ↓↓ Hb.
- Treatment involves removing precipitating factors and decreasing the percentage of HbS.
- *Rx:* Keep warm and well hydrated, O_2, analgesia. Empirical antibiotics if any evidence of sepsis. Blood transfusion if ↓ Hb, exchange transfusion.

Thalassaemia

Thalassaemias are genetic diseases of Hb synthesis, resulting in underproduction of one chain, which results in destruction of red cells whilst they are still in the bone marrow. Common in the Mediterranean to the Far East.

α-thalassaemia leads to ↓ α-chain production with unbalanced β-chain production, and β-thalassaemia leads to ↓ β-chain production.

- *Prognosis*: severity correlates with the genetic deficit.
- In severe cases, death may result by age 1y without transfusion, and at 20–30y due to cardiac siderosis. Symptoms of iron overload after 10y: endocrine failure, liver disease, and cardiac toxicity.

Polycythaemia

- *Relative*—↓ plasma volume. Dehydration from alcohol or diuretics.
- *Absolute*—↑ red cell mass.
  - 1°—polycythaemia rubra vera.
  - 2°—altitude, smoking, COPD, tumours, e.g. fibroids.
- Rx: Treat underlying cause; consider venesection.

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Chapter 4

Practical procedures

The anaesthesia face mask

Key facts

The anaesthesia face mask is the device most commonly used to deliver O_2 and anaesthetic gases, as well as to ventilate patients who have been apnoeic.

Despite the many advances in airway management, it remains a mainstay in the delivery of anaesthesia and in resuscitation. It is an essential fall-back technique for maintaining oxygenation during a failed or difficult intubation.

Face mask ventilation is minimally invasive and virtually universal and requires the least sophisticated equipment, thus making it critical to management of the airway.

Equipment

- Face mask, reservoir bag, and tubing.
- O_2 source.
- Suction.
- Oropharyngeal airway/nasopharyngeal airway.

How to use?

- Inform the patient about placement of a face mask on their face.
- Place the mask on the patient's face; cover the nose and mouth without extending it over the chin.
- Gently hold the mask on the patient's face with the left hand, leaving the right hand free for other tasks.
- Appropriate positioning of the mask against the patient's face is paramount.
- Achieve a tight fit of the face mask by downward displacement of the mask between the thumb and the first finger, with concurrent upward displacement of the mandible with the remaining fingers (jaw thrust).
- Hold the mask with two hands in patients who are obese, have a beard, or are edentulous. In this situation, a second person is required to ventilate the patient.
- Oral or nasal airways create a passage to facilitate bag–mask ventilation in difficult situations.

Tips and pitfalls

- Air leak around the face can be prevented by use of the two-hand technique.
- Applying excessive pressure on the face may be uncomfortable for the patient.
- Applying excessive pressure during ventilation can lead to gastric distension and may cause the patient to feel sick.
- Aspiration is a risk and the best way to prevent aspiration is with a good technique, including low pressure and low volume with slow insufflation.
- Also in emergency situations, prior to endotracheal intubation, cricoid pressure can be applied to prevent aspiration.
- Difficulty during mask ventilation may be caused by malposition of the face mask, inadequate patient position, inadequate depth of anaesthesia, inadequate muscle relaxation, enlarged tonsils, laryngospasm, and bronchospasm, and if the patient is edentulous or has a beard.

Laryngeal mask airway

Key facts

The laryngeal mask airway (LMA) is a supraglottic airway device. It is designed to sit in the patient's hypopharynx and cover the supraglottic structures. The LMA is used in the operating room, emergency department, and out-of-hospital care.

Equipment

- LMA.
- Syringe for cuff inflation.
- Bag–valve mask.
- O_2 source.
- Yankauer suction device.
- End-tidal CO_2 ($ETCO_2$) detector.
- Intubation equipment.

Preparation

- Preoxygenate the patient with 100% O_2.
- Choose appropriate size of LMA
- Check the LMA for cuff leaks; apply water-soluble lubricant to the posterior surface of LMA.
- Insert the LMA when the patient is ready, i.e. when there is loss of cough and gag reflexes.

Placement

- Hold the LMA like a pen, with the index finger of the dominant hand at the junction of the cuff and the tube.
- Slide the LMA along the hard palate, pushing it against the palate as it is advanced towards the hypopharynx.
- Advance with gentle pressure until resistance is met.
- Once in place, inflate the cuff without holding the LMA, to allow it to acquire its position.
- Confirm the position of the LMA by auscultating breath sounds bilaterally, observing chest rise with ventilation and monitoring $ETCO_2$.

Tips and pitfalls

- Do not insert LMA in patients with cough or gag reflex present.
- Avoid placing LMA in patients with restricted mouth opening, as this may make insertion difficult and can cause trauma.
- Potential pharyngeal or laryngeal injury if excessive force is applied during insertion.
- LMA does not prevent aspiration of gastric contents.

Endotracheal intubation

Key facts

Performed to secure the airway in both elective and emergency situations, e.g. GA, cardiac arrest, impaired consciousness, respiratory failure, trauma, etc.

- Effective bag-and-mask ventilation is better than ineffective attempts at endotracheal intubation in the arrest setting.
- Endotracheal intubation should only be attempted by those with experience in advanced airway management and placing an endotracheal tube (ETT).

Equipment

- ETT (7.0–8.0 for ♀ and 8.0–9.0 for ♂).
- Laryngoscope, conventional and advanced in case of anticipated difficult airway (e.g. McCoy, Airtraq, GlideScope, etc.).
- Syringe (10 or 20mL), lubricating jelly, ETT-securing device (tape, ribbon, etc.), bougie.
- Anaesthetic circuit or Ambu bag, O_2 supply (anaesthetic machine, O_2 cylinder or wall connection).
- Working suction equipment (machine, tubing, yankauer, and catheter).
- Personal protection equipment (gloves, mask, plastic gown, etc.).

Drugs

- Induction agent (propofol, thiopental).
- Muscle relaxant (suxamethonium, atracurium).
- Analgesia (fentanyl, alfentanil).
- Other drugs (ketamine, midazolam, resuscitation medications, etc.).

Monitoring

- Respiratory (O_2 saturation, $ETCO_2$).
- Cardiovascular (ECG, BP, HR).

Preparation

- Confirm integrity of ETT cuff.
- Ensure all airway equipment is working.
- Secure IV access and commence monitoring.
- Move bed/trolley forward, so that you can stand behind the patient's head, and raise it so that you are working at a comfortable height.
- *Elective setting*. Pre-oxygenate the patient with 100% O_2 using the anaesthetic circuit.
- *Emergency setting*. Pre-oxygenate the patient with 100% O_2 using self-inflating bag.
- Remove any dentures.
- Extend the neck (sniffing position); insert the laryngoscope from the right corner of the mouth; push the tongue to the left and advance the laryngoscope blade anterior to the epiglottis, and pull gently upwards (not levering on upper teeth) to expose the vocal cords.
- Insert the lubricated ETT between the cords into the trachea (see Fig. 4.1a). Inflate the cuff till no leak is heard.

- Confirm correct placement (seeing the ETT passing through the vocal cords, observing chest movement, fogging of the ETT, auscultation for breath sounds, $ETCO_2$), and secure the tube in place using tape or tie (see Fig. 4.1b).
- To keep the patient tolerant of ETT, sedation is required. In the operating theatre, this is provided by GA, but outside the operating theatre, sedative infusions are given to keep the patient tolerant of ETT.

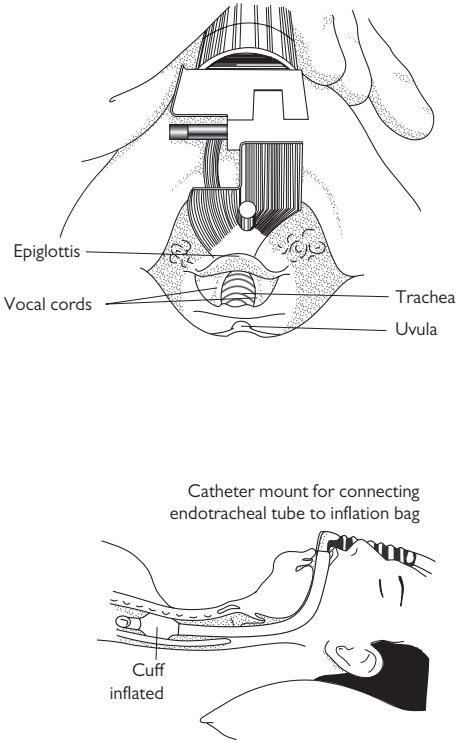


Fig. 4.1 (a) Diagram of the larynx as seen at intubation. (b) Correct position of inflated endotracheal tube cuff.

Tips and pitfalls

- Difficult intubation, e.g.:
  - Receding mandible.
  - Buck teeth.
  - Short neck.
- Cannot visualize vocal cords—use airway aids like GlideScope or Airtrac.
- Cannot intubate, cannot ventilate situation—seek help.
- Oesophageal intubation—potentially fatal if not recognized.
- Inadequate cuff pressure—check cuff integrity, pressure, and any leak around the tube.
- Dental damage.
- Aspiration is a risk and the best way to prevent aspiration is with a good technique, including low pressure and low volume with slow insufflation.
- To prevent aspiration, cricoid pressure can be applied where there is a high risk of regurgitation, e.g. emergency surgery, Caesarean section under GA, patients with severe acid reflux.

Percutaneous dilatational tracheostomy

Key facts

Percutaneous dilatational tracheostomy (PDT) is carried out in adult intubated patients. It is an elective procedure to secure the airway by creating a passage between the tracheal rings in order to place a tracheostomy tube. PDT is a fast, safe, cost-effective, and relatively easy-to-perform technique to secure the airway for long-term mechanical ventilator support. It can be performed at the bedside by an adequately trained team of doctors and nurses.

Prolonged mechanical ventilation using an ETT is problematic and causes numerous complications. It requires considerable sedation for ETT to be tolerated, making weaning patients from a ventilator more difficult; also prolonged ETT placement carries the risk of formation of laryngeal and subglottic stenosis.

Equipment

- PDT kit with appropriate-sized tracheostomy tube.
- Bronchoscope, swivel connector with a port for the bronchoscope.
- Curved artery forceps, tracheal dilator.

Planning and preparation

- Recent CXR, Hb, platelets, coagulation screen.
- Perform ultrasound of the neck to ensure presence/absence of any blood vessel.
- Documents: Consent form 4, WHO surgical checklist, operative document.
- Ensure adequate number of personnel are present (operator, endoscopist, nurse to secure ETT, circulating nurse).

Landmarks

- Thyroid cartilage, cricoid cartilage, tracheal rings, supra-sternal notch.

Technique

- Strict asepsis.
- Deepen sedation to ensure the patient is not conscious; muscle relaxation.
- Position the patient—gently extend the neck using a rolled towel under the shoulders and a head-ring to support head.
- Deliver 100% O_2 and monitor vital signs: HR, ECG, BP, O_2 saturation, and $ETCO_2$.
- Operator 2:
  - Performs direct laryngoscopy and pulls the ETT out, ensuring that the cuff is just below the vocal cords.
  - Performs bronchoscopy and places the bronchoscope at the distal end of the ETT, ensuring that the trachea is clearly visible.
  - Guides operator 1 at every step of the procedure, from needle insertion to correct placement of the tracheostomy tube.
- Operator 1 infiltrates the incision line with 1% lidocaine with adrenaline.
- Once skin incision is made, separate subcutaneous tissues using artery forceps and the little finger until the tracheal rings are palpable.

- Insert a 14G Teflon catheter needle between the second and third tracheal rings, taking care not to pierce through the posterior tracheal wall. Remove the needle and leave the catheter in the trachea; pass the guide wire through the catheter, and remove the catheter. Pass a guide catheter and a graduated curved dilator over the guide wire, and separate the tracheal rings gently to accommodate the graduated dilator up to the black mark. Keep the dilator in this position for 15–30s.
- Remove the graduated dilator, leaving the guide wire and guide catheter in place.
- Now pass an appropriately sized tracheostomy tube over the guide wire and guide catheter.
- Once placement of the tracheostomy tube is confirmed (auscultation, direct bronchoscopy, $ETCO_2$), secure the tracheostomy tube and remove the ETT.
- Now ventilate the patient through the tracheostomy tube.

Complications

- Bleeding.
- Surgical emphysema or pneumothorax.
- Laceration of posterior tracheal wall.
- Accidental decannulation or displacement of tracheostomy tube.
- False tract formation.
- Tracheal stenosis.

Cricothyroidotomy

Key facts

Cricothyroidotomy is an important emergency procedure that is used to obtain an airway when other (more routine) methods are ineffective. This is a lifesaving procedure.

Indications

- Trauma causing oral, pharyngeal, or nasal haemorrhage.
- Upper airway deformities.
- Tumour or other disease process or trauma causing mass effect.
- Airway oedema (anaphylaxis).
- Foreign body.
- Maxillofacial injuries.

Equipment

- Antiseptic solution.
- Lidocaine.
- Sterile drapes/gloves/gown.
- Bag–valve–mask.
- Tracheal dilator, tracheal hook, tracheostomy tube or ETT.

Procedure

- Place the patient in a supine position; clean the skin.
- Apply cervical spine stabilization.
- Identify the cricothyroid membrane, and with the dominant hand, insert the introducer needle attached to a fluid-filled 5mL syringe through the cricothyroid membrane; as the needle is advanced, apply negative pressure to the syringe. A distinct pop can be felt as the needle traverses the membrane and enters the trachea; in addition, air bubbles will appear in the fluid-filled syringe.
- Remove the syringe from the needle and advance the guide wire through the needle. Remove the needle once the guide wire is in place.
- Make a small stab with the scalpel close to the guide wire.
- Place the dilator over the airway catheter, and insert the two devices together over the wire. Once in place, remove the wire and dilator, leaving the airway tube in the trachea.
- Confirm correct placement of the tube and secure it appropriately.

Complications

- Haemorrhage (may be life-threatening).
- Subcutaneous emphysema.
- Injury to the oesophagus, thyroid, vocal cords, and other adjacent structures.
- Infection.
- Late complications: scarring, dysphonia, persistent stoma, trachea–oesophageal fistula.

Non-invasive ventilation

Key points

NIV refers to provision of ventilatory support through the patient's upper airway using either a tight-fitting face mask or a hood.

Generally, two modes of ventilatory support are provided: CPAP or bi-level positive airway pressure (BiPAP).

This can be provided in specialized areas where trained nursing staff is present to deliver the service led by a dedicated team of doctors.

NIV has become an integral tool in the management of both acute and chronic respiratory failure, in both the home setting and the critical care unit.

Indications

- COPD with respiratory acidosis.
- Respiratory failure due to chest wall deformity and neuromuscular disease.
- Cardiogenic pulmonary oedema.
- Weaning from mechanical ventilation.
- Sleep hypoventilation/apnoea.

Contraindications

- Coma.
- Cardiac arrest.
- Respiratory arrest.
- GI bleeding.
- Inability to protect airway (impaired cough or swallowing).
- Potential for upper airway obstruction.

Method of delivery

- Ventilator support can be achieved through a variety of interfaces (face mask, nasal mask, or helmet mask).
- Patient cooperation is essential.

Tips and pitfalls

- NIV is well suited for patients with cardiogenic pulmonary oedema.
- Both CPAP and BiPAP modalities are effective, with CPAP possibly being more effective.
- The greatest benefits are realized in the relief of symptoms and dyspnoea.
- A decrease in intubation and mortality rates is not a universal experience.
- Patients with hypercapnic respiratory acidosis may derive the greatest benefit from NIV.
- Importantly, adjust to standard therapy, including diuresis.
- Benefit may be seen within 2h of support.

Venepuncture

Key facts

The term venepuncture describes the procedure of inserting a needle into a vein. This is a mandatory skill to learn for all doctors.

IV cannulation is placement of a plastic cannula in the vein which is loaded over a needle and is used for long-term use.

Common veins for venepuncture and IV cannulation are the superficial veins of the arm.

Indications

- Venepuncture is performed to obtaining venous blood samples for laboratory analysis.
- IV cannulation provides venous access for administration of IV fluids, blood, and IV drugs.

Equipment

- Tourniquet.
- Venepuncture (23G or 21G) needle, vacuum tube system to collect blood, syringes, alcohol swabs, appropriate laboratory sample tubes.
- Venous cannula (20G or 18G), adhesive dressing, cleaning wipes.

Preparation

Wear appropriately sized gloves; apply tourniquet above or below the elbow, as required, and inspect the arm for suitable engorged veins.

Method

- Clean the skin thoroughly with alcohol wipes; wear gloves and clean the skin with chlorhexidine solution.
- For venepuncture, select the median cubital vein, as this is easily accessible, and after the procedure, it is easy to bend the elbow to stop any post-procedure bleeding.
- For peripheral IV cannulation, select a distal vein first before trying to puncture proximal veins. Avoid veins above joints or bony prominences.
- Common sites are the dorsum of the hand and veins on the forearm.
- Tether the skin distal to the site with the thumb of your non-dominant hand.
- Pass the needle obliquely through the skin at a point ~2mm distal to the point of planned entry to the vein.
- Advance the needle gently until a 'give' is felt as the vein is entered and a 'flashback' is seen in the chamber of the needle or cannula.
- For venepuncture, attach a vacutainer to draw blood in appropriate sample bottles or draw blood in a syringe and aspirate the required volume of blood.
- For cannulation, after penetrating the vein, the needle is withdrawn and the cannula is pushed into the vein.
- Remove the needle and leave the cannula in place. Now you can withdraw blood or give fluids and medications if required.
- REMEMBER! Always release the tourniquet after completion of blood collection (venepuncture) and placing a cannula in place. Ensure safe disposal of sharps and needles.
- Apply pressure to the site to arrest any bleeding. Do not assume the patient can help with this, e.g. stroke patients.

Tips and pitfalls

- *Difficult venous access*. In obese patients, post-chemotherapy patients, hypovolaemic patients, IV drug users, and those who naturally have poor venous access.
- *Needle phobia*.
- *Failed attempts*. Repeated failed attempts would distress the patient, so ask for help.
- *Ultrasound*. Occasionally ultrasound may be required to locate deep veins to gain venous access.
- *Blood cultures*. These must be taken according to local policy. However, strict asepsis and appropriate technique are recommended to avoid sample contamination.
- *Labels and forms*. Ensure that all samples are labelled correctly and forms filled to provide required patient information (demographics, clinical details, reason for request) and tests requested. Failure to do so may give the wrong result that may either expose the patient to inappropriate antibiotic therapy or delay the diagnosis and deprive the patient of receiving appropriate treatment.
- *Electronic requests*. If you are requesting electronically, please follow the same rules as followed on paper request form.

Key revision points—venous drainage of the upper limb

- Superficial venous system:
  - *Cephalic vein*. Commences from the lateral end of the dorsal venous network overlying the anatomical snuffbox, ascending the lateral and anterolateral aspect of the arm to the deltopectoral groove and piercing the clavipectoral fascia to join the axillary vein.
  - *Basilic vein*. Commences from the medial end of the dorsal venous network, ascending along the medial and anteromedial aspects of the forearm, piercing the deep fascia to join the venae comitantes of the brachial artery, which eventually joins the axillary vein.
  - *Median cubital vein*. Connects these two veins in the cubital fossa.
- Commonest sites for venepuncture and cannulation:
  - Dorsal venous network.
  - Median cubital vein.
  - Cephalic vein in the forearm.
  - Other sites—dorsum of the foot or the saphenous vein.

Peripheral intravenous cannulation

Key facts

A skill similar to that of venepuncture. IV cannulation is placement of a plastic cannula in the vein which is loaded over a needle and is used for long-term use. Common veins for venepuncture and IV cannulation are the superficial veins of the arm.

Indications

- IV cannulation provides venous access for administration of IV fluids, blood, and IV drugs.

Contraindications

- Presence of injury or damage (fracture, oedema, lymphoedema, thrombosis, etc.).
- Avoid veins over bony prominences.

Equipment

- Tourniquet.
- Venous cannula (20G or 18G), adhesive dressing, cleaning wipes.
- Safety cannulae must be used to prevent sharps injuries.

Selecting a site for cannulation

Select veins that are accessible, unused, and easily detected by palpation and/or visual inspection, appear healthy, and are patent (feel soft and bouncy to touch and refill quickly following compression).

The commonest veins used are basilic or cephalic veins of the arm. The antecubital fossa veins should only be used as a last resort.

Preparation

Apply a tourniquet above or below the elbow, as required, and inspect the arm for suitable engorged veins.

Wearing gloves is recommended, particularly in patients known to have blood-borne diseases.

Method

- Infection control—thorough hand cleaning, gloves worn, and the patient's skin cleaned with chlorhexidine.
- Select a distal vein first before trying to puncture proximal veins.
- Anchor the vein by holding the surrounding skin taut using your non-dominant hand.
- Pass the cannula over the needle, obliquely through the skin at a point ~2mm distal to the point of planned entry to the vein.
- Advance the needle gently until a 'give' is felt as the vein is entered and a 'flashback' is seen in the chamber of the cannula.
- After penetrating the vein, withdraw the needle and push the cannula into the vein.
- Remove the needle and leave the cannula in place. Now you can withdraw blood or give fluids and medications.
- REMEMBER! Always release the tourniquet after completion of blood collection (venepuncture).

- Ensure safe disposal of sharps and needles.
- Flush the cannula with 5mL of saline and secure the cannula in place with sterile dressing.

Tips and pitfalls

- *Observe cannulation being performed and practise to become competent.*
- *Difficult venous access*. In obese patients, post-chemotherapy patients, hypovolaemic patients, IV drug users, and those who naturally have poor venous access.
- *Difficult venous trolley*. In key areas, e.g. operating theatres, ICUs, and emergency departments, where there is a difficult IV trolley kept which contains various equipment that may be needed to assist in venous cannulation.
- *Needle phobia*. Establish if the patient has genuine phobia for needles. Anxiety may make successful needle/cannula placement difficult. A careful explanation and a confident manner are essential.
- *Failed attempts*. Ask for help or consider central venous cannulation or a PICC line for long-term IV access.
- *Ultrasound*. Occasionally, ultrasound may be required to locate deep veins to gain venous access.

Factors affecting success of venous cannulation

- Patient factors: anxiety, patient cooperation, temperature, dehydration.
- Other: thrombosed veins, cannula size, skill of performer.

Complications

- Accidental damage to adjacent structures.
- Phlebitis (advice on changing cannula after 2–3 days).
- Bleeding and haematoma formation.
- Extravasation.
- Residual anaesthetic or sedative drugs may be left in IV lines and cannulae, unless they are effectively flushed at the end of the procedure. If they are not, the residual drug can be later inadvertently introduced into the patient's circulation, causing muscle paralysis, unconsciousness, and respiratory and cardiac arrest (Patient Safety Alert, 9 November 2017).

Failure to cannulate

- Is a cannula necessary?
  - Can medication or fluids be given PO or via NGT?
  - In the case of antibiotics, discuss with the microbiologist about changing the route of administration if possible.
  - Can insulin regimes be modified to be given SC?
  - Many painkillers and antiemetics can be given PR or IM.
- Ask another colleague to try.
- Contact the anaesthetist on-call if they can help, but remember they are *not* a cannulation service.
- Think of using ultrasound scan (USS) and alternative routes—PICC line, central line.

Intraosseous access

Key points

The intraosseous (IO) route is a reliable and extremely valuable alternative to venous access. IO access can be used in trauma, emergencies, and cases requiring volume resuscitation.

Inserting an IO needle is easy to learn, but success depends on familiarity with the equipment and correct technique.

IO access provides an alternative method to providing venous administration of drugs and fluids in the emergency department, at cardiac arrests, in paediatrics, and in cases where IV access is challenging or time-critical.

Preparation

Equipment

- IO device to gain access.
- Pressurized system to administer fluids.

Common access sites

- Humeral head.
- Proximal tibia.
- Sternum.

Procedure

- Sterilize the skin at needle site insertion.
- Manually stabilize the bone during insertion.
- Inject LA at the injection site to reduce pain.
- Aspirate after needle insertion—blood/bone marrow confirms successful placement.
- Ensure the needle is flushed with at least 10mL of saline after drug administration.
- Frequently assess the IO site for signs of extravasation.

Tips and pitfalls

- The humeral site is the least painful and quickest to access.
- All resuscitation and anaesthetic drugs can be given via the IO route.
- Fluids need to be administered under pressure.
- Improper placement may lead to extravasation, which can then lead to compartment syndrome.
- There is a risk of osteomyelitis, which is particularly high when the inserting site is infected, damaged, or burnt, and also if the device is left for >24h.
- Sternal insertion carries the risk of pneumothorax, damage to great vessels, and mediastinitis.

Central venous cannulation

Key facts

Central venous cannulation (CVC) is placement of a single or multi-lumen catheter in one of the central veins, i.e. SVC or IVC. Access to these veins can be gained via the internal jugular, subclavian, or femoral veins.

CVC can be used for giving vasoactive drugs, nutrition, drugs in high concentration, chemotherapeutic agents, and blood products and also to obtain blood for laboratory analysis.

Internal jugular and subclavian lines can allow measurement of CVP.

Equipment

- Central venous catheter (CVC) tray.
- Sterile gloves, gown, drapes.
- Antiseptic solution.
- Ultrasound.
- 1% lidocaine, syringe.

Indications

- Emergency venous access—cardiac arrest, hypovolaemic shock, septic shock.
- CVP monitoring.
- Nutritional support.
- Administration of caustic medications, e.g. inotropes, cytotoxic drugs.
- Transvenous pacing wire introduction.
- Haemodialysis.
- Pulmonary artery catheterization.
- Difficult venous access.

Contraindications

- Distorted local anatomy.
- Infection at insertion site.
- Presence of anticoagulation or bleeding disorder.
- Current thrombolysis.

Preparation (internal jugular venous cannulation)

- Explain the procedure, benefits, risks, and complications to the patient in the presence of a nurse, and obtain verbal consent if the patient is awake.
- Place the patient in a supine position, with the bed raised to a height that is comfortable for the operator. Now place the patient in 10–15% Trendelenburg position to distend the internal jugular vein (IJV) and reduce the risk of air embolism.
- Turn the patient's head to the left side for right IJV cannulation.
- Identify anatomical landmarks (clavicle, sternocleidomastoid muscle, palpate the carotid artery).
- Using the ultrasound probe, identify the IJV (collapsible on applying gentle pressure) and carotid artery (non-collapsible on pressure) at the level of the cricoid cartilage. In the majority of cases, the vein lies lateral to the artery.

- Using a needle attached to a 5mL syringe, insert the needle at an angle of 30–45° to the skin surface, between the two heads of the sternocleidomastoid muscle and pointing towards the ipsilateral nipple.
- Once blood is aspirated, pass the guide wire through the needle; remove the needle and introduce a dilator; once the vein is dilated, pass the CVC over the guide wire.
- Once the CVC is in place, remove the guide wire and confirm correct placement of the CVC by aspirating blood from all lumens.
- Secure the catheter and complete documentation.
- Post-insertion of CVC—request a CXR to confirm placement and exclude pneumothorax.

Other insertion sites for central venous catheters

- Subclavian vein.
- Femoral vein.

Complications

- Arterial puncture, haematoma.
- Air embolism.
- Pneumothorax.
- Haemothorax.
- Arrhythmias.
- Infection.
- Loss of guide wire, catheter fracture.

Peripherally inserted central catheters

Key facts

PICCs are used for medium- to long-term IV access. The catheter is inserted into a peripheral vein in the arm at the antecubital fossa or in the upper arm and threaded along the vein until the distal tip rests in the SVC.

PICCs are a reliable alternative to CVCs, with a lower potential for complications than short-term CVCs.

These are relatively economical and have a lower risk of complications. They can be inserted by trained doctors and nurses and also can be managed in the hospital and in community.

Indications

- For infusing highly concentrated medications.
- Long-term antibiotic treatment.
- Patients with limited venous access.
- Parenteral nutrition.
- Withdrawing blood for laboratory sampling.

Cautions and contraindications

- Presence of upper extremity venous or subclavian vein thrombosis.
- Patients with chronic renal failure may need to preserve veins for future dialysis fistulae.

Troubleshooting

- Unable to flush PICC.
- Unable to obtain blood sampling.
- Leakage of fluid at the exit site or along PICC.
- The patient is experiencing pain in the shoulder, neck, or chest, redness, swelling, or exudate at the exit site, redness tracking up the arm, or pyrexia or rigor post-PICC flush.

Refer the patient to the hospital for advice.

Procedure

- Obtain patient consent.
- Full aseptic precautions.
- Insert the cannula and stylet into a vein in the antecubital fossa.
- Remove the stylet and insert the catheter through the cannula.
- Pull back the cannula and peel it away from the catheter.
- The catheter is secured in position with sutures.
- Another method is the Seldinger technique.

Complications

- Thrombus formation.
- Non-thrombotic occlusions.
- Phlebitis.
- Catheter malposition.
- Catheter damage.

Care and maintenance

- Proper dressing and care are required for the PICC line to last.
- Regular flushing is required; use a 10mL or larger syringe to prevent excessive flushing force that can damage the catheter.
- The PICC must be removed with caution by pulling it out gently. If resistance is encountered, which may be due to venospasm, coil the exposed catheter and apply a sterile dressing over it. Try again later, applying gentle traction.

Arterial puncture and arterial cannulation

Key facts

Arterial puncture is needed to sample arterial blood gases (ABGs).

If continuous monitoring of BP is required or if there is a clinical need to monitor ABGs on a regular basis, then an arterial cannula is placed in the artery.

Equipment

- In order to perform an arterial puncture, a 22G needle and a blood gas syringe are required.
- An arterial cannula needs to be placed under aseptic conditions—either use prepacks or have the following equipment available:
  - Arterial cannulae.
  - Transducer set with specific tubing.

Preparation

- Inform the patient.
- Position the insertion site appropriately; for example, for radial artery insertion, place the forearm on a pillow and slightly dorsiflex the wrist, and for femoral artery cannulation, abduct and flex the hip slightly.

Common arterial line cannulation sites

- Radial artery—most commonly used.
- Brachial artery.
- Ulnar artery.
- Femoral artery.
- Dorsalis pedis artery.

Technique

- Performed under sterile conditions (prepare skin, sterile gloves, gown, etc.).
- Infiltrate LA (1% lidocaine) at the site of arterial cannulation. This provides analgesia and prevents vasospasm.
- Palpate the artery with the tips of the index, middle, and ring fingers to confirm pulsation and direction of the artery.
- Pass the needle or cannula at 45° into the skin.
- Arterial cannula can be placed by either the transfixion technique or the non-transfixion technique (see Fig. 4.2).
- *Transfixion technique*. The cannula is passed through the artery until flashback of blood stops; the needle is then partially withdrawn and very slowly, the cannula is withdrawn as well. Once there is flashback of blood, only the cannula is advanced to its full length and secured.
- *Non-transfixion technique*. During insertion of the arterial cannula, once there is flashback of blood, do not advance the needle anymore—just advance the cannula only. Once the entire length of the cannula is in the artery, remove the needle and secure the cannula.
- You can place a 2mL syringe without the plunger at the end of the arterial cannula to trap blood in order to avoid spillage of blood.
- Once it is confirmed that the arterial cannula is in place, secure it by suturing (for long-term use); alternatively, the cannula can be secured thoroughly with cannula-securing tape (see Fig. 4.2).

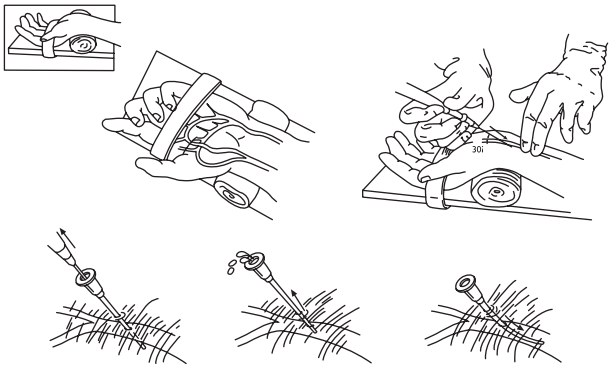


Fig. 4.2 Transfixion technique of arterial cannula insertion.

Complications

- Bleeding, distal ischaemia, thrombosis, damage to adjacent nerves, infection, occlusion of the artery.

Contraindications

- Inadequate circulation to the extremities.
- Raynaud syndrome.
- Infection at cannulation site.
- Vascular graft.

Caution

- Disconnection.
- Inadvertent intra-arterial injection of drugs.

Allen's test

- Allen's test is performed before radial artery cannulation is initiated.
- It is designed to evaluate for adequate collateral circulation to the palmar arch of the hand.
- Apply pressure to ulnar and radial arteries so as to occlude them, and simultaneously ask the patient to make a fist for 30s.
- The patient is then asked to open the hand, which should appear blanched.
- Pressure over the ulnar artery is released and the time it takes for colour to return is measured. If colour returns to the hand in 5s, the radial artery can safely be cannulated.

Chest drain (intercostal drain/pleural drain) insertion

Key facts

Chest drain insertion is an essential skill for hospital doctors. It is a flexible plastic tube that is inserted through the chest wall into the pleural space. It is used to drain pneumothoraces or pleural effusions and can be performed at the bedside.

There are three options for most patients with pleural effusions or pneumothoraces that need intervention:

- *Needle thoracentesis*. Used for first-time treatment of simple effusions or pneumothoraces with low likelihood of recurrence.
- *Pigtail chest drain*. A 16G tube inserted using modified Seldinger technique; good for simple effusions or pneumothoraces.
- *Chest tube*. Large-bore tube inserted, either blunt or using a trocar to treat tension pneumothorax, recurrent pneumothorax, haemothorax, or emphyema.

Indications

- Pneumothorax >2cm.
- Pleural effusion (unilateral) causing breathlessness.
- Pleural effusion (bilateral).
- Palliation of breathlessness in malignant pleural effusions.
- Pleural effusion (haemothorax, empyema, parapneumonic, malignancy, and chylothorax).
- Penetrating chest injury.
- Pleurodesis (for recurrent malignant effusion).
- Bronchopleural fistula.

Contraindications

- Coagulopathy.
- Local infection.

Preparation

- Explain to the patient the procedure and potential complications.
- Obtain written consent.
- Correct any coagulopathy.
- Recent CXR.
- Conscious sedation is desirable using short-acting opioid and benzodiazepine.
- Essential monitoring (ECG, HR, BP, SpO_2).

Vasovagal reactions can happen, so monitor patients appropriately. If sedation is required, then monitor patients according to the Association of Anaesthetists of Great Britain and Ireland (AAGBI) guidelines.

Equipment

- Ultrasound.
- Sterile conditions:
  - Sterile gown and gloves.
  - Sterile dressing pack.
  - Chlorhexidine or povidone iodine.
- Analgesia.
  - 1% lidocaine in 5mL syringe (orange and blue needles).
- Scalpel with blade, forceps, sutures (2/0 silk).
- Chest drain (size depending upon type of fluid).
- Closed drainage system.
- Sterile dressing.

Procedure

- Perform WHO safety check.
- Position the patient and choose the insertion site.
- In awake cooperative patients, position the patient in the semi-decubitus position on the bed at 45°, with the arm behind the head to expose the axillary area.
- Identify the 'safe triangle', which is delineated by the anterior border of the latissimus dorsi, the lateral border of the pectoralis major and a horizontal line at the level of the nipple (see Fig. 4.3).
- Use an ultrasound probe to identify pleural effusion, particularly in difficult cases or loculated pleural effusions.
- For pneumothorax, the second intercostal space in the mid-clavicular line is also suggested as an alternative, particularly in emergency situations, e.g. tension pneumothorax.
- Prepare the area of insertion with antiseptic solution and drape.
- Anaesthetize 2–3cm area of skin and subcutaneous tissue using 1% lidocaine. Inject lidocaine cutaneously, SC, and into the pleural space.
- Insert the Seldinger needle with a 10mL syringe attached in the anaesthetized area, aspirating as the needle is advanced. Once air or fluid is aspirated, insert it 0.5cm further and confirm aspiration of air or fluid.
- Remove the syringe; place a thumb over the open needle, and pass the Seldinger wire through the needle. Hold the needle in place all the time as you advance the wire.
- Remove the Seldinger needle over the wire.
- Make a 0.5cm skin incision (the scalpel sharp edge should always be facing away from the wire).
- Pass the Seldinger dilator over the wire; gently, but firmly, insert the dilator over the wire through the skin and intercostal muscles.
- Once dilated, remove the dilator and pass the chest drain over the wire.
- Attach the three-way tap over the drain and ensure it is closed.
- Secure the drain in place by suturing it to adjacent skin.
- Remove the cap of the drain and then attach it to chest tubing, which is then connected to an underwater seal drainage.

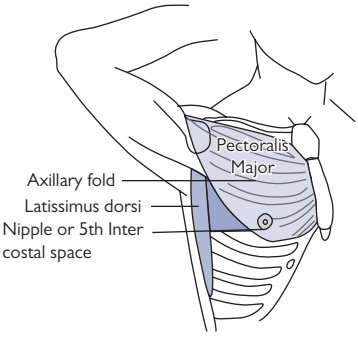


Fig. 4.3 Anatomy of the intercostal space.

Management of chest drains

Patients with a chest drain *in situ* should be managed in a clinical environment where nursing staff are competent in managing chest drains.

Immediate post-drain care

- Ensure the drain is secured and sutured. The chest drain bottle should be kept below the drain insertion site and upright all the time.
- Confirm the drain is swinging and whether there is active fluid drainage or bubbling.
- Obtain a CXR after insertion.

Types of drainage system

Underwater seal

- Suitable for any condition requiring pleural drainage.
- They can be used with or without suction.

Heimlich valves

The Heimlich valve is a one-way valve, which can be connected to a standard chest drain. It allows air and fluid out of the chest cavity but prevents both from going back.

Heimlich valves are usually considered in patients with a permanent air leak for whom surgery is not appropriate and for whom the main goal of therapy is discharge home or to palliative care.

Portable chest drainage system

- Designed as an ambulatory chest drainage system. It consists of a Heimlich valve within a drainage bag which has a capacity of about 1500mL and can be emptied intermittently. This cannot be connected to suction.

Suction

Most of the conditions can be safely managed by an underwater seal system without suction. However, if a patient cannot reinflate his/her own lung or a persistent air leak is preventing reinflation of the lung, high-volume, low-pressure thoracic suction in the range of 3–5kPa (20–40cmH_2O) can be used to help reinflate the acutely collapsed lung and improve drainage of fluid.

The use of suction may cause continuous bubbling from the tube, and swinging of fluid may not be visible.

Clamping drains

Temporary clamping of the drainage tube may be necessary when changing the drain bottle to prevent ingress of air into the pleural cavity. If a three-way tap is *in situ*, then this can be switched off.

Do not clamp a bubbling chest drain.

There are certain circumstances when a chest drain can be clamped, as below.

Post-pneumonectomy

The post-pneumonectomy chest drain is usually clamped for an hour at a time and unclamped briefly to allow blood to drain.

Massive haemothorax or effusion

In massive haemothorax, the effect is to attempt to tamponade the bleed, buying time to organize surgical exploration.

In massive pleural effusion, this allows time for the lung to expand without re-expansion pulmonary oedema and to reduce mediastinal shift caused by rapid drainage.

Decision-making in long-term drains

In patients with chronic pleural effusion or air leak, clamping is applied to see if a patient can manage without a drain. There is a risk of developing tension pneumothorax in patients with air leak. Such patients must be closely observed for any sign of respiratory or haemodynamic compromise or surgical emphysema. If the patient tolerates the clamp for 24h, it is usually possible to remove the drain.

Defibrillation and cardioversion

Key facts

Defibrillation is a non-synchronized random administration of shock during a cardiac cycle. Cardioversion is a synchronized administration of shock during the R waves or QRS complex of a cardiac cycle.

During defibrillation and cardioversion, electric current travels from the negative to the positive electrode by traversing the myocardium. This causes all of the heart cells to contract simultaneously, which interrupts and terminates the abnormal electrical rhythm. This, in turn, allows the sinus node to resume normal pacemaker activity.

Indications

Defibrillation

- Pulseless VT.
- VF.
- Cardiac arrest due to, or resulting in, VF.

Cardioversion

- SVT.
- AF.
- Atrial flutter.
- VT with pulse.

Anaesthesia

Defibrillation

Defibrillation is an emergent manoeuvre and is promptly performed.

Cardioversion

- Patient is adequately sedated/anaesthetized using short-acting agents (e.g. midazolam or propofol) and painkiller (fentanyl).
- Monitoring of O_2 saturation, ECG, HR, and BP is essential.
- Airway can be managed with bag and mask.
- Patient needs to be fasting for 6h for solids prior to elective cardioversion
- There is no patient preparation for emergency cardioversion.

Equipment

- Defibrillator (automated external defibrillator (AED) or standard defibrillator with monitor).
- ECG monitor with recorder.
- Defibrillator patch.
- Intubation kit.
- O_2 equipment, monitoring (pulse recorder, O_2 saturation monitor).
- IV access.
- Suction device.
- ALS medications.

Energy selection

Defibrillation

- VF or VT: 200J.

Cardioversion

- AF: 100–200J.
- Atrial flutter: 50–100J
- VT with pulse: 100J.

Complications of DC cardioversion

- Complications of GA.
- Systemic embolism if patient is not anticoagulated.
- Failure to cardiovert.
- Burns from incorrect placement of pads.
- Muscle pain from involuntary contraction.
- Arrhythmias, including asystole and VF.
- Pulmonary oedema.

Special populations

- Cardioversion in patients with digitalis toxicity.
- Cardioversion in patients with permanent pacemakers/ICDs.
- Cardioversion during pregnancy.

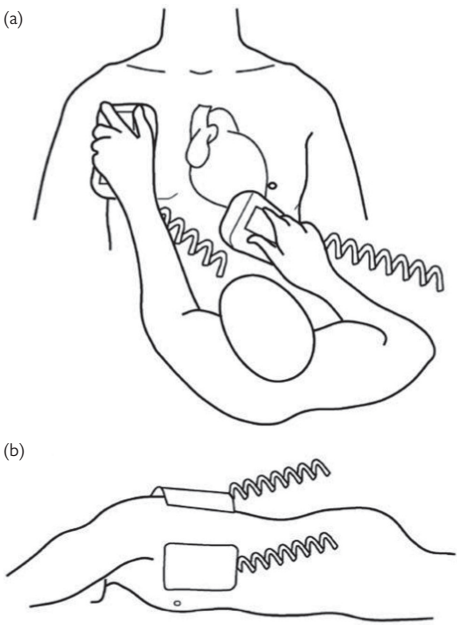


Fig. 4.4 Using defibrillators. (a) Correct positioning for defibrillation and cardioversion. (b) Alternative positioning for synchronized DC cardioversion.

Pericardiocentesis

Key facts

Pericardiocentesis is aspiration of fluid from the pericardial space that surrounds the heart. This can be a lifesaving procedure in patients with cardiac tamponade.

The detection of pericardial fluid is facilitated by echocardiography. With echocardiography, the location can be identified, the size can be estimated, and haemodynamic effects can be examined as well.

Emergency pericardiocentesis is performed in the presence of life-threatening haemodynamic changes in a patient with suspected cardiac tamponade.

Non-emergency pericardiocentesis is aspiration of pericardial fluid in haemodynamically stable patients for diagnostic, palliative, or prophylactic reasons. Performed under ultrasound or CT guidance.

Indications

- To determine the aetiology of an effusion.
- To relieve the symptoms of cardiac tamponade in an emergency.

Equipment

- Antiseptic solution, sterile drapes, gown, gloves, mask.
- LA (1% lidocaine), syringes, and needles.
- Pericardiocentesis kit.
- Ultrasound machine, sterile ultrasound probe cover.
- Spinal needle 18G, 90–120cm.

Preparation

- Explain the procedure to the patient.
- Ensure the patient has continual ECG monitoring.

Landmarks

- Identify the xiphoid process and fifth and sixth ribs. A common site for needle insertion is half a centimetre below and to the left of the xiphoid process along the sternocostal margin.
- Needle insertion at 45° to the skin, pointing towards the left shoulder (see Fig. 4.5).

Technique

- Position the patient in a semi-recumbent position at a 30–45° angle. This position brings the heart closer to the anterior chest wall.
- Ensure that the patient has at least one secured IV access line and is receiving supplemental O_2, and monitoring is in place (ECG, HR, O_2 saturation, BP).
- Prepare and drape the skin; infiltrate LA and puncture the skin using a No. 11 blade scalpel.
- Connect a 20mL syringe to the spinal needle and aspirate 5mL of saline in the syringe.
- Insert the spinal needle through the skin incision at 45° angle and direct it towards the left shoulder. Using ultrasound guidance, slowly advance the spinal needle up to a depth of 5cm,

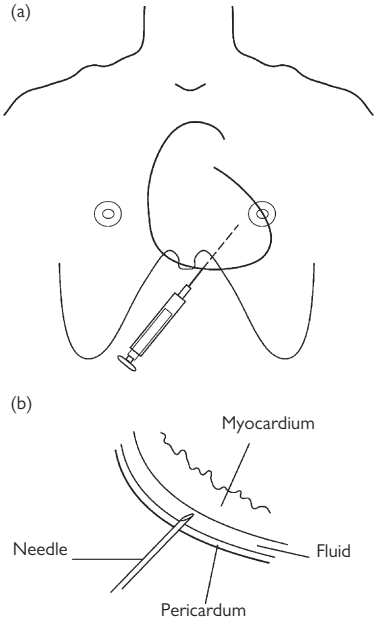


Fig. 4.5 Technique of pericardiocentesis. (a) Landmarks for needle. (b) Pericardiocentesis.

- If ECG waveform shows ST elevation (this suggests needle in direct contact with myocardium), withdraw the needle a few millimetres.
- Withdraw as much fluid as possible.
- Alternatively, you can use a pericardiocentesis catheter; using the same technique, once pericardial fluid is aspirated, withdraw the needle and use the catheter to aspirate.
- For recurrent pericardial effusion, use Seldinger technique to put a pericardial drain in place.

Complications

- Cardiac arrhythmias, cardiac tamponade.
- Arterial puncture (coronary, left internal mammary).
- Pneumopericardium, haemothorax, pneumothorax.
- Hepatic injury.
- False negative aspiration (clotted blood in the pericardium).
- False positive aspiration (intracardiac aspiration).
- Reaccumulation of pericardial effusion.
- Infection.

Nasogastric tube insertion

- NGTs are used to decompress the stomach and to administer enteral feeding and drugs in patients who cannot manage oral intake.
- Inadvertent placement of NGT into the bronchial tree can cause aspiration pneumonia or even respiratory arrest. Placement of a tube must always be confirmed.

Indications	Contraindications
Gastric emptying, e.g. bowel obstruction, ileus	Patient refusal
Enteral nutrition	Basal skull fracture or other facial trauma
Gastric lavage or aspiration after poisoning or drug overdose	Recent nasal surgery
Administration of medication	Oesophageal strictures
Administration of contrast for radiological investigation	Known oesophageal varices
• Upper GI bleed: • Evaluation (e.g. presence, volume, etc.) • Sengstaken–Blakemore tubes help in controlling variceal bleeds	Caution in unconscious patients and in patients with coagulopathy
Identifying oesophagus and/or stomach on CXR	Alkaline ingestion

Equipment

- NGT.
- Gloves.
- Lubricating gel.
- Lidocaine throat spray.
- Litmus paper.
- 50mL syringe.
- Glass of water with a straw if patient is able to swallow.
- NG collection bag.
- Tape to secure the tube.

Preparation

- Explain the procedure to the patient where appropriate and obtain verbal consent.
- Patients with swallowing problems should be assessed by a speech and language therapist.
- Lubricate the NGT with gel and pass horizontally along the nasal cavity, aiming towards the occiput.
- All NGTs must be confirmed to be in position by X-ray to exclude inadvertent bronchial intubation.
- Tape the tube securely to the nostril and attach the end to a bag.

Tips and pitfalls

- If the patient has problems swallowing only the NGT, ask the patient to swallow sips of water as the tube is passed.
- Constant coiling in the mouth. Tube may be soft—cool in the fridge.
- Resistance to passing. There may be an anatomical reason for this, e.g. oesophageal stricture. The tube may need to be passed under X-ray control.
- Tube migration. Just because the tube was in the correct position yesterday does not mean it is today; patients pull at these and work them out of the oesophagus with their tongue into a coil at the back of the throat. If called to assess, always look at the back of the pharynx ('Open your mouth and say Ah') and get a CXR.
- Aspiration of tube feeds. In the hypoxic or obtunded patient on NGT feeds, think of aspiration. Stop the feeds. Sit the patient up and give O_2. Assess the tube position. If you suspect aspiration (tube feeds visible in the mouth, coughing up feeds, tube in bronchus on CXR), call for senior help; the patient may need a bronchoscopy and/or intubation.

Nasal restraining loop (NRL)/nasogastric bridle

This is a method of preventing inadvertent displacement or removal of fine-bore NG feeding tubes in patients requiring external administration of feeds, fluid, or medication. It comprises a rigid probe and a flexible probe with a tape attached. The flexible probe has a removal guide wire. Each probe has a magnet at the end. The probes are inserted into each nostril until the magnets join at the back of the nostril, bringing the flexible probe and the loop of the tape around the back of the nasopharynx and exiting from each nostril. The tapes from each nostril are then secured to the NGT with a clamp, reducing the risk of inadvertent removal.

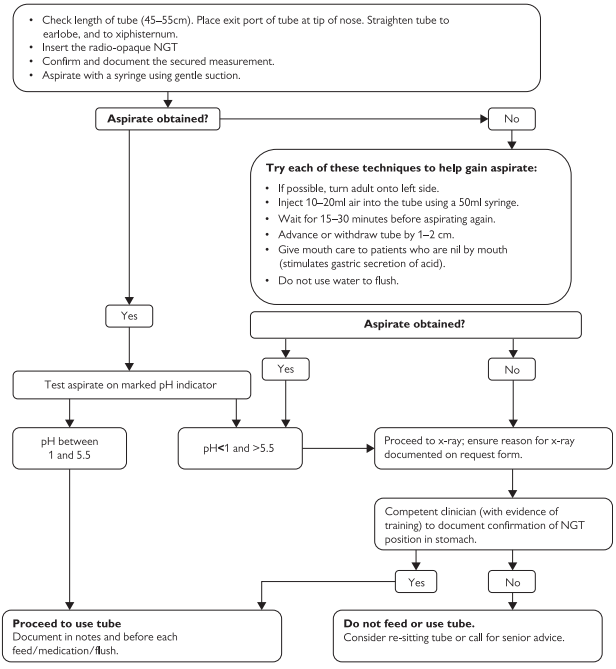
Considered in patients

- Who pulled out at least two NGTs.
- Who poorly tolerate NGTs for feeding and medicines.
- With post-endoscopically placed NJTs.

Flow chart for NGT placement checks in adults

- Check length of tube (45–55cm). Place exit port of tube at tip of nose. Straighten tube to earlobe and to xiphisternum.
- Insert the radio-opaque NGT.
- Confirm and document the secured measurement.
- Aspirate with a syringe using gentle suction.

Flowchart for NGT placement checks in adults



Urethral catheterization

Key facts

- Foley catheters are useful to monitor urine output hourly (renal failure, fluid balance) and in immobile patients.
- Catheterization of ♀ patients is usually performed by nursing staff; it is useful to learn the technique as you will be asked to try if they fail!

Indications

- Perioperative monitoring of urinary output.
- Acute urinary retention.
- Chronic urinary retention.
- Aid to abdominal or pelvic surgery.
- Incontinence.

Male catheterization

Equipment

- Foley catheter (size 12–20G, 14G most commonly used).
- Dressing/catheter pack containing drapes.
- Cleansing solution, sterile gloves (two pairs).
- Lidocaine gel.
- Gauze swabs, drainage bag, and/or universal specimen pot for midstream urine (MSU).

Preparation

- Consent the patient, explaining the procedure.
- Lay the patient supine.
- Expose the genital area and cover with a sterile drape with a hole in it.

Method

- Clean hands and put on sterile gloves.
- Pick up the glans penis with your non-dominant 'dirty' hand through the hole in the drape; the other hand will be your 'clean' hand.
- Holding a swab soaked in sterile saline with your clean hand, retract the foreskin and clean the urethral orifice and glans thoroughly so your gloved fingers only touch the swab, not the glans penis.
- Without letting go of the penis, discard the swab and pick up the sterile lidocaine gel with your clean hand and inject into the urethra.
- Still holding the penis in a vertical position, introduce the catheter with the clean hand and advance gently for ~10cm.
- Lower the penis to lie horizontally and advance the catheter fully (through the prostatic urethra) up to the hilt.
- Inflate the balloon now in the bladder via the smaller catheter channel with 10mL of sterile water; some catheters have an integral bulb of air which, when squeezed, inflates the balloon.
- NEVER inflate the balloon until the catheter is fully inserted as this risks inflating the balloon within the prostatic urethra, causing urethral rupture; ideally you should see urine before inflating the balloon.
- Attach a catheter bag firmly to the catheter.
- Replace the foreskin to avoid paraphimosis.

Tips and pitfalls

- *Difficulty identifying the urethral orifice*. Sometimes the orifice is located in the glans penis, e.g. hypospadias. If there is difficulty in retracting the foreskin, use plenty of gel.
- *No urine immediately*.
  - The bladder has just been emptied; insert a 2mL syringe into the end of the catheter and aspirate any residual urine.
  - The catheter tip may be blocked with lidocaine gel; try gently instilling 15–20mL of sterile water and gently aspirating.
- *Still no urine*. The patient may be anuric or a false passage may have been created; palpate to see if the bladder is empty or if you can feel the catheter balloon (which should not normally be palpable).
  - Treat anuria appropriately.
  - Consult a senior colleague if a false passage may have been created.
- *Inability to insert*. Try a smaller catheter or a silastic (firmer). If unsuccessful, ask a senior for help; suprapubic catheterization (SPC) may be needed (➲ Insertion of SPC, p. 280).
- *Decompression of grossly distended bladder*. Rapid decompression of a distended bladder (e.g. from chronic retention) may result in mucosal haemorrhage. Empty the bladder by 250–500mL every 30min until empty. Then monitor urine output closely as a brisk diuresis and dehydration may follow.
- *Bypassing catheter*. Usually due to catheter blockage. Check urine output, flush the catheter, and observe. If urine is flowing down the catheter and bypassing it, the catheter may be too small; try a slightly larger size.
- *Catheter stops draining*. The catheter may be kinked or blocked. Flush as above; if unsuccessful, try inserting a new catheter. Is the patient oliguric or anuric? Treat appropriately

Female catheterization

In many hospitals, ♂ are not allowed to catheterize awake ♀. Check before doing so and request a ♀ chaperone.

Equipment As for ♂ catheterization.

Preparation Lie patient on the back with knees bent. Ask the patient to place heels together and allow knees to fall apart as far as possible.

Method

A similar technique is employed here to ♂ catheterization, but note the following:

- Separate the labia minora with the left hand and ensure the whole genital area is adequately cleaned using the right hand.
- Identify the external urethral orifice. If this proves difficult in obese patients, an assistant may help by retracting the dependent fat from the pubic area.
- Lubricate the tip of the catheter with sterile water or lidocaine gel and pass gently into the urethra.

Tips and pitfalls

- *Difficulty identifying urethral orifice*. After warning the patient, place an index finger in the vagina to elevate the anterior vulva. Guide the catheter along the finger into the urethra.

Suprapubic catheterization

Indications

- Acute or chronic urinary retention—especially if urethral catheterization is difficult or potentially dangerous.
- Neurological disease such as multiple sclerosis and SCI.
- Urinary incontinence.
- Post-operative.
- Urethral trauma.
- Palliative use—especially in elderly frail patients, to enhance patient care and comfort.
- Urodynamic investigations.

Contraindications

- Carcinoma of the bladder.
- Patients on anticoagulation and antiplatelet treatment.
- Abdominal wall sepsis.
- Presence of subcutaneous vascular graft in the suprapubic region, e.g. femoro-femoral crossover graft.
- Clinical history taking must include previous abdominal surgery, especially to the suprapubic region and pelvis.
- Ultrasound may be used as an adjunct to SPC insertion.
- Informed consent must be obtained for all patients undergoing SPC insertion.
- SPC insertion can be performed under LA if adequate bladder distension can be achieved; if not, GA or regional anaesthesia is required.
- Antibiotic prophylaxis is recommended if urinary infection is suspected.
- Bladder aspiration with a 21G or smaller needle can be attempted to relieve symptomatic urinary retention temporarily.

Equipment

- Dressing pack.
- Gloves.
- Cleansing solution.
- Two 10mL syringes.
- 25G and 21G needles.
- 10mL 1% lidocaine.
- Prepacked SPC set (usually containing catheter, trocar, and scalpel).
- Catheter bag.

Insertion of SPC

- *Closed technique*. Bladder has to be adequately distended, in excess of 300mL. A variety of techniques include Seldinger's technique, trocar systems, or use of urethral sound.
- *Open technique*. In patients with a history of lower abdominal surgery and the bladder cannot be distended.

Preparation

- Explain the procedure and consent the patient.
- Lie patient supine and expose abdomen.
- Confirm clinically an enlarged, tense bladder.
- Identify catheterization site, 2cm (two finger breadths) above the symphysis pubis (see Fig. 4.6).

Method

- Clean the skin thoroughly around the site and apply drapes.
- Inject lidocaine into skin and subcutaneous tissues, injecting and aspirating in turn until urine is withdrawn.

'Nottingham' introducer (uses trocar)

- Make a 5mm incision at the identified site.
- Advance the catheter, with the trocar in place, through the incision and subcutaneous tissues. A 'give' will be felt as the bladder is entered.
- Withdraw the trocar and ensure that there is free flow of urine from the catheter.
- Inflate the catheter balloon and suture the flange of the catheter to the skin.
- Attach a catheter bag.

Bonanno (modified Seldinger's technique)

- Make a 5mm nick in the skin.
- Take the introducer needle and advance it, aspirating until urine is withdrawn.
- Remove the syringe and pass the guide wire down the needle into the bladder, then remove the needle, holding the guide wire in place.
- Pass the dilator firmly over the wire into the bladder.
- Remove the dilator and pass the catheter into the bladder, securing it as above.

Urethral sound

- Urethral sound is passed into the bladder and is manipulated to press onto the anterior abdominal wall.
- A cut-down is then performed to attach the catheter to the urethral sound.
- The sound is then withdrawn and the catheter is disconnected and pulled back into the bladder.

Tips and pitfalls

- *Bypassing urine*. With some types of catheter and trocar, urine may initially bypass the catheter. This will cease with full advancement of the catheter and decompression of the bladder.
- *No urine or faeculent matter in catheter*. Obtain help; you may have entered the peritoneum or bowel.

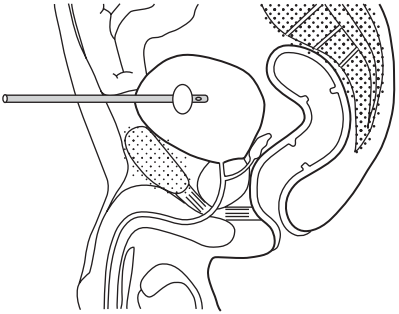


Fig. 4.6 Site of typical suprapubic catheter insertion.

Paracentesis abdominis

Key facts

This is a useful technique in some patients for the diagnosis and management of ascites, often in a patient with malignancy.

Indications

- Diagnostic evaluation of ascites:
  - Fluid for analysis.
- Therapeutic drainage of ascites:
  - To relieve physical symptoms.

Contraindications

- Bleeding disorders/DIC.
- Bowel obstructions/ileus.

Equipment

- Dressing pack.
- Gloves.
- Cleansing solution.
- 10mL syringe and 21G and 25G needles.
- 10mL of 1% lidocaine.
- 60mL syringe with 16G aspiration needle for diagnostic 'tap'.
- Bonanno catheter or paracentesis catheter, three-way tap, and collecting bag for therapeutic drainage.
- Specimen container if appropriate.
- Dressing.

Preparation

- Explain the procedure and consent the patient.
- Position the patient supine and expose the abdomen.
- Percuss and identify the position of ascites.
- Identify a suitable tap site; the RLQ is the commonest, with the patient turned semi-lateral to ensure the ascites fills this area (see Fig. 4.7).

Method

- Prepare the skin at the appropriate site and place sterile drapes.
- Infiltrate LA into the skin and subcutaneous tissues down to the peritoneum. Aspirate as the needle is advanced to avoid accidental vessel puncture.

Diagnostic tap

- Introduce the aspiration needle through the skin and subcutaneous tissues while aspirating. A 'give' should be felt and fluid freely aspirated as the peritoneal cavity is entered.
- Withdraw 15–20mL of fluid for diagnostic evaluation.
- Remove the aspiration needle carefully and apply an occlusive dressing.

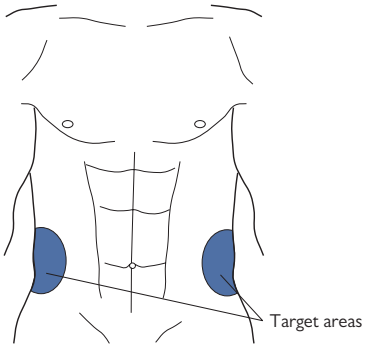


Fig. 4.7 Target areas for ascitic tap at the level of the umbilicus, 3–4cm lateral to the mid-inguinal line.

Therapeutic drainage

- Introduce the catheter into the abdominal wall until a 'give' is felt. Trial aspirate with a syringe to ensure ascites returned.
- Slide the catheter over the needle into the peritoneal cavity. Stop if resistance is encountered.
- Allow up to 1000mL of ascitic fluid to drain slowly over 1–2h.

Complications

- Persistent leak from needle site.
- Bowel or vessel injury.
- Bacterial peritonitis.
- Hypovolaemia.
- Electrolyte imbalance.

Tips and pitfalls

- *Unable to aspirate adequate quantity of fluid*. The ascites may be loculated. Drainage under ultrasound guidance may be helpful.
- *Blood or faeculent material*. Continual staining of the ascitic fluid with fresh blood or any staining with faeculent material may indicate puncture of a vessel or viscus. This is potentially serious; inform a senior colleague.
- *Peritoneal catheter*. Some patients who require repeated ascitic taps might benefit from placement of a temporary intraperitoneal catheter to allow daily drainage of ascites for symptomatic relief. There is a risk of peritonitis with these devices and only a short period of use is usually recommended, e.g. 2–3 days.
- The volume of ascites drained should be closely monitored, along with the patient's serum albumin and overall fluid balance. A maximum drainage of 2L/day is usually advised.

Rigid sigmoidoscopy

Key facts

This is a useful skill to learn. It is usually performed in the outpatient department as part of the investigation of lower GI complaints, but may have to be performed on the ward, e.g. acute admissions with rectal bleeding.

Indications

- Investigation of anorectal symptoms.
- Visualization of the rectum.

Equipment

- Rigid sigmoidoscope with obturator and light source.
- Lubricating jelly.
- Gloves.
- Gauze swabs.

Preparation

- Explain the procedure and consent the patient.
- Position the patient in the left lateral position, with the hips flexed as fully as possible and knees partially extended.
- Carry out a digital examination of the rectum to identify low-placed lesions or faecal loading, which may prevent safe insertion or obscure a useful view.

Method

- Lubricate the sigmoidoscope with jelly.
- With the obturator in place, introduce the scope gently through the anal sphincter in the direction of the umbilicus for ~5cm.
- Remove the obturator; attach light source, insufflator, and eyepiece.
- Introduce small amounts of air to open up the lumen.
- Advance the instrument slowly under direct vision, ensuring that a patent lumen is identified prior to advancing the scope further.
- Note the appearance of the mucosa and the presence of any mucosal lesions. The level of any lesion should also be noted using the marked scale on the outer casing of the sigmoidoscope.
- If the patient experiences significant discomfort, do not persist.
- Withdraw the scope slowly, again under direct vision.
- Clean the area around the patient's anus.

Tips and pitfalls

- Biopsy. Unless experienced in the skill, do not attempt biopsy of lesions. Note and document their position and inform a senior colleague.
- Unable to see the upper rectum. Remember that the rectum has a sacral curvature, often pronounced in women; GENTLY use the tip of the scope as a 'lever' to push the anterior wall of the rectum forward to open to the lumen. If this is not easy and painless, do not persist; it may represent pathology.
- Recto-sigmoid junction. Negotiation of the recto-sigmoid junction can be difficult. The best view that can be hoped for is to see the last sigmoid fold above the junction. Do not attempt to pass the scope into the distal sigmoid; this is the role of flexible sigmoidoscopy.

Key revision points—anatomy of the rectum

- The rectum is said to start at the level of S2, but a distance of 15cm from the anorectal junction is used to define pathology which is termed 'rectal'.
- The rectum has two main angles:
  - The first is the acute anorectal angle which slopes posteriorly and is formed, in part, by the pull of the sling of the levator ani.
  - The second is the sacral curvature which runs throughout the rectum, sloping progressively anteriorly up to the level of the recto-sigmoid junction.
- Three 'lateral valves' are commonly described but are only the mucosal folds of the rectum equivalent to the colonic folds.
- The peritoneal-lined 'pouch of Douglas' (or recto-vesical pouch in ♂) extends a variable distance down the anterior wall of the rectum. Its contents (e.g. sigmoid colon) may be easily palpable, particularly in elderly ♀.
- The upper third is covered by the peritoneum anterolaterally, the middle third just anteriorly, and the lower third is entirely extraperitoneal.
- The rectum has a complete outer longitudinal muscle coat (thus, diverticular disease does not occur in the rectum).
- The rectum and associated mesorectal fat, blood vessels, and lymph nodes are enclosed and separated from the 'true' pelvic organs by a fascial sheet—the mesorectum.

Local anaesthesia

An LA is a drug that causes reversible LA and a loss in nociception. LA is used in a variety of settings and is easy to deliver.

LA can be esters (cocaine, procaine, tetracaine, etc.) or amides (bupivacaine, levobupivacaine, prilocaine, etc.)

Pharmacokinetics

- *Absorption*: absorption of LAs into the systemic circulation varies, depending on the site of injection, characteristics of the agent, and presence of added vasoconstrictor.
- *Metabolism*: esters (cocaine, procaine, tetracaine, etc.) are metabolized rapidly by plasma esterase to inactivated compounds. Amides (bupivacaine, levobupivacaine, prilocaine, etc.) are metabolized in the liver. Their metabolism is slower, hence more prone to accumulation, especially in the case of liver dysfunction.

Mechanism of action

LAs act by blocking the Na^+ channel in the nerve membrane, preventing propagation of the action potential.

Small, non-myelinated pain fibres are blocked first, and large, myelinated fibres that conduct impulses from pressure senses are the last to be blocked.

LAs are generally ineffective when used to anaesthetize infected tissue.

Common agents

Lidocaine

- Lidocaine is a common LA with anti-arrhythmic properties.
- Concentrations: 0.5%, 1%, and 2%. Solution can be plain or contain adrenaline.
- Duration of action: rapid onset (2–3min) and lasts 30–90min.

Clinical usage

- Skin infiltration.
- Bier's block or IV regional anaesthesia (IVRA): 40mL of 0.5% lidocaine (without adrenaline).
- Peripheral nerve blocks.
- Dental blocks.
- Topical anaesthesia.
- Subarachnoid block (spinal anaesthesia).

Recommended maximum safe doses of lidocaine are as follows

- Lidocaine without adrenaline (plain solution): 3mg/kg.
- Lidocaine with adrenaline: 7mg/kg as systemic absorption is much slower.

Bupivacaine

Bupivacaine has been in clinical practice for >40y. It has a slower onset with a longer duration of action. Available as 0.25% and 0.5% solution (with or without adrenaline), and a special preparation (heavy bupivacaine) 0.5% with 6% glucose is available for use in spinal anaesthesia.

Bupivacaine may be administered by infiltration, intrathecally, and epidurally. Commonly used for epidural infusions (with or without opioid) for labour analgesia and post-operatively.

Because of its potential for cardiovascular toxicity, bupivacaine is contra-indicated in IVRA.

Recommended maximum safe doses of bupivacaine are as follows:

- Bupivacaine without adrenaline (plain solution): 2.0mg/kg.
- Bupivacaine with adrenaline: 2.5mg/kg.

Levobupivacaine

- Levobupivacaine is a relatively new agent. Compared with bupivacaine, it is said to have greater vasoconstrictive action and less motor block. The real advantage is that it is apparently less cardiotoxic.
- Maximum safe dose: 2.5–3.0mg/kg.

Toxicity

LAs may be toxic if sufficient amounts are absorbed into the systemic circulation. Of these, bupivacaine appears to be the most dangerous. Features of toxicity usually occur at a peak of 10–25min after SC injection but occur immediately with IV injection.

Signs and symptoms

- *Neurological*. Drowsiness, confusion, slurred speech, light-headedness, tingling of lips, tongue, or mouth; tinnitus, slurred speech, seizures, reduced level of consciousness, and coma.
- *Cardiovascular*. Early tachycardia and HTN; late bradycardia, hypotension, cardiac arrhythmias, reduced myocardial contractility, and cardiac arrest. In the case of bupivacaine, the cardiac effects are particularly difficult to treat.

Treatment

- Stop procedure.
- Maintain airway and provide O_2.
- Ensure IV access.
- Monitor patient's ECG, BP, HR, and O_2 saturation.
- *Convulsions*. Diazepam 5–10mg IV.
- *Hypotension*. IV fluids, inotropes.
- *Bradycardia*. Usually resolves; atropine is rarely required.
- *Severe toxicity*. Intralipid® 20% IV in cases of refractory toxicity or cardiac arrest.

Regional anaesthesia

Below is a brief description of some regional anaesthesia techniques commonly used either as sole anaesthetic technique or to supplement GA.

Intercostal nerve block

Indications Pain due to fractured ribs, post-thoracotomy pain relief.

Preparation Ensure all equipment required is available (20mL syringe, needle, LA for injection, e.g. bupivacaine).

The patient is positioned as for pleural aspiration and the site for infiltration is identified:

- *Broken ribs*: medial to the site of fracture on the posterior aspect of the chest wall.
- *Post-thoracotomy*: medial to the posterior edge of the scar on the posterior chest wall.

Method Ensure that the skin is prepared thoroughly with antiseptic solution and drapes are placed properly.

Insert the needle with syringe containing LA through the skin, just inferior to the rib associated with the nerve to be blocked.

Aspirate the syringe to ensure that the needle has not entered a blood vessel or the pleural space. After negative aspiration, the site is infiltrated with 4–5mL of LA. Repeat this at various sites.

Obtain a CXR post-procedure to rule out pneumothorax.

Spinal anaesthesia

Spinal (subarachnoid) anaesthesia is a safe and effective alternative to GA when the surgical site is located on the lower extremities (hip or knee arthroplasty), perineum, or lower body (inguinal hernia, Caesarean section). Spinal anaesthesia produces intense sensory and motor blockade. It is achieved by instilling LA into the CSF at the lumbar level below the spinal cord.

Contraindications include patient refusal, lack of patient cooperation, difficulty in positioning, raised ICP, allergy to LA, coagulation disturbances, fixed CO (aortic stenosis), bacteraemia, and infection at the site of needle insertion.

Epidural

Epidural anaesthesia is performed as sole anaesthesia or in combination with spinal anaesthesia or GA. The special epidural needle is placed in the epidural space using loss of resistance technique. Epidural injection can be performed from cervical to sacral spine (caudal analgesia).

Paravertebral block

Paravertebral block is performed by placement of LA around the nerve bundles as they arise from their corresponding intervertebral foramina.

Paravertebral block provides analgesia for chest wall incisions (thoracotomy, breast surgery).

Transversus abdominis plane block

TAP block is used to provide analgesia to the anterior and lateral abdominal wall. Mid-/lower thoracic and upper lumbar spinal nerves travel in the fascial plane between the transversus abdominis and the internal oblique muscles. TAP block is an adjunctive technique for analgesia.

Indications Abdominal surgery (laparotomy, hernia repair, open appendicectomy, Caesarean section, abdominal hysterectomy, etc.).

Techniques

- *Landmark technique*— accesses the TAP via the lumbar triangle of Petit (bound by the external oblique anteriorly, the latissimus dorsi posteriorly, and the iliac crest inferiorly) (see Fig. 4.8). After piercing the skin, the needle is advanced until a pop is felt (fascial extension of the external oblique). The needle should be further advanced until a second pop is felt (fascial extension of the internal oblique). The needle should now lie superficial to the transversus abdominis muscle in the TAP. After aspiration, the LA is injected—20mL on each side, keeping in mind not to exceed the maximum safe dose of LA.

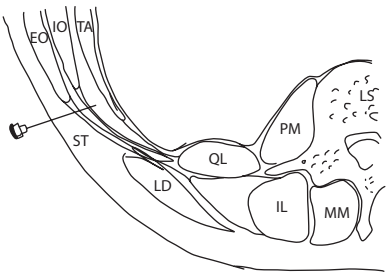


Fig. 4.8 Diagram of transverse section of abdominal wall during landmark TAP block (N, Needle; ST, subcutaneous tissue; EO, external oblique; IO, internal oblique; TA, transversus abdominis; LD, latissimus dorsi; QL, quadratus lumborum; LS, lumbar spine; PM, psoas major; MM, multifidus muscle; IL, Longissimus, Iliocostalis),

- *Ultrasound-guided technique*: high-frequency linear probe is used to identify abdominal muscle. Moving the probe from close to the midline (identifying the rectus abdominis) laterally between the iliac crest and the costal margin, the three layers of muscles can be identified (external oblique, internal oblique, and transverse abdominis).

Brachial plexus block

Common approaches to the brachial plexus are interscalene, supraclavicular, infraclavicular, and axillary.

Interscalene approach is the technique of choice for proximal surgery, including shoulder (see Fig. 4.9).

Supraclavicular approach provides the most reliable anaesthesia of the entire arm. The injection is made at the level of the trunks. It also has the highest incidence of pneumothorax.

Infraclavicular approach provides anaesthesia to the forearm, wrist, and hand; the risk of pneumothorax is less than with the supraclavicular approach.

Axillary approach is the most commonly used and easy to perform and has few side effects. Suitable for surgery to the forearm, wrist, and hand. Injection is made at the terminal branches. There is a risk of developing haematoma and intravascular injection.

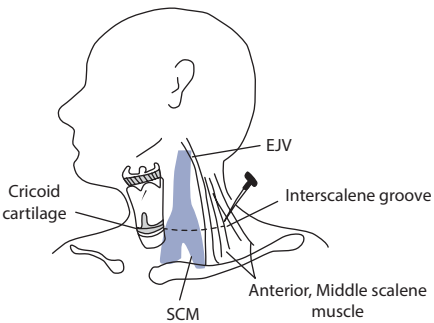


Fig. 4.9 Interscalene brachial plexus block.

Intravenous regional anaesthesia (Bier's block)

Regional anaesthesia can be achieved by injecting LA IV distal to a tourniquet. A reliable and simple technique of providing anaesthesia for minor procedures to the extremities of <1h duration.

- 0.5% prilocaine is the drug of choice (3–5mg/kg); an alternative is 0.5% lidocaine (3mg/kg).
- *Caution*: risk of LA toxicity in cases of tourniquet failure, local infection.
- Contraindicated in patients with sickle cell anaemia, allergy to LA, and peripheral vascular disease, and in cases of patient refusal.

Chapter 5

Head and neck surgery

Thyroglossal cyst, sinus, and fistula

Key facts

- A thyroglossal cyst is a fluid-filled sac resulting from incomplete closure of the thyroglossal duct.
- A thyroglossal sinus results from persistence of the whole duct.
- Incidence <1%; ♂:♀, 1:1.

Anatomy (see Fig. 5.1)

The thyroglossal duct arises embryologically between the first and second pharyngeal pouches. It runs as a hollow tube from the foramen caecum on the dorsal surface of the tongue, becoming a solid cord of cells migrating through the tongue and into the midline of the neck. The tract usually passes in front of the hyoid bone and then loops up behind it before descending in the midline of the neck where the cells divide to form the two lobes of the thyroid gland on either side of the midline. The duct normally atrophies in the sixth week of gestation.

Clinical features

- Usually presents in children or young adults.
- Ninety per cent present as a painless midline cyst.
- Ten per cent appear on one side of the midline, usually the left.
- Seventy-five per cent appear in front of the hyoid bone and most of the rest at any point to the root of the neck.
- The cyst elevates on protruding the tongue if attached to the hyoid or elevates on swallowing if attached to the isthmus of the thyroid.
- Five per cent become infected, presenting as a painful, red neck swelling.
- Fifteen per cent have a fistula to the skin (due to infection or incomplete excision).
- Papillary carcinoma of the thyroglossal ductal cells is rare (about 1%). Treatment is by excision.

Diagnosis and investigations

- USS is the investigation of choice.
- CT scan will often reveal a well-circumscribed cyst related to the midline of the hyoid bone.
- Fine needle aspiration (FNA) may reveal a cloudy infected fluid or a straw-coloured fluid.

Treatment

Infected thyroglossal cyst

- Most respond to antibiotics.
- Surgical drainage if abscess persists or failure to respond to antibiotics.
- Elective excision of the cyst once the acute infection has resolved.

Surgery

- Excision is recommended for most cysts.
- Remove through a transverse midline incision in a skin crease.
- Divide the platysma muscle and excise the cyst using sharp and blunt dissection.
- On the deep surface, it is attached to the hyoid bone; excise ~1cm of the bone in the midline, removing any underlying thyroglossal duct epithelium. This is the Sistrunk's procedure.
- Close the wound in layers, with a suction drain if needed.
- If there is a fistula or sinus in the neck, excise it through a transverse elliptical incision. Again use blunt dissection and remove the middle part of the hyoid bone.

Complications These are usually very few. Remove the drain and discharge the patient—same day or next day.

The important structures that must be considered when operating on the thyroid gland include:

- Recurrent laryngeal nerve.
- Superior laryngeal nerve.
- Parathyroid glands.
- Trachea.
- Common carotid artery.
- IJV (not depicted).

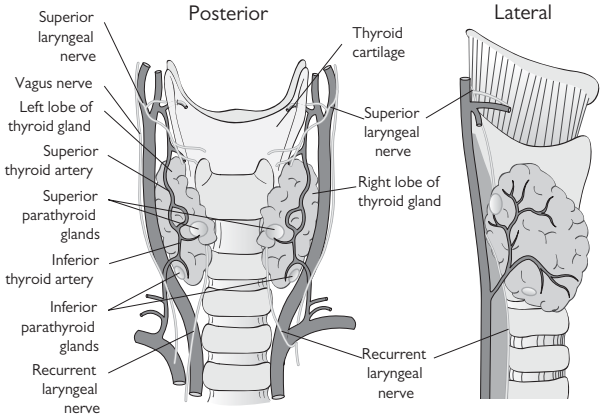


Fig. 5.1 The anatomy of the region of the thyroid gland.

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Branchial cyst, sinus, and fistula

Key facts

- Disputed aetiology. Theories include:
  - Cystic degeneration of epithelial derivatives of the first, second, or third branchial clefts.
  - Cystic degeneration of epithelial elements in a cervical lymph node.
- A branchial fistula is a tract running from the neck skin through to the posterior pillar of the fauces; these are very rare.
- A branchial sinus occurs when the lower part of this tract remains open on to the neck skin surface.
- A branchial abscess is an infected branchial cyst.

Clinical features

- Presents as a neck lump, usually painless.
- They typically present in early adulthood.
- Sixty to seventy per cent are anterior to the upper third of the sternomastoid muscle, with the posterior border lying beneath the sternomastoid. Other sites include:
  - Parotid gland.
  - Anterior to the lower two-thirds of the sternomastoid.
  - Anterior to the pharynx.
  - In the posterior triangle of the neck.
- Two-thirds occur on the left side; 2% are bilateral.
- May present with an acute branchial cyst abscess causing pain, ↑ swelling, and occasionally pressure symptoms (difficulty swallowing or breathing).

Diagnosis and investigations

For branchial cyst or abscess

- USS is the first investigation of choice. CT/MRI for complex cases.
- Fine needle aspiration biopsy (FNAB):
  - Abscesses. Purulent fluid is obtained that may culture organisms.
  - Cysts. Straw-coloured fluid containing cholesterol crystals.

Treatment

Branchial abscess

- Drain via a transverse incision in the neck at the point of maximum convexity.
- Suture a Yeates-type drain.
- Give antibiotics and make no attempt to remove the cyst until the infection has resolved completely.

Branchial cyst

- Most cysts are excised to achieve a diagnosis and prevent symptoms or complications.
- Make a transverse incision over the cyst, preferably in a transverse skin crease, long enough to match the size of the cyst.
- Divide the platysma and the deep fascia over the anterior border of the sternomastoid and retract the muscle posteriorly.
- Remove the cyst, usually by blunt/sharp dissection.

- Use suction drainage and close the wound in layers.
- If the cystic lesion is in the parotid gland and cannot be distinguished from any other parotid lesion, extend a preauricular incision into the neck as for a superficial parotidectomy.

Branchial fistula

- Excise a sinus or fistula through a horizontal elliptical incision around the neck opening.
- Blunt and sharp dissection of sinus tract as far as possible.
- If the upper end of the tract cannot be reached, make a further transverse incision at a higher level ('stepladder' incisions).
- Sometimes the tract runs between the internal and external carotid arteries and sometimes up to the pharyngeal wall in the region of the middle constrictor.
- Close the wounds in layers with suction drainage.

Complications

A branchial cyst at any site often lies near important nerves. Previous infections causing fibrosis will increase the risk of damaging them. The following nerves are at risk:

- Hypoglossal nerve (tongue deviates to affected side on protrusion).
- Mandibular branch of the facial nerve (movement of lower lip).
- Great auricular nerve (numb ear).
- Accessory nerve (paralysis of trapezius: weakness of arm abduction, asymmetry, and chronic pain).

Salivary calculi

Key facts

- Occur in up to 1% of general population, but asymptomatic in half of these.
- Salivary gland calculi occur most commonly within the submandibular ductal tree (80%), and 20% in the parotid.
- Composed of calcium phosphate and carbonate; may be related to sialadenitis (inflammation of a salivary gland).
- Commonest in adults. Commonest between third and sixth decade.

Clinical features

- Pain and swelling of the affected gland on eating and drinking ('mealtime syndrome').
- If there is partial obstruction of the duct, the swelling can last minutes to several hours.
- Complete obstruction leads to persistent swelling and infection.
- The patient may also experience colicky pain in the duct when eating.

Points in the examination of the submandibular gland

- Examine the gland from behind and feel the swelling by running the finger backwards under the jaw. If you cannot feel a lump, ask the patient to suck a sour sweet and re-examine them.
- Examine the duct orifice from the front. Ask the patient to open their mouth wide and point their tongue upwards. The ducts lie near the midline at the root of the tongue. Are they red? Is there pus? Can you see an impacted stone?
- Examine the gland bimanually from the front. Wear gloves and place the finger of one hand over the gland. The index of the other hand is placed in the mucosal surface of the mandible and the gland palpated between the two.

Diagnosis and investigations

- Radiographs of the submandibular gland, parotid gland, and ducts are helpful. Twenty per cent of submandibular calculi and 80% of parotid calculi are radiolucent.
  - A lower occlusal X-ray of the teeth will show a stone in the distal portion of submandibular duct.
  - A lateral oblique X-ray or orthopantomogram (OPT) of the mandible will show a calculus in the submandibular gland.
- Submandibular duct radiography (sialography) is technically difficult and rarely done.
- Parotid sialography may show a filling defect. Sialectasis is often seen. May provide therapeutic benefit due to flushing out of debris in the ductal tree.
- USS of parotid and submandibular glands is often the choice of investigation by head and neck radiologists.

Treatment

- Stones in the intra-oral part of the ducts can be removed under LA. Steady the stone with Babcock's forceps and incise directly over it. Remove the stone; leave the duct marsupialized.
- Stones within the submandibular gland require removal of the gland itself.
- Removal of a calculus from the parotid gland is a rare operation. Most calculi are at the distal end of the parotid duct (as it does an 'S' bend through the buccinator muscle) and can be released by intra-oral incision of the parotid duct papilla.
- Most parotid gland obstructive/inflammatory disease is treated conservatively with sialogogues and intermittent massage of the gland towards the duct. Duct dilatation using lacrimal probes is useful as most strictures/obstruction occur at the 'S' portion noted above.

Key revision points—anatomy and physiology of the salivary glands

- The salivary glands produce: saliva-containing water; electrolytes, especially potassium (K+) and bicarbonate (HCO3–); and varying amounts of mucus and enzymes.
- The parotid is a pure serous gland. It responds to salivary stimuli, e.g. food in the mouth, smell. There is little resting flow. The submandibular gland is mixed with serous and mucous acini, responds to salivary stimuli, and has a resting flow, which contributes, along with sublingual and minor glands, to maintaining mouth moisture.
- Saliva functions to lubricate, aid mastication, aid taste, suppress oral bacteria, and initiate starch digestion.
- The submandibular duct is palpable in the floor of the mouth and enters the mouth from the gland on the sublingual papilla near the midline.
- The parotid duct is palpable over the anterior border of the masseter and enters the mouth on the medial wall of the cheek after passing through the buccinator muscle via an 'S' bend.
- The facial nerve trunk lies between the deep and superficial parts of the parotid gland and divides into five branches (pes anserinus) within the superficial portion.

Acute parotitis

Key facts

- Parotitis is inflammation of the parotid gland. Causes include:
  - Acute or chronic obstruction (now the commonest cause).
  - Bacterial (ascending parotitis)—less common.
  - Viral infection, e.g. paramyxovirus (mumps), HIV.
  - Inflammatory disorders, e.g. Sjögren's syndrome, sarcoidosis.
  - Any cause of inflammation of lymph nodes within the parotid gland.
- Most patients develop this condition as an acute episode of a chronic obstructive sialadenitis.

Clinical features

- Obstructive parotitis occurs more commonly in adults.
- Presents as an acutely painful preauricular swelling.
- There is often a history of recurrent, intermittent swelling of the gland.
- The gland is usually tender on palpation.
- The patient may be toxic with fever and a raised WCC, and pus may exude from the opening of the parotid duct opposite the crown of the second upper molar tooth.
- Elderly, debilitated, dehydrated patients with poor oral hygiene or who are on anticholinergic drugs are at greatest risk.

Diagnosis and investigations

- Plain X-rays to determine whether radio-opaque calculi are present in the duct or gland.
- USS or CT scanning may help differentiate between stones, inflammation, and tumour.
- If pus is present, take a bacteriology swab and send it to the lab. The commonest infecting organism is Staphylococcus aureus.

Treatment

Acute parotitis

- Most patients respond to antibiotics:
  - Give amoxicillin 500mg tds, IV if necessary.
  - Rehydrate dehydrated and debilitated patients.
  - Good oral nursing care with chlorhexidine mouth rinses.
- Review patients by clinical examination after the infection has subsided to make sure that the obstruction was not due to a parotid tumour.
- If a parotid abscess develops, it should be drained surgically:
  - Make an incision over the abscess under GA where it appears to be pointing, parallel to the branches of the facial nerve to avoid damaging them.
  - Open the abscess with sinus forceps and place a Yeates drain in the wound.

Recurrent parotitis

- Teach patients with recurrent parotitis to massage the gland in order to express saliva from the duct.
- Dilatation of the duct with lacrimal probes can assist drainage.
- Remove radio-opaque calculi, if possible.
- Advise the patient to keep an emergency supply of antibiotics at home.
- If recurrent parotitis persists for months or years, a total parotidectomy is curative.

Salivary gland tumours

Key facts Salivary gland tumours are rare, accounting for 0.4% of all malignant tumours; 80% arise in the parotid gland.

Clinical features Most patients present with a slow-growing lump in the affected gland. Pain, paraesthesiae (e.g. lingual nerve in the submandibular gland), and facial palsy (parotid gland) imply malignancy. Salivary tumours of minor glands in the upper aerodigestive tract (UADT) present as a lump. Fifty per cent of these are malignant.

Clinicopathological features

Pleomorphic adenoma

- Eighty per cent of benign parotid tumours; ♂:♀, 1:1.
- Peak incidence 30–50y.
- Composed of epithelial and mesothelial cells that form a mucous matrix, often with chondromatous components.
- The tumour grows slowly and has no true capsule, so these strands of tumour cells protrude into normal surrounding tissue. Hence there is a high rate of recurrence of up to 50%.
- Malignant change (adenocarcinoma) occurs in 20% after 10y and is seen in asymptomatic deep lobe parotid tumours.

Warthin's tumour (adenolymphoma)

- Usually affects men >50y; 10% are bilateral.
- Strong association with smokers.
- Benign and presents as a slow-growing, soft swelling.
- Very low malignant potential.
- Successfully treated by wide local excision.

Malignant tumours

Mucoepidermoid tumour

- Occurs typically in 30–50y.
- Low-grade malignancy, though variable behaviour.
- Most grow slowly, invading locally and eventually metastasizing to neck lymph nodes, the lung, and the skin.

Adenoid cystic carcinoma

- A slow-growing, locally aggressive, malignant tumour with indolent behaviour.
- Perineural invasion propensity with facial palsy common, with extension through the stylomastoid foramen. Lung metastasis common.
- Often regarded as incurable, but individuals can lead a normal life over 20–30y before succumbing.

Acinic cell carcinoma

- ♀ > ♂; slow-growing and rare, but may metastasize unexpectedly. Surgery is the treatment of choice.

Squamous cell carcinoma, adenocarcinomas, and undifferentiated carcinomas

- Generally high-grade malignant tumours.
- Often rapid local invasion into extraparotid tissues and infratemporal fossa, leading to pain and trismus.
- There may be skin fixation or ulceration with facial nerve palsy and invasion of the external auditory canal; incurable; palliative radiotherapy.

Diagnosis and investigations

- Clinical examination is still of great importance in assessing extent.
- CT scanning may help differentiate between stones, inflammation, and tumour.
- MRI scanning offers the most sensitive investigation for assessment of local invasion and involvement of surrounding structures.
- Positron emission tomography (PET) CT is useful for assessing metastases.

Treatment

Benign parotid tumours

- Excise the parotid gland superficial to the facial nerve (superficial parotidectomy). Deep lobe tumours should have a facial nerve-sparing total parotidectomy.
- Enucleation is inadequate and often leads to local recurrence that is difficult to manage.

Benign tumours in other salivary glands

- Excision of the entire gland (e.g. simple submandibulectomy).

Malignant tumours

- Radical local excision (to sacrifice or preserve the facial nerve in parotid tumours is controversial).
- May be accompanied by neck dissection, especially in parotid tumours.

Complications of parotid surgery

- Facial nerve injury (risk varies according to procedure: lowest in primary surgery for benign tumours < redo surgery < surgery for malignancy). Seventy-five per cent neurapraxia with complete or extensive recovery of function; 25% neurolysis with little or no recovery (may be treated by nerve interposition grafting).
- Frey's syndrome:
  - Late complication of surgery in up to 25% of patients.
  - Facial flushing and sweating of the skin innervated by the auriculotemporal nerve when the patient salivates.
  - Caused in this case by division of the parasympathetic secretomotor fibres that innervate the parotid gland; they may regenerate erratically to control cutaneous secretomotor functions.
  - SC Botox injection is useful.

Prognosis

- Recurrence of benign tumours. May develop 20y after surgery, especially in the patient where enucleation, rather than superficial parotidectomy, has been performed.
- Five-year survival rate for all malignancies ~60%.

Head and neck cancer

Key facts

- Head and neck cancer refers to cancer of the UADT; 90% are SCCs.
- UK incidence 8–15 in 100 000 and rising. Wide geographical variation, e.g. Indian subcontinent: 40% of all cancers. ♂:♀, 2:1; ♀ incidence rising.
- Predisposing factors:
  - Carcinogens. Tobacco, alcohol, betel nut chewing.
  - Infection. Hyperplastic candidiasis, HPV 16.
  - Extrinsic factors. UV light in lip cancer.
  - Intrinsic factors. Diet poor in fruit, vegetables, and fish oils; immunodeficiency/suppression.

Clinical features

- Peak incidence 40+y (increasing incidence in younger patients).
- Persistent oral ulcer with induration, bleeding, often painful.
- Persistent oral swelling, e.g. large tonsil, unexplained loose teeth.
- Unexplained earache: common in tongue, oropharyngeal tumours.
- Dysphagia and odynophagia occur in oro-/hypopharyngeal cancer.
- Hoarseness lasting >3 weeks.
- Persistent unilateral serosanguineous nasal discharge.
- Unresolved head or neck swellings of >3 weeks.
- Examination of the neck is mandatory and should include all levels of neck lymph nodes. Bilateral nodal spread common.
- Six per cent of patients have a synchronous SCC present in the aerodigestive tract (mouth, larynx, lungs, oesophagus).

Diagnosis, investigations, staging, and assessment

- Fibreoptic nasendoscopy to examine the nasopharynx, base of the tongue, hypopharynx, and larynx.
- Fine needle cytology for neck mass.
- Imaging. CT of head and neck and chest, with MRI in selected cases. PET CT for unknown 1° tumours and metastatic disease assessment.
- Haematology, biochemistry, ECG, and lung function tests as patients usually have high comorbidities.
- EUA. Measure tumour size, biopsy. Panendoscopy to exclude synchronous tumours of the UADT.
- Extraction of any diseased teeth, especially if in possible radiotherapy treatment field, to prevent osteoradionecrosis.
- All patients should be seen by a dietician, speech and language therapist, clinical nurse specialist, and restorative dentist.

- In the tumour, nodes, metastasis (TNM) system, T1–4 stage is complex and depends on the anatomical site; N1–3 stage applies to all sites.

Treatment

- Surgery, radiotherapy ± chemotherapy, or a combination of all, and may be done with curative intent or palliation.
- Function and quality of life are important outcomes. Gastrostomy/NGT feeding often required during treatment.

Treatment of primary tumour

- Approximately equal cure rate for T1 and T2 tumours with surgery or primary radiotherapy. Surgery is usually offered for oral cancer, sometimes for T1 larynx (laser surgery). Radiotherapy ± chemotherapy have better functional outcome in pharyngeal and posterior one-third tongue cancers.
- Larger T3 and T4 tumours involving bone/cartilage are best managed surgically, e.g. laryngectomy, and often require adjuvant radiotherapy.
- Monoclonal antibody treatment (cetuximab) is licensed for the treatment of locally advanced SCC of the head and neck ± radiotherapy.

Treatment of the neck

- N0 necks may have occult nodal metastases, depending on tumour site, e.g. >50% for pharynx, and should have either a selective neck dissection or radiotherapy.
- Single node disease (N1) should have either a neck dissection or radical radiotherapy.
- Bulky nodal disease (N2, N3) should have a comprehensive neck dissection followed by radiotherapy, or vice versa.

Neck dissections

These are either comprehensive or selective. Selective dissection removes groups of nodes likely to have occult metastases. Comprehensive dissection includes radical neck dissection (removal of all five levels of lymph nodes, accessory nerve, internal jugular vein, and sternomastoid muscle) and modified or functional neck dissection:

- Type 1 preserves the accessory nerve.
- Type 2 preserves the accessory nerve and IJV.
- Type 3 preserves the accessory nerve, IJV, and sternomastoid muscle.

Reconstruction of surgical defect

- Good functional outcome (speech, eating, swallowing) is the aim of reconstruction of surgical defect in the UADT.
- Options include:
  - 1° closure, e.g. small tongue tumour.
  - Local flap, e.g. nasolabial to floor of mouth.
  - Regional flap, e.g. pectoralis major to retromolar region.
  - Free microvascular transfer flaps which offer great versatility, e.g. radial forearm for lining, fibula for bone, anterior thigh for bulk.
  - Prosthesis, e.g. obturator for palatal defect.

Prognosis

- Crude overall 5y survival is 30–40% and of those deaths, 50% die from other causes, usually tobacco-related.
- HPV 16-positive cancers appear to have better outcome.

Facial trauma

Key facts

- Eighty-five per cent of facial injuries are from assault, often with alcohol/drugs involved; the remaining from falls, sports, road accidents, and industrial injuries.
- Ten to twenty per cent have associated head injury, 2% cervical spine injury.
- Fracture incidence: nose > zygoma > mandible > maxilla. Panfacial fractures indicate high-energy impact or multiple blows.

Emergency situations in facial injuries

As part of 1° and 2° surveys, pay special attention to:

- Airway. Severely displaced fractures, tissue swelling (which may get worse), blood, and dislodged teeth can compromise the airway, especially with associated head injury; intubate if in doubt.
- Bleeding. Profuse bleeding can occur in midface fractures or deep tongue wounds, requiring early theatre for suturing and nasal packing/fracture stabilization. Swallowed blood is often vomited.
- Retrobulbar bleed. May follow even minor injury. Orbital swelling can mask it. Cardinal signs are pain, proptosis, and falling visual acuity. Treatment is lateral canthotomy under LA, then theatre for orbital drainage via infra-orbital incision to open the ocular muscle cone; 90min window before blindness sets in.

Key clinical examination points

- Examine the eye, even if it means opening swollen eyelids—check visual acuity. Any diplopia indicates orbital fat/muscle entrapment in orbital complex fracture. An orbital blow-out fracture may have enophthalmos.
- Dental occlusion (bite): ask the patient if bite feels normal. If not, then a fracture is likely. Manually check continuity of the mandible. Fractures in teeth-bearing segment are compound fractures. In the maxilla, grasp the upper incisor teeth and any movement suggests maxillary fracture.
- Mental nerve or infra-orbital nerve paraesthesiae indicates mandibular or orbital floor/zygoma fracture, respectively.
- Look for deformity, e.g. nose deviation, flattened cheek, forehead hollow.

Investigations

- Imaging. Plain X-rays, OPT, and posterior–anterior (PA) skull for fractured mandible; occipitomental 30° and 45° views for zygoma fracture. For complex fractures, CT with 3D reconstruction. Coronal CT/MRI is useful in orbital complex injuries.
- Clinical photographs as a record which may be used in court.
- Other tests, e.g. ECG, Hb, U&Es for falls in the elderly.

Treatment

- Head injuries, soft tissue lacerations, and direct trauma to the eye take precedence.
- Direct pressure to be placed over bleeding sites.
- Fractures involving the teeth are compound and antibiotics are required, e.g. amoxicillin or erythromycin if allergic to penicillin.
- Timing: mandibular fractures involving tooth-bearing segments and any soft tissue lacerations should be treated within 24h. Uncomplicated fractures of orbit/malar/frontal bone/nose/maxilla are best treated when facial swelling has settled. Optimum time is 5–10 days.
- All patients with orbital/malar/maxillary fractures must not blow their nose for 10 days to prevent surgical emphysema of soft tissues.
- Undisplaced fractures may be treated conservatively. Advise soft diet if tooth-bearing fragments are involved.
- The aim of active treatment is to restore function and correct any deformity, e.g. diplopia from orbital complex fracture; decompression of any nerves involved in fracture line (infra-orbital, inferior dental, frontal nerves); restoration of dental occlusion to correct bite (mandibular/maxillary fractures); correction of deformity (fractured nose/zygoma).
- Fractures may be treated by closed reduction, e.g. intermaxillary fixation with wires or open reduction using mini-fracture plates. Surgical access to fractures may be intra-oral, incisions around the eye for the orbit, submandibular for the mandible, bicoronal to frontal bone.
- Patients who have had an unprovoked assault may experience post-traumatic stress disorder and benefit from referral to a clinical psychologist.

Neck space infections

Key facts

- Ninety per cent of neck space infections are of dental origin, especially lower molar teeth.
- Ten per cent are from the tonsils and infected epidermoid, branchial, and thyroglossal cysts.
- Their importance is the risk of airway obstruction, septicaemia, and mediastinitis; mortality risk from overwhelming sepsis.

Anatomy

The cervical fascia is divided into a superficial and a deep layer.

The deep layer is further subdivided into a superficial (investing) layer, a middle (visceral) layer, and a deep layer.

The superficial layer covers the platysma and extends to cover the muscles of the face.

The investing layer of the cervical fascia is attached to the mastoid, superior nuchal line, lower border of the mandible, and hyoid and descends to the clavicle. It splits to enclose the sternomastoid and trapezius muscles and thus forms a structural collar to the neck. Medially lie the pharynx, larynx, trachea, and upper oesophagus which is in direct continuity with the mediastinum. As it splits to enclose the parotid gland, a deep layer is formed, attached to the base of the skull, merging with the upper end of the carotid sheath and pharyngobasilar fascia posteriorly. It also splits to enclose the submandibular gland, with the deep layer attached to the mylohyoid line. As a result, a number of important anatomical compartments or potential spaces exist (see Fig. 5.2).

- Sublingual. Floor of mouth above mylohyoid.
- Submental. Anterior upper neck below mylohyoid.
- Submandibular. Below mylohyoid around submandibular gland.
- Parapharyngeal. Deep to parotid, lateral to pharynx.
- Pterygoid. Pterygomaxillary fissure.

These are all interconnected and continue inferiorly down the neck, following outside the tough carotid sheath into the mediastinum. Related are buccal and submasseteric spaces that are not connected.

Clinical features

- Infection may present as a localized, fluctuant swelling or it may present as spreading cellulitis with a brawny, hard, tender, hot, erythematous mass. Often it is a mixture of both. Necrotizing fasciitis is rare and has a high mortality.
- There is usually a history of toothache, sore throat, and previous neck swelling, e.g. branchial cyst.
- Cardinal signs of severity include: fever, trismus, hot potato speech, dysphagia, stridor, tachycardia, and respiratory rate increase.
- Bilateral sublingual/submental/submandibular swelling (Ludwig's angina) is particularly aggressive.

Investigations

- Temperature, HR, BP, respiratory rate.
- WCC.
- Imaging. OPT if dental cause expected. USS can localize any deep space collection. CT, including chest, is useful in severe cases.

Treatment

- Admit if systemically unwell or any cardinal signs of severity as above.
- IV antibiotics. Co-amoxiclav or clindamycin if allergic to penicillin, and then guide according to M, C, & S.
- Contact the anaesthetist as may need fibreoptic intubation.
- Surgery before sunset if systemic sepsis.

Surgical management

- Remove cause of infection, e.g. extract offending teeth, incise quinsy of tonsil.
- Incise and drain at dependent point any localized abscess.
- Send pus sample for culture and sensitivity.

Exploration of neck spaces

Use a submandibular incision, and incise the platysma and cervical fascia. Using the Hilton's method, find the lower border of the mandible, and then explore medially—this is the submandibular space; go anteriorly to open up the sublingual space. To open the parapharyngeal and pterygoid spaces, push forceps up the medial ramus of the mandible and open the forceps. If there is swelling extending to the root of the neck, make a second incision above the clavicle and medial to the sternomastoid. Suture in a corrugated-type drain.

If intubation is difficult or the airway compromised, e.g. unrelieved trismus on induction, do a tracheostomy. The swelling often gets worse before it gets better. You may need to re-explore the neck. Book an ITU bed in severe cases.

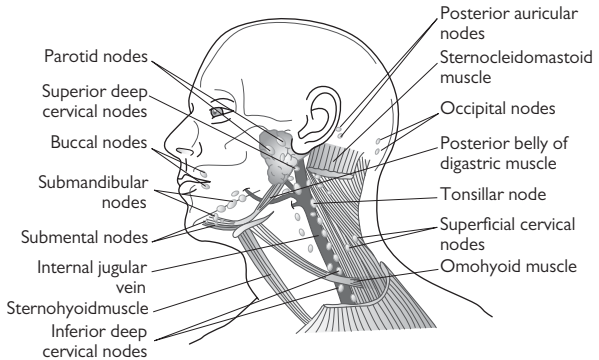

Fig. 5.2 The distribution of lymph nodes in the neck.

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Chapter 6

Breast surgery

Breast cancer

Key facts

- Incidence 55 000/y in the UK (Cancer Research UK, 2014), mortality 11 000/y.
- Lifetime risk for women: 1 in 8.
- Commonest in Western Europe, least common in Japan and Africa.
- Overall, 1/3 are screen-detected (50% for patients aged 50–70).
- Breast cancer mortality rates have ↓ by 35% since 1970s.

Risk factors for breast cancer

- ♀ gender (<1% of breast cancers occur in men).
- Increasing age (80% are aged over 50).
- Dense breast tissue.
- Genetic factors account for <10% of breast cancers.
  - Family history of breast/ovarian cancer.
  - Specific mutations account for 5% (*BRCA1*, *BRCA2*, *TP53*, *CDH1*, *PTEN*).
  - Ashkenazi Jewish heritage (ten times more likely to have *BRCA* gene mutations).
- Previous ionizing radiation, e.g. for lymphoma.
- DCIS: pre-invasive breast cancer.
- Hormonal changes: nulliparity, first pregnancy age >30, early menarche, late menopause, use of combined hormone replacement therapy (HRT) >5y after menopause.
- Lifestyle: alcohol, obesity, smoking.
- Risk is multifactorial.

BRCA and breast cancer risk

- Tumour suppressor genes with dominant inheritance.
- *BRCA1* lifetime risk of cancers:
  - Breast cancer 60–90%.
  - Ovarian cancer 40–60%.
  - ♂ breast cancer 0.1–1%.
  - Prostate cancer 10%.
- *BRCA2* lifetime risk of cancers:
  - Breast cancer 45–85%.
  - Ovarian cancer 10–30%.
  - ♂ breast cancer 5–10%.
  - Prostate cancer 20–25%.
- Managing breast cancer risk in *BRCA* mutation carriers:
  - Earlier screening for early detection from age 30.
  - Advise modification of any lifestyle risk factors.
  - Chemoprophylaxis: tamoxifen or raloxifene.
  - Surgical prophylaxis: risk-reducing mastectomy (reduces breast cancer risk to <5%), risk-reducing salpingo-oophorectomy (risk of ovarian cancer reduced to <5%, breast cancer risk reduced by up to 50%).

Clinical features of breast cancer

Breast lump

- Commonest presenting symptom.
- Usually painless, firm, irregular shape.
- May be tethered/fixed to chest wall or skin.

Skin changes

- Peau d'orange: orange peel appearance of skin due to skin oedema from locally advanced cancer/local lymph node involvement.
- Dimpling or puckering.
- May present late with ulceration or fungating tumour.
- Inflammation of the skin may be due to inflammatory breast cancer (rare, but aggressive, form of breast cancer).

Nipple abnormalities

- Nipple inversion/deviation may occur due to underlying malignancy.
- Paget's disease of the nipple:
  - Rare presentation of breast cancer.
  - Nipple changes may be confused with eczema of the areola.
  - May progress to ulceration/destruction of nipple areola complex.
  - Investigation: triple assessment (see Box 6.1) and a punch biopsy (see Box 6.2).
- Nipple discharge:
  - Unilateral, single duct, bloodstained, spontaneous discharge may be a feature of breast cancer.
  - 10% of bloody nipple discharge is due to breast cancer; 80% are due to benign intraductal papilloma.
  - Milky discharge may be physiological or due to pituitary tumour (check prolactin level).
  - Multiduct, multicoloured discharge is due to duct ectasia (dilated ducts—benign).

Systemic features

- Systemic features of metastatic breast cancer include weight loss, anorexia, bone pain, jaundice, and pleural/pericardial effusions.

Pathological features

- 80% invasive ductal; 20% invasive lobular, mucinous, medullary, or tubular.
- Pathological grades 1–3 relate to degree of cell differentiation.
- DCIS:
  - Pre-invasive breast cancer confined by the basement membrane.
  - 90% asymptomatic, usually detected by screening.
  - May progress to invasive breast cancer.
  - Classified as low/intermediate/high grade.
  - Subtypes: comedo, solid, micropapillary, papillary, cribriform, flat.
- Oestrogen receptor (ER) and progesterone receptor (PR):
  - Steroid receptors in cell nucleus involved in cell growth.
  - Positive receptor is predictive of response to endocrine therapy.
  - 70–80% of breast cancers are ER+.
  - ER status measured by immunohistochemistry (Allred score 1–8).

- Human epidermal growth factor receptor 2 (HER2):
  - HER2 proteins are receptors on breast cells that normally control cell growth.
  - HER2 is amplified in 15–20% of breast cancers, which makes them grow faster and divide in an uncontrolled way.
  - HER2+ status is associated with poorer survival.

Diagnosis and investigations

Diagnostic tests

- All breast lumps/suspected carcinoma undergo triple assessment.
- Clinical assessment:
  - History: duration of symptoms, past history, family history, medical comorbidities.
  - Examination: breasts, axillae, systemic examination if symptoms suggestive of metastatic disease.
- Radiological assessment:
  - Mammography if over 40y of age.
  - USS if age <40 (mammography is less accurate for the assessment of young, dense breasts), as an adjunct to mammography in women age >40, to assess axillary lymph nodes, and guide core biopsies/fine needle aspiration for cytology (FNAC) of breast tissue/axillary nodes.
  - MRI is not used routinely but is used to screen *BRCA1/2* women and is used for the assessment of multifocal/mammographically occult tumours or those not seen well on mammogram, e.g. invasive lobular cancers, to assess suitability for breast conservation, and to monitor response to neoadjuvant therapy.

Box 6.1 Triple assessment

- Comprises clinical examination of breasts and axillae (P), imaging (mammography (M) and/or ultrasound (U)), and histopathological assessment (core biopsy (B) or cytology (C)), each of which is scored according to the suspicion of malignancy.
- Scoring system (e.g. P1 = normal physical examination):
  - 1 = normal (exception is C1 which means inadequate sample).
  - 2 = benign.
  - 3 = indeterminate.
  - 4 = suspicious of malignancy.
  - 5 = malignant.
- Helps to convey the degree of concern regarding a breast lump, determines whether a biopsy or further intervention is warranted in order to achieve a diagnosis, and aids communication among health care professionals.

Tissue diagnosis

- Obtained by USS-guided core biopsy of breast lump ± FNAC/core biopsy of abnormal axillary nodes.
- Punch biopsy of suspicious skin/nipple lesions.
- Core biopsy provides more information than FNAC as it can differentiate between DCIS and invasive cancer and provides tissue for assessment of ER/PR and HER2 status.

Box 6.2 How to perform a punch biopsy

- Check patient's identity and indication for procedure.
- Obtain verbal or written informed consent from the patient.
- Prepare equipment needed for procedure.
  - Sample pot containing appropriate medium.
  - Clean trolley/tray.
  - Skin biopsy pack containing forceps and scalpel.
  - Sterile non-woven swabs and towels.
  - Sterile gloves.
  - Disposable apron.
  - Antiseptic skin prep, e.g. chlorhexidine.
  - LA, e.g. lidocaine 1%.
  - 2mL syringe.
  - Disposable punch biopsy needle.
  - Orange needle.
  - Blue needle.
  - Wound closure strips, e.g. Steri-Strip™.
  - Dressing.
- Position patient and expose biopsy site.
- Wash hands; put on gloves and apron, and clean the biopsy site.
- Draw up LA using syringe and blue needle.
- Remove blue needle and change to orange needle.
- Inject LA SC at biopsy site.
- Check biopsy site is adequately anaesthetized.
- Insert punch biopsy needle at 90° to skin surface.
- Rotate punch biopsy needle several times to ensure a disc of skin is cut.
- Remove punch biopsy needle and apply pressure to biopsy site with swab.
- If the specimen retained in end of needle, aid removal with forceps, scalpel, or needle. Handle carefully to prevent crushing the sample.
- Place specimen into sample pot containing appropriate medium, and label container and histology request form correctly.
- Apply wound closure strips and dressing to biopsy site.
- Dispose of sharps and used equipment and protective clothing appropriately.
- Wash hands; document procedure, and send sample in a sealed specimen bag to the laboratory.
- Provide post-procedure information leaflet to patient.

Staging investigations

- Axillary lymph node status is the most significant prognostic marker.
- Staging investigations for distant metastases not usually performed for patients with early breast cancer (i.e. <5cm and node negative).
- Indications for staging:
  - Locally advanced breast cancer.
  - Recurrent breast cancer.
  - Symptoms of metastatic disease in nodes, bone, liver, lung, and brain.
- Investigations may include: blood tests (LFTs, serum Ca^{2+}), staging CT (chest, abdomen, and pelvis), and isotope bone scan.

Surgical treatment of breast cancer

Surgery is the mainstay of treatment for early breast cancer. Options for treating 1° breast disease are breast-conserving surgery or mastectomy, with or without breast reconstruction.

Breast-conserving surgery

This aims to provide local disease control with minimum morbidity and acceptable cosmetic outcomes. Long-term results are equivalent to mastectomy, providing surgery is followed by radiotherapy for invasive cancers (reduces the risk of local recurrence).

Wide local excision

- Aim for complete excision with a clear pathological margin.
- Preoperative localization using ultrasound or stereotactic methods may be required for impalpable lesions.
- Considerations: size of tumour relative to breast volume and tumour location.
- Complications: haematoma (2% require evacuation), infection (5–10% delay adjuvant treatment), involved margins requiring re-excision (10–25%), poor cosmetic outcome.
- In-breast tumour recurrence rate: <0.5%/y.

Oncoplastic techniques

- Excision of >10% breast volume is associated with poorer cosmesis.
- Volume replacement techniques now include the use of local flaps/rotation of breast tissue to fill defects or lipofilling.

Mastectomy

- Options: simple mastectomy or skin-preserving mastectomy.
- Indications: large cancer (relative to breast size) or multifocal tumours, widespread *in situ* changes, cancer in *BRCA1/2* patient, patient choice, risk reduction.
- Tumour location may be a relative indication, e.g. retroareolar cancer.
- Adjuvant breast radiotherapy not required if early breast cancer.
- Complications: haematoma, infection, flap necrosis (1–2%).

Breast reconstruction

- Can be immediate (at same time as mastectomy) or delayed.
- Factors to consider: adjuvant therapy, smoking status, comorbidities.
- Techniques include:
  - Implant-based ± dermal sling or acellular dermal matrix.
  - Autologous flap-based: pedicled flap, e.g. latissimus dorsi (LD) flap or free flap (using microvascular techniques).

Surgery for metastatic breast cancer

- Limited to procedures which provide symptom control of local disease such as a fungating tumour.

Management of the axilla

Sentinel lymph node biopsy (SLNB)

- Allows accurate staging of the axilla in patients with invasive breast cancer without clinically palpable/radiologically abnormal nodes (70–80% of patients)—patients with –ve nodes can be spared the morbidity of axillary node clearance (ANC).
- Dual technique (technetium 99m + patent blue V dye) improves the accuracy of localization. Preoperative lympho-scintigram may be used.
- SC injections of radioisotope and blue dye spread to the sentinel nodes (i.e. first drainage site of cancer cells).
- Less invasive than ANC, with a lower risk of complications such as lymphoedema, seroma, or shoulder stiffness.
- Risk of severe anaphylactic reaction to blue dye (1:2000).
- Selective removal of sentinel lymph node(s) which are assessed by histopathological examination or intraoperative polymerase chain reaction (PCR)-based assays to determine whether ANC is required (if nodes +ve).
- If nodes are +ve, current practice is to perform ANC or axillary radiotherapy.

Axillary node clearance

- Traditional management for axillary metastases.
- Complications: lymphoedema, reduced shoulder movement, injury to structures including the long thoracic nerve (winging of scapula), thoracodorsal pedicle, intercostobrachial nerves, and axillary vessels.
- New trials suggest that ANC may not always be necessary in early breast cancer with a limited number of positive nodes on SLNB. Further evidence is awaited.

Management of DCIS

- Commonly presents as asymptomatic microcalcification seen on screening mammograms.
- Diagnosis made by mammogram and stereotactic-guided biopsies.
- Surgical options are the same as for invasive breast cancer and depend on the size of the known abnormality.
- Often need preoperative localization, as impalpable.
- Breast-conserving surgery for high- or intermediate-grade DCIS is followed by adjuvant radiotherapy.
- Axillary surgery and adjuvant drug treatment are not required, as no potential for lymph node metastases.

Adjuvant treatment of breast cancer

Radiotherapy

Whole breast radiotherapy (WBRT)

- Recommended for all patients treated with an invasive cancer who have had breast-conserving surgery and for patients who have undergone mastectomy for locally advanced breast cancer (T3/T4) or who have axillary lymph node metastases.
- Dosing schedule: 40Gy in 15 fractions over 3 weeks.
- Adjuvant use for DCIS traditionally guided by Van Nuys Prognostic Index (based on tumour size, margins, grade, presence of necrosis, and patient age).
- Side effects: skin changes, pain, lymphoedema, reduced range of arm movement, tiredness, brachial plexopathy, respiratory complications (less common with modern techniques).
- Clinical trials evaluating the use of localized breast radiotherapy in place of WBRT are currently being undertaken.

Tumour bed boost and/or supraclavicular fossa radiotherapy

- May be given in addition to WBRT for patients with poor prognosis disease or a heavy nodal burden.

Axillary radiotherapy

- May be used to treat patients with axillary nodal metastases, instead of ANC, due to patient choice/fitness for further surgery.

Chemotherapy

- Combination chemotherapy reduces breast cancer recurrence and improves survival. It is offered to patients with high-risk features (young patients, +ve nodes, poor histological grade, large size) as adjuvant treatment after surgery.
- Potential benefits are predicted using computer-based models, e.g. Adjuvant Online/Predict, or multiple gene expression microarray analysis, e.g. Oncotype DX.
- Benefits must be balanced against the toxicity of chemotherapeutic agents.
- Anthracycline-based chemotherapy has replaced CMF (cyclophosphamide, methotrexate, and fluorouracil) regimes.
- Taxane-based chemotherapy regimes are used in anthracycline-resistant metastatic breast cancer and in HER2+ disease.
- Neoadjuvant chemotherapy can be used to downsize larger tumours to enable breast conservation or to make locally advanced tumours operable.
- Side effects: neutropenic sepsis, hair loss, nausea, cardiomyopathy, neuropathy.

Endocrine therapy

Used in patients with ER+ breast cancer. It can be used in the neoadjuvant setting to decrease the size of a tumour before surgery, or as a 1° treatment in patients who are not fit for surgery, or in the adjuvant setting after chemotherapy (if indicated).

Tamoxifen

- Used as first-line endocrine therapy in pre-menopausal patients.
- Reduces the risk of recurrence and improves survival.
- Side effects: hot flushes, vaginal bleeding, vaginal discharge, VTEs, endometrial cancer in post-menopausal women.

Aromatase inhibitors, e.g. letrozole, anastrozole

- First-line treatment in post-menopausal patients (contraindicated in pre-menopausal women).
- Improves survival.
- Side effects: hot flushes, musculoskeletal problems, ↑ risk of bone fractures so women should have a bone density scan at the start of treatment.

Targeted molecular therapy

Trastuzumab

- Recombinant humanized monoclonal antibody to HER2.
- Can be used in the neoadjuvant or adjuvant setting in patients with HER2+ breast cancer.
- Improves disease-free and overall survival in HER2+ breast cancer; benefits greater when given with taxane-based chemotherapy.
- Side effects: cardiotoxicity (worse if given with anthracycline-based chemotherapy regimes). Cardiac function is monitored with multigated acquisition (MUGA) scans/echocardiograms.

Pertuzumab

- Recommended in conjunction with chemotherapy and trastuzumab as neoadjuvant treatment of locally advanced/inflammatory/early-stage HER2+ breast cancers.
- Improves pathological complete response and disease-free survival.

Management of disseminated (stage IV) breast cancer

- Aims: symptom control, to improve quality of life, to prolong survival.
- Treatments: endocrine therapy, chemotherapy, radiotherapy, surgery, targeted molecular therapy.
- Isolated doses/short courses of radiotherapy may be used with palliative intent to treat metastases in bone/skin (reduces tumour burden and pain).
- A variety of medications can be used in the metastatic setting that are not used as adjuvant treatments.

Male breast cancer

Key facts

- Accounts for <1% breast cancers and 0.1% of ♂ cancer deaths.

Risk factors

- Family history (15–20%): up to 10% are due to *BRCA2* mutation.
- Age.
- Previous radiation to chest.
- Excess alcohol.
- Liver cirrhosis.
- Obesity.
- Klinefelter's syndrome.

Clinical features

- 85% present with painless mass.
- Increasing incidence with age.
- May present later than in women.

Pathological features

- 90% invasive (80% ductal cancer), 10% DCIS.
- 90% are ER+.

Diagnosis and investigations

- As described for ♀ breast cancers (➲ Breast surgery, p. 314).

Treatment

- Mastectomy is the usual surgical treatment due to limited tissue and often late presentation.
- Adjuvant chemotherapy may be needed if ER –ve.
- Tamoxifen is the drug of choice for ER+ disease, after surgery.
- Side effects of endocrine therapy: reduced libido, impotence, hot flushes and sweats, disordered mood, headaches, nausea.

Breast cancer screening

Aims

- To identify asymptomatic/early breast cancer.
- To identify DCIS.

Abnormalities seen on screening mammography

- Spiculated lesion.
- Asymmetric density.
- Calcification/microcalcification.

NHS Breast Screening Programme (NHSBSP)

- Initiated after the *Forrest Report* published in 1986.
- Population-based screening programme introduced in 1988.
- Screening age is 50–70y, but planned age extension from 47 to 73y currently being rolled out.
- Screening by two-view mammography every 3y.
- Women older than screening age can self-refer for screening.
- Disadvantages of screening: risk of false positive screen causing unnecessary anxiety/psychological morbidity, risk of false negative screen resulting in false reassurance (~10% of cancers are not seen on mammograms), radiation exposure, risk of overtreatment.

Results

- Around 70% of women offered screening accept invitation.
- Around 1 in 25 women are recalled for further investigation. Of these, 1 in 4 are diagnosed with breast cancer (80% invasive, 20% non-invasive).
- Screen-detected cancers generally have better prognostic features than symptomatic cancers (smaller, lower grade, node –ve).
- One breast cancer death is prevented for every 250 women invited (1300 deaths from breast cancer are prevented per year in the UK).
- For each breast cancer death prevented, three women will be overdiagnosed and treated.
- 1% of women invited to screening have an overdiagnosed cancer (i.e. a cancer diagnosed by screening that would not otherwise have come to attention in the woman's lifetime).
- Breast cancer mortality reduced by 20% in those invited for screening.

Increased risk groups (after genetic assessment)

- Annual mammographic surveillance for:
  - Moderate-risk patients aged 40–59y.
  - High-risk patients aged 30–59.
  - Patients with known *BRCA* mutation aged 30–69y.
  - Followed by NHSBSP screening programme.
- MRI surveillance for patients with:
  - Known *BRCA* mutation aged 30–49y.
  - Known *TP53* mutation (Li Fraumeni syndrome) aged 20–49y.

Benign breast disease

Most benign breast conditions arise from minor aberrations of normal processes of development, cyclical activity, and involution of the breast (aberrations in normal development and involution (ANDI)), infections, or trauma.

Fibroadenoma

- Common benign breast lesion, usually in women <30y.
- Smooth, rubbery, mobile, discrete, painless breast lump ('breast mouse').
- May be multiple or giant (commoner in Afro-Caribbeans).
- Composed of mixed stromal and epithelial elements.
- Investigation: USS ± core biopsy.
- Treatment: conservative; excise if large size, concern over diagnosis or cosmesis, or if symptomatic.

Breast cyst

- Common, benign, symmetrical, round lumps; may be tender.
- May be solitary or multiple.
- Investigations: USS (aspiration of straw-coloured fluid resulting in collapse is diagnostic); biopsy any residual mass.
- Treatment: USS-guided needle aspiration; may recur.

Fibrocystic change

- Affects 30–60% of women, usually aged 30–50y.
- Lumpy breast texture, due to localized fibrosis and cyst formation, usually in upper outer quadrant.
- May be associated with cyclical breast pain and swelling.
- Due to fluctuating hormone levels, usually subsides after menopause.
- Treatment: cyst aspiration, treat associated breast pain, reduce caffeine, hormone manipulation, evening primrose oil, vitamin E.

Phyllodes tumour

- Rare (<1% of breast neoplasms) fibro-epithelial tumour of unknown cause with varying malignant potential (up to 25–30% malignant).
- Presents with firm, rapidly growing palpable mass, usually in women aged 40–50y.
- Classical 'leaf-like' appearance on histopathological examination.
- Similar pathology to fibroadenomas, but ↑ cellularity and activity of stromal element.
- Treatment: wide local excision to prevent local recurrence.
- Metastases are rare overall, but up to 20–25% in malignant phyllodes with pattern of spread similar to sarcoma (lung, bone, abdominal viscera).

Fat necrosis

- Firm, irregular mass that often develops after trauma, bruising in the breast.
- Needs to be differentiated from a cancer.
- Mammograms may show oil cysts.
- Resolves spontaneously.

Breast infections

Usually present with breast pain and inflammation ± palpable mass. Follow up all breast infections to ensure complete resolution and exclude inflammatory breast cancer, which may present similarly.

Lactational mastitis

- Affects 5% of breastfeeding women.
- Usually due to staphylococcal infection.
- Treatment: advise continued feeding/expressing, antibiotics (flucloxacillin).

Non-lactational mastitis

- Associated with smoking, due to mixed bacterial growth.
- Often chronic/recurring, may develop mammary fistula(e).
- Treatment: antibiotics to cover anaerobic bacteria and Gram-negative bacilli (e.g. amoxicillin + metronidazole).

Breast abscess

- Can occur 2° to lactational/non-lactational mastitis.
- Treatment: needle aspiration under USS guidance/mini-incision and drainage only if failed aspiration or necrotic overlying skin.

Breast pain

Common presenting complaint, but <0.1% of breast cancers present with breast pain alone.

Causes and features

True breast pain

- Cyclical breast pain.
  - Exaggerated response to hormonal fluctuations.
  - Fibrocystic disease.
- Non-cyclical breast pain.
  - Mastitis.
  - Breast abscess.

Non-breast origin

- Musculoskeletal: may be worse on movement.
  - Costochondritis—local inflammation of a costochondral joint (Tietze's syndrome).
  - Lateral chest wall pain.
- Visceral: angina, acute coronary syndrome, pleural disease.
- Skin pathology: infected sebaceous cyst, cellulitis, skin abscess.

Investigations

- History and examination.
- Imaging ± biopsy if any suspicion of breast cancer.
- USS may help identify breast abscess if breast acutely inflamed.
- Pain chart may be useful if no pathology found.

Management

- Reassure.
- Advise reduction in dietary caffeine.
- Medical management:
  - Analgesia: paracetamol, PO/topical NSAIDs.
  - Evening primrose oil (contains gamma linolenic acid).
  - Tamoxifen.
  - Danazol.
- No role for surgical management.

Gynaecomastia

Key facts

- Hyperplasia of ♂ breast tissue.
- Commonest ♂ breast condition.
- Presents with concentric swelling of breast tissue ± tenderness.
- Differential diagnosis: cancer, pseudogynaecomastia (excess fat).

Causes and features

Physiological

- Typically occurs in neonates/during puberty or senescence.
- Usually self-limiting.

Pharmacological

- Hormones: anabolic steroids, oestrogen agonists, anti-androgens.
- Cardiovascular drugs: digoxin, spironolactone, amiodarone, nifedipine, ACE inhibitors, verapamil.
- Anti-ulcer drugs: omeprazole, ranitidine, cimetidine.
- Antibiotics: metronidazole, minocycline, ketoconazole.
- Psychiatric drugs: diazepam, tricyclics, phenothiazines.
- Antiretroviral drugs.
- Recreational: alcohol, cannabis, heroin.
- Others: metoclopramide, phenytoin, methyldopa, penicillamine, domperidone.

Underlying disease processes

- ↑ oestrogen: produced by testicular/adrenal/lung tumours.
- ↑ peripheral oestrogen aromatization: liver/adrenal/thyroid disease, starvation refeeding.
- ↓ androgen production: bilateral cryptorchidism/torsion, orchitis, hyperprolactinaemia, chromosomal abnormalities (Klinefelter's syndrome), renal failure.
- Androgen resistance: testicular feminization.

Classification of gynaecomastia

- I: mild, little/no excess skin.
- II: moderate with (IIa) or without (IIb) excess skin.
- III: severe + excess skin.

Investigations

- Exclude breast cancer: history and examination of breast, axillae, testes, and abdomen ± USS breast ± core biopsy.
- Exclude pathological causes: U&Es, LFTs, GGT, prolactin, alpha-fetoprotein (AFP), β-HCG, testosterone.

Management

- Reassurance (80% resolve).
- Address underlying disease processes/change or withdraw drug.
- Medical: tamoxifen (but not a licensed indication).
- Limited role for surgery (IIb or III not responding to above): options are liposuction/open excision ± reduction of excess skin.

Endocrine surgery

The thyroid gland

Anatomy

- The thyroid gland consists of *two lobes*, which constitute 90% of its substance.
- They are joined in the midline by a *central isthmus*, with a variable *pyramidal lobe* extending cranially.
- Most of the thyroid gland comprises *follicles* that contain *colloid* and are lined by epithelial (follicular) cells that synthesize thyroxine. In addition, there is a small population of *parafollicular C cells* that secrete *calcitonin*, which has negligible function in normal physiological conditions.
- Arterial supply to each lobe is via the *superior thyroid artery* (branch of the *external carotid artery*) and the *inferior thyroid artery* (from the *subclavian artery*).
- Venous drainage is via a plexus draining to the superior, middle, and inferior thyroid veins.
- Lymphatic drainage is typically to *central cervical lymph nodes*, followed by *lateral cervical lymph nodes*.
- The thyroid is intimately related to the *larynx* and *trachea*, *oesophagus*, *parathyroid glands*, and *recurrent laryngeal nerves (RLNs)*.

Physiology

- The principle role of the thyroid gland is the *synthesis* and *regulation* of *thyroid hormones*.
- *Thyroid-stimulating hormone (TSH)* from the *anterior pituitary* increases the uptake of iodide (I^-) from the circulation.
- *Thyroglobulin (Tg)* is released from the apical membrane of the follicle cell and facilitates *iodination* of tyrosine residues to form *monoiodotyrosine* (*MIT*) or *diiodotyrosine (DIT)*.
- *MIT* and *DIT* are coupled in the follicular lumen to form *triiodotyrosine (T_3)* and *tetraiodothyronine (T_4, L-thyroxine)*.
- T_3 and T_4 are subsequently cleaved from Tg within the follicular cells and secreted as an active hormone under *TSH* control.
- T_3 is by far the most biologically active, and T_4 is deiodinated peripherally to produce *active* T_3, or *reverse* T_3, or *DIT*.
- Both T_4 and T_3 can be administered therapeutically in either synthetic or biological forms.

Terminology

- *Thyroid lobectomy or hemithyroidectomy* are interchangeable terms. This operation involves removal of a single lobe of the thyroid in conjunction with the *thyroid isthmus* ± *pyramidal lobe*.
- *Total thyroidectomy* means removal of all thyroid tissue, including both lobes, the isthmus, and the pyramidal lobe (when present).

Goitre

Key facts

- A normal thyroid gland is impalpable.
- Goitre (Latin *guttur* = *throat*) refers to an enlargement of the thyroid gland.
- In the UK, 15–40% of people have a palpable goitre; up to half have a goitre when assessed by ultrasound.
- ♀:♂ is up to 10:1.
- Most patients with goitre are euthyroid.
- Thyroid cancer is very rare (1% of new malignancies).

Pathological features

- Goitre is caused by hyperplasia of thyroid follicular tissues; causes include diet, genetics, iodine deficiency, and environmental and endocrine factors.

Goitre can be classified as follows:

- *Epidemiology*. Endemic (iodine deficiency), sporadic, familial, drug-induced.
- *Morphology*. Diffuse, nodular (multinodular, solitary nodule).
- *Functional status*. Toxic, non-toxic, hypothyroid.
- *Location*. Cervical or retrosternal.

Clinical features

- The WHO classifies goitre clinically as 0 (not visible or palpable), 1 (palpable), 2 (visible), and 3 (large and visible from a distance).
- Sporadic multinodular goitre (MNG) is the commonest presentation in the UK, usually as an asymptomatic neck mass.
- Patients may present with compressive symptoms such as dysphagia (oesophagus), stridor and respiratory compromise (trachea), and distended neck veins and facial plethora (obstruction of venous drainage).
- 'Red flag' features associated with thyroid cancer include rapid growth of mass, hoarse voice (invasion of the RLN), lymphadenopathy, dysphonia (invasion of upper airways), weight loss, and signs of metastatic disease.
- Risk factors for thyroid cancer include exposure to ionizing radiation (especially in childhood), family history of thyroid cancer, and ♂ gender.

Goitre may also be associated with thyroid dysfunction:

- Graves' disease may be accompanied by a hyperplastic goitre and thyrotoxicosis.
- Hashimoto's disease may be associated with a modest, firm, 'woody' goitre.
- Thyrotoxicosis may be seen in the context of a toxic nodule (adenoma) or a toxic MNG (Plummer's disease).

Diagnosis and investigations

- *History and examination* will determine the nature of the goitre and suggest a potential thyroid function disorder.
- *Thyroid function tests (TFTs)*. TSH initially ± free T_4 as initial screening tests. Usually normal.
- *CXR*. In cases of a large goitre, this may suggest retrosternal extension ± tracheal deviation. Rarely used.
- *CT scanning*. Cross-sectional imaging in patients when retrosternal extension is suspected.
- *Flexible laryngoscopy*. Mandated in cases of hoarse voice to assess vocal cord (RLN) function.

Treatment

Surgical treatment

- Surgery is the most effective treatment; medical therapy may be considered in patients unfit or unsuitable for surgery.
- Indications include:
  - Local compressive symptoms.
  - Proven/suspected malignancy.
  - Enlarging goitre with retrosternal extension.
  - Patient choice due to cosmetic effects.
  - Failed medical therapy.
- *Hemithyroidectomy* is a feasible option in largely unilateral disease and will avoid long-term hypothyroidism in up to 85%.
- *Total thyroidectomy* is required for bilateral disease and additional indications, and removes the risk of recurrence. All will require post-operative levothyroxine.
- Complications of thyroidectomy include RLN injury (requires tracheostomy if bilateral), hypocalcaemia (total thyroidectomy), and haemorrhage.

Medical treatment

- *Levothyroxine* treatment is indicated in hypothyroidism and may reduce the size of a goitre in this circumstance and in endemic (iodine-deficient) goitre, by reducing the stimulus from excess TSH.
- *Radioactive iodine (RAI; ^{131}I)* induces gradual destruction of the thyroid and may reduce goitre size by up to 50%. May require repeated doses. Used for non-toxic goitre (more in Europe than the UK or USA).
- Risks of RAI are:
  - Radiation thyroiditis (acute thyroid swelling may be dangerous in patients with large retrosternal goitre).
  - Temporary thyrotoxicosis (rapid release of preformed thyroid hormones from thyroid follicles).
  - Late hypothyroidism (common) due to over-destruction of the gland.

Thyrotoxicosis

Key facts

- The prevalence of thyrotoxicosis is ~2% in ♀, with an annual incidence of 0.5–1 per 1000 in Europe.
- Graves' disease (75%) is the commonest cause, followed by toxic multinodular (Plummer's disease) and toxic adenoma (5%).

Pathological features and causes

- *Autoimmune*. Graves' disease involves stimulation of thyrocytes by TSH receptor antibodies. Early phase of Hashimoto's disease can be associated with thyrotoxicosis.
- *Autonomous function*. Seen in toxic solitary or MNG (benign follicular adenomas).
- *Release of preformed thyroid hormones*. Seen in subacute (viral) thyroiditis (*subacute granulomatous or de Quervain's thyroiditis*) and post-partum thyroiditis.
- *Factitious thyrotoxicosis*. Exogenous ingestion of excess thyroid hormone.

Other rare causes include:

- *Drug-induced*. Amiodarone, lithium, tyrosine kinase inhibitors, antiretroviral therapy (all can cause release of preformed hormone or induce autoimmune reaction).
- *TSH-secreting pituitary adenoma*.
- β-*HCG-mediated hyperthyroidism*. Gestational, hydatidiform mole.
- *Thyroid cancer*.

Clinical features

Multisystem disease—common signs and symptoms include:

- *Neurological*. Tremor (common), anxiety (common), restlessness, emotionally labile, insomnia/lethargy.
- *GI*. Diarrhoea, ↑ appetite, weight loss (common).
- *Cardiac*. Palpitations (common), sinus tachycardia/AF (common), dyspnoea, chest pain.
- *Dermatological*. Thin hair, pretibial myxoedema/thyroid acropachy, erythema.
- *Reproductive*. Menstrual irregularities, loss of libido.
- *Thyroid*. Goitre, either diffuse or nodular, bruit.
- *Ophthalmological*.
  - *Graves' ophthalmopathy*. Proptosis (exophthalmos), periorbital oedema, chemosis, ↓ acuity, visual field loss, ophthalmoplegia.
  - *All causes*: lid lag.
- *Other*. Heat intolerance (common), muscle weakness.

Rarely, patients can present with *life-threatening thyrotoxic crisis ('thyroid storm')* due to a variety of triggering causes.

Diagnosis and investigations

- *History and examination*, including all systems to evaluate disease and thyrotoxicosis-related comorbidities.
- *Biochemical tests*.
  - TSH and free T_4 ± free T_3 to confirm diagnosis.
  - Thyroid receptor antibodies (TRAbs); Graves' disease.
  - Thyroid peroxidase antibodies (TPO); Hashitoxicosis.
- *Imaging* (often not required).
  - USS.
  - Technetium scintigraphy; useful in diagnosis of toxic nodule.

Treatment

Medical treatment

- *Antithyroid drugs (ATDs)*. Up to 50% long-term remission in Graves' disease.
  - *Carbimazole* 20–40mg daily initially (preferred in the UK).
  - *Propylthiouracil* 200mg bd initially.
- Following initial treatment, (euthyroid) patients treated with either *'block and replace'* regime or titrated dose of ATDs.
- Warn ALL patients of risk of *agranulocytosis* whilst on ATDs. Presents as sore throat, mouth ulcers, and high fever.
- β-*blockers* (propranolol 40–120mg/day) are used to control tachycardia and tremor.
- *RAI (^{131}I)* is an effective treatment for thyrotoxicosis. Contraindicated in active Graves' ophthalmopathy (may worsen condition), pregnancy (teratogenic), and in those with young children (relative, requires isolation).

Surgical treatment

- Render all patients euthyroid preoperatively.
- Resistant thyrotoxicosis may be managed with preoperative β-*blockers* and *Lugol's iodine/potassium iodide*.
- *Total thyroidectomy* is the treatment of choice for ATD-resistant or relapsed Graves' disease in patients not suitable for ^{131}I or those with moderate to severe active eye disease.
- *Hemithyroidectomy* may be considered in unilateral toxic nodules.
- Other indications for surgery include:
  - Symptomatic or large (goitre).
  - Poor response to ^{131}I.
  - Coexisting parathyroid disease.
  - Paediatric thyrotoxicosis.
  - Patient choice.
- Surgery for thyrotoxicosis, especially Graves' disease, is associated with higher complication rates than other benign indications for thyroidectomy.

Differentiated thyroid cancer

Key facts

- Thyroid cancer is rare, accounting for 1% of all new cancers.
- The incidence is increasing due to ↑ use of neck imaging.
- Differentiated thyroid cancer (DTC) comprises papillary (PTC; 85%) and follicular (FTC; 15%) thyroid cancer.

Clinical presentation

- The commonest presentation is with an asymptomatic nodule.
- Clinical nodules are present in ~5% of women and 1% of men; high-resolution ultrasound identifies nodules in up to two-thirds.
- The risk of malignancy in a thyroid nodule is 7–15%.
- Extremes of age (<16y or >70y) are risk factors for cancer. Children typically present with more advanced disease.
- Other malignant features include voice change, cervical lymphadenopathy, and a rapidly enlarging goitre.

Pathological features

- *Papillary carcinoma*. Cells show typical cytological features, including nuclear grooves, intranuclear inclusions, or optically clear nuclei ('orphan Annie cells'). Metastasis is typically lymphatic.
- High-risk cell types include *tall cell* and *diffuse sclerosing* types.
- *Follicular carcinoma*. Variable degrees of invasion. *Minimally invasive* types invade the thyroid capsule only. *Widely invasive* variants can invade local structures and display haematogenous spread.
- *Hurthle cell (oncocytic) carcinoma* is a high-risk subtype.
- *Genetic mutations* associated with DTC are *BRAF*, *RAS*, and *TERT*.
- High-risk prognostic factors include age >45, ♂ gender, high-risk cell type, local invasion, and high-risk mutations (e.g. *p53*, *TERT*).

Diagnosis and investigations

- *History and examination*—evaluate risk factors and presenting mass.
- *Blood tests*—including TFTs, thyroid autoantibodies, and Ca^{2+}.
- *FNAC* is a highly sensitive test for thyroid cancer in experienced hands. USS-guided FNAC may improve diagnostic yield. The Royal College of Pathology reporting system (UK) and Bethesda 2 (USA) are commonly used.
  - *Thy1*, non-diagnostic; *Thy1(c)*, benign cyst; *Thy2*, benign; *Thy3a*, cellular atypia; *Thy3f*, follicular neoplasm; *Thy4*, suspicious for malignancy; *Thy5*, malignant.
- *USS* can provide detailed description of thyroid nodules and is the most sensitive imaging modality in identifying malignancy.
- *Genetic sequencing* tools are commercially available and provide additional information regarding malignant potential of a nodule.

Staging

- *TNM staging* remains the best predictor of survival in DTC.
- *MACIS (Metastases, Age, Completeness of resection, Invasion, Size)/AMES (Age, Metastases, Extent, Size)* and other prognostic scoring systems are commonly utilized in some specialist centres.
- *USS ± CT (without iodinated contrast) or MRI* provide accurate preoperative staging of the 1° tumour/cervical lymph nodes.
- *Distant metastases* are assessed by cross-sectional imaging.

Treatment

Surgical treatment

- All thyroid cancer patients should be managed by a designated *thyroid cancer MDT*.
- The most effective treatment for thyroid cancer is adequate surgery.
- *Hemithyroidectomy* is appropriate for diagnostic purposes in patients with indeterminate lesions (*Thy3a*) and follicular lesions (*Thy3f*; FNAC cannot distinguish follicular adenoma from follicular carcinoma) and for suspicious (*Thy4*) lesions.
- *Hemithyroidectomy* is also an acceptable approach to small unifocal tumours (*Thy5*) <2cm (possibly <4cm) with no high-risk features.
- *Clinical ± radiological* observation may be utilized in *micropapillary* carcinoma (<1cm) with no high-risk features.
- *Total thyroidectomy* is the procedure of choice for larger tumours and tumours with intermediate- to high-risk factors for recurrence.
- *Therapeutic neck dissection* is appropriate for clinical or radiological evidence of cervical lymph node metastasis. *Prophylactic neck dissection* is not routine practice for DTC.
- *Flexible laryngoscopy* preoperatively assesses vocal cord function.
- *Complications*, including RLN injury and hypocalcaemia (devascularization or excision of parathyroid glands), are commoner following surgery for malignant disease. Adjuncts such as intraoperative neural monitoring may reduce the risks.
- *Preservation of function* should be attempted wherever possible, including conservation of the RLN, IJV, and sternocleidomastoid (SCM) muscle, if clinically appropriate.

Additional treatment and follow-up

- *Suppressive doses* of levothyroxine are generally utilized to reduce the stimulatory effect of TSH on any residual malignant cells.
- *RAI remnant ablation* is utilized for patients with risk factors for recurrence/death. This requires a period of social isolation. The efficacy of this treatment is improved by thyroid hormone withdrawal or recombinant human TSH administration.
- *Tg* is an excellent post-operative tumour marker for DTC. Any post-operative increase in Tg should raise the possibility of disease recurrence and prompt further investigation.
- *Dynamic risk stratification* is utilized in most thyroid cancer centres and involves USS assessment and Tg (either stimulated or not).
- Patients considered *low risk* can be considered for relaxation of TSH-suppressive levothyroxine and have their follow-up regimen relaxed.
- *High-risk* patients will undergo serial Tg measurements and imaging, and remain on suppressive treatment for at least 5y.
- Overall 10y survival rates for PTC approach 95%; in FTC, it will exceed 85% in experienced centres.

Medullary thyroid cancer

Key facts

- Medullary thyroid cancer (MTC) is a rare condition arising from the parafollicular calcitonin-secreting C cells of the thyroid—in effect, a neuroendocrine thyroid tumour.
- MTC accounts for <5% of all thyroid cancers.
- MTC may be sporadic (75%) or familial (*FMTC*), or associated with *MEN2* or *MEN3*.
- *RET* proto-oncogene mutations occur in genetically determined cases.

Clinical presentation

Sporadic cases

- Often present in the fourth or fifth decade with a thyroid mass.
- ~50% of patients present with cervical lymphadenopathy, whilst others may present with symptoms of advanced disease: diarrhoea (hypercalcitoninaemia), flushing, or bone pain.
- Metastatic disease (liver, lungs, bone) occurs in 20% at presentation.
- Some patients will present with incidental findings of a thyroid mass on imaging or with unexplained ↑ calcitonin.

Familial/multiple endocrine neoplasia cases

- Most *kindred* will be identified through family tree analysis from the index case and *genetic screening* of at-risk individuals. As a result, many will be under surveillance from a young age.
- Patients presenting as the *index* case or those patients not under surveillance (lost to follow-up/adopted, etc.) will tend to present earlier than sporadic disease.
  - *MEN3* usually presents in infancy or childhood.
  - *MEN2* presents in the second or third decade.
  - *FMTC* presents at a similar time to sporadic MTC.
- In genetic disease, MTC arises on a background of *diffuse C cell hyperplasia*.
- MTC is usually the first manifestation in MEN2/3, but rarely patients will present due to other components of the condition such as phaeochromocytoma or perioral neuromas.

Diagnosis and investigations

- Non-kindred patients presenting with a neck mass should be investigated in the same way as for any thyroid nodule.
- *FNAC* of either a thyroid nodule or lymph node may show spindle-shaped cells with pleomorphic nuclei, dense chromatin, and eosinophilic cytoplasm, thus allowing a diagnosis of MTC.
- Immunohistochemistry will stain positive for *calcitonin* ± *CEA*.
- *Tumour markers*: serum calcitonin and CEA.
- *Genetic testing/mutation analysis* may identify *RAS* mutations, especially high-risk mutations such as codons 918, 634, and 630. This is critically important in the index case.
- *Urinary or plasma metanephrines* should be performed in ALL patients with MTC to exclude phaeochromocytoma, which occurs in ~50% of MEN2/3 patients.
- *Serum (Ca^{2+}) measurement* will screen for hyperparathyroidism.

Staging

- *TNM staging* system. Most accurate predictor of survival.
- *Detailed high-resolution USS ± CT* of the neck should be used to accurately stage the patient preoperatively.
- In patients where preoperative serum *calcitonin* is significantly raised (>400pg/mL), the presence of distant disease should be actively sought. This will involve high-resolution CT scanning ± MRI scanning in the first instance. PET-CT and bone scintigraphy can provide additional information.

Treatment

Surgical treatment

- The aim of surgery is to provide local control, with the aim of clinical and biochemical cure. Patients with phaeochromocytoma should have this treated before the thyroid cancer.
- *Total thyroidectomy and level VI (central compartment) lymph node dissection* is the minimum required operation in preoperatively diagnosed MTC.
- Central compartment lymph node metastases are seen in up to 80%.
- *Selective lateral compartment (levels II to V) lymph node dissection* is guided by radiological/clinical evidence of disease.
- *Prophylactic lateral neck dissection* in the absence of clinically apparent disease is controversial but may be guided by baseline calcitonin levels.
- *Prophylactic thyroidectomy* is performed in childhood in *RET proto-oncogene-positive* individuals. The timing of surgery depends on the codon affected, with highest-risk individuals recommended for *total thyroidectomy* in the first year of life.
- Lymphadenectomy can be avoided in such cases when preoperative calcitonin is normal. In patients with elevated preoperative calcitonin, the procedure is no longer prophylactic, but therapeutic and should be managed appropriately.

Follow-up and prognosis

- 10y survival rates vary from 56% to 96% at 10y, with survival in >95% when biochemical cure is achieved.
- Calcitonin ± CEA are used as post-operative tumour markers; calcitonin *doubling time* of <12 months indicates a worse prognosis.
- Rising *tumour markers* should prompt a search for recurrent/persistent disease using the imaging modalities listed above.
- *Adjunctive treatments* for non-resectable/symptomatic disease: *external beam radiotherapy*, *tyrosine kinase inhibitors*, *radiolabelled somatostatin analogues*, *chemotherapy*, and *radiofrequency ablation*.
- For newly identified FMTC/MEN kindred, all 'at-risk' family members should be offered genetic testing and counselled appropriately.

Anaplastic thyroid cancer

Key facts

- *Anaplastic (ATC)* or *undifferentiated* thyroid cancer is a rare (1–2 per million), aggressive disease with a dismal prognosis.
- It accounts for <2% of thyroid cancers, but >50% of thyroid cancer-related deaths.

Clinical presentation

- Patients typically present with variable 'red flag' symptoms, including *rapidly enlarging* neck mass, *dysphagia*, *hoarse voice*, *stridor*, *lymph node* mass, weight loss, anorexia, or symptoms from metastatic disease.
- *Elderly* ♀ (>70y) are most commonly affected.

Pathology

- Linked to loss of *p53* tumour suppression gene.
- Squamoid, spindle cell, and giant cell types.
- At least 50% are thought to arise from pre-existing DTC.
- *Poorly differentiated* thyroid cancer has an intermediate prognosis between DTC and ATC.
- *Staged* with TNM following USS of the neck and whole-body CT.

Management and prognosis

- Median survival 7 months for localized disease; 3 months with metastases.
- *Total thyroidectomy ± selective resection of structures and nodes* is possible in some, if deemed resectable disease.
- *Palliative surgery*, including *tracheostomy*, sometimes required to maintain an airway.
- *External beam radiotherapy* as adjuvant therapy post-operatively or as palliation for 1° disease or distant metastases.
- Limited role for *chemotherapy*, but clinical trials should be considered to determine the role of novel therapeutic agents.

Thyroid lymphoma

Key facts

- Rare thyroid malignancy.
- *Tumour of mucosa-associated lymphoid tissue (MALToma)*; classified as diffuse B-cell non-Hodgkin's lymphoma.
- Can rarely occur as a complication of long-standing Hashimoto's thyroiditis.
- Recognition of diagnosis is critical as the optimal management is oncological (*chemoradiotherapy*), with little or no role for surgery.

Post-operative emergencies

Haemorrhage

- The reporting of *post-operative haematoma* varies. Most have no particular clinical consequences, will resolve without complication, and can be managed expectantly.
- ~1% of thyroidectomy procedures will be complicated by a significant haematoma, which compromises the upper airway.
  - The mechanism is *laryngeal oedema*, not direct compression.
  - Patients present with respiratory distress and stridor.
  - Most commonly within the first *4h* post-operatively, but may occur within the first 24h (1° *bleed*) or up to several days post-operatively in conjunction with an infection (2° *bleed*).

Management

- *Call for senior help* (an anaesthetist with 'difficult airway' skills preferable).
- *Open the wound*; acute respiratory distress and pending airway loss mandate opening the wound and evacuating the haematoma at the bedside.
- *High-flow O_2 therapy, IV access, IV fluids ± IV steroid treatment* and *tranexamic acid* (if no contraindications).
- In life-threatening situations, *intubation* and *return to theatre* to control the bleeding should be undertaken as early as possible.

Acute bilateral recurrent laryngeal nerve palsy

- *Very rare occurrence*.
- Causes include *poor surgical technique*, *misplaced cervical plexus nerve block*, and *unilateral injury (temporary/permanent)* with failure to diagnose existing *contralateral injury* preoperatively.
- Presents with *acute upper airway compromise* due to *bilateral vocal cord palsy*, usually on extubation.
- May be *temporary* (commonest) or *permanent*.

Management

- *Anaesthetist* should attempt re-intubation.
- Patient will need management on ITU. A *tracheostomy* is often required and may be temporary or permanent.
- 1° *prevention* with *preoperative vocal cord check* and sound intraoperative technique should be the aim.
- Adjuncts such as *steroids* may be utilized under specialist supervision.

Hypocalcaemia

- Can occur following *total thyroidectomy*, subtotal or total *parathyroidectomy*, or *re-operative neck surgery*.
- *Temporary hypocalcaemia* may occur in up to 20% of patients, post-total thyroidectomy. *Permanent hypocalcaemia* rates should not exceed *5%*.
- Caused by *devascularization/trauma* or removal of parathyroid glands (PGs); usually requires injury to ≤2 PGs before clinical sequelae noticeable.
- Patients usually present with *perioral paraesthesiae* and/or *paraesthesiae in the limb extremities*.

- More *severe* hypocalcaemia can present with *carpopedal spasms* and *tetany* and *chest pain*.
- *Laryngospasm* and *cardiac arrhythmias*, with associated long QT changes on ECG, are *life-threatening* complications.
- *Chvostek's sign* (tapping on the facial nerve at the parotid) and *Trousseau's sign* (inducing carpal spasm with upper limb sphygmomanometer) may be *positive*.

Management

- Units should have an established local policy for management of post-operative hypocalcaemia (see https://www.baets.org.uk for an example).
- *Severe symptoms* or Ca^{2+} *<1.80mmol/L* should be managed with *calcium gluconate* (10mL of 10% in 100mL of normal saline over an hour, followed by ongoing infusion) and PO calcium supplementation ± *1α-calcidol using cardiac monitoring*.
  - *Serum* Mg^{2+} should be checked in severe hypocalcaemia due to the metabolic link between these cations.
- *Asymptomatic patients* with Ca^{2+} *>2.00mmol/L* or patients with *symptoms* and Ca^{2+} *>2.10mmol/L* require reassurance and repeat Ca^{2+} assay.
- Patients falling between these groups are generally managed according to symptoms with *PO calcium supplementation* (e.g. *Calcichew® D3 Forte tds to qds)* ± *PO or IV* alfacalcidol until asymptomatic with stable serum Ca^{2+}.

Thyroid storm (thyrotoxic crisis)

- Rare. Extreme manifestation of thyrotoxicosis with severe metabolic disturbances. Precipitated by surgery, major trauma, iodinated contrast administration, and infection. Usually encountered in patients on ITU.
- Insomnia, anorexia, vomiting, fever, tachyarrhythmias, diarrhoea, and sweating. High mortality rate unless recognized and treated rapidly.

Management

- Initial treatment is aggressive rehydration with *IV crystalloids via large-calibre cannula*.
- Supplement with *high-flow* O_2 via non-rebreathing mask.
- Urinary catheterization ± *central venous/arterial monitoring* to guide resuscitation should be undertaken in a high-care environment (*HDU/ITU*).
- Further management should be guided by a *specialist* and may include *ATDs*, drugs to inhibit thyroid hormone release (*propylthiouracil*), drugs to limit the peripheral effects (β-*blockers*), steroids (*hydrocortisone, dexamethasone*), and *paracetamol*.

The parathyroid glands

Anatomy

- Most people have *four PGs*, with a pair of *superior* and *inferior* glands in the central neck *bilaterally*.
  - Occasionally patients have supernumerary glands, and rarely <4 glands.
- The *superior PGs* develop *embryologically* from the fourth pharyngeal pouch and have a consistent position in close relation to the upper pole of the thyroid.
- The *inferior PGs* have an embryological origin common with the *thymus* (*third pharyngeal pouch*), and have a more variable position between the *lower pole of the thyroid and the thymus* in the anterior mediastinum, due to their longer pathway of decent.
- *Ectopic PGs* occur and pose a challenge to the parathyroid surgeon in pathological states.
- The *blood supply* to the PGs is derived from the *superior and inferior thyroid arteries*, with venous drainage via the thyroid veins.

Physiology

- The function of the PGs is to maintain *serum Ca^{2+}* concentrations via its hormone *parathyroid hormone* (parathormone, *PTH*).
- This is achieved by *Ca^{2+}-sensing receptors* on the surface of parathyroid cells, which detect fluctuations in serum Ca^{2+}.
- Stimulation of these cells due to a fall in Ca^{2+} leads to the secretion of PTH, which has a half-life of 4min, and an increase in serum ionized Ca^{2+}.
- PTH exerts its actions *directly* with *metabolic effects* on the bone and *kidneys*, and indirectly by increasing the synthesis (via hydroxylation) of *active vitamin D (calcitriol, 1,25-dihydroxyvitamin D3)*.
  - Its action on bone leads to demineralization and release of Ca^{2+} and phosphate.
  - Its renal effects are to increase Ca^{2+} reabsorption, whilst increasing phosphate excretion. In addition, it increases hydroxylation to calcitriol undertaken by the kidneys, whilst potentiating its actions.
- Calcitriol acts to increase Ca^{2+} absorption from the GI tract.
  - In the kidneys, calcitriol reduces renal excretion of Ca^{2+} and phosphate.
- *Vitamin D deficiency is very common* in many regions and can lead to a physiological increase in serum PTH (*2° hyperparathyroidism*).

Primary hyperparathyroidism

Key facts

- Primary hyperparathyroidism (pHPT) is the commonest cause of hypercalcaemia.
- Population prevalence is 1 in 500, but occurs in up to 190/100 000 post-menopausal women.

Pathological features

- 85% are caused by autonomous function of a *single parathyroid adenoma*.
  - *Hyperplasia* is seen in ~10–15%.
  - *Double adenoma* is present in 2% of pHPT.
  - *Parathyroid carcinoma* is rare, accounting for <1%.
- pHPT occurs as the major component of *MEN1* and should be considered in patients presenting <40y old. Mild pHPT is associated with *MEN2*.
- *Normocalcaemic pHPT* is described, but its clinical significance is unclear.

Clinical features

- Most cases of pHPT are diagnosed incidentally due to a serum Ca^{2+} check for other reasons.
- Many of the symptoms associated with pHPT/hypercalcaemia are non-specific, although a classical combination of symptoms is described.
  - Many use the mnemonic groans, bones, stones, and psychic groans.
- pHPT is a disorder of multiple systems:
  - *Bone disease*: reduced bone mineral and bony pain, and rarely *osteitis fibrosa cystica*.
  - *Renal disease*: Ca^{2+}-predominant *renal calculi*, polyuria, polydipsia, reduced renal function.
  - *GI*: nausea, vomiting, constipation, *pancreatitis*, *peptic ulcer*.
  - *Psychological*: lethargy, low mood or depression, confusion, coma.
  - *Cardiovascular*: short QT, bradycardia, LV dysfunction.

Diagnosis and investigations

- The diagnosis is biochemical with a high serum Ca^{2+} and either elevated (85–90%) or inappropriately normal (10–15%) PTH level.
- Vitamin D levels should be checked to ensure replete stores.
- The main differential diagnosis is the rare *autosomal inherited* disorder *familial hypocalciuric hypercalcaemia (FHH)*.
  - Ca^{2+}-sensing receptor threshold altered.
  - Excluded by calculating Ca^{2+}/Cr clearance ratio.
  - *Genetic testing* may be offered, but no treatment is required.
- Drugs which lead to a clinical picture mimicking pHPT include *lithium* and *thiazide* diuretics.
- *Preoperative localization* studies include high-resolution USS and 99mTc-sestamibi nuclear medicine scans.
- *Four-dimensional (4D) CT scanning* is utilized routinely in some centres but involves high doses of ionizing radiation. It is a useful imaging technique following failed 1° surgery, to identify ectopic PGs.
- *Single-photon emission CT (SPECT)/CT or MRI* may also be used for re-operative surgery; *CT angiography* and *selective venous catheterization* are rarely used.

Management

- Parathyroidectomy in pHPT is indicated for symptomatic disease and asymptomatic disease in the following circumstances:
  - Age <50y.
  - Serum Ca^{2+} >0.25mmol/L above the normal range.
  - Cr clearance <60mL/min.
  - Reduced bone mineral density at key skeletal sites.
  - 24h urine Ca^{2+} >400mg/day.
  - Proven urinary tract calculi.
- Negative localization studies increase the likelihood of *multi-gland disease*.
- *Intraoperative PTH (ioPTH) assay* can increase the proportion of patients achieving biochemical success at initial surgery.
- The presence of >1 enlarged PG, negative localization (in the absence of ioPTH), or failure of ioPTH to fall following removal of a gland should mandate *bilateral neck exploration* due to the high likelihood of multi-gland disease.
- Patients with concordant preoperative localization studies ± significantly elevated serum Ca^{2+} may be suitable for a *targeted (unilateral) parathyroidectomy*.
- *Video-assisted parathyroidectomy*, *endoscopic parathyroidectomy*, and *robotic parathyroidectomy* have all been described.

Prognosis and follow-up

- In experienced centres, biochemical cure can be achieved in 97% of patients when the adenoma is localized preoperatively, and 93% when localization studies are negative.
- Patients should be followed up for a minimum of 6 months to ensure ongoing biochemical normality and cure.

Parathyroid cancer

Key facts

- Rare (<1%) cause of hypercalcaemia in pHPT.
- May present as a firm neck mass or with symptoms of invasion (e.g. hoarse voice). Often diagnosed peri-operatively.
- Associated with significantly elevated Ca^{2+} and PTH levels.
- If suspected, *en bloc resection* of tumour and adjacent thyroid lobe should be performed.
- Post-operative *radiotherapy* may be considered. Prognosis is generally poor.

Renal hyperparathyroidism

Key facts

- *Secondary hyperparathyroidism (sHPT)* is characterized by excessive levels of PTH in response to a chronic reduction in serum Ca^{2+}.
- *Vitamin D deficiency* is the commonest underlying mechanism and may be either dietary or due to renal failure (*renal HPT*).
- 10% of patients with renal HPT will require *parathyroidectomy* after 10–15y of RRT.

Pathology

Renal HPT results from:

- Phosphate retention from a *reduction in glomerular filtration*. This leads to a reduction in serum Ca^{2+}.
- *Hyperphosphataemia* stimulates PTH secretion.
- *Reduced renal activation of vitamin D* leads to reduced intestinal Ca^{2+} absorption.
- Elevated PTH acts on bones to demineralize them and may lead to compensatory hypercalcaemia.
- *Four-gland hyperplasia* is seen in renal HPT.

Clinical presentation

- *Biochemically*, patients will have evidence of *chronic kidney disease*, usually severe or end-stage, and excessive *PTH*. Serum *Ca^{2+}* can be low, normal, or elevated.
- Patients may be asymptomatic.
- Reduction in bone mass density or *pathological fractures* may be the presenting feature.
- *Symptoms* such as *bone pain*, *pruritus* (due to excessive PTH), *muscle weakness*, and *psychiatric* symptoms, such as irritability or depression, can often be experienced.
- *Rarely*, patients present with *calciphylaxis*; vascular calcification and skin necrosis associated with high morbidity and mortality rates.
  - This usually occurs in the context of known renal HPT.

Management

Medical treatment

- The aims of medical treatment are to maintain serum Ca^{2+} and phosphate levels within target ranges, restore bone minerality, and reduce morbidity from an excessively elevated PTH. Options include:
  - *Low phosphate diet*.
  - *Phosphate-binding compounds*.
  - *Vitamin D supplementation* (alfacalcidol); this may be the only treatment required in vitamin D deficiency alone.
  - *Calcimimetics* (e.g. cinacalcet) which inhibit the Ca^{2+}-sensing receptors and temper PTH secretion.

Surgical treatment

- There are several relative indications for surgery, most relating to failure of medical therapy in severe renal HPT.
- Failure to maintain Ca^{2+} or phosphate targets; often calculated as a *Ca^{2+} × phosphate product*.
- A *significantly elevated PTH* refractory to treatment, especially when combined with intractable pruritus.
- *Osteoporosis, pathological fracture, bone pain*.
- *Calciphylaxis*.
- Severe *psychological symptoms*.
- *Patient preference* following counselling.

There are several options available to endocrine surgery, with no consensus regarding the optimum procedure:

- *Total parathyroidectomy with autotransplantation*. Excision of all four PGs, with a 'minced' remnant of a single gland autotransplanted into the forearm or the SCM muscle.
- *Subtotal parathyroidectomy*. Excision of three glands, with a small remnant of the fourth gland left *in situ*.
- Preoperative localization is NOT required.
- Care should be taken not to rupture the PG capsule due to the risk of *parathyromatosis* resulting from the ongoing stimulus for PTH secretion.
- Procedures retaining some parathyroid tissue may be preferred in patients likely to undergo *renal transplantation* due to the potential for recovery of 'normal' Ca^{2+} homeostasis.
- Post-operative management of renal HPT patients can be complicated and should be undertaken in close liaison with a nephrologist or endocrinologist.

Tertiary hyperparathyroidism

Key facts

- Only occurs in patients with a history of renal HPT who undergo successful *renal transplantation*.
- Once the metabolic disarray of renal failure is corrected, the majority of patients will regain normal Ca^{2+} homeostasis.
- A small proportion of patients (4%) will become (remain) hypercalcaemic with now *inappropriately elevated PTH* (as in pHPT).
- This is due to the development/persistence of autonomous secretion from all *PGs*.
- Homeostasis can take 12–18 months to normalize, and surgery (rarely indicated) should not be considered during this period of time.

Adrenal incidentaloma

Key facts

- An *asymptomatic adrenal mass* discovered by chance on *imaging for non-adrenal disease*.
- Found in *5%* of high-resolution abdominal scans.
- The *risk of malignancy* in unselected adrenal incidentaloma is *0.1%*.
- The vast majority of cases will be benign, *non-functioning adenomas*.

Clinical presentation

- By definition, patients will be *asymptomatic* at presentation.
- In some patients, a detailed *history and examination* may elicit signs or symptoms suggestive of a *functional adrenal lesion*.
- Review of *previous imaging* may provide further information regarding the chronicity of the presenting lesion.

Diagnosis and investigations

- The diagnosis of 'incidentaloma' will be made based on imaging.
- Key questions in investigation are:
  - Is the tumour *functional* or non-functional?
  - Is the tumour benign or *malignant*?
  - Are there *indications to resect* the tumour?
- *Functionality* is assessed with a minimum of a *low-dose (1mg) overnight dexamethasone suppression test* (*cortisol-secreting/Cushing's* tumour) and a *24h urinary metanephrine* analysis (or equivalent) (*phaeochromocytoma*).
- In patients with *hypertension*, plasma K^+ (Na^+) and *aldosterone:renin activity* should be measured (*Conn's* tumour).
- A *non-contrast CT* scan is the initial investigation of choice.
- *MRI or PET-CT* can provide additional information if required.
- Malignant potential can be assessed with radiological and clinical information.
  - Tumours under *4cm* are rarely malignant (≤1%).
  - Lesions that are *lipid-rich* on non-contrast CT are less likely malignant.
  - A rapid increase in size, irregular outline, heterogenous contrast uptake, or excess secretion of multiple hormones, including sex hormones, are associated with malignancy.
  - Local invasion or distant metastases indicate malignancy.
- *Adrenal biopsy* is rarely indicated, although may be utilized in the diagnosis of an adrenal metastasis.
  - Biopsy of a phaeochromocytoma may precipitate a life-threatening *hypertensive crisis*.
  - Tumour seeding may occur following biopsy of an adrenocortical carcinoma (ACC).

Management

Conservative management

- *Non-functioning* tumours, <4cm, and with no evidence of malignancy can be safely observed.
  - Follow-up cross-sectional imaging at 3–12 months is generally recommended.
- *Functioning* tumours can be managed medically or operatively, depending on local protocols.
- The overall risk of malignancy in *tumours >4cm* is ~10%, and most recommend *surgery* beyond this size threshold.
  - Some advocate *conservative management* for *tumours 4–6cm* when certain low-risk criteria are present, although there is no consensus.
- *Functional tumours* can be managed medically in patients with responsive disease or risk factors which preclude surgical intervention.
- In patients presenting with *advanced ACC*, the anti-adrenal treatment *mitotane* or *palliative chemotherapy* may be used. This should be done in the context of *clinical trials*.

Surgical management

- *Laparoscopic adrenalectomy* is the procedure of choice in the majority of adrenal incidentalomas.
  - Can be considered in tumours <6cm with some suspicion of malignancy, but *no obvious local invasion*.
  - For benign ± functioning tumours, the upper size limit for laparoscopic adrenalectomy will be determined by local expertise, with the risk of conversion ↑ only if a tumour >6–8cm is encountered.
  - This procedure can be undertaken via a *transperitoneal* approach or a *posterior (retroperitoneal)* approach.
- *Open adrenalectomy* is recommended for large tumours in which the chances of successful laparoscopic resection are low.
  - Open adrenalectomy is mandated in cases demonstrating local invasion. *En bloc resection* of local structures is recommended to improve survival.
  - *Debulking open adrenalectomy* is sometimes indicated in advanced ACC as *palliation* for complications from hormone excess.
- *Partial adrenalectomy* is occasionally performed for bilateral disease to preserve some endogenous adrenal function and limit reliance on post-operative steroid treatment (especially in MEN2).
- In patients with preoperative cortisol excess, *perioperative steroids* should be administered to prevent *Addisonian crisis*.
- Following adrenalectomy, a 9.00 a.m. random cortisol test may be useful in identifying patients in need of steroid replacement. A short Synacthen® test should be arranged post-operatively to ensure contralateral gland recovery prior to stopping steroid supplementation.

Cushing's syndrome

Key facts

- A highly comorbid condition driven by *excessive* levels of circulating *glucocorticoids*.
- Rare condition affecting 4 per million; predominantly women.
- Untreated has a *mortality rate* approaching *50% at 5y*.
- *Iatrogenic Cushing's syndrome* is caused by administration of *exogenous steroids* and is commoner.

Pathology

- The spectrum of disease is driven by *excess glucocorticoid secretion* from the *zona fasciculata* of the adrenal cortex.
- *Cushing's disease* is an *adrenocorticotrophic hormone (ACTH)-dependent* disease caused by *pituitary pathology* (commonest).
- *Ectopic ATCH secretion* (e.g. small cell lung cancer) can also produce a similar clinical picture.
- *Cushing's syndrome* can be ACTH-independent, caused by 1° adrenal pathology.
  - Unilateral disease: adenoma or ACC.
  - Bilateral disease: macronodular adrenal hyperplasia commonest.
- *Subclinical Cushing's syndrome* can occur in asymptomatic patients with elevated levels of circulating glucocorticoids.

Clinical presentation

- Peak presentation ages *30–40y*.
- Multisystem disease due to widespread effects of glucocorticoids; many presenting features are common in the general population and non-specific.
- *Weight gain*. Central or truncal obesity, with 'buffalo hump'.
- *Muscle wasting*. Typically proximal limb muscle groups.
- *Facial plethora ('moon' face)*.
- *Hirsuitism, acne, thin skin, easy bruising, striae*.
- *Dysmenorrhoea*.
- *Osteoporosis*; metabolic bone disease.
- *Psychological disturbances*.
- *HTN*; partial mineralocorticoid effects.
- *Congestive cardiac failure*.
- 2° *DM*; significant metabolic changes.
- *Red flag symptoms*, *virilization*, and symptoms/signs of local *compression/infiltration* may suggest ACC.
- *Skin pigmentation* may occur in ectopic ACTH secretion.
- Symptoms/signs of *pituitary mass (bilateral hemianopia)* suggest *Cushing's disease*.

Diagnosis and investigations

- High *index of suspicion* is the key to diagnosis.
- A detailed history and examination for *Cushing's-related comorbidities* should be undertaken.
- *Establishing hypercortisolism* (loss of circadian rhythm).
  - Low-dose (1mg) overnight dexamethasone suppression test.
  - Late night salivary cortisol assay.
  - Urinary free cortisol has lower specificity.

- *Establishing cause*.
  - *Plasma ACTH*. Elevated levels suggest ACTH-dependent cause and an *MRI pituitary* should be undertaken to identify *pituitary adenoma*.
  - *Confirmed ACTH independence*. *CT abdomen* indicated to identify possible adrenal lesion.
- In ACTH-dependent disease with a normal MRI pituitary, further imaging (*CXR/CT/MRI chest/abdomen*) is indicated to look for a source of ectopic ATCH secretion.
- *Bilateral inferior petrosal sinus sampling* may distinguish pituitary from an ectopic cause in ACTH-dependent disease.

Management

Medical treatment

- All patients with adrenal Cushing's syndrome should be managed in conjunction with a *specialist endocrinologist*.
- *Metyrapone, ketoconazole, or etomidate* may be utilized preoperatively in severe disease under the guidance of a specialist.
- *Perioperatively*, most patients will require steroid cover.
  - IV hydrocortisone 50–100mg tds until PO diet established.
  - *PO hydrocortisone 50–100mg tds/PO prednisolone 3mg od* until recovery of the suppressed contralateral gland (may take up to 12 months).
  - *Bilateral adrenalectomy*. Lifelong steroid treatment and mineralocorticoid replacement (*fludrocortisone 0.1mg od PO*).
  - Patients on steroids should be given a *steroid card*.

Surgical treatment

- Cushing's disease should be referred to a neurosurgeon for consideration for *trans-sphenoidal resection* of the pituitary adenoma.
- *Laparoscopic adrenalectomy* is the procedure of choice for the majority of cases of ACTH-independent Cushing's syndrome.
- *Open adrenalectomy* is undertaken if ACC is suspected preoperatively.
- *Bilateral laparoscopic (or open) adrenalectomy* is occasionally required for ACTH-dependent disease following failed pituitary surgery or gamma-knife ablation, or for palliation from ectopic secretion.
  - May be required in cases of bilateral adrenal hyperplasia.
- An adrenal tumour with *subclinical Cushing's* syndrome should also be considered for surgery if the patient is a suitable candidate.

Conn's syndrome

Key facts

- 1° *hyperaldosteronism* or Conn's syndrome is a cause of refractory HTN (up to 10% of cases of HTN in 2° care).
- ♀:♂ ratio is 3:1.
- Caused by *autonomous mineralocorticoid (aldosterone) secretion* from the *adrenal zona glomerulosa*.

Pathological features

- A significant proportion of cases are caused by a *solitary aldosterone-secreting adenoma*, as originally described by Jerome Conn.
- Conn's adenomas are generally small (<2cm), with a significant proportion constituting *microadenomas* (<10mm).
- *Idiopathic bilateral adrenal hyperplasia* (up to 60% of cases) can be asymmetrical but needs excluding prior to consideration of surgery.
- Rare familial causes of hyperaldosteronism exist:
  - *Type I. 11β-hydroxylase mutation*—suppressed with dexamethasone.
  - Type II. Autosomal dominant—not suppressible.

Clinical presentation

- *HTN* is the commonest presentation. This can be moderate to severe and refractory to treatment.
  - Should be considered in young patients on multiple antihypertensive medications.
- *Hypokalaemia* may be identified incidentally on biochemical analysis or present with symptoms such as *cramps, muscle weakness, fatigue, thirst, polyuria, or nocturia*.
  - Severe hypokalaemia may present with cardiac complications or ECG changes.
  - 50% are normokalaemic.
- *Cardiac* or *cerebrovascular* disease are commoner than with essential HTN.

Diagnosis and investigations

- A diagnosis or suspicion of Conn's syndrome should prompt referral to a *specialist endocrinologist*.
- A thorough history and examination should include a detailed *family history* and *BP* evaluation
- U&E measurements may demonstrate *hypokalaemia* and any end-organ renal dysfunction. There may be concurrent *alkalosis*.
- *Plasma aldosterone:renin ratio (ARR)*.
  - *Renin* will be suppressed (<0.5pmol/mL/h) in the context of raised *aldosterone* levels (>250pmol/L).
  - Medications that interfere with this should be discontinued prior to testing (e.g. β-*blockers*, *spironolactone*, ACE inhibitors, angiotensin II blockers).
  - *Hypokalaemia* should be corrected prior to testing.
  - An ARR of >2000 is likely diagnostic of Conn's syndrome.
- Aldosterone will not suppress with *fludrocortisone* or *saline suppression testing* in cases of Conn's syndrome.

- *Adrenal CT scanning* may show *unilateral adenoma*, *bilateral hyperplasia* or *nodularity*, or be *normal*.
  - *Adrenal MRI* has a similar diagnostic yield to CT.
- Patients with 1° hyperaldosteronism under consideration for surgical resection should undergo *lateralization testing* preoperatively due to the high prevalence of non-functioning adrenal adenomas in the general population.
  - *Adrenal venous sampling*. Adrenal and cortisol levels compared prior to, and following, Synacthen® stimulation.
  - *^{11}C-metomidate PET-CT* is a non-invasive alternative to venous sampling studies, but its availability is limited in most centres.
- Failure to lateralize is suggestive of *bilateral disease* and *medical management* should be considered as first-line treatment.

Treatment

Medical management

- *Spironolactone* is an aldosterone receptor inhibitor and may be used to *correct K^+* and *lower BP*.
  - High affinity for *sex hormone receptors*, with consequential side effects, including *gynaecomastia*, reduced libido, and *menstrual irregularities*.
- Coexisting essential HTN may require treatment with additional classes of antihypertensive drugs.

Surgical management

- The relative indications for surgery are:
  - Unilateral Conn's adenoma.
  - True unilateral (and localized) adrenal hyperplasia.
  - *Adrenal carcinoma* secreting aldosterone ± additional hormones.
  - Rarely type II familial hyperaldosteronism.
- *Laparoscopic adrenalectomy* is the surgical approach of choice in the vast majority of Conn's syndrome patients.
  - Transperitoneal or retroperitoneal approaches possible.
- *Open adrenalectomy* is utilized in cases of known or suspected ACC.
- *Hypokalaemia* must be corrected preoperatively to minimize risks of perioperative period.
  - *Potassium supplementation* and *spironolactone* can usually be withdrawn post-operatively.
  - *K^+* levels should be closely monitored in the post-operative period.
- *50%* of patients will *stop all antihypertensive medications* post-operatively, with a further *40%* able to reduce their medication requirements.

Phaeochromocytoma

Key facts

- Rare catecholamine-secreting tumours of the adrenal medulla.
- Incidence is 2 per million.

Pathology

- The majority of tumours secrete predominantly *noradrenaline* or *adrenaline*. Some tumours have *mixed* secretion or secrete *dopamine*.
- Phaeochromocytomas: *benign (90%)* or *malignant*, *uni- or bilateral*.
- *Paragangliomas* are similar tumours originating from other tissues of *neural crest origin* such as the sympathetic chain.
- A proportion of phaeochromocytomas (up to 30%) occur as part of a genetic disorder. Such tumours are more likely to be *malignant*, *bilateral*, or *extra-adrenal*.
  - MEN2 or MEN3, von Hippel–Lindau (VHL) syndrome, neurofibromatosis type 1, *succinate dehydrogenase* gene mutations.

Clinical presentation

- Patients *may be asymptomatic* or present with classical symptoms.
- Symptoms/signs classically include *continual or intermittent HTN* (>90%), *headache* (90%), *sweating* (70%), and *palpitations*.
- Other features include *anxiety*, *pallor*, and *flushing*.
- *Paroxysmal 'attacks'* may be precipitated by alcohol, stress, labour, GA, and direct manipulation (trauma, surgery).
- *Hypotension* can be the dominant feature in predominantly *dopamine-secreting* phaeochromocytomas.
- *10%* are discovered *incidentally* on abdominal imaging.

Diagnosis and investigations

- Thorough *history and examination* should cover a comprehensive review of the *cardiovascular system* and coexisting disease.
- *Hypercalcaemia* and *glucose intolerance* may be present, and evidence of coexisting endocrine disease should be excluded.
- *24h urinary or plasma metanephrine assays* are the most effective tests for confirming the diagnosis. False negative/positive results may occur with certain medications.
- *CT* or *MRI* abdomen is the most appropriate initial investigation.
- *^{123}I-metaiodobenzylguanidine (^{123}I-MIBG)* or whole-body cross-sectional imaging may be required to identify a *paraganglioma*.
- *^{18}F-flurodopamine PET* scanning is more sensitive for extra-adrenal disease but is not widely available in many centres.

Treatment

Medical management

- *All patients* should have their *BP controlled* preoperatively.
  - α-*blockade* (e.g. *phenoxybenzamine 10mg bd* titrating upwards to achieve postural hypotension—patients will often complain of nasal congestion when adequately blocked).
  - β-*blockade* (e.g. *propranolol*) can be initiated once α-blockade and HTN are controlled, to control β-adrenergic effects.
  - *Doxazosin* is an alternative method of blockade.
- *Adjuvant therapies* are available for *metastatic phaeochromocytoma/paraganglioma*; 'debulking' surgery can improve treatment efficacy.
  - Radioactive ^{131}*I-MIBG*, *chemotherapy*, or *tyrosine kinase inhibitors* all have a role in malignant disease.

Surgical treatment

- *Laparoscopic transperitoneal* or *retroperitoneal adrenalectomy* is preferred for phaeochromocytomas, even when tumour is >10cm.
- *Open surgery* (laparotomy/thoracotomy) is preferred for *extra-adrenal* disease due to the higher incidence of malignancy.
  - This approach may convey *oncological benefit* in malignant cases; en bloc excision of the tumour and involved structures preferred.
- Care should be taken when handling the tumour to limit catecholamine secretion and capsular disruption.
- Close liaison with an *experienced anaesthetist* is required, especially as the tumour is devascularized and the BP can drop markedly.
- *HDU/ITU care* may be required post-operatively as *vasopressor agents* may be required to support BP following surgery.

Adrenocortical carcinoma

Key facts

- *Very rare* tumour: 1–3 per million population, with poor prognosis.
- *Bimodal distribution*: <5y and 30–40y.

Presentation and investigations

- May be *asymptomatic*, present with symptoms/signs of *hormone secretion* or with *evidence of metastatic or advanced disease*.
- *Virilization* (♀) and *feminization* (♂) suggests malignancy.
- *CT abdomen*. Irregular, heterogenous mass.
- *PET scanning*. Useful to assess or exclude metastatic disease.

Treatment

Surgical treatment

- *Open adrenalectomy* is mandated for ACC surgery.
  - Radical en bloc resections are required for locally advanced, operable disease to increase possibility of negative resection margins.
- *Cytoreductive surgery* is occasionally required as palliation from intractable symptoms associated with hormone excess.
- The *Weiss scoring system* is used for post-operative diagnosis.

Medical treatment

- *Mitotane* anti-adrenal chemotherapy is utilized in patients with advanced/metastatic disease.
- *IV steroids* and *thromboprophylaxis* should be administered perioperatively.

Multiple endocrine neoplasia

Key facts

- Rare group of *autosomal dominant* familial conditions affecting *multiple endocrine organ systems*.
- Known kindred are referred to specialist centres for management.

Clinical and pathological features

Multiple endocrine neoplasia type 1 (MEN1)

- Results from mutation in the *menin* gene on *chromosome 11*.
- Syndrome of the 'three Ps'; parathyroid, pituitary, and pancreas.
- Most (95%) will develop *pHPT* by 40y; all during lifetime—this almost always involves all four glands and should be treated as multi-gland disease.
- *Pancreatic neuroendocrine tumours*: 30–75%; can be uni- or *multifocal*, *benign* or malignant, and secrete one or multiple *polypeptides*.
  - *Insulinoma* and *gastrinoma* are the commonest.
  - *Gastrinoma* can result in *Zollinger–Ellison syndrome*; multiple peptic ulcers, severe reflux oesophagitis, with secretory diarrhoea.
  - Glucagonoma, somatostatinoma, and VIPoma are very rare.
- Rarely, foregut neuroendocrine tumours, adrenal carcinomas, lipomas, and tumours of the pineal gland are associated with MEN1.
- *Anterior pituitary tumours* occur in 15–40%.
  - Most commonly prolactinoma, followed by growth hormone (GH) (acromegaly) and ACTH-secreting tumours (Cushing's disease).

Multiple endocrine neoplasia type 2 (MEN2)

- Results from *RET proto-oncogene* mutations on *chromosome 10*.
- *MTC* usually presents in second or third decade.
  - Background of C cell hyperplasia; often multifocal tumours.
- *Phaeochromocytoma* occurs in ~50% of patients.
- *pHPT* occurs in ~10–15%. This is *usually mild*, and can be *single* or *multiple* gland disease.
- *High-risk codon mutations* are associated with a more aggressive pattern of disease, with earlier malignant transformation in the thyroid and ↑ likelihood of *malignant phaeochromocytoma*.

Multiple endocrine neoplasia type 3 (MEN3)

- *RET mutations* associated with *non-endocrine features* and an *absence of hyperparathyroidism*.
- Many are *de novo* mutations, making identifying patients difficult.
- *MTC* which *presents early*, in virtually *all patients*, has a *poorer prognosis* than sporadic MTC.
- *Phaeochromocytoma* in over 50%; frequently bilateral.
- *Mucosal neuromas*, *ocular* features, and *musculoskeletal* and *intestinal ganglioneuromas* (may present with GI bleeding).
- *Marfanoid body habitus* seen in 90% of patients.

Diagnosis and investigations

MEN1

- Known gene carriers will be entered into a *biochemical screening programme*, beginning in the *second decade* of life.
- 10% of *menin* mutations will occur *de novo*; therefore, siblings are not always at risk; appropriate *genetic counselling* should be offered.
- Patients will be screened for *Ca^{2+} and PTH*, and with *gut hormone profiles* (insulin, glucose, gastrin, insulin-like growth factor (IGF)-1, chromogranins), and *anterior pituitary hormone assay*.

MEN2

- *Genetic screening* of at-risk individuals will allow a diagnosis, and a risk assessment if the specific mutation is known.
- In known carriers, *surveillance* is with *thyroid USS and calcitonin*, and annual *24h urinary/plasma metanephrine analysis*.
- *Ca^{2+} and PTH* screening annually or if symptoms of *pHPT*.

MEN3

- Due to the high rate of *de novo* mutations and the poorer prognosis (patients rarely have children), *pre-disease identification is rare*.
- All paediatric patients presenting with MTC should be screened and be under surveillance for *phaeochromocytoma*.

Treatment

MEN1

- Once a *threshold Ca^{2+}* (e.g. 2.75mmol/L) has been exceeded, patients should be offered *subtotal parathyroidectomy and bilateral cervical thymectomy*. *Recurrence* is almost *inevitable*.
- Most *pancreatic tumours* are *benign*—where possible, *enucleation* or *distal pancreatectomy* is preferred.

MEN2

- Unless indicated earlier following surveillance, a *planned prophylactic total thyroidectomy* will be undertaken, the timing of which depends on the mutation identified.
- Phaeochromocytoma needs excluding preoperatively.
- The management of *phaeochromocytoma* is discussed under ➔ Medical management, p. 358.
  - *Malignant* disease and *bilateral* disease are commoner in the setting of MEN and should be managed appropriately.
- *Hyperparathyroidism* is often mild and should be treated as for sporadic pHPT, with excision of only abnormal glands.

MEN3

- *Prophylactic thyroidectomy* is rarely possible due to early onset of disease; therefore, a *therapeutic total thyroidectomy* and *central compartment lymph node dissection* as a minimum should be performed.
  - Some centres may perform *lateral neck dissections*, either as a routine or as guided by preoperative calcitonin levels.
  - All patients with *radiological evidence* of lateral neck involvement should undergo *therapeutic lateral neck dissection*.
- *Phaeochromocytoma* is managed as in *MEN2* patients.

Upper gastrointestinal surgery

Upper gastrointestinal endoscopy

Four types of endoscopic modality are commonly used to examine the upper GI (UGI) and pancreaticobiliary tracts.

Gastroscopy

Correctly termed oesophago-gastro-duodenoscopy (OGD). Allows direct visualization of pathology, biopsy, and therapeutic interventions.

Indications

- Investigation of dysphagia (Dysphagia, p. 370) (see Fig. 8.1).
- Investigation of dyspepsia, reflux disease, and upper abdominal pain.
- Investigation of acute or chronic UGI bleeding.
- Investigation of iron deficiency anaemia (with colonoscopy) and unexplained weight loss.
- Surveillance of Barrett's oesophagus and gastric ulcers.
- Therapeutic interventions for UGI pathology:
  - Balloon dilatation of benign strictures.
  - Endoluminal stenting of malignant strictures.
  - Injection, coagulation, or banding of bleeding sources, including ulcers, varices, tumours, and vascular malformations.
  - Resection of early neoplastic lesions in oesophagus and stomach (EMR).

Preparation and procedure

- Patient should be fasted for 4h (except in emergency indications).
- Can be performed with LA throat spray (lidocaine).
- Often performed with IV sedation (e.g. midazolam 2 mg).

Risks and complications

- *Cardiopulmonary adverse events*. Related to sedation and analgesia; includes aspiration pneumonia, respiratory depression and arrest, and cardiac events; commoner in frail, underweight, and elderly patients.
- *Perforation* (usually of the oesophagus). Median risk ~1 in 3000; highest in the elderly, with oesophageal pathology, and during therapeutic intervention.
- *Bleeding*. Commonest after biopsy or therapeutic procedure.

Endoscopic ultrasound (EUS)

Utilizes a video endoscope with an ultrasound probe at its tip.

Indications

- Staging of oesophageal, gastric, and pancreatic cancer.
- Investigation of pancreatic cysts/tumours.
- Investigation of possible distal common bile duct (CBD) stones.
- Guided biopsy of pancreatic, peri-oesophageal, perigastric mass, and lymph nodes.
- Guided drainage of pancreatic (pseudo) cysts.
- Guided coeliac plexus blockade.

Endoscopic retrograde cholangiopancreatography

Utilizes a side-viewing endoscope to access the biliary tree via the ampulla of Vater/duodenal papilla in the second part of the duodenum.

Indications

- Therapeutic interventions for pancreaticobiliary disease.
  - Extraction of CBD stones.
  - Stenting for CBD stones, strictures, tumours, post-operative bile leak, and biliary sepsis.
  - Obtain cytology for suspected cholangiocarcinoma/pancreatic tumours.
- Investigation of pancreatic disease (pancreatic duct strictures, pancreatic duct abnormalities).

Preparation and procedure

- Patient should be fasted for 4h (except in emergency indications).
- IV access required.
- Always performed with LA throat spray (lidocaine).
- Always performed with IV sedation (e.g. midazolam 5mg) and occasionally analgesia (e.g. pethidine 50mg or fentanyl 50mg).
- Performed under X-ray screening guidance, often in X-ray department.
- May be performed under GA.
- LFTs and coagulation screen obligatory prior to procedure.
- Post-procedure non-steroidal anti-inflammatory suppositories have been reported to reduce the risk of post-ERCP pancreatitis.

Risks and complications

- *Perforation* (of oesophagus or retroduodenum). Median risk ~1 in 1000; commoner in the elderly, with pathology, and during therapeutic interventions, especially sphincterotomy.
- *Bleeding* (1.3%). Commonest after biopsy or therapeutic procedures, especially sphincterotomy; usually controlled by balloon pressure, may require open surgery.
- *Post-ERCP pancreatitis* (3.5–5%). Risk factors for post-ERCP pancreatitis include: normal bilirubin, young age, ♀ sex, sphincter of Oddi dysfunction, pancreatic duct injection, sphinterotomy, and balloon dilatation of the sphincter.
- *Post-ERCP cholangitis* (1%). Particularly in jaundiced patients in whom the procedure has been unsuccessful.
- *Respiratory depression and arrest*. Related to oversedation; commonest in frail, underweight, and elderly patients.

Enteroscopy

Often termed 'push endoscopy'. Performed with long-length, thin-calibre endoscope, aiming to intubate beyond the duodeno-jejunal flexure/junction and visualize the upper small bowel.

Indications

- Investigation of undiagnosed UGI bleeding (possibly due to proximal small bowel pathology).
- Investigation of abdominal pain.
- Investigation of upper small bowel Crohn's disease.

Preparation and procedure As for gastroscopy.

Risks and complications As for gastroscopy.

Video capsule endoscopy (VCE)

Non-invasive method for diagnostic imaging of the entire small bowel.

Indications

- Locate suspected small bowel bleeding site.
- Diagnosis of suspected Crohn's disease.
- Diagnosis of suspected small bowel tumours.

Contraindications

- Swallowing disorders.
- Gastroparesis.
- Intermittent/subacute small bowel obstruction.
- Pacemakers/ICD.

Preparation and procedure

- Ambulatory or inpatient setting.
- Patients should fast for 12h.
- Patient is provided with a belt-worn sensor array fastened to the abdomen.
- Capsule ingestion—clear liquids after 2h and food/medication after 4h.
- Image acquisition and review—sensor arrays are removed after 8–12h and the recorded images are downloaded and processed on a workstation.

Risks/complications

- Capsule retention.
- MRI—patients should not undergo MRI until capsule passage has been confirmed.

Dysphagia

Key facts

The subjective sensation of difficulty or abnormality of swallowing. Dysphagia is an alarm symptom which always warrants investigation to exclude potential malignancy.

Classification

Oropharyngeal dysphagia

Difficulty initiating a swallow.

Oesophageal dysphagia

Difficulty swallowing several seconds after initiating a swallow and a sensation of food getting stuck.

Causes

Mechanical

- Malignancy—oropharyngeal, oesophageal (➲ Oesophageal tumours, p. 380), gastro-oesophageal.
- Benign stricture—peptic stricture, oesophageal web/ring.
- Extrinsic pressure—lung cancer, mediastinal lymph nodes, goitre.
- Pharyngeal pouch (➲ Pharyngeal pouch, p. 374).

Motility disorders

(➲ Oesophageal motility disorders, p. 372.)

- Achalasia.
- Diffuse oesophageal spasm.
- Systemic sclerosis.
- Neurological (bulbar palsy, myasthenia gravis).

Others

- Oesophagitis—candidiasis, reflux oesophagitis (➲ Gastro-oesophageal reflux disease, p. 378).
- Globus hystericus.

Diagnosis and investigations

An algorithm for the investigation of dysphagia is shown in Fig. 8.1.

'Urgent suspected cancer' referral (within 2 weeks)

- Dysphagia.
- Age >55y, with weight loss and any of the following:
  - Upper abdominal pain.
  - Reflux.
  - Dyspepsia.
  - Anaemia.
  - Abdominal mass.
  - History of previous abdominal surgery/malignancy.
- Suspicious finding on CT/barium swallow.

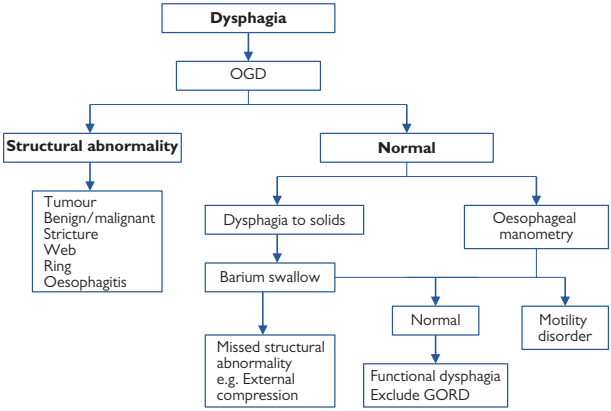

Fig. 8.1 Algorithm for investigation of oesophageal dysphagia.

Oesophageal motility disorders

Key facts A spectrum of diseases involving failure of coordination or contraction of the oesophagus and its related muscular structures.

Pathological features In some cases, degeneration of the ganglion cells within myenteric plexuses can be demonstrated, but often no structural abnormality is seen. Symptoms may be associated with other disease (e.g. systemic sclerosis).

Clinical features

Achalasia

- Peak ages of incidence in young adulthood (idiopathic) and old age (mostly degenerational).
- Slowly progressive dysphagia. Initially worse for fluids than solids.
- Frequent regurgitation of undigested food common late in the disease.
- 2° recurrent respiratory infection due to aspiration.

Diffuse oesophageal spasm

- Commonest in young adults; ♂ > ♀.
- Characterized by acute pain along the length of the oesophagus, induced by ingestion, especially of hot or cold substances (odynophagia).

Diagnosis and investigations

Suspect a motility disorder in any patient with oesophageal dysphagia, non-cardiac chest pain, or reractory GORD. It is crucial to first exclude malignancy.

Achalasia

- *OGD*. To exclude benign and malignant strictures.
- *Barium swallow*. A characteristic failure of relaxation of the lower oesophagus, with a smooth-outline 'rat's tail' or 'bird beak'.
- *Oesophageal manometry*. Hypertonic lower oesophageal high-pressure zone with failure of relaxation normally induced by swallowing; in chronic cases, the proximal oesophagus may be adynamic.

Diffuse oesophageal spasm

- *OGD*. Required to exclude underlying associated malignancy.
- *Video barium swallow*. 'Corkscrew' appearance of the oesophagus caused by discoordinated diffuse contractions.
- *Oesophageal manometry*. Diffuse hypertonicity and failure of relaxation; little or no evidence of coordinated progressive peristalsis during episodes, but normal peristalsis when asymptomatic.

Treatment

Treatment is aimed at decreasing the resting pressure of the LOS.

Achalasia

- *Endoscopically guided controlled balloon dilatation* (fixed pressure). Successful in up to 80% of patients; low complication rates (perforation); multiple procedures may be required over time.

- *Botulinum toxin injections*. Successful in some patients failing dilatation. Good for patients not suitable for invasive therapy.
- *Surgical myotomy (Heller's cardiomyotomy)*. Usually performed laparoscopically with division of the lower oesophageal circular muscle fibres; highly successful in resistant cases; mostly applicable to young patients. Specific complications include reflux, obstruction of gastro-oesophageal junction (GOJ), and oesophageal perforation.
- *Per-oral endoscopic myotomy (POEM)*. A relatively new endoscopic myotomy technique performed at specialized centres. The role of POEM in the treatment of achalasia remains controversial.

Diffuse oesophageal spasm

- Oral calcium channel blockers or relaxants, e.g. benzodiazepines.
- Long-acting nitric oxide donors (smooth muscle relaxant).
- Widespread oesophageal pneumatic dilatations (often repeated).
- Long surgical open myotomy, rarely performed.

Key revision points—anatomy and physiology of the oesophagus

- Upper two-thirds. Stratified, squamous, epithelial-lined (develops squamous carcinoma), striated skeletal muscle, lymphatic drainage to neck and mediastinal nodes, somatic innervation of sensation (moderately accurate location of level of pathology).
- Lower third. Transition to columnar epithelium (develops adenocarcinoma), transition to smooth muscle, lymphatic drainage to gastric and para-aortic nodes, visceral innervation (poor localization of pathology).
- The GOJ is site of portosystemic anastomosis (between left gastric and (hemi)azygos veins); may develop gastric or oesophageal varices.
- Upper oesophageal sphincter (UOS) = cricopharyngeus.
- LOS = functional zone of high pressure above the GOJ. Relaxants include alcohol.
- Swallowing requires intact and coordinated innervation from vagus nerve (UOS, oesophagus, LOS) and intramural myenteric plexus.

Pharyngeal pouch

Key facts

- An acquired 'pulsion' diverticulum arising in the relatively fibrous tissue between the inferior constrictor and the cricopharyngeus muscle in the laryngopharynx—'Killian's dehiscence'.
- Arises primarily as a result of failure of appropriate coordinated relaxation of the cricopharyngeus, causing ↑ pressure on the tissues directly above during swallowing.
- Typically occurs in the elderly.
- Associated with lower cranial nerve dysfunction (e.g. motor neuron disease, previous CVA).

Pathological features

- Acquired diverticulum (fibrous tissue and serosa without muscle fibres in most of the wall).
- Tends to lie to one side of the midline due to the cervical spine directly behind.

Clinical features

- Upper cervical dysphagia.
- Intermittent 'lump' appearing to the side of the neck on swallowing.
- Regurgitation of undigested food.
- Nocturnal aspiration—'waking up coughing'.

Diagnosis and investigations

- Diagnosis may be made on observed swallowing with a transient neck swelling appearing.
- Video barium swallow will show filling of pouch.

Gastroscopy should be avoided unless there is a question of associated pathology, since the pouch is easily missed and easily damaged or perforated by inadvertent intubation.

Treatment

Endoscopic stapled pharyngoplasty—side-to-side stapling of pouch to the upper oesophagus, which also divides the cricopharyngeus muscle.

Hiatus hernia

Key facts

The presence of part or all of the stomach within the thoracic cavity, usually by protrusion through the oesophageal hiatus in the diaphragm (see Fig. 8.2).

- Very common; ♀ > ♂; majority are asymptomatic.
- May or may not be associated with GORD.
- Predisposing factors include obesity and previous surgery.

Clinico-pathological features

Type 1—sliding hernia (95%)

- Displacement of upper stomach through the oesophageal hiatus, usually with stretching of the phrenico-oesophageal membrane.
- By far, the commonest form; may result in GORD.

Type 2—rolling (para-oesophageal) hernia

- Displacement of part or all of the fundus and body of the stomach through a defect in the phrenico-oesophageal membrane, such that it comes to lie alongside the normal oesophagus.
- Much less common.
- Symptoms include hiccough, 'pressure' in the chest, and odynophagia.
- May present acutely with gastric volvulus, incarceration, or obstruction—chest pain, unproductive vomiting, epigastric distension.

Type 3

- Combination of both type 1 and type 2 hernias (mixed).

Type 4

- Characterized by herniation of organs other than the stomach.

Diagnosis and investigations

- *UGI endoscopy (OGD)*. To exclude associated oesophageal mucosal pathology (oesophagitis, Barrett's oesophagus, neoplasia).
- *Video barium swallow*. Usually identifies type and extent.
- *CT thorax*. Investigation of choice in acute presentation.

Treatment

Medical (mainly for GORD symptoms)

- *Reduce acid production*. Stop smoking, lose weight, reduce alcohol consumption.
- *Counteract acid secretion*. PPIs, symptomatic relief with antacids, mucosal protectants.
- *Promote oesophageal and gastric emptying*. Promotilants, e.g. metoclopramide.

Surgical

Rarely required.

Indications

- Persistent symptoms despite maximal medical therapy.
- Established complications of rolling hernia such as volvulus or obstruction.

Elective procedure of choice is laparoscopic (or open) hiatal hernia repair, usually with plication of the oesophageal opening (crural plication) and usually with a fundoplication (e.g. Nissen's operation) if GORD symptoms predominate. Acute presentations may require a partial gastrectomy if stomach is non-viable.

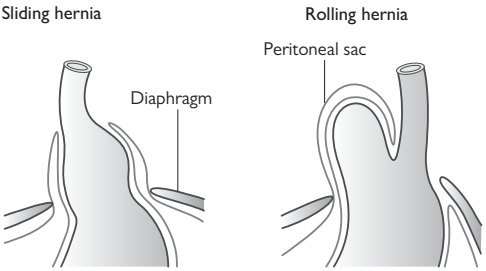

Fig. 8.2 Hiatus hernia—sliding and rolling.

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Gastro-oesophageal reflux disease

Key facts

- Pathologically excessive entry of gastric contents into the oesophagus, causing mucosal damage and/or symptoms that have a detrimental effect on quality of life.
- Physiological reflux occurs in up to 5% of the population.
- Commonest in middle-aged adults.
- Usually due to gastric acid, but may also be due to bile reflux.
- Contributory factors include:
  - *Reduced tone in the LOS*. Idiopathic, alcohol, hiatal hernia, drugs, previous surgery, 2° to existing peptic stricture.
  - ↑ *intragastric pressure*. Coughing, delayed gastric emptying, obesity.

Clinical features

- 'Heart burn', regurgitation, commonly worse at night, after large meals, and when recumbent.
- May present with cough, asthma, hoarseness, or aspiration pneumonia.
- Dysphagia may occur if there is associated ulceration or a peptic stricture.

Pathological features

Oesophagitis

- Results in inflammatory changes in the squamous-lined oesophagus.
- Varies in severity from minor mucosal erythema and erosions to extensive circumferential ulceration and stricturing (graded I to IV).

Strictures

- Chronic fibrosis and epithelial destruction may result in stricturing.
- Eventually shortening and narrowing of the lower oesophagus.
- May lead to fixation and susceptibility to further reflux.

Barrett's oesophagus (oesophageal metaplasia)

- Metaplastic columnar epithelium replaces normal stratified squamous epithelium of the distal oesophagus.
- Risk of conversion to oesophageal cancer (0.2–0.5% per annum).
- ♂ gender, old age, GORD, and obesity are established risk factors.
- Asymptomatic and normally identified incidentally during investigation of GORD.
- Current guidelines recommend 2- to 5-yearly endoscopic surveillance for Barrett's without evidence of dysplasia.
- Low-grade dysplasia—6-monthly endoscopic surveillance.
- High-grade dysplasia—endoscopic therapy (resection or radiofrequency ablation (RFA)).

Diagnosis and investigations

Diagnosis can be based on clinical symptoms alone

- Symptoms are relatively common and can be treated empirically.
- Investigation is only required if symptoms fail to respond to treatment or if there are any significant associated worrying symptoms, e.g. dysphagia, weight loss.

Investigations

Upper GI endoscopy

- GORD symptoms and any significant associated alarm symptoms, e.g. dysphagia, weight loss.
- Persistent symptoms despite 4–8 weeks' maximal medical therapy.

Ambulatory pH monitoring/oesophageal manometry

- Persistent GORD symptoms without endoscopic evidence of mucosal damage.
- Exclude other oesophageal pathology, e.g. motility disorders.
- Always in patients being considered for surgery.

Treatment

Medical

- *Lifestyle modification*. Smoking, weight, alcohol consumption; avoid dietary triggers (e.g. caffeine, spicy food).
- *Counteract acid secretion*. PPI (e.g. omeprazole), symptomatic relief with antacids (e.g. Gaviscon®).
- *Improve gastric and oesophageal emptying*. Promotilants (e.g. metoclopramide).

Surgical

Rarely required. Indicated for:

- Persistent symptoms despite maximal medical therapy.
- Young patients who may require lifelong therapy.
- High-volume reflux with complications, including stricture, severe ulceration, or respiratory complications.

Procedure of choice is laparoscopic fundoplication—'*Nissen's operation*' (wrapping the fundus of the stomach around the intra-abdominal oesophagus to augment the high-pressure zone).

Uncertain role in the prevention of progressive dysplasia in Barrett's oesophageal metaplasia in the absence of symptoms.

Oesophageal tumours

Key facts and pathological features

Typically present late with 50–60% of patients presenting with incurable locally advanced or metastatic disease.

Types of oesophageal tumours

Adenocarcinoma

- Rapidly increasing incidence in Western world; ♂ > ♀, 5:1.
- Commonest in Western Europe.
- Risk factors: GORD and Barrett's metaplasia, smoking, obesity.
- Commonly occurs in the lower third of the oesophagus and GOJ.

Squamous carcinoma

- Incidence falling in Western world. Commonest in northern Iran, northern China, and central Asia; ♂ > ♀, 3:1.
- Risk factors: smoking, alcohol, diet poor in fresh fruit and vegetables, chronic achalasia, chronic caustic strictures.
- Commonly affects mid- to upper oesophagus.

Lipoma and GISTs (➔ Small bowel tumours, p. 396) Rare.

Rhabdomyo(sarco)ma Malignant tumour of skeletal muscle wall of the oesophagus; very rare.

Clinical features

- *Dysphagia*. Progressive symptoms, initially to liquids, then to solids. May present with food bolus.
- *Haematemesis*. Rarely the presenting symptom.
- *Incidental/screening*. Occasionally identified as a result of follow-up/surveillance for Barrett's metaplasia, achalasia, or reflux disease. Presence of high-grade dysplasia in Barrett's is associated with the presence of an occult adenocarcinoma in 30%.
- *Features of disseminated disease*. Cervical lymphadenopathy, hepatomegaly due to metastases, epigastric mass due to para-aortic lymphadenopathy.
- *Symptoms of local invasion*. Dysphonia in RLN palsy, cough and haemoptysis in tracheal invasion, neck swelling in SVC obstruction, Horner's syndrome (sympathetic chain invasion).
- *Anaemia and weight loss*.

Diagnosis and investigations

- *OGD 9 biopsy*. First-line investigation. Incidental diagnosis during Barrett's surveillance.
- *CT thorax and abdomen*. Occasionally diagnosed incidentally during investigation for weight loss.
- *Barium swallow*. Only indicated after failed OGD or suspected post-cricoid carcinoma (often missed during intubation).

Staging investigations

Prognosis is strongly associated with stage of disease (TNM staging) (see Table 8.1).

Table 8.1 TNM staging of oesphageal cancers (8th edition, in use from January 2018)*

T (tumour)	N (nodes)	M (metastases)
Tis, high-grade dysplasia	N0, no lymph nodes	M0, no distant metastases
T1, tumour invades lamina propria, muscularis mucosae, or submucosa	N1, involves 1–2 regional lymph nodes	M1, distant metastases
T2, tumour invades muscularis propria	N2, involves 3–6 regional lymph nodes	
T3, tumour invades adventitia	N3, involves 7+ regional lymph nodes	
T4, tumour invades adjacent structures		

* In the 8th edition, tumours with the epicentre within 2cm of the GOJ extend into the oesophagus and tumours with the epicentre within the proximal 2cm of the cardia are to be staged as oesophageal.

Radiological staging investigations

Local staging

- *EUS*. Assesses depth of tumour invasion and local lymph node involvement.

Regional staging

- *CT*. Evaluate region of the 1° tumour and assess for distant metastases (liver, lung, bone, peritoneum).
- *CT-PET*. Used to exclude occult disseminated disease in patients considered for potentially curative treatment. Also useful in the re-staging of disease after neoadjuvant treatment.
- *Staging laparoscopy*. Used to assess for occult intra-abdominal metastasis in GOJ tumours not visible on CT (liver and peritoneum).

Physiological staging investigations

- Cardiopulmonary exercise (CPEX) testing.
- Vital to assess cardio-respiratory fitness prior to consideration of radical surgery (anaerobic threshold and peak VO_2—key variables).

Treatment

An MDT approach is crucial in determining treatment strategy.

Potentially curative

- *Squamous cell carcinoma*. Radical external beam chemoradiotherapy or neoadjuvant chemotherapy or neoadjuvant chemoradiotherapy followed by surgery (radical resection).
- *Adenocarcinoma (large)*. Neoadjuvant chemotherapy followed by surgery (radical resection). Definitive chemoradiotherapy in patients deemed unfit for surgery or patient choice.
- *Adenocarcinoma (Tis/T1) or high-grade dysplasia in Barrett's*. EMR, RFA, or occasionally surgical resection.

Palliative

- Dysphagia can be treated by endoscopically inserted endoluminal self-expanding metal stenting (SEMS), external beam radiotherapy; surgery is no longer indicated for palliation.
- Metastases. Systemic chemotherapy if symptomatic.
- Other palliative measures: analgesia, antiemetics, ascitic fluid drainage.

Peptic ulcer disease

Key facts

- A breach in the gastric or duodenal mucosa that extends through the muscularis mucosa into the deeper layers of the wall.
- May be acute and transient (e.g. stress ulceration after surgery or in acutely unwell ITU patients).
- If the repair system fails to deal with the breakdown of the mucosa or the causative mechanisms are not treated, it may become chronic.
- The term 'peptic' refers to ulcers in the columnar mucosa in the lower oesophagus, stomach, duodenum, or small bowel, usually due to the corrosive action of gastric acid.

Classification

Multiple classification systems:

- Region: gastric, duodenal, oesophageal.
- Modified Johnson: type 1 (body), type 2 (duodenal and gastric), type 3 (pyloric channel), type 4 (proximal gastric), type 5 (throughout stomach, chronic NSAID use).
- Forrest classification (for bleeding ulcers identified during gastroscopy).

Gastric ulceration

- ♂ > ♀, 3:1; peak age of incidence 50y.
- Associated with *Helicobacter pylori* in 45% of cases and with NSAID use, high alcohol intake, and smoking.

Duodenal and type II gastric ulceration

- ♂ > ♀, 5:1; peak age of incidence 25–30y.
- Associated with *H. pylori* in 85% of cases and with high acid secretion, smoking, and NSAID use.

Atypical ulceration

- Usually due to either atypical sites of gastric acid secretion (e.g. ectopic gastric mucosa in a Meckel's diverticulum) or abnormally high levels of acid secretion (e.g. Zollinger–Ellison syndrome; ➔ Peptic ulcer disease, p. 384).
- Associated with ulceration that fails to respond to maximal medical therapy, multiple ulcers, and ulcers in abnormal locations (e.g. distal duodenum or small bowel).

Clinical features

- *Dyspepsia*. Often related to food.
- Duodenal ulceration typified by hunger pains, with central back pain *relieved by food*; pain is often cyclical and occurs in the early hours of the morning.
- Gastric ulceration typified by pain *precipitated by food* with associated weight loss and anorexia; pain less cyclical.
- Vomiting and upper abdominal distension suggest gastric outlet obstruction (due to pyloric scarring from ulceration).

Management and investigations

Initial investigation of upper abdominal symptoms can be challenging (see Fig. 8.3).

- H. *pylori* testing and eradication initially.
- *Gastroscopy*. Commonest diagnostic test. Biopsy to assess for associated malignancy.
- *Barium meal*. May be used if gastroscopy contraindicated.
- *Fasting serum gastrin levels*. If hypergastrinaemia suspected.

Complications

- Acute UGI bleeding (➲ Upper gastrointestinal bleeding, p. 398).
- Iron deficiency anaemia due to chronic low-level bleeding.
- Perforation (➲ Acute upper GI perforation, p. 402).
- Gastric outlet obstruction due to chronic scarring at pylorus.

Treatment

Medical

- H. *pylori* eradication (usually triple therapy of metronidazole, PPI, and clarithromycin).
- Advise to avoid NSAIDs/aspirin, reduce alcohol intake, and smoking cessation.
- PPIs (e.g. omeprazole 20–40mg PO or H2 blockers (e.g. ranitidine 150mg PO bd) if intolerant of PPI.
- Topical antacids (e.g. Gaviscon®, sucralfate, colloidal bismuth), especially for acute ulceration post-operatively or in ITU patients.

Surgical

Rarely necessary with highly effective acid-reducing drugs and *H. pylori* eradication therapy. Indications include:

- Gastric outlet obstruction not responsive or suitable for endoscopic dilatation. Usual procedure is pyloroplasty or partial gastrectomy (Polya).
- Failure to respond to maximal medical treatment with severe symptoms or due to habitual recidivism.
- Emergency indications include:
  - Perforation.
  - Bleeding.

Zollinger–Ellison syndrome

- Due to hypergastrinaemia causing extensive, persistent, or typical ulceration.
- Commonest cause is benign secretory gastrinoma (usually intrapancreatic); occasionally the cause is malignant gastrinoma (associated with MEN syndromes).
- Diagnosed by raised serum gastrin level, tumour located by CT scanning, angiography, selective pancreatic venous cannulation at surgery.
- Treatment: resection of pancreatic tissue containing tumour.

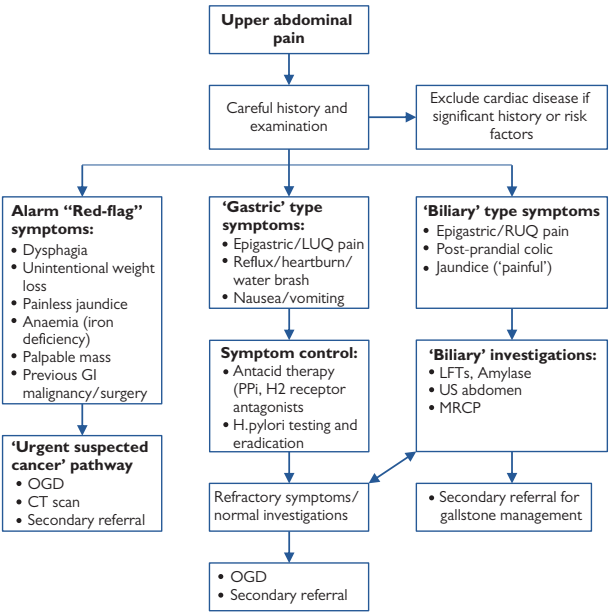

Fig. 8.3 Symptom-based algorithm for upper abdominal pain investigation.

Gastric tumours

May arise from the tissues of the mucosa (adenocarcinoma), connective tissue of the stomach wall (GISTs; ➲ Gastrointestinal stromal tumours, p. 396), the neuroendocrine tissue (carcinoid tumours; ➲ Small bowel tumours, p. 396), or the lymphoid tissue (lymphomas).

Typically present late, with ~50% having disease that extends beyond the locoregional confines.

Key facts

Adenocarcinoma; commonest age of incidence >50y; ♂ > ♀, 3:1. Predisposing factors include:

- Chronic *H. pylori* infection.
- Diet rich in nitrosamines (cured meat, pickled fruit).
- Chronic atrophic gastritis.
- Blood group A.
- Obesity.
- Smoking.

Clinical features

Symptoms

- Dyspepsia (any new onset of dyspepsia over the age of 55 should be considered to be due to adenocarcinoma until proven otherwise).
- Weight loss, anorexia, and lethargy.
- Anaemia (iron deficiency due to chronic blood loss).
- Early satiety.
- Occasionally presents as acute UGI bleeding (➲ Upper gastrointestinal bleeding, p. 398).
- Dysphagia uncommon unless involving the proximal fundus and GOJ.

Signs

- Weight loss.
- Palpable epigastric mass.
- Palpable supraclavicular lymph node (Virchow's signal node, Troisier's sign) suggests disseminated disease.

Diagnosis and investigations

- *OGD ± biopsy*. First-line investigation.
- *CT thorax, abdomen, and pelvis*. Occasionally diagnosed incidentally during investigation for weight loss.
- *Barium meal*. Only indicated after failed OGD.
- *Screening*. Japan/East Asia. Not currently performed in the UK/Europe.

Staging investigations

Prognosis is strongly associated with stage of disease (TNM staging; see Table 8.2).

Local staging

- *EUS*. Assesses depth of tumour invasion and local lymph node involvement in proximal gastric tumours.

Regional staging

- *CT*. Evaluate region of the 1° tumour and assess for distant metastases (liver, lung, bone, peritoneum).
- *Staging laparoscopy*. To assess for occult peritoneal metastasis not visible on CT.
- *CT-PET*. Used in staging proximal gastric tumours encroaching on the GOJ to exclude occult disseminated disease in patients otherwise considered for potentially curative treatment. Also useful in re-staging of disease after neoadjuvant treatment.

Table 8.2 TNM staging of gastric cancers (8th edition, in use from January 2018)*

T(tumour)	N (nodes)	M (metastases)
Tis, *in situ* within mucosa	N0, no lymph nodes	M0, no distant metastases
T1, tumour invades lamina propria, muscularis mucosae, or submucosa	N1, involved 1–2 regional lymph nodes	M1, distant metastases
T2, tumour invades muscularis mucosa	N2, involves 3–6 regional lymph nodes	
T3, tumour penetrates subserosal connective tissue without invasion of visceral peritoneum or adjacent structures	N3, involves 7+ regional lymph nodes	
T4, tumour invades serosa (visceral peritoneum) or adjacent structures		

* In the 8th edition, tumours with the epicentre within the proximal 2cm of the cardia are to be staged as oesophageal and those tumours with the epicentre >2cm distal to the GOJ will be staged as gastric.

Treatment

An MDT approach is crucial in determining the treatment strategy. When a tumour is considered potentially curable, the treatment offered is stage-directed.

Early gastric cancer (Tis/T1, N0/1, M0)

- Suitable for attempted curative resection if patient medically fit enough.
- EMR/ESR (endoscopic mucosal/submucosal resection) in tumours <2cm in diameter.
- Neoadjuvant/adjuvant chemotherapy when lymph node metastases suspected.

Advanced gastric cancer (T2 or more or any of N2/3, M1)

- Patients will be offered neo/adjuvant chemotherapy.
- Complete surgical eradication of the gastric tumour with resection of adjacent lymph nodes represents the best chance for long-term survival.
- Surgery includes: total/subtotal/partial gastrectomy.
- May be undertaken for palliative treatment.
- Local ablation for symptom control occasionally possible.
- Palliative chemotherapy occasionally effective for disseminated disease.

Key revision points—anatomy and physiology of the stomach

- The fundus is predominantly a storage zone with few active cells.
- The body contains mostly chief cells (secrete pepsinogen; stimulated by gastrin and local ACh release) and oxyntic cells (secrete H+; stimulated by gastrin, histamine, and ACh; inhibited by H+, secretin, and gastric inhibitory polypeptide (GIP)).
- The antrum contains G cells (secrete gastrin; stimulated by ACh from vagus and stretch; inhibited by vasoactive intestinal polypeptide (VIP), secretin, and H+).
- The pyloric sphincter is a functional sphincter of circular muscle.
- Arterial supply is profuse (gastric ischaemia is rare) via the coeliac axis—left gastric, splenic, and common hepatic arteries.
- Lymphatic drainage follows arteries and is profuse (significant lymph node metastases are usually fatal).

Chronic enteric ischaemia

Caused by chronic reduction in intestinal blood flow without acute threat to bowel viability.

Key facts

Chronic intestinal ischaemia is uncommon. Usually presents with vague symptoms and diagnosis is often prolonged.

Causes include:

- Progressive multi-vessel mesenteric atherosclerosis, e.g. superior mesenteric artery (SMA) occlusion and coeliac artery stenosis.
- Obliterative small vessel disease/vasculitis (e.g. thromboangiitis obliterans, systemic sclerosis, severe diabetic vasculopathy).

Clinical features

Symptoms

- *Mesenteric angina*. Postprandial, chronic central abdominal pain, usually within an hour of eating.
- May present with weight loss, general anorexia, and malnutrition.
- Typically associated with other features of extensive vascular disease such as coronary disease, claudication, and renal impairment.

Signs

- Weight loss, central abdominal tenderness, abdominal bruit.

Diagnosis and investigations

- High index of clinical suspicion.
- Patients often first investigated for other aetiologies as an explanation of weight loss, e.g. malignancy.
- Imaging modalities of visceral vessels include:
  - CT angiography (CTA).
  - MR angiography (MRA).
  - Transfemoral digital subtraction angiography/aortography.
- Other investigations should include:
  - Assessment of renal function.
  - Assessment of coronary circulation.
  - Exclusion of aneurysmal disease.
- Small vessel disease may require autoimmune screen.

Complications

- Acute intestinal ischaemia is less common due to the development of collateral vessels, although, if undiagnosed, loss of all visceral vessels may result in eventual pan-intestinal infarction.
- Chronic ischaemic intestinal strictures due to focally severe ischaemia.

Treatment

Medical

- Smoking cessation.
- 2° prevention measures to limit atherosclerosis (HTN, hyperlipidaemia, diabetes).
- Aspirin 75mg od to prevent thromboembolic events.

- Treatment of autoimmune disease if present.
- Nutritional assessment and support.
- Anticoagulation only for acute-on-chronic thromboembolism.

Radiological/surgical

Revascularization indicated for significant symptoms plus documented evidence of mesenteric vessel stenosis.

- Percutaneous transluminal angioplasty ± stent.
- Surgical revascularization: endarterectomy, aortomesenteric/coeliac bypass.

Overall prognosis: vessel restenosis (7–34%); mortality (4–6%).

Key revision points—anatomy and physiology of the small intestine

- The duodenum is a secretory and digestive organ; described in four parts (second part admits the common bile and pancreatic ducts via the ampulla of Vater on the medial wall).
- The jejunum is a secretory and digestive organ. Typical features include: thick, red-purple wall; prominent plicae circulares; and single arterial arcades with long mesenteric vessels.
- The ileum is predominantly an absorptive organ. Typical features include: thin, blue-purple wall; prominent lymphoid aggregates; and multilayered mesenteric arterial arcades.
- The terminal ileum is a specialized area of the ileum, particularly concerned with absorption of bile salts and vitamin B12/intrinsic factor (IF) complex.

Surgery for morbid obesity

Key facts

- Obesity is defined as a BMI of >30kg/m².
- In 2015, in the UK, 58% of women and 68% of men were overweight or obese, with an obesity prevalence increasing from 15% in 1993 to 27% in 2015.
- Admissions to hospital due to obesity-related disease have ↑ 8-fold in the last 10y.
- Obese patients are exposed to an ↑ risk of chronic diseases, particularly type 2 diabetes, HTN, hyperlipidaemia, and arthritis.
- ↑ risk of some forms of cancer (breast, colon, endometrial).

Clinical features

Symptoms

- The symptoms of obesity are generally related to the underlying condition that develops in association with it.
- Commonly, patients find their physical capacity is reduced and they become short of breath more easily on exertion.

Signs

- Examination of obese patients can be challenging because their size can mask the ability to elicit clinical signs.
- Assess for signs of respiratory disease and dyspnoea at rest.
- Investigation of possible biliary disease (CBD stones, biliary strictures, biliary tumours, biliary injuries, intrahepatic biliary disease).
- Investigation of pancreatic disease (pancreatic duct strictures, pancreatic duct abnormalities).
- Therapeutic interventions for pancreaticobiliary disease:
  - Stenting for CBD stones, strictures, and tumours.
  - Sphincterotomy for the extraction of biliary stones.

Diagnosis and investigations

- Exclude underlying endocrine disorders—Cushing's disease (➲ Cushing's syndrome, p. 354), hypothyroidism, and polycystic ovarian syndrome.

Treatment

Medical

- Diabetic control. Oral hypoglycaemics and insulin, if necessary.
- Medication for antihypertension and cardiovascular disease.
- Psychological and dietetic support for weight loss programme.
- Anti-obesity medication. Orlistat (reduces fat absorption) is the commonest.

Surgical

- Should only be considered after non-surgical treatments have failed and the patient has been through a thorough preoperative assessment by an MDT, including a dietician, a medical bariatric specialist, a psychologist/psychiatrist, a nurse specialist, and a surgeon, to ensure their ability to adhere to the necessary post-operative dietary and lifestyle changes.
- Indications include BMI ≤40kg/m² without comorbid illness or BMI 35–39.9kg/m² and at least one serious comorbidity, including, but not limited to:
  - Type 2 diabetes.
  - Obstructive sleep apnoea.
  - HTN.
  - Hyperlipidaemia.
  - Obesity hypoventilation syndrome.
  - Non-alcoholic liver disease.
- Surgical procedures have either a volume-restrictive or a nutrient-malabsorptive effect.
- Affect satiety, absorption, and insulin sensitivity via hormonal and enteric pathways.
- Procedures are typically performed laparoscopically due to associated improved recovery and lower morbidity, compared with open surgery.
- Laparoscopic adjustable gastric banding (LAGB):
  - The commonest restrictive operation (the band is placed around the cardia of the stomach and restricts the volume of food, but not liquid, that can be ingested at one time). This has now lost favour among specialist bariatric surgeons in view of the high medium-term complication rate (15%).
- Roux-en-Y gastric bypass (RYGB):
  - Involves a restrictive element (division of the stomach to create a small gastric pouch) and a malabsorptive element (division and re-anastomosis of the small bowel to reduce its ability to absorb food).
- Sleeve gastrectomy (SG):
  - Involves a restrictive element (reduced stomach volume), reduces gastric motility, and has fewer ghrelin-producing cells (a gut hormone involved in regulating food intake).

Small bowel tumours

Key facts

Diagnosis is difficult due to their rarity and variable presenting symptoms. Tumours may arise from:

- *Mucosa*—adenocarcinoma (<5% of all GI malignancies).
- *Neuroendocrine tissue*, e.g. carcinoid tumours.
- *Lymphoid tissue* (lymphoma).
- *Connective tissue* of the bowel wall, e.g. GIST, lipoma.

Adenocarcinoma of small bowel

- 25–40% of all small bowel tumours.
- Commonest in the duodeno-jejunal junction and proximal jejunum; least common in mid- and distal ileum.
- Associations:
  - Lynch Syndrome (➲ Pathological features, p. 500).
  - Familial adenomatous polyposis (FAP) (➲ Colorectal cancer, p. 500).
  - Peutz–Jegher's syndrome (➲ Colorectal polyps, p. 498).
  - Crohn's disease (➲ Crohn's disease, p. 494).
  - Untreated long-standing coeliac disease.

Neuroendocrine tumours (NETs)

- 25% of all small bowel tumours.
- Commonest in the distal small bowel; may arise in Meckel's diverticulum or appendix.
- Commonest are carcinoid tumours; other rarer tumours include gastrinoma and duodenal somatostatinoma.
- Majority benign (non-metastatic).
- May produce enteric hormones (e.g. 5-HT, kallikrein, substance P); hormonal effects only occur when the 1° is able to secrete hormones into the systemic circulation or when hepatic metastases secrete into the vena caval circulation (carcinoid syndrome).
- Associated with syndromes such as Zollinger–Ellison syndrome (gastrinoma).

Primary lymphoma

- 20% of small bowel tumours.
- Arise from the lymphoid tissue of the small bowel wall.
- Almost always non-Hodgkin's; commonest are B-cell lymphomas arising from MALTomas.

Gastrointestinal stromal tumours

- 10% of small bowel tumours.
- Arise from the mesenchymal tissues of the bowel wall and mesentery (smooth muscle cells, fibroblasts, lipocytes).
- Previously called variously leiomyo(sarc)oma and lipo(sarco)ma.
- Tumours of myenteric plexus tissues are a variant called gastrointestinal autonomic nerve tumours (GANTs).

Clinical features

- *Adenocarcinoma of small bowel*. Often presents late with metastases; may present with small bowel obstruction, recurrent abdominal pain, or recurrent/occult GI bleeding.
- *Carcinoid tumours*. Commonest presentation is incidental finding after appendicectomy or Meckel's diverticulectomy.
- *Carcinoid syndrome*. Rare (typified by flushing, tachycardia, colicky abdominal pain, diarrhoea, and wheezing).
- *GIST*. Often slow-growing abdominal mass, vague abdominal pain; may present with occult GI bleeding due to tumour ulceration.
- 1° *lymphoma*. Presents with malaise, abdominal pain, and diarrhoea; may present with acute perforation or obstruction.

Diagnosis and investigations

Radiological

- *CT/CT-PET*. Identifies 1° tumour; assesses extent of involvement of other tissues and/or metastatic disease.
- *CT/MR enterography*. Better visualization of bowel lumen.
- *CTA*. For assessment of particularly vascular tumours.

Endoscopic

- *Gastroscopy*. For tumours located in duodenum.
- *Capsule endoscopy*. Non-invasive, visualizes entire small bowel.
- *Enteroscopy*. Passage of scope beyond ligament of Treitz, able to biopsy/perform therapeutic interventions.

Complications

- *Bleeding*. Common with adenocarcinoma and some GISTs.
- *Obstruction*. Especially adenocarcinoma, GIST/GANTs, lymphoma.
- *Perforation*. Most commonly lymphoma, especially shortly after starting chemotherapy (due to bowel wall replacement by tumour); also occurs with adenocarcinoma.
- *Malabsorption*. Often with lymphoma if widespread.

Treatment

Surgical

- Wide segmental resection with macroscopic clearance of tumour for adenocarcinoma, carcinoid, GISTs, and GANTs.
  Potentially curative for non-metastatic disease.
  Palliative to prevent complications if disease is metastatic.
- Pancreaticoduodenectomy for duodenal (D1/D2) tumours.
- Surgical resection may be indicated for 1° lymphoma prior to chemotherapy if there is a high risk of perforation of 1° tumour.

Medical

- *Chemotherapy*. Unresectable/metastatic tumours, adjuvant therapy for node-positive completely resected tumours, lymphomas.
- *Hepatic ablation/embolization*. Used to treat carcinoid metastases to treat symptoms of carcinoid syndrome.
- *Imatinib (anti-CD113)*. Treatment for GISTs (c-Kit positive).

Upper gastrointestinal bleeding

Key facts

Current guidelines suggest a lead clinician be responsible for all GI bleeding pathways (upper and lower), including clinical governance.

It is recommended that patients with UGI bleeds should only be admitted to centres with 24/7 access to endoscopy, interventional radiology, GI surgery, critical care, and anaesthesia.

- *Haematemesis*. Vomiting of fresh blood, usually due to bleeding proximal to the duodeno-jejunal junction.
- *Melaena*. Malodorous, black, tarry stools (altered blood), usually due to bleeding distal to the GOJ.
- *Haematochezia*. Red/maroon blood in stool, typically lower GI bleed origin but may occur in massive UGI bleeding.
- *Coffee ground vomiting*. Often used to describe vomiting of 'old' blood.
- Annual incidence 1 in 1000 adults in the UK.
- Intervention required in 20% because of ongoing bleeding or rebleeding.

Causes and features

- *Peptic ulceration* (50% of UGI bleeds). Fresh red blood with clots, occasionally mixed with food.
- *Oesophageal varices*. Copious dark red venous blood with little mixing with food; features of portal HTN (e.g. caput medusa).
- *Oesophageal ulceration*. Small volumes of bright red blood/streaks typical.
- *Oesophageal trauma* ('Mallory–Weiss tear'). Small volumes of fresh bright blood preceded by violent vomiting or retching.
- *Vascular malformations/lesions* (e.g. 'Dieulafoy').
- *Gastric tumours* (adenocarcinomas, GISTs) (➲ Gastric tumours, p. 388).
- Aortoenteric fistula. Copious bright red blood (often rapidly fatal), usually previous history of aortic graft surgery.

Associated or predisposing conditions

- Agents affecting mucosal health—NSAIDs, steroids, alcohol, major trauma, or massive burns (stress response).
- Agents worsening risk of bleeding—anticoagulants.

Emergency management

Resuscitation

- IV access with large-calibre IV cannula; give crystalloid fluid up to 1000mL if tachycardic or hypotensive. Only use group O negative blood if the patient is *in extremis*; otherwise wait for cross-matched blood if transfusion required.
- Urinary catheterization and fluid balance chart if hypotensive.
- Blood tests for FBC, U&Es, LFTs, INR, cross-match (at least 4U if large haematemesis), and clotting profile.
- Early discussion with haematology.
- Always consider alerting HDU/ITU if very unwell.

- Insertion of a Sengstaken–Blakemore gastro-oesophageal tube may be a lifesaving resuscitation manoeuvre (variceal compression).
- Thorough history from patient or next of kin, particularly with regard to NSAID/steroid use, possible anticoagulation, and alcohol intake.
- All major bleeds should be discussed with the on-call consultant responsible for major GI bleeds within an hour of diagnosis.

Haemodynamically unstable

- Resuscitate as above, prepare for emergency endoscopy, early consultation with surgery, and/or interventional radiology in the event of unsuccessful endoscopy.
- Emergency OGD (in theatre) as soon as patient stable.
- If no UGI source identified, proceed with urgent colonoscopy.
- Surgery may be required if massive haemorrhage requiring ongoing resuscitation, failed initial endoscopic therapy, and rebleeding not suitable for repeat endoscopic therapy.

Haemodynamically stable

- Resuscitate as necessary.
- OGD within 24h.
- If no UGI source identified, proceed with colonoscopy.
- If source remains unidentified, consider CTA.

Risk stratification

Blatchford score

- Calculates risk of requirement for endoscopic intervention.
- Does not require endoscopic finding and can be calculated at presentation.
- Identifies patients who may be safely managed as outpatients (see Table 8.3).

Rockall score

- Based on patient age, presence of shock, comorbidity, diagnosis, and endoscopic stigmata of recent haemorrhage.
- Mortality risk calculation (see Table 8.4).

Treatment

- *Medication*. IV PPI (e.g. omeprazole 40mg IV); stop all NSAIDs, correct coagulopathy, *H. pylori* eradication, IV vasopressin or analogues for varices.
- *OGD*. Combination therapies include: thermal coagulation, haemostatic clips, adrenaline injection, and banding for varices.
- *Angiography*. Localization, embolization, or vasoconstriction.
- *Surgery*. Cause-dependent: local ulcer excision, partial gastrectomy (ulcers or tumours), or under-running of peptic ulcer.

Table 8.3 Blatchford score for predicting need for intervention of acute upper GI bleeding*

Admission risk marker		Score component value	
Blood urea			
6.5–8.0		2	
8.0–10.0		3	
10.0–25.0		4	
≤25.0		6	
Haemoglobin (g/L) for men		**Haemoglobin (g/L) for women**	
12.0–13.0	1	10.0–12.0	1
10.0–12.0	3	<10.0	6
<10.0	6		
Systolic blood pressure (mmHg)			
100–109		1	
90–99		2	
<90		3	
Other markers			
Pulse ≤100/min		1	
Presentation with melaena		1	
Presentation with syncope		2	
Hepatic disease		2	
Cardiac disease		2	

* A score of 6 or more is associated with a >50% risk of needing an intervention

Table 8.4 Rockall score for predicting mortality of acute upper GI bleeding*

	Rockall score			
	0	1	2	3
Age (y)	<60	60–79	>80	
Shock	None	HR >100bpm	Systolic BP <100mmHg	
Major comorbidities	None		Cardiac	Hepatorenal disease Carcinoma
Diagnosis	Mallory–Weiss tear	Other non-malignant GI diagnoses	UGI malignancy	
Endoscopic stigmata of haemorrhage	None		Adherent clot/spurting vessel	

* Percentage mortality for scores: 0, <1%; 1, 3%; 2, 6%; 3, 11%; 4, 25%; 5, 40%; 6, >80%.

Acute upper GI perforation

Causes and features

- *Peptic ulceration*. Gastric or duodenal.
- *Malignancy*. Gastric cancer.
- *Barotrauma*. Oesophageal perforation (Boerhaave's syndrome).
- *Iatrogenic*. Endoscopic perforation (gastroscopy/ERCP).
- *Ingestion/foreign body*, e.g. caustic injury, fish bone perforation.
- *Ischaemic*. 2° to gastric volvulus.

Symptoms

- Acute-onset upper abdominal pain. Severe, constant, worse with breathing and moving; may radiate to back or shoulders.
- Prodrome of upper abdominal pain (in benign or malignant ulceration).
- Severe retching followed by retrosternal chest pain may suggest Boerhaave's syndrome.
- Prodrome of weight loss, dyspepsia, and anorexia suggests carcinoma.

Signs

- Generalized peritonism common ('board-like' generalized rigidity with marked guarding and tenderness).
- Localized upper abdominal peritonism may occur, especially with previous surgery where adhesions may act to contain the contamination.
- Mild fever, pallor, tachycardia, and hypotension (often profound due to autonomic reaction); typically respond quickly to modest fluid resuscitation.

Emergency management

Resuscitation

- IV access, large-calibre cannula; give crystalloid fluid up to 1000mL if tachycardic or hypotensive.
- Urinary catheterization and fluid balance chart.
- Blood tests for FBC, U&Es, LFTs, group and save, clotting, ABG, including serum lactate.
- Antibiotic therapy.

Establish a diagnosis

- *Erect CXR* (looking for free gas).
- *CT*. Providing haemodynamically stability, most patients will undergo CT imaging which may provide additional information, e.g. metastatic gastric carcinoma, or help tailor treatment, e.g. laparoscopic approach.

Treatment

- *Surgery*. See definitive management.
- *Conservative management*. IV PPI, NBM; has a limited role in management where there is evidence that the perforation has sealed at the time of presentation, there is no haemodynamic instability, and there are no signs of peritonism, or when patient is deemed unfit to survive surgery.
- *Endoscopic therapy*. Limited role for endoscopic stents to cover oesophageal perforations whilst healing occurs. Typically for small iatrogenic perforations with minimal or no contamination.

Definitive management

Boerhaave's syndrome

- Depends on anatomical location. Operative closure around a T-tube drain to fashion a controlled fistula considered the safest option requiring thoracotomy and/or laparotomy.

Gastric/duodenal ulcer

- Sutured closure with omental patch (open or laparoscopic).
- Empirical oral triple therapy for *H. pylori*.

Gastric carcinoma

- Partial gastrectomy (usually palliative).

Traumatic

- Sutured closure.

Volvulus with ischaemia

- Subtotal gastrectomy.

Chapter 9

Liver, pancreatic, and biliary surgery

Jaundice

Key facts

High serum bilirubin levels. Clinically manifests at levels >35μmol/L. Derived from the French word '*jaune*' meaning yellow.

Bilirubin metabolism

- Production of 250–300mg/day of unconjugated bilirubin by breakdown of Hb by the reticuloendothelial system largely in the spleen.
- Unconjugated bilirubin is insoluble and transported attached to albumin in the bloodstream.
- Taken up into the liver by active transport and in hepatocytes is conjugated with glucuronide. Conjugated bilirubin is water-soluble and can be excreted in urine (bilirubinaemia), accounting for its dark colour.
- Excreted with bile into the duodenum bound to cholesterol, lipids, and bile salts as micelles.
- Bile contains bile salts (1°: cholate and chenodeoxycholate; 2°: deoxycholate and lithocholate), bicarbonate, cholesterol, steroid, and water.
- In the terminal ileum, conjugated bilirubin is converted into urobilinogen by bacterial proteases.
- 90% of urobilinogen is oxidized to stercobilinogen and excreted in faeces; 10% is reabsorbed via the portal system and ultimately excreted in the urine.

Classification

Pre-hepatic jaundice (haemolytic, increased breakdown of red blood cells)

Unconjugated hyperbilirubinaemia, normal-colour stool and urine, no bilirubin in urine.

- Haemolytic anaemia.
- Congenital abnormalities of red cell structure (spherocytosis, sickle cell disease).
- Transfusion reactions.
- Drug reactions.

Hepatic jaundice (hepatocellular)

Excess of both unconjugated and conjugated bilirubin due to hepatocyte failure to conjugate, enzyme deficiency, or ↓ uptake. Mixed jaundice with dark urine and normal stool. ALT > ALP. PT ↑ and not correctable with vitamin K.

- ↓ conjugation.
  - Cirrhosis (➲ Liver cirrhosis, p. 426).
  - Infection. Viral (hepatitis A/B/C, EBV, CMV); bacterial (liver abscess); parasitic (amoebic).
  - Prematurity.
  - Crigler–Najjar syndrome—congenital inability to conjugate bilirubin due to enzyme inhibition.
  - Gilbert's syndrome—inhibition of conjugation and defective uptake by hepatocytes. Exacerbated by concurrent illness.

- ↓ hepatocyte uptake.
  - Sepsis.
  - Drugs (paracetamol excess, amiodarone, diclofenac, fluconazole).
  - Non-infective hepatitis (alcohol-related).
- Liver tumours (malignant and benign).

Post-hepatic jaundice (obstructive)

Biliary tree obstruction (intraluminal, mural, or extrinsic). Normally conjugated hyperbilirubinaemia, dark urine with bilirubin present, pale stools due to reduced stercobilinogen. ALP > ALT. PT prolonged, but correctable with vitamin K.

- Intraluminal biliary tree abnormalities.
  - Gallstones.
  - Parasites (flukes).
- Mural biliary tree abnormalities
  - Biliary stricture (intra-/extra-hepatic, primary sclerosing cholangitis, chronic cholangitis, primary biliary cirrhosis, iatrogenic, traumatic).
  - Tumours (cholangiocarcinoma, periampullary, pancreatic cancer).
  - Congenital atresia.
- Extrinsic compression of biliary tree.
  - Pancreatitis.
  - Tumours (head of pancreas).
  - Lymphadenopathy.

Painful versus painless jaundice

Differentiating between surgical and medical causes of jaundice can be challenging. A useful diagnostic stratification tool can be used to ascertain if the jaundice is painful or painless (see Fig. 9.1).

History, examination, and clinical presentation

- History should include key features to elicit a list of likely differential diagnosis. Important questions include:
  - Family history of blood disorders, recent foreign travel, medications, gallstones, alcohol, weight loss, recent infections, and urine and stool colour and consistency.
  - Likewise, clinical examination and presentation are dependent on the cause but usually includes yellowing of the sclera and skin, dark urine, and pruritus.
  - Examination findings such as spider naevi, xanthomas, and hepatosplenomegaly should be looked for and may delineate the underlying pathophysiology.

Investigations

Investigating jaundice should aim to elucidate the underlying cause and, as such, investigations should be guided by history and examination.

- *Blood tests*. FBC, U&Es, LFTs, clotting profile, Coombs' test (haemolytic disorders), reticulocyte count, hepatitis screen, EBV, CMV, ferritin, α1-antitrypsin, immunoglobulins, anti-smooth muscle antibodies (chronic active hepatitis), anti-mitochondrial antibodies (primary biliary cirrhosis), autoantibodies, AFP, copper (Wilson's).
  - *LFTs*—useful in identifying pre-hepatic, hepatic, and post-hepatic cause, based on ratios of ALP and aminotransferase increases, compared to normal.

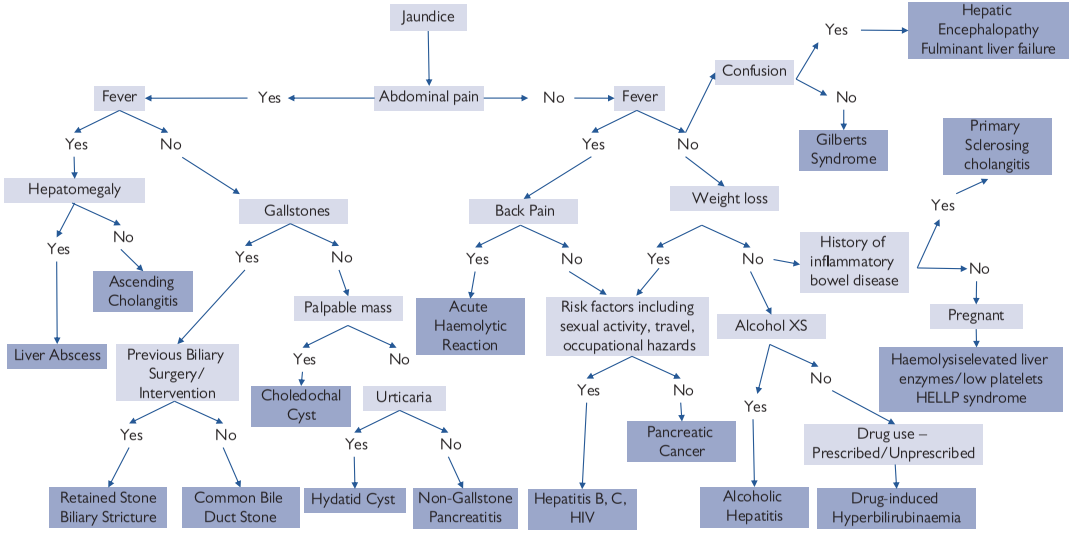

Fig. 9.1 Jaundice algorithm.

- *Tumours markers* may be useful if suspicion of underlying malignancy.
- *Urinalysis*—presence or absence of bilirubin, urobilinogen.
- *USS*—excludes the presence of extra-hepatic obstruction and may identify underlying pathology.
- *MRCP*—more sensitive to identify underlying lesions as cause of biliary obstruction.
- *CT*—cross-sectional imaging may be useful, depending on the cause.
- *EUS*—microlithiasis, head of pancreas lesions, and allows for EUS-guided biopsy if required.
- *Liver biopsy*—for suspected hepatitis and cirrhosis.

Treatment

General

Management of acute presentation of jaundice will depend on the underlying cause. Acute general management should be performed in an ABCDE approach with care to:

- Correct dehydration and monitor urine output (caution in patients with pre-existing or suspected liver disease).
- Treat sepsis, antibiotics as required after appropriate cultures.
- Correct clotting abnormalities—give vitamin K 10mg IV stat if prolonged PT.
- Consider nutritional requirements and enteral feeding options.

Specific treatments

- Pre-hepatic.
  - Immunoglobulin infusion is 1° treatment for pre-hepatic jaundice.
  - Phototherapy may be considered for high bilirubin levels.
  - Rarely splenectomy may be considered if failed medical therapy.
- Hepatic jaundice.
  - Supportive therapy (fluids, analgesia) for hepatitis A.
  - Interferon therapy for hepatitis B and C.
  - Steroids and immunosuppressant may be used in autoimmune hepatitis.
  - Detoxification and abstinence for alcohol-induced hepatitis/cirrhosis.
  - Cessation of causative agents, e.g. medications.
  - Liver transplantation may be required if end-stage in liver failure.
- Post-hepatic (mechanical decompression).
  - ERCP and sphincterotomy ± stent insertion (plastic or expanding metal).
  - Percutaneous transhepatic cholangiogram (PTC)—allows insertion of stent and temporary external drainage of obstructed biliary tree.
  - Surgical drainage and/or surgical resection (➲ Chronic pancreatitis, p. 18).

Complications

- *Infection*. Cholangitis 2° to obstructed biliary system. Typically Gram-negative bacteria (*Escherichia coli*, *Pseudomonas*).
- *Renal failure*. Hepatorenal syndrome. Caused by a combination of dehydration, underlying sepsis, and high bilirubin levels having a toxic effect on the kidney. High associated mortality in >65y.

- *Coagulopathy*. Reduced synthesis of vitamin K-dependent clotting factors (II, VII, IX, X) and impaired platelet function.
- *Immunosuppression*. Hyperbilirubinaemia predisposes to opportunistic systemic infection and poor wound healing because of reduced protein synthesis.

Prognosis in acute jaundice—risk factors

- Age >65y.
- Elevated urea.
- Bilirubin >200g/L.
- Underlying malignancy.
- Sepsis and multiorgan failure.

Gallstones

Key facts

Common; present in ~15% of adult population.

Pathophysiology

Gallstones principally develop as a result of supersaturation and excessive secretion of cholesterol or conjugated bilirubin.

Two principle types of gallstone:

- *Cholesterol* (20% pure, 50% mixed). Cholesterol solubility is dependent on bile acids and phospholipids (90% lecithin). Imbalance (↑ secretion of cholesterol 2° to obesity or hormones) leads to stone formation.
- *Pigment stones* (30%). Black and brown.
  - Black: form in sterile gall bladder, comprise calcium bilirubinate, associated with haemolytic disorders (e.g. sickle cell anaemia).
  - Brown: outside gall bladder, unconjugated bilirubin and calcium salts, associated with biliary infection and parasites.

Risk factors

- Ethnicity (cholesterol stones predominate in Western population).
- ♀ sex—52 times more likely to form stones than ♂.
- Family history and genetics—five times elevated risk.
- Age—frequency increases with age. Marked increase >40.
- Obesity—25% of morbidly obese have evidence of gallstones.
- DM—a correlation with lipid abnormalities.
- Diet—↑ with high-cholesterol, high-carbohydrate diet.
- Drugs—OCP, octreotide, thiazide diuretics.
- Chronic diseases—cirrhosis, Crohn's disease, cystic fibrosis (CF), sickle cell.

Clinical features

- *Asymptomatic*. Only 1–4% per year develop symptoms.
- *Biliary colic*. Classically intermittent abdominal pain localized to epigastrium/RUQ. Often postprandial and related to fatty food intake. Associated nausea and vomiting. Typically lasts 1–5h in duration before self-resolving. Non-specific abdominal examination findings.
- *Acute cholecystitis*. Severe, persistent, well-localized abdominal pain in RUQ. Radiation to back and around flanks. Associated nausea, anorexia, and fever. Abdominal rebound and guarding, positive Murphy's sign (non-specific) on examination.
- *Chronic cholecystitis*. Repeated episodes of infection resulting in a thick-walled and fibrotic gall bladder.
- *Mucocele*. A distended gall bladder filled with mucoid, resulting from outlet obstruction, typically 2° to impacted gallstones in the neck of the gall bladder. Presents with RUQ pain.
- *Empyema*. Gall bladder abscess.

Key revision point—Murphy's sign

Inspiration causes the gall bladder to descend onto the fingers, producing pain if the gall bladder is inflamed.

Diagnosis and investigations

- FBC, U&Es, LFTs, amylase/lipase, clotting.
- Blood cultures if spike in temperature.
- AXR—exclude perforation, 10% stones radio-opaque.
- Ultrasound—first line, 95% sensitivity, rapid, and non-invasive.
- MRCP if suspicion of CBD stones, bile duct dilated on USS, but no stones identified and/or LFTs abnormal.
- Other. EUS, hepatobiliary iminodiacetic acid (HIDA) scan, CT.

Management

- *Reassurance*—if asymptomatic, with normal gall bladder and normal biliary tree, no treatment required.
- *Laparoscopic cholecystectomy (LC)*—first-line treatment of choice for all patients presenting with symptomatic gallstone disease or asymptomatic patients with a risk of complication (diabetics, gall bladder polyps (>10mm), porcelain gall bladder (20% associated with adenocarcinoma). Elective day case procedure if uncomplicated, but early LC (within 1 week of diagnosis) recommended for those patients presenting with acute cholecystitis. Risk of LC—bleeding (1%), conversion to open (~5%), bile leak (1%), bile duct injury (0.3–0.7%).
- *On-table cholangiogram*—used intraoperatively to confirm anatomy or exclude CBD stones. Evidence for routine versus selective use remains equivocal.
- *Percutaneous drainage of the gall bladder (cholecystostomy)*—for those patients deemed unfit or not suitable for LC. Two options—transhepatic or transperitoneal performed by USS or CT guidance. LC should be reconsidered after resolution of symptoms.
- *ERCP* (➲ *Endoscopic retrograde cholangiopancreatography*, p. 415). CBD stones.

Gallstones and pregnancy

↑ progesterone results in reduced gall bladder contraction, causing biliary stasis, with 10% of women developing sludge or stones during the course of their pregnancy. Mostly asymptomatic, but acute cholecystitis affects 8/100 000 pregnancies and pancreatitis (1 in 1000). Early surgery is recommended usually in second trimester if patients present with acute cholecystitis or gallstone pancreatitis.

Common bile duct stones

Key facts

Incidence of 10–20% in those patients with symptomatic gallstones.

Pathophysiology

- For types of gallstones, ➲ Gallstones, p. 412.
- Most are migratory and originate in the gall bladder before moving to the CBD.
- *De novo* 1° CBD stones may form due to parasitic infection.
- Normal USS and LFTs at time of gallstone precipitation suggests incidence of CBD stones is <5%.

Clinical considerations

- Can be asymptomatic and found as an incidental finding on imaging.
- Obstructive jaundice (➲ Jaundice, p. 406).
- Ascending cholangitis—classically Charcot's triad: RUQ pain, obstructive jaundice, and high fever 2° to an infected (*Escherichia coli* 27%, *Klebsiella* 16%, *Enterococcus* 15%) and obstructed biliary system. Managed by treating sepsis and shock, IV antibiotics, and emergency decompression of biliary tree.
- Acute pancreatitis (➲ Acute pancreatitis, p. 416).

Risk factors

A combination of the following variables predicts the probability of a stone being found to be 70%.

- Age >55.
- Dilated ducts on USS.
- Serum bilirubin >30 micrograms/L.

Diagnosis and investigations

- FBC, U&Es, LFTs (? obstructive jaundice), amylase/lipase, clotting, blood cultures.
- USS—first line. Identifies duct dilatation and CBD stones. Low sensitivity and user variability. Interpretation can be limited in obese and as a result of overlying bowel gas.
- MRCP—gold standard. Non-interventional, 100% sensitive for stones >1cm, 71% sensitive for stones <5mm. No radiation.
- EUS—invasive, operator-dependent, limited availability, no intrahepatic duct imaging. High sensitivity.
- ERCP—most sensitive and specific test for CBD stones. However, interventional nature and associated risks means use as diagnostic test is no longer justified.

Management

All patients with symptomatic or asymptomatic CBD stones should undergo LC where possible and either surgical or endoscopic CBD stone extraction. Options for duct clearance include the following.

Endoscopic retrograde cholangiopancreatography

- Biliary sphincterotomy and stone extraction with balloon catheter.
- Stent insertion for unextractable stones with no surgical option.
- Risks (➔ Endoscopic retrograde cholangiopancreatography, p. 366).
- Sphincterotomy to prevent recurrence may be considered a definitive procedure in the elderly and frail not fit for further surgical intervention.

Laparoscopic bile duct clearance

- Laparoscopic transcystic exploration at time of LC. Patients with stones smaller than 10mm in the distal CBD, below the cystic duct insertion are suitable. Stones are retrieved with a Dormia basket or a Fogarty® biliary catheter.
- Laparoscopic/open bile duct exploration (cholodochotomy). Allows for removal of larger occlusive stones via 10–15mm longitudinal incision of the bile duct. Following stone removal, closure should either be with a biliary stent or with a T-tube. Stenting is associated with lower morbidity, shorter hospital stay, and earlier return to daily activities.

Others

- Mechanical lithotripsy.
- Extracorporeal shock wave lithotripsy (ESWL).
- PTC—used as a therapeutic procedure following failure of ERCP. Allows biliary drainage as a temporizing measure prior to definitive intervention where possible. Risks include sepsis, tube displacement, leakage, and dehydration.

Acute pancreatitis

Key facts

Incidence is increasing; 50% result of gallstones, 25% related to alcohol.

Pathophysiology

Inflammatory disease resulting from premature intra-acinar cell activation of digestive zymogen and lysosome enzymes. Cell damage induces an inflammatory response, resulting in interstitial oedema, inflammatory cell recruitment, activation, and cytokine release.

Aetiology and risk factors

- Gallstones (50%)—usually small <6mm, need definitive management on index admission if mild and moderately severe.
- Ethanol (25%).
- Hyperlipidaemia/hypercalcaemia/hypothermia.
- Iatrogenic causes (ERCP).
- Trauma.
- Drugs (azathioprine, diuretics).
- Idiopathic (should be no more than 20% of cases).

History and clinical features

- History of previous gallstones, alcohol intake, family history, drug intake, any prodromal symptoms, recent infections or virus.
- Severe epigastric pain radiating to back, nausea and vomiting.
- Signs of sepsis—oliguria, hypotension, tachycardia.
- Clinical examination may reveal epigastric tenderness with local or diffuse peritonism.
- Grey Turner's sign (left flank ecchymosis) and Cullen's sign (periumbilical ecchymosis) are rare but can be indicative of retroperitoneal bleeding and associated with pancreatic necrosis and haemorrhagic pancreatitis.

Diagnosis and investigations

- Any two of: (1) abdominal pain; (2) serum amylase three times above upper limit of normal; and (3) CT evidence of pancreatitis.
- FBC, U&Es, LFTs (? obstructive jaundice), clotting, Ca^{2+}.
- Amylase. Diagnostic if three times normal, but may not be elevated, even in severe cases; should be interpreted in relation to onset of pain and is raised in alternative diagnosis (ischaemic bowel, perforated viscus, leaking AAA).
- Serum/urinary lipase. Pancreas is only source of lipase and therefore superior specificity and sensitivity, but not always widely available.
- Plain AXR/erect CXR—exclusion of differential diagnosis (perforated viscus).
- USS—within 24h of admission to exclude gallstones and CBD dilatation.
- CT—only if diagnostic uncertainty or to exclude early complications.

Prognostic features of severity and severity scoring

Classification (Atlanta 2012)[1]

- Mild—no organ failure, no complications.
- Moderate—transient (<48h) organ failure ± local complications.
- Severe—persistent organ failure >48h.
- Marshall scoring system of organ dysfunction.
- Glasgow score no longer used routinely.

Management

- Aim for euvolaemia (effective MAP and urine output >0.5mL/kg).
- Catheter and strict fluid balance.
- Supplemental O_2 to maintain arterial saturation >95%.
- Early critical care involvement—invasive monitoring.
- Antibiotics remain controversial—usually not indicated; discuss with tertiary centre.
- Do not routinely fast—early enteral feeding via NGT if tolerated. Nasojejunal (NJ) feeding if not. TPN as last resort.
- Urgent ERCP in patients with concomitant cholangitis.

Complications

- *Local*:
  - Acute fluid collection (<4 weeks from onset).
  - Acute necrotic collection (<4 weeks from onset).
  - Gastric outlet dysfunction.
  - Splenic or portal vein thrombosis.
  - Colonic necrosis.
- *Systemic*:
  - Exacerbation of pre-existing comorbidity.
  - Multiorgan failure.

Reference

1 Banks PA, Bollen TL, Dervenis C, *et al*. (2013). Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut* **62**: 102–11.

Chronic pancreatitis

Key facts

Chronic inflammatory condition characterized by irreversible pancreatic tissue destruction. Increasing incidence and prevalence.

Pathophysiology

Irreversible inflammatory process characterized by progressive fibrotic destruction of the pancreatic secretory parenchyma. Various theories regarding underlying mechanism. Recent data suggest a key role of pancreatic stellate cells. Process driven by a complex interplay of inflammatory mediators leading to extracellular matrix deposition and fibroblast proliferation. Pain results from ductal obstruction, parenchymal hypertension, and inflammation.

Causes

- Alcohol is commonest cause in Western world (60–70%).
- Cryptogenic/idiopathic (*of uncertain origin*) (30%).
- Metabolic disorders (hypercalcaemia).
- Pancreatic duct obstruction, e.g. pancreatic trauma, iatrogenic (post-ERCP stricture), pancreatic divisum, peri-ampullary tumours.
- Hereditary (mutations in the *trypsinogen 3* gene, *CFTR* gene).
- Autoimmune—usually associated with raised immunoglobulin G4 (IgG4) levels.

Clinical features and presentation

- Persistent, recurrent episodes of epigastric pain radiating through to back; 5–10% of patients, however, remain pain-free.
- Nausea and vomiting.
- Anorexia and weight loss.
- Exocrine dysfunction.
  - Steatorrhoea and fat malabsorption—soft, greasy, foul-smelling stools that float. Gross steatorrhoea present if pancreas exocrine function <10% of normal level.
  - Protein malabsorption leading to anorexia and weight loss.
- Endocrine dysfunction.
  - 2° to loss of β islet cells.
  - Two-thirds of patients have abnormal glucose tolerance tests, 30–50% type 1 DM.
- Incidental pancreatic calcification findings on plain X-ray or CT.

Diagnosis and investigation

- Serum amylase is usually normal.
- Plain AXR *may* show pancreatic calcification
- Triple-phase pancreatic protocol CT—may show calcification, pancreatic atrophy, duct dilatation, structural duct abnormalities, or pseudocyst.
- MRCP—may help identify ductal strictures and localize calculi.
- EUS—most sensitive. Demonstrates parenchymal calcification, loss of lobularity, and gland texture using elastography. Rosemont classification used to standardize.

Treatment

- *Non-surgical*:
  - Cessation of causative habits—smoking and alcohol.
  - Pancreatic enzyme replacement therapy—Creon® (pancrelipase).
  - Control of DM (type 3c).
  - Medical pain control and referral to chronic pain team.
  - Nutritional support and dietary advice (high protein, low fat).
  - ERCP trial stenting of pancreatic duct strictures.
- *Surgical*:
  - Surgical intervention is aimed at reducing pain and preventing further parenchymal damage via pancreatic duct drainage or resection of the inflammation in the head of the gland.
  - Drainage procedures (relieve ductal pressure) include:
    - Duval procedure—distal pancreatectomy, splenectomy, and end-to-side pancreaticojejunostomy.
    - Puestow–Gillesby procedure—distal pancreatectomy, splenectomy, and longitudinal pancreaticojejunostomy.
    - Partington–Rochelle variant of the Puestow—longitudinal pancreaticojejunostomy without distal pancreatectomy and splenectomy.
  - Resection procedures (remove inflammatory sequelae) include:
    - Whipple's procedure—pancreaticoduodenectomy.
    - Pylorus-preserving pancreaticoduodenectomy (PPPD).
    - Beger procedure—duodenum-preserving pancreatic head resection.
  - Combined drainage and resection:
    - Frey procedure—pancreatic head resection and longitudinal pancreaticojejunostomy.
  - Total pancreatectomy is reserved as a salvage operation for those patients with continued symptoms despite multiple less radical surgical interventions. Results in brittle diabetes.
- Islet cell autotransplantation. May provide delayed onset or prevention of DM in those patients undergoing radical pancreatectomy.
- Surgical options for pain management include:
  - Splanchnicectomy (EUS-guided or thoracoscopic).
  - RFA.

Complications

- Pseudocyst formation.
- Biliary duct obstruction.
- Gastric outlet or duodenal obstruction.
- GI bleed 2° to pseudoaneurysm formation or variceal bleeding (splenic vein thrombosis may lead to gastropathy and bleeding; splenectomy may be indicated).
- Pancreatic cancer—4% lifetime risk in patients with chronic pancreatitis.

Pancreatic cancer

Key facts

Rapidly increasing incidence and prevalence. Poor prognosis; >95% of those affected will die as a direct result, with 5y survival rate of ≈5%.

Key revision point—Courvoisier's Law
'*A palpable, non-tender gallbladder in the presence of jaundice is unlikely to be due to gallstones.*'

Pathophysiology

- 65% head, 15% body, 10% tail, 10% multifocal.
- Associated with mutations in *KRAS2* oncogene.
- 90% adenocarcinoma, 10% mucinous, <5% pancreatic islet cell tumours.

Risk factors

- Smoking—3-fold increase in risk.
- Hereditary—10% may have an inherited component, e.g. Peutz–Jeghers syndrome, *BRCA2* mutation, Lynch syndrome, *p16* tumour suppressor gene mutations.
- Other potentially associated factors include high-fat diet, obesity, alcohol excess, chronic pancreatitis, and DM.

Clinical presentation

- Often presents with late, advanced disease.
- Painless obstructive jaundice, abdominal mass (late), non-specific abdominal pain, weight loss and anorexia, Courvoisier's sign.
- 80% of cases present between 65 and 75y of age.

Diagnosis and investigations

- FBC, U&Es, LFTs, clotting, random blood glucose.
- CA 19-9—sensitivity 70%, specificity 90% (false positives 2° to obstructive jaundice or chronic pancreatitis). Level correlates with tumour volume.
- USS—may demonstrate lesions >2cm and identify sequelae of obstruction; Doppler may identify portal vein flow and associated vasculature.
- Pancreatic protocol CT—identifies size and location of lesion, and extent of local and distant spread; 90% accurate in predicting resectability.
- MRCP—detailed information regarding duct involvement.
- ERCP—invasive, but useful if diagnostic uncertainty. Provides symptomatic relief from biliary obstruction and cytology/histological samples for tissue diagnosis.
- EUS—increases sensitivity to CT for small tumours (<3cm) and specific vascular involvement. Can be used also for FNA.
- PET—identification of local and distant disease involvement.
- Staging laparoscopy—useful for borderline resectable lesions. Identifies peritoneal, liver capsule, and serosal involvement.

Treatment and management

Most pancreatic tumours (90%) are not suitable for resection, due to local vascular involvement, or distant spread, or patient factors. Decisions should be via an MDT and consideration should also be given to available novel therapy clinical trials.

- Stages I and II—resectable disease:
  - Surgical options depend on site and extent of disease.
  - Pancreatic head tumours. Tumour without involvement of SMA or coeliac axis, patent superior mesenteric–portal confluence, no evidence of metastases:
    - Whipple's procedure—pancreaticoduodenectomy with antrectomy.
    - PPPD.
  - Body and tail tumours:
    - Laparoscopic/open distal pancreatectomy ± splenectomy.
    - Adjuvant chemotherapy—3- to 6-month overall survival benefit.
    - Pancreatic replacement therapy (500–2500 lipase units/kg/meal).
    - Nutritional support may be required.
- Stage III—locally advanced unresectable disease:
  - Tumours involving nearby structures, making them unresectable (SMA, coeliac axis), but in the absence of distant metastasis:
    - ERCP and self-expanding covered metal stent insertion.
    - Biliary bypass surgery if endoscopic stenting fails.
    - Gemcitabine-based combination chemotherapy.
    - Some evidence for benefit of consolidation radiotherapy.
- Stage IV—metastatic disease:
  - Patients have limited survival of 3–6 months and, as such, treatment is based on symptom control:
    - ERCP and palliative biliary stenting.
    - Symptoms of gastric outlet obstruction due to mass effect or infiltration into duodenum may require surgical bypass with gastroenterostomy.
    - Palliative chemotherapy with gemcitabine monotherapy or combination strategies for those patients with good performance status.
    - Relief of pain—analgesic pain ladder often requiring morphine, percutaneous or EUS-guided coeliac block, or splanchnicectomy.
    - Psychosocial support—use of palliative care team.

Hepatocellular carcinoma

Key facts

Commonest 1° liver cancer, representing >90% of all liver cancers; however, accounts for <1% of all new cancers in the UK. Third leading cause of cancer death worldwide.

Pathophysiology

- Underlying mechanism results from chronic inflammation with necrosis and fibrosis leading to cellular regeneration and the development of HCC. Underlying conditions are summarized below, but 90% of cases have established cirrhosis.
- Often solitary nodules. Diffuse HCC is rare.

Aetiology and risk factors

- Cirrhosis (congenital or acquired).
- Hepatitis B and C.
- Haemochromatosis (iron deposition).
- Wilson's disease (copper deposition).
- Alcohol.
- Non-alcohol fatty liver disease (NAFLD).
- Aflatoxins (e.g. *Aspergillus*).
- Once established cirrhosis present, independent risk factors include:
  - ♂ (high androgens, low oestrogens), age, and DM.

Clinical presentation

- Often incidental finding on routine screening for liver disease.
- Acute presentation symptoms may include RUQ pain, obstructive jaundice 2° to biliary invasion, or signs and symptoms of portal HTN.
- In rare cases, a ruptured HCC may present as an acute abdomen.

Investigations

- Bloods. LFTs may be deranged.
- Serum AFP >400ng/mL, with concordant imaging is diagnostic.
- Triple-phase CT—will identify hepatic and extra-hepatic disease. HCC lesions have distinctive CT findings:
  - Hypervascular lesions.
  - Show arterial enhancement and rapid washout in portal phase.
- MRI liver—good at identifying HCC from other lesions.
- Aim to avoid biopsy as risk of tumour seeding is 3–7%.

Management

Options depend on tumour burden, grade, and underlying liver function.

- Sorafenib (tyrosine protein kinase inhibitor)—advanced disease.
- Surgical resection.
- Transarterial chemoembolization (TACE).
- Selective internal radiotherapy (SIRT).
- RFA.

Cholangiocarcinoma

Key facts

Arise from intrahepatic or extra-hepatic biliary epithelium; 90% adenocarcinoma, 10% squamous cell.

Risk factors

- Primary sclerosing cholangitis.
- Congenital cystic disorders (Carolis disease—15% lifetime risk).
- Liver fluke (*Clonorchis sinensis*) infection.
- Cirrhosis.
- Chronic viral hepatitis B and C infection.

Pathophysiology and classification

- Affects three anatomic regions: intrahepatic (within the liver), extrahepatic (outside the liver), and perihilar.
- Perihilar lesions are classified using the Bismuth–Corlette classification system.
- Three unique subtypes: sclerosing, nodular, and papillary.

Diagnosis

Patients typically present with obstructive jaundice and/or systemic manifestations of malignancy (weight loss, lethargy, anorexia). Usually thereafter, a radiological finding on USS/MRCP. Staging is by CT/liver MRI, and EUS.

Management

- Often presents late with advanced presentation, and placement of metal stent (via ERCP or PTC) allows biliary drainage.
- Surgical resection or rarely transplant can offer potential cure. Only one-third of patients at presentation have resectable disease.
- Highly chemoresistant in nature.

Gall bladder cancer

Key facts

Rare; diagnosed late with very poor outcomes.

Pathophysiology

- Adenocarcinoma (80%): fundus (60%), body (30%), neck (10%).
- Genetic susceptibility and association with chronic gall bladder inflammation.
- 90% associated with gallstones.
- Overall survival <10% at 5y.

Treatment

- T1—open cholecystectomy and regional node sampling.
- T2/T3—cholecystectomy and formal resection of segments IVb/V.
- T4—best supportive care.

Colorectal liver metastases

Key facts

Seventy per cent of patients with colorectal metastases will have disease confined to the liver alone; 50% of colorectal cancer (CRCa) patients will be diagnosed with liver metastases, of which only 15% will be resectable.

Surveillance

(➲ Colorectal cancer, p. 500.)

Imaging

- If recurrence suspected, or as part of initial CRCa staging, imaging should be via triple-phase CT (hypoattenuating lesions).
- If liver lesion confirmed and further surgery considered, liver MRI (hypodense on T1, hyperdense on T2-weighted image) and CT-PET to exclude extra-hepatic disease should be performed.

Liver remnant function

- Liver is only a visceral organ capable of regeneration and will restore any lost mass and adjust to the size of the organism, while continuing to provide complete homeostasis to the host.
- Restoration to original size will normally occur within 3 months.
- Adequate liver volume to provide sufficient liver function is linked to physiological state of underlying liver. The future liver remnant (FLR), the liver remnant remaining after resection, should be at least 20% for healthy liver and 40% if underlying liver disease.
- FLR may be optimized prior to resection with selective preoperative portal vein embolization.

Classification of resectable disease

- Usually resectable—≥4 involved segments and a residual liver volume of >40%.
- Potentially resectable—5–6 segments involved, central hepatectomy or need for vascular reconstruction.
- Not resectable—extra-hepatic disease, involvement of two portal branches or three hepatic veins.

Management

Depending on disease profile and liver physiology described above, options include:

- Neoadjuvant chemotherapy followed by laparoscopic/open liver resection.
- 1° laparoscopic/open liver resection.
- Synchronous colorectal and liver resection—generally not a favoured approach.
- Adjuvant biological therapies—cetuximab/panitumumab in *KRAS*-positive patients.
- RFA or microwave ablation.

Benign liver lesions

Haemangioma

- Commonest benign tumour of the liver.
- Very low risk of rupture or bleeding.
- Separated from liver with ring of fibrous tissue.
- Usually no indication for resection.

Focal nodular hyperplasia (FNH)

- Well-circumscribed areas with a central scar on imaging.
- Nodular polyclonal proliferation within the liver.
- Usually no indication for resection, unless required for symptom control or inability to exclude malignant potential.

Liver cell adenoma

- Predominantly found in women in third to fifth decade of life.
- Linked to use of oestrogen-containing OCP.
- Surgical resection should be considered in lesions >5cm.
- Can be differentiated from FNH on MRI.

Hamartomas

- Second commonest benign liver lesion in children.
- Pathogenesis is poorly understood; however, typically present in <3.
- Can grow to enormous sizes and usually require resection.

Hydatid cysts

- Result from *Echinococcus* infection.
- Cysts have no epithelial lining.
- Present with malaise, RUQ pain, abnormal LFTs, and raised eosinophils.
- Percutaneous aspiration prior to sterilization is contraindicated. Cysts are sterilized with albendazole or mebendazole prior to surgical resection or PAIR technique (puncture, aspiration, injection, reaspiration).

Liver abscess

- Usually precipitated by biliary sepsis.
- Symptoms include fever, RUQ, malaise, and jaundice (50%).
- USS/CT usually demonstrate a fluid-filled cavity.
- Bacterial or amoebic in origin.
- If small, may resolve with antibiotics or amoebicides.

Liver cysts

- 5% of people.
- Symptomatic cysts or those at risk of rupture should undergo laparoscopic/open de-roofing and marsupialization.

Hepatic cyst adenoma

- Rare, but malignant potential.
- Can occur at any age, but usually seen in middle-aged women.
- Solitary, multi-loculated lesions, 85% hepatic, 15% extra-hepatic biliary system.
- USS—large, anechoic fluid-filled area with irregular margins.
- Due to malignant potential, should all undergo surgical resection.

Liver cirrhosis

Key facts

- Commonest cause of liver failure in the UK.
- Multiple causes, but commonest is alcoholic liver disease (30%).

Pathophysiology

- Characterized by chronic hepatic injury of various underlying pathology, with healing occurring through nodular regeneration and fibrosis of the liver parenchyma. Fibrosis leads to disruption of liver architecture, hepatocellular necrosis, and portal HTN.
- Micronodular form.

Causes

- Toxins:
  - Alcoholic liver disease.
  - Drugs—amiodarone, methotrexate.
- Infection:
  - Chronic viral hepatitis B and C.
  - Schistosomiasis (common in South America and Africa).
- 1°/autoimmune:
  - Primary biliary cholangitis.
  - Primary sclerosing cholangitis.
  - Autoimmune hepatitis.
- Metabolic:
  - NAFLD/non-alcoholic steatohepatitis (NASH).
  - Obesity.
  - Haemochromatosis.
  - Wilson's disease.
  - α1-antitrypsin deficiency.
- Biliary obstruction (2° biliary cirrhosis):
  - Mechanical obstruction (gallstones, cholangitis, stricture).
  - Biliary atresia.
  - CF.
- Other:
  - Sarcoidosis.
  - Cryptogenic.

Clinical features

- Relate to end-stage outcomes of cirrhosis, portal HTN, liver insufficiency, and ultimately liver failure.
- Compensated cirrhosis:
  - Biochemical, radiological, and histological findings consistent with cirrhosis; however, no evidence of portal HTN or loss of hepatic synthetic function.
- Decompensated cirrhosis:
  - Evidence of reduced liver function or hepatocellular failure and portal HTN (Portal hypertension, p. 428).

- Signs of hepatocellular failure:
  - Jaundice.
  - Spider naevi.
  - Ascites.
  - Bruising, coagulopathy, and clotting disorders.
  - Gynaecomastia and testicular atrophy.
  - Encephalopathy.
  - Hepatorenal syndrome.

Diagnosis and investigations

- FBC (platelets reduced), U&Es, (Na^+ reduced), LFTs (deranged, albumin reduced), clotting screen (prolonged PT).
- Hepatitis screen (A, B, C, D, E; EBV and CMV).
- Metabolic screen (haemochromatosis, iron studies; Wilson's, serum copper and caeruloplasmin (low)).
  - Autoimmune screen—anti-mitochondrial antibodies (primary biliary cirrhosis), anti-smooth muscle antibody and antinuclear antibodies (ANA) (autoimmune hepatitis), plasma α1-antitrypsin and serum protein electrophoresis (α1-antitrypsin deficiency).
- Abdominal ultrasound—small liver, surface nodularity, ascites, portal vein diameter and flow, splenomegaly.
- CT and MRI.
- Upper GI endoscopy—identification of varices, gastropathy.
- Ultrasound-guided liver biopsy—confirm type, activity, and cause.

Treatment

- Treatment is focused on removing causal factors or preventing disease progression:
  - Abstinence from alcohol, avoidance of hepatotoxic drugs (NSAIDs, high-dose paracetamol).
  - Interferon therapy for hepatitis B.
  - Oral antiviral agents for hepatitis C.
  - Immunosuppression for autoimmune causes.
  - Ursodeoxycholic acid for primary biliary cirrhosis.
- Treatment of complications:
  - Portal HTN (➔ Portal hypertension, p. 428).
  - Ascites—diagnostic paracentesis to confirm diagnosis. A serum ascites albumin gradient (SAAG) >1.1g/dL indicates portal HTN. Treatment should involve sodium restriction and use of diuretics (furosemide and spironolactone). Paracentesis and albumin replacement for tense symptomatic ascites.
  - Encephalopathy—aim to reduce nitrogen absorption. Dietary protein restriction, PO lactulose, PO antibiotics (rifaximin).
  - Decompensated hepatocellular failure—liver transplant.
- Liver transplantation (➔ Liver transplantation, p. 832).

Portal hypertension

Key facts

Occurs as a result of ↑ resistance in the portal venous systems. Normal pressure is 5–10mmHg; however, is said to be pathological at >12mmHg.

Key revision points—anatomy of the portal circulation

- Portal circulation carries blood from the GI tract (distal oesophagus to the anorectal junction) to the liver.
- Stomach, pancreas, and portions of the large bowel drain into the splenic vein via various anatomical tributaries, including the inferior mesenteric vein.
- Small bowel, portion of large bowel, stomach, and pancreas drain ultimately into the superior mesenteric vein.
- The splenic vein and superior mesenteric vein merge to form the portal vein, which splits into right and left, supplying each respective half of the liver.
- Portosystemic anastomoses occur in junctional areas of venous drainage between the two systems:
  - Left gastric vein (portal) and oesophageal veins (azygos veins).
  - Superior rectal vein (portal) and inferior rectal vein (pudendal veins) in lower rectum.
  - Umbilical vein (portal) and epigastric veins at umbilicus.

Causes

- *Pre-hepatic*.
  - Portal vein thrombosis (congenital malformation, pancreatitis, tumour, trauma).
- *Hepatic*.
  - Cirrhosis (➔ Liver cirrhosis, p. 426).
  - Schistosomiasis (leading cause globally).
  - Intrahepatic tumours (cholangiocarcinoma, HCC).
- *Post-hepatic*.
  - Budd–Chiari syndrome (thrombosis or obstruction of hepatic vein, 2° to malignancy, haematological disease, or OCP).
  - Constrictive pericarditis.
  - Right-sided heart failure.
- ↑ *portal blood flow*.
  - Arterial–portal venous fistula.
  - ↑ splenic flow.

Complications

Increases in portal pressure cause development of portosystemic collateral circulation, with resultant portosystemic shunting. Resistance in portal veins is normally lower than that of the systemic system, so blood flows from systemic system to portal system. Increases in portal pressure causes reversed flow. Various complications can result.

- Gastro-oesophageal varices—30% will bleed, with a mortality of 50% per episode. Bleeding can be catastrophic and often patients have concomitant coagulopathy 2° to underlying liver disease.
- Ectopic varices—large portosystemic varices occurring anywhere in the abdomen, except the gastro-oesophageal region.
- Portal hypertensive gastropathy—characteristic snake skin appearance on endoscopy.
- Gastric antral vascular ectasia (GAVE)—collection of ectatic vessels in antrum of stomach.
- Ascites and spontaneous bacterial peritonitis (SBP)—cirrhotic portal HTN accounts for ~75% of patients with ascites. Patients with ascites are prone to infection of the ascitic fluid—SBP.
- Hepatorenal syndrome.
- Hepatic encephalopathy.
- Splenomegaly—can be associated with thrombocytopenia, leucopenia, and anaemia. Results from congestion of splenic venous drainage.

Classification

Child–Pugh classification is used to assess severity (see Table 9.1).

Table 9.1 The Child–Pugh classification of portal hypertension

	1 point	2 points	3 points
Bilirubin (¼mol/L)	<34	34–51	>51
Albumin (g/L)	>35	28–35	<28
PT (s)	<3	3–10	>10
Ascites	None	Moderate	Moderate to severe
Encephalopathy	None	Moderate	Moderate to severe

Grade A: 5–6 points; Grade B: 7–9 points; Grade C: 10–15 points.

Treatment

- Oesophagogastric varices:
  - β-blockers (propranolol) reduce portal venous pressure.
  - Endoscopic injection sclerotherapy and variceal ligation.
  - Portosystemic shunts.
- Rectal varices:
  - Injection sclerotherapy.
- Splenomegaly (if symptomatic)
  - Laparoscopic or open splenectomy.
- Ascites
  - PO diuretics (spironolactone and/or furosemide).
  - Paracentesis.

Chapter 10

Abdominal wall

Abdominal wall hernias

A surgeon can do more for the community by operating on hernia cases and seeing that his recurrence rate is low than he can by operating on cases of malignant disease.

Sir Cecil Wakely (1940)[1]

Definition of a hernia
The protrusion of a viscus or part of a viscus through the wall of a cavity in which its contents are contained.

Aetiology

- *Congenital*. Associated with a variation in embryological anatomy—persistent process us vaginalis (infantile inguinal hernia) or failure of complete closure of umbilical opening (infantile umbilical hernia).
- *Acquired*. Weakness of the abdominal wall due to ageing or previous surgery; risk increases in conditions where there is ↑ intra-abdominal pressure (heavy lifting, chronic cough, straining on urination or defecation, abdominal distension, ascites, pregnancy).

Characteristics of hernias

- *Reducible*. A hernia that can disappear either spontaneously or on manipulation.
- *Irreducible*. A hernia that cannot be manipulated or reduced to its original cavity.
- *Incarcerated*. An irreducible, painful hernia that requires surgical intervention.
- *Strangulated*. A hernia in which blood supply has been compromised and its contents become ischaemic.

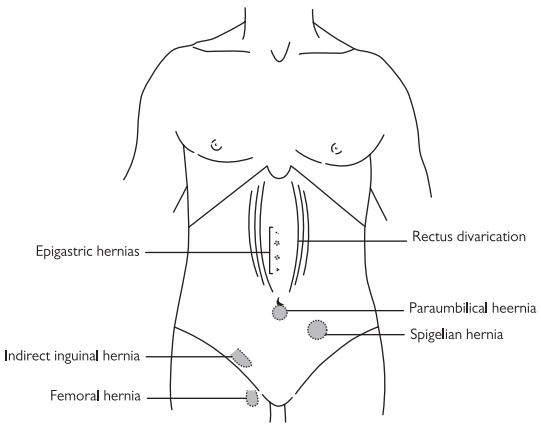

Fig. 10.1 Common sites for hernias.

Reproduced with permission from Callaghan, Chris, et al, Emergencies in Clinical Surgery, Oxford University Press.

1 Reproduced from Wakeley, C. P. G. Treatment of certain types of external herniae. Lancet (1940): 1:6088, p822–826.

Key revision points—general considerations in assessing a patient with a hernia

History

- Ask the patient! Self-diagnosis of a swelling is common.
- Where is the swelling?
- Is it reducible/irreducible? Is it painful? If suspecting incarceration, ask about symptoms of bowel obstruction (any vomiting?).
- Is it a 1° or recurrent hernia? Any previous surgery?
- Patient risk factors? Recent heavy lifting, history of coughing or constipation/difficulty in micturition?

Physical examination

- Confirm swelling and characteristics (reducible/irreducible, cough impulse, anatomical location).
- Examine the patient lying down and standing. Ask them to cough or strain (to elicit reducible hernias).
- Any skin changes or overlying cellulitis?
- Any previous surgical scars?
- eneral examination, including signs of bowel obstruction (distended abdomen).

Investigations

- Diagnosis is usually clinical.
- Plain X-ray is of little value in hernia diagnosis (may show signs of bowel obstruction in strangulated hernias).
- USS may be useful for simple hernias or ruling out other causes of abdominal wall swellings.
- CT is used in complex hernias to determine size and defect location and in the acute situation.

Inguinal hernia

Anatomy of the inguinal canal

- The inguinal canal is the oblique passage through the lower abdominal wall. It runs from the deep to the superficial ring (i.e. from the internal to the external inguinal ring).
- Boundaries of the inguinal canal:
  - *Anteriorly*—aponeurosis of external oblique (internal oblique laterally).
  - *Posteriorly*—transversalis fascia.
  - *Superiorly (roof)*—transversalis fascia, internal oblique, and transversus abdominis.
  - *Inferiorly (floor)*—inguinal ligament (lacunar ligament medially).
- The inguinal canal transmits the spermatic cord (round ligament in the ♀) and the ilioinguinal nerve.
- Contents of the spermatic cord are:
  - Three vessels (testicular artery, cremasteric artery, artery to the vas deferens).
  - Three nerves (genital branch of genitofemoral, autonomic supply to the testicle, ilioinguinal nerve).
  - Three structures (vas deferens, pampiniform venous plexus, testicular lymphatics).
  - Three coverings (external spermatic fascia, cremasteric fascia, internal spermatic fascia).
- The **deep (internal) inguinal ring** is formed through the transversalis fascia (TVF) and lies 1–2cm above the inguinal ligament, midway between the symphysis pubis and the anterior superior iliac spine.
- The **superficial (external) inguinal ring** is a V-shaped defect in the aponeurosis of the external oblique, above, and medial to the pubic tubercle.

Key facts

- It has been estimated that worldwide, >20 million repairs of inguinal hernia are carried each year, and in the UK 100 000.
- Commonest type of abdominal wall hernia; ♂:♀, 8:1.
- Abdominal contents protrude through the inguinal canal.
- Classified as indirect (lateral) and direct (medial), according to its relationship to the inferior epigastric artery (see Table 10.1).
- Direct inguinal hernias pass through a weakened TVF in the **Hesselbach's triangle**, an area bounded by the inguinal ligament inferiorly, the inferior epigastric artery laterally, and the lateral border of the rectus muscle medially.
- A 'pantaloon hernia' is the coexistence of direct and indirect hernias descending on either side of the inferior epigastric vessels.

Clinical features

- Many have no symptoms until a lump is noticed in the groin.
- Ache or dragging sensation, especially towards the end of the day.
- Some patients relate the onset of pain and lump to a specific activity (e.g. lifting).

Table 10.1 Comparison between indirect and direct inguinal hernias

	Indirect	Direct
Age	Any age, but usually young	Uncommon in children and young adult
Aetiology	Congenital (patent processus vaginalis)	Acquired weakness in abdominal wall
Relationship to inferior epigastric artery	Lateral	Medial
Descending to scrotum	Often when neglected	Rarely
Occluding the internal ring	Controls it	Does not control it
Neck	Narrow	Wide
Strangulation	More likely	Rare
Treatment	Infant—herniotomy (ligation and excision of the sac) Adult—open mesh repair, laparoscopic repair	Open mesh repair, laparoscopic repair

Diagnosis and investigations

Often a clinical diagnosis. Radiological investigations may be utilised if diagnosis is uncertain.

- Ultrasound. Least invasive, but may lead to false results and operator-dependent.
- CT and MRI. Highly accurate, but CT involves exposure to radiation.
- Herniography (intraperitoneal contrast injection and subsequent X-ray). Useful in cases of groin pain where no hernia can be palpated, but rarely performed nowadays.

Treatment

- Patients with symptoms or have had episodes of irreducibility or bowel obstruction documented should be offered repair promptly.
- Elderly, immobile patients or those with high morbidity for operation may be safely observed if asymptomatic or mildly symptomatic (annual risk of incarceration is 2–3 per 1000 patients per year).
- A groin truss is generally of limited benefit.

Technical aspects

- Elective surgery for inguinal hernia repair is common.
- Open repairs may be performed under either GA or LA.
- Tension-free reinforcement of the TVF layer (usually with non-absorbable mesh); in open repairs, this lies in front of the TVF and in laparoscopic repairs, behind it).
- Mesh may be fixed in place by sutures (open) or 'tacking' devices (laparoscopic approach).

- Laparoscopic repair can be either transperitoneal (transabdominal pre-peritoneal (TAPP)) or in the pre-peritoneal space (totally extraperitoneal (TEP)).
- Laparoscopic approach is recommended for recurrent and bilateral hernias and should be carried out by experienced surgeons in well-equipped units (NICE guidelines).
- Patients should be advised to avoid heavy lifting and straining for at least 1–2 weeks post-op.
- Lifetime recurrence of mesh repairs is ~<5%.

Femoral hernia

Anatomy of the femoral canal

- Lies medial to the femoral vein within femoral sheath.
- Contains loose areolar tissue and a lymph node known as the lymph node of Cloquet.
- The femoral ring is the abdominal opening of the femoral canal. The ↑ diameter of the true pelvis in ♀ proportionally widens the femoral canal.
- Boundaries to the femoral ring:
  - *Anteriorly*—inguinal ligament.
  - *Medially*—lacunar ligament.
  - *Posteriorly*—pectineal ligament.
  - *Laterally*—femoral vein.
- In the ♀ pelvis, the canal is larger in diameter, thus increasing the risk of femoral herniation. Similarly, the defect can increase in size in the elderly.

Key facts

- Less common than inguinal hernia.
- Commoner in women than in men (but inguinal still commoner than femoral in women).
- Occurs through tissues of femoral canal.
- Easily missed on clinical examination (remember to think of it!).
- Has a high risk of strangulation due to the neck of the sac having bony and ligamentous structures limiting it on three sides.
- ~30% of femoral hernias present as emergencies; 50% of these require bowel resection for strangulation and ischaemia.

Clinical features

- Appears below and lateral to pubic tubercle, medial to femoral pulse.
- Typically not reducible.
- May be asymptomatic until incarceration or strangulation occurs.
- May be mistaken for an upper medial thigh swelling if large.

Diagnosis and investigations

- Differential diagnosis includes:
  - Femoral lymph node.
  - Saphena varix (compressible, disappears when lying flat, palpable thrill on coughing or percussion of the saphenous vein).
  - Femoral artery aneurysm (pulsatile).
  - Psoas abscess (fluctuant and lateral to femoral artery).
- Clinical diagnosis in routine cases.
- Patients with unexplained small bowel obstruction should undergo careful examination for a femoral hernia.
- Ultrasound or CT should be performed if there is any uncertainty in diagnosis.

Treatment

- Should all be repaired because of significant risk of strangulation.
- Truss has no place in the management of femoral hernias.
- The aim of repair is to reduce the hernia and then narrow the femoral canal with the use of interrupted sutures or mesh to prevent recurrence. Care must be taken not to narrow the adjacent femoral vein which may lead to thrombosis.
- There are three main open approaches to repair.
- Although laparoscopic techniques can also be performed.

Low approach (Lockwood)

- Transverse incision is made directly over the hernia.
- Has the advantage of not interfering with the inguinal structures but provides little or no scope for resecting any compromised small bowel and so is best reserved for elective surgery.
- Can be performed under LA.

Inguinal approach (Lotheissen)

- Incision above the inguinal ligament, approaching the femoral canal from above by dissecting through the posterior wall of the inguinal canal (this can be quite disruptive).
- Requires repair of the inguinal canal on closure but offers excellent access to the peritoneal cavity, should small bowel surgery be required.

High approach ('open posterior')

- Ideal in emergency situations.
- Can be performed through a transverse (Henry) or vertical (McEvedy) incision.
- Allows access to peritoneal cavity and easy bowel inspection/resection.

Ventral hernias

Key facts

Hernias of the anterior abdominal wall. Umbilical and epigastric are the two commonest types.

Umbilical hernias

- Often small and occur through the umbilical cicatrix.
- Common in infants but often resolve spontaneously. The umbilical defect is present at birth but often closes within the first few weeks of life. Any delay can lead to presentation of hernia.
- Contents usually pre-peritoneal fat, and rarely bowel/omentum.
- May be painful but rarely strangulate.

Paraumbilical hernias

- Variable in size and occur through the periumbilical tissues.
- Common in obese and parous women.
- Contents can include bowel/omentum, and hernias will occasionally strangulate.

Epigastric hernias

- Defects in the linea alba at various sites between the xiphisternum and the umbilicus.
- They may be small and contain pre-peritoneal fat, or large and contain bowel/omentum, especially if neglected or long-standing.
- Moderate risk of strangulation.

Diagnosis

- Diagnosis is usually clinical. USS or, more commonly, CT, can be utilized in less obvious cases and to rule out other causes of swelling such as lipoma or subcutaneous lesions.

Treatment

- Congenital umbilical hernias only require surgical intervention when they persist beyond the age of 3y.
- All other ventral hernias can be repaired by either open, laparoscopic or robotic technique.
- Small defects are usually repaired by an overlapping sutured repair using non-absorbable suture.
- Larger defects or recurrent hernias may require mesh augmentation.

Incisional hernias

Key facts

- Occur at sites of previous abdominal wall surgery.
- Incidence varies greatly; 10–50% of midline laparotomy incisions can suffer herniation to some degree. Up to 5% of laparoscopic port-site incisions can also develop hernias.
- Predisposing factors include:
  - Patient factors—obesity, malnutrition, steroid administration, chronic cough.
  - Wound factors—infection, dehiscence.
  - Surgical factors—incorrect suture material, poor surgical technique.

Clinical features

- Present as swelling over site of previous incision.
- Wide variation in size. May encompass only a portion of previous incision or present as a diffuse swelling of entire length of incision.
- Most incisional hernias are broad-necked, with low risk of strangulation.
- Can present with signs and symptoms of intestinal bowel obstruction.

Treatment

- Asymptomatic incisional hernias or those in elderly/frail patients may not require any treatment.
- Open, laparoscopic or robotic repair can be performed.
- Small incision hernias (<4cm) can undergo simple sutured repair.
- Medium and large incision hernias (>4cm) often require insertion of a mesh or an 'abdominal wall component separation' to close defects.
- Laparoscopic repair is now commoner. The hernia is reduced before an intraperitoneal mesh is used to adequately cover (with overlap) the edges of the defect. The ideal route of repair remains debatable.

Other types of hernia

Spigelian

- Commonest in the elderly but can occur at any age.
- Arise through defect in aponeurosis of transversus abdominis muscle (Spigelian fascia).
- Typically presents with swelling at central edge of rectus sheath (linea semilunaris).
- CT is often required to confirm diagnosis.
- High risk of complications.
- Surgery can be either open or laparoscopic.

Lumbar

- 1° lumbar hernias are rare.
- Majority occur through the 'inferior lumbar triangle'.
- Usually contain retroperitoneal fat and rarely bowel.
- Mesh repair is often required as defect may be large.

Parastomal hernia

- Herniation around a colostomy or ileostomy.
- High occurrence following stoma formation (may be 50% or more at follow-up).
- Open suture and mesh repair techniques both available.
- Re-siting of stoma may be required in recurrent cases.

Obturator

- Commoner in women and the elderly.
- Herniation through the obturator canal.
- Many cases present with referred pain to knee (along obturator nerve).
- Hernias are often strangulated at time of presentation due to narrow neck.
- Repair is often via laparotomy for acute small bowel obstruction.

Perineal

- Very rare.
- Often a consequence of previous surgery and herniation through defects in pelvis floor.

Rectus sheath haematoma

Key facts

A consequence of haemorrhage within the rectus sheath that often arises from terminal branches of the superior and inferior epigastric arteries.

Clinical features

- Rare.
- Presents with acute pain and localized tenderness.
- A history of strenuous physical exercise or trauma may be elicited.
- In elderly patients, particularly those on anticoagulation, it may present as a mass in the lower abdominal wall.

Diagnosis and investigations

- Differential diagnoses include appendicitis, diverticulitis, and abdominal wall hernias.
- FBC and coagulation profile should be checked.
- Ultrasonography and, more commonly, CT scan can be used to confirm diagnosis.

Treatment

- Treatment is either conservative or surgical.
- Small haematomas and patients who are haemodynamically stable can be managed conservatively.
- Reversal of anticoagulation or blood transfusion may be required in more unstable patients.
- Surgical intervention involves evacuation of the haematoma and ligation of the contributing vessels.
- Arterial embolization can be utilized in large, expanding haematomas.

Groin 'disruption'

Key facts

- Also known as 'sports hernia' or 'Gilmore's groin' (although a true hernia is rarely present).
- Commonest in young ♂ athletes involved in sports that require sudden changes in movement/direction (e.g. rugby, football, tennis).
- An overuse syndrome that results in muscular imbalances of the pelvis and abdominal wall muscles.

Anatomical features

Features include a torn external oblique aponeurosis, a torn conjoined tendon, a dilated superficial inguinal ring, and a dehiscence between the inguinal ligament and the conjoined tendon.

Clinical features

- Unilateral groin pain that is often insidious and gradually worsens. Pain is felt 'deep' in the groin area.
- Some patients identify a distinct provocative event like kicking (forced flexion) whilst playing.
- Pain can prevent the patient from exercising but is almost always absent at rest.
- Usually no hernia is felt, but there may be tenderness in the region of the inguinal canal or external inguinal ring.

Diagnosis and investigations

- The differential diagnosis includes other causes of nearby injury such as adductor tendinopathy and pubic symphysis diastasis.
- Diagnosis is often clinical. MRI is essential in ruling out other causes of pain.

Treatment

- Patients should be initially managed conservatively, whilst other causes of pain are excluded and a firm diagnosis is made.
- Surgery should be considered as a **last** resort and only in cases of persistent pain.
- Mesh reinforcement may be utilized during repair.

Acute groin swelling

Causes of chronic testicular swelling are discussed under ➔ Scrotal swellings, p. 466.

Causes and features

- *Incarcerated groin hernia* (inguinal or femoral). May be associated with bowel obstruction; often red, hot, and tender.
- *Acute epididymo-orchitis* (in ♂). Tenderness is particularly over the spermatic cord and the epididymis.
- *Torsion of the testis*. May present with pain radiating into the groin; however, tenderness is primarily over the scrotum (and affected testis). Testicle is tender, swollen, and high-riding. Elevation of the scrotum, unlike epididymitis, makes the pain worse.
- *Iliopsoas abscess*. Tenderness is primarily below the inguinal ligament; swelling can be fluctuant, associated with RIF or left iliac fossa (LIF) tenderness.
- *Acute iliofemoral lymphadenopathy* (e.g. from infected toenail). Tender diffuse swelling; often multiple palpable lumps (nodes).
- *Acute saphena varix*. Compressible, cough thrill.
- Acute complications of *femoral artery aneurysm*.

Emergency management

Resuscitation

- Establish IV access; IV fluids will be required if a complicated hernia (e.g. bowel obstruction) is suspected. In such cases, catheterization and fluid balance monitoring will also be required.
- Send bloods for FBC, U&Es, and CRP.
- Group and save should be requested if surgical intervention is likely.

Establish a diagnosis

Torsion of the testis is a true surgical emergency and treatment should not be delayed.

Colour flow Doppler assessment may be able to confirm the presence of a hyperaemic testis but, unless immediately available, should not delay operative intervention.

If there is a strong clinical history in a young ♂, immediate operation remains the diagnostic investigation of choice.

Definitive management

Incarcerated groin hernia

- Repair is indicated for all patients, except those considered unfit for surgical intervention or those declining treatment.
- Femoral hernias may be approached by three different incisions (Femoral hernia, ➔ p. 438).
  - Infra-inguinal (Lockwood) incision is made directly over the hernia but has limited exposure.
  - A trans-inguinal approach gives access to the peritoneal cavity but can be disruptive to the inguinal canal.
  - High ('open posterior') approach is ideal in emergency situations and allows for more extensive bowel inspection/resection.

- Inguinal hernias should be approached through a conventional groin crease incision. Repair may require mesh insertion, although there is an ↑ risk of infection if bowel resection is performed.
- Surgery can be performed under LA or GA.

Torsion of the testis

- Once identified, the affected testis should be assessed; if non-viable, a simple orchidectomy is performed, and if viable, an orchidopexy.
- If the diagnosis is confirmed, the contralateral testicle should be fixed by orchidopexy to prevent subsequent torsion (➔ Torsion of the testicle, p. 488 for further details).

Epididymo-orchitis

- Antibiotics PO (e.g. ciprofloxacin 500mg od) for 14 days or IV if severe.

Psoas abscess

- The underlying cause should be identified as a matter of priority. Incision and drainage of the groin collection may be indicated, but more formal treatment of underlying aetiology is often required.

Chapter 11

Urology

Symptoms and signs in urology

Symptoms

Pain

- May be located over the site of pathology, e.g. testes.
- May radiate in accordance with innervation of the structure involved.
  - *Kidney pain*. In the renal angle (between the lower border of the twelfth rib and the spine).
  - *Ureteric pain*. Between the renal angle and the groin.
  - *Bladder pain*. In the suprapubic region.
  - *Prostatic pain*. In the perineum, but may radiate along the urethra to the tip of the penis.
- May be related to function, e.g. suprapubic pain exacerbated by bladder filling.

Haematuria (visible)

- Frequently a sinister symptom of malignant disease, especially the bladder or kidney when it is normally painless.
- When associated with painful voiding, it is usually due to bladder infection or stones.

Lower urinary tract symptoms (LUTS) see Fig. 11.1

- Refers to a group of symptoms that typically affect the ageing ♂.
- Often caused by bladder outflow obstruction (BOO) related to prostatic enlargement.
- Includes symptoms related to both voiding and storage.
- Voiding symptoms: poor urine flow, hesitancy, strainuria, post-micturition dribbling.
- Storage symptoms: frequency, nocturia, urgency, incontinence.
- More than two-thirds of men will have symptoms of voiding and storage.
- The International Prostate Symptom Score (IPSS) is a validated questionnaire to estimate the patient's perception of severity of symptoms.

Urinary incontinence

- Affects women more commonly than men.
- *Stress incontinence*. Involuntary urine leakage that occurs at times of ↑ abdominal pressure, e.g. during coughing, sneezing, lifting.
  - Results from incompetence of urethral sphincter and bladder neck mechanism; usually related to pregnancy and childbirth.
- *Urge incontinence*. Urine leakage that occurs in association with a strong desire to void.
  - Urine leaks from the bladder before the patient is able to reach the toilet.
  - Usual cause is overactivity of the detrusor muscle.
  - May be idiopathic or 2° to other bladder disease.
  - Stress and urge incontinence frequently coexist.

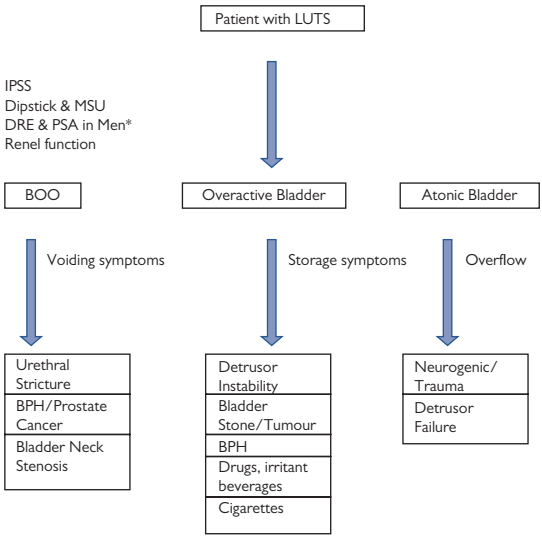

Fig. 11.1 Symptoms and signs in urology.

* If recurrent UTI, PSA elevated for age, digital rectal examination (DRE) abnormal, unexplained visible haematuria (non-visible haematuria, age >60), refer to a urologist.

- *Insensible urine leakage*. Occurs without any associated symptoms.
  - Urine leaks from the bladder continuously and the patient is sometimes unaware.
  - *Causes*. Overflow incontinence from chronic retention, fistulation (commonly between bladder and vagina), and gross sphincter disturbance resulting from surgery or neurological disease.

Male sexual dysfunction

- Erectile dysfunction (ED), commonly known as impotence.
  - Inability to attain and maintain an erection adequate for satisfactory sexual intercourse.
  - There are degrees of ED.
  - Men with incomplete ED respond more satisfactorily to treatment.
  - The majority of cases have an organic basis.
  - All cases have a degree of psychogenic involvement.
- Premature ejaculation.
  - Commoner in younger men.
  - Invariably has a psychogenic basis.
  - Often associated with performance anxiety.
- Loss of libido.
  - Loss of normal sex drive.
  - Either psychogenic or related to hypogonadal states.

Haematospermia

- Presence of blood in ejaculate.
- Single episode usually benign.
- Rarely associated with significant pathology, but investigate lower urinary tract appropriately, particularly if age >40.

Signs

Inspection

Examination of the penis must include retraction of the foreskin if uncircumcised, and inspection of the glans and external urethral meatus for signs of infection, inflammation, or tumour.

Palpation

- Tenderness in the renal angle or a palpable loin mass may indicate renal pathology.
- Suprapubic dullness to percussion is an indication of a bladder mass or full of urine.
- Check for testicular asymmetry, masses, or tenderness (underdevelopment, tumours, and infection).
- Intrascrotal mass may include the testis (e.g. hydrocele) or be separate from it (e.g. epididymal cyst).
- Do a DRE to determine the size, consistency, regularity, and symmetry of the prostate.

Investigations of urinary tract disease

Laboratory investigations

Urinalysis

- Dipstick analysis for blood, leucocytes, protein, nitrites, and glucose.
  - Nitrites, blood, and leucocytes—infection.
  - Blood—non-visible haematuria (NVH).
  - Protein and leucocytes—intrinsic renal disease.
- Microbiology, cytology for presence of urinary infection or malignant cells. MSU specimens are required for bacteriological culture; take care to avoid contamination, particularly in women.
- Matched urine and serum biochemistry to assess glomerular function, e.g. matched osmolarities, Na^+, and K^+.

Blood

- Serum Cr levels. Provides a crude assessment of overall renal function.
- Cr clearance (requires 24h urine collection and measurement of serum Cr).
  - Cr clearance (mL/min) = $u \times v/p$
  - where u is urine Cr concentration, v is 24h urine volume, and p is plasma Cr concentration.
- Serum PSA. Indicator of prostate disease.
  - Interpreted according to age-specific reference range.
  - High levels are found in BPH, prostate cancer, acute retention, and urinary infection.
- Sex hormone measurements. Occasionally useful in the assessment of ♂ sexual dysfunction and infertility.

Radiology investigations

Ultrasound

- Renal and bladder scans. For haematuria and UTI.
- TRUS.Measures prostate accurately, compared to transabdominal. Is used for systematic biopsy for detection of prostate cancer.
- Scrotal and testicular ultrasound. Evaluates acute scrotal and testicular pathology from cancer, inflammation, and abscesses—USS is not reliable to rule out testicular torsion.
- Intravenous urogram (IVU). Provides greater functional information than ultrasound. Provides superior imaging of the ureter.

CT

- Pre- and post-contrast scans provide some functional information with regard to arterial and venous blood flow and excretory function of the kidneys. Also, whether enhancing kidney tumours are enhancing or not.
- Vital for staging of renal, bladder, and testicular cancers.

MRI

- Provides greater accuracy than CT in assessment of the prostate and its capsule, and seminal vesicles.
- Sensitive test for the presence of bone metastases.

Isotope bone scan

- Demonstrates abnormal area of bone turnover.
- A useful screening test for the presence of bone metastases using ^{99m}Tc methylene diphosphonate.
- Plain films are taken to aid interpretation if the site or pattern of hot spots are indeterminate.

Isotope renography

- Provides anatomical and functional information about the kidneys.
- Dimercaptosuccinate (DMSA) scan provides an image of functioning renal parenchymal tissue.
- ^{99m}Tc-mercaptoacetyltriglycine (MAG3) renogram provides dynamic information regarding excretion from the kidneys and determines whether or not obstruction is present.

Endoscopy

Flexible cystoscopy

- Examines urethra and bladder.
- Performed using LA gel.
- Interventions can be undertaken such as biopsy, laser destruction of tumours, and injection of agents into the bladder such as botulinum toxin.

Rigid cystoscopy Under GA, permits deeper biopsy and the larger *resectoscope* allows resection of bladder or prostate tissue.

Ureteroscopy

- Rigid and flexible ureteroscopes provide access to the ureter and pelvicalyceal system.
- Allows the passage of biopsy forceps and laser fibres for stone and upper urinary tract tumour management.

Urinary tract stones

Key facts

- Prevalence of stones in the population is around 3% and is increasing year by year.
- The commonest reason for emergency urological admissions.
- Peak presentation in the summer months.
- Commonest age of presentation of urinary calculi is 20–50y.
- 90% of urinary calculi are radio-opaque.

Aetiology

- Dehydration with low urinary volume is the commonest cause. High animal protein intake.
- Metabolic. Hyperparathyroidism, idiopathic hypercalciuria, disseminated malignancy, sarcoidosis, hypervitaminosis D.
- Familial metabolic causes. Cystinuria, errors of purine metabolism, hyperoxaluria, hyperuricuria, xanthinuria.
- Infection.
- Impaired urinary drainage, e.g. medullary sponge kidney, pelviureteric junction (PUJ) obstruction, horseshoe kidney, ureteric stricture, extrinsic obstruction.

Pathological features

Calcium stones

- 80% of all urinary calculi.
- Usually combined with oxalate or phosphate.

Magnesium, ammonium, phosphate 'struvite' stones

- 15% of all calculi.
- Commonly occur against a background of 'urease'-producing bacteria and may grow rapidly.
- 'Staghorn' calculi (fill the calyceal system) are a sequelae.

Uric acid stones

- As a consequence of high levels of uric acid in the urine.
- 5% of all urinary stones; radiolucent.

Cystine stones

- Relatively rare; 1–2% of all cases.
- Difficult to treat due to extremely hard consistency; radiolucent.

Other stones

Xanthine, 'indinavir'- or 'triamterene'-associated, pyruvate, and other stones; 1% of all calculi.

Clinical features

- 'Ureteric colic'. Severe, intermittent, stabbing pain radiating from loin to groin.
- Non-visible or, rarely, visible haematuria (VH).
- Systemic symptoms such as nausea, vomiting, tachycardia, and pyrexia.
- Loin or renal angle tenderness due to infection or inflammation.
- Iliac fossa or tip of penis pain if the calculus has passed into the distal ureter.

Investigations

- Basic tests.
  - Raised WCC and CRP suggest superimposed infection (should be confirmed by MSU); raised Cr suggests renal impairment.
  - Stones often visible on plain AXR—'kidneys/ureters/bladder' (KUB).
  - Serum Ca^{2+}, PO_4, and uric acid.
  - 24h urine for Ca^{2+}, PO_4, oxalate, urate, cystine, and xanthine.
- Advanced tests.
  - Non-contrast spiral CT is the gold standard for locating stones and assessing evidence of complications.
  - IVU will locate stones and show any proximal obstruction.
  - USS for hydronephrosis, as cannot reliably assess whole ureter.

Treatment

Acute presentations (renal colic, ureteric obstruction)

- Analgesia, e.g. diclofenac 100mg PR; antiemetic, e.g. metoclopramide 10mg IV; IV fluids.
- Small stones (<0.5cm) may be managed expectantly, as most will pass spontaneously.
- Emergency treatment with percutaneous nephrostomy or ureteric stent insertion is necessary if uncontrolled pain, or severe renal impairment, or infection/sepsis is present.

Elective presentations

- ESWL.
  - Focused, externally generated electrohydraulic or ultrasonic shock waves.
  - Targeted onto the calculus using ultrasound, X-ray, or a combination.
  - Causes stone disintegration and the fragments are then voided.
  - CT's Hounsfield Unit density of the stone, and skin to stone distance must be considered.
  - Contraindicated in aortic aneurysms, pregnancy, distal ureteric obstruction, and uncontrolled HTN.
- Percutaneous nephrolithotomy (PCNL).
  - For large stones in the renal pelvis or calyces and occasionally for stones in the upper ureter.
  - Percutaneous track into the renal pelvis using fluoroscopic and/or ultrasound guidance.
  - Nephroscope is inserted and the calculus removed either in total or, if large, following fragmentation.
- Endoscopic treatment.
  - Ureteroscope is inserted, and the stone visualized.
  - Stone is fragmented using ultrasound, electrohydraulic intracorporeal lithotripsy, or laser.
- Open, laparoscopic, or robotic nephrolithotomy/ureterolithotomy. For large staghorn calculi or complex stones/anatomy, e.g. above ureteric stricture, horseshoe kidney.

Prevention of recurrence

- Increase oral fluid intake and moderate calcium, salt, and protein intake.
- Correct metabolic abnormalities.
- Treat infection promptly.
- Urinary alkalinization, e.g. sodium bicarbonate 5–10g/24h PO in water (mainly for cystine and urate stones).
- Thiazide diuretics (for idiopathic hypercalciuria).

Obstruction of the ureter

(See Fig. 11.2.)

Key facts Ureteric obstruction leads to hydronephrosis (ureteric and pelvicalyceal dilatation).

Pathological features Hydronephrosis can be unilateral or bilateral.

Unilateral

- Extramural.
  - Aberrant vessels at the PUJ.
  - Extrinsic tumour. Carcinoma of the cervix, prostate, or large bowel, or retroperitoneal endometriosis.
  - Idiopathic retroperitoneal fibrosis.
  - Post-radiation fibrosis.
  - Retrocaval ureter.
  - AAA.
- Intramural.
  - TCC of the renal pelvis or ureter.
  - Urinary calculi.
  - Ureteric stricture.
  - Aperistaltic segment. Almost always congenital.

Bilateral

- All causes of unilateral obstruction may cause bilateral hydronephrosis.
- Congenital posterior urethral valve.
- Congenital or acquired urethral stricture.
- Benign enlargement of the prostate.
- Locally advanced prostate cancer.
- Large bladder tumours.
- Gravid uterus.

Clinical features

- Loin pain.
- Fever and/or rigors (if complicated by infection).
- Symptoms and signs of renal failure (if obstruction long-standing).

Investigation and diagnosis

- Serum biochemistry and haematology.
- MSU.
- KUB X-ray/IVU.
- USS and/or CT scan.
- Isotope renogram.
- Retrograde pyelogram.

Complications

- Infection, pyonephrosis.
- HTN.
- Renal failure.

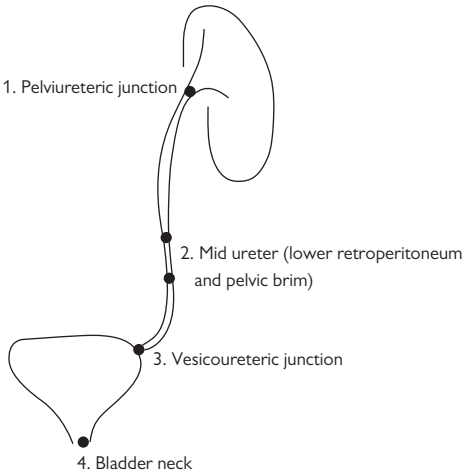

Fig. 11.2 Commonest sites of renal stone impaction. 1, pelviureteric junction; 2, pelvic brim; 3, vesicoureteric junction.

Treatment

Emergency presentation

- Emergency treatment is indicated if there are signs of infection, established renal failure, and uncontrolled symptoms.
- Treatment is drainage of the kidney via a percutaneous nephrostomy or retrograde ureteric stent.

Elective presentation

- Definitive treatment is directed at the underlying cause. Possible interventions include:
  - Treatments of calculi (see ➲ Urinary tract stones, pp. 456–8).
  - Long-term ureteric stenting (unilateral or bilateral).
  - Pyeloplasty for PUJ obstruction.
  - Ureterolysis and ureteric transfer (for retroperitoneal fibrosis).
  - Prostatic resection.
  - Bladder drainage using a urethral or suprapubic catheter.
- In cases where the kidney is deemed non-functional, a nephrectomy is performed to avoid infective complications.

Benign prostatic hyperplasia

Key facts

- BPH is a non-malignant enlargement of the prostate gland. There is an increase in both stromal and glandular components.
- Incidence of BPH is about 25% in age 40–60y, and 40% in over 60s.
- Commonest cause of LUTS in middle-aged and elderly men.

Pathological features

Aetiology is largely unknown. Possible factors include:

- Androgens.No BPH in those who have had early castration.
- Oestrogens.↑ oestrogen:testosterone ratio with age.
- Growth factors, e.g. high concentration of transforming growth factor α (TGF-α) in BPH.

Clinical features

Symptoms

(See ➲ Symptoms and signs in urology, p. 450.)

- Storage symptoms such as frequency, urgency, nocturia, and incontinence.
- Voiding symptoms, including hesitancy, poor stream, intermittency, terminal dribble, and abdominal straining.
- Superimposed infection may cause dysuria and haematuria.
- Incomplete emptying and chronic or acute retention of urine.

Signs

- Smooth enlargement of the prostate detected by DRE.
- Possible palpable bladder if chronic retention.
- Always examine for neurological signs in those with LUTS.

Complications of BPH

- Intractable LUTS.
- Haematuria.
- UTI.
- Stone formation.
- Acute retention of urine.
- Chronic retention of urine with overflow incontinence.
- Obstructive renal failure.

Diagnosis and investigations

- Prostate symptom score to assess severity: IPSS.
- DRE and serum PSA measurement to assess anal tone and prostate size for features of malignancy.

Basic investigations

- Serum Cr and urinalysis in all patients.
- Urine flowmetry and residual volume estimation in those considered for intervention.

Advanced investigations

- Cystoscopy. To exclude urethral stricture or bladder pathology.
- TRUS ± guided biopsy. If concern over underlying malignancy.
- Renal ultrasound.
- Urodynamic studies.

Treatment

Recommended for those with LUTS that are impacting on quality of life or those with above complications.

Medical treatment

- Patients with mild symptoms and no complications may be observed (watchful waiting).
- α-adrenergic antagonists. Relax smooth muscle of prostatic urethra and bladder neck to decrease outlet resistance; side effects include dizziness, postural hypotension, and retrograde ejaculation.
- 5α-reductase inhibitors. Block conversion of testosterone to dihydrotestosterone (DHT) and shown to cause involution of BPH; side effects include loss of libido and ED.
- Combination drug therapy with both above agents has been proven to reduce clinical progression and decrease the need for surgery.

Surgical treatment

- Reserved for those with any of the complications or symptoms not responding to medical therapy.
- Intermittent self-catheterization (requires dexterity of the patient or carer).
- Long-term indwelling urethral or suprapubic catheterization.
- Surgical options include:
  - Transurethral resection of the prostate (TURP), the most commonly performed procedure for BPH in the UK.
  - Bladder neck incision.
  - Laser enucleation or vaporization of the prostate.
  - Microwave thermotherapy ablation of the prostate.
  - Open transvesical or simple prostatectomy.
  - 'Urolift' endoscopic-placed anchoring implants to hold the occlusive lateral lobes apart.
  - Prostate artery embolization (interventional radiology).

Key revision points—anatomy of the prostate

- Three glandular zones:
  - Peripheral (70%), most prone to carcinoma formation.
  - Central (25%), most prone to BPH.
  - Transitional (5%), most prone to BPH.
- Blood supply:
  - Arterial supply is triple, mainly inferior vesical, some from inferior rectal and internal pudendal.
  - Venous drainage—extensive plexus beneath capsule.
- Innervation:
  - Autonomic—extensive from inferior hypogastric plexus as a capsular plexus supplying prostate, seminal vesicles, and urethra, which also supplies the penile structures (glands, corpora, and urethra).
  - Somatic—from pudendal nerve (S2, 3, 4) to supply external urethral sphincter.

Stricture of the urethra

Key facts

- Classified according to site and aetiology, e.g. post-inflammatory bulbar stricture or traumatic membranous stricture.
- Graded according to length and (in the anterior urethra) degree of fibrosis of corpus spongiosum (spongiofibrosis).
- Any part of the urethra may be involved.
- The propensity for stricture recurrence parallels the severity (grade).

Aetiology

- Trauma.
  - Pelvic fracture.
  - Falls astride, e.g. on bicycle crossbar.
  - Urinary tract instrumentation/surgery.
- Infection.
  - *Neisseria gonorrhoeae* and *Chlamydia trachomatis*.
  - Catheter-associated UTI.
- Lichen sclerosis et atrophicus.

Pathological features

- Annular narrowing by scar tissue composed of dense collagen and fibroblasts, which may extend into corpus spongiosum.
- Lichen sclerosus (balanitis xerotica obliterans (BXO)) consists of dermal sclerosis with epidermal atrophy; urethral lesions are usually confined to the meatus and fossa navicularis.

Clinical features

- History of urethritis, trauma, or urinary tract instrumentation.
- LUTS, divergent or diminished stream, straining to void, urgency, frequency.
- Haematuria ('initial' or 'terminal', i.e. at the beginning or end of the stream).
- UTI (often recurrent).
- Urinary retention, acute or chronic.
- Overflow incontinence.

Complications

- Urinary tract calculi formation.
- Infection, including UTI, prostatitis, epididymitis, and (rarely) Fournier's necrotizing fasciitis.
- Renal failure 2° to chronic obstruction.

Diagnosis and investigations

- Urinalysis (microbiology, biochemistry, and cytology).
- U&Es.
- Uroflowmetry and measurement of post-void residual bladder volume. Uroflowmetry shows a 'box' pattern and flat curve.
- Voiding cystourethrogram with or without retrograde urethrogram.
- Endoscopy.
- USS (transperineal or endoluminal) to assess extent of spongiofibrosis.
- Magnetic resonance tomography.

Treatment

Initial

- Treat infection before surgical treatment.
- For acute urinary retention (AUR), severe symptoms, or renal failure—temporary SPC.

Definitive

- All urethral reconstructive surgery has an 'attrition rate'.
- Initial internal urethrotomy may cure up to 30%, but repeat procedures are rarely successful and may cause progression.
- Urethroplasty may be used for some cases.
  - Short strictures are excised and the urethra primarily re-anastomosed.
  - For longer or complex strictures, pedicled flap reconstruction or graft reconstruction is used; the graft or flap may be applied as an onlay (to augment the native urethra) or as a tube.
  - For free grafts, buccal mucosa is currently favoured.
  - More complicated repairs are best managed with staged repairs.
- Perineal urethrostomy may be required.

Scrotal swellings

Major causes

Intratesticular lesions

- Malignant testicular tumours.
- Benign intratesticular lesions. Simple intratesticular cyst or epidermoid cysts and benign teratoma (especially in the prepubertal testis).

Inflammatory/Infectious lesions

- Acute epididymo-orchitis or viral orchitis.
- Chronic tuberculous epididymo-orchitis, schistosomal epididymitis, sperm granuloma.
- Be vigilant of patients with poor hygiene, immunocompromised, with or without catheters, who present with scrotal cellulitis; this could be Fournier's gangrene which is a urological emergency.

Traumatic lesions

- Scrotal haematoma.
- Haematocele (haematoma within tunica vaginalis).
- Testicular haematoma (within tunica albuginea testis).

Derangement of testicular, adnexal, or cord anatomy

- Epididymal cysts or spermatocele of the epididymis.
- Varicocele (varicosities of the pampiniform plexus).
- Inguinal hernia (patent processus vaginalis in children).
- Hydrocele (communicating, non-communicating, or of the cord).
- Late presentation/missed to torsion of the spermatic cord.
- Persistence of embryological vestigial structures.
  - Müllerian duct remnant (appendix testis).
  - Wolffian duct remnants (appendix epididymis, vas aberrans of Haller, paradidymis).

Miscellaneous

- Acute idiopathic scrotal oedema.
- Cutaneous lesions, e.g. sebaceous cysts.
- Henoch–Schönlein purpura.

Clinical features/diagnosis

- Testicular tumours:
  - Firm, intratesticular, or progressively enlarging lesions are tumours until proven otherwise.
  - Tumours may present with acute pain from a bleed.
- Tuberculous epididymitis may occur with or without evidence of pulmonary, systemic, or other GU involvement.
- Non-communicating hydroceles, large spermatoceles, and epididymal cysts are transilluminable, may be fluctuant, and are usually confined to the scrotum.
- Communicating hydroceles or patent processus vaginalis are as above, but swelling can be seen in the groin.
- Varicocele. Often associated with subfertility or dragging discomfort that worsens on standing and settles when recumbent. More obvious as a fluctuant swelling with the patient standing. A cough impulse may be felt. Ipsilateral testis may be atrophied. Examine the abdomen to exclude an associated renal tumour.

- Inguinal hernias tend to be intermittent, associated with groin discomfort, and when 'out', the examining hand cannot 'get above' the swelling in the cord/inguinal canal. A history of long-standing enlargement is typical.

Investigations

- Urinalysis for M, C, & S.
- Blood tests. Consider tumour markers (AFP, β-HCG) and inflammatory markers (CRP, WCC).
- USS of the scrotal contents has several important uses.
  - Distinguishes intratesticular from paratesticular swellings, solid from cystic lesions, cellulitis from abscess, etc.
  - Can examine impalpable testes, e.g. within large hydroceles.
  - Can identify rupture of the tunica albuginea testis.
  - Examination of the abdomen can identify renal mass associated with varicocele or ascites with hydrocele/scrotal oedema, etc.
  - Colour flow Doppler ultrasound can identify hyperaemia, varicocele, and underperfusion (not reliable for acute testicular torsion).

Treatment

- Testicular tumours, acute epididymo-orchitis (see ➔ Testicular tumours, pp. 482–3; ➔ Acute testicular pain, pp. 488–9).
- Suspected paratesticular tumours are approached surgically in the same way as testis tumours. Surgery may be conservative with benign testicular and paratesticular tumours.
- Sperm granuloma may be excised or epididymectomy may be performed. Reassurance may be all that is required in many cases. Recurrence rates after surgery are high.
- Epididymal cysts/spermatocele may be aspirated, but recurrence is common. Excision risks loss of epididymal patency, is associated with risk of recurrence, and should probably be discouraged in men who have not completed their family.
- Hydroceles. Treat if symptomatic. Procedures usually reconfigure the serosal remnant of tunica vaginalis, so as to allow lymphatic drainage via scrotal lymphatics. Reduction, inversion (Jaboulay), or imbrication (Lord's) of the tunica vaginalis is used.
- Embryological remnants. No treatment if asymptomatic.
- Varicocele. Treat if symptomatic, associated with infertility or with failure of testicular growth. Venous embolization and retroperitoneal ligation (laparoscopic or open) of the testicular vein have similar results. Minimally invasive treatments are preferable. Gubernacular veins and other collaterals may account for failures. Open surgical ligations via an inguinal incision can deal with these.
- Acute idiopathic penoscrotal oedema of childhood usually settles with conservative treatment. Antihistamines and antibiotics are frequently prescribed, but there is little evidence to support this.

Disorders of the foreskin

Phimosis

(See Fig. 11.3.)

Key facts

- Narrowness of the preputial opening, preventing retraction and exposure of the glans.
- May be physiological in infants and young children and will resolve.
- Pathological phimosis is 2° to scarring such as BXO or following monilial balanoposthitis.
- Pathological phimosis may be associated with discomfort, UTI, and balanoposthitis.

Clinical features

- In childhood, physiological phimosis may be identified by absence of scarring of the preputial tip and by pouting of the inner layer of the prepuce when a gentle attempt is made to retract it.
- With physiological phimosis, gentle protraction of the foreskin, the glans is usually visible. Ballooning of the foreskin; this a natural process to break down glanular adhesions.
- Whitish, sclerotic preputial skin with no pouting characterize BXO.
- Parents may complain of recurrent balanoposthitis.

Treatment

- Physiological phimosis needs no treatment, only reassurance.
- Early BXO may be treated by application of topical steroid cream but will eventually require surgery.
- Surgical treatment of phimosis includes dorsal slit, preputioplasty, or circumcision.

Paraphimosis

(See Fig. 11.3b.)

Key facts/diagnosis

- The retracted foreskin acts as a constricting ring, reducing lymphatic and venous drainage of tissues distal to the ring (glans, inner layer of foreskin). Subsequent oedema makes reduction more difficult.
- Involved tissues typically appear oedematous and inflamed, with progression to infection, ulceration, and necrosis if left untreated.

►► *Treatment*

- Early cases. Gentle manual compression of the oedematous tissues within a saline-soaked swab will allow reduction of the foreskin after a few minutes without anaesthesia.
- Established cases. LA penile block or anaesthesia may be necessary. Hyaluronidase injection under the constriction has been described.
- Neglected/severe cases. A relaxing dorsal incision may be made through the constriction and the tissues immediately proximal and distal to it. Subsequent circumcision is often offered.

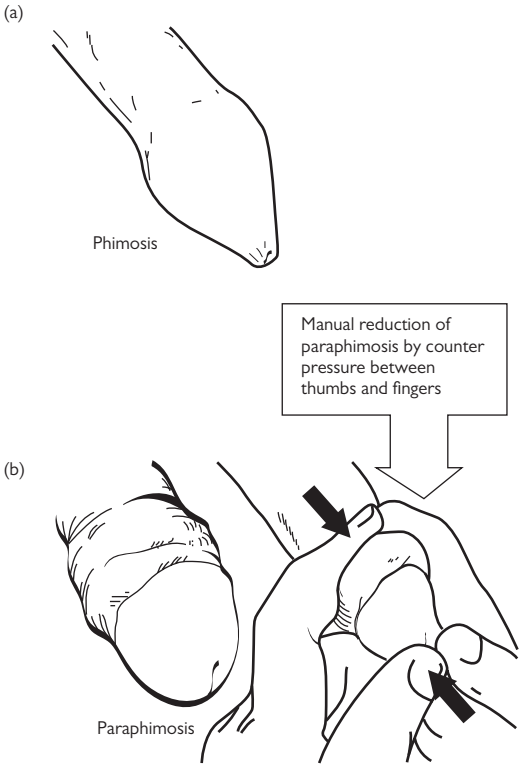

Fig. 11.3 (a) Phimosis. (b) Reduction of an acute paraphimosis.

Common conditions of the penis

Peyronie's disease

Key facts

- Damage to tunica albuginea penis, forming inelastic penile plaques mostly in the dorsal midline, causing local pain and deformity.
- Associated with Dupuytren's contractures, plantar fascial contractures, and tympanosclerosis.
- One-third of patients have ED.

Clinical features

History and examination

- Gradual or sudden development of palpable penile plaques, with painful distortion of the erect penis during the acute phase.
- Occasional history of penile microtrauma.
- ED may involve the whole or part of the penis.
- Pain resolves in chronic phase, but deformity does not.
- Penetrative sexual intercourse may be more difficult or impossible because of pain, angulation, or buckling.
- The plaque is palpable with the flaccid penis stretched.
- Erection is induced pharmacologically and a photo record kept.

Investigations

- Grey-scale ultrasound may demonstrate calcification, an indicator of plaque maturity. Doppler used if concurrent ED to offer best surgical options.
- MRI scan can be used for severe forms ± with a 'waist' deformity.

Treatment

Medical

Indicated for those able to engage in intercourse and whose disease is still in the acute phase.

- Oral treatment options. Colchicine, vitamin E, or Potaba®.
- Intralesional injections. Verapamil or collagenase (investigational).
- Treatment of associated ED.

Surgical

Indicated for those with stable lesions in the chronic phase, severe deformity. Curvature angle (> or <60°) and ED predict techniques.

- Plication techniques. Plication of the tunica opposite the deforming plaque.
- Grafting techniques. The plaque is incised/excised and the defect grafted.
- Prosthesis insertion within corpora cavernosa. Generally reserved for patients with severe ED refractory to medical therapy.

Priapism

Key facts

Abnormally sustained erection unrelated to sexual stimulation. It is classified as high flow (arterial) or low flow (veno-occlusive).

Low-flow priapism

- Congestion secondarily reduces arterial blood flow, leading to local hypoxia, acidosis, and hypercapnia.
- Causes include:
  - Drugs (intracavernosal prostaglandin and papaverine, psychotropics, e.g. trazodone, chlorpromazine).
  - Abnormal blood viscosity (sickle cell, myeloma, leukaemia, thalassaemia, TPN).
  - Neurological disease, e.g. spinal or cerebrovascular disease.
  - Miscellaneous, e.g. infiltration by solid tumour.
- Characterized by painful, persistent erection, not involving the glans and corpus spongiosum.

High-flow priapism

- Most commonly follows blunt trauma to penis or perineum, but has been caused by intracavernosal injection or revascularization.
- The mechanism is arterio-cavernosal fistula with unregulated arterial inflow and ↑ venous outflow.
- Characterized by partial, painless swelling of the glans and corpus spongiosum.

Investigations

- Colour Doppler ultrasonography or cavernosal blood gases.
- pH <7.25, pO_2 <30mmHg, and pCO_2 >60mmHg suggest low flow.
- FBC and differentials; Hb electrophoresis.

Treatment—low flow

- Correct underlying abnormalities, e.g. sickle cell (rehydration, O_2, transfusion), myeloma (plasmapheresis).
- Corpora cavernosa aspiration (up to 50mL) + manual pressure.
- Intracavernosal phenylephrine (patient must be on a cardiac monitor).
- PO β-agonists, e.g. terbutaline 5mg + further 5mg 15min later.
- Surgical techniques, such as distal and proximal shunts, augment venous drainage of the corpora cavernosa.

Treatment—high flow

- Selective pudendal arteriography and embolization.

Erectile dysfunction

Key facts

- ED is the inability to achieve or maintain an erection satisfactory for sexual intercourse.
- Distinct from premature, retrograde, or delayed ejaculation.
- Causes include:
  - *Psychogenic.* Anxiety, depression.
  - *Drugs.* Antihypertensives, recreational drugs, tobacco, alcohol.
  - *Vascular.* Hypercholesterolaemia, atheroma, DM.
  - *Metabolic/endrocrine.* Azotaemia, hypercholesterolaemia, hypogonadism, hyperthyroidism, hyperprolactinaemia, DM.
  - *Neurological.* Parkinson's disease, CVA, spinal injury, neurological damage following pelvic surgery, pelvic fracture, autonomic neuropathies.
  - *Penile.* Cavernositis, Peyronie's disease, previous priapism.

Clinical features

- Specific validated questionnaires have been developed as investigational tools and can be used in practice.
- The presence of morning erections strongly suggests a psychogenic cause.
- Small testicles and lack of 2° sexual characteristics suggest an endocrine cause.
- Lack of lower limb pulses suggests a possible vascular cause.
- Neurological deficits in S2, 3, 4 distributions suggest a neurological cause.

Investigations and diagnosis

- Check blood glucose, lipids and serum electrolytes, hormone profiles (testosterone, follicle-stimulating hormone (FSH)/luteinizing hormone (LH), prolactin), and thyroid function for underlying cause.
- Dynamic cavernosometry confirms if there is a true vasculogenic cause (venous or arterial).
- Angiography demonstrates arterial anatomy if revascularization is contemplated.

Treatment

- Psychotherapy or specialist sexual counselling for psychogenic causes.
- PO phosphodiesterase-5 inhibitors—sildenafil, vardenafil, tadalafil.
- Apomorphine SL.
- Intracavernosal—prostaglandins, α-blockers, papaverine.
- Intraurethral—prostaglandin.
- Vacuum device-induced pseudoerection.
- Penile implant: malleable rods, inflatable penile prosthesis.

Adenocarcinoma of the kidney

Key facts

- Accounts for 2% of all cancers.
- Incidence is 2–5 per 100 population.

Clinical features

- May be asymptomatic at presentation, the tumour being detected during imaging of the abdomen for an unrelated condition (e.g. CT scan or USS).
- Symptoms include painless haematuria, groin pain, and awareness of a mass arising from the flank.
- Chest symptoms and bone pain may be present with metastases to these sites.
- Positive family history or clinical evidence of neurological or ocular disease should raise the possibility of VHL.
- Renal carcinomas are often small and may be multiple.
- Local spread often includes spread via intravascular invasion into the renal vein and IVC.

Diagnosis and investigations

- Blood tests. Hb and ferritin to check for anaemia (iron-deficient); electrolytes and Cr to check for overall renal function. Raised corrected Ca^{2+} and ALP suggest possible bony metastases.
- Diagnostic and staging investigation of choice is a pre- and post-IV contrast-enhanced CT scan of abdomen and chest (delineates size, local extent, local invasion, likely sites of possible metastases).
- Isotope bone scan if there is clinical or biochemical evidence of bony metastases.

Treatment

Surgery

- Recommended as the only curative treatment, except in the very elderly, extensive (inoperable) local invasion, and the presence of metastases.
- May be via open, laparoscopic, or robotic approach.
- Radical nephrectomy is recommended for large tumours.
- Partial nephrectomy may be suitable for peripheral tumours <7cm in size.
- Resection of the 1° cancer is occasionally appropriate with the presence of metastasis (the deposit must be low volume, solitary, and itself amenable to complete local resection, e.g. in the liver or lungs).
- RFA or cryotherapy may be appropriate in elderly and/or comorbid patients with small renal tumours.

Medical therapy

- Used for metastatic disease.
- Biological therapy can be with immune modulators such as IFNs and ILs. Partial response rates of 15–20% can be achieved, but the treatment carries significant morbidity. This is reserved for patients with a good performance status.

- Targeted therapy, such as tyrosine kinase inhibitors and mammalian target of rapamycin, have now become the first-line therapy for metastatic renal cell carcinoma over immune modulators.
- Chemotherapy is rarely used as the tumours are not chemosensitive.
- Hormonal therapy (androgens and tamoxifen) may have some benefit.
- Radiotherapy is useful to palliate painful bony metastases.

Prognosis

- The outcome following nephrectomy is unpredictable.
- Tumours that are pathologically confined to the kidney confer a good prognosis. Adverse risk factors include extracapsular spread, invasion of the renal vein, and lymph node involvement.
- Cure is likely if the tumour is <4cm in diameter and if there are no adverse pathological features.
- Periodic radiological follow-up is recommended in most cases, so that locally recurrent or metastatic disease can be detected at an early stage.

Key revision points—anatomy of the kidney

Usually five segments (apical, anterior superior, anterior inferior, posterior, and inferior), each supplied by its own artery.

- Fascial coverings.
  - Perirenal, anterior and posterior layers, enclosing kidney, and perirenal fat.
  - Lateral conal fascia, formed from anterior and posterior perirenal fascia; fused with transversalis and iliac fascia laterally.
- Blood supply.
  - Arterial supply, direct from the aorta; renal arteries also give supply to the renal pelvis and upper ureter and adrenal gland.
  - Venous drainage, direct to IVC; left renal vein also drains the left gonadal vein.
- Common anatomical variants.
  - Unilaterally absent kidney (1 in 1200).
  - Pelvic kidney (1 in 2500).
  - Joined (horseshoe) kidney (1 in 400).

Transitional cell tumours

Key facts

- TCC may affect any part of the urinary epithelium (renal pelvis, ureter, bladder, or, very rarely, urethra).
- TCC have a spectrum of disease, from superficial papillary growth to solid sessile invasive tumour.
- TCC of the bladder. Fifth commonest cause of cancer deaths and the commonest form of bladder cancer in the UK.
- TCC of the upper urinary tract is similar in spectrum of disease and management to bladder tumours, but much less common.
- TCC is associated with:
  - Exposure to aromatic hydrocarbons, e.g. workers in the petrochemical, industrial dye, and rubber industries, chimney sweeps.
  - Smoking (especially in women).
- Risk is probably due to excretion of carcinogenic products excreted and concentrated in the urine and tumours are more likely in locations exposed to urine for the longest periods, i.e. bladder.

Pathological features

- The majority (70%) are superficial in nature at diagnosis, being confined to the mucosa.
- Invasion into the lamina propria, muscle, and perivesical fat can occur with lymphatic and distant spread occurring in advanced cases.
- Carcinoma in situ of the bladder whilst pre-invasive and can progress to high-risk muscle-invasive disease if not adequately treated.
- TCC must be differentiated from other forms of bladder cancer.
  - *SCC.* Usually caused by chronic irritation due to schistosomiasis infestation (bilharzia), indwelling catheter, and repeated previous surgical interventions.
  - *Adenocarcinoma.* Rare; presents in middle age and is usually located in the dome of the bladder in association with the urachus.

Clinical features

- The majority of cases present with painless haematuria.
- Other features are painful micturition, renal colic due to blood clot, disturbance of urinary stream, and retention of urine.

Diagnosis and investigation

Urine cytology May reveal malignant cells; if positive, is associated with high-grade TCC or carcinoma in situ will probably be present.

Cystoscopy

- Usually carried out using a fibreoptic flexible cystoscope and LA gel.
- Images the bladder and urethra; suspect lesions usually require transurethral resection under GA for diagnosis.

Transurethral resection
- Carried out using a rigid resectoscope under GA.
- Permits resection of all or part of the tumour using a diathermy 'loop', with the tumour resected in fragments or en bloc; resection should be carried out into deep tissue (the muscle wall of the bladder beneath the tumour).
- Subsequent pathological examination will determine the histological grade and the pathological stage, e.g. depth of invasion.
- Bimanual examination pre- and post-transurethral resection of bladder tumour (TURBT) determines whether or not the mass is mobile.

Upper tract imaging
- USS permits examination of the renal cortex and will detect tumours of 1cm in diameter in the pelvicalyceal system, ureter, and bladder.
- CT urogram used to identify and assess pelviureteric tumours.
- Bladder tumour may show as a filling defect in the cystogram phase.

Local staging MRI and CT scanning to detect local or systemic spread.

Treatment

Superficial TCC
- Remove completely by endoscopic resection.
- Recurrence is common and regular endoscopic surveillance with check cystoscopy is mandatory.
- Intravesical chemotherapy reduces the risk of tumour recurrence (single dose of mitomycin instilled after resection of the tumour).
- For multiple or recurrent TCC, six intravesical treatments are given.

Carcinoma in situ
- Requires thorough therapy to prevent invasive TCC.
- Immunotherapy with intravesical bacille Calmette–Guérin (BCG) is effective in 60% of cases.
- Needs close endoscopic surveillance with regular bladder biopsy.

Invasive TCC
- Muscle-invasive tumours are high risk and cannot be managed endoscopically only.
- Curative therapy can be offered with radical cystectomy (combined with a continent or incontinent urinary diversion) or radical radiotherapy.

Squamous cell carcinoma and adenocarcinoma
- Radical cystectomy, provided general condition allows.
- Usually resistant to radiotherapy and chemotherapy.

Prognosis
- ~30% develop muscle-invasive disease.
- The 5y survival rate for muscle-invasive bladder cancer is 40–50%.
- Metastatic TCC has poor prognosis, with a median survival of 13 months.
- Systemic chemotherapy with cis-platinum-containing regimes provides a long-term response in 15% of cases.
- Neoadjuvant chemotherapy offers a survival advantage of 25%.

Adenocarcinoma of the prostate

Key facts

- Most commonly diagnosed cancer affecting men in the Western world.
- ~30 000 new cases diagnosed annually in the UK, with 10 000 deaths.
- Peak incidence in eighth decade.
- ~40% of cases present with early disease; 20% of cases have metastases at presentation.

Clinical features

- The majority of men present with LUTS (see ➲ Symptoms and signs in urology, pp. 450–2).
- Bone pain, pathological factures, and features of hypercalcaemia are occasional presenting features due to metastases.
- May be diagnosed by DRE; areas of firmness or palpable nodules are suggestive of malignant change.

Diagnosis and investigations

- *Serum PSA*. Can be used as a screening test; high sensitivity, but low specificity; elevated age-specific levels are an indication to consider prostate biopsy.
- *TRUS*. Permits detailed imaging of the prostate. Systematic needle biopsy is performed, guided by ultrasound images, with antibiotic prophylaxis; graded using the Gleason grading system which assigns a numerical score to adverse features, from a minimum of 2 to a maximum of 10.
- *Pelvic MRI*. More informative when done pre-biopsy. Used to detect the presence of extracapsular extension or the presence of pelvic lymphadenopathy (suggests spread).
- *Laparoscopic node biopsy*. May be performed to sample enlarged nodes prior to considering radical treatment.
- *Isotope bone scan*. Will detect the presence of bone metastases.

Treatment

Localized disease (confined to prostate)

Patients with a life expectancy of <10y

- Active monitoring, with treatment deferred until there is evidence of disease progression (rising serum PSA).
- Hormonal therapy or α-blocker treatment offered for troublesome LUTS.
- TURP is considered for severe symptoms with features of obstruction.

Patients with a life expectancy of >10y

- Counselled in detail about radical treatment aimed at cure.
- The options are as follows:
  - Radical prostatectomy. Operation to remove the prostate and seminal vesicles; complications include incontinence (severe in 3%) and ED (<30% if bilateral nerve spare performed).
  - External beam radiotherapy. Radiation is delivered at a radical dose of 55–70Gy in 20–25 fractions over a 4- to 5-week period; complications include cystitis, proctitis, and ED.
- Brachytherapy. Radioactive seeds placed into the prostate using TRUS guidance.

Locally advanced disease (spread beyond the prostate)

- Incurable and treatment is therefore palliative.
- 80% are androgen-dependent. Hormone therapy reduces androgenic drive to the prostate cancer cell using two methods:
  - Luteinizing hormone-releasing hormone (LHRH) agonists. Given by 3-monthly depot injections, suppresses testosterone production by the testes; side effects include hot flushes, lethargy, and loss of sexual function. Must be started with temporary cover of anti-androgens, as LHRH agonists causes an initial testosterone surge.
  - *LHRH antagonists* act as above but do not require initial co-administration with anti-androgens.
  - Anti-androgens. Given PO, act as competitive inhibitors at the level of the androgen receptor, reduce androgenic stimulus to the prostate cancer cell without reducing serum testosterone levels; side effects include gynaecomastia and nipple tenderness (60%); potency is sometimes preserved.

Metastatic disease

- Treated with hormonal therapy using LHRH analogues. Addition of an anti-androgen provides a 2° response in some cases of PSA relapse. Pain from bone metastases usually responds to radiotherapy.

Hormone-resistant disease

- All prostate cancers will eventually become hormone-resistant.
- Chemotherapy is appropriate for patients who have a good performance status.
- Palliative radiotherapy and bisphosphonates are used for bone metastases.

Prognosis

- Localized prostate cancer. Excellent prognosis, with 70–90% 10y disease-specific survival figures.
- Locally advanced, non-metastatic disease. Median survival of 7y.
- Metastatic disease. Median survival of 2–3y.
- Once the state of hormone-resistant disease has been reached, the median survival is 6–12 months.

Carcinoma of the penis

Key facts

- Rarest of the urological cancers.
- Occurs primarily in older, uncircumcised men. Usually associated with poor socio-economic status and immunocompromised state.

Clinicopathological features

- Over 95% are SCC.
- Usually affects the glans but may involve the shaft.
- Associated with chronic infection of the penis, particularly in the presence of phimosis.
- Early cases present with a painless ulcer, nodule, or 'warty' outgrowth on the penis that may also involve the foreskin.
- Advanced disease presents with a fungating mass, usually ulcerated. Inguinal lymphadenopathy may be present on examination. Nodes are often reactive, rather than being metastatic, and antibiotics should be given prior to further assessment.

Diagnosis and investigations

- Biopsy lesion to confirm the diagnosis.
- Pelvic and abdominal CT scanning provides further evidence of nodular involvement in cases with positive inguinal nodes.

Treatment

- If 1° tumour confined to glans, treatment involves either partial amputation or radiotherapy.
- Superficial lesions can be treated by excision of the glans followed by glans reconstruction.
- More advanced carcinomas require total penectomy.
- Inguinal and iliac lymph node dissections are considered.
- Non-palpable inguinal nodes should undergo dynamic sentinel lymph node biopsy, depending on tumour stage.

Prognosis

- Early-stage penile cancer has a high cure rate with either surgery or radiotherapy.
- Long-term survival is sometimes seen, even in cases of lymph node involvement.

Testicular tumours

Key facts

- Commonest malignancy in men between the ages of 18 and 40.
- Annual incidence 6 per 100 000 ♂ per year.
- Associated with testicular maldescent.
- ↑ risk is associated with higher levels of exogenous oestrogens, either prenatally or in childhood.
- ↑ level of awareness has led to more tumours being detected on self-examination, particularly among younger men.

Pathological features

- Common types are seminoma and non-seminomatous germ cell tumours (NSGCTs). Sex cord stromal tumours and lymphomas are rare testicular tumours.
- Seminoma.
  - Peak incidence 30–40y.
  - Lymphatic spread commoner than haematogenous.
  - Lymphatic spread to iliac and para-aortic nodes.
- NSGCTs.
  - Peak incidence 20–30y.
  - Haematogenous spread commonest to lungs, brain, and liver.
- Lymphomas. Peak incidence over 60y.
- Marsden staging (after investigations and treatment).
  - Stage 1, confined to testis.
  - Stage 2, abdominal nodal spread.
  - Stage 3, nodal disease outside the abdomen.
  - Stage 4, extralymphatic spread.

Clinical features

- The usual presentation is with a painless testicular mass.
- Typical features are irregular, firm, and fixed, and does not transilluminate.
- Palpate the abdomen for intra-abdominal masses (either para-aortic node masses or hepatomegaly).
- Check for supraclavicular lymphadenopathy and signs of lung or neurological disease.

Diagnosis and investigation

Any clinically suspicious mass requires urgent testicular USS. Typical features are a non-homogeneous mass with ↑ vascularity.

- Serum tumour markers, β-HCG, and AFP. ↑ levels suggest metastatic disease in NSGCTs, but may be normal in localized or metastatic disease; very rarely elevated in seminoma, even if metastatic.
- CT scan of abdomen and chest. To assess for presence of metastases.
- CT brain, bone scan. Only if clinically indicated.

Treatment

- Orchidectomy is carried out at the earliest opportunity; this is performed via an inguinal approach, so that the spermatic cord can be clamped prior to mobilization of the testis.
- Seminoma is radiosensitive and even widespread local disease responds well to radiotherapy.
  - Stage 1. May be treated by orchidectomy only, orchidectomy + prophylactic iliac and para-aortic radiotherapy, or orchidectomy + prophylactic chemotherapy.
  - Stages 2/3/4. Orchidectomy + radiotherapy to involved node or chemotherapy.
  - Visceral metastases are treated with a combination of chemotherapy and radiotherapy.
- NSGCTs are chemosensitive and even widespread metastatic disease responds well.
  - Stage 1. Orchidectomy.
  - Stages 2/3/4. Orchidectomy + chemotherapy; if lymphadenopathy is still present following chemotherapy, a retroperitoneal lymph node dissection is performed.
- Retroperitoneal lymph node dissection commonly performed for residual masses post-chemotherapy for NSGCTs in the UK. High risk of retrograde/anejaculation.

Prognosis

- Cure rates of >95% for stage 1 tumours.
- Metastatic disease also has excellent long-time survival rates with combination therapy.

Haematuria

Causes and features

May be NVH or VH.

- UTI. Commonest cause; usually associated with LUTS, particularly cystitis.
- Renal/ureteric stones. Often associated with pain (ureteric colic).
- Malignancy (TCC, renal adenocarcinoma, prostate adenocarcinoma). Most likely to be macroscopic, often with few other acute symptoms.
- Post-interventional. For example, post-TURP, post-cystoscopy, post-catheterization.
- Renal disease. For example, glomerulonephritis, vasculitis; usually causes microscopic haematuria, is often asymptomatic, and rarely presents as an emergency.

Complications

- Suprapubic colicky pain or acute retention of urine suggests clots in the bladder/urethra.
- Cardiovascular collapse is rare.

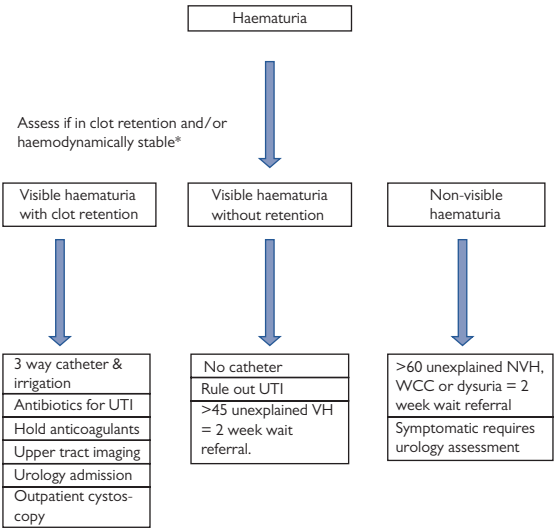

Fig. 11.4 Haematuria. If VH persists after UTI treated, refer as 2-week wait. For haematuria, check BP, glomerular filtration rate (GFR), and albumin:creatinine ratio if no urology cause.

Emergency management

Resuscitation

- Establish large-calibre IV access if the bleed is large; give crystalloid fluid, up to 1000mL, if tachycardic or hypotensive.
- Do not catheterize without seeking senior advice if there is any suggestion of lower urinary tract pathology or post-interventional bleeding.
- Irrigation ('3-way') catheters may be used to relieve acute symptoms of clot colic or clot retention, but should be placed by experienced staff.
- Send blood for FBC (Hb, WCC), U&Es (Na^+, K^+), group and save, and clotting.

Establish a diagnosis

- Full clinical examination. Particularly check the prostate on PR exam.
- Ultrasound kidney. To identify renal tumours and cysts.
- Cystoscopy (usually rigid, may be flexible). To identify bladder tumours.
- CT scan abdomen (contrast). If renal/ureteric tumour is suspected.

Early treatment

- Ensure all clotting abnormalities are corrected.
- Ensure fluid balance is correct; promote active diuresis to prevent clot formation and retention.
- Transfuse blood only if Hb <8g/dL or the patient is symptomatic or high risk.
- Start antibiotics according to local protocol if infection is suspected (before cultures are available).

Definitive management

Transitional cell tumours

Transurethral resection will control symptoms, establish a diagnosis, and start treatment (see ➲ Testicular tumours, pp. 482–3).

Renal stones

(See ➲ Urinary tract stones, pp. 456–8.)

- May pass spontaneously.
- May need endoscopic removal, lithotripsy, or percutaneous treatment.

Post-discharge Flexible cystoscopy may be required if not performed recently.

Acute urinary retention

Causes and features

Defined as a painful inability to pass urine.

Local causes

- Prostatic enlargement (BPH or carcinoma) (see ➔ Benign prostatic hyperplasia, pp. 462–3). Often acute-on-chronic retention.
- Post-urological surgery, e.g. post-TURP, clot impaction.
- Bladder or urethral stone impaction.
- Pressure on bladder, e.g. late pregnancy, faecal impaction.
- UTI.

General causes

- Pharmacological, e.g.:
  - Anticholinergic side effects of many drugs.
  - Anaesthetic drugs.
  - Alcohol intoxication.
  - α-sympathomimetics.
- Post-non-urological surgery:
  - Precipitated by recent catheterization.
  - Abdominal surgery with lower abdominal pain.
  - Epidural or spinal anaesthesia.
- Loss of normal neurological control:
  - Spinal injury (trauma, 'slipped disc', neurological disease).
  - Epidural/spinal anaesthesia.

Symptoms

- Suprapubic pain, inability to pass urine despite desire.
- May dribble urine in small volumes, especially if there is underlying chronic retention.
- Palpable/percussible bladder strongly suggests pre-existing chronic retention/lower urinary tract disease.

Signs

- Prostatic enlargement on PR examination.
- Check for signs of neurological disease.

Emergency management

Resuscitation

- Give analgesia (e.g. morphine 5–10mg IV); it will also help relaxation and may aid spontaneous micturition.
- A warm bath may aid micturition in drug-induced retention.
- Catheterize if retention persists. Seek senior advice before starting if there are concerns about local pathology as a cause or if there is a history of previous surgical instrumentation of the urethra.
- Flexible cystoscopy-guided or suprapubic catheterization may be required for known or suspected urethral disease or failed urethral catheterization (see ➔ Urethral catheterization, pp. 276–7).
- Document initial urine volume passed after catheter inserted; large volumes without abdominal pain suggests underlying chronic retention.
- Send urine for M, C, & S.
- Send blood for FBC (Hb, WCC), U&Es (Na^+, K^+), and Cr.

Establish a diagnosis

- Check medications, especially recent changes.
- Cystoscopy may be required.
- Review full clinical examination, including neurological findings and rectal examination.

Early treatment

- Monitor renal function; should improve after relief of obstruction.
- Monitor fluid balance as diuresis may occur, necessitating IV fluid supplementation.
- Start antibiotics according to local protocols if there is evidence of a UTI.

Definitive management

Prostatic disease

(See ➲ Benign prostatic hyperplasia, pp. 462–3.)

- α-blocker may enable successful trial of voiding.
- TURP may be required.

Acute testicular pain

Causes and features

This is an acute emergency in men. Torsion of the testicle must be dealt with immediately to preserve testicular function. It is the commonest cause for referral for acute testicular pain.

Torsion of the testicle

Key facts

- Occurs due to anatomical variants in testicular anatomy, e.g. 'bell clapper' testicle. Deficient posterior anchoring of the testis due high insertion of tunica vaginalis allows free rotation.
- Peak age of incidence 12–18y.
- Torsion initially causes venous obstruction, but with prolonged ↑ venous pressure, arterial compression occurs and the testicle rapidly develops irreversible ischaemia and necrosis.
- Testicular salvage depends on the degree of torsion and time spent torted. Speed of presentation, diagnosis, and treatment are all important. Torsion >360° lasting longer than 24h results in near-universal complete or severe atrophy.
- Spermatogenic cells are more susceptible to ischaemia than Leydig cells. Subfertility may occur, even if the testicle is macroscopically normal after treatment.

Features

- Sudden onset of moderate to severe, constant, unilateral scrotal pain, often with nausea, vomiting, and central abdominal pain.
- May have been preceding episodes of intermittent pain that suddenly resolved.
- The testis is globally tender and high in the scrotum, and may have a transverse axis and be slightly enlarged. If it is infarcted, scrotal wall oedema and tenderness may be present. Absence of ipsilateral cremasteric reflex is the most reliable sign.

Torsion of the testicular appendages

- Occurs in testicular appendix 'hydatid of Morgagni' or epididymal appendages (e.g. cysts, ductal remnants).
- Similar features and symptoms to testicular torsion.
- The 'blue dot sign' is said to be pathognomonic when present.
- The testis and epididymis may be non-tender and the cremasteric reflex should be preserved.

Acute epididymo-orchitis

- Peak incidences vary according to cause, ages 35y and >55y.
- Common organisms include *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in the young (STIs).
- *Escherichia coli* and *Proteus* occur in chronic BOO or urinary tract instrumentation.
- One-third of ♂ adolescents with mumps develop orchitis, which is unilateral in 80%; a third of these testes atrophy.

Features
- Gradual onset of pain (hours or days).
- Dysuria, urethral discharge, and pyrexia are common.
- Tenderness and induration are localized to the epididymis and spermatic cord in epididymitis.
- Cremasteric reflex is preserved.
- Prehn's sign (relief of pain with scrotal elevation).

Idiopathic scrotal oedema
- Often less painful and tender than appears.
- Swelling is mostly cutaneous and normal size and texture testicle may be palpable with care.

Acute inguinal lymphadenopathy
- May occur 2° to lower limb, buttock, or perineal infections.
- Rarely part of systemic infection of lymphatic disorder.

Emergency management

Resuscitation Give analgesia (e.g. morphine 5–10mg IV).

Establish a diagnosis

Immediate surgical exploration is indicated for all cases where the diagnosis of torsion is considered possible and the history is short (i.e. testicular viability is still at issue).
- Testicular colour duplex ultrasound where symptoms have been present for days and testicular viability is unlikely if torted.
- Send MSU, urethral swab, and Chlamydia serology if suspected infection.

Definitive management

If a torted testicle is found at surgery:
- A viable testicle is detorted and fixed.
- A clearly non-viable testicle is excised.
- The opposite testicle is fixed (orchidopexy) to prevent the opposite side from torting in future.

Torsion of testicular or epididymal appendage Excise appendage.

Epididymo-orchitis
- Suspected STI, e.g. ceftriaxone 250mg IM single dose, doxycycline 100mg PO bd 14 days. Refer to GU medicine clinic with partner.
- Suspected UTI-related—ciprofloxacin 500mg PO bd 10–14 days.
- Scrotal elevation, local ice therapy, and oral NSAIDs may help.
- Abscess formation may require drainage or orchidectomy.
- Treatment of acute viral orchitis if symptomatic.

Chapter 12

Colorectal surgery

Ulcerative colitis

Key facts

- An acute and chronic inflammatory disease originating in the colonic columnar mucosa.
- Precise aetiology is unknown, but an environmental trigger combined with a genetic predisposition (family history) are factors.
- Often precipitated by an apparent acute GI infection; peak age of diagnosis is late teens and twenties, but may present in late adulthood.
- Commonest in white Anglo-Saxon Caucasians.

Pathological features

- Granular, hypervascular, and mildly oedematous mucosa with loss of vascular pattern seen at endoscopy.
- Acute neutrophil infiltration of the colonic mucosa and submucosa; mucosal crypt abscesses with goblet cell mucin depletion.
- With more severe inflammation, there are multiple aphthous ulcers, which may become confluent with only islands of inflamed mucosa and granulation tissue remaining ('pseudopolyposis').
- Transmural inflammation may occur in severe disease 2° to the widespread loss of mucosa and subsequent severe inflammation.
- Chronic 'burnt-out' disease leads to a pale, featureless, ahaustral pattern to the colon.
- Disease tends to be present in the distal colon and rectum and spread proximally with increasing extent of disease.

Clinical features

- *Proctitis.* Commonest presentation. Rectum 'always' involved unless already on topical treatment. Symptoms of urgency and frequency of defecation due to rectal irritability; bloody mucus mixed with loose stools (frank bloody diarrhoea rare).
- *Left-sided colitis.* Disease up to the splenic flexure. Symptoms of rectal irritation plus extensive bloody mucus in stools, often leading to bloody diarrhoea; mild associated systemic features.
- *Pancolitis.* Disease involving the entire colon. May be associated with mild 2° inflammation of the terminal ileum ('backwash ileitis'). Diarrhoea predominant feature; systemic features common (fever, malaise, anorexia, tachycardia). May be associated with anaemia (due to blood loss), hypoalbuminaemia, and hypokalaemia (due to mucus loss).

Diagnosis and investigations

Basic tests

High WCC and CRP; low Hb and albumin, especially during episodes of inflammation. AXR may show oedematous colonic mucosa ('thumbprinting') but is unreliable for diagnosis or extent of disease. Proctosigmoidoscopy usually shows erythematous, granular, or frankly ulcerated rectal mucosa with mucus and blood. Biopsies should be taken before starting treatment. Always send stool for M,C,&S and test for parasites and cysts in any acute presentation to exclude infectious causes.

Advanced tests

Extent of disease is best assessed with colonoscopy and biopsies; will also usually exclude colonic Crohn's disease.

Treatment

(See ➔ pp. 994–5 for management of acute severe colitis.)

Medical treatment

Principles are to reduce inflammation and prevent complications. Acute derangements in blood results should be corrected (e.g. blood transfusion for severe anaemia, potassium supplementation, nutritional support for hypoalbuminaemia).

Proctitis

Topical treatment is the mainstay of treatment

- Topical steroids—Predsol® or Budenofalk® suppositories.
- Topical 5-aminosalicyclic acid (5-ASA) suppositories or enemas.

Left-sided colitis

- Topical steroids—Predsol® or Budenofalk® foam enemas (penetrate up as far as the splenic flexure).
- Topical 5-ASA foam enemas.

Left Sided and Pancolitis

- Topical steroids or 5-ASA treatments for local symptoms.
- Usually need systemic treatment, e.g. PO steroids (prednisolone), 5-ASA treatment.
- *For moderate to severe Flare*.
- Cortiment 9mg OD for 1 month (budesonide MMX)
- Oral Steroids such as Budesonide
- *For severe Flare not requiring hospital admission*
- Prednisolone orally for 2 weeks
- After 2 courses step up to thiopurine (Azathioprine) and if this is not tolerated or unsuccessful step up to anti TNF
- Surgical treatment

Surgery is indicated for acute colitis that fails to respond to treatment (➔ Acute severe colitis, pp. 994–5) and for chronic colitis when:

- Chronically symptomatic despite maximal medical therapy.
- Medical therapy controlling symptoms, but associated with unacceptable side effects, e.g. osteoporosis, immunosuppression.
- Recurrent exacerbations affecting growth or development in adolescents.
- Confirmed diagnosis of either high-grade dysplasia or dysplasia-associated lesion or mass (DALM), or carcinoma of colon.

Surgical treatment may be:

- Proctocolectomy (removal of colon and rectum) with ileoanal pouch formation (➔ Restorative pelvic surgery, pp. 504–5).
- Panproctocolectomy (removal of colon, rectum, and anus) with end-ileostomy formation (permanent).
- Subtotal colectomy (removal of colon) with ileostomy (used when the patient is too unwell for major pelvic surgery, e.g. for acute severe colitis).

Crohn's disease

Key facts

- A chronic inflammatory, non-caseating, granulomatous disease affecting any part of the GI tract.
- Associated with several extraintestinal disorders (see Box 12.1).
- Precise aetiology is unknown, but products from the bacterial flora combined with a genetic predisposition (family history) are factors.
- Peak age of onset of symptoms is the teens and early twenties, but diagnosis is often several years later.
- Commonest in white Anglo-Saxon Caucasians.

Pathological features

- Commonly focused in the terminal ileum and caecum, but may affect the anus, colon, or entire small bowel.
- Anal Crohn's disease is not common but may be severe and associated with active small bowel disease.
- Colonic Crohn's disease is a long-term risk factor for CRCa formation.
- Affected bowel looks blue-grey and thickened, with spiral surface vessels and encroachment of the mesenteric fat around the bowel ('fat wrapping').
- Transmural inflammation in the form of lymphoid aggregates, particularly in the subserosal tissues ('Crohn's rosary'), mucosal crypt ulceration, and fissuring ulceration.
- Mucosal thickening and serpiginous longitudinal ulceration combine to give the appearance of 'cobblestoning'.
- Perforation, fistulation, and abscess formation are occasional 'fistulizing' sequelae of transmural inflammation.
- Extensive fibrosis and smooth muscle hyperplasia may occur, giving rise to stenosis.

Clinical features

- *Inflammatory features.* Fever, malaise, abdominal pain (often RIF), change in bowel habit (usually diarrhoea without blood), and weight loss. Children and adolescents may have failure to thrive or have retarded growth. Rectal bleeding is rare, except in Crohn's colitis.
- *Fistulizing features.* Para-enteric abscess formation, often with a tender abdominal mass, fistula formation (ileocolic, ileoileal, ileocutaneous); rarely free perforation with features of peritonitis.
- *Stenosing features.* Colicky abdominal pain, weight loss due to poor food intake ('food fear'), palpable or visible distended small bowel loops.
- *Anal disease.* Atypical severe anal fissures, fistula in ano, anal mucosal thickening, and discoloration.

Diagnosis and investigations

Basic tests

High WCC and CRP; low Hb and albumin, especially during episodes of inflammation.

Advanced tests

- In acute presentations, abdominal CT may show an inflammatory mass, abscess formation, and localized or free perforation.
- In subacute or chronic presentations, small bowel disease may be shown by a small bowel contrast study (showing mucosal irregularity and narrowing) or a white cell scan showing ileal 'hot spots'.
- Crohn's colitis is diagnosed by endoscopy and biopsy.
- Anal disease may require EUA, anal ultrasound, or MRI scanning for assessment.
- OGD and biopsies may show features of Crohn's in gastric mucosa.

Treatment

Medical treatment

Principles are to reduce inflammation and control complications. Acute derangements in blood results should be corrected.

- Systemic (5-ASA) drugs are first-line acute and long-term treatment.
- Systemic steroids (hydrocortisone, prednisolone) control acute exacerbations of inflammation, and steroids with very high first-pass metabolism (budesonide) can be used chronically.
- Immunosuppressives (azathioprine, 6-mercaptopurine) are used as maintenance therapy and anti-TNF-α antibodies (infliximab, adalimumab) may be effective in fistulizing complications.
- Dietary manipulation (elemental diet) may reduce inflammatory factors.

Surgical treatment

Principles are to deal with septic complications, relieve significant bowel obstruction, and remove as little bowel as possible. Indications for surgery include the following.

- *Acute.* Free perforation, severe haemorrhage, acute severe colitis, complete intestinal obstruction.
- *Subacute.* Inflammatory mass, subacute obstruction, abscess formation, symptomatic fistulation.
- *Chronic.* Steroid dependency or complications, growth retardation, cancer treatment or prevention.

Box 12.1 Extraintestinal manifestations of Crohn's disease

Associated with disease activity

- Pyoderma gangrenosum
- Erythema nodosum
- Primary biliary cirrhosis

Independent of disease activity

- Ankylosing spondylitis
- Polyarthritis
- Chronic active hepatitis

Other forms of colitis

Key facts and pathological features

Various insults of widely differing origin may give rise to colitis other than idiopathic inflammatory bowel disease.

Acute infective colitis

- Typically caused by pathological variants of normal enteric organisms, e.g. enteropathogenic Escherichia coli; only rarely progresses to acute severe colitis.
- Typhoid colitis (Salmonella typhi) (rare in the UK). Typified by acute bloody diarrhoea, but few if any colonic mucosal neutrophils on biopsy due to bone marrow suppression.

Clostridium difficile-related colitis

Caused by Clostridium difficile infestation. Associated with antibiotic use, particularly third-generation cephalosporins (even as a single dose), and prolonged inpatient stay. Toxin A produced by the organism causes acute severe inflammation in the mucosa.

Clinical picture may be varied.

- *C. difficile diarrhoea.* Foul green liquid without bloody stools.
- *Acute C. difficile colitis.* Caused by progressive rapid mucosal loss, acute neutrophil infiltration, and inflammation.
- *Pseudomembranous colitis.* Exudate and slough forms grey-white 'plaques' of material on the denuded colonic surface called pseudomembranes. May rapidly progress to acute severe 'invasive' colitis, especially in the immunocompromised or acutely unwell. Typified by 2° infections associated with mucosal loss.

Neutropenic colitis

Occurs in the severely immunocompromised with neutropenia and/or neutrophil dysfunction. Caused by multiple, normally non-pathogenic, enteric organisms colonizing the colonic mucosa.

Radiation colitis

Acute, transient colitis caused by mucosal injury 2° to external beam radiotherapy. May progress to chronic mucosal damage, haemorrhagic telangiectasia, and possible stricturing after months or years.

Ischaemic colitis

Commonest in the upper left colon where the collateral blood supply between the middle and inferior colic arteries is poorest. Usually precipitated by an acute occlusion of part or all of the inferior mesenteric artery. May progress to infarction but often presents with acute-onset bloody diarrhoea and abdominal pain; may settle spontaneously, although occasionally forms an ischaemic stricture.

Clinical features

Broadly similar, independently of the underlying cause. Typical features are vague abdominal pain, mild fever (absent in neutropenic colitis), and diarrhoea (may be bloody, especially in ischaemic, radiation, severe pseudomembranous, and typhoid colitis). Cessation of diarrhoea, except with treatment, suggests acute severe colitis is developing and should be investigated urgently.

Diagnosis and investigations

Depending on the suspected cause:

- Stool sent for C. difficile toxin (CDT), three samples.
- Stool for M,C,&S (if atypical infective causes possible, also send for cysts, parasites, and ova (C,P,&O)).
- Plain abdominal radiograph may show thickened colonic haustrae.
- CT abdomen often shows typical mucosal thickening in colitis and may be diagnostic for pseudomembranous colitis.
- Flexible endoscopy (usually flexible sigmoidoscopy) with biopsy.

Treatment

Medical treatment

- *Acute infective colitis.* Antibiotics only if severely symptomatic.
- *C. difficile diarrhoea/colitis.* Oral vancomycin up to 200mg PO daily or metronidazole 400mg PO tds; treatment may be as for pseudomembranous colitis if severe.
- *Pseudomembranous colitis.* Oral vancomycin up to 200mg PO daily or metronidazole 400mg PO tds; adjuvant systemic treatment may be added in severe cases, e.g. IV vancomycin or tigicycline.
- *Neutropenic colitis.* Broad-spectrum antibiotics, bone marrow support.
- *Radiation colitis.* Symptomatic treatment only; anti-diarrhoeals.
- *Ischaemic colitis.* Supportive treatment; anticoagulation may be appropriate if the underlying cause is thromboembolic.

Surgical treatment

Rarely indicated. Any form of colitis may progress to acute severe colitis and require emergency colectomy (➲ Acute severe colitis, pp. 994–5). Indications are:

- Failure to respond to maximal medical therapy with life-threatening colitis (usually requires perioperative ITU support).
- Complications of colitis. Uncontrollable bleeding, perforation (especially in ischaemic or neutropenic colitis).

Key revision points—colorectal resections

Anastomosis of the colon/rectum may be in several ways:

- Hand-sewn. Either end-to-end or end-to-side, usually a single layer of sutures (dissolvable), either interrupted or continuous.
- Stapled colonic. By mechanical stapler ('linear stapler'), usually side-to-side.
- Stapled colorectal. By mechanical stapler ('endoluminal circular stapler'), end-to-end.
- Defunctioning (loop) ileostomy typically for rectal anastomosis when:
  - Below the peritoneal reflection.
  - Comorbidities (diabetes, age, previous radiotherapy acute illness).

Colorectal polyps

'Polyp' is a purely descriptive term and any growth from the lining of the large bowel can be described as a polyp. Polyps may be predominantly raised with a stalk attachment (pedunculated), flat and spreading over the surface of the bowel wall (sessile), or occasionally a combination of the two.

Key facts and pathological features

Polyps may arise for many different reasons.

Juvenile polyps

Mucin-filled cystic swellings of the lower rectal mucosa. Rarely, part of a hereditary syndrome (juvenile polyposis) with multiple juvenile polyps throughout the colon; small ↑ risk of CRCa.

Hamartomatous polyps

Polyps containing excessive amounts of the normal architectural components of the bowel wall, usually isolated. May be part of a hereditary syndrome (Peutz–Jeghers syndrome, with polyps characterized by extensive branched growth of the muscularis mucosa); small ↑ risk of CRCa and other GI cancers.

Hyperplastic polyps

Small sessile polyps formed from normal elongated mucosal crypts. Only associated with risk of CRCa if numerous ('hyperplastic polyposis').

Adenomatous polyps

- True neoplastic polyps formed by excessive growth of the colorectal epithelium; divided by the morphology of the glandular tissue into tubular, tubulovillous, and villous types.
- May be sessile, pedunculated, or mixed.
- Thought to be the precursor of most CRCa; the risk of cancerous change within an adenomatous polyp increases with size (particularly >1cm), villous morphology, and sessile form.
- Majority are sporadic (either isolated or in small numbers), although occasionally part of a hereditary syndrome.

Familial adenomatous polyposis

Caused by an autosomal dominant defect in the *APC* gene on chromosome 5. Characterized by between dozens and thousands of adenomatous polyps in the colorectum and an ↑ risk of polyp formation in the stomach and duodenum. The risk of cancerous transformation in any given polyp is similar to that in normal polyps, but the overall risk is very high due to the vastly ↑ number present.

Associated with:

- Desmoid formation, particularly in the abdominal tissues.
- Multiple osteomata, fibromata, and thyroid inflammation (called Gardner's syndrome).

Hereditary non-polyposis colorectal cancer

A range of abnormalities of the mismatch repair (MMR) genes predispose adenomas to acquiring multiple genetic defects, and so progressing more rapidly than normal to cancer, although the overall rate of adenoma formation is similar to that in normals.

Clinical features

Most polyps are asymptomatic, although symptoms may occur with increasing size and with proximity to the anus. Typical symptoms are:

- *Bleeding*. Usually low volume, dark red, often flecks or mixed with stool.
- *Mucus discharge*. White, clear, or watery; commonest with large villous adenomas and may cause hypokalaemia and hypoproteinaemia if the villous adenoma is large with copious mucus discharge.
- *Prolapse*. If pedunculated and low in the rectum, polyps may prolapse out of the anus.

Diagnosis and investigations

- Most polyps are diagnosed by colonoscopy.
- Most patients with polyps require further follow-up investigations to keep them under surveillance for future polyp formation; the frequency and length of follow-up depend on the number, size, and histology of the polyp.[1]
- Hereditary polyposis syndromes may be investigated by genetic mutation analysis.

Treatment

Medical treatment

- Colonoscopic polypectomy (➔ Abdominal investigations, pp. 64–5). Simple, is carried out for pedunculated polyps larger than 1–2mm; others which are larger or sessile and spreading, may be removed by EMR/ESD (➔ Minimally invasive colorectal surgery, pp. 506–7).
- Patients with FAP require regular gastroscopy and upper GI surveillance to identify premalignant polyps.

Surgical treatment

- Surgical excision is required for polyps that are too large or unsuitable for colonoscopic removal and in which there is a risk of current or future malignant change; for colonic polyps, this means either resection or open excision.
- Rectal polyps may be removed by transanal endoscopic microsurgery (TEMS) or transanal minimally invasive surgery(TAMIS) (see ➔ Minimally invasive colorectal surgery, pp. 506–7).
- FAP is usually treated by proctocolectomy (usually with ileoanal pouch formation) or colectomy and ileorectal anastomosis before early adulthood; other polyposis syndromes may also be treated by prophylactic colectomy.

Reference

1 Atkin WS, Saunders BP (2002). Surveillance guidelines after removal of colorectal adenomatous polyps. Gut **51**(Suppl V): v6–9.

Colorectal cancer

Key facts

CRCa is the second commonest tumour and commonest GI malignancy. One in 18 of the population will suffer CRCa (♂:♀ ≈ 3:1). Peak age of incidence 55–75y, but is increasing in younger ages.

Pathological features

The predominant type is adenocarcinoma (mucinous, signet ring cell, and anaplastic subtypes). Classified as well, moderately, or poorly differentiated. Predisposing factors include:

- Polyposis syndromes (including FAP, Lynch Syndrome, juvenile polyposis).
- Strong family history of colorectal carcinoma.
- Previous history of polyps or CRCa.
- Chronic UC or colonic Crohn's disease.
- Diet poor in fruit and vegetables.

Morphology

CRCa may occur as a polypoid, ulcerating, stenosing, or infiltrative tumour mass. The majority (75%) lie on the left side of the colon and rectum (rectum, 45%; descending–sigmoid, 30%; transverse, 5%; right-sided, 20%). Three to five per cent have a synchronous carcinoma at the time of diagnosis.

Clinical features

Rectal location

- PR bleeding. Deep red on the surface of stools.
- Change in bowel habit. Difficulty with defecation, sensation of incomplete evacuation, and painful defecation (tenesmus).

Descending–sigmoid location

- PR bleeding. Typically dark red, mixed with stool, sometimes clotted.
- Change in bowel habit. Typically ↑ frequency, variable consistency, mucus PR, bloating, and flatulence.

Right-sided location

Iron deficiency anaemia may be the only elective presentation.

Emergency presentations

Up to 40% of colorectal carcinomas will present as emergencies.

- Large bowel obstruction (colicky pain, bloating, bowels not open).
- Perforation with peritonitis.
- Acute PR bleeding.

Algorithm for referral of colorectal symptoms

Urgent suspected cancer pathway referral (2-week wait, 2WW)

- Patients aged 40 and over with unexplained weight loss and abdominal pain, or
- Patients aged 50 and over with unexplained rectal bleeding, or
- Patients aged 60 and over with:
  - Iron deficiency anaemia, or
  - Changes in their bowel habit, or
  - Tests showing occult blood in their faeces.

Consider a suspected cancer pathway referral (for an appointment within 2 weeks) for CRCa in adults with a rectal or abdominal mass.

Consider a suspected cancer pathway referral (for an appointment within 2 weeks) for CRCa in adults aged under 50 with rectal bleeding **and** any of the following unexplained symptoms or findings:

- Abdominal pain.
- Change in bowel habit.
- Weight loss.
- Iron deficiency anaemia.

Protocol for rectal bleeding outside the 2WW criteria

- Emergency admission for patients with profuse rectal bleeding causing hypotension or drop in Hb.
- Colorectal referral (direct access or otherwise) for patients with persistent unexplained rectal bleeding.
- Colorectal referral (direct access or otherwise) for patients with persistent rectal bleeding and/or anorectal symptoms resistant to local first-line treatments.

Diagnosis and investigations

Elective diagnosis By PR examination or rigid sigmoidoscopy for rectal carcinoma. Colonoscopy is the preferred diagnostic investigation (alternatives are barium enema and CT colonography).

Emergency presentations Commonly diagnosed by abdominal CT scan. Single-contrast enema may be used when the diagnosis of large bowel obstruction is possible and CT scanning is unavailable. Acute PR bleeding is sometimes investigated by urgent colonoscopy.

Staging investigations

- Assessment of the presence of metastases (liver, lung, or para-aortic). Thoracoabdominopelvic CT scanning is gold standard; CT-PET scan may be used to evaluate equivocal lesions.
- Assessment of local extent. For colonic carcinoma, CT scanning is adequate; for rectal cancer, pelvic MRI is commonly used.
- Assessment of synchronous tumours. If not diagnosed by colonoscopy or barium enema, one of these two tests is usually performed to identify synchronous tumours.
- Tumour marker (CEA) is of no use for diagnosis or staging but can be used to monitor disease relapse if raised at diagnosis and falls to normal after resection.

Pathological staging

Duke's (~5y survival, %)

- A, confined to bowel wall only (75–90)
- B, through bowel wall (55–70)
- C, any with +ve lymph nodes (30–60)
- D, any with metastases (5–10)

TNM

- T1–4, stages of invasion of bowel wall
- N0/1/2, no/up to 4/>4 lymph nodes involved
- M0/1, metastases not present/present

Treatment

Potentially curative treatment

Suitable for technically resectable tumours with no evidence of metastases (or metastases potentially curable by liver or lung resection).

- Surgical resection (with lymphadenectomy) is the only curative treatment. Typical operations:
  - Right/transverse. Right/extended right hemicolectomy.
  - Left. Left hemicolectomy.
  - Sigmoid/upper rectum. High anterior resection.
  - Lower rectum. Low anterior resection/abdominoperineal resection (APER).
  - Anorectal· APER.
- Preoperative (neoadjuvant) chemoradiotherapy may be used in rectal cancer to increase the chance of curative resection.
- Adjuvant chemotherapy—5-fluorouracil (5-FU)-based—is offered for tumours with positive lymph nodes or evidence of vascular invasion.
- Hepatic or lung resection may be offered to patients with suitable metastases and a clear resected/resectable 1° tumour.

Palliative treatment

For unresectable metastases or unresectable tumours.

- Chemotherapy may effectively extend life expectancy with a good quality of life.
- Obstructing tumours may be endoluminally stented with self-expanding metal stents or transanally ablated if rectal.
- Surgery reserved for untreatable obstruction, bleeding, or severe symptoms.

Restorative pelvic surgery

Low/ultra-low anterior resection

Anterior resection = removal of part or all of the rectum and anastomosis of the left colon to the remaining stump of tissue.

- Low anterior resection refers to a join that takes place below the level of the peritoneal reflection, i.e. to a short stump of the rectum.
- Ultra-low anterior resection refers to a join that takes place onto the top of the anal canal, i.e. no native rectum remains. The anastomosis may be stapled or sewn by hand.

The lower the level of the anastomosis, the higher the risk of complications of anastomosis, particularly anastomotic leakage (➲ Post-operative anastomotic leakage, pp. 518–19). Most low and almost every ultra-low anastomoses will have a temporary loop ileostomy formed to reduce the risk of major septic complications and consequences of leakage, but cannot prevent them.

Indications

- Rectal carcinoma.
- Rectal adenoma untreatable by other means—very rare with Transanal endoscopic microsurgery (TEMS) or Transanal minimally invasive surgery (TAMIS).
- Severe or complex anorectal sepsis (including rectovaginal fistula).

Ileoanal pouch formation

For operations that remove all the colon and rectum, but do not require removal of the anus, a permanent stoma can be avoided by the formation of an ileal pouch. Formed from a side-to-side double fold of the ileum ('J' pouch) or three folds sewn together ('W' pouch), joined either by hand or by staples to the upper anal canal (ileoanal anastomosis). A temporary loop ileostomy is often formed for the same reasons as for a low anterior resection (above).

Indications

- UC not responding to medical management.
- FAP or multiple colorectal polyposis.
- Multiple colonic tumours, including the rectum.

Complications of pelvic anastomosis (anterior resection and ileoanal pouch)

- *Leakage.* Occurs in up to 15% of cases; highest in the lowest anastomosis. Typically presents as fever, abdominal pain, and tachycardia (➲ Post-operative anastomotic leakage, pp. 518–19).
- *Bleeding.* Uncommon; usually settles with supportive treatment.
- *Ischaemia.* The proximal bowel involved in the anastomosis may become ischaemic. This may present as a leak, bleeding PR, or fever and tachycardia. Diagnosis is by careful flexible sigmoidoscopy. May resolve spontaneously; progressive ischaemia results in perforation and death if not corrected by surgery.
- *Stenosis.* Narrowing of the anastomosis or bowel used to form it is an occasional late complication. Presents with difficulty in defecation and small-volume frequent stools. Treatment is dilatation under anaesthesia or endoscopic dilatation; very rarely requires re-operation.

- *Pouchitis.* Inflammation of the ileoanal pouch resulting in pain, high frequency, bleeding, and other less specific symptoms. Can be treated with antibiotics, steroids, or probiotics.
- Poor function. The low nature of these anastomoses can lead to variable function in patients, resulting in diarrhoea, urgency, incontinence, and discomfort (termed anterior resection syndrome). This is treated on an individual basis and may require manoeuvres such as biofeedback, irrigation, nerve stimulation, and, at its worst, surgical intervention.

Minimally invasive colorectal surgery

Transanal endoscopic microsurgery TEMS or Transanal minimally invasive surgery

- TEMS. Large-calibre operating protoscope with an operating microscope allows microsurgery within the rectum.
- Transanal minimally invasive surgery (TAMIS). A specialized TAMIS device is placed inside the anal canal, insufflating it with CO_2 in a pressure-controlled environment, allowing access and visibility for utilization of laparoscopic instruments.

Indications

- Excision of large adenomas of the rectum (up to the recto-sigmoid junction) in a single specimen either by mucosectomy (or occasionally full-thickness).
- Excision of early rectal carcinoma (only if <3cm in size, early tumour (T1), no adverse features, or in elderly/comorbid patients) by single-specimen, full-thickness excision.
- Repair of a rectovaginal fistula.

Advantages Include entirely endoscopic technique; very low risk of pararectal/pelvic sepsis; single complete specimen for histological assessment; may avoid more radical surgery.

Complications Include bleeding (rarely requires active treatment), infection in the pelvic tissues (rare; presents with deep pelvic pain, fever, tachycardia, and disturbance of bowel habit; treat with IV antibiotics).

Laparoscopic surgery

Several variants now exist. All use the same principles: minimal incisions, avoidance of exposure of viscera, light anaesthetic techniques with minimal opiates, and often enhanced recovery post-operatively.

Typical indications

- Any colorectal resection can be performed by laparoscopic surgery.
- Rectopexy for prolapse.
- Combined treatment of large/extensive colonic polyps.
- Formation of some stomas.

Two newer variants have been used:

- Single-incision laparoscopic surgery (SILS). One larger port for camera and instruments used at the umbilicus.
- Combined laparoscopic and endoscopic single-site surgery (LESS). Combines laparoscopic mobilization and handling with endoluminal endoscopic techniques to remove very large lesions.

Advanced polypectomy

Several advanced endoscopic techniques are used to remove large and often sessile colonic polyps.

- *EMR.* Excision (usually piecemeal) of (presumed) adenoma with use of submucosal fluid injection to facilitate snaring of the polyp.
- *ESD.* Attempted complete excision of (presumed) adenoma using submucosal injection and endoscopic diathermy 'knife'.

Robotic colorectal surgery

Robotics using the Da Vinci system has been adopted across other fields of surgery and is gaining popularity in colorectal surgery. It allows surgeons to see 3D images and better dexterity through ↑ rotation and angulation of the laparoscopic instruments, which is particularly significant in pelvic surgery (anterior resection). There are, however, cost implications and no clear patient outcome benefit has yet been seen over standard laparoscopic surgery.

Diverticular disease of the colon

Key facts

Colonic diverticula are acquired outpouchings of the colonic mucosa and overlying connective tissue through the colonic wall.

- Tend to occur along the lines where the penetrating colonic arteries traverse the colonic wall between the taenia coli.
- Associated with hypertrophy of the surrounding colonic muscle, with thickening of the colonic mucosa. This is probably due to the underlying pathological process, which is high-pressure contractions of the colon, causing chronic pressure on the colonic wall.
- Peak age of presentation is 50–70y, but diverticular disease is increasing in frequency and occurring at a progressively younger age.

Clinical and pathological features

Asymptomatic

The majority of diverticular disease is found incidentally.

Painful diverticular disease

Intermittent LIF pain may be due to diverticular disease, but irritable bowel syndrome commonly coexists and may be the cause of symptoms.

Acute diverticulitis ➲ *pp. 992–3*

Rapid onset of LIF pain, nausea, fever, and frequently with loose stools. Usually febrile, with moderate tachycardia and LIF tenderness. Colonic wall shows acute neutrophil infiltration around the inflamed diverticulum and in the subserosal tissues.

Bleeding diverticular disease

Usually spontaneous in onset, with no prodromal symptoms. Presenting with large-volume, dark red, clotted rectal blood. Due to rupture of a peridiverticular submucosal blood vessel. Not typically associated with inflammation.

Complications

Pericolic/paracolic mass/abscess ➲ *pp. 966–7*

Acute diverticulitis may progress to persistent pericolic infection, with thickening of surrounding tissues and the formation of a mass. If this suppurates, a pericolic abscess forms. Enlargement and extension of this into the paracolic area lead to a paracolic abscess. The features are those of acute diverticulitis, with a swinging fever, fluctuating tachycardia, unresolving abdominal pain, and a tender LIF mass.

Peritonitis ➲ *pp. 968–9*

Perforation of a pericolic or paracolic abscess usually leads to purulent peritonitis. Direct perforation of the acute diverticular segment leads to faeculent peritonitis. The features are of acute diverticulitis with high fever, severe abdominal pain, and generalized guarding and rigidity.

Diverticular fistula

Acute infection with paracolic sepsis may drain by perforation into adjacent structures. This is typically the posterior vaginal vault in women or the bladder in either sex. Colovesical fistula leads to recurrent UTI caused by enteric organisms, with pneumaturia and debris in the urine. Colovaginal fistula leads to faeculent per vagina (PV) discharge.

Stricture formation

Chronic or repetitive inflammatory episodes may lead to fibrosis and narrowing of the colon. A history of recurrent diverticulitis with recurrent colicky abdominal pain, distension, and bloating suggests stricture formation.

Diagnosis and investigations

- Elective diagnosis is usually by colonoscopy, although this is a substandard investigation to assess the number and extent of diverticula.
- Contrast CT scanning is the test of choice to identify complications, including abscess formation, stricture, and perforation.
- Hb, WCC, and CRP during acute episodes of inflammation.
- Elective endoscopic visualization is used to exclude coexisting malignancy when CT diagnosis is initially made.

Treatment

Medical treatment

- High-fibre diet, high fluid intake, and stool softeners to reduce intracolonic pressure.
- IV antibiotics (amoxicillin 500mg IV tds, metronidazole 500mg IV tds, gentamicin IV od) during acute infective exacerbations.
- Significant paracolic abscesses may be drained by radiological guidance.

Surgical treatment

- Resection is indicated for:
  - Acute inflammation failing to respond to medical management.
  - Undrainable paracolic sepsis.
  - Free perforation.

The affected region should be resected (segmental colectomy). The ends may be re-anastomosed if they are healthy and the patient's general condition is suitable and covering with a defunctioning ileostomy is necessary. If not, a proximal end-colostomy and oversewing of the distal end is usual (Hartmann's type resection).

- Stricture may be treated by elective resection or emergency resection of causing acute obstruction not settling with conservative management.
- Recurrent episodes of diverticulitis, symptoms impacting on quality of life, or diverticular fistula may be treated by elective resection to prevent recurrent infections.

Rectal prolapse

Key facts

Rectal prolapse may be partial thickness (usually just mucosa) or full thickness involving all the layers of the rectal wall. Full thickness may be contained within the rectum (internal prolapse, also called intussusception). Commonest in post-menopausal women, multiple vaginal deliveries, associated with chronic straining and chronic disorders of defecation (which cause weakness of the pelvic floor and sphincter complex), and slow-transit constipation. Occasionally occurs in children suffering constipation (usually self-limiting).

Pathological features

Mucosa involved in prolapse undergoes chronic changes.

- Typically glandular branching and occasional gland misplacement.
- Thickening of the muscularis mucosae and excess submucosal collagen deposition.
- Mucosal inflammation and focal ulceration may also occur. Extensive mucosal ulceration associated with mucosal prolapse may result in an appearance called 'solitary rectal ulcer'.

Clinical features

- *Mucosal prolapse.* Discharge of mucus and small-volume faecal staining, pruritus ani, and occasionally small-volume, bright red rectal bleeding.
- Internal full-thickness prolapse. Sensation of rectal fullness/mass, incomplete defecation, dissatisfaction after defecation, and repeated defecation.
- External full-thickness prolapse. External prolapsing mass after defecation (usually requiring manual reduction), mucus and faecal soiling, occasional bright red rectal bleeding (may be large volume if prolapse becomes ulcerated).

Diagnosis and investigations

- Rigid sigmoidoscopy may show features of mucosal inflammation, particularly the anterior rectal mucosa.
- Prolapse may be demonstrable on straining in clinic.
- Defecating or MRI proctogram may be performed to confirm the diagnosis if it is unclear and surgery is contemplated. Proctogram required to confirm the diagnosis of internal prolapse if suspected. May also demonstrate associated problems of pelvic floor and rectocele.
- Colonic transit studies may be used if there is suspected slow-transit constipation and resection is possible.

Treatment

Medical treatment

- Avoidance of straining and adaptation of defecatory habit (biofeedback).
- Avoidance of constipation (stool softeners and bulking agents, rather than stimulants).

Surgical treatment

Mucosal prolapse

- Recurrent banding or dilute phenol injection of excess mucosa.
- Mucosal excision.

Full-thickness prolapse (internal or external)

Surgery indicated for failure of control of symptoms. Choice of operation depends on age and extent of prolapse.

- Delorme's perineal rectopexy (mucosal excision with sutured plication of the excessively long rectal muscle tube in an effort to shorten it to prevent prolapse). Ideal for very frail and elderly, but least successful, with highest recurrence rate of all surgical procedures.
- Altemeier's perineal rectal resection (mucosal and rectal muscle tube excision with sutured perineal anastomosis). Avoids abdominal operation but has ↑ morbidity due to perineal anastomosis.
- Transabdominal rectopexy (mobilization of the rectum and suturing to the pre-sacral fascia). May be done via laparotomy or laparoscopically. May be just to ventral surface of the rectum with suspensory mesh (ventral mesh rectopexy). Highest success rate for prevention of recurrence of prolapse. May be combined with a sigmoid resection if there is marked associated constipation on transit studies.

Key revision points—anorectal physiology

- The internal anal sphincter is smooth muscle and under involuntary control of the pelvic autonomic system. Relaxants include nitric oxide donors (e.g. GTN) and calcium antagonists (e.g. diltiazem).
- The external anal sphincter is skeletal muscle and under voluntary control of the pudendal nerve (S2, 3, 4). Relaxation (by temporary partial paralysis) may be achieved by botulinum toxin injection.
- Defecation is a complex sensorimotor process that requires intact pelvic autonomics, sacral spinal nerves, and pelvic floor muscle function.

Pilonidal sinus disease

Key facts

Single or multiple sinuses ('pits') that exist in the midline of the buttock clefts. Usually contain hair, inspissated secretions, and debris. Commonest in men and dark-haired, hirsute people, especially eastern Mediterranean races. Probably caused by local trauma, causing retention of hairs within initially normal midline pits. May be precipitated by long periods seated, e.g. lorry drivers, computer operators.

Pathological features

Typified by chronic inflammation. Once inflammation has started, sinuses often extend and may become interlinked. Lateral tracks may run out into the neighbouring buttock tissue.

Clinical features

- *Irritative features.* Intermittent discharge and inflammation, with pain and swelling.
- *Acute sepsis.* Acute abscess formation is common with swelling, pain, and erythema; may discharge spontaneously or may cause fistulation, with sinuses appearing in the lateral buttock tissue.
- *Chronic sepsis.* Usually follows unresolved acute sepsis either after spontaneous discharge or surgical drainage.

Diagnosis and investigations

- Ensure the patient is tested for occult DM.
- Very extensive sinus formation and fistulation may be assessed by MRI scanning of the natal cleft and buttocks.

Treatment

Medical/non-surgical

- Shaving of local hairs and washing of accessible cavities (usually by a partner or family member) may control local symptoms.
- Intermittent courses of antibiotics may be required for septic episodes.
- Formed pilonidal abscess or collection requires surgical drainage (under LA or GA).
- Recurrent acute sepsis or persistently symptomatic chronic sepsis usually requires surgical treatment.

Surgical

Principles of surgical treatments are:

- Excision of all sinus openings.
- Obliteration of all infected or chronically inflamed tissue.
- Obliteration of the natal cleft by flattening (thought to be most important in the prevention of recurrence by reducing the risk of further hair implantation).

Surgical options are:

- 1° excision with laying open of wound and closure by 2° intention are very rarely used, except in extensive recurrent disease; it requires daily dressings for many weeks or months.
- Tension-free apposition of the skin edges (may be by lateral flaps, e.g. Karydakis or Bascom procedures, or by plastic surgical flaps, e.g. rhomboid, rotational, or Z plasty flaps).

Key revision points—anatomy of the large bowel

- Main features of large intestine structure include:
  - Complete layer of circular smooth muscle throughout, but incomplete bands of longitudinal muscle (taeniae coli) in colon (complete in rectum).
  - Fatty appendages along taeniae (appendices epiploicae).
  - Folded internal mucosal appearances (haustrations).
  - 'Segmented' external appearances (sacculations).
- Four main arterial (and lymph node) territories (used for resections):
  - Ileocolic and right colic arteries (from SMA): last terminal ileal loop, caecum, and ascending colon.
  - Middle colic artery (from SMA): transverse colon up to the splenic flexure.
  - Left colic (from inferior mesentery artery (IMA)): splenic flexure and descending colon.
  - Superior rectal artery and its sigmoid branches (from IMA): sigmoid, rectum, and upper anal canal.
- Autonomic nerve supply:
  - Sympathetic, mainly from greater splanchnic nerves via SMA and IMA plexuses.
  - Parasympathetic, from vagus via SMA and IMA plexus from caecum to splenic flexure and from pelvic parasympathetics (S2, 3, 4) via hypogastric plexuses and retroperitoneal nerves from splenic flexure to upper anal canal.

Fistula-in-ano

Key facts

A fistula is an abnormal connection of two epithelial surfaces and the two surfaces joined in fistula-in-ano are the anorectal lining and the perineal or vaginal skin. Very common, especially in otherwise fit young adults. May occur in the presence of Crohn's disease; minor association with obesity and DM, very rarely due to trauma or ulceration of anorectal tumours.

Pathological features

Commonest cause is sepsis arising in an anal gland that forces its way out through the anal tissues to appear in the perianal, or in women vaginal, skin (cryptoglandular theory of fistula-inano). Often presents initially as an acute perianal abscess. The tissues through which the track pushes determines the classification of fistulae (see Fig. 12.1).

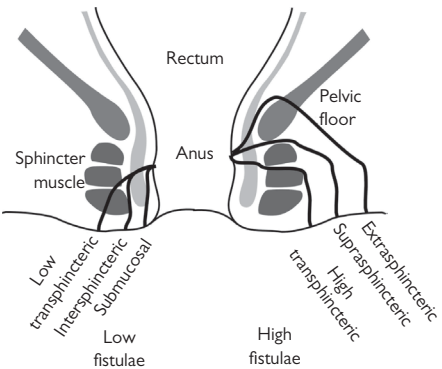

Fig. 12.1 Classification of fistula-in-ano.

Clinical features

- *Acute perianal abscess.* Rapid onset of severe perianal or perineal pain. Swelling and erythema of the perianal skin, with fever and tachycardia.
- *Recurrent perianal sepsis.* Recurrent intermittent sepsis typified by gradual build-up of 'pressure' sensation and swelling in the perianal skin and eventual discharge of bloodstained purulent fluid.
- *Chronic perianal discharge.* Persistent low-grade sepsis of the track, with chronic discharge of seropurulent fluid via a punctum that is usually clearly identified by the patient.

Diagnosis and investigations

Diagnosis and investigations should aim to confirm the presence of a fistula and identify the course of the track to determine the type of fistula:

- Examination of the perineum and rectal examination may reveal a palpable fibrous track.
- EUA with probing of any external opening to aid identification of the course of the track.
- Endoanal ultrasound (sometimes with hydrogen peroxide injected into the track) identifies the course of the track.
- MRI scanning is probably the most sensitive method of determining the course of the track and identifying any occult perianal or pelvic sepsis, particularly in complex/recurrent pathology.
- Flexible sigmoidoscopy if associated colorectal disease, e.g. Crohn's disease, is suspected.

Treatment

Medical treatment

- Antibiotics may reduce symptoms from recurrent sepsis but cannot treat the underlying fistula.
- Medical treatment of inflammatory bowel disease may dramatically reduce symptoms from associated fistulae.

Surgical treatment

Principles of surgical treatment are as follows:

- Drainage of any acute sepsis if present.
- Prevention of recurrent sepsis. Usually by insertion of a loose seton suture, e.g. silastic sling.
- Low fistula-in-ano. Lay open track; remove all chronic granulation tissue, and allow to heal spontaneously (fistulotomy); little risk of impairment of continence due to minimal division of sphincter tissues.
- High fistula-in-ano:
  - Remove fistula track and close the internal opening (core fistulectomy and endorectal flap advancement).
  - Slowly divide the sphincter tissue between the fistula and the perianal skin (cutting seton); low risk of incontinence.
  - Fill the fistula with fibrin glue (which has variable results).

Haemorrhoids

Key facts

Broad term, often incorrectly used to refer to any perianal excess tissue. True haemorrhoids are excessive amounts of the normal endoanal cushions that comprise anorectal mucosa, submucosal tissue, and submucosal blood vessels (small arterioles and veins). Commonest age of onset is in young adulthood. Associated with constipation, chronic straining, obesity, and previous childbirth. May become ulcerated and inflamed if recurrently prolapsing.

- If confined to the tissue of the upper anal canal, they are referred to as 'internal'.
- If extend to the tissues of the lower anal canal, they are referred to as 'external'.
- Typically occur in the same location as the main anal blood vessel pedicles (described as 3, 7, and 11 o'clock positions as seen in the supine position).

Clinical features

- *Features of irritation*. Pruritus ani, mucus discharge, and perianal discomfort.
- *Features of damage to mucosal lining*. Recurrent post-defecatory bleeding—bright red, not mixed with stools, on paper or splashing in the toilet pan.
- *Features of prolapse*. Intermittent lump appearing at anal margin, usually after defecation, may spontaneously reduce or require manual reduction.

Diagnosis and investigations

- Diagnosis is usually by rigid sigmoidoscopy and proctoscopy.
- Flexible sigmoidoscopy or colonoscopy may be appropriate if there is concern about the cause of symptoms; remember—haemorrhoids rarely start over the age of 55y and it is often best to assume another cause until proven otherwise in these cases.

Treatment

Medical treatment

Avoidance of constipation and straining; bulking or softener laxatives.

Surgical treatment

- Banding of excessive tissue. Best for internal haemorrhoids (not possible for external components due to excellent nerve supply of lower anal canal resulting in pain).
- Dilute phenol injections (5% in almond oil). Best for bleeding symptoms, although not as effective as banding.
- Haemorrhoidal devascularization procedures (e.g. arterial ligation 'HALO' either Doppler ultrasound-guided or blind). For failed topical treatments and surgery not indicated/desired.
- Stapled anopexy (also called procedure for prolapse and haemorrhoids (PPH)). Sometimes used for circumferential prolapsing haemorrhoids, although increasingly uncommon.
- Haemorrhoidectomy for large external haemorrhoids or haemorrhoids failing to respond to conservative treatment. Associated with a small risk of impaired continence and anal stenosis.

Key revision points—anatomy of the anus

- Lower third of the anal canal is somatic tissue in origin—stratified squamous epithelium; very sensitive (pudendal and distal sacral nerves); relatively poor blood supply and healing.
- Upper third of the canal is visceral tissue in origin—columnar epithelium; insensitive; excellent blood supply and healing.

Post-operative anastomotic leakage

Causes and features

Any intra-abdominal anastomosis may leak. Highest risk of leak occurs with oesophageal and rectal anastomosis and lowest with small bowel anastomosis (see Table 12.1).

Anastomotic leakage may present as one of several clinical pictures.

▶▶ *Peritonitis pp. 968–9*

Acute, severe generalized abdominal pain, with generalized guarding and rigidity. Fever, tachycardia, and tachypnoea are common. Diagnosis is usually clinical but may require CT scanning if unsure.

Intra-abdominal abscess

(➲ pp. 966–7.) Swinging fever and tachycardia, commonly around 5–7 days post-operatively. Localized tenderness related to the anastomosis may be present. Diagnosis should be sought by CT scanning.

Enteric fistula

A fistula between the anastomosis and the wound or another organ may occur. Usually occurs as a result of a subclinical leak and abscess formation that discharges through a pathway of low resistance. Often presents late as an apparent wound infection that discharges with enteric content. Diagnosis made by CT scanning or, occasionally, fistulography if presents very late.

▶ *Cardiovascular complications*

Sepsis originating from an initially subclinical leak may present with apparent cardiovascular complications, e.g. AF, SVT, chest pain, and sinus tachycardia. A wise precautionary rule is 'Any acute post-operative disturbance of physiology in a patient with an intra-abdominal anastomosis is due to leak until proven otherwise.'

Emergency management

Resuscitation

- Establish large-calibre IV access; give crystalloid fluid up to 1000mL if tachycardic or hypotensive.
- Catheterize and place on a fluid balance chart if hypotensive.
- Send blood for FBC (Hb, WCC), U&Es (Na+, K+), LFTs (albumin), group and save, clotting.
- Give appropriate analgesia if not on an epidural or PCA.

Establish a diagnosis

- Acute peritonitis needs no diagnostic investigation. Emergency re-look laparotomy should be organized immediately.
- CT scanning with IV and PO contrast is the investigation of choice for all other suspected leaks.
- For rectal anastomoses, a water-soluble contrast study may delineate a leak.

Early treatment

- Give IV antibiotics (e.g. IV cefuroxime 750mg tds + metronidazole 500mg tds).
- Monitor fluid balance hourly.

Table 12.1 Risk factors associated with high risk of leak

Patient factors	Disease factors	Operative factors
• Chronic malnutrition • Immunosuppression • Obesity	Unprepared bowel, e.g. obstruction	Poor blood supply or bowel ends
• High-dose steroid use • Diabetes mellitus	• Local or generalized sepsis • Metastatic malignancy	Tension on bowel ends

Definitive management

▶▶ *Peritonitis*

Always requires surgical intervention unless the patient is deemed unfit.

Prepare for theatre. Ensure blood results from resuscitation are available.

Stoma care review is not always necessary and often not practical or useful.

Once the leak has been identified, options for management include:

- Dividing the anastomosis, closing the distal end, and forming the proximal end into a stoma.
- Emptying the bowel (lavage) and forming a proximal defunctioning stoma.
- Re-forming or repairing the anastomosis (only suitable for fit patients with minimal contamination and an otherwise healthy anastomosis).
- Placing a large drain(s) next to the anastomosis.

Intra-abdominal abscess

- Radiologically guided drainage and antibiotics, provided patient does not become peritonitic or show signs of 2° complications.
- Open surgical drainage if inaccessible, unresponsive to radiological drainage or patient acutely unwell with sepsis and peritonitis.

Enteric fistula

- Usually managed by antibiotics. May close spontaneously; if fails to close, surgical repair may be required.
- Treat as for abscess or peritonitis if either develops.

Chapter 13

Paediatric surgery

Principles of managing paediatric surgical cases

Key facts

- Children are not small adults.
- Children come in different sizes; always obtain a weight before starting treatment and consider weight to surface area.
- Fluids and drug doses depend on body weight.
- Babies and children have different differential diagnoses from adults.
- Children often differ from adults in physiology and anatomy (see Table 13.1).
- Babies and young children have difficulty communicating symptoms and non-verbal cues need to be sought.

Special problems with babies

- Thermoregulation is impaired (immature sweating, high surface area to body weight increases rate of heat loss). Prone to hypothermia.
- Minimal glycogen stores. Prone to acute hypoglycaemia.
- Principal breathing pattern is diaphragmatic. Prone to breathing difficulties with abdominal distension.
- Immature body physiology. Much less biological functional reserve than adults; acute disturbances of physiology more serious, with less room for error.
- May not metabolize drugs as expected.

Intravenous fluids

Maintenance fluids

(See Box 13.1.)

Box 13.1 Paediatric fluid regimen

- 4mL/kg/h total fluid for each of the first 10kg of weight (0–10kg).
- 2mL/kg/h total fluid for each of the next 10kg of weight (11–20kg).
- 1mL/kg/h total fluid for each subsequent kilogram of weight (over 21kg).

- Always calculate fluid and Na^+ requirements according to weight.
- Remember to add glucose, especially for neonates.
- Adjust the fluid regimen according to clinical setting for neonates and premature babies.

Resuscitation fluids

- Surgical emergencies—20mL/kg bolus; repeat as necessary.
- 10mL/kg is more typical for medical conditions.

Table 13.1 Physiological changes with age

	Neonate	Infant	Child	Adolescent
Blood volume (mL/kg)	80–90	70–80	75	70
Oral fluid intake (mL/kg/day)	150	100	75	40
Systolic BP (mmHg)	60–80	70–100	90–110	100–120
Resting pulse rate (bpm)	120–160	110–150	100–140	80–120

Oesophageal atresia

Key facts

- Congenital abnormality of the formation of the oesophagus and trachea, usually resulting in discontinuity of the oesophagus (see Fig. 13.1).
- Often associated with other congenital abnormalities (VACTERL*).

Clinicopathological features

- May be diagnosed on prenatal ultrasound. Features include maternal polyhydramnios, absent stomach bubble, and associated abnormalities.
- At birth, these infants bubble at the mouth and seem to have excessive secretions. They should not be fed.
- If oesophageal atresia is suspected, a large-bore feeding tube should be passed via the mouth, if necessary, to exclude the diagnosis (small-calibre tubes can coil).
- If fed, cyanosis and aspiration can occur.
- Tracheo-oesophageal fistula without oesophageal atresia is unusual and presents later with recurrent aspiration/chest infections.

Diagnosis and treatment

- CXR and AXR, NGT coiled in oesophagus.
- Presence of stomach gas suggests distal tracheo-oesophageal fistula.
- Assess the spine and ribs on these images.
- Early echocardiography. Associated cardiac abnormalities may have implications for surgery and anaesthesia.
- Prior to discharge: renal tract and spinal ultrasound for associated VACTERL anomalies.

Medical treatment

- Nurse head-up.
- NBM.
- Replogle (oro-oesophageal) tube—this is a sump drain with continuous suction to decompress the blind-ending upper oesophagus.
- Consider antibiotics for possible aspiration pneumonia.

Surgical treatment

- Atresia with tracheo-oesophageal fistula (85%). Thoracotomy, ligation and division of fistula, and 1° anastomosis of oesophageal atresia.
- Isolated atresia.
  - Gastrostomy for feeding + continuous drainage of upper pouch.
  - Delayed closure of defect (may require interposition graft if a long segment involved).
- Isolated tracheo-oesophageal fistula. Ligation of fistula (usually through neck).

* VACTERL association: a constellation of congenital anomalies—Vertebral, Anorectal, Cardiac, Tracheo-oEsophageal, Renal, and Limb.

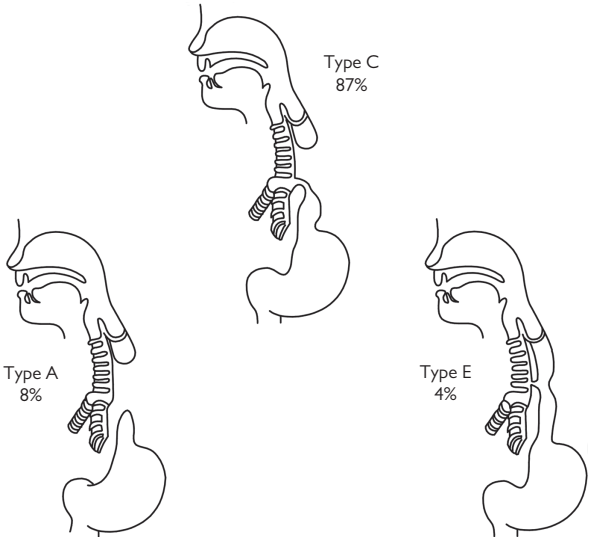

Fig. 13.1 Three varieties of oesophageal atresia. Type C (III), distal fistula; type A (I), atresia without fistula; type E (V), H-type fistula.

Further reading

Rothenberg SS (2019). Esophageal atresia and tracheoesophageal fistula malformations. In: Holcomb III GW, Murphy JP, St Peter SD (Eds). *Holcomb and Ashcraft's Pediatric Surgery*, 7th edn. Elsevier, New York, NY; pp. 437–59.

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Pyloric stenosis

Key facts (infants)

- Incidence 1 in 300 infants.
- ↑ familial risk.
- 30% occur in first born boys (♂:♀, 4.5:1).

Pathological features There is hypertrophy of the pyloric smooth muscle in early infancy, which occurs at about 3–6 weeks.

Clinical features

- Classically projectile, forceful vomiting.
- Often has been treated as gastro-oesophageal reflux
- Baby appears active and hungry, especially after vomiting.
- Small, green starvation stools passed infrequently.
- Poor weight gain or weight loss.
- Dehydration with hypochloraemic alkalosis in untreated, established condition.
- Baby usually looks well in early state.
- Dehydration, pallor, and underweight only in advanced condition.
- Epigastric fullness with left-to-right gastric peristaltic wave.
- Test feed is usually performed to palpate a pyloric 'tumour' (see Box 13.2).

Diagnosis and investigations

- If a pyloric 'tumour' is felt, surgery may be performed without further imaging.
- Ultrasound shows thickened (>4mm), elongated (>16mm) pyloric muscle and ↑ muscle-to-lumen ratio with ↓ movement of fluid through narrow canal (measurements for term infants).
- Barium meal (rarely necessary) shows an enlarged stomach, ↑ gastric peristalsis, and elongated, narrowed pyloric canal.
- Electrolytes and capillary blood gases (↓ Na+, ↓ K+, ↓ Cl–, base excess, and pH).

Treatment

- Resuscitation with IV rehydration.
- Correct hypovolaemia with boluses of 0.9% saline.
- Correct hypochloraemic alkalosis and hypokalaemia (may take 24–48h)—0.45% NaCl/5% glucose with added potassium chloride (KCl) at a rate of 120–150mL/kg/24h.
- Monitor correction with 6-hourly capillary blood gases.
- NGT drainage to prevent aspiration of vomited secretions.

Surgical treatment

- Pyloromyotomy (division of pyloric muscle fibres without opening of bowel lumen).
  - Traditionally performed via RUQ incision, now a periumbilical or laparoscopic approach is more typical.
  - Caution not to open mucosa and avoid the prepyloric vein ('of Mayo').
  - Feeds are started within the first 6h post-operatively and ↑ as tolerated.

Box 13.2 How to perform a test feed

- The principle is to keep the baby relaxed and suckling to allow examination.
- Position the baby—classically on mother's lap, with the left side presented to you. This is often modified with bottle feeds or Sweet Ease® on a dummy, often allowing adequate examination.
- When the baby is suckling, use fingertips of your left hand to palpate the epigastric and left hypochondrium areas under the liver edge.
- Minimal movement is needed and the pylorus presents itself to the fingertips.
- It feels similar to an olive in size and consistency.
- Overdistension of the stomach can cause vomiting and obscure the pylorus—aspirate the NGT whilst examining, to avoid this and sometimes demonstrate pylorus.
- Patience and calmness are the key. Allow time for a relaxed examination.

Malrotation and volvulus

Key facts and clinical features (neonates)

'*Bilious vomiting should be assumed to be malrotation volvulus until proven otherwise.*'

Malrotation

- This can present at birth or soon after, and symptoms may be due to duodenal obstruction or to complete small bowel volvulus.
- The caecum is typically in an abnormally high midline position, with attachments to the retroperitoneum.
- These 'Ladd's bands' may cross over the duodenum and cause obstruction.
- This leads to bile-stained vomiting.

Volvulus

- Twisting, in a clockwise direction, of non-fixed midgut loop on its narrow-based mesentery through 360° or more.
- Results in obstruction of bowel and superior mesenteric blood vessels.
- Signs. Sudden onset of abdominal pain, bilious vomiting, progression to shock, passage of blood PR.
- May be less dramatic.
- Potential for rapid development of gut necrosis—a surgical emergency.
- Older children may present insidiously or as rapid onset of shock with other less prominent symptoms.

Diagnosis and investigations

If in doubt, operate. Viability of twisted bowel is very time-dependent—delays in diagnosis can be very serious.

- Plain AXR. 'Double bubble' sign with some distal gas—*but may be normal*.
- Barium meal—gold standard. Abnormal position of the duodenojejunal flexure implies malrotation (should be to the left of left pedicles of lumbar spine at level of pylorus). Duodenal obstruction and a 'corkscrew' appearance of proximal small bowel loops suggest volvulus.
- USS. Reversed relation of superior mesenteric artery and vein—a normal USS does not exclude the diagnosis.

Treatment

- Resuscitation, including decompression with NGT.
- Prompt surgery to avoid irreversible bowel damage.
- Laparotomy may reveal:
  - Obstructed, but viable, bowel.
  - Patchy ischaemic changes.
  - Established necrosis.
- Resection of ischaemic gut may risk 'short gut syndrome'.
- 'Second look' laparotomy (24–48h) may allow reassessment of questionably viable bowel prior to resection.

Further reading

Millar AJW, Rode H, Cywes S (2003). Malrotation and volvulus in infancy and childhood. *Semin Pediatr Surg* **12**: 229–36.

Intussusception

Key facts (infants)

- Incidence ~1 in 250 infants.
- Peak age of presentation at 6–9 months.
- ♂ predominance.
- Fewer than 10% have a pathological cause that starts the intussusception (more likely in older children).

Clinicopathological features

- Invagination/telescoping of the proximal bowel (called the intussusceptum, e.g. terminal ileum/ileocaecal valve) into the distal bowel (called the intussuscipiens, e.g. caecum/ascending colon).
- May be due to enlargement of lymphatic patches of Peyer ('idiopathic').
- A pathological (rather than idiopathic) lead point may be:
  - Meckel's diverticulum.
  - Polyp.
  - Lymphoma.

Clinical features

- Classic triad (seen in less than one-third) is:
  - Abdominal pain (associated with pallor, screaming, and restlessness).
  - Palpable sausage-shaped mass (mid-abdominal or RUQ).
  - Passage of 'redcurrant jelly' stool (rectal examination may reveal bloody mucus and the intussusceptum may be rarely palpable).
- Typically, the infant is quiet and lethargic between bouts of pain.
- Multiple boluses of fluid resuscitation are often needed.
- Features of obstruction (distension and vomiting) may occur.

Diagnosis and investigations

- Ultrasound (diagnostic test of choice). Intussusception in cross-section ('doughnut' or 'target' sign).
- Plain X-ray. May show soft tissue mass, small bowel obstruction, and free air indicating perforation.
- Air enema. Diagnostic and, more importantly, therapeutic (see below).

Treatment

- Immediate IV fluid resuscitation to correct fluid losses and to restore fluid, electrolyte, and acid–base balance.
- Antibiotics to cover for translocation.
- NGT for transfer with IV fluid maintenance and replacement of NG losses.
- Reduction only attempted once fluid balance restored.
- Analgesia and sedation may aid process of reduction.

Methods of reduction

Radiological reduction

- Air enema therapeutic in 75% of cases.
- Performed in radiology department under fluoroscopy.
- Surgeon should be present.
- Evidence of perforation mandates immediate halt and a compromised infant must have percutaneous decompression of a tension pneumoperitoneum via RIF.
- Partial or incomplete reduction may warrant repeat attempt after 4–6h.
- Informed consent includes risk of perforation.

Surgical reduction

- Surgery indicated without enema if evidence of peritonitis or perforation.
- Laparoscopic or open reduction practised.
- Manual reduction by squeezing both ends of intussusception.
- Resection and anastomosis if bowel viability is in doubt (10% require resection).
- Post-reduction septic shock may occur with release of bacterial products from viable, but damaged, bowel segment.
- Most recover rapidly, with resumption of oral feeding in 24–48h.

Complications

- Recurrence rate is 5–7% in non-operative cases and about 3% for operative reduction.
- Morbidity is low, but delayed diagnosis, inadequate resuscitation, and failure to recognize ischaemic or perforated bowel account for 1% mortality.

Hirschsprung's disease

Key facts (neonates/children)

- Incidence 1 in 5000 live births.
- Commoner in ♂.

Pathological features

- Due to incomplete migration of neural crest cells into the hindgut, resulting in distal aganglionosis and failure of coordinated peristaltic waves, abnormal anorectal relaxation, and loss of recto-anal inhibitory reflexes.
- May involve:
  - The rectum and recto-sigmoid 'standard segment' (75–80%)—usually presents in infancy or early childhood.
  - Extensive colonic involvement—'long segment' (15–20%).
  - Total colonic aganglionosis, with variable length of small bowel involved (5–10%).
- The aganglionic segment fails to relax and causes distal bowel obstruction with proximal distension.

Clinical features

- There is failure to pass meconium within 24–48h, abdominal distension, and bile vomiting.
- It may be associated with Down's syndrome.
- It may present late with poor weight gain, offensive diarrhoea, or enterocolitis.

Diagnosis and investigations

- AXR shows multiple distended bowel loops, consistent with distal bowel obstruction.
- PR examination may result in an explosive release of stool.
- Warm saline rectal washouts may be used to decompress the abdomen and protect against enterocolitis.
- Contrast enema shows a transition from collapsed distal to dilated proximal bowel. The level of suspected transition is not reliable.
- Suction rectal biopsy confirms the diagnosis; aganglionosis and thickened nerve fibres, with characteristic stains with acetylcholine and calretinin.
- Anorectal manometry (in older children) may show loss of recto-anal inhibitory reflexes.

Treatment

- Fluid resuscitation, broad-spectrum antibiotics, and analgesia.
- Decompression of the colon with regular saline rectal washouts.
- If decompression is not successful, a defunctioning stoma is necessary.
- Definitive surgery is to remove the aganglionic bowel and bring normally innervated bowel to the anus (pull-through technique—Soave, Swenson, or Duhamel types).
  - Surgery can be performed by a single-stage technique or with a covering stoma.
  - The pull-through can be performed transanally or abdominally.
  - Laparoscopy is sometimes used for the abdominal component.

Complications

- One-third of infants require further surgery within 30 days.
- Obstruction.
- Twisted pull-through.
- Anastomotic leak or stenosis.
- Long-term bowel function management needed with issues with constipation and incontinence.
- Enterocolitis is less common after pull-through but can affect 20–50% of children pre- and post-operatively (uncommon >5y of age, unless an obstructive component exists).

Further reading

Langer JC (2019). Hirschsprung disease. In: Holcomb III GW, Murphy JP, St Peter SD (Eds). *Holcomb and Ashcraft's Pediatric Surgery*, 7th edn. Elsevier, New York, NY; pp. 557–76.

Neonatal intestinal obstruction

Duodenal atresia

- 1 in 6000.
- Caused by failure of recanalization of the duodenum.
- May be complete (e.g. entirely separate proximal and distal duodenum) or partial (e.g. an hourglass narrowing or web obstruction in the second part of the duodenum).

Diagnostic features

- Antenatal—polyhydramnios and double bubble.
- Bile-stained vomiting or large-volume aspirate from birth.
- Epigastric fullness.
- One-third associated with Down's syndrome.
- Plain AXR. 'Double bubble' sign.

Management

- NGT, fluid resuscitation.
- Assess for cardiac anomalies.
- Surgical bypass (duodeno-duodenostomy).

Jejuno-ileal atresia

- 1 in 5000 (one-third premature).
- Caused by in utero mesenteric vascular event.
- May occur in a single or multiple segments and may be short segments or long stretches of small bowel involved.

Diagnostic features

- Presents as bowel obstruction in the neonatal period.
- The level of atresia influences the timing of bilious vomits and degree of distension.

Investigations and management

- NGT, fluid resuscitation.
- Contrast enema can differentiate from intraluminal obstruction.
- Surgical anastomosis between atretic ends.

Meconium ileus

- 1 in 10000–20 000.
- Caused by the presence of impacted, abnormally thick meconium within the normal lumen of the small bowel.
- Pathognomonic of CF in term neonates.
- Preterm infants have a similar presentation in the absence of CF.

Diagnostic features

- May be identified during antenatal ultrasound examination ('bright spots' or 'echogenic' bowel) or family history with antenatal testing.
- Presents in neonatal period with features of distal obstruction: vomiting becoming bilious, and abdominal distension.

Investigations and management

- Resuscitation, IV fluids, NGT.
- Plain AXR—distal obstruction with soap bubble appearance of impacted meconium.
- Contrast enema may be diagnostic and therapeutic.
- Surgical removal of meconium via an enterotomy (may require a temporary ileostomy).
- Confirmation of CF: sweat test, immunoreactive trypsin, and CF genes (commonest ΔF508).

Anorectal malformations

- 1 in 5000.
- Best described anatomically by location of the associated fistula.
- Commonest lesion in ♂ imperforate anus with a rectoprostatic fistula, and in girls an imperforate anus with a rectovestibular fistula.
- Malformations may be part of a VACTERL association or linked to chromosomal abnormalities.

Diagnostic features

- Should be detected at neonatal check.
- Missed more often following less use of rectal thermometer.
- Abdominal distension and vomiting can ensue.
- It may take up to 24h before meconium passes through a fistula.

Investigations and management

- Lateral prone X-ray of pelvis at 24h (assists in level assessment and sacrum).
- Perineal and renal ultrasound, and echocardiography.
- Contrast loopogram and micturating cystogram after stoma formation (position of fistula/renal anomalies).
- Prophylactic antibiotics.

Surgical treatment

- A perineal fistula may be managed with a single-stage perineal approach (anoplasty or dilatation).
- Other lesions usually managed with a defunctioning colostomy.
- Posterior sagittal anorectoplasty (PSARP). At 1–6 months and colostomy closure thereafter.

Prognosis

- Low anomalies often have a tendency to constipation in later life.
- Higher anomalies are more likely to have impaired function with need for management of soiling/incontinence.

Further reading

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Levitt M, Pena A (2010). Imperforate anus and cloacal malformations. In: Holcomb III GW, Murphy JP, St Peter SD (Eds). *Ashcraft's Pediatric Surgery*, 5th edn. Elsevier, New York, NY; pp. 468-90.

Abdominal wall defects

Exomphalos (omphalocele)

- Incidence 1 in 7000 births.

Clinicopathological features

- Herniation of the abdominal viscera through an umbilical defect that is covered by a membrane (unless ruptured).
- Exomphalos minor. The defect is <5cm and only bowel is herniated.
- Exomphalos major. The defect is >5cm and bowel, liver, and other abdominal organs lie in the hernial sac.
- May present antenatally with an abnormal scan or raised maternal serum AFP; in postnatal presentation, there is an obvious defect.

Diagnosis and investigations

- Investigations directed at identifying the associations (see Box 13.3).
- Check blood sugar.
- All newborn babies should have cardiac imaging prior to further management.

Treatment

- Parents may opt for termination in antenatally detected defects with associated major cardiac or chromosomal anomaly (mortality ~80%).
- Postnatal management involves protection of sac, insertion of NGT, IV access, and fluid management.
- Minor exomphalos should be suitable for reduction and 1° closure of umbilical defect.
- Major exomphalos may be associated with underdeveloped abdominal cavity, precluding 1° reduction. Epithelialization of the sac can be encouraged with application of topical agents, resulting in a large ventral hernia that is suitable for delayed closure at around 1y of age.

Surgical treatment

- 1° reduction of smaller defects. Excision of sac, closure of umbilical defect (linear or purse string), and closure of umbilical skin.
- Application of silo and staged reduction may be used for a larger defect, rather than conservative management as described above.

Box 13.3 Associations of exomphalos

- Chromosomal abnormality (trisomy 18, 13, 21).
- Cardiac and renal anomalies found in up to 40%.
- Pulmonary hypoplasia caused by abnormal diaphragm function.
- Beckwith–Wiedemann syndrome: exomphalos, macroglossia, gigantism, hyperinsulinism in infancy, renal/hepatic tumours.
- Pentalogy of Cantrell: exomphalos, sternal cleft, ectopia cordis, anterior diaphragmatic hernia, ventricular septal defect.

Gastroschisis

- Incidence 1 in 3000 births (increasing).

Clinicopathological features

- There is a defect to the right of the umbilicus, with protrusion of the stomach, small bowel, and large bowel.
- Associated with young maternal age and antenatal smoking or recreational drug use.
- Most present antenatally with an abnormal scan or raised maternal serum AFP.
- Antenatal diagnosis allows planned delivery (no evidence to recommend Caesarean section).
- Extraintestinal associated anomalies are uncommon.
- Intestinal atresia found in 10–20%.

Diagnosis and investigations Associated anomalies are rare. No formal investigations are required.

Treatment

Management of gastroschisis at birth

- Planned vaginal delivery as close as possible to neonatal surgical unit.
- Standard neonatal resuscitation (clean, dry, stimulate, facial O_2, etc.).
- Cling film wrap to protect herniated bowel against trauma, contamination, heat loss, drying, and fluid loss (ensure mesentery not under tension).
- Insertion of NGT to decompress stomach.
- Fluid balance must include considerable evaporative losses from gut.
- Broad-spectrum antibiotics.

Non-surgical treatment Manual reduction and non-sutured closure of defect.

Surgical treatment

- If possible, the defect is delineated and closed.
- If herniated contents are unable to be reduced, application of a 'silo' to cover gut and delayed closure once the gut is reduced (7–10 days).
- Increasingly preformed silos are used to reduce the bowel in all gastroschisis babies.

Further reading

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Necrotizing enterocolitis

Key facts

- Range of intestinal inflammation, ranging from mild mucosal injury to full-thickness necrosis and perforation.
- Perforated necrotizing enterocolitis (NEC) associated with 40% mortality in neonates.

Clinicopathological features

- Associated with:
  - Premature delivery.
  - Formula milk feeds.
  - Hypoxia.
  - Systemic sepsis.
  - 'Micro-epidemic' outbreaks in neonatal units.
- Typically affects premature babies on ventilatory support.
- Features of vomiting, distension, and bloody mucus passing PR.
- May show signs of severe sepsis/shock (tachypnoea, poor perfusion, temperature instability).

Diagnosis and investigations

- Plain AXR—pneumatosis intestinalis; portal venous gas; free gas if perforation; dilated, thick-walled (oedematous) bowel.

Treatment

Medical

- Fluid resuscitation.
- IV antibiotics.
- Bowel rest and TPN.

Surgical treatment

- Indicated by complications (perforation, failure to respond to medical treatment, abdominal mass, systemic sepsis).
- May include:
  - Peritoneal drainage.
  - Bowel resection (usually with stoma formation).

Complications

- Septicaemia.
- Enteric fistulation.
- Peritonitis.
- Adhesions.
- Enteric stricture.
- Short gut syndrome.
- Death.

Inguinal hernia and scrotal swellings

Inguinal hernia

Key facts

- Childhood inguinal hernias derive from a persistent processus vaginalis and are almost invariably indirect.
- ♂:♀, 7:1.
- Right-sided hernias (60%) are commoner than left-sided (25%); 15% are bilateral.
- Higher incidence of complications (incarceration) than adult hernias (➲ Inguinal hernia, p. 434 for adult hernias).

Clinical features

- Usually noticed as a painless swelling, variable in size in the inguinoscrotal or labial area.
- More prominent when the baby cries, and may disappear intermittently.
- Bowel entrapment causes pain and irreducibility leads to strangulation, intestinal obstruction, perforation, and peritonitis.
- Ovarian entrapment may occur in ♀.
- Bile vomiting in a young infant should always prompt examination of the inguinoscrotal area.
- Cardinal feature is a swelling in the groin above which the examining fingers cannot define the inguinal canal ('cannot get above').
- Asymmetrical thickening of the spermatic cord in the presence of a history compatible with a hernia is strongly suggestive of the diagnosis.

Treatment

- Prompt surgical treatment is important in premature/young infants to avoid risks of complications.
- Herniotomy alone is adequate. No need to repair the walls of the canal; usually a day case procedure.
- Acute surgery can be very difficult when it is irreducible or strangulated or in very young infants.

Hydrocele

- Congenital fluid-filled processus vaginalis and tunica vaginalis.
- Communicates with the peritoneal cavity in children.
- Scrotum is usually smoothly enlarged and sometimes bluish in colour, and the testis is often surrounded by the hydrocele.
- Occasionally acquired due to trauma, infection, or testicular tumour.
- Simple hydroceles may resolve spontaneously up to age 3 or even older, though this becomes less likely with age; surgical intervention is deferred till at least 2.5y.
- At operation, ligation of the patent processus vaginalis and drainage of the fluid are adequate; there is no need to excise the hydrocele wall.

Varicocele

- Due to a dilated pampiniform venous plexus of the spermatic cord.
- Onset usually after puberty.
- Has the feel of a 'bag of worms' during palpation of the cord.
- Indications for treatment include discomfort (aching), cosmesis, and concern about fertility. The procedure is carried out by ligation of the vessels, typically by laparoscopic surgery.
- Beware an acute left varicocele in childhood due to obstruction of the left renal vein by tumour (nephroblastoma).
- Treatment may be surgical ligation or radiologically guided embolization.

Idiopathic scrotal oedema

- Aetiology unknown; possibly due to an acute allergic reaction.
- Characterized by painless, red scrotal swelling which can cross the raphe and extend to the groin or perineum.
- The oedema is superficial to the testis which allows it to be palpated and confirmed to be non-tender.
- Rapidly resolves spontaneously; the clinical diagnosis precludes the need for investigation.

Other childhood hernias

Umbilical hernia

Key facts

- Persistence of the physiological umbilical defect beyond birth.
- Usually closes spontaneously (especially in premature infants).
- Has a low incidence of complications (incarceration/strangulation).

Clinical features Usually noticed as a painless, intermittent swelling at the umbilicus.

Treatment

- Delay repair until 5y of age.
- Simple sutured closure of defect in surgery required.

Epigastric hernia

Key facts

- Defect in the midline linea alba between the umbilicus and the xiphoid process.
- Very rarely closes spontaneously.
- Has a low incidence of complications (incarceration/strangulation).

Clinical features Usually noticed as a painless, intermittent swelling above the umbilicus.

Treatment Simple sutured closure of the defect may be undertaken, although some surgeons advocate conservative management.

The foreskin and circumcision

Key facts

- The variable physiology of the foreskin leads to a large number of referrals and parental anxiety.
- Phimosis, simply a non-retractile foreskin, is normal at birth and can, in some cases, persist until puberty.
- Ballooning and spraying are the result of this physiological phimosis and are not indications for surgery.
- Ballooning is a method of physiological separation of the prepuce from the glans.
- Familiarity with the range of normal healthy foreskins often allows expectant management.
- Smegmal cysts (or preputial pearls) are a common cause of anxiety as a white lump is noted under the foreskin near the corona. These resolve as the foreskin becomes retractile.

Balanitis

- Balanitis (inflammation of the glans) or balanoposthitis (inflammation of the prepuce in addition to the glans) cause discomfort and swelling and can lead to urinary retention.
- Typically, these episodes are short-lived and respond to NSAIDs; micturition can be facilitated by a warm bath.
- A similar appearance occurs with separation of the glans and prepuce; the freshly exposed surface can become inflamed for a few days—which can lead to discomfort and anxiety but is effectively managed with similar measures and topical petroleum jelly.
- Frequency and severity of episodes can be reduced by meticulous hygiene and ensuring urine is not left under the foreskin.
- Only when balanitis becomes recurrent and interferes with activities is a circumcision considered. This may reduce the frequency of episodes.
- Topical mild steroids and gentle stretching exercise can reduce the frequency and severity of episodes. A fully retractile prepuce is not the aim of treatment here.

Balanitis xerotica obliterans

- Diagnosed by recognizing a pale white scar causing a pathological phimosis.
- Treated by circumcision.
- Scarring may extend onto the glans and cause meatal stenosis. Urethral involvement, although rare, does occur.

Acute urinary retention

- This can follow on from an episode of balanitis.
- Early recognition and supportive measures can negate the need for catheterization or surgical intervention.
- Adequate analgesia, considering anxiolytics and a warm bath usually are effective if started early.
- Constipation is a common cause.

Paraphimosis

- This occurs after a forcible retraction of the prepuce which is left retracted whilst still tight.
- The tight ring acts as tourniquet, leading to progressive oedema.
- Prompt recognition and reduction prevent worsening oedema.
- When this is impossible with an awake or sedated child, it usually is achievable under anaesthesia.
- Rarely, a dorsal slit is needed to release the tourniquet effect.
- Circumcision should not be performed in the context of gross oedema.
- Often when seen in clinic after a few weeks, the appearance is entirely normal and no surgical intervention is required.

How to examine a child's foreskin

- Try to ensure the boy is happy and relaxed, lying on the examination couch or parental knee.
- Normal foreskin often appears long and 'redundant'.
- Asking the child to retract his own foreskin often demonstrates enough of the prepuce to make hands-on examination redundant.
- Do not forcibly retract the foreskin, as it will cause distress and may cause trauma.
- If retraction attempted, perform gently to show pouting of inner pseudo-mucosa.
- Blanching of skin below preputial opening—normal.
- Tight, white, contracted preputial orifice indicates fibrotic phimosis ('muzzling').

Further reading

Malone P, Steinbrecher H (2007). Medical aspects of male circumcision. *BMJ* **335**: 1206–9.

Undescended testis

Key facts

- Testicular descent from the fetal abdominal site into the scrotum is normally complete by birth.
- Absence of a scrotal testis (cryptorchidism) usually results from arrested descent (superficial inguinal pouch, within canal, intra-abdominal). It can be due to an ectopic testis (in the lower part of the abdomen, front of the thigh, femoral canal, skin of the penis, or behind the scrotum) or may be due to agenesis (rare).
- Incidence 4% of newborn term boys, falling to 1% at 3 months.
- Commoner on the right side.

Clinical features

- Undescended testis can be noted at the postnatal check, by parents, or by the GP.
- Rarely presents acutely as torsion (tender mass in inguinal region).
- A retractile testis is one that can be brought down into the scrotum with gentle manipulation and stays there without tension, but retracts into the groin because of the cremasteric reflex (see Box 13.4).

Diagnosis and investigations

- No investigations are required in palpable undescended testis.
- Chromosomal studies and HCG stimulation test may be requested in bilateral impalpable testes.
- Diagnostic laparoscopy is definitive and allows further management.

Treatment

- Testis should be brought to the scrotum at 6 months to 1y of age to avoid 2° damage due to trauma, torsion, and ↑ ambient temperature.
- Hormone manipulation is ineffective in true undescended testis.
- Intracanalicular or ectopic testis should be managed by one-stage orchidopexy.
- Intra-abdominal testis can be brought down by one- or two-stage laparoscopic orchidopexy (50–90% success).
- Laparoscopy for bilateral impalpable testes, which should prompt consideration of a disorder of sex development (DSD).
- Scrotal position facilitates self-examination to detect signs of neoplastic change.

Complications

- Post-operative atrophy of the testis (<2%) unless intra-abdominal position (10–50%).
- Retraction.

Box 13.4 How to exclude retractile testis

- In a warm room …
- A cooperative, relaxed little boy is essential. Examine on carer's knee or lying down.
- Control inguinal canal with finger pressure (prevents retraction of testis).
- Palpate tissues superficial to external inguinal ring, working down to scrotum.
- Try to manipulate testis into scrotum—then release.
- True retractile testis should remain in scrotum briefly; a testis which pings out of the scrotum or can only get there under tension requires intervention.

Solid tumours of childhood

Neuroblastoma

- Commonest solid abdominal tumour of childhood.
- Spectrum of tumours derived from neuroblasts found in the adrenal gland, along the sympathetic chain, or extra-adrenal sympathetic tissues.
- Aggressive tumour with early spread to lymph glands, liver, bone (cortex or marrow), orbits, and skin.
- Presents as painless, large abdominal mass in children <2y.
- May present as weight loss, hypertension, or metastatic disease.
- Urinary vanillylmandelic acid (VMA) and homovanillic acid (HVA) elevated.
- CT scan. Optimal investigation for suspected neuroblastoma.
- Treatment by combination of chemotherapy, surgery, and radiotherapy.
- Survival of between 30% and 90%, depending on the site and stage at presentation.

Nephroblastoma (Wilms' tumour) of the kidney

- Fast-growing tumour of the kidney.
- Ranges from benign mesoblastic nephroma of infancy to poorly differentiated, malignant nephroblastoma in the older child.
- Malignant tumours frequently metastasize to regional lymph nodes, liver, and lungs.
- Usually presents as a large, relatively painless abdominal mass in an otherwise well child.
- Treatment by combination of chemotherapy, surgery, and radiotherapy, according to histology and spread at diagnosis.
- 5y survival:
  - Early stage, 90%.
  - Disseminated disease, 30%.

Rhabdomyosarcoma

- Tumour of striated muscle origin from the bladder, vagina, prostate, parameningeal tissue, and limbs.
- Haematuria, vaginal bleeding, and appearance of grape-like cysts (sarcoma botryoides) at the vaginal introitus.
- Variable histology (embryonal is most favourable), which determines the prognosis.
- Survival of up to 70% from surgery and chemotherapy.

Hepatoblastoma

- This presents as a right hypochondrial mass extending across the midline.
- Chemotherapy may render initially inoperable tumours resectable.
- Depending on staging, size, and histology, survival of up to 70% is possible.

Neck swellings

Key facts

- Childhood neck lumps may be due to embryological abnormalities, as well as the same spectrum of conditions in adults (➲ Neck swellings, p. 293).
- Embryological abnormalities may relate to:
  - Descent of the thyroid from the foramen caecum of the tongue—thyroglossal cysts (➲ Thyroglossal cyst, p. 294).
  - Formation of second, third, and fourth branchial arches and clefts—branchial cysts (➲ Branchial cyst, p. 296).
  - Formation of lymphatic vessels and veins—cystic hygroma and cavernous haemangiomata.
- Lymphadenopathy is very common in children but typically 'waxes and wanes'.
- If lymphadenopathy persists for longer than 2 months and measures >2cm diameter, it should be biopsied.

Causes and clinicopathological features

Thyroglossal cyst (➲ Thyroglossal cyst, p. 294).

Branchial cysts (➲ Branchial cyst, p. 296).

Lymphadenopathy

- The neck contains large numbers of lymph glands draining areas of potential infection in the mouth, nose, tonsils, and ears.
- Common causes of lymphadenopathy include upper respiratory tract infection, middle ear infections, tonsillitis, parotitis, dental abscess, and atypical mycobacterial infection.
- Malignant lymphadenopathy is much less common.
  - May be 1° lymphoma.
  - 2° deposits, e.g. from neuroblastoma.

Salivary gland swellings

- May be due to duct obstruction (stones or duct stenosis), infection (mumps), autoimmune disorders (recurrent parotitis), and neoplasia (adenoma).
- Commonest in the submandibular, sublingual, and parotid glands.

Skin lesions

- Dermoid cysts. Usually in the midline above the hyoid bone and are rarely infected. Also seen in the outer eyebrow (external angular).
- Sebaceous cysts. Rare in children. Epidermal origin with a small central punctum; may occur anywhere, but most commonly on the scalp or back of the neck.

Lymphovascular lesions

- Haemangiomas. Can be mixed capillary or cavernous haemangiomas or haemangioendotheliomas within the neck and parotid area; may grow rapidly in size and lead to high-output cardiac failure or even carotid steal syndrome.
- Lymphatic vascular malformation (cystic hygroma or lymphangioma). Commonly in the posterior triangle of the neck.

Treatment

- Excision of dermoid cysts, sebaceous cysts, thyroglossal cysts, thyroid neoplasms, salivary gland enlargements, and lymph gland enlargements (biopsy).
- Haemangioma. Usually managed conservatively unless symptomatic. Airway involvement may require aggressive treatment.
- Lymphatic vascular malformation. Sclerosant injection is effective in lesions with few large cysts.

Differential diagnosis of neck lump

Neck lumps may be lateral or midline.

Lateral

- Lymph node
- Branchial sinuses and cyst
- Cystic hygroma
- Sternomastoid tumour
- Haemangioma
- Lymphangioma
- Submandibular gland
- Parotid gland

Midline

- Submental lymph nodes
- Thyroglossal cyst
- Thyroid swelling
- Dermoid cyst

Neck swellings by cause

Congenital

- Thyroglossal cyst
- Branchial cyst
- Cystic hygroma
- Haemangioma
- Dermoid cyst

Acquired

- Reactive lymphadenopathy
- Infective lymphadenopathy
- 2° tumour deposits

Chapter 14

Paediatric orthopaedic surgery

Childhood growth and the physis

Children's bones have a number of unique properties. They are covered in a very thick periosteum that provides O_2 and nutrients to the bone, allowing for rapid growth and repair. The periosteum also contributes to the overall strength of the bone, which has greater elasticity than adult bone, allowing it to bend. The immature skeleton has an extensive potential to remodel its shape and orientation, should a deformity occur.

The growth of the paediatric skeleton is driven by a specialized area typically found at the ends of bones called physes (growth plates) (see Fig. 14.1). On a radiograph, this region appears radiolucent and can sometimes be mistaken for a fracture. Whilst the physis remains 'open', the bone can continue to grow and remodel.

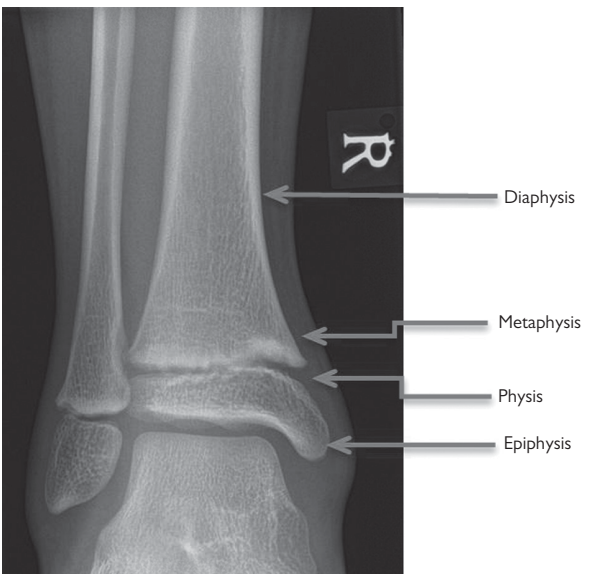

Fig. 14.1 Anteroposterior (AP) radiograph of a skeletally immature right ankle.
Reproduced courtesy of Dr Jeremy Jones, Royal Hospital for Sick Children Edinburgh.

Remodelling potential is greatest when the deformity is close to a joint (i.e. the physis) and angulated in the plane of motion of that joint. For example, deformities affecting the distal radius and ankle have the greatest potential to remodel when in the sagittal plane (flexion/extension).

Management of paediatric fractures

Fractures in the paediatric population are common, affecting ~40% of boys and 25% of girls prior to 16y of age. Boys are almost twice as likely as girls to sustain a fracture. The majority of fractures are minor, with almost half affecting the forearm 2° to a fall onto an outstretched hand. There are seasonal variations in the frequency of childhood fractures, with a rise in injuries seen during the summer months due to an increase in outdoor play.

When a child presents with a fracture, acquisition and documentation of a clear history and thorough examination are vital, giving due consideration to a number of key features.

History

- Mechanism of injury (low versus high energy).
- Time of injury and last meal (milk counts as a meal).
- Previous history of fracture (consider differential diagnoses).
- Past medical history.
- Allergy status.
- Social circumstances.
- Handedness (if fracture affects upper limb).

Examination

Most children with a fracture will be distressed and scared. It is vital to earn their trust before beginning an examination. It is good practice to sit or kneel so that you are at eye level. Try to create a calm environment and interact with them as much as possible. If they are old enough, explain what you are about to do before doing so and warn them if something will hurt. Always examine the uninjured side first to gauge their response and gain their confidence.

- *Localization*. Children will often complain of pain and be visibly distressed. However, the younger the child, the less able they are to localize their injury. In a young child, the only sign of injury may be a limp, refusal to weight-bear, or pseudoparalysis of the injured limb.
- *External signs*. Deformity of the fractured limb may be evident. Look for wounds (open fractures), abrasions, swelling, and bruising (not a constant feature due to the thick subcuticular fat layer). Remember to actively examine the whole child for associated injuries or evidence of non-accidental injury (NAI).
- *Neurovascular status*. Check and clearly document the neurovascular status of the limb. Assess for the presence/absence of distal pulses and capillary refill times. Examine distal neurology for power and sensation. This can be difficult to determine in the child. Box 14.1 shows a number of tips to use for the assessment of upper limb neurology. Demonstrate these so the child can copy you.

Box 14.1 Tips for assessment of upper limb neurology

- **ROCK** (*clenched fist*): median nerve.
- **PAPER** (*finger extension*): radial nerve.
- **SCISSORS** (*finger abduction*): ulnar nerve.
- **OK sign**: anterior interosseous nerve.

- *Analgesia*. Provide systemic analgesia prior to splinting the limb or obtaining imaging. Application of a form of splint will improve the pain and minimize bleeding.
- *Investigations*. Radiographs of the affected limb should be undertaken to adequately determine and document the pattern of injury. The joint above and below the zone of injury should be included, and two orthogonal views taken. 'Comparison' views of the unaffected side may be used occasionally when there is radiological uncertainty, but this should be discussed with a senior team member first. A sparing approach to the number of radiographs obtained should be adopted to minimize radiation exposure.

Types of fracture

- As children's bones are more elastic and have a thick periosteum and open physes, a number of specific fracture patterns can occur.
- The thick periosteal sleeve can act to hold the fractured bone ends in approximation and the degree of displacement can therefore be less than that seen in an adult.
- *Greenstick fracture*. This is where one cortex of the bone (convex side) breaks and the other (concave side) bends (plastic deformation) (see Fig. 14.2a). It is akin to trying to snap the young green branch of a tree. These are inherently stable injuries.
  - *Treatment*. Undisplaced stable fractures: cast for 3–4 weeks.
- *Buckle/torus fracture*. This is where one or both cortices have undergone a compressive force, crushing down or buckling the cortex (see Fig. 14.2b). It is akin to crushing an empty aluminium drink can. These are inherently stable injuries.
  - *Treatment*. Undisplaced stable fractures: splint/cast for 3 weeks.
- *Complete fracture*. This is where both cortices of the bone have been fractured through (see Fig. 14.2c). These injuries can be unstable and displacement can occur, particularly if the periosteum is torn.
  - *Treatment*. Cast immobilization: 4–6 weeks. Inclusion of the joint above and below the fracture is often required to assist in controlling movement and rotation at the fracture site. Check radiographs at 1 week in fracture clinic are required to ensure no early displacement.
- *Toddler's fracture*. Also known as childhood accidental spiral tibial (CAST) fractures—undisplaced or minimally displaced spiral fractures of the distal tibial diaphysis. They typically occur in a child of walking age <3y. They can be difficult to diagnose as the radiological features can be subtle. In equivocal cases, repeat radiographs are taken at 1–2 weeks to assess for callus formation.
  - *Treatment*. Above-knee cast for 3–4 weeks.
- *Fractures involving the physis*. The physis is susceptible to injury and is often involved in paediatric fractures. There are a number of patterns of injury in which the physis can be affected, as described by the Salter–Harris classification system (see Fig. 14.3).
  - *Treatment*. Undisplaced stable fractures: cast for 3–4 weeks.

When damage to the physis occurs, disturbances in normal growth can sometimes result. This is known as growth arrest. When growth arrest is complete, the limb overall will be shorter. When growth arrest is partial, angular deformity will result as the unaffected side of the physis continues to grow.

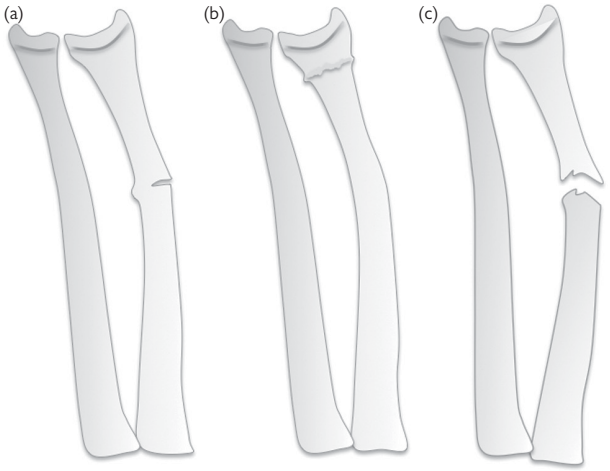

Fig. 14.2 Illustration of (a) greenstick, (b) buckle, and (c) complete fracture.

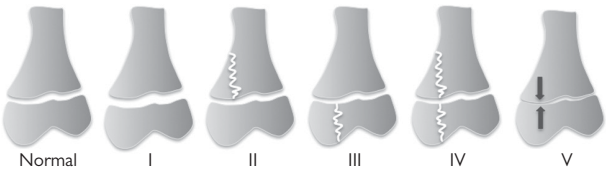

Fig. 14.3 Salter–Harris classification of physeal fractures. I, through the physis—the physis may appear widened. II, through the physis into the metaphysis. III, through the physis into the epiphysis. IV, through the metaphysis, physis, and epiphysis. V, crush injury to the physis.

Accepted parameters

There are a number of accepted parameters with regard to angulation, displacement, and joint involvement of any fracture. The amount of deformity accepted is dependent on the bone fractured, site of fracture, and age of the child. However, as a rule of thumb:

- *Angulation*. Best tolerated in the plane of motion of the closest joint. Acceptable remodelling of the bone will occur with: 15° of angulation in a child <6y old and 10° of angulation in a child 6–12y old. Angulation is unacceptable in children >12y with <2y of growth remaining.
- *Bayonetting*. This is the amount of overlap (or shortening displacement) of the bones; <1cm is acceptable in a child <12y old.
- *Rotation*. Rotation is usually not acceptable.

- *Joint involvement*. <2mm of displacement at the joint surface may be accepted. However, the greater the displacement in the articular surface, the greater the predisposition to traumatic arthritis in later years.

If the fracture is displaced or angulated beyond acceptable limits, manipulation or percutaneous/open reduction and fixation under GA may be required.

Box 14.2 Safety rules for cast application

- Ensure casting material does not contact the skin to avoid iatrogenic burns.
- Use a maximum of seven layers cast of cast material.
- After application, do not place on a pillow as this increases cast temperature.
- Water temperature <23°C (tepid to the ungloved hand).
- Ensure cast is free from sharp indentations which could cause pressure sores/necrosis.
- Ensure cast edges are smooth.
- Ensure cast is not applied too tightly to allow for swelling.
- Do not overlay with resin until the cast in completely dry.

Consideration in cast application

Whether paediatric fractures are treated conservatively or operatively, casts are often applied. Although they appear innocuous, serious complications, such as iatrogenic burns, are a risk. To help prevent these risks, a number of cast rules should be followed (see Box 14.2).

Non-accidental injury

Abuse in the broadest term is any form of maltreatment of a child. NAI is the infliction of physical harm upon a child. It is part of a spectrum of abuse that includes neglect, physical, sexual, and psychological subtypes where neglect is the commonest form. These types of abuse may occur in isolation or in combination. In 2016, The National Society for the Prevention of Cruelty to Children (NSPCC) determined that 58 000 children were at risk of abuse in the UK. However, this is likely to be an underestimation as many cases remain unreported.

In children, physical abuse often presents with fractures and soft tissue manifestations, including bites, burns, and bruising. If undetected, ongoing abuse can lead to physical and intellectual developmental delay, social and emotional withdrawal, and even fatality. NAI is the second commonest cause of childhood death after accidental injury.

Children at risk

It is essential to appreciate that NAI can affect any child, regardless of their age, sex, cultural and socio-economic background, or how happy and well they may first appear. A high index of suspicion should always be maintained. However, certain cohorts of children remain at greater risk:

- Lower socio-economic class.
- Recent loss of parental employment.
- Children with disabilities.
- Stepchildren.
- Unplanned pregnancy.
- Preterm children.
- Twins.
- <2y of age (80% of non-accidental fractures are in children <18 months).

Concerning features in the history

- Inexplicable delay in seeking medical attention.
- Frequent attendances at the emergency department.
- Changing or inconsistent history.
- Pattern of injury incompatible with history or age of child.
- Past medical history of multiple soft tissue or bony injuries.
- Failure to thrive or meet developmental milestones.
- Past involvement of social services.

Concerning features on examination

- Poor child–parent/guardian bonding.
- Child may appear withdrawn or display unusual behaviour.
- Multiple bruises—torso, ears, neck, back, or genitalia. Look for patterns of bruising, including fingerprints and clustering and bruising of differing ages.
- Bites, burns, and scratches.
- Multiple fractures—especially if of different stages of healing. Up to 74% of abused children have two or more fractures, in comparison to only 16% in accidental trauma.

Concerning features on imaging

- Unusual fracture patterns (see Box 14.3).
- Metaphyseal corner and bucket handle fractures represent shear injuries, usually in children <2y, caused by vigorous shaking of the child or forceful twisting of a limb (see Fig. 14.4). A flake of bone is seen at the metaphyseal flare. Although considered pathognomonic, diaphyseal fractures are four times commoner in NAI.
- Sometimes the only radiological sign of NAI may be a periosteal reaction from the pressure of a strong adult grip.

Box 14.3 Radiological features suggestive of NAI

- Metaphyseal corner fractures
- Bucket handle fractures
- Posterior rib fractures
- Transverse long bone fractures
- Scapular fractures
- Vertebral fractures/subluxation
- Complex skull fractures
- Lateral clavicle fractures
- Digital fractures
- Multiple fractures—varying age

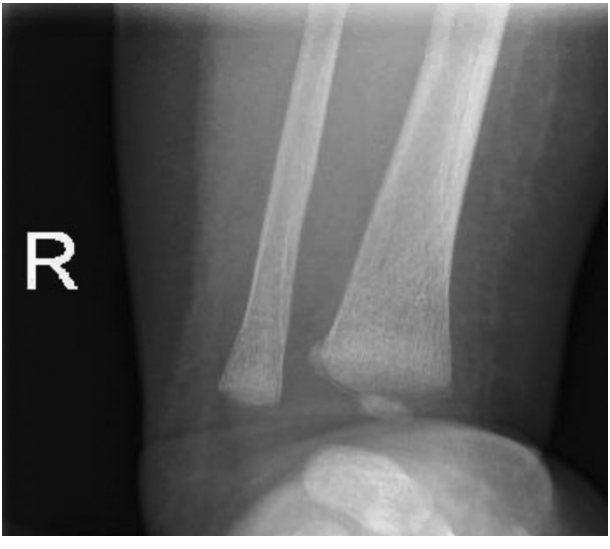

Fig. 14.4 Radiograph of right distal tibia showing a bucket handle fracture.

Differential diagnosis

Certain medical conditions can mimic the features of NAI. These include osteogenesis imperfecta, scurvy, idiopathic juvenile osteoporosis, osteopenia of prematurity, haemophilia, malignancy, and true accidental injury and should be ruled out.

Management

The main priority is the safety of the child. Any doubt regarding the child's well-being should prompt action to protect the child. Each unit will have their own protocol, but a number of universal principles are listed below:

- Safeguard the child against immediate danger. This may range from simply ensuring that there is a chaperone with the child at all times to physically removing the child away from the parent/guardian. Briefly ascertain if there are any other children in the family who could also be in danger.
- Discuss the case with the most senior member of the team.
- Referral to the child protection team is required.
- Arrange for hospital admission so that the case can be investigated further, even if there is no medical reason for the admission.

The paediatric child protection team will undertake:

- A full examination of the child and document any injuries found.
- Radiographic skeletal survey (skull, chest, pelvis, and limbs).
- Blood tests (FBC, U&Es, clotting screen, bone profile, Cu, caeruloplasmin, Mg^{2+}, 25-hydroxyvitamin D, and PTH).

A knowledge of normal child development will assist in recognizing when an injury may not be consistent with the history given. As a child's nervous system matures, predictable developmental motor milestones are reached. These advance from head to toe (cephalocaudal), so the older the child becomes, the more distal the developmental achievement (see Fig. 14.5).

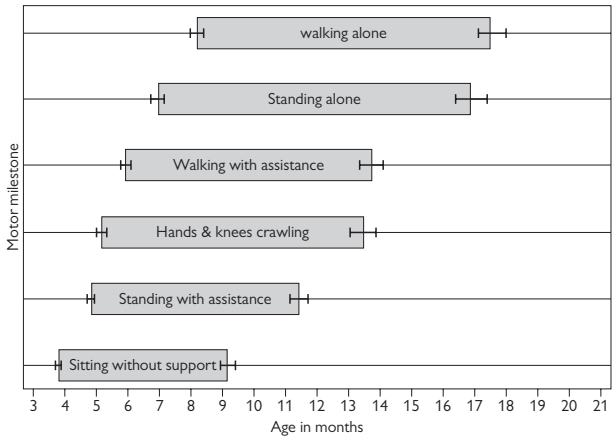

Fig. 14.5 WHO Multicentre Growth Reference Study Group (2006).
WHO Motor Development Study: Windows of Achievement for Six Gross Motor Development Milestones. Acta Paediatrica Supplement 450: 86–95.

The limping child

The limping child may represent anything from an innocuous soft tissue injury to a serious life-threatening infection or malignancy. As such, the child with a limp requires prompt assessment and a high index of suspicion.

History

It is important to document:

- The affected side.
- If the child has always limped.
- When it started and what the child was doing at the time.
- If there is a history of trauma.
- Any exacerbating or relieving features.
- Any red flags—night pain, night sweats, weight loss, back pain.
- Any systemic features—malaise, pyrexia, vomiting, dry nappies.
- Recent illness, including respiratory tract infections and chickenpox.
- Past medical history, including birth history and endocrine disorders.
- Family history of musculoskeletal complaints.
- Social history, including recent foreign travel.

A thorough history, coupled with the child's age, will narrow down the list of differential diagnoses (see Table 14.1). It is important, however, to appreciate that what causes an older child to limp will often cause a younger child to refuse to weight-bear.

Table 14.1 Differential diagnoses of the limping child

• Young • 0–3y	• Child • 4–10y	• Adolescent • 11–16y	All ages
• DDH • Juvenile rheumatoid arthritis • Toddler's fracture • Limb length discrepancy • Haemophilia	• Perthes • Juvenile rheumatoid arthritis • Haemophilia	• SUFE • Overuse syndrome • Osteo chondritis dissecans • Gonococcal septic arthritis	• *Infection*: • Septic arthritis • Osteomyelitis • Discitis • Psoas abscess • Reactive arthritis • Fracture/soft tissue injury • NAI • Malignancy • Neuro muscular

DDH, developmental dysplasia of the hip; SUFE, slipped upper femoral epiphysis.

Examination

- Does the child look well? Look for evidence of systemic sepsis by assessing vital signs, including temperature.
- Assess the child walking and decide if a limp is present.

  - Antalgic: less time spent on the affected side due to pain.
  - Trendelenburg: pelvis dips and torso moves over the affected limb due to weak hip abductors—developmental dysplasia of the hip (DDH), Perthes, and slipped upper femoral epiphysis (SUFE).
  - Circumduction: excessive hip abduction in leg length discrepancy.
- Inspect the lower limbs and spine. Look for skin changes, swelling, asymmetry, and posture. A flexed, abducted, and externally rotated hip is the position of comfort in hip effusion or sepsis.
- Feel for tenderness, crepitus, and temperature.
- Examine the lower limb joints for range of motion and pain. Examine the unaffected side first to gauge what is normal. Examine the joint above and below the affected joint. Common mistakes include missing hip pathology in a child presenting with knee pain. Foot pathology is also overlooked and can originate from the spine or brain.
- Measure limb lengths (true/apparent). *If discrepancy is found, determine if this lies within the femur or tibia by flexing both hips and knees, keeping the ankles together (Galeazzi sign)* (see Fig. 14.6).

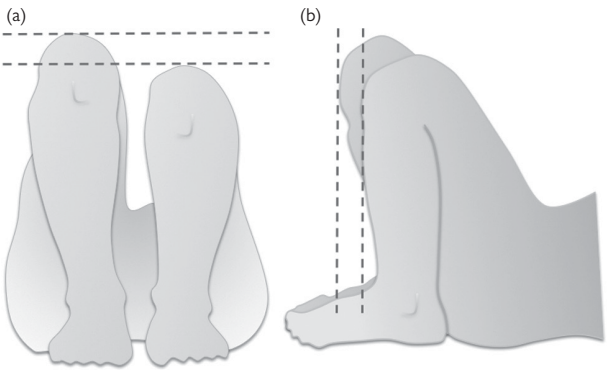

Fig. 14.6 Galeazzi sign demonstrating (a) tibial shortening and (b) femoral shortening.

Investigations

- Blood tests: FBC, U&Es, LFTs, CRP, erythrocyte sedimentation rate (ESR), and blood cultures.
- Radiographs: image the bone/joint involved and consider imaging the joint above and below if no abnormality is found. In the younger patient, it is often necessary to image the whole limb.
- Ultrasound: can be used to detect joint effusion.
- MRI: helpful in diagnostic uncertainty but may require GA.

Management

- Dependent on diagnosis. If diagnostic uncertainty exists, have a low threshold to admit the child for observation.

Developmental dysplasia of the hip

DDH is defined as the abnormal development of the hip 2° to capsular laxity and mechanical influences. DDH covers a wide spectrum of abnormalities, from a mildly underdeveloped, but stable, hip through to an irreducible dislocation or acetabular dysplasia. The incidence is 1–2/1000 newborns in the UK. Both hips can be affected, but it is usually unilateral. Subluxation or dislocation of the hip can occur pre-, peri-, or postnatally, meaning it is not exclusively a congenital condition. Detection within the first 6 weeks of life is important in order to initiate treatment and improve long-term outcomes.

Risk factors

- ♀ (6:1).
- First born.
- Breech presentation after 35 weeks of gestation.
- Oligohydramnios.
- Family history of affected first-degree relative.
- Foot abnormalities, including calcaneovalgus or metatarsus adductus.
- Moulded appearance of newborn.

Clinical features

- Affected neonates may lie with hips in extended and/or adducted position. Affected hips have reduced abduction when tested with hips flexed at 90°. The Ortolani/Barlow tests of hip stability are unreliable in inexperienced hands and commonly provide a false negative result in infants older than 6 weeks.
- The affected leg may be short, but this can be subtle. It is best assessed with the hips flexed to 90°, allowing comparison of knee height (Galeazzi sign) (see Fig. 14.6).

Screening

All newborns should have their hips examined by trained examiners as part of the newborn physical examination. Hips thought to be abnormal on examination or those with risk factors should be secondarily screened by expert examiners or with hip ultrasonography.

Radiology

- Ultrasound. Investigation of choice for children <1y of age. Ultrasound can show if the femoral head is located and centred in the acetabulum and provides information on the slope of the acetabular roof (alpha angle).
- Radiographs. Used in children >1y of age (see Fig. 14.7).

Treatment

- *0–3 months of age*. Most can be treated with an abduction harness (e.g. Pavlik harness), which holds the hips in flexion and abduction, seating the femoral heads in the (acetabula). Improper fitting of an abduction harness can result in avascular necrosis (AVN) of the femoral head, femoral nerve palsy, or loss of hip position. In expert hands, abduction harnesses result in successful hip development in 90% of cases.

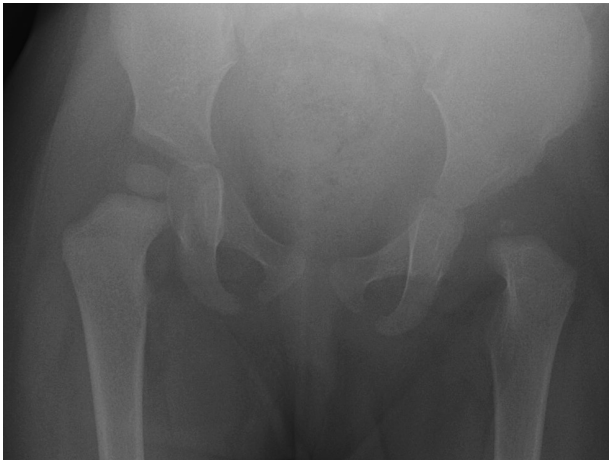

Fig. 14.7 AP pelvic radiograph showing DDH of the right hip.
Reproduced courtesy of Dr Jeremy Jones, Royal Hospital for Sick Children Edinburgh.

- *6–18 months*. Children in this age group commonly require closed or open surgical relocation of the hip under GA. This is followed by a prolonged period in a hip spica cast (from chest to ankles) for 12–18 weeks. Femoral osteotomy may also be required to reduce tension on the relocated femoral head which can cause AVN.
- *≤18 months*. Most of these children will require open surgical hip relocation with femoral and acetabular osteotomies to improve stability. A spica cast is then used for 12–18 weeks.

The best results are obtained when affected children are identified whilst abduction harnessing is still possible. The long-term outcomes deteriorate significantly once surgical treatment is required.

Further reading

Paton RW, Srinivasan MS, Shah B, *et al*. (1999). Ultrasound screening for hips at risk in developmental dysplasia: is it worth it? *J Bone Joint Surg Br* **81**: 255–8.

Legg–Calvé–Perthes disease

Legg–Calvé–Perthes disease, more commonly known as Perthes, is an idiopathic AVN of the immature femoral head. It affects around 1 in 10 000 children, most frequently between the ages of 4 and 10y. Boys have a greater predisposition to the disease (5:1) and those in lower socio-economic classes are more often affected. Disruption in the vascular supply to the femoral head, in combination with micro-trauma, leads to resorption and collapse, changing the sphericity and congruency of the joint.

Risk factors

- Family history of Legg–Calvé–Perthes disease.
- Low birthweight.
- Passive smoking.
- Attention-deficit/hyperactivity disorder (ADHD).

Clinical features

- Limp: usually antalgic but can occasionally be painless.
- Pain: groin, hip, thigh, or knee.
- Reduced range of motion (internal rotation and abduction).
- Absence of systemic features.
- Limb length discrepancy: late sign.

Imaging

The diagnosis is made using plain radiographs of the hip. A frog lateral view may show the earliest changes. Medial joint space widening and ↑ density of the epiphysis are followed by a lucent line within the outer margin, representing a subchondral fracture (crescent sign). The epiphysis subsequently collapses anterolaterally and loses height. Cysts may be seen just proximal to the femoral physis. If the radiographic features are subtle, MRI may be used to aid diagnosis.

Waldenström described four stages of Legg–Calvé–Perthes disease:

1. *Initial*. Infarction and medial widening.
2. *Fragmentation*. Femoral head collapses.
3. *Reossification*. Ossific nucleus reossifies.
4. *Remodelling*. Femoral head remodels.

Classification

Herring's lateral pillar classification is described to assist in directing treatment (see Fig. 14.8). It is based on the appearance of the femoral epiphysis on radiograph imaging during the fragmentation stage.

- A: no loss of height of lateral pillar, compared with opposite hip.
- B: <50% loss of height of lateral pillar.
- C: >50% loss of height of lateral pillar.

Management

- *<7y old*: pain relief, activity restriction, and physiotherapy—maintain range of hip motion.
- *>7y old and Herring A/C*: pain relief and physiotherapy—maintain range of hip motion.
- *>7y old and Herring B*: prompt surgical management (femoral and/or pelvis osteotomy) to improve containment of the femoral head.

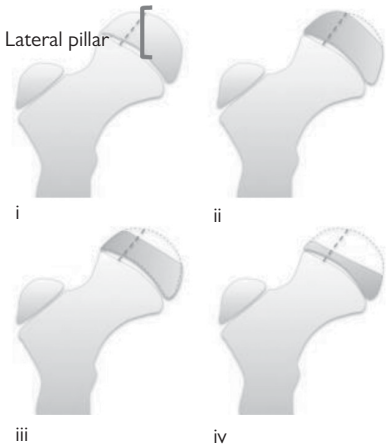

Fig. 14.8 Herring's classification showing (a) normal hip, (b) Herring A, (c) Herring B, and (d) Herring C.

Prognosis

- Acute pain and stiffness variable over 2–3y.
- Long-term outcome depends on the sphericity of the femoral head after healing and how well it is matched to the acetabulum.
- *<7y of age and Herring A*: 90% excellent outcome.
- *Age of presentation >7y (especially ♀) and Herring B*: poorer outcomes with persistent stiffness and pain.
- *Herring C*: universally poor outcomes, regardless of age.

Further reading

Annamalai SKM, Buckingham R, Cashman J (2007). Perthes disease: a survey of management amongst members of the British Society for Children's Orthopaedic Surgery (BSCOS). *J Child Orthop* ***1***: 107–13.

Slipped upper femoral epiphysis

This is an orthopaedic emergency requiring immediate admission for treatment. SUFE has an incidence of ~8–10 per 100 000 population. It occurs when there is failure within the hypertrophic zone of the physis, allowing abnormal movement between the epiphysis and the metaphysis. This can result in a minor slip of a few degrees to complete dissociation. Boys are affected more than girls (3:2) and it usually occurs in children aged 10–14y, but other age groups can be affected.

Risk factors

- Obesity (single greatest risk factor).
- Rapid growth.
- Femoral retroversion.
- Endocrinopathies—these include hypothyroidism, hypogonadism, and pituitary disorders.
- Renal disease.
- Radiotherapy in proximity to the hip.

Classification

SUFE is classified according to the ability of the child to weight-bear.

- *Stable*: child able to weight-bear. The slip is deemed stable, even if a limp is present and crutches are required to mobilize.
- *Unstable*: child cannot weight-bear, even with crutches.

SUFE can also be classified according to the degree of slip measured on frog lateral radiographs of the pelvis (Southwick angle):

- *Mild*: <30°.
- *Moderate*: 30–50°.
- *Severe*: >50°.

Clinical features

- Symptoms may be mild or prolonged over several weeks or very sudden following minor trauma.
- Often presents as thigh or knee pain. It is important to always examine the ipsilateral hip in a child complaining of knee discomfort.
- The leg may appear short and externally rotated. Comparison of foot position when supine will help identify rotational differences.
- Gentle rotation of the leg is often painful.
- Passive hip abduction is often reduced.

Radiology

- *Frog lateral radiograph of the hips*. AP pelvic radiographs can miss a subtle SUFE.
- *AP radiograph of the pelvis*. A line drawn along the superior border of the femoral neck should intersect the epiphysis on a normal hip (Klein's line) (see Fig. 14.9).

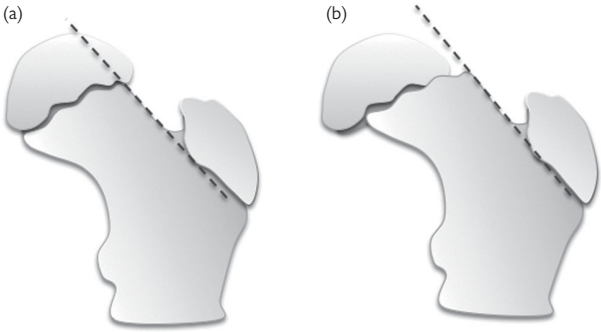

Fig. 14.9 Normal (a) and abnormal (b) Klein's lines.

Management

A child with SUFE should be admitted immediately for orthopaedic management.

- *Mild/moderate slip*. Stabilization is achieved without attempted reduction and a screw is passed across the epiphysis to prevent further slip. Some remodelling of deformity up to 50° will take place. Any residual deformity following skeletal maturity can be addressed with further operative procedures. Attempted reduction at initial presentation runs the risk of AVN of the femoral head and is a devastating complication.
- *Severe slip*. A slip of >50° often necessitates complex open reduction of the slip and carries a risk of AVN.

Prophylactic fixation

There is a risk of 30–60% that the contralateral hip may slip in the future. Many surgeons advocate prophylactic screw fixation of the opposite hip to prevent this from happening.

The acute paediatric knee

Knee pain is a common presentation in children and adolescents, with many differential diagnoses, of which infection and malignancy must be first ruled out. Paediatric knee pain is often attributable to trauma or congenital or growth-related pathology

Patella sleeve fracture

- Fracture between the cartilaginous sleeve and body of the patella.
- Common in children 8–12y of age.
- Caused by contraction of the quadriceps when the knee is flexed.
- Can be easily missed.

Clinical presentation

Pain, limp, haemarthrosis, and difficulty in active extension. If the sleeve is completely torn distally, a palpable gap may be felt at the inferior pole of the patella, which will be riding high (patella alta). If torn proximally, a proximal gap and patella baja can be found.

Investigations

- Radiographs may demonstrate a small flake of bone at the inferior pole of the patella. Patella alta/baja can be seen (Fig. 14.10).
- Ultrasound and MRI used when diagnosis equivocal.

Treatment

- Cylinder cast for 6 weeks: undisplaced and extensor mechanism intact.
- Surgical repair: displacement >2mm and extensor mechanism in discontinuity. The majority of injuries require operative repair.

Osgood–Schlatter's disease

- Osgood–Schlatter's disease is an apophysitis of the tibial tubercle resulting from traction forces from the patella tendon.
- Common in children 8–15y of age; boys > girls.
- Bilateral in up to 30%.
- Power-burst sports increase the risk, e.g. sprinting and basketball.

Clinical presentation

- Anterior knee pain that is worse on jumping, kneeling (direct pressure), and resisted knee extension. The tibial tubercle may be tender to palpate and is often enlarged.

Investigations

- Radiographs may demonstrate irregularity of the tibial apophysis with fragmentation (which can be also seen in asymptomatic knees).

Treatment

- *Conservative*: activity modification, rest, ice, NSAIDs, and physiotherapy. Spontaneous resolution common in 91% of children.
- *Operative*: excision of ossicle when skeletal maturity reached for those children who do not respond to conservative treatment.

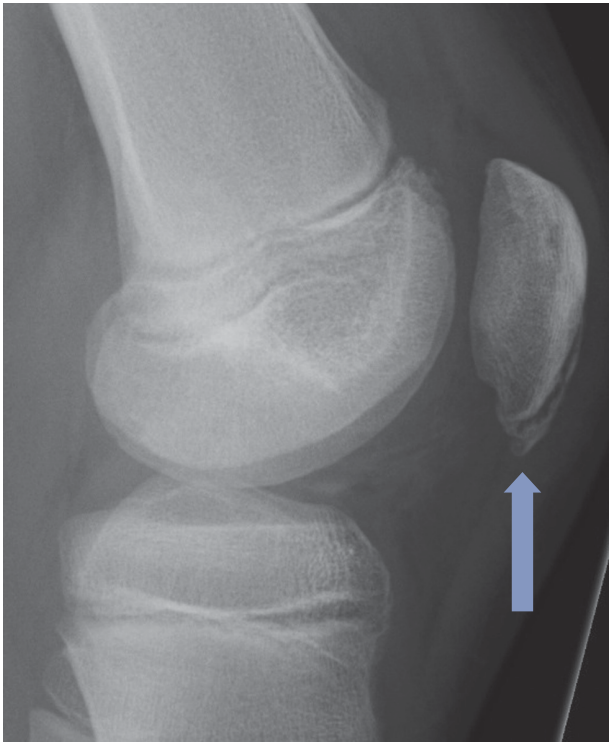

Fig. 14.10 Lateral radiograph of the right knee showing a patella sleeve fracture. Note the flake of bone at the inferior pole of the patella.
Reproduced courtesy of Dr Jeremy Jones, Royal Hospital for Sick Children Edinburgh.

Osteochondritis dissecans

- Osteochondritis dissecans is a pathological lesion characterized by varying degrees of softening, separation, and detachment of the articular cartilage from the underlying subchondral bone.
- Causes include repetitive micro-trauma, vascular insult, and genetic predisposition.
- 70% of lesions affect the medial femoral condyle.
- Common in children 10–15y of age; boys > girls.

Clinical presentation

- Pain: poorly localized. Exacerbated by exercise.
- Limp.
- Effusion: may be intermittent.
- Mechanical symptoms: locking, crepitus, and giving way indicate a loose fragment/flap of cartilage and represents advanced disease.

Investigations

- Radiographs of the knee: AP, lateral, and tunnel views.
- MRI: useful in determining location, size, and condition of fragment.

Treatment

- Conservative: stable lesions/open physes—restrict weight-bearing and provide symptomatic treatment.
- Operative: stable lesions/closing physes, unstable/enlarging lesions—fragment fixation, subchondral drilling, chondral resurfacing.

Prognosis

- Stable lesions spontaneously heal over 18 months.
- Good prognostic indicators: young age, open physes.
- Bad prognostic indicators: lesions of the lateral femoral condyle and patella, sclerosis on plain film, and synovial fluid behind the lesion on MRI. These children are at risk of early-onset osteoarthritis.

Congenital talipes equinovarus or clubfoot

Congenital talipes equinovarus (CTEV), more readily known as clubfoot, is considered the commonest congenital abnormality affecting 1 in 500 children in the UK, with a higher prevalence found in those of Maori or Hawaiian origin. Boys are affected more than girls and 50% of cases are bilateral. It can be diagnosed at prenatal screening after 12 weeks, but a high false positive rate exists.

Clubfoot can be idiopathic or syndromic whereby it occurs in combination with other developmental disorders such as arthrogryposis, tibial hemimelia, and spina bifida.

Clinical features

The foot lies in a supinated, adducted, and varus position, so that the sole of the foot is directed towards the midline. The forefoot appears supinated but is, in fact, pronated in relation to the midfoot. Characteristic features of clubfoot can be recalled using the aide memoire *CAVE*:

- C, midfoot cavus.
- A, forefoot adductus.
- V, hindfoot varus.
- E, hindfoot equinus.

Management

- *Conservative*: Ponseti method involves a minimum of 4y of treatment. The first 2 months of serial casting is undertaken to laterally rotate the foot around a fixed talus. During the next 3 months, the child wears a foot abduction orthosis with a Dennis-Brown bar for 23h a day. They continue to wear this until age 4 during sleep.
- *Operative*: percutaneous tenotomy is often used to assist correction of deformity. In resistant, recurrent, or late-presenting cases, bony procedures and tendon transfers are required to correct the foot position.

Pes planus (flat foot)

Paediatric flat feet are frequently seen in the orthopaedic clinic. Often, parental concern over the foot's appearance in the asymptomatic child instigates referral. Children do not develop arches until 3y of age. Determination if the deformity is flexible or rigid is key.

If the medial arch is reconstituted on passive dorsiflexion of the great toe (Jack's test), the deformity is flexible and does not require treatment. Rigid flat feet are associated with an underlying abnormality such as congenital vertical talus and tarsal coalition and require further investigation.

Scoliosis

Scoliosis is a condition that causes the spine to curve in the coronal plane and rotate in the axial plane. The commonest type of scoliosis affecting children is idiopathic, but others exist, including congenital and neuromuscular. Idiopathic scoliosis is further sub-categorized as:

- *Infantile* (<3y). Infantile idiopathic scoliosis accounts for ~5% of all idiopathic scoliosis. Boys are more commonly affected than girls and a strong family history is often present. Associated conditions include thoracic insufficiency syndrome and plagiocephaly. The curve leans towards the left side. The majority of curves spontaneously resolve.
- *Juvenile* (4–10y). Juvenile idiopathic scoliosis accounts for 15% of all idiopathic scoliosis. In this variant, it is girls that are affected more than boys and the curve leans towards the right. Associated conditions include syringomyelia, tethered cord, and tumour. The vast majority progress and thus require treatment.
- *Adolescent* (10–18y). This is the commonest type of idiopathic scoliosis where for small curves, boys and girls are equally affected. However, girls have a 10-fold predisposition to curves >30°. A strong family history is regularly present. Curves tend to be apex to the right, and if left-sided curves are detected, MRI scanning is mandated to rule out serious underlying pathology. Scoliosis in this age group is often treated surgically with posterior fusion in order to prevent chronic pain and progression.

Clinical features

When examining the spine, look for:

- Skin changes: hairy patches, café-au-lait spots, skin dimples.
- Leg length discrepancy.
- Rotational deformity of the ribs, pelvic tilt, and waist asymmetry.
- Differences in shoulder height.
- Adams forward bending test—on bending forwards (flexion), one side of the thorax is higher, exposing spinal malrotation.
- Asymmetrical foot deformities and abdominal reflexes. Assess the neurology of the lower limbs and consider undertaking an MRI scan.

Investigations

- Radiographs: standing PA and lateral.
- MRI: if underlying neural axis abnormality suspected.

Management

- Dependent on age and degree of curve.
- Options: observation, bracing, growing rods, and spinal fusion.

Complications

- Untreated, progressive curves can lead to delay in cardiopulmonary development, cardiopulmonary compromise, and death.

Cerebral palsy

Cerebral palsy is an upper motor CNS disorder caused by injury to the neonatal brain before, during, or after birth, which subsequently affects muscle control and movement. The manifestations of the condition have made cerebral palsy the commonest cause of childhood disability, with an incidence of 2 in 1000 live births. The diagnosis can take up to 2y to make and sometimes longer in subtle cases. Whilst the CNS pathology is static, musculoskeletal consequences can emerge over time.

Risk factors

- Prematurity. This is the commonest risk factor.
- Delayed/traumatic birth causing anoxia.
- Perinatal infections, e.g. CMV, rubella, herpes simplex.
- Congenital cerebral malformations

Features

Children presenting with cerebral palsy display a number of orthopaedic manifestations to varying degrees, depending on their level of affliction.

Primary

- Abnormal tone: hypotonia, spasticity.
- Ataxia.
- Dyskinesia.

Secondary

- Joint contractures.
- Hip subluxation/dislocation.
- Hand and foot deformities.
- Gait disturbances.
- Fractures.

Investigations

- *Radiographs*: AP and lateral radiographs of the hip and, if possible, standing radiographs of the spine.
- *MRI brain*: help establish the diagnosis and extent of damage.
- *Gait analysis*: assists in quantifying gait disturbances and in treatment recommendations and assesses the value of interventions offered.

Management

Aim to keep the child as comfortable and as mobile as possible.

- Physiotherapy and orthotics: maintain muscle strength, joint mobility, and optimize movement.
- Medical: Botox and baclofen to address spasticity.
- Operative:
  - Soft tissue procedures, e.g. tenotomies.
  - Selective dorsal rhizotomy.
  - Bony procedures, e.g. osteotomies.

Chapter 15

Major trauma

Management of major trauma

Key facts

- Trauma is the leading cause of death in the first four decades of life, and every minute, >9 people die from injuries and violence.
- Trimodal distribution of death implies death from injury occurs in one of three time periods.
  - *First peak*. Within seconds to minutes. Very few can be saved due to the severity of their injuries.
  - *Second peak*. Within minutes to several hours. Deaths occur due to life-threatening injuries.
  - *Third peak*. After several days to weeks. Deaths from sepsis and multiple organ failure.
- The 'golden hour' refers to the period when medical care can make the maximum impact on death and disability. It implies the urgency, and not a fixed time period of 60min.

The advanced trauma life support (ATLS) system

- Accepted as a standard for trauma care during the 'golden hour' and focuses on the 'second peak'. Emphasizes that injury kills in certain reproducible time frames in a common sequence: loss of airway, inability to breathe, and loss of circulating blood volume, expanding intracranial mass.
- Involves rapid assessment of injuries and institution of life-preserving therapy.
- Asystematic, rapid initial assessment is essential and this includes: preparation, triage, 1° survey (ABCDE), resuscitation, 2° survey, continued monitoring and re-evaluation, and definitive care.

Prehospital care and the trauma team

- Effort is made to minimize scene time, emphasizing immediate transport to the closest appropriate facility (scoop and run).
- Aprehospital system is set up to notify the receiving hospital is informed before patient transport from the scene.
- Trauma team usually comprises an anaesthetist, a 'general' surgeon, an orthopaedic surgeon, and an A&E specialist, A&E nurses, and radiographers.
- Information from paramedics should include: **M**echanism of injury, **I**njuries identified, **S**ymptoms and **S**igns at the scene, and any **T**reatment initiated (MIST).
- Triage is the process of prioritizing patients according to treatment needs *and* the available resources (those with life-threatening conditions **and** with the greatest chance of survival are treated first).

Management

Primary survey

Identify and treat life-threatening conditions according to priority (ABCDE).

Airway maintenance with cervical spine protection

- Protect spinal cord with immobilization devices or using manual in-line immobilization. Protect until cervical spine injury is excluded.
- Access airway for patency. If patient can speak, airway is not immediately threatened.
- Consider foreign body and facial, mandibular, or tracheal/laryngeal fractures if unconscious. Perform chin lift/jaw thrust. Consider nasopharyngeal/oropharyngeal airway.
- If patient unable to maintain airway integrity or GCS score 8 or less, secure a definitive airway (orotracheal, nasotracheal, cricothyroidotomy).

Breathing and ventilation

- To maximize oxygenation and eliminate CO_2, adequate gas exchange is required.
- Administer high-flow O_2 using a non-rebreathing reservoir.
- Inspect for chest wall expansion, symmetry, respiratory rate, and wounds. Percuss and auscultate the chest. Look for tracheal deviation and surgical emphysema.
- Identify and treat life-threatening conditions: tension pneumothorax, open pneumothorax, flail chest with pulmonary contusion, and massive haemothorax.

Circulation with haemorrhage control

- Look for signs of shock. Haemorrhage is a predominant cause of preventable deaths after an injury.
- If tension pneumothorax has been ruled out as a cause of shock, hypotension is usually considered to be hypovolaemic in origin, unless proved otherwise. Think: chest, abdomen, retroperitoneum, muscle compartment, and open fractures ('blood on the floor and four more').
- Control external bleeding with pressure.
- Obtain IV access using two large-calibre cannulae (minimum 16G). Send blood for cross-match, appropriate lab analysis, toxicology studies, and pregnancy testing for all ♀ of childbearing age.
- Commence bolus of warmed Ringer's lactate solution; unmatched, type-specific blood only for immediate life-threatening blood loss.
- Excessive fluid administration is not a substitute for definite control of bleeding and can activate the inflammatory cascade by exacerbating the lethal triad of coagulopathy, acidosis, and hypothermia.
- Consider surgical control of haemorrhage (laparotomy, thoracotomy).

Disability

- Perform a rapid neurological evaluation. AVPU method (**A**lert, responds to **V**ocal stimuli, responds only to **P**ainful stimuli, **U**nresponsive to all stimuli), Glasgow coma scale (GCS) score.
- After excluding hypoxia and hypovolaemia, consider changes in level of consciousness due to head injury.
- Prevention of 2° brain injury by maintaining adequate oxygenation and perfusion.

Exposure/environment control

- Undress patient for thorough examination.
- Prevent hypothermia by covering with warm blankets/warming device. Use warm IV fluids.

Adjuncts to primary survey

- Monitoring. Pulse, non-invasive BP, ECG, pulse oximetry, ABG.
- Urinary catheter (after ruling out urethral injury) and gastric catheter.
- Diagnostic studies. X-rays (lateral cervical spine, AP chest, and AP pelvis), focused abdominal sonography for trauma (FAST) and diagnostic peritoneal lavage (DPL), CT scan.
- Essential radiological imaging should be obtained, even in pregnant patients.

Secondary survey

Begin only after 1° survey is complete, resuscitation is continued, and normalization of vital functions.

- Take history. AMPLE (**A**llergy, **M**edication, **P**ast medical history, **L**ast meal, **E**vents of the incident).
- Perform a head-to-toe physical examination.
- Continue reassessment of all vital signs.
- Perform specialized diagnostic tests that may be required.

Re-evaluation

Crucial to identify deterioration of previous findings and to ensure new findings are not missed.

Thoracic injuries

Key features

- Thoracic injuries account for 25% of deaths from trauma.
- 50% of patients who die from multiple injuries also have a significant thoracic injury.
- Open injuries are caused by penetrating trauma from knives or gunshots. Closed injuries occur after blasts, blunt trauma, and deceleration—road traffic accidents (RTAs) are the commonest cause.
- <10% of blunt chest injuries and <30% of penetrating chest injuries require operative intervention.

Management—primary survey

Identify and treat major thoracic life-threatening injuries.

Tension pneumothorax

- Aclinical diagnosis. There is no time for X-rays.
- Patient has respiratory distress and is tachycardic and hypotensive.
- Look for tracheal deviation, ↓ movement, hyperresonant percussion note and absent breath sounds over affected hemithorax, and distended neck veins.
- Treat with immediate decompression. Insert a 12G cannula into the second intercostal space in the mid-clavicular line. Follow this with insertion of an underwater seal chest drain into the fifth intercostal space between the anterior and mid-axillary line.

Open pneumothorax

- Occlude with a three-sided sterile dressing.
- Follow by immediate insertion of an intercostal drain through a separate incision.

Flail chest

- Results in paradoxical motion of the chest wall. Restricted chest wall movement and underlying lung contusion cause hypoxia.
- If the segment is small and respiration is not compromised, nurse patient in HDU with adequate analgesia. Encourage early ambulation and vigorous physiotherapy. Do regular blood gas analysis.
- In more severe cases, endotracheal intubation with positive-pressure ventilation is required.

Massive haemothorax

- Rapid accumulation of >1500mL of blood or one-third or more of the patient's blood volume in the pleural cavity.
- Suspect when shock is associated with dull percussion note and absent breath sounds on one side of chest, and flat neck veins.
- Simultaneously restore blood volume and carry out decompression by inserting a wide-bore chest drain. Blood from chest tube should be collected in a device appropriate for autotransfusion.
- Consider need for urgent thoracotomy to control bleeding if there is continued brisk bleeding and a need for persistent blood transfusion. Consult with a regional thoracic centre.

Cardiac tamponade

- Most commonly results from penetrating injuries, but blood can also accumulate in pericardial sac after blunt trauma.
- Beck's triad—hypotension, distended neck veins, and muffled heart sounds.
- If critically ill with suspected tamponade, perform 'blind' pericardiocentesis and call cardiothoracic or general surgeons to consider emergency thoracotomy.
- If unwell, but responding to treatment, arrange urgent TTE or focused abdominal ultrasound in A&E.

Management—secondary survey

In stab injuries, expose the patient fully and position them in order to assess the front, back, and sides of the chest for any wounds missed in the 1° survey.

An erect CXR will aid in identifying the following injuries.

Simple pneumo-/haemothorax Best treated with a chest drain if large or symptomatic or in any patient likely to undergo GA or receive positive-pressure ventilation.

Pulmonary contusion Commonest potentially lethal chest injury. Respiratory failure develops over a period of time, rather than immediately. Treat with analgesia, physiotherapy, and oxygenation. Consider respiratory support for a patient with significant hypoxia.

Tracheobronchial rupture

- Potentially fatal.
- Haemoptysis, subcutaneous emphysema, or tension pneumothorax.
- Suspect when there is persistent large air leak after chest drain insertion. Seek immediate (cardiothoracic) surgical consultation.
- Bronchoscopy confirms the diagnosis

Blunt cardiac injury (myocardial contusion/traumatic infarction)

- Associated with chest wall contusion/sternal or rib fractures.
- Suspect when there are significant abnormalities on ECG or echocardiography.
- Seek cardiology/cardiothoracic surgical advice.

Aortic disruption

- Patients survive immediate death because the haematoma is contained.
- Suspect when history of decelerating force and where there is widened mediastinum on CXR.
- Thoracic CT scan is diagnostic.
- Consider cardiothoracic surgical referral.

Diaphragmatic rupture

- Usually 2° to blunt trauma in restrained car passengers (seat belt compression causes 'burst' injury commonly on the left side).
- Suspect in patients with a suitable history and a raised left hemidiaphragm on CXR.
- Penetrating trauma below the fifth intercostal space can produce a perforation.
- Thoracoabdominal CT scan usually diagnostic.

Abdominal trauma

Key features

- Abdominal injuries are present in 7–10% of trauma patients. These injuries, if unrecognized, can cause preventable deaths.
- Blunt trauma. Most frequent injuries are the spleen (40–55%), liver (35–45%), small bowel (5–10%), and retroperitoneum (10%).
  - Direct pressure—compression or crushing, causing rupture of solid or hollow organs.
  - Deceleration injury due to differential movement of fixed and non-fixed parts of organs, causing tearing or avulsion from their vascular supply, e.g. liver tear and vena caval rupture.
- Penetrating trauma.
  - Stab wounds and low-velocity gunshot wounds. Cause damage by laceration or cutting; stab wounds commonly involve the liver (40%), small bowel (30%), diaphragm (20%), and colon (15%).
  - High-velocity gunshot wounds transfer more kinetic energy and also cause further injury by cavitation effect, tumble, and fragmentation; commonly involve the small bowel (50%), colon (40%), liver (30%), and vessels (25%).
- Explosive trauma—can combine both blunt and penetrating injuries.

Management—primary survey

- Any patient persistently hypotensive despite resuscitation, for whom no obvious cause of blood loss has been identified by 1° survey, can be assumed to have intra-abdominal bleeding.
- If the patient is stable, an emergency abdominal CT scan is indicated.
- If the patient remains critically unstable, an emergency laparotomy is usually indicated.

Management—secondary survey of the abdomen

History

- Obtain from patient, other passengers, observers, police, and emergency medical personnel.
- Mechanism of injury. Seat belt usage, steering wheel deformation, speed, damage to vehicle, ejection of victim, etc. in automobile collision; velocity, calibre, presumed path of bullet, distance from weapon, etc. in penetrating injuries.
- Prehospital condition and treatment of patient.

Physical examination

- Inspect anterior abdomen, which includes lower thorax and perineum, and log roll to inspect posterior abdomen. Look for abrasions, contusions, lacerations, penetrating wounds, distension, and evisceration of viscera.
- Palpate abdomen for tenderness, involuntary muscle guarding, rebound tenderness, and gravid uterus.
- Auscultate for presence/absence of bowel sounds.
- Percuss to elicit subtle rebound tenderness.
- Assessment of pelvic stability must be done only once as repeated testing can result in further haemorrhage.
- Penile, perineum, rectal, and vaginal examinations, and examination of gluteal regions.

Investigations

- Blood and urine sampling. Raised serum amylase may indicate small bowel or pancreatic injury.
- Plain radiography. Supine CXR is unreliable in the diagnosis of free intra-abdominal air.

Focused abdominal sonography for trauma

- It consists of imaging of the four Ps—Morrison's **p**ouch, **p**ouch of Douglas (or **p**elvic), **p**erisplenic, and **p**ericardium.
- It is used to identify the peritoneal cavity as a source of significant haemorrhage.
- It is also used as a screening test for patients without major risk factors for abdominal injury.

Diagnostic peritoneal lavage

- Mostly superseded by FAST for unstable patients and by CT scanning in stable patients. Useful, when these are inappropriate or unavailable, for identification of the presence of free intraperitoneal fluid (usually blood).
- Aspiration of blood, GI contents, bile, or faeces through the lavage catheter indicates laparotomy.

Computed tomography

- The investigation of choice in haemodynamically stable patients in whom there is no apparent indication for an emergency laparotomy.
- It provides detailed information relative to specific organ injury and its extent and may guide/inform conservative management.

Indications for resuscitative laparotomy Blunt abdominal trauma with unresponsive hypotension despite adequate resuscitation and no other cause for bleeding found.

Indications for urgent laparotomy

- Blunt trauma with positive DPL or free blood on ultrasound and an unstable circulatory status.
- Blunt trauma with CT features of solid organ injury not suitable for conservative management.
- Clinical features of peritonitis.
- Any penetrating injury associated with visible viscera, haemodynamic instability, or developing fever/signs of sepsis.
- Any gunshot wound.

Vascular injuries

Key features

- Stab wounds cause most of the upper extremity vascular injuries, whilst gunshot wounds cause the majority of lower extremity vascular injuries in penetrating vascular injury.
- Blunt trauma causes more morbidity than penetrating injuries due to associated fractures, dislocations, and crush injuries to muscles and nerves.

Management—primary survey

- Apply direct pressure to open haemorrhaging wound.
- Initiate aggressive fluid resuscitation.
- Arapidly expanding haematoma suggests a significant vascular injury.
- Realign and splint any associated fracture. Immobilize dislocated joint.
- Seek surgical consultation.

Management—secondary survey

- Begin only after 1° survey is complete and resuscitation is continuing successfully.
- Identify limb-threatening injuries.
  - 'Hard' signs are external pulsatile bleeding, expanding or pulsatile haematoma, absent or diminished distal pulses, palpable thrill or audible bruit, and signs of distal ischaemia. These patients require urgent operative intervention.
  - 'Soft' findings include history of active bleeding at the scene, proximity of penetrating or blunt trauma to a major artery, small non-pulsatile haematoma, and neurological deficit.
- Measure distal systolic Doppler pressures of the injured arm or leg and compare with uninjured brachial systolic pressure. An index of <0.9 is a predictor of arterial injury.
- Intraoperative arteriography helps in planning the operative approach. Some minimal arterial injuries can be managed non-operatively. Embolization can be used to manage selected arterial injuries.

Principles of operative management

- Obtain proximal and distal control prior to exposing the injury.
- Inspect the injured vessel and debride as necessary.
- Remove intraluminal thrombus by using a Fogarty catheter.
- Flush lumen with heparinized normal saline solution.
- Consider temporary intraluminal shunting if limb is ischaemic and there is a delay (if revascularization is anticipated).
- The type of vascular repair depends on the extent of damage. Techniques used are lateral repair, patch angioplasty, end-to-end anastomosis, interposition graft, or bypass graft.
- Use systemic anticoagulation if there is no contraindication.
- Consider intraoperative completion arteriography.
- Ensure completed vascular repair is free of tension and covered with viable soft tissue.

- In patients with combined vascular and orthopaedic injuries, perform arterial repair first to restore circulation before orthopaedic stabilization. Where there is massive soft tissue injury, debride all non-viable tissue.
- Anticipate development of compartment syndrome. Perform fasciotomies to decompress all four compartments of the leg.

Head injuries

Causes and features

- Commonest reasons for head injuries—falls, RTAs, and assaults.
- About 75% of head injuries are mild, 15% moderate, and 10% severe.
- Up to half of deaths from trauma under the age of 45 are due to a head injury. Sequelae are common in survivors.
- Classification:
  - Mild—GCS score 13–15.
  - Moderate—GCS score 9–12.
  - Severe—GCS score 3–8.

Management—primary survey

- Prevent potentially devastating 2° brain injury by providing adequate oxygenation and maintaining BP sufficient enough for brain perfusion.
- Determine consciousness level on the AVPU or GCS (see Table 15.1; use a standard head injury proforma).
- Involve an anaesthetist to provide appropriate airway management in patients with GCS score <8 or AVPU = P or U.
- Avoid systemic analgesia until full neurological assessment is made.

Management—secondary survey

- Fully assess the head and neck, including:
  - Examination of skull vault.
  - Looking for signs of base of skull fractures (haemotympanum, 'panda' eyes, CSF otorrhoea or rhinorrhoea, Battle's sign).
  - Repeated monitoring of vital signs.
  - Repeated assessment of consciousness level (GCS or AVPU).
- Follow guidelines when transferring patients to the neurosurgical unit. Ensure resuscitation and stabilization of patient is complete before transfer.
- Give verbal advice and a written head injury advice card to patients who are being discharged from A&E.

Indications for CT scanning in head injuries

- GCS score <13 at any point since injury; GCS score <15 at 2h after injury.
- Suspected open or depressed skull fracture.
- Any sign of basal skull fracture.
- Post-traumatic seizure.
- Focal neurological deficit.
- >1 episode of vomiting.
- Amnesia for >30min of events before impact.
- Age ≤65y, coagulopathy, dangerous mechanism of injury, loss of consciousness or amnesia has been experienced.

Indications for neurosurgical referral in head injuries

- Major intracranial injury (extradural haematoma, moderate or larger subdural haematoma, intracerebral haematoma).
- Progressive focal neurological signs.
- Definite or suspected penetrating head injury.
- CSF leak or base of skull fracture.
- Persisting coma (GCS score ≥8) after initial resuscitation or deterioration in GCS score after admission.

Indications for admission in head injuries

- Patients with new, clinically significant abnormalities on imaging.
- Patient has not returned to GCS score 15 after imaging, regardless of the imaging results.
- Patient fulfils the criteria for CT scanning, but this cannot be done within the appropriate period, either because CT is not available or because the patient is not sufficiently cooperative to allow scanning.
- Continuing worrying signs, e.g. persistent vomiting, severe headaches.
- Other sources of concern, e.g. drug or alcohol intoxication, other injuries, shock, suspected NAI, meningism, CSF leaks.

Table 15.1 Glasgow coma scale*

Feature	Scale	Score
Eye opening (E)	Nil	1
	In response to pain	2
	In response to speech	3
	Spontaneous	4
Motor response (M)	Nil	1
	Extension	2
	Abnormal flexion	3
	Flexion away from pain	4
	Localizes pain	5
	Obeys commands	6
Verbal response (V)	Nil	1
	Sounds	2
	Inappropriate words	3
	Confused sentences	4
	Orientated fully	5

* Minimum score, 3; maximum score, 15.

Reproduced from Teasdale, G. and Jennett, B. (1974). The Lancet, 304: 7872, with permission from Elsevier.

Disaster management

Approach

- Unpredictable due to the nature, location, and timing, and hence preparedness enhances the ability to respond.
- Fundamental principle—do the greatest good for the greatest number.
- Incidents or events where the patient needs overwhelm the resources needed to care for them.

Phases of disaster management

- *Preparation*—undertaken by hospital to identify risks and resources and to build capacity. Includes risk assessment of the area, disaster plan that is reviewed and revised regularly, and provision of training.
- *Mitigation*—involves adoption of an incident command system (ICS) to lessen the severity and impact of a potential disaster. No substitute for adequate training and drilling.
- *Response*—involves treating victims of actual disaster, include activation of hospital disaster plan and ICS.
- Prehospital response occurs in four stages:
  - Stage 1, chaos stage—15–20min.
  - Stage 2, organizational phase—1–2h.
  - Stage 3, site clearing and evacuation—time depends on complexity of incident.
  - Stage 4, gradual recovery.
- *Recovery*—local public health system plays a major role and this phase facilitates resumption of operations after an emergency.

Pitfalls

- Communication—communication systems will need to be ensured to be fully interoperable.
- Supplies—supplies needed for disasters must be available and stored in secure areas.
- Security—must be ensured for providers, patients, supplies, and systems.
- Volunteers—properly trained and must participate only as part of a planned and organized disaster response, in order to avoid danger to themselves and the patients.

Triage

- Process of prioritizing patient treatment during mass casualty events.

Principles

- Do the most good with available resources.
- Make a decision—time is the essence of triage.
- Good understanding of the resources available.
- Planning and rehearsal.

Triage category

- Black, dead.
- Red, priority 1—immediate attention.
- Yellow, priority 2—can wait for a short time for transport.
- Green, priority 3—can be delayed before transport.

Retriage

- Not a one-time one-place event—continuous and repetitive at each level.

Chapter 16

Orthopaedic surgery

Examination of a joint

- Applying a systematic approach will avoid missing vital clues.
- Always begin with a history, followed by examination.
- The classical orthopaedic triad of 'look, feel, and move' applies.[1]
- Remember to examine the patient as a whole, not just the joint!

Ask

- Is the joint painful?
- Is there a specific area of tenderness?
- Does the pain radiate? Where to?
- Is the joint swollen?
- Can the joint be moved actively?
- Has there been an injury to the joint?

Look

- Remember, always compare unaffected with affected side.
- Is there any swelling? If so, is it an effusion, synovitis, or bony deformity?
- Where is the swelling?
- Are there any colour changes? Bruising or erythema?
- Is there any skin involvement, i.e. rheumatoid nodules at elbow or psoriatic plaques?
- Are there any scars or wounds? If so, are they traumatic, surgical, or infective?
- Look for muscle wasting, generally around the joint and specifically in the whole limb.
- Examine the patient as a whole for clues to the disease process at the joint.

Feel

- Always gain verbal consent and explain what you are doing to the patient.
- Remember, ask the patient if it is painful first!
- Examine the unaffected or least painful side prior to examining the affected side.
- Is the joint hot, cold, or moist?
- Is there any local tenderness? Look at the patient's face, not the joint.
- Is the joint swollen? An effusion can occur after trauma (haemarthrosis) or with infection (septic arthritis). Does the fluid shift with sweeping? Is synovitis present (non-movable fluid feel), or is it a bony swelling?

Move

- Compare affected with unaffected side.
- Test active movements first before passive. This gives an idea of the patient's pain and reduces further discomfort.
- Ask the patient to move the joint through a full range of movement.
- Look for pain (patient's face) and limitation of movement.
- Is the limitation mechanical (blocked by loose body, meniscal tear, or contracture) or restrictive (resisted by the patient due to pain)?
- *Shoulder*. Flexion, extension, abduction, internal and external rotation.
- *Elbow*. Flexion, extension, pronation, and supination (ensure humerus at patient's side).

- *Wrist*. Flexion, extension, radial and ulnar deviation, pronation, and supination.
- *Metacarpophalangeal joint (MCPJ)*. Flexion, extension, abduction, and adduction.
- *Proximal interphalangeal joint (PIPJ) and distal interphalangeal joint (DIPJ)*. Flexion and extension.
- *Thumb*. Flexion, extension, abduction, adduction, and opposition.
- *Hip*. Flexion, extension, internal and external rotation.
- *Knee*. Flexion and extension.
- *Ankle*. Plantar flexion and dorsiflexion, eversion and inversion.
- *Cervical spine*. Flexion, extension, and lateral rotation and flexion.
- *Lumbar spine*. Flexion, extension, and lateral rotation and flexion.
- Muscle power is graded via the Medical Research Council (MRC) system (➲ Examination of the limbs and trunk, p. 608).
- Always get the patient to walk to test gait if the problem is lower limb.

Special tests

These depend on the individual joint examined and are numerous for each joint!

They normally involve:

- Either tests of instability, e.g.:
  - Collateral testing in the knee, elbow, or finger joints.
  - Cruciate ligament testing (anterior draw, Lachman's).
  - Apprehension tests (shoulder instability).
- Or provocation tests that aim to locate the cause of intra-articular pain, e.g.:
  - Grind tests (thumb base osteoarthritis, knee meniscal injury).
  - Meniscal provocation tests (McMurray's test).

Reference

1 Blom A, Warwick D, Whitehouse M (Eds) (2017). *Apley and Solomon's System of Orthopaedics and Trauma*, 10th edn. CRC Press, Boca Raton, FL.

Examination of the limbs and trunk

Develop your own system with which you feel comfortable. Always compare affected with unaffected side. Make allowances for the dominant side.

Look

Is there any swelling, deformity, asymmetry, muscle wasting, twitching (fasciculation), scars, skin colour changes, rashes, or discharging wounds (sinuses)?

Feel

Is there any tenderness, temperature changes, solid or fluid swellings, or muscle bulk?

Move

- Move each joint through its full active and passive range.
- Is the limb tone normal, reduced (flaccid or floppy), or ↑ (rigidity)? Is there any spasm? Are there any joint contractures?
- Is the rigidity through whole movement or only initially (spasticity)?
- Is the alteration in movement from a neuromuscular disorder, a mechanical block, or pain from the joint?

Power (MRC grading)

- Grade 0, no movement.
- Grade 1, flicker of movement only.
- Grade 2, movement with gravity eliminated.
- Grade 3, movement against gravity.
- Grade 4, movement against resistance.
- Grade 5, normal power.

Test all muscle groups within their relevant myotomes, according to the patient's history.

Coordination

- Ask the patient to touch their nose with their index finger, with their eyes open and then shut. Compare side to side.
- Alternatively, ask the patient to put their right heel onto their left knee and to run it down their shin, and vice versa. Note whether these movements are smooth or jerky.
- Romberg's test. Stand with feet together and eyes shut. Positive result will cause the patient to become unstable or fall; be prepared!

Reflexes

- Biceps jerk, C5/6.
- Abdominal, T8–T12.
- Triceps jerk, C6/7.
- Knee jerk, L2/3/4.
- Brachioradialis, C5/6.
- Ankle jerk, S1/2.
- Plantar response. Normal flexor, abnormal extensor (Babinski's sign).
- Clonus at ankle (normal two beats or less).

Grading

- 0, absent.
- 1, hypoactive.
- 2, normal.
- 3, hyperactive, no clonus.
- 4, hyperactive with clonus.

Sensation

Explain what you are about to do clearly to the patient and perform the test with their eyes closed. Compare symmetrical sides of the body at the same time. Map out the abnormalities.

- Pinprick, light touch, and temperature tested in a dermatomal pattern.
- Vibration sense tested with a 128MHz low-pitched tuning fork on a bony prominence. Start distal and if abnormal, move from proximal.
- Proprioception (joint position sense) tested by moving the metatarsophalangeal joint (MTPJ) of the hallux, up and down; the patient confirms the correct movement.

Fracture healing

Fracture healing occurs as either 1° or 2° bone union.

- 2° bone healing produces callus. It occurs when fractures are immobilized with 'relative stability' (some minimal movement at fracture site, e.g. a plaster cast). It involves two simultaneously occurring, but distinct, processes—intramembranous and endochondral ossification, producing periosteal bony callus and fibrocartilagenous bridging callus, respectively.
- 1° bone healing does not produce callus. It occurs when fracture fragments are reduced 'anatomically' and 'interfragmentary compression' is achieved with 'absolute stability'. There is no motion between fracture surfaces (e.g. compression plating techniques or lag screw fixation).

Secondary bone healing (callus)

Initial phase: haematoma and inflammation

- Torn vessels at fracture site bleed, producing a haematoma and subsequent clot.
- The size of the haematoma depends upon the blood supply to the bone and the violence of the injury; it can continue to expand during the first 36h.
- Injured tissue and platelet activation cause an inflammatory cascade via the release of growth factors and various cytokines.
- Inflammatory cell migration to the haematoma occurs (macrophages, fibroblasts, osteoclasts, chondroblasts).
- Fibroblasts and chondroblasts organize the haematoma into collagen and granulation tissue, with new capillary ingrowth (angiogenesis).
- Osteoclasts and macrophages remove dead bone and tissue, respectively.
- This stage usually lasts up to 1 week.

Second phase: callus formation (soft and hard)

- Cell (osteoblasts) proliferation and differentiation results in callus formation.
- Intramembranous (or periosteal) hard callus forms peripherally, with endochondral (fibrocartilagenous/bridging) soft callus forming alongside. A third type 'medullary callus' forms later if the above fails.
- The amount and type of callus produced are dependent upon local factors such as the type of fracture, proximity of the bone ends, amount of haematoma, and is inversely proportional to the amount of movement present.
- Soft callus is calcified by chondroblasts and subsequently resorbed by chondroclasts.
- New blood vessel invasion into the callus brings osteoblastic-type cells, resulting in ossification into woven bone.
- By this point, the fracture will have united and be pain-free.
- This stage lasts 1 week to 4 months.

Third phase: remodelling

- Woven bone is resorbed by osteoclasts and osteoblasts replace this with lamellar bone, which is very hard and dense.
- Final remodelling occurs when swelling around the fracture site decreases; trabeculae can be seen crossing the fracture site on radiographs and the medullary canal is recreated.
- Remodelling is most marked in children and follows the mechanical forces applied to the bone in a physiological environment.
- This process is identical in both 1° and 2° bone healing and can last for several years.

Primary bone healing (absolute stability)

- The inflammatory response is much reduced.
- Areas of direct contact undergo some activity.
- Any gaps are invaded with blood vessels and cells differentiate into osteoblasts, laying down woven and lamellar bone (gap healing).
- Osteoclasts acting as 'cutting cones' pass directly across the fracture site, leaving channels that are filled with blood vessels and allowing osteoblasts to fill them with lamellar bone.
- No callus is formed and union takes much longer to achieve, with the strength of the healing process being borne by the mechanical properties of the fixation device.
- Remodelling occurs as above.

Factors adversely affecting fracture healing

- Degree of local trauma (bone loss, soft tissue trauma and interposition, neurovascular injury, open fractures).
- Inadequate reduction and immobilization.
- Infection.
- Location of fracture. Which bone and where on bone, i.e. metaphysis versus diaphysis (see below)?
- Disturbances of ossification, e.g. metabolic bone disease, osteoporosis, local pathological tumour.
- Age, poor nutrition, smoking, drugs (especially NSAIDs), diabetes.

How long do fractures take to unite?

Perkins rules

- Fractures of cancellous (metaphyseal) bone (e.g. those around joints) will take 6 weeks to unite.
- Fractures of cortical (diaphyseal) bone (e.g. shafts of long bones) will take 12 weeks to unite.
- Fractures of the tibia (because of poor blood supply) will take 24 weeks to unite.
- Time to union for children equals the age of the child in years plus 1, e.g. tibial fracture in a 2y-old child will unite in 3 weeks. Common sense needs to be applied when applying the rule to fractures of cancellous bone in older children.

Delayed union

Defined as failure of union to occur in 1.5 times the normal time for fracture union.

Non-union

Defined as failure of union to occur within twice the normal time to fracture union. However, expect open fractures to normally take twice the normal Perkins rule.

- *Hypertrophic non-union*. Excess mobility or strain at fracture site. There is a good blood supply, with healing potential. Appears as large callus (elephant's foot pattern) on X-rays. Usually requires stabilization to allow callus progression.
- *Atrophic non-union*. Due to poor blood supply resulting from initial injury or surgical intervention. There is poor healing potential. Usually requires stabilization and biological augmentation to heal.

Further reading

Blom A, Warwick D, Whitehouse M (Eds) (2017). *Apley and Solomon's System of Orthopaedics and Trauma*, 10th edn. CRC Press, Boca Raton, FL.

Ramachandran M (2007). *Basic Orthopaedic Sciences: The Stanmore Guide*. Hodder Arnold, London.

Reduction and fixation of fractures

Caveat

A fracture is a soft tissue injury with an associated broken bone. Treat the soft tissues with utmost respect to ensure fracture healing.

Modern fracture reduction and treatment were pioneered by the AO group and centres around four key principles:[1]

- Fracture reduction and fixation to restore anatomical relationships.
- Stability by fixation or splintage as the personality of the fracture and the injury dictates.
- Preservation of blood supply to the soft tissue and bone by careful handling and gentle reduction techniques.
- Early and safe mobilization of the part and patient.

Fracture reduction can be achieved by closed[2] (indirect) or open (direct and indirect) methods. Maintenance of the reduction may also be achieved via closed methods which can be non-surgical (plaster or brace) or surgical (intramedullary nail, external fixation, Kirschner (K) wires), or via open methods such as rigid internal fixation with plates and screws[3].

Casting

- Application of a plaster of Paris (or modern alternatives) cast over appropriate padding to stabilize a reduced fracture.
- Typically involves splinting of joints on either side of a long bone fracture to provide additional rotational stability.
- Simplest and cheapest to apply.
- Lowest risk of septic complications.
- It will provide pain relief.
- 'Half casts' or 'backslabs' can be utilized to immobilize a fracture prior to definitive management.
- Complications include problems caused by the cast (pressure sores, itching, and rashes), loosening of the cast and breakdown (losing reduction), thromboembolic events, and coverage of wounds.
- It is a very involved process, requiring regular follow-up with clinical and radiographic assessment to ensure maintenance of reduction.

Cast bracing

- Stabilization of a fracture across a joint with a cast, but the joint itself is left free to move by incorporation of a hinge across it.
- Has the advantage of allowing early movement of the joint without the use of weight-bearing, e.g. tibial shaft fractures.

Internal fixation

Indications

- *Intra-articular fractures*. To prevent or reduce the incidence of osteoarthrosis.
- *Unstable fracture patterns*.
- *Neurovascular damage*. Fracture stability must be achieved before the delicate repair of vessels or nerves takes place. If not, these repairs may be damaged.
- *Polytrauma*. Multiple injuries are managed by fixation to facilitate nursing care and to allow early mobilization.

- *To prevent complications of decubitus*. Elderly patients tolerate immobilization and prolonged bed rest poorly (fractured neck of femur).
- *Fractures of long bones* (e.g. forearm, femur, tibia). Rehabilitation is facilitated more quickly with internal fixation after reduction.
- *Failure of conservative therapy* (loss of acceptable alignment).
- *Pathological fractures*.

Methods

- Compression plates and screws, locking plates and screws, K-wires, intramedullary nails, tension band wiring.

Complications

- Infection which increases with size and ↑ time of exposure required.
- Nerve and vessel injury.
- Compartment syndrome.
- Non-union (↑ with iatrogenic soft tissue and periosteal injury).
- Implant failure.
- Loss of fixation or reduction.
- Thromboembolic event.
- Stiffness in adjacent joints.

External fixation

Indications

Temporizing measure for:

- Open fractures (commonly tibia or femur) associated with significant soft tissue damage or nerve and vessel injury.
- Highly comminuted or unstable fractures and fracture dislocations.
- Lifesaving splintage procedure in pelvic fractures.
- Initial stabilizing device for any fracture where 'damage limitation' surgery may be appropriate in the multiply injured patient (damage control orthopaedics).
- Definitive treatment of complex periarticular fractures (pilon and tibial plateau).
- As a salvage option in the face of mal-union, non-union, or significant bone loss.

Methods

- Pin-and-rod construct most commonly used (tibia–pelvis).
- Modern systems incorporate ring fixators with pins and rods (hybrid).
- Circular fixators (Ilizarov) can be used for definitive fracture fixation or as a salvage option.

Complications

- Pin site infection and possible osteomyelitis.
- Septic arthritis if placed within joint capsule.
- Nerve, vessel, ligament, and tendon injury (good understanding of cross-sectional anatomy required).
- Over-distraction, resulting in non-union.

Locking plates

- Modern implants in which the screw heads are threaded and engage and lock into threads in the plate holes.
- These act as 'internal, external fixators' where forces are transmitted from bone to screw to plate.
- The locking plate provides angular stability and is much stronger than a normal plate, as all screws act in unison.
- Advantages are:
  - Excellent holding power as all locked screws have to fail at once for construct to fail. Thus, excellent choice of fixation in osteoporotic fractures.
  - Spares periosteal blood supply as does not rely on compression of plate on bone.
  - They can be placed percutaneously (avoiding stripping soft tissue and blood supply from a fracture site).
  - They do not require contouring.
  - The screws are usually self-drilling and self-tapping.

References

1 Ruedi TP, Buckley R, Moran C (2007). *AO Principles of Fracture Management*, 2nd edn. Thieme Medical Publishers, New York, NY.
2 McRae R, Esser M (2008). *Practical Fracture Treatment*, 5th edn. Churchill Livingstone, Edinburgh.
3 National Institute for Health and Care Excellence (2016). *Fractures (non-complex): assessment and management*. NICE guideline [NG38]. Available at: https://www.nice.org.uk/guidance/ng38

The skeletal radiograph

This is the most important investigation in orthopaedics but does not substitute for accurate history and examination. Always remember a fracture is a clinical, not a radiological, diagnosis.

Evaluation

Have a system. The following is only one example:

- Note the history, race, occupation, handedness, pastimes, age, sex, and recent laboratory results of the patient.
- Accurate history and clear requests to be documented on X-ray forms. Always write the side in full, e.g. 'right'.
- Always take two views at 90° to each other (orthogonal).
- Examine the film carefully.
- Most hospitals use computer-based X-ray viewing systems, but if using a viewing box, have a bright spotlight and magnifying glass available.
- When describing the lesion, think of the side, anatomical site, nature, displacement, and soft tissue components.
- Keep it simple.
  - A. Adequate views and alignment.
  - B. Bones.
  - C. Cartilage (soft tissues).
- Look for cortical/medullary changes, periosteal reactions, deformity, soft tissue swelling, and cortical breach (definition of a fracture).
- Supplement radiological findings with further biochemical investigations, bone scanning, and biopsy if indicated.

Radiological features

Osteoporosis

- The commonest form of bone disease.
- Characterized by low bone mass and deterioration of the microarchitecture of bone tissue, with consequent increase in bone fragility and susceptibility to low-trauma fractures.
- Affects middle-aged and elderly women, predisposing them to fractures of the distal radius, femoral neck, and vertebral bodies.
- Localized osteoporosis follows disease, e.g. after joint fusion.
- The cortices are thin, with reduced medullary trabeculae, i.e. the bone is essentially normal; there is just too little of it.

Osteomalacia

There is reduced mineralization of osteoid.

- The trabeculae are blurred.
- Symmetrical transverse or oblique cortical defects appear (Looser's zones, pseudofractures).
- In children, changes are most marked at the metaphysis (rickets).

Hyperparathyroidism

There is bone resorption. Best place to see it is in the phalanges of the hands in the subperiosteal cortex. Note generalized cortical striations. Usually diagnosed with PTH levels after incidental finding of raised Ca^{2+} levels.

Diffuse increase in density

Think of neoplasia, fluorosis, sarcoidosis, and bone dysplasia (osteopetrosis).

Abnormalities of bone modelling

Developmental disorders, e.g. osteochondrodysplasia, are often present from birth. Look for abnormalities of the eyes, heart, and ears. Thorough assessment by biochemical and genetic specialist is required.

Local abnormalities may occur in congenital disorders, e.g. endochondromatosis (Ollier's disease), fibrous dysplasia, neurofibromatosis, or acquired disorders, e.g. Paget's disease.

Solitary lesions

Always think of sepsis, 1° bone tumours, or 2° metastasis. Location and age are important, e.g. an epiphyseal lesion in a child may be a chondroblastoma and a subarticular lesion in a young adult may be a giant cell tumour. The older the patient, the more likely it is a metastasis.

Describing a fracture

- First, check details match patient (i.e. date of X-ray, patient age, side, hospital number).
- Ensure the appropriate X-ray is taken, with two views at 90° to each other (e.g. an X-ray of an ankle, rather than the whole lower leg).
- Which bone is fractured?
- Where is the fracture in the bone? Joint (intra-articular), proximal, middle, or distal third, or metaphysis (flares at end of bones), diaphysis (shaft), physis (growth plate), and epiphysis (end part of bone) in children.
- What is the pattern? Transverse, oblique, spiral, comminuted or multifragmentary, segmental.
- Is there displacement? Quantify this (e.g. 50% of bone width or completely 'off-ended' >100%).
- Is there any angulation? Which direction (varus, valgus, recurvatum)?
- If a joint is involved, comment on whether it is 'in joint' or dislocated.
- Other things to look for are gas in soft tissues (suggests open fracture or gas-forming infection), foreign bodies (metal, glass, grit), fluid in joints (e.g. lipohaemarthrosis in knee suggests fracture), and fat pad signs in the elbow (suggest fracture and are prominent due to blood in joint).
- A ring-like structure (e.g. the bony pelvis) rarely fractures in only one place; if you find one fracture, look hard for another one!
- Comment on implants if present and the proximity and involvement of this to the fracture—periprosthetic fractures of a total hip replacement (THR) or total knee replacement (TKR).
- *Pitfalls*. Is it a fracture? Structures that may be mistaken for fractures include suture lines between bones, vascular channels, and physes in immature skeletons. Anatomic structures are more likely to be symmetrical, if not midline.

Further reading

Chan O (2013). *ABC of Emergency Radiology*, 3rd edn. John Wiley and Sons, Chichester.

Raby N, Berman L, Morley S, de Lacy G (2014). *Accident and Emergency Radiology: A Survival Guide*, 3rd edn. Saunders, London.

Injuries of the phalanges and metacarpals

Thumb

Mechanism

Direct blows to thumb, forced opposition of the thumb.

Extra-articular

- *Metacarpal shaft fractures*. Undisplaced fractures can be managed in cast. Displaced fractures require reduction and fixation, either open or closed.
- *Metacarpal base fractures*. Often displaced or angulated due to deforming forces of the tendon attachments. If fractures undisplaced, can manage with closed reduction and immobilization in cast. For displaced/significantly angulated fractures, closed reduction with K-wire fixation or open reduction with internal fixation (ORIF) is required.

Up to 30° of angulation can be accepted due to the vast range of movements at the base of thumb.

Intra-articular (± fracture dislocation)

- *Bennett's fracture dislocation*. Volar/ulnar fragment left behind due to strong ligament attachments; remaining distal metacarpal dislocates proximally and dorsally. Treatment involves closed reduction and K wire fixation to either carpus (trapezium) and/or index finger metacarpal. Open reduction is rarely needed.
- *Rolando fractures*. Multifragmentary fracture, at least three parts in a 'T' or 'Y' pattern ± dislocation. Treatment depends on degree of fragmentation. Reduction and K wire fixation or external fixation should be considered. ORIF only if large fragments.

Thumb dislocation

- At the MCPJ, usually results in ulnar collateral ligament (UCL) injury (gamekeeper's thumb).
- Tear of the UCL can be partial or complete, with adductor aponeurosis stuck in the joint (Stener lesion), preventing reduction.
- Partial tears (stable) are immobilized in cast for 6 weeks.
- Complete tears (unstable or Stener lesion) require surgical repair.
- Chronic tears are treated with tendon reconstruction of UCL or MCPJ fusion.

Metacarpal fractures

Mechanism

Usually 'punch' injury ('Friday night' or 'boxer's' fracture). The little finger most commonly affected. Remember to check for rotational deformity, as this is not an acceptable deformity.

Metacarpal neck fractures

Accept up to 15° angulation in index/middle fingers and 35° in ring/little fingers. Most treated conservatively (neighbour strapping). Reduction and pinning or ORIF if significant angulation.

Metacarpal shaft fractures

Check for rotation. Transverse or unstable fractures (especially ring and little fingers)—treat with ORIF or K wire fixation. Undisplaced fractures can be treated conservatively (neighbour strapping). Similar degrees of angulation to neck fractures can be accepted.

Metacarpal base fractures (± subluxation)

Always get a true lateral X-ray of the hand to assess for subluxation. These are usually stable fractures and can be treated conservatively in cast for 3 weeks. If subluxed (little finger akin to Bennett's fracture), treatment involves closed reduction and K wire fixation to carpal bone for 4 weeks.

Distal phalanx fractures

Mechanism

Crush injury that is comminuted and often compound. Tuft-type fractures.

Treatment

Wound toilet, simple nail bed repair if needed, and 1° suture or Steri-strip® with pressure dressing. Wound inspection at 48h and as required. Can be dealt with in A&E department. Antibiotics if open.

Mallet finger

- Sudden flexion injury of distal phalanx (i.e. stubbing finger), resulting in either avulsion of extensor tendon insertion with flake or large fragment of bone, but can be purely tendinous.
- Small fragments or purely tendinous types are treated in 'mallet splint' (extension) for 8 weeks continuously, followed by 2–4 weeks just at night. If large displaced fragment or joint subluxation on lateral view, fixation can be undertaken if unable to maintain in splint.

Proximal and middle phalanx fractures

Mechanism

Direct blow or twisting injuries.

Treatment

- Dependent on fracture configuration. Check for rotational deformity.
- Undisplaced stable fractures are treated with neighbour strapping for 2–3 weeks, with early mobilization.
- If unstable, rotated, severely angulated, or involving the joint, consider closed reduction and K wire fixation or ORIF with mini-fragment screws ± plate. Stable fixation to allow early mobilization is the goal.

PIPJ dislocations

- Dorsal dislocation is the commonest. It is associated with avulsion of volar plate or fracture of volar base of middle phalanx.
- Dislocations require reduction under ring block.
- If stable, they can be treated with extension blocking splint with PIPJ flexed for 4–6 weeks.
- If unstable or associated with significant fracture, they require manipulation under anaesthesia (MUA) and K wiring, or ORIF, or volar plate arthroplasty.

Immobilization for hand injuries (Edinburgh position)

To prevent stiffness, the metacarpal joint should be immobilized in 90° of flexion and the PIPJ in extension, with the wrist extended at 30°. This places the ligaments on maximal stretch, whilst immobilized.

Further reading

Blom A, Warwick D, Whitehouse M (Eds) (2017). *Apley and Solomon's System of Orthopaedics and Trauma*, 10th edn. CRC Press, Boca Raton, FL.

Wrist injuries

Scaphoid fractures

Mechanism

Fall onto the outstretched hand, with forced dorsiflexion.

Examination

- Fullness in the anatomical snuffbox means an effusion.
- Tenderness on the volar surface of the scaphoid, i.e. the tubercle, is more predictive than snuffbox tenderness (dorsal), which is unreliable.
- Wrist movement, particularly pronation, followed by ulnar deviation, may be painful.
- Pain on compression of the thumb longitudinally or on gripping may be present.
- However, clinical examination is highly variable and skill-dependent.

Investigations (radiographs)

'Scaphoid series' films (PA wrist in ulnar deviation, lateral wrist in neutral; PA in 45° pronation and ulnar deviation; and AP with 30° supination and ulnar deviation). False negative rate of <5%. Fractures are usually of the waist but may be more proximal.

Treatment

- Below elbow, cast in neutral position (RCTs show that the thumb does not need to be included) for 8 weeks, but the fracture may take 12 weeks to unite. At 12 weeks, remove plaster, regardless of symptoms.
- If initial X-rays negative, but clinical suspicion persists, cast the wrist and repeat films in 2 weeks.
- If 2-week films negative, but clinical suspicion of fracture, get MRI (within 2 weeks) to look for occult fracture. This may also reduce length of immobilization in cast if negative.
- Displacement of >1mm or angulation requires ORIF with compression screw.
- Proximal pole fractures are a relative indication for fixation, as high chance of non-union due to poor blood supply.

Complications

- *Non-union*. ↑ with proximal fractures due to blood supply running from distal to proximal in the bone. If not united at 12 weeks, proceed to ORIF (compression screw) ± bone grafting.
- *AVN*. ↑ with proximal and displaced fractures (see above). Treatment is by internal fixation and bone grafting which may need to be a 'vascularized' graft.
- *Degenerative change*. May occur after non- or mal-union. Treated by limited wrist fusion (four-corner fusion, scaphoidectomy, and radial styloidectomy).

Other carpal fractures

- The commonest is *hamate fracture*. The hook is fractured by direct blow to the palm of the hand or repeated direct contact (e.g. motorcyclists, golfers, racquet sports, and cricketers).
- Treatment is usually excision, but internal fixation may be attempted if the fragment is large.

Ligamentous injuries of the wrist

- Common—difficult to diagnose, so easily missed.
- If left untreated, they can cause long-term disability.
- The proximal row of carpal bones forms an intercalated segment, i.e. they are connected and work together as a unit. Injury may occur to the ligaments connecting the bones.

Scapholunate ligament Common in isolation or in association with fractures (especially distal radius). 'Terry Thomas' sign, i.e. increase in the space between scaphoid and lunate on PA view of clenched fist. Acute ruptures may be repaired, but chronic injuries may require reconstruction or fusion.

Lunotriquetral ligament Less common; acute repair may be successful, but chronic injuries require lunotriquetral fusion.

Carpal dislocation

Complete ligamentous injury may allow the carpus to dislocate.

- Occurs either with the lunate remaining in place (a perilunate dislocation) or with the carpus staying in place and the lunate moving (a lunate dislocation).
- On rarer occasions, the scapholunate ligament remains intact and the scaphoid fractures, resulting in a *trans-scaphoid perilunate dislocation*.

Treatment Severe injury requires reduction—best open as it allows formal repair of the disrupted ligaments, as well as stabilization of the carpus. If the scaphoid is fractured, it should be internally fixed as well.

Carpometacarpal fracture dislocation

- Usually as a result of a punch injury. Affects little or ring fingers.
- Commonly missed due to poor history and examination.
- Indicated by tenderness at the carpometacarpal base.
- Diagnosed with a true lateral (not the standard lateral oblique) X-ray (shows subluxation or dislocation at the carpometacarpal joint).

Treatment Unstable injury—reduce with traction and local pressure, then stabilize the joint with K wire fixation for 4 weeks.

Triangular fibrocartilage complex injury

Triangular fibrocartilage complex (TFCC) and the ulnar small ligaments of the hand. An acute tear is usually peripheral and the result of trauma, including a fracture to the ulnar styloid. It will present with ulna-based wrist pain in ulnar deviation with or without rotation.

Treatment If associated with a large ulnar styloid fracture, this can be internally fixed with a tension band wiring technique. Arthroscopic debridement or repair of the tear has been attempted but is technically demanding.

References

E-Hand.com. *The Electronic Textbook of Hand Surgery*. Available at: http://www.eatonhand.com

Clay NR, Dias JJ, Costigan PS, *et al*. (1991). Need the thumb be immobilized in scaphoid fractures? A randomised prospective trial. *J Bone Joint Surg Br* **73**: 828–32.

Fractures of the distal radius and ulna

- Usually caused by a fall onto the outstretched hand.
- Very common; ~1 in 6 of all fractures treated.
- Bimodal incidence. Peaks in childhood (6–10y) and early old age (60–70y).
- Scaphoid and ligamentous wrist injuries may also be present.

Classification

- Classification systems are the AO system and the Frykman system.
- Historical eponymous terms ('Colles', 'Smith's') are still used.
- To avoid confusion, stick to describing the fracture by anatomical methods, e.g. dorsally displaced fracture of the distal radius with shortening and ulnar deviation.
- In children, the fracture usually involves the epiphyseal region and these fractures are classified by the Salter–Harris system. Salter–Harris type II is easily the commonest injury of the distal radius.

Important radiological features to assess

- These parameters give an idea of the severity of the injury, and thus the stability of the fracture; the more features, the more unstable.
- Dorsal cortex comminution.
- Intra-articular extension—radiocarpal and distal radioulnar joint (DRUJ).
- Ulnar styloid fracture (suggests a TFCC injury which is a strong DRUJ stabilizer).
- Loss of radial inclination (normally ~22°).
- Loss of palmar tilt or dorsal angulation (normal tilt ~11°).
- Loss of radial height (~11mm from distal ulna to tip of radial styloid).

Treatment

Children

- Fractures of the distal radius. Usually treated by closed reduction (manipulation) and application of a well-moulded plaster.
- If very unstable in theatre (radial and ulnar complete fractures, displaced) or if the fracture has slipped position in plaster after manipulation, then internal fixation with percutaneous K wires or, more rarely, open fixation with plates and screws may be used.

Adults

Fractures with dorsal displacement/angulation

- Undisplaced + stable. Moulded below-elbow plaster immobilization for 6 weeks.
- Displaced + stable. Closed reduction and moulded below-elbow plaster immobilization for 6 weeks.
- Displaced + unstable. Closed reduction and percutaneous K wire fixation (two or three wires), or ORIF with plates and screws, or external fixation.
- Complex intra-articular fractures or highly unstable patterns can be treated with anatomic pre-contoured distal radius locking plates.

Traditionally, a difficult area to know the best treatment method.

- Take each case on its own merits. There are many patient and fracture factors to allow for.
- Dorsal comminution is a common problem and must be taken into account in the method chosen.
- Bone structural substitutes, e.g. Biobon, lack RCT data to back up their use and have considerable expense.

Fractures with volar displacement ('Smith's fracture')

Unstable and are treated by a volar buttress plate (supports a fracture like a shelf, propping up or supporting the distal fragment).

Intra-articular fractures with volar displacement ('Barton's fracture')

Internal fixation is mandatory as it is a highly unstable fracture.

Ulnar styloid fractures

Do not often require fixation unless the fragment is large, in which case, it may represent a TFCC injury (➲ Fractures of the radius and ulnar shaft, p. 628) treated by internal fixation.

References

Frykman G (1967). Fracture of the distal radius including sequelae—shoulder–hand–finger syndrome, disturbance in the distal radio ulna joint and impairment of nerve function. A clinical and experimental study. *Acta Orthop Scand* Suppl 108: 3.

Salter RB, Harris WR (1963). Injuries involving the epiphyseal plate. *J Bone Joint Surg Am* 45: 587–632.

Fractures of the radius and ulnar shaft

Mechanism

- Commonly a fall onto the outstretched hand or a direct blow injury.
- High energy may be involved; therefore, look closely for neurovascular status and compartment syndrome.
- As displaced or angulated fractures affect the proximal (Monteggia) and distal (Galeazzi) radioulnar joints, it is essential to get orthogonal X-rays of the wrist and elbow.

Fracture types

Children

- Usually transverse fractures of the radius and ulna.
- May be angulated only with one of the cortices still intact ('greenstick fracture').
- Be aware of plastic deformation (no obvious fracture, but bowing of one or both bones).
- May sustain a fracture dislocation as in adults.

Adults

- Usually either a transverse or an oblique fracture of the radius and ulna.
- Isolated ulnar shaft fractures ('nightstick fractures', named after mechanism of defending direct blow from a policeman's nightstick or truncheon).
- Displacement and significant angulation are indications for fixation.
- Remember, the forearm is a 'force parallelogram' and a fracture of only one bone will usually result in a dislocation of the other bone at the proximal or distal joints. These fracture dislocations are:
  - *Monteggia fracture*. Proximal ulnar fracture with dislocation of the proximal radial head.
  - *Galeazzi fracture*. Distal radial fracture with dislocation of the DRUJ.

Treatment

Children

- Greenstick fractures. Closed reduction and cast immobilization from wrist to above the elbow.
  - In-line traction is always the key to any initial reduction and often all that is required to realign, given patience.
  - Use minimal force. If the periosteal hinge is broken during reduction, the fracture may displace completely and become unstable.
- Plastic deformation needs to be corrected.
- Displaced fractures are often unstable and can be treated by ORIF with plates and screws or by flexible intramedullary nail fixation.
- Fracture dislocations (Monteggia and Galeazzi). Closed manipulation and cast immobilization (failed reduction may require open reduction).

Adults

- Usually impossible to achieve or maintain a closed reduction for adult forearm shaft fractures.
- Undisplaced fractures can be managed in an above-elbow cast.
- Displaced fractures are treated with open reduction and compression plate fixation.
- 'Nightstick fractures' of the ulna are splinted by the intact radius, so if undisplaced, then early protected motion with an elbow cast–brace is indicated.
- If the fracture is displaced (>50% displacement or >10°), open reduction and compression plate fixation should be used.
- Fracture dislocations. Treated with ORIF to accurately reduce and hold the associated dislocations.

Complications

- Mal-union or non-union. Close follow-up of closed, manipulated fractures. An X-ray at 1 and 2 weeks is mandatory to watch for slip of position. Mal-union can present with functional problems with forearm rotation.
- Non-union is normally treated by open reduction, debridement of the non-union site, and compression plate fixation with or without bone grafting.
- It is not usually necessary to remove metalwork from the radius and ulna, unless they cause significant problems after the fracture has healed. Radial plate removal has been associated with a significant risk of neurovascular complications.

Fractures and dislocations around the elbow in children

- Second commonest injury in children (8% of childhood fractures).[1]
- Cause is usually a fall onto the outstretched hand. The result is related to age:
  - <9y, supracondylar fracture of the humerus.
  - >10y, dislocated elbow.
  - >12y, shoulder injuries.
- Salter–Harris injuries of the elbow occur through the lateral condyle and radial neck.

Supracondylar fractures

Types

- Based on the mechanism of injury, *extension type* (~95%), and *flexion type* (5%).
- Classified using the modified *Gartland system:*[2]
  - Type I. Undisplaced.
  - Type II. Angulated/displaced, but posterior cortex is intact, acting as a hinge.
  - Type III. Complete displacement.
  - Type IV. Completely displaced and unstable in flexion and extension.

Treatment

Displaced supracondylar fractures (types III/IV) are an orthopaedic emergency, especially if complicated with an absent distal pulse. Do not delay.

- Assess neurovascular status and document beforehand.
- Reduce under GA by straight arm traction (up to 5min may be required).
- Then manipulate to correct rotation, varus/valgus tilt, and finally any extension deformity.
- Try to flex the elbow up past 90°, with the forearm pronated (may be difficult due to anterior soft tissue swelling; the reduction technique itself can cause loss of pulse in the flexed position).
- Displaced (type III/IV) fractures should be reduced and stabilized with K wires. Some advocate the same for type II injuries.
- Common configuration is two crossed condylar K wires, one medial (beware of ulnar nerve) and one lateral used to fix the fracture.
- An above-elbow cast is then used to supplement fixations, and wires are removed at 4 weeks.
- Long arm traction may be used as definitive treatment but involves a long inpatient stay until the bone has united (usually 3 weeks).

Undisplaced fractures (type I) can be treated with a collar and cuff with or without a plaster backslab.

Complications

Vascular

- Injury to the brachial artery is rare, as the pulse usually returns after fracture reduction.
- Examination is the key. An absent pulse with a well-perfused hand does not require any immediate vascular management; however, a pulseless, cold hand or a pulse that is lost post-reduction and pinning does!
- True loss of the radial pulse may be due to:
  - *Vascular spasm*. Typified by good capillary refill after reduction, but slow return of the pulse. Failure of pulse return may be due to other injuries and requires a vascular surgical opinion. Partial injury (endothelial flap) is treated by direct repair.
  - *Complete transection or disruption*. May be treated by direct repair or, more often, interposition vein graft.
- Contracture. Untreated vascular injury will result in fibrosis and contracture of the forearm ('Volkmann's ischaemic contracture'). This is a devastating and debilitating condition and should be avoidable with early (<12h) exploration and/or repair of vascular damage.

Neurological

- Neuropraxia is commonest, with gradual recovery. May involve:
  - Radial nerve.
  - Anterior interosseous (branch of medial nerve).
  - Median nerve and, rarely, ulnar nerve.

Mal-union

- Incorrectly reduced fractures will not remodel and can lead to cubitus valgus and a 'gunstock deformity'. Much less common with K wire fixation.
- Recurvatum common following cast management of type II/III; remodels poorly.

Lateral condyle fractures

Types

Classified according to Milch, depending on how much of the intra-articular surface is involved:

- *Type I*. Fracture through growth centre of capitellum (Salter–Harris type IV).
- *Type II*. Fracture medial to growth centre and can involve trochlea (Salter–Harris type II).

Treatment

- Displaced fracture. ORIF with either two cannulated screws or two K wires.
- The fragment is always considerably larger than expected from the X-ray due to the condyle not being fully ossified.
- If not reduced and fixed, the fragment will displace. This is due to the pull of the wrist extensors arising from the lateral epicondyle. This will lead to a cubitus valgus deformity and can present in later life with an ulnar nerve palsy, as it has been chronically stretched ('tardy' ulnar nerve palsy).

Medial condyle fractures

- Not to be confused with epicondyle fractures.
- These fractures occur in a similar pattern to the lateral condyle (types I and II).
- Treatment is essentially as described for lateral condyle fractures.
- The key is recognition of this injury as it is intra-articular and, if missed, can be associated with valgus instability of the elbow and subluxation.

Epicondyle fractures

Medial epicondyle fractures Avulsion-type injuries of the apophysis. High association with elbow dislocations. The fragment can remain undisplaced or displaced, or become trapped in elbow joint. Treatment is usually conservative in a long arm cast. Surgery is indicated for trapped fragments.

Lateral epicondyle fractures Essentially the same as medial; treated with a long arm cast, unless fragment entrapped in joint.

Radial head and neck fractures

- Usually result from a valgus force to the elbow, associated with dislocation or fractures (Monteggia).
- Fractures of the neck are often angulated or displaced, or both.
- Head fractures are of the Salter–Harris type.
- Fractures associated with dislocation happen at the time of injury or as a result of reduction (radial head pushed into ulnar–humeral joint).

Treatment

- <30°. Angulation acceptable; sling is provided and early mobilization.
- 30–60°. Reduction should be attempted, but ongoing debate.
- >60°. Reduction is required under GA, usually stable; once reduced, do not require any further fixation.
- Occasionally open reduction is required.
- Intra-articular fractures that are displaced may require fixation with K wires or screws.

Olecranon fractures

Can often be difficult to spot. The proximal epiphysis appears between 8 and 10y. Isolated fractures do occur but are more commonly associated with fracture dislocations of elbow/radial neck.

Treatment

- Undisplaced fractures require a cast in extension (removes pull of triceps) for 4 weeks.
- Displaced fractures or those associated with dislocation require ORIF with tension band wiring.

References

1 Wenger DR, Pring ME, Pennock A, Upsani V (2017). *Rang's Children's Fractures*, 4th edn. Lippincott, Williams, and Wilkins, Philadelphia, PA.

2 British Orthopaedic Association. *BOAST 11: supracondylar fractures of the humerus in children*. Available at: https://www.boa.ac.uk

Fractures of the humeral shaft and elbow in adults

Humeral shaft fractures

Mechanism

- Usually as a result of a fall with direct blow or torsional forces.
- Can be low energy (osteoporotic) or high energy (younger age group).
- X-rays of joints (above and below) important to rule out intra-articular extension. High-energy injuries, especially to rule out floating elbow or shoulder.
- Remember to evaluate neurovascular status (radial nerve at risk).

Types

- Transverse, oblique, spiral, multifragmentary.
- Distal third fractures associated with radial nerve palsy, known as Holstein–Lewis fracture.

Treatment

Conservative management

Is the mainstay.

- Initial sugar-tong cast for 1–2 weeks, then convert to functional brace until union (usually by 3 months); requires regular clinic evaluation and tightening of brace.
- Can accept 20° anterior/posterior angulation, 30° varus/valgus angulation, 15° rotations, and 1–3cm of shortening.
- Remember to mobilize elbow and shoulder or will stiffen!

Surgical treatment Indicated if there is an open fracture, vascular injury, associated intra-articular fracture, floating joint (proximally or distally), or pathological fracture.

- Relative indications include multiple injuries, inability to maintain reduction closed, segmental fractures, or transverse fractures in young athletes.
- ORIF with plate and screws (in compression) is commonly used. Locking plates can be utilized if poor bone quality.
- Intramedullary nailing is an alternative, good for pathological or segmental fractures or for medically labile patients due to small exposure. It is associated with shoulder pain and rotator cuff dysfunction.

Humeral condylar fractures

Mechanism

Usually due to impaction injury (the olecranon driven into the humerus via a direct fall and the condyle usually splits into a 'T'- or 'Y'-shaped pattern). Pattern depends upon bone quality and angle of flexion at time of injury. Careful neurovascular evaluation required.

Types

- Intercondylar fracture (commonest). The fracture line extends from the articular surface to the supracondylar region in a 'T'- or 'Y'-shaped pattern.
- Supracondylar.
- Isolated medial or lateral condyle fracture.
- Isolated capitellum fracture.

Treatment

Intercondylar

- Intra-articular fractures. The principles are open anatomical reduction with absolute stability, providing stability to allow early mobilization.
  - Posterior approach with either a triceps split or, if more complex, a trans-olecranon osteotomy to visualize the articular surface.
  - Fixation by plate and screw constructs (reconstruction plates). Compression across the intra-articular segments may be required. Pre-contoured distal humerus locking plates more commonly used.
- Can be difficult to treat if heavily comminuted and the bone quality is poor.
  - An option is conservative management in cast, but early mobilization to try to maintain as much function as possible ('bag of bones' technique).
  - Non-union is not uncommon following these fractures, and sometimes salvage surgery in the form of elbow replacement may be considered.
  - In the elderly with a low fracture pattern, 1° elbow replacement is sometimes used.

Supracondylar

- Conservative management in a cast if undisplaced or highly comminuted in the elderly. Mobilize at 2 weeks in hinged brace. Cast. Discontinue when healed (6–8 weeks).
- ORIF if displaced. 90/90 plating was the classical method (medial and posterolateral), but now utilize pre-contoured, bicolumnar locking plates. Again stable fixation with early mobilization is the goal.

Transcondylar

- A very distal fracture and within the joint capsule.
- Management follows same principles as for supracondylar type.

Capitellum

- Radiographs. The capitellum aligns with the radial head on AP and lateral views. Classification dependent on size of fragment (best seen on lateral).
  - Type I is a large osseous fragment, often involving the trochlea.
  - Type II is a thin articular fragment with little osseous composition.
  - Type III is multifragmentary.
- Treatment. Type I requires ORIF. A common technique is headless compression screws from a posterior to anterior direction. Type II is usually not amenable to fixation and is excised, as are the loose components of type II injuries.
- Kocher's approach (interval between anconeus and extensor carpi ulnaris) often used.

Olecranon fractures

Mechanism

A fall onto the point of the elbow, but can occur as a fall onto the outstretched hand where the triceps avulses the olecranon process.

Colton classification

- *Type I*. Undisplaced (<2mm separation, able to extend elbow against gravity).
- *Type II*. Displaced (subtypes: IIA, avulsion; IIB, oblique/transverse; IIC, comminuted; IID, fracture–dislocation).

Treatment

- Undisplaced. Place in a cast at 90°. At 4 weeks, begin mobilization.
- Displaced. ORIF with tension band wiring technique or pre-contoured locking plate.
- Comminuted. Plate and screw ± bone graft with pre-contoured locking plate.

Radial head fractures

Mechanism Fall onto the outstretched hand, forearm in pronation.

Mason classification

- *Type 1*. Minimally displaced.
- *Type 2*. Displaced.
- *Type 3*. Comminuted and displaced.

Treatment

- *Type 1* (<3mm displacement). Sling or half cast for 1–2 weeks, then mobilization (aspiration of the joint haematoma acutely can give pain relief and injection of LA can rule out any mechanical block).
- *Type 2* (displaced, mechanical block to motion or part of more complex injury pattern). ORIF with compression screw and/or mini-plate.
- *Type 3* (too comminuted to allow ORIF). Radial head replacement indicated. Excision considered at a later stage and contraindicated if other destabilizing ligamentous injuries.

Complications of elbow fractures

- Joint stiffness (rotational).
- Non- or mal-union.
- Degenerative joint disease (osteoarthritis).
- Heterotopic ossification.
- Neurovascular injury and its sequelae.
- Compartment syndrome

References

Colton CL (1973). Fractures of the olecranon in adults: classification and management. *Injury* **5**: 21–9.

Sarmiento A, Zagorski JB, Zuch GA, *et al*. (2000). Functional bracing for the treatment of fractures of the humeral diaphysis. *J Bone Joint Surg Am* **82**: 478–86.

Dislocations and fracture dislocations of the elbow

Mechanism

- *Simple*. No bony component.
- *Complex*. Associated bony injury.
- *Posterior and posterolateral*. Commonest type; fall onto an outstretched hand, with the elbow in extension or slight flexion (with supination and valgus forces).
- *Anterior (rare)*. Fall onto a flexed elbow or as a direct blow from behind ('side swipe injury').
- *Divergent (rare)*. Radius and ulna separated proximally.

Associated fractures (complex injury)

- Elbow stability dependent on bony and ligamentous component integrity—radial head, coronoid, olecranon, medial and lateral collateral ligaments.
- Coronoid fractures. Occur as distal humerus is driven against it during subluxation, dislocation, or instability. Any injury to the coronoid suggests an episode of instability.
- Regan and Morrey classification.
  - Type 1, tip fractured (consider anterior capsule injury).
  - Type 2, <50% of the process.
  - Type 3, >50% of the process.

Treatment

- Aim to reduce under GA to allow thorough assessment of stability.
- In-line traction. Supinate forearm (clears coronoid); flex elbow from an extended position, whilst pulling olecranon in an anterior direction.
- Check elbow stability once reduced, and X-ray to confirm.
- If stable, collar and cuff at 90° for 7–10 days, with early motion.
- If unstable (redislocates), place forearm in pronation if lateral collateral ligament disrupted or supination if medial collateral ligament disrupted, and immobilize in above-elbow cast for 2–3 weeks.
- If there are associated injuries, then most fractures will require ORIF to aid stability and allow early mobilization. This may include radial head prosthetic replacement.

The 'terrible triad'

- Posterior elbow dislocation associated with radial head fracture, coronoid process fracture, and lateral collateral ligament tear.
- The elbow will require surgical stabilization via ORIF of coronoid and anterior capsular repair, ORIF or replacement of radial head, and lateral collateral ligament repair.

Complications

- Neurovascular injury.
- Compartment syndrome.
- Chronic elbow instability.
- Articular cartilage damage.
- Heterotopic calcification.
- Stiffness (especially extension). Early motion at 1 week to try to prevent this.

Further reading

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Fractures around the shoulder

Clavicle

Mechanism

Fall or direct blow to lateral shoulder (5–10% of all fractures).

Types

- Occurs in the middle (75%), lateral (20%), or medial (5%) third.
- The pattern of lateral third fractures depends on relationship and integrity of the coracoclavicular ligaments and involvement of the acromioclavicular joint (ACJ).
- Medial third fractures are assessed with displacement and involvement of sternoclavicular joint in mind.

Treatment

- *Middle third*. Virtually all can be treated conservatively with a broad arm sling (not a collar and cuff). Indications for ORIF (plate and screws) are open fractures, significant neurovascular injuries, skin tenting, and floating shoulder. Fractures with >100% displacement and 2cm of shortening may have better outcomes with ORIF.
- *Lateral third*. Undisplaced can be treated non-surgically; however, the presence of displacement suggestive of coracoclavicular ligament disruption will require fixation with plate and screw constructs, or hook plate, or ligament reconstruction (Weaver–Dunn procedure).
- *Medial third*. Most are undisplaced and treated conservatively in a sling. Displacement, especially posterior, into the root of the neck may warrant surgery.

Complications

- *Metalwork*. Failure or subcutaneous irritation.
- *Non-union*. Associated with displacement and shortening.
- *Acute complications*. Neurovascular injury (including brachial plexus injury), neurovascular compression (costoclavicular syndrome), and pneumothorax from bony penetration of the pleura.

Scapula

Mechanism Direct trauma, usually a high-velocity injury such as an RTA. Always have a high clinical suspicion of other possible injuries such as rib fracture, pulmonary contusion, and pneumo-/haemothorax; 20–40% have ipsilateral clavicle fractures.

Treatment

- Simple (no involvement of the glenoid—glenohumeral joint)). Adequate analgesia (very painful injury; may require HDU admission) and early mobilization.
- Complex (involving the glenoid and glenoid neck). May need ORIF after further imaging such as CT/MRI scanning.
- Floating shoulder will require ORIF of clavicle.

Proximal humerus

Mechanism

Young—high-energy injury; elderly—low-energy falls. Full neurovascular assessment (especially axillary nerve) is essential, alongside pre-injury function (aids management decision).

The Neer classification

Based on Codman's fracture lines along old physeal scars; four segments or parts. A fracture part is considered when it is >1cm displaced or >45° angulated. Thus, defined as 1-, 2-, 3-, or 4-part fractures.[1]

Treatment

- Undisplaced or impacted. Collar and cuff, with early pendular mobilization.
- Displaced. Can be treated non-operatively or operatively, depending on the pattern of fracture.[2] ORIF using anatomical 'locking' plate or proximal humeral intramedullary nail; less commonly, cannulated screws, K wires with or without tension band wiring.
- Severely comminuted fractures (4-part), especially including fracture dislocations, have a high rate of AVN; usually treated with hemiarthroplasty or reverse polarity shoulder replacement and soft tissue reconstruction of the rotator cuff to the prosthesis.

Complications Non- and mal-union, avascular necrosis of the humeral head, and osteoarthritis of the shoulder joint are the commonest. High-velocity injuries may also cause neurovascular injuries, particularly of the brachial plexus.

Paediatric humeral fractures

- Usually occur at the surgical neck or through and around the proximal humeral epiphysis.
- May be indicative of NAI.
- Most require no treatment apart from collar and cuff with mobilization, as for adults. Remodelling potential is good in this area.

References

1 Neer CS (1970). Displaced proximal humeral fractures. I. Classification and evaluation. *J Bone Joint Surg Am* **52**: 1077–89.

2 Rangan A, Handoll H, Brealey S, et al. (2015). Surgical vs nonsurgical treatment of adults with displaced fractures of the proximal humerus. The PROFHER Randomized Clinical Trial. *JAMA* **313**: 1037–47.

Dislocations of the shoulder region

(See Fig. 16.1.)

Sternoclavicular joint

- Uncommon injury.
- *Mechanism*. Indirect force to lateral shoulder or direct impact on medial end of clavicle.
- *Types*. Usually dislocates anteriorly; posterior dislocation is rare. The deformity is at the medial clavicle.
- *Complications*. Tracheal and oesophageal compression may occur with posterior dislocation. Careful assessment is required.

Treatment

- *Anterior dislocation*. Treated symptomatically with a sling, analgesia, and early mobilization.
- *Posterior dislocation* with tracheal compression. Requires closed reduction or open if this fails (with cardiothoracic surgical help).

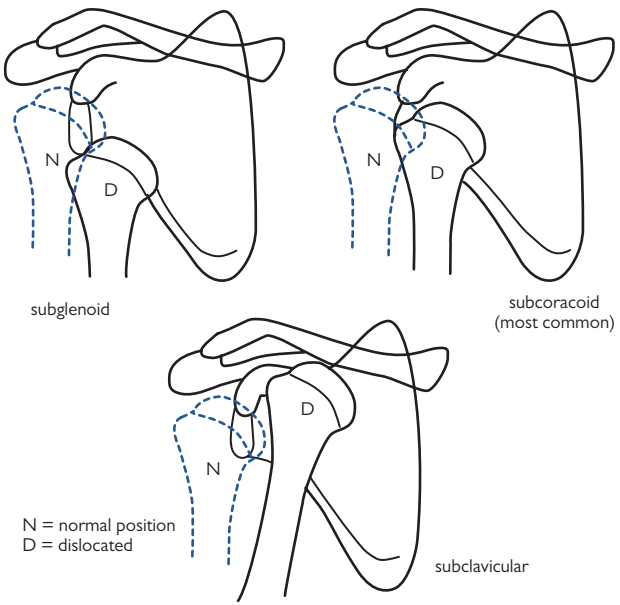

Fig. 16.1 Types of shoulder dislocation.

Reproduced with permission from Collier, J. et al. (2006). Oxford Handbook of Clinical Specialties, 7th edn. Oxford University Press, Oxford.

Acromioclavicular joint

- Usually an injury of second to fourth decade, commoner in ♂.
- *Mechanism*. Fall or direct impact onto the point of the shoulder.
- *Rockwood classification*.[1] Six types with increasing numbers relating to increasing severity of ligamentous disruption (acromioclavicular and coracoclavicular) and displacement.
  - Type I. Sprained acromioclavicular ligament (no displacement).
  - Type II. Acromioclavicular ligaments disrupted, ACJ subluxed.
  - Type III. Acromioclavicular and coracoclavicular ligaments disrupted (>100% displacement).
  - Type IV. Both ligaments disrupted, with posterior displacement.
  - Type V. All ligaments torn and massively displaced.
  - Type VI. All ligaments torn and inferior displacement (very rare).

Treatment

- *Types I and II*. Broad arm sling and early mobilization when pain allows.
- *Type III*. Controversial—treat as above in most cases. Persistent pain or functional limitation is treated by reconstruction of the coracoacromial ligament.
- *Type IV and above*. Acute repair indicated. Soft tissue reconstruction better than hook plate.

Anterior dislocation of the glenohumeral joint

Mechanism

Traumatic event, leading to forced abduction and external rotation (fall onto the outstretched arm).[2, 3]

Associated features

- Young. 90% have traumatic injury to bony and/or soft tissue restraints in the shoulder—the Bankart lesion (anteroinferior glenoid labrum tear, with or without a glenoid rim fracture), Hill–Sachs lesion (impression fracture as the anterior glenoid impacts on humeral head).
- *Rotator cuff tears*. ~30% of those >40y and 80% of those >60y will have a tear.
- *Greater tuberosity fractures*. Common over the age of 50y.

Clinical findings

- History of injury and whether had previous dislocations.
- The shoulder looks 'square' as the deltoid is flat and a sulcus can be visible where the humeral head may be.
- The patient supports the arm which is abducted and very painful.
- Assess neurovascular status (axillary nerve).
- X-rays (AP and axillary or scapular 'Y' lateral views) show the humeral head anterior and inferior to the glenoid. Used to exclude a fracture of the humerus or glenoid.

Treatment

- Reduce as an emergency in A&E.
- Give IV morphine 5–10mg + inhaled N_2O (IV midazolam 5mg is usually unnecessary).
  - Simplest, extremely reliable method, gentle, continued straight line traction with the arm abducted about 10–20° from the trunk. May take 10–15min, but patience is the key, not force.
  - Avoid rotation (such as in a 'Kocher's manoeuvre'), as this is dangerous and may cause fracture of the humerus.
  - Countertraction can be placed across the trunk with a broad sheet.
- Alternative technique is patient prone on the trolley, with arm hanging freely down and weighted (e.g. 3L bag of saline) ('Stimson's technique').
- If there is an associated humeral neck fracture, then the reduction should be done under GA.
- Place the arm in a collar and cuff sling under the clothes. Repeat the X-ray to confirm reduction and that there has been no iatrogenic fracture.
- Always document the neurological status (axillary nerve) before and after reduction.
- Follow-up in clinic mandatory to assess for associated injuries.

Posterior dislocation of the glenohumeral joint

Mechanism

Rare. Due to forced internal rotation or direct blow to the anterior shoulder (e.g. after an epileptic fit or electric shock). Commonly missed.

Features

- The arm is held internally rotated and no external rotation is possible.
- The humeral head should be palpable posteriorly.
- AP X-rays may show the humeral head as a 'light bulb' shape (internally rotated), but this is not diagnostic of posterior dislocation.
- Lateral X-ray shows the dislocation.

Treatment

- In-line traction method (as above), but consider GA if difficult—avoid excessive force.
- May be very unstable; occasionally, the 'broomstick' plaster may be used.

Recurrent dislocation of the shoulder

- Usually due to a Bankart lesion or capsular redundancy (stretched and floppy).
- Commonest in young age of first dislocation (90% recurrence if <20y, 60% if 20–40y, <10% if older than 40y).

Treatment—surgery

- Repair and fixation of the anterior 'Bankart lesion'.
- May be done open or arthroscopically.
- Capsular laxity is treated by capsular shift, an overlapping 'pants-over-vest' procedure to improve proprioceptive joint sensation.

References

1 Tornetta P, Court-Brown CM, Heckman JD, McKee M, McQueen MM (2014). *Rockwood and Green's Fractures in Adults*, 8th edn. Lippincott, Williams, and Wilkins, Philadelphia, PA.

2 Robinson CM, Dobson RJ (2004). Anterior instability of the shoulder after trauma. *J Bone Joint Surg Br* **86**: 469. [review]

Fractures of the ribs and sternum

Mechanism

- Single rib fractures occur as a result of direct injury such as a fall.
- Fractures of the lower ribs can occur with coughing.
- Sternal fractures occur with direct injury, e.g. contact with steering wheel or restraint by a seat belt.
- High-velocity (RTA) or large crush injuries can result in a 'stove-in' chest with a flail segment, i.e. multiple rib fractures, each fractured at two sites.

Treatment

- *Single rib fracture*. Symptomatic with analgesia.
- *Multiple rib fractures*. If ≤3 ribs involved, should admit for observation overnight, but treatment symptomatic with analgesia and chest physiotherapy.
- *Sternal fracture*. Symptomatic treatment, but observe for associated injuries (see below).
- *Flail chest or extensive multiple rib fractures*. Potentially life-threatening injury and may present with severe respiratory distress.
  - Flail segments move paradoxically, preventing adequate ventilation.
  - Multiple fractures restrict respiratory effort, severely impairing ventilation.
  - Treat with high-flow O_2 and analgesia. CPAP and even IPPV may be required.
  - Consider rib fixation—regional referral centres/major trauma centres.

Complications

Incidence of complications rises dramatically if the injury involves:

- >3 ribs.
- First, second, or third rib.
- Sternum.
- Scapula.

They are all indicators of high-energy transfer injury.

Cardiac tamponade

- Bleeding into the pericardial cavity causes severe haemodynamic shock and may be the cause of a cardiac arrest at presentation.
- Diagnosis is by high clinical suspicion, muffled heart sounds, raised JVP, and no signs of a tension pneumothorax.
- Treat by immediate pericardiocentesis, with transfer to cardiothoracic unit for repair of the defect.

Pneumothorax

- Usually due to direct pleural injury by bone fragments during injury.
- Often associated with haemothorax.
- Signs are respiratory distress, absent breath sounds, hyperresonant percussion note (pneumothorax), or shock.
- Tension pneumothorax is life-threatening and requires immediate decompression via a 16G needle placed into the second anterior intercostal space, followed by definitive chest drain placement.
- If in any doubt, always treat first on clinical grounds, rather than wait for investigations (X-rays, etc.).

References

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British Orthopaedic Association. *BOAST 15: the management of blunt chest wall trauma*. Available at: www.BOA.ac.uk

Fractures of the pelvis

Mechanism

- *Age <60y*. High energy—RTA or falls at work (building sites) or sport (horse riding).
- *Age >60y*. Low energy (insufficiency fracture)—fall from standing height.

The force required to fracture the pelvis in the young is considerable and as a result, morbidity and mortality can be as high as 20%. It is the main cause of death in multiple trauma patients.

Types

- The pelvis is a ring consisting of two innominate bones and the sacrum.
- Anteriorly are the ligaments of the symphysis pubis and posteriorly, the ligaments to the sacrum (sacrospinous, sacrotuberous, and sacroiliac).
- An isolated break at any part is generally stable (the ring will not separate).
- Two breaks in the ring make it unstable (able to displace or open). Remember, this can be due to a fracture or ligament disruption!
- Young and Burgess classification (descriptive):[1]
  - AP compression (impact from the front or rear).
  - Lateral compression (impact from the side).
  - Vertical shear (usually a fall from height).
  - Combined mechanical (mixture of all of the above).

Assessment

- ATLS approach.
- Mechanism of injury important as gives insight into degree of injury.
- Assessment and documentation of other injuries is mandatory.
- Look for neurological, GI, and GU injury.

Treatment

Initial treatment of all pelvic fractures should include ATLS protocols. Once stable, an AP pelvis X-ray, supplemented with inlet and outlet views, is required.

A CT scan is helpful to assess the posterior pelvic structures that can be obscured on a plain X-ray.

Single ring fracture (stable)

- Check for an occult sacroiliac ligament injury (local bruising and tenderness with pain on stressing the joint); this suggests the injury could be unstable.
- Fractures of both superior and inferior pubic rami on the same side are a single break in terms of ring stability.
- Other stable patterns include ilium fractures into sciatic notch or sacrum.
- Remember that if a significant single break in the ring is present, there is a chance that concurrent ligamentous injury exists. Thus, a CT scan is often required prior to mobilization to confirm stability.
- Isolated fractures of the ilium, ischium, or pubis are normally treated with bed rest, analgesia, and early mobilization as soon as the pain allows.

- Fractures that extend to the acetabulum (joint) require further investigation to plan treatment but are usually stable if isolated.
- Simple pubic rami fractures in the elderly (osteoporotic) can be treated conservatively with bed rest and early rehabilitation.

Multiple ring fractures (unstable)

Unstable fractures are liable to massive haemorrhage within the soft tissues of the pelvis. This is mostly because the pelvic ring is grossly displaced during the injury and tearing of the extensive posterior pre-sacral venous plexus occurs. A patient's entire blood volume can be lost, hence the high mortality rate.

Establish haemodynamic control

- Approach patient according to ATLS guidelines (ABCDE).
- Haemorrhage is leading cause of death with pelvic injuries.
- Tachycardia and hypotension suggest bleeding.
- Establish at least two large-calibre IV infusions and commence resuscitation with warm crystalloid (2000mL), and then reassess.
- Send bloods for urgent cross-match of 4–6U. O –ve blood is given if no response to initial fluid challenge.
- Continued bleeding must be controlled by reducing the pelvic volume. This may be achieved by:
  - Pelvic binder or binding a bedsheet tightly around the pelvis and internally rotating the hips—very effective as an emergency procedure.
  - Application of an external pelvic fixator—a more definitive solution, but should be done in the trauma theatre.
- Laparotomy is contraindicated, unless there are life-threatening intra-abdominal injuries that must be treated, since this effectively 'decompresses' the pelvis again superiorly.
- Urethral injury occurs, especially with anterior compression bony injuries. If there is blood at the urethral meatus, perineal bruising, haematuria, or a high-riding prostate on rectal examination, then catheterization should only be performed by an experienced urologist (very often suprapubically). Investigation for injury is with a retrograde urethrogram or IVU once the patient is stable.

Definitive management

- Significant AP compression mechanism can result in an 'open book' pelvis. Severe subtypes of the lateral compression or vertical sheer fractures are very unstable and associated with other injuries.
- Once ATLS stabilization is complete, CT scanning is required to define the fracture and plan treatment.
- External fixation is excellent to temporarily control unstable fractures and manage definitively. It can help to control haemorrhage in a haemodynamically unstable patient.
- Once stable, liaise with local pelvic fixation centres to arrange definitive fixation if required.
- ORIF with screws and plates for the symphysis pubis of ilium fractures. Posterior pelvic instability involving the sacrum requires screw fixation.

Complications

- Haemorrhage, shock, and death from exsanguination.
- Open fractures carry 50% mortality and need to be treated aggressively by both orthopaedic and general surgical teams.
- Urogenital injury.
- Thromboembolism (35–50% develop DVT and 10% PE).
- Neurological injury.
- Paralytic ileus.
- Mal-union may lead to difficulty with pregnancy.
- Osteoarthritis.

Acetabular fractures

- Usually high-energy injury in the young from RTA (dashboard impact) or fall from a height. Associated with hip dislocation.
- Assess as per all pelvic fractures (i.e. ATLS).
- X-rays required include AP pelvic views plus Judet views (45° internal and external views); CT is routine.
- Letournel–Judet classification. Fractures as posterior wall, posterior column, anterior wall, anterior column, or transverse.
- Non-surgical management is reserved for fractures that are undisplaced (except posterior wall as hip unstable) and do not involve the 'dome', the superior acetabular roof (weight-bearing area).
- Surgical management, ORIF, in displaced dome fractures, fractures resulting in joint instability, or trapped intra-articular fragments.

Sacral fractures

- High-energy injury in the young or low-energy in the elderly.
- Assess as per all pelvic fractures (i.e. ATLS). Must assess for sacral nerve root injury.
- X-rays include AP pelvic (inlet and outlet) views; CT is required as difficult to fully appreciate on X-ray.
- Denis classification. Alar lateral to foramen, involving the foramen, or central portion medial to foramen.
- Non-surgical management is reserved for fractures that are undisplaced or impacted.
- Surgical management if displaced or loose fragments impinging on nerve roots.

Reference

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British Orthopaedic Association. *BOAST 14: the management of urological trauma associated with pelvic fractures.* Available at: https://www.boa.ac.uk

Femoral neck fractures

Mechanism

- Commonest fracture in the elderly with an exponential increase in incidence with age.[1]
- The risk increases with decreasing bone mass (osteoporosis).
- In the elderly, usually result from a trip/fall onto side (low energy).
- Fractures in the young are usually a result of high-energy trauma.

Classification

Fractures should be described anatomically:

- *Intracapsular fractures*. Occur within the joint capsule (proximal to the intertrochanteric line).
- *Extracapsular fractures*. Occur distal to the joint capsule (involving or distal to intertrochanteric line).
- It is then important to describe fractures as displaced or undisplaced.
- The relationship to the blood supply is key:
  - The femoral head receives its supply via the medial and lateral femoral circumflex arteries, which form the extracapsular ring and give rise to the cervical arteries (the lateral being most important). There is also supply via the intraosseus nutrient vessels and the ligamentum teres.
  - Displaced intracapsular fractures disrupt their blood supply and have a high rate of AVN of the femoral head and non-union.
  - Extracapsular fractures maintain the blood supply to the head (thus reduced AVN and generally heal well).
- Other classification systems are not generally required.
- *Subtrochanteric fractures*. Occur below the level of the lesser trochanter. May be through an area of pathological bone (metastasis) or high energy in young.

Assessment

- Inability to weight-bear following a fall in the elderly is a common presenting feature.
- Consider medical cause of fall (stroke, MI, etc.).
- Other comorbidities essential to ascertain.
- Pre-injury level of function and home circumstances important.
- If the fracture is displaced (common), the leg will be shortened and externally rotated. Straight leg raise and hip movements are globally inhibited by pain. Neurological state important.
- Most fractures do not require any temporary stabilization; however, subtrochanteric fractures may benefit from a Thomas splint for pain relief.
- X-rays. AP pelvis and lateral of affected hip (long leg views if history of malignancy to look for metastases).
  - Displaced fractures are usually obvious.
  - Undisplaced intracapsular compression fractures may be difficult to see on X-ray. If high clinical suspicion of fracture, then further investigation is warranted; isotope bone scan (highly sensitive, poor specificity), MRI (gold standard), or CT scan.

Treatment

The majority of hip fractures require either surgical stabilization or replacement (see below) to allow early mobilization and prevent displacement. This will reduce the risks of long periods of immobilization and bed rest (pressure sore, DVT, UTI, chest infection).

Consider the NICE guidelines on fractured neck of femur management.

In the UK, a tariff is offered for best clinical practice related to hip fractures. Six criteria are measured (all patients aged 60 and above):

1. Time to theatre (all cases) <36h—36h from arrival in emergency department (or time of diagnosis if an inpatient) to the start of anaesthesia.
2. Admitted under the joint care of a consultant geriatrician and a consultant orthopaedic surgeon.
3. Admitted using an assessment tool agreed by geriatric medicine, orthopaedic surgery, and anaesthesia.
4. Assessed by geriatrician in perioperative period (defined as 72h of admission)—geriatrician defined as consultant, specialty and associate specialist (SAS), or specialty trainee year 3 (ST3) and above.
5. Post-operative geriatrician-directed:
   a. Multi-professional rehabilitation team.
   b. Fracture prevention assessments (falls and bone health).
6. Pre- and post-operative Abbreviated Mental Test Score (AMTS).

Intracapsular

- *Undisplaced impacted in the elderly*. Treated by early mobilization with analgesia; 15% late displacement rate, requiring operative intervention.
- *Undisplaced*. Treated by internal fixation with either cannulated screws or a 2-hole dynamic hip screw (DHS).
- *Displaced*. Treated by hemiarthroplasty.[2]
- Total hip arthroplasty can be used if symptomatic pre-existing arthritis or those with few comorbidities and high functioning (controversial).
- In children or young adults, reduction (open or closed) and fixation is employed.

Extracapsular

- Closed reduction on the traction table and open fixation with the use of a DHS.
- Intramedullary hip screw (IMHS) may be used in 4-part fractures.
- Subtrochanteric and reverse obliquity fractures require stabilization, utilizing an intramedullary nail or a fixed-angle plating system.

Complications

- Overall mortality in the elderly is 10% at 30 days; 20% at 90 days. This is indicative of the fact that the fracture is more a marker of generally poor condition, rather than due to acute surgical perioperative complications.
- AVN of the femoral head.
- Dislocation of arthroplasty.
- Loss of fixation.
- Non-union.
- Lower limb thromboembolic disease.

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Femoral shaft fractures

Mechanism

- High-energy RTA in young adults (dashboard injury) or fall from height.
- Stress fractures.
- Low energy in elderly.
- Pathological (metastases).

Classification

- AO system can be used but is complex.
- Anatomical description is the simplest:
  - Location (proximal, mid- or distal shaft, divide into thirds, or metaphyseal or diaphyseal).
  - Configuration (transverse, oblique, spiral, segmental, comminuted).
  - Number of fragments.

Associated injuries

- Polytrauma is common. Look for head, chest, and abdominal injuries.
- Ipsilateral femoral neck fracture (up to 5%).
- Pelvic and acetabular fractures.
- Knee joint injuries. Both bony or ligamental, e.g. anterior cruciate ligament (ACL) rupture.
- Soft tissue injury to skin, muscle, and neurovascular structures.
- Sciatic nerve traction injury (uncommon).
- Bilateral femoral shaft fractures associated with 25% mortality.

Treatment

- *ATLS* (ABCDE).
- Establish two large-calibre IV access and give 2000mL of crystalloid; initial haemodynamic compensation is common in the young and may hide a large blood loss. Can lose up to 4U (1500mL) of blood into tissues around a femoral fracture.
- Send bloods for FBC, U&Es, and group and save.
- Realign and splint the leg with skin traction and a Thomas splint. This will help to control pain and haemorrhage.
- *An X-ray of a femoral fracture not in a splint should never be seen! Diagnose clinically; splint, then get the X-ray.*
- If an 'OPEN' fracture, photograph the wound and socially clean it; place a povidone-iodine dressing over it, and stabilize it. Commence IV antibiotics and tetanus toxoid if required.
- Full 2° survey, looking for associated injuries.

Children

- Nearly always heal and remodel.
- Age 0–2y. Treat in gallows (suspension) traction until callus seen (2–4 weeks) or Pavlik harness/hip spica.
- Age 2–6y. Treat with closed manipulation and hip plaster spica (allows discharge) or continuation of the Thomas splint.
- Age 6–14y. Options are a flexible intramedullary nail ('elastic' nail), ORIF with a plate and screws, or external fixation.
- Age >14y. Can consider locked intramedullary fixation.

Adults

- Non-operative treatment with traction if patient too sick for surgery (be aware of complications: pressure sores, DVT, etc.).
- Locked and reamed intramedullary nailing is the common treatment regime (provides rotational stability).
- Plate and screw construct can be used if there is distal metaphyseal extension. Usually much larger exposure.
- Temporizing external fixation is occasionally required in damage control scenarios. This can be exchanged for a nail once patient stable enough.

Complications

- Compartment syndrome.
- Fat embolus (1%) and possible ARDS.
- Infection (5% after open, 1% after closed nailing).
- Non-union.
- Thromboembolic disease.
- Neurological injury.
- Mal-union, rotation being the most symptomatic.
- Pressure sores, bronchopneumonia, UTI on conservatively treated patients.

Further reading

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Fractures of the tibial shaft

Mechanism

- High-energy injuries in young as a result of RTA, sporting injury, or fall from height.
- Direction of force dictates fracture pattern—torsional (spiral), direct blow (transverse or short oblique). Higher-energy patterns suggested by multifragmentary fractures with or without bone loss.
- Soft tissue injuries common as tibia subcutaneous. Be aware of 'OPEN' fractures.

Assessment

- ATLS approach recommended.
- Inspect for angulation, deformity, and malrotation.
- Subcutaneous crepitation may be present or obvious open wound.
- Neurovascular status needs to be assessed and documented.
- Watch for 'compartment syndrome'. Presents as pain, uncontrolled by analgesia and out of proportion to injury. Look for pallor, paraesthesiae, and pulselessness (late signs). Passive dorsiflexion of joint distal to injury stretching the muscles in the affected compartment is usually diagnostic.
- Compartmental pressures can be measured; an absolute pressure of >40mmHg or <30mmHg difference between compartment and diastolic BP diagnostic.
- *This is a clinical diagnosis and requires immediate management via fasciotomies of affected compartments.*

Classification

- AO system can be used but is complex.
- Anatomical description is the simplest:
  - Position (mid-shaft, junction of distal third, etc.).
  - Configuration (transverse, oblique, spiral, segmental, comminuted).
  - Number of fragments.
- Open injuries are normally classified additionally by the Gustillo–Anderson system.[1]

Treatment

There is no one method of treatment that is appropriate for all fractures. There is a continual contentious debate about the pros and cons of different modalities. The best rule is to judge each fracture and associated soft tissue injury on an individual basis and treat appropriately.[2, 3]

Non-surgical (plaster of Paris)

- Used for undisplaced fractures and low-energy displaced fractures in children that can be closed-reduced.
- Long leg cast, with the knee flexed 20° and the ankle in neutral.
- Mobilize non-weight-bearing on crutches, with X-rays weekly for the first 4 weeks to check alignment.
- Start weight-bearing at 8 weeks in a weight-bearing contact 'Sarmiento' cast.
- Union takes around 14–16 weeks.
- *Advantages*. Simple and avoids all operative risks.

- *Disadvantages*. Takes a long time; requires good follow-up and patient compliance. Stiffness at the knee and ankle is common and unstable injuries are very difficult to manipulate and control in plaster alone.

Cast bracing A variation of plaster where Sarmiento plaster is applied from day 1.

Surgical

- Indicated if unable to maintain closed reduction, i.e. >50% displaced, >10° angulated, >10° rotational deformity, >1cm shortening.
- Unstable fracture patterns. Multifragmentary and same-level tibial fractures.
- Open fractures.

Locking plate fixation

- Mostly used for fractures near the joint surface.
- Plates used in the shaft have a high rate of infection and non-union caused by the large soft tissue exposure required.
- *Advantages*. Simple, quick, rapid mobilization, and avoids the need for plaster.
- *Disadvantages*. Risk of infection, non-union, and implant failure.

Intramedullary nailing

- Currently the treatment of choice in most centres, but requires ↑ operating time and experience.
- May be used in compound fractures, especially where soft tissue flaps are required, since it gives relatively unlimited access to the tibia 'fix and flap'.[4]
- Best for mid-shaft fractures and is poor at controlling fractures within 5–10cm of the knee and ankle joints.
- *Advantages*. Early mobilization, quicker rehabilitation than closed methods, soft tissue undisturbed by technique, easy access for flaps.
- *Disadvantages*. Technically demanding, high rate of chronic anterior knee pain (site of nail insertion—not recommended for kneeling profession, e.g. carpet fitters).

External fixation

- Often used in compound fractures as it allows least disturbance of soft tissue. Can be placed in an extremely rigid configuration to allow stability. Rigidity can then be reduced sequentially in outpatients, if required.
- Tensioned wire circlage frames pioneered in Russia (Ilizarov) can be used for difficult fractures around the knee or ankle.
- *Advantages*. Technically simple (not Ilizarov); allows early mobilization, avoids further soft tissue damage.
- *Disadvantages*. Pin site infections common, but usually easily treated. Requires good nursing backup and patient compliance. Pin sites need to be planned carefully with plastic team if flaps used, so as not to compromise soft tissue cover.

Open fractures

- Guided by the British Orthopaedic Association and British Association of Plastic, Reconstructive, and Aesthetic Surgeons guidelines.[5]
- Recommend a multidisciplinary approach in a specialist centre, if possible.
- High-energy patterns of fracture with soft tissue injury (skin loss, degloving, muscle damage or loss, arterial injuries) need to be acted upon promptly.
- Initially ATLS approach.
- Assessment of affected limb. Neurovascular status essential and repeated regularly.
- Give IV broad-spectrum antibiotics (within 3h of injury).
- Treat limb-threatening injuries immediately (vascular or compartment syndrome).[6]
- Remove gross contamination from open wounds, photograph, and wrap in saline-soaked gauze and film dressing. Immobilize the whole affected limb in a splint. Tetanus status checked.
- Combined management approach to plan definitive treatment of fracture and soft tissues. Aim to do within 24h of injury if isolated open fracture.

References

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Further reading

British Orthopaedic Association. *BOAST 4: management of severe open lower limb fractures*. Available at: https://www.boa.ac.uk

British Orthopaedic Association. *BOAST 10: diagnosis and management of compartment syndrome of limbs*. Available at: https://www.boa.ac.uk

Fractures of the ankle

- Commonest fracture of the lower limb.
- Usually low-energy rotational force, resulting in simple to complex configurations.
- The talus rotates in the mortise and produces different patterns, dependent on whether the foot is inverted or everted.
- Axial load causes fracture of the tibial plafond.
- The ankle should be thought of as a ring and stability is conferred by:
  - *Bones*. Medial and lateral malleoli and the talus (form 'mortise').
  - *Ligaments*. Laterally, the tibiofibular ligamentous complex (syndesmosis) and the lateral collateral ligaments (talofibular and calcaneofibular); medially the deltoid ligament.
- Remember that a fracture of the proximal fibula (at the knee) is associated with an ankle fracture or dislocation until proven otherwise.

Assessment

- Initially, ATLS approach.
- Inspect for bruising, swelling, obvious deformity, open wounds, skin tenting, and signs of neurovascular compromise.
- Depending on the degree of injury, some patients may walk in for assessment.
- Remember to examine the whole fibula for proximal tenderness.
- Examine for medial tenderness. The medial injury may be ligamentous only, but this is enough to destabilize the ankle and allow talar shift.
- X-rays are mortise AP (15° internal rotation) and lateral views.

Classification

- AO/Danis–Weber system (based on level of fibula fracture):
  - Type A. Below the syndesmosis.
  - Type B. At the syndesmosis.
  - Type C. Above the syndesmosis.
- The Lauge–Hansen classification is more complex and based upon mechanism of injury. Foot supinated and adducted or externally rotated or foot pronated and abducted or externally rotated.
- A distal tibial fracture involving the joint is known as a pilon fracture.

Treatment

Displaced fracture/dislocation

- A displaced fracture dislocation is an orthopaedic emergency and is always clinically obvious. Displacement is often more than expected due to soft tissue swelling.
- Reduce the fracture immediately in A&E and apply a below-knee backslab before sending the patient for an X-ray. An X-ray of a dislocated ankle should never be seen!
- Check neurovascular status before and after reduction.
- Give plenty of analgesia ± sedation (usually done in resuscitation area).

Stable injuries

- Lateral malleolus only (Weber A or B), with no talar displacement (shift) in the mortise.
- Ensure no medial tenderness exists.

- These can be managed in a well-fitting below-knee cast with the foot at 90°(neutral) or fixed-angle removable orthotic (FARO).
- Obtain a post-cast X-ray to ensure position acceptable.
- Regular follow-up and serial X-rays required to ensure reduction remains.
- Total of 6 weeks' cast.
- Weight-bearing is allowed.
- Some simple Weber A fractures require just a supportive elasticated stocking.

Unstable injuries

- Minimal displacement (≥2mm) is acceptable in the elderly and treated by plaster as above.
- Weber B or C fractures with medial tenderness or talar shift.
- Initially placed into backslab for comfort, elevated, and iced (reduces swelling).
- If too swollen, skin closure is compromised; thus, aim to do within first 24–48h.
- Check for significant blisters around areas of incision.
- ORIF is used, with the lateral fracture reduced and held with a 'lag screw' and 'neutralization' plate and screw construct.
- The medial malleolus is fixed directly with two partially threaded cancellous screws (compression) or if a small fragment, a tension band wiring construct.
- A cast is applied following fixation and the patient remains in this, non-weight-bearing, for up to 6 weeks.
- If unable to control fracture in a cast or too swollen, consider spanning external fixator.

Pilon fractures

- Intra-articular fractures of the tibial plafond due to axial force (high energy).
- Initial management is as for ankle fractures.
- Careful assessment for swelling, skin compromise, blisters, and neurovascular status.
- Standard AP and lateral X-rays required; often a CT scan is needed to define fracture pattern further and plan treatment.
- Consider external fixator prior to CT—'span and scan'.
- Non-surgical management only for undisplaced fractures.
- Surgical management is related to soft tissue status.
- External and internal fixation techniques are applicable.

Further reading

British Orthopaedic Association. *BOAST 12: management of ankle fractures*. Available at: https://www.boa.ac.uk

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Fractures of the tarsus and foot

Talus

Mechanism

- Usually a fall from height or an RTA (high energy).
- Foot forcibly dorsiflexed against the tibia.
- Look for associated ankle fractures.
- Blood supply to the talus often compromised. Talus has limited soft tissue attachments, thus relies on extraosseous vessels, which are easily disrupted.

Assessment

- ATLS.
- Look for associated injuries.
- Look for compartment syndrome of foot.
- Neurovascular status.
- AP and lateral of ankle plus CT.

Classification

- By anatomical site, i.e. head, neck, body, or lateral process.
- 'Hawkins' classification for talar neck fractures (types I–IV, increasing levels of displacement and subluxation with increasing grade).

Treatment

- Body fractures. Treated surgically with ORIF unless undisplaced.
- Neck fractures:
  - *Undisplaced*. Strict non-weight-bearing in below-knee plaster for 6 weeks.
  - *Displaced*. ORIF is required. If dislocated, urgent management required as soft tissue can be compromised.

Complications

- AVN. Rate increases with displacement (10% in type I to 90% in type III).
- Osteoarthritis of tibiotalar and subtalar joints.
- Mal-union.

Calcaneum

Mechanism

- Axial load. Fall from height or RTA.

Assessment

- ATLS.
- Assess for associated injuries. Spinal (thoracolumbar) fracture and upper limb injuries.
- Swelling can be significant. Assess for compartment syndrome acutely.
- AP ankle and lateral plus axial Harris view.
- CT may be required.

Classification

- Extra- or intra-articular.
- Intra-articular fractures involve the subtalar joint and are classified by their CT appearance according to the 'Saunders' system.

Treatment

- Extra-articular or undisplaced intra-articular fractures.
  - Conservative. Elevation, ice, bed rest, and observation of soft tissues overnight.
  - Mobilize non-weight-bearing with a removable splint to stop equinus at the ankle. Early subtalar passive mobilization should be initiated.
- Displaced intra-articular fractures. Operative treatment is still controversial. ORIF is usually delayed 10–14 days for swelling to resolve. Caution is exercised if patient a smoker, advanced age, complex patterns, multiple trauma, compensation, or bilateral fractures.

Complications

- Wound breakdown.
- Mal-union.
- Subtalar arthritis.
- Peroneal tendon pathology.

Tarsometatarsal fracture–dislocations (Lisfranc)

Jacques Lisfranc de Saint-Martin described an amputation technique across the five tarsometatarsal (TMT) joints as a solution to forefoot gangrene 2° to frostbite. This became known as the Lisfranc joint.

Mechanism and assessment

- Direct dorsal force (RTA) or indirect rotational injury to a plantar-flexed and fixed forefoot (foot caught in a riding stirrup and rotation of body around it).
- The Lisfranc ligament runs from the base of the second metatarsal to the medial cuneiform. It is the only link between the first ray and the rest of the forefoot. The recessed base of the second metatarsal also provides bony stability.
- Disruption of the Lisfranc ligament, with or without a bony component, results in incongruity of the TMT joint.
- Neurovascular status and compartment syndrome must be assessed for.
- AP, oblique, and lateral X-rays are required. Consider weight-bearing views.
- The medial cortex of second metatarsal should align with medial cuneiform. Look for 'fleck' sign, suggesting avulsion of Lisfranc ligament.
- These injuries are commonly missed, so a high index of suspicion is required.

Treatment

- ORIF is required for all displaced injuries using screws, plates and screws, and supplementary wires.

Complications

- Foot compartment syndrome in acute injuries.
- Metatarsalgia.
- Post-traumatic arthritis.
- Purely ligamentous injuries have the worse outcome.

Metatarsal and phalanges

Mechanism

- Crushing or twisting injuries (e.g. the foot being run over).
- Fifth metatarsal fracture occurs after an inversion injury and can be mistaken for an ankle fracture if not examined correctly.
- Always be suspicious of compartment syndrome in severe crush injuries.

Treatment

- *Metatarsal fractures*. If minimal displacement or angulations, conservative treatment with mobilization as pain allows. Plaster only if mobilization is too painful. Multiple fractures may require reduction and fixation.
- Non-union of the fifth metatarsal sometimes requires ORIF with grafting if problematic (rare).
- *Phalangeal fractures*. Neighbour strapping.

References

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Sanders R (2000). Displaced intra-articular fractures of the calcaneus. *J Bone Joint Surg Am* **82**: 225–50. [Current concepts review]

Injuries and the spinal radiograph

If a patient complains of central pain in the spinal column after trauma, always obtain radiographs. This should not delay resuscitation, as a spinal fracture can be immobilized and life-threatening problems corrected first.

Spinal injuries can be associated with other injuries and the patient may not be able to communicate this to you because they are:

- Unconscious.
- Intubated.
- Shocked.
- Intoxicated.
- Anaesthetized distal to a cord lesion.

Common injuries associated with spinal trauma are:

- Bilateral calcaneal fractures—thoracolumbar fractures.
- Facial fractures—cervical fracture/dislocation.
- Severe head injury—cervical injuries, especially C1/C2.
- Sternal dislocation—thoracic spine fracture.
- Ankylosing spondylitis—cervical and thoracic fractures.
- Cervical fracture—10% rate of fracture at another level.

An awake, alert, oriented patient who can demonstrate a normal painless range of motion of the cervical spine does not need radiographic evaluation.

X-ray interpretation

- Develop a mental picture of the normal spinal radiograph. If you feel that the X-ray 'just doesn't look right', then it probably isn't!
- Try to develop a system and use this for every fracture you see, even when you know it will be normal. It gets you into the habit.
- A systematic approach has been shown to reduce the risk of missed spine injuries.

C-spine

- AP, lateral, and open mouth views for C1/C2 are required.
- You must be able to see from C1 to T1; if not, request further views (swimmer's view).
- Look at the bones and their alignment.
- On the lateral film, a smooth line should run down the anterior aspect of the vertebral body, the posterior aspect, the anterior aspect of the spinous process (spinolaminar line), and the posterior aspect of the spinous process.
- Look for any obvious steps between vertebral bodies (up to 25% displacement may suggest unifacet dislocation; >25% suggests complete facet joint instability) and angulation.
- Examine each vertebral body for integrity.
- Look at the facet joints for congruity (facet joint dislocations).
- Look at the distance between the spinous processes (increase suggests injury).
- Assess the soft tissues. Disc space for narrowing or widening.
- Assess the soft tissues anterior to the spine. This should be no more than 7mm at C3 and 3cm at C7. Any increase suggests swelling, and thus injury.

- Look at the odontoid peg and its relationship to C1. Look for fractures and the gap in front of the peg (usually <3mm).
- On the AP film, assess the vertebral body height and alignment.
- The interpedicular distances (increase may suggest fracture).
- Look for central alignment of the spinous processes. Beware the empty vertebrae; this may imply damage to the spinous process.
- Check the transverse processes; they become displaced when fractured.

Thoracolumbar spine

- AP and lateral views required.
- Thoracic spine is difficult to interpret; look for other clues such as multiple rib fractures.
- On the AP, look at alignment.
  - Vertebral body height.
  - Interpedicular distance. An absent or broken pedicular ring may suggest fracture of the posterior elements.
  - Central spinous process alignment.
  - Integrity of the transverse processes.
  - Disc height.
- On the lateral X-ray, assess alignment and look for steps.
  - Look at the vertebral body and check for anterior wedging (>50% loss of body height suggests instability).
  - Any bony fragments displaced into the vertebral canal posteriorly.
  - Check disc height.
  - Overall angulation of the spine (kyphosis in lumbar spine or ↑ kyphosis of thoracic).

Further imaging

- If a fracture is found on the X-rays that is deemed unstable or the films are difficult to interpret and there is a high suspicion of injury, a CT scan should be requested.
- A CT scan will assess the bony spine.
- If soft tissue or spinal cord injury (SCI) is suspected, an MRI scan will be required.

Signs that imply spinal instability

Denis divides the spine into three structural columns:

- *Anterior*. Anterior half of vertebral body and the anterior longitudinal ligament.
- *Middle*. Posterior half of vertebral body and the posterior longitudinal ligament (PLL).
- *Posterior*. All structures posterior to the PLL (facet joints, pedicles, ligamentum flavum, spinous processes, and their interspinous ligaments).

With increasing column involvement, there is increasing instability, i.e. one-column injury usually stable, three-column injury highly unstable.

- Complete vertebral dislocation or translocation.
- Significant anterior wedging (>50%).
- Fractures in a previously fused spine, especially ankylosing spondylitis.
- Signs of movement. Malalignment, avulsion fractures, and evidence of paravertebral swelling.
- ↑ interspinous or interpedicular distance.

Further reading

British Orthopaedic Association. *BOAST 2: spinal clearance in the trauma patient*. Available at: https://www.boa.ac.uk

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Spinal injuries

Any patient with major trauma arriving in A&E should be assumed to have a cervical injury unless proven otherwise. Remember that the A in the 1° survey of ATLS resuscitation stands for airway with cervical spine control, i.e. it is top priority.[1]

- Cervical spine is the commonest area to have a major spinal injury.
- Other areas of concern are where mobile areas are at a junction with a less mobile one, e.g. C7/T1, T12/L1, and L5/S1 junctions.
- The main reason for delay in diagnosis of spinal injuries is failure to have a high clinical index of suspicion in all major trauma patients.

Principles of treatment

- Begin with ATLS approach and C-spine immobilization (rigid collar, lateral head supports, and strapping).
- Particular attention needs to be paid to this, as some studies have suggested up to 25% of spinal cord injuries occur in the early management phase, after the initial injury!
- All trauma patients should be assumed to have a spinal injury until proven otherwise, especially in the presence of altered mental state or blunt head injury.
- A thorough 1° and 2° survey needs to be performed, looking for mechanisms that would increase the risk of spine injury (RTA, motorcyclist, seat belt marks) and signs suggestive of other injuries (boggy swelling along the spine on log rolling).
- Until injuries of the spine have been deemed as stable, log rolling should take place to prevent any SCI from occurring.
- A full neurological examination is mandatory (if patient conscious).
- Once resuscitation and stabilization have occurred, appropriate radiological studies need to be undertaken.
- In the trauma setting, the ATLS manual now recommends CT scanning of the C-spine, rather than a lateral X-ray. However, you must still be able to assess an X-ray if CT is unavailable!

Definitive management

Cervical spine

- *C1 (Jefferson fracture)*.
  - If stable, semi-rigid collar or halo fixator.
  - If unstable, halo fixator or traction ± surgical fixation.
- *C2 (odontoid peg fracture)*.
  - Type 1 (tip). Treat with semi-rigid collar.
  - Type 2 (waist). In elderly, consider cervical collar or C1/C2 fusion; in young patients, if undisplaced—halo fixator, if displaced—internal fixation or C/C2 fusion.
  - Type 3 (base and extends into body of C2). Stable, treat with cervical collar.

- *C3–C7*.
  - Anterior compression fractures treated in semi-rigid collar or halo (if >25% loss of anterior height or kyphosis >11°, may need operative fusion).
  - Burst fractures (from axial load). Associated with cord injury; treatment with decompression and fusion may be required.
- *Facet joint dislocations*. Both unilateral and bilateral dislocations require reduction with progressive traction. Once reduced, unifacet dislocations are stable and treated in a cervical collar; bilateral dislocations require surgical stabilization.

Thoracolumbar injury

- Commonest at T11–L2 as transitional segment (rigid to mobile).
- Up to 12% incidence of fracture at different spinal level.
- Denis classified into compression, burst, flexion–distraction, and fracture–dislocations, based on 3-column theory.
- Stable fractures (<50% loss of vertebral body height, <25% kyphosis, <50% spinal canal compromise) and neurologically intact are treated conservatively with a thoracolumbar spine orthosis (TLSO) for 3 months.
- Unstable fractures or neurological deficit require stabilization via fusion and decompression of spinal canal.

Sacral injury

(➔ Fractures of the pelvis, p. 648.)

Spinal cord injury

- Assess acutely as previously described (ALTS and so forth).
- Full neurological evaluation.
- Establish level of SCI. C5 or above requires intubation.
- Complete or incomplete lesions. Find motor level, then establish presence of sacral sensation (intact suggests incomplete).
- Look for other injuries and treat accordingly.
- Steroid use for SCI is controversial, with a paucity of level 1 evidence.
- High-dose methylprednisolone administered within 8h of injury and continued for 48h has been shown to improve outcomes.[2]
- Always adhere to local policy.

Neurogenic shock

- Neurological injury causing failure of descending sympathetic pathways of cervical and upper thoracic cord. Affects vasomotor tone and cardiac function.
- Results in vasodilatation and bradycardia (unopposed parasympathetic).
- Be careful of excessive fluid therapy to treat hypotension as may result in fluid overload.

Spinal shock

- Loss of muscle tone (flaccidity), loss of reflexes (areflexia), and anaesthesia following SCI.
- Duration variable (usually 48h).
- End defined by onset of spasticity below level of SCI.
- No recovery by 48h suggests complete cord injury and poor prognosis.

'Incomplete' spinal cord syndromes

If there is preservation of some modalities of cord function distal to the injury level, the cord lesion is referred to as 'incomplete'. Several recognized patterns exist.

Central cord syndrome

- Commonest.
- Hyperextension injury to a spine with stenosis; usually age >50y.
- Weakness affects upper limbs > lower limbs. Deficits worse distal than proximal. Variable sensory loss.
- Due to vascular compromise to cord (anterior spinal artery supplies central cord).
- Recovery. Lower extremities, then bladder, then proximal upper limbs, and then hands.

Anterior cord syndrome

- Flexion injury.
- Poorest prognosis (10–20% recovery).
- Dense motor (paraplegia/quadriplegia) and sensory loss below level of injury. Affects spinothalamic and corticospinal tracts.
- Proprioception and vibration sense (posterior column) spared.

Brown-Séquard syndrome

- Penetrating trauma.
- Hemisection of the cord, giving ipsilateral motor (corticospinal), vibration, and proprioceptive loss (posterior columns), and contralateral loss of pain and temperature (spinothalamic).
- Variable, but best chance of recovery.

Posterior cord syndrome

- Proprioception and vibration sense lost, but intact motor power (rare).

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Acute haematogenous osteomyelitis

This is a disease of growing bones. It is common in infants and children, but rare in adults unless they are immunocompromised or diabetic.

Aetiology

- Infants (<1y). *Staphylococcus aureus*, group B streptococci, and *Escherichia coli*.
- Children (1–16y). *S. aureus*, *Streptococcus pyogenes*, *Haemophilus influenzae*.
- Adults. *S. aureus*, *Staphylococcus epidermidis*.
- Sickle cell patients. *Salmonella* spp.
- Rare causes. *Brucella*, TB, spirochetes, and fungi.

Pathological features

The organisms settle near the metaphysis at the growing end of a long bone. The following stages typically occur.

- *Inflammation*. Acute inflammation with venous congestion.
- *Suppuration*. After 2–3 days, pus forms in the medulla and forces its way out to the periosteum.
- *Necrosis*. After 7 days, blood supply is compromised and infective thrombosis leads to necrosis and formation of a pocket of dead tissue (sequestrum).
- *Repair*. At around 10–14 days, new bone is formed from the subperiosteal layer that was stripped with the swelling (involucrum).
- *Discharge*. Involucrum can develop defects (cloacae), allowing discharge of pus and sequestrum to allow resolution. This can also be achieved by surgical release and debridement.

Clinical features

- Usually a child with a preceding history of trauma or infection (skin or respiratory).
- Fever, pain, and malaise develop after a few days.
- The child may be limping or refusing to weight-bear.
- On examination, there may be localized swelling or redness of a long bone.
- Infants may present with failure to thrive, drowsiness, or irritability.
- Neonates may present with life-threatening septicaemia in which obvious inflammation of a long bone develops, or a more benign form in which the symptoms are slow to develop, but bone changes are extensive and often multiple.

Investigations

- Plain X-rays may be normal for the first 10 days. Do not be reassured!
- ^{99}Technetium bone scan is usually positive in the first 24–48h and is effective in confirming diagnosis early.
- ^{67}Gallium bone scan and ^{111}indium-labelled white cell scans are more specific, but generally not available in most units.
- MRI is very sensitive, but not specific and difficult for children.
- CT scanning can define extent of bone sequestration and cavitation.

X-ray features

- Soft tissue swelling is an early sign; look for displacement of fat planes.
- Patchy lucencies develop in the metaphysis at around 10 days.
- Periosteal new bone may be seen.
- Involucrum formation is only apparent at around 3 weeks.
- Sequestrum appears radiodense, compared to the surrounding bone which is osteopenic.
- Normal bone density occurs with healing.

Laboratory test results

- FBC. ↑ WCC, normally with a raised neutrophil count.
- ↑ ESR.
- ↑ CRP, but returns to normal quickly post-treatment.
- Blood cultures positive in 50% of cases (use to inform and adjust antibiotic therapy).
- Perform U&Es, LFTs, and glucose.

Treatment

- Pain relief by bed rest, splintage, and analgesics.
- Give IV antibiotics according to local guidelines (after blood cultures and pus swab samples taken), e.g. flucloxacillin IV, then PO qds for up to 6 weeks, dose adjusted according to age, clindamycin if penicillin-allergic, vancomycin if MRSA, ampicillin for *Haemophilus*.
- Surgical drainage of mature subperiosteal abscess, with debridement of all necrotic tissue, obliteration of dead spaces, adequate soft tissue coverage, and restoration of an effective blood supply.

Complications

- Disseminated systemic infection, e.g. septicaemia, cerebral abscess.
- Chronic osteomyelitis.
- Septic arthritis.
- Deformity due to epiphyseal involvement.

Further reading

Lazzarini L, Mader JT, Calhoun JH (2004). Osteomyelitis in long bones. *J Bone Joint Surg Am* **86**: 2305–18.

Chronic osteomyelitis

Causes

- Occasionally following acute haematogenous osteomyelitis.
- Commonest following contaminated trauma and open fractures.
- After joint replacement surgery.
- 1° chronic infections of bone.

Secondary to acute osteomyelitis

Features

- Sinus formation due to sequestra or resistant bacteria.
- Prevented by adequate treatment of the initial acute attack.

Treatment

- Conservative (simple dressings) may be appropriate (elderly). Recurrent attacks with spontaneous recovery may occur and surgery should be reserved for cases where an abscess forms.
- Chronic abscess. May require drainage, debridement of all necrotic tissue, and obliteration of dead spaces. May involve plastic surgery to achieve soft tissue cover and restoration of an effective blood supply.
- Closed suction drainage/irrigation systems can be effective, especially if irrigation fluid contains antibiotics. The disadvantage is that early blockage of the system can occur.
- Antibiotic (gentamicin)-impregnated beads or sponges deliver high local levels and may be beneficial in areas of poor blood supply, hence systemic antibiotic penetration.
- Unresolving cases may require amputation.

Secondary to trauma (open fractures)

- Prevention by early aggressive approach to compound fractures with debridement and lavage of contaminated tissue.
  - Excise all dead tissue widely and remove all devitalized bone fragments, i.e. with no soft tissue connections.
  - Copious lavage is necessary as '*the solution to pollution is dilution*' (≤10L is common).
  - Skeletal stabilization is mandatory.
  - IV antibiotics, e.g. IV cefuroxime ± metronidazole if anaerobes may be involved (soil).
- Treat established chronic infection as above, with removal of internal foreign bodies, e.g. metalwork, and possible application of external fixation.

Secondary to joint replacement surgery

- Rare (≥1%), but is often a disaster for the elective patient.
- Prevention is better than cure. Dedicated laminar flow theatres, strict theatre discipline, and prophylactic IV antibiotics are mandatory.
- 50% will require surgical intervention.
  - Initial joint irrigation, debridement, and tissue sampling can be attempted if the prosthesis is still solid and not 'loose'.
  - If grossly infected, the prosthesis must be removed, the surfaces debrided, and an antibiotic cement spacer placed on the raw bone ends to allow the soft tissue envelope to settle.

- Once inflammatory markers have settled (CRP is the best) and the clinical infection has resolved, second-stage replacement of the spacer with a new prosthetic joint can go ahead. This may take 12 months and may not be possible.

Chronic osteomyelitis as an initial presentation

Brodie's abscess

- An isolated well-contained chronic abscess.
- Treatment. Operative drainage with excision of the abscess wall and antibiotics.

Tuberculosis

- Usually associated with other systemic features of the disease.
- May present acutely.
- Muscle atrophy develops and spontaneous discharge of a 'cold' abscess may lead to sinus formation and destruction of bone.
- Spinal TB may cause vertebral collapse, leading to acute neurology ('Pott's paraplegia').

Syphilitic osteomyelitis

- Associated with advanced tertiary disease in adults. Features diffuse periostitis (with sabre tibia) or localized gummata with sequestra, sinus formation, and pathological fractures. X-rays show periosteal thickening with 'punched-out' areas in sclerotic bone.
- Infants with congenital disease have epiphysitis and metaphysitis. X-rays show areas of sclerosis near the growth plate separated by areas of rarefication.

Mycotic osteomyelitis

- Typically occurs in immunocompromised patients.
- Bone granulomas, necrosis, and suppuration present without worsening acute illness.
- Usually occurs as spread from 1° lung infections such as coccidioidomycosis, cryptococcosis, blastomycosis, and histoplasmosis.
- Treatment. Amphotericin B and/or surgical excision.

Further reading

Lazzarini L, Mader JT, Calhoun JH (2004). Osteomyelitis in long bones. *J Bone Joint Surg Am* **86**: 2305–18. [Current concepts review]

Septic arthritis

A condition due to infection of a joint space and its synovium. It is common in infants and children, but rare in adults unless they are immunocompromised or diabetic.

Aetiology

The infective organisms are the same as for acute osteomyelitis.

Pathological features

- 1° seeding of the synovial membrane.
- 2° infection from adjacent metaphysis or directly from epiphysis.
- In some joints, the metaphysis lies partly within the joint capsule (shoulder, elbow, hip, and ankle); osteomyelitis can break through the metaphysis and into the joint.
- Proteolytic enzymes are released from synovial cells and proteases from chondrocytes, causing destruction of the articular cartilage:
  - By 5 days, proteoglycans are lost from cartilage.
  - By 9 days, collagen is lost.

Clinical features

- Usually a child with a preceding history of trauma or infection (skin or respiratory).
- Acute onset with pyrexia and irritability.
- The child may be limping or refusing to weight-bear.
- The affected joint is held still in position of maximal comfort (e.g. hip flexed, abducted, and externally rotated gives largest joint volume).
- Look for an erythematous, hot, swollen joint with an effusion.
- A neonate may present with none of the above—just irritable, lethargic, off feeds, and not moving affected limb.
- In the adult, the joint will be exquisitely painful, hot, red, and swollen, and they will usually not allow passive motion.

Investigations

- Plain X-rays may show joint space widening, joint subluxation or dislocation, and soft tissue swelling.
- USS can demonstrate effusion and guide aspiration.
- The mainstay of diagnosis is aspiration of the affected joint with immediate Gram stain and microscopy, followed by cultures and sensitivities.
- Always ask for the sample to be analysed for crystals and some septic-looking joints in adults are actually due to crystalopathies (gout or pseudogout).

Laboratory test results

- FBC. ↑ WCC, normally with a raised neutrophil count.
- ↑ ESR.
- ↑ CRP, but returns to normal quickly post-treatment.
- Blood cultures positive in 50% of cases (use to inform and adjust antibiotic therapy).
- Perform U&Es, LFTs, and glucose.

Treatment

- Septic arthritis is a surgical emergency and as such, rapid diagnosis and management are required.
- Open (or arthroscopic) drainage of the affected joint with copious irrigation.
- Resuscitation and antibiotics.
- Re-exploration should be considered for those not settling.
- IV antibiotics (broad-spectrum), then tailored once culture results are available for 2 weeks, then PO for further 4 weeks.

Peripheral nerve injuries

Common; ~9000 people a year are admitted to hospital with an injury to a peripheral nerve.

- Causes include traction, trauma, inflammation, and compression.
- The degree of injury depends on the mechanism (open or closed injury, acute or chronic) and health of nerve prior to injury.

Pathological features

- Seddon classification.

Neuropraxia

- Stretching or compression of the nerve which remains anatomically intact.
- Conduction block with normal conduction above and below.
- Focal demyelination occurs at the site of injury, which is repaired by the Schwann cells.
- Recovery is usually complete, occurring in days or weeks. There is no axonal degeneration.

Axonotmesis

- The axon is divided, but the covering connective tissue component remains intact, i.e. the nerve cylinder remains.
- Usually a traction or severe crush injury.
- Axonal ('Wallerian') degeneration occurs distal to the injury and is followed by nerve regeneration (by sprouting from the severed nerve end) after 10 days.
- Nerve growth occurs at a rate of 1mm per day.
- Prognosis is generally good as the cylinder is intact, but the more proximal the lesion, the less the distal recovery.
- Sensation recovery is generally better than motor recovery, especially if the lesion is proximal and muscle wasting occurs whilst it is 'denervated'.

Neurotmesis

- The nerve is completely divided or irreparably damaged with loss of apposition of the severed nerve bundles and their respective distal parts.
- Usually a high-energy injury, penetrating trauma, severe traction, ischaemia, or high-pressure injection injury.
- Minimal recovery is possible without operative intervention to repair or graft a new nerve to the injury.
- Surgical repair may allow axon regeneration to the correct end-organ, but recovery will not be complete as often 'miswiring' occurs.

Diagnosis

- What is the injury? Is there an open wound, fracture, recent surgery, or prolonged immobility?
- Complete neurological examination. You must know the motor and sensory supplies of peripheral nerves! Use a pin or your finger for sensory testing. Compare the area of normal and injured side sequentially.
- *Tips*. Anaesthetic skin looks shiny and does not sweat. Denervated skin will not wrinkle in water.

- There are specific features of different levels of injury in peripheral nerves of the upper limb.
- Examination very soon after injury can be misleading as sensory loss may take time to appear.
- Diagnosis of a peripheral nerve injury is clinical but can be supplemented with nerve conduction studies and electromyography (EMG).

Treatment

Closed injury

- Injuries in continuity. Neuropraxia and axonotmesis (vast majority) can be expected to recover spontaneously, so exploration is not indicated.
- Compression injuries should have compressive forces removed, e.g. external, such as plaster, or internal such as carpal tunnel syndrome.
- Physiotherapy and splintage should be used whilst awaiting recovery; this will maintain functionality and prevent contractures.

Open injuries

- 1° *repair (suture)*. Within 24h is ideal, but an uncontaminated operative field, adequate skin cover, and proper equipment (e.g. microscopes) must be present.
- *Delayed* 2° *repair*. Can be done at any time after injury once the soft tissues have healed (3–6 weeks acceptable). The nerve can be mobilized to allow a no-tension repair after resection of the cut nerve stumps. Usually, however, a nerve graft has to be used to bridge the defect (sural nerve as a donor is the commonest).

Further reading

Blom A, Warwick D, Whitehouse M (editors) (2017). *Apley and Solomon's System of Orthopaedics and Trauma*, 10th edn. CRC Press, Boca Raton, FL.

British Orthopaedic Association. *BOAST 5: peripheral nerve injuries*. Available at: https://www.boa.ac.uk

Brachial plexus injuries

The brachial plexus is formed from the ventral rami of C5 to T1. It is subsequently divided into root, trunks, divisions, cords, and terminal branches. The lesion can be at any of the above levels.

Terminal branches arising from the root level (phrenic nerve, C3–5); dorsal scapular nerve, C5; and long thoracic nerve, C5–7) are important to recognize as if they are spared, it suggests the lesion is post-ganglionic.

Causes

- *Child*. Obstetric, i.e. difficult deliveries with traction on the plexus.
- *Adult*. Almost all traumatic.
  - Usually closed, e.g. motorcycle accidents, falls, and traction injuries with forced abduction of the arm.
  - May be open, e.g. stab or gunshot wounds.
- Always look for associated injuries, e.g. head, neck, chest, abdominal, and vascular.

Types

- *Erb–Duchenne (upper)*. Involves C5 and C6; the arm classically hangs at the side, with the arm flaccid, internally rotated, and adducted and the wrist flexed (waiter's tip position).
- *Klumpke's (lower)*. Involves C8 and T1; the hand is clawed due to intrinsic muscle paralysis and if the sympathetic trunk is involved, there is Horner's syndrome.

Try to localize the lesion to preganglionic (intraspinal) or post-ganglionic (extraspinal) by clinical means, as described earlier. At the T1 level, look for signs of Horner's syndrome (ptosis, miosis, ipsilateral anhydrosis), suggesting a preganglionic lesion.

If histamine is injected into the skin of the supplied area, vasodilatation, wheal, and flare indicate a positive result and a preganglionic injury is present. If there is no flare, the lesion is post-ganglionic.

Anatomic level of injury should be delineated.

Treatment

- Physiotherapy to prevent joint contracture and stiffness. If no return of function at 2 months, consider myelography or histamine tests to localize the injury level.
- Open injuries should be explored acutely, but not if there are more life-threatening injuries (which is usually the case). 1° repair may be possible in this group.
- Delayed surgery (if required at all) is normal for most patients.
- Preganglionic injuries are irreparable and should not be explored.
- Post-ganglionic injuries may be explored for up to 6 months post-injury. 2° repair with nerve grafting can then be attempted if a clear lesion is isolated.

Prognosis

- If there are no EMG abnormalities at 3–4 weeks, prognosis is good with conservative treatment.
- Causalgic pain, Horner's syndrome, and the presence of root avulsion on myelogram (hence intraspinal lesion) indicate a poor prognosis.
- Recovery is generally very slow and often unsatisfactory.
- Salvage surgery with tendon transfers or shoulder arthrodesis may improve function and give better results than amputation.

Further reading

Birch R (1996). Brachial plexus injuries. *J Bone Joint Surg Br* **78**: 986–92. [Review]

Osteoarthrosis (osteoarthritis)

This is degenerative joint disease; it is a disease of cartilage, not the joint.

- It is limited to the joint itself and there is no systemic effect.
- It may involve any synovial joint but is commonest in the hip, knee, and hands.
- It is the commonest form of arthritis, with an estimated radiographic incidence of moderate to severe changes in 5 million people in the UK.
- ~2 million people visit their GP with osteoarthritis per year and it is predicted that there will be a 66% increase in the number of people with osteoarthritis-related disability by 2020.

Types

Primary osteoarthritis (idiopathic)

Mainly affects the following joints: DIPJ, first carpometacarpal, hips, knees, and apophyseal joints of the spine. Women are more affected than men and there may be a hereditary component, but the aetiology is unknown.

Secondary osteoarthritis

Affects previously damaged joints and is commoner in weight-bearing joints. Both sexes are equally affected. Local causes are fractures, acquired or congenital deformities, joint injury (chondral lesions), diabetic neuropathy (Charcot joints), and AVN.

Clinical features

- Characteristic pain, swelling, and deformity.
- Dull, aching pain with morning stiffness of the affected joint.
- Pain becomes steadily worse throughout the day and may disturb sleep.
- Acute onset. Swollen, hot, and painful joint, with raised inflammatory markers.
- Look for Heberden's nodes at the DIPJs and Bouchard's nodes at the PIPJs.
- Physical symptoms may not correlate with the severity of the radiographic changes, so judge each patient on an individual basis.

X-ray changes

- Loss of joint space.
- Subchondral bone sclerosis.
- Cyst formation (especially at the hip).
- Osteophyte formation.

Treatment

Relieve pain, improve mobility, and correct deformity in that order.

Medical

- Pain relief with simple analgesics (paracetamol, codeine), in combination with NSAIDs, helps control symptoms and increase mobility. Beware of GI bleeding, especially in the elderly, and of worsening asthma.
- Radiant heat in the form of infrared light or a hot water bottle frequently helps.
- Weight loss, physiotherapy, and aids to daily living, such as walking sticks, heel raises, raised chair, and household aids, should all be in place before contemplating surgery.
- Joint injections of steroid and LA may help in up to 50% of patients.

Surgical treatment

This is indicated for pain relief, improved mobility, and correcting deformity only when conservative measures have failed.

Options include:

- Osteotomy. Realignment of a joint to unload an arthritic area.
- Arthrodesis. Permanent stiffening of a joint by excision and fusion to stop pain.
- Excision. Removal of the joint without fusion.
- Arthroplasty. Replacement of all or part of the joint surface by an artificial material.

Carpal tunnel syndrome

- Compression of the median nerve at the wrist.
- Boundaries of tunnel are: radially—scaphoid tubercle and trapezium, ulnarly—hook of hamate and pisiform, transverse palmar ligament, palmar aspect (roof).
- Contents—flexor tendons (flexor pollicis longus (FPL), flexor digitorum superficialis (FDS), flexor digitorum profundus (FDP)) and median nerve.
- Commonest in middle age.
- Often bilateral, but when unilateral, most commonly affects the dominant hand.

Aetiology

Remember, the commonest is idiopathic.

Compression of the tunnel wall

- Trauma, e.g. distal radial fracture.
- Rheumatoid arthritis (thickening of the surrounding synovium and tissues).
- Subluxation or dislocation of the wrist.
- Acromegaly (soft tissue thickening and enlargement).

Compression within the tunnel

- Fluid retention, e.g. pregnancy.
- Myxoedema.
- Space-occupying lesion, e.g. benign tumour.
- Chronic proliferative synovitis.

Changes in the median nerve

- DM.
- Peripheral neuropathies.

Clinical features

Symptoms

- Aching pain and paraesthesiae (pins and needles) over radial three-and-a-half fingers and palm.
- Pain typically at night and can disturb sleep.
- Relieved by shaking the hand.
- May notice dropping items (weak pinch grip), clumsiness.
- Can be made worse by activity.
- Atypical symptoms can be common.

Signs

- Hand looks normal.
- Thenar muscle wasting if chronic and severe.
- Weakness of thumb abduction.
- Tinel's test. Tapping over the nerve at the wrist in neutral produces symptoms.
- Phalen's test. Rest elbows on the table and passively flex the wrist. If symptoms appear within 60s, test is positive.
- Median nerve compression test. Extend elbow, supinate forearm, flex wrist to 60°, and press on carpal tunnel. Positive if symptoms within 30s.

Investigations Nerve conduction studies are gold standard but still show only 90% accuracy.

Treatment

Conservative

- Splintage.
- NSAIDs.
- Injection of corticosteroids.
- Avoidance of precipitating factors.

Surgical

- Surgical decompression.
- Use a tourniquet.
- Skin incision in line with ulnar border of the ring finger. This is to avoid the motor branch of the median nerve.
- Protect the nerve with a MacDonald's dissector and visualize the nerve directly throughout.
- Do not extend the skin incision beyond the wrist crease to protect the palmar cutaneous branch of the median nerve.

Complications

- Complex regional pain syndrome.
- Tender, hypertrophic scar giving pillar pain (pain in the heel of the scar on pressure).
- Neuroma of the palmar cutaneous branch.
- Recurrence.
- Bowstringing of flexor tendons.

Reference

Tetro AM, Evanoff BA, Hollstien SB, Gelberman RH (1998). A new provocative test for carpal tunnel syndrome: assessment of wrist flexion and nerve compression. *J Bone Joint Surg Br* **80**: 493–8.

Ganglion

This is a degenerative mucinous cyst swelling that can arise from a tendon sheath or joint. It contains clear, colourless, gelatinous fluid.

Common sites

- Dorsum of the wrist, arising from the scapholunate ligament or mid-carpal joint (70% of all cases).
- Radial aspect of the volar wrist normally from scaphotrapezial joint (20% of all cases).
- Base or DIPJ of finger.
- Dorsum of the foot.
- Around the knee.

Clinical features

- Slow-growing, cystic lump, commonly presenting as dorsal wrist pain.
- Increase and decrease in size.
- Firm, smooth, and rubbery, and will usually transilluminate.
- May be more obvious with wrist in palmar flexion.

Diagnosis

- Needle aspiration gives gelatinous fluid. If no fluid can be aspirated, then investigate further since a soft tissue tumour (including sarcoma) is possible.
- MRI scanning should be used if there is serious concern over a soft tissue tumour.
- Occult ganglia (no palpable lump) can yield symptoms in the wrist or foot. USS will confirm the diagnosis.

Treatment

- 50% will disappear spontaneously. Therefore, treat conservatively, unless pressed by the patient.
- Aspiration may be curative in 50% of cases.
- Deliberately induced traumatic rupture often leads to recurrence.

Surgery

- Excision is not guaranteed success (recurrence ~10%; painful scar ~10%).
- Use a tourniquet.
- If not occult or excessively large, then LA as day case procedure appropriate.
- Excise thoroughly and transfix the base to prevent recurrence.
- Volar wrist ganglia often surround, or are very close to, the radial artery!

Bone tumours

Diagnosis

The key to successful management of bone tumours is early detection and treatment; always having a clinical suspicion is essential!

Presenting features

- Pain. Persistent, at night, response to analgesics?
- Mass or swelling (? getting bigger, rate of progression).
- If there is a fracture, is there a history of trauma?
- Neurological symptoms.
- Systemic symptoms.
- Previous tumours, radio- or chemotherapy.
- Any family history.
- Watch for the 'red herring history' of trivial injury.

Examination

- Extract features of the mass/swelling and palpate for lymphadenopathy.
- Is it around a joint? Is it deep to the fascia? Size, relationship to surrounding structures.

Radiological investigations

- A plain X-ray (AP and lateral) of affected area is mandatory.
- Where is the lesion? What are the effects on bone? Is there a bone reaction (new bone, periosteal reaction, Codman's triangle, sunburst spiculation)? Is there a matrix?
- Once a diagnosis has been considered/made, further imaging is required; MRI scanning is usually gold standard.
- Accurately stage and assess local or systemic spread (CT, bony architecture; MRI, soft tissue or bony extensions).

Blood tests

- Rarely diagnostic and often non-specific (e.g. ↑ ALP, ↑ ESR, ↑ Ca^{2+}).

Urgent referral

- Once a diagnosis is made, urgent referral (even before MRI is obtained in highly suspicious lesion) to local tumour services is required.
- They will advise and guide further local management and arrange definitive treatment if required.

Other considerations

- Angiography. Helps plan radical surgery and possible limb salvage.
- Open biopsy. Best to achieve histological diagnosis and required for treatment planning.
- Must be performed by the surgeon who is going to do the definitive surgery.
- The biopsy track must be excised as part of the definitive excision and placed to maximize the chance of limb salvage surgery.

Metastatic tumours

- 2° metastases are the commonest tumours of bone (breast, prostate, lung, thyroid, and kidney 1°).
- Bone pain—worse at night and with weight-bearing.
- Systemic symptoms (fatigue, weight loss, no appetite).

- History of cancer (personal or family).
- Pathological fractures common.
- If a patient is admitted with a history of pathological fracture with unknown 1°, full examination should be performed to find source (breast, rectal, prostate, etc.).
- Blood tests, including Ca^{2+}, phosphate, tumour markers (PSA, CEA, CA125).
- Bone scan, staging CT/MRI may be required.
- Treatment:
  - Treat electrolytes first if raised.
  - Internal fixation (ideally prophylactic before fracture occurs) allows early weight-bearing and hopefully early discharge from hospital.
  - Radiotherapy for pain.
  - Manage in conjunction with oncologists.

Benign tumours

Osteochondroma ('exostosis')

- Commonest benign tumour.
- A cartilaginous capped outgrowth of bone from the cortex, normally near an epiphysis. Lesion grows until skeletal maturity.
- Usually pain-free and present as lump. If painful, usually due to inflammation of overlying bursa.
- Usually solitary. Multiple lesions require close follow-up (hereditary).
- Any sudden increase in size may indicate malignant transformation to chondrosarcoma (<1%).

Chondroma

A non-calcified cartilaginous growth in the medulla (enchondroma) or cortex (ecchondroma) of tubular bones such as phalanges and metacarpals/tarsals.

Osteoid osteoma

- Occurs in young patients (5–30y).
- Progressive pain (night) of long bone, referred to other joints, classically relieved by NSAIDs.
- Commonly long bones (diaphysis), can be intra-articular, and a cause of painful scoliosis.
- Radiology shows a 'nidus' which is a small osteolytic area surrounded by a rim of dense sclerosis. Look for periosteal reaction.

Non-ossifying fibroma

- Fibrous tissue tumour which usually appears radiologically as an oval cortical defect with sclerotic rim. Common incidental finding on X-rays; usually needs no treatment.
- Treatment. Principles of treatment are simple local excision or removal by curettage if symptomatic or likely to cause pathological fracture (likely if >50% diameter of bone involved).
  - Cavities should be packed with bone graft or bone cement.
  - Internal fixation may be used once large tumours have been removed.
  - Difficult tumours should be managed in a 'bone tumour centre'.

Primary malignant tumours (rare)

All 1° malignant tumours require a 'multidisciplinary team' (orthopaedic surgeon, oncologist, musculoskeletal radiologist, histopathologist).

Osteosarcoma

- Commonest 1° bone tumour.
- Long bones of young adults (peak incidence 10–20y) or as a consequence of Paget's disease in the elderly (➲ Paget's disease (osteitis deformans), p. 700).
- Presents with progressive pain (rest/night) refractory to analgesia.
- Swelling, reduced joint movement, limp.
- ? trivial sporting injury—not related to tumour development!
- X-ray features. Bone destruction, soft tissue invasion, radiating spicules of bone ('sun ray' appearance), subperiosteal elevation with new bone formation ('Codman's triangle').
- MRI shows extent of tumour, skip lesions, and soft tissue involvement.
- Metastasis via blood to the lungs and bone.
- Treatment:
  - Neoadjuvant chemotherapy, followed by surgical resection and further chemotherapy.
  - Limb salvage surgery possible in about 90% (rare for pelvic tumours).
  - Local recurrence rate 5% (poor prognosis).
  - Radiotherapy may be used as an alternative in the elderly.
- 5y survival 60–70% (localized) or 25% (pelvic) with surgery; 20% if present with metastases.

Chondrosarcoma

- Commonest in older patients (30–75y).
- Usually occurs in a flat bone, e.g. ilium of pelvis, ribs, scapula.
- Location of presentation gives clues to type (scapula malignant and hand benign).
- May present de novo or arise from a pre-existing osteochondroma.
- Graded. Low to high (1, 2, 3, undifferentiated); 60% present grade 1.
- Metastasis is not common and is via blood.
- Local invasion is more usual but is normally slow-growing.
- High grade present with bone destruction and soft tissue mass.
- Treatment:
  - Low grade requires wide resection. Local recurrence 20% at 10y.
  - High grade requires wide resection ± amputation.
  - No real role for chemo- or radiotherapy unless undifferentiated or elderly).
  - 5y survival dependent on grade. Grade 1, up to 90%; grade 2, 60–70%; grade 3, 30–50%; undifferentiated, 10%.

Ewing's sarcoma

- Children and young adults (<20y).
- Pain, associated hot/erythematous swelling with associated pyrexia so that osteomyelitis may be suspected.
- Commonly pelvis, long bones, and scapula.
- ↑ ESR and ↑ WCC.
- X-rays show lytic bone destruction with periosteal reaction in multiple layers ('onion skin' appearance).

- MRI scan shows soft tissue involvement, which would not usually be the case in osteomyelitis.
- Metastasis to the lung is very fast and most people present with this.
- Treatment. Preoperative neoadjuvant chemotherapy (12 weeks) and then re-evaluate and re-stage.
- Isolated lesions managed with wide excision or amputation.
- Radiation can be used if metastases present.
- Response to chemotherapy predicts prognosis.
- Prognosis is still poor. Isolated extremity Ewing's ~65% 5y survival; metastatic disease at presentation <20% 5y survival.

Giant cell tumour (osteoclastoma)

- This is rare before the age of 20y.
- Usually benign, but may undergo malignant transformation (~10%).
- Rarely metastasizes; usually to lungs, but may be locally invasive.
- Treatment. Local excision and defect filled bone graft or cement.
- Recurrence is common (~20%), especially with malignancy.

Further reading

Excellent summaries of bone tumours can be found at: https://www.bonetumor.org

Low back pain

- Very common condition in the UK; 60–80% of adults will be affected during their lifetime.
- Common cause of absence from work.
- In the majority of cases, it is a self-limiting condition requiring no surgical intervention.[1]
- Multifaceted condition with overlapping aetiology.
- Always consider neoplasia—metastasis, myeloma, osteoid osteoma.

Types of pain

Mechanical

- Pain (low back, buttock, and thigh), like 'toothache'.
- Rarely radiates below knee.
- Worse over course of day and with activity.
- Cannot get comfortable and wakes from sleep when turn.
- Acute pain from trivial movement which settles with rest.
- Typically midline and made worse by lordotic postures, e.g. bending and lifting (discogenic).
- Pain is from the annulus fibrosis layer of the disc when it is being stretched.

Nerve root entrapment

- Pain radiates down the leg from buttock to calf/foot.
- Commonly caused by a lumbar disc prolapse, compressing and irritating the nerve root as it exits the spinal foramina.
- Pain from spinal stenosis is worsened with extension (walking down a hill) and relieved with flexion (riding a bike).
- Radiation should match the sensory dermatome of the nerve root involved.
- Other features are pain on sneezing, coughing, and straining. Numbness or paraesthesiae in the dermatome of the affected root may also be present.
- The earliest and most persistent feature of nerve root compression is loss of a tendon reflex, e.g. knee L3/4, ankle L5/S1.

Referred

May arise from retroperitoneal pathology (aortic aneurysm, pancreatic and biliary tree pathology, rectal pathology, renal stones, lymphadenopathy, and hip arthroses).

Psychosocial (yellow flags)

- The commonest cause for chronicity.
- A diagnosis of exclusion, i.e. exclude all other pathology before labelling people as 'neurotic'.
- Typical features of non-organic pain are pain on axial compression or pelvic rotation, non-dermatomal sensory loss, non-anatomical tenderness, cogwheel (give way) weakness, and overreaction (Waddell's signs).[2]

Common pathological (organic) causes

- *Mechanical* (80–90%).
  - Unknown cause. Muscle strain or ligamentous injury.
  - Degenerative disc or joint disease.
  - Vertebral fracture.
  - Congenital deformity (such as scoliosis, kyphosis, and transitional vertebrae).
  - Spondylolysis.
  - Instability (spondylolisthesis).
- *Neurogenic* (5–15%).
  - Herniated disc.
  - Spinal stenosis.
  - Osteophytic nerve root composition.
  - Failed back surgery syndrome (such as arachnoiditis, epidural adhesions, recurrent herniation). May cause mechanical back pain as well.
  - Infection (such as herpes zoster).
- *Non-mechanical spinal conditions* (1–2%).
  - Neoplastic (such as 1° or metastatic).
  - Infection (osteomyelitis, discitis, abscess—staphylococcal or TB).
  - Inflammatory arthritis (such as RA and spondyloarthropathies, including ankylosing spondylitis, reactive arthritis, and enteropathic arthritis).
  - Paget's disease.
- *Coccydynia*. Pain in the coccyx may be due to lumbosacral disc disease.

Assessment

- History must distinguish between 'simple' back pain and that requiring urgent care.
- '*Red flags*' of serious spinal pathology:
  - Retention of urine or incontinence.
  - Onset over age 55 or under 20.
  - Symptoms of systemic illness (weight loss, fever).
  - Severe progressive pain (unrelenting).
  - Trauma.
  - A prior history of cancer.
  - IV drug use.
  - Prolonged immunosuppressant or steroid use.
- Examination must cover:
  - Palpation, movements, straight leg raising, femoral stretch test, power, sensation, reflexes.
- If signs of significant spinal pathology present (i.e. cauda equina), perianal sensation and PR examination must be performed.

Investigations

- Spinal X-rays (AP and lateral of affected level).
- CT scan good for assessing bony structures.
- MRI scan good for soft tissue problems (discs, spinal cord, nerve root involvement), but only relevant in planning, not in diagnosing treatment. Should be left for treating surgeon as it is often not required.

Treatment

Non-operative

- Majority of patients require non-operative treatment.
- Initial rest (1–2 days only).
- Analgesia (paracetamol, NSAIDs, muscle relaxants, opioids).
- Early mobilization with strong encouragement.
- Physiotherapy (massage, acupuncture, hydrotherapy).
- Counselling and psychosocial support.

Surgery

- Only a minority requires surgery. Should be clearly focused on proven pathology demonstrated by imaging where possible.
- Procedures used include:
  - Discectomy.
  - Chemonucleosis/percutaneous disc removal.
  - Nerve root decompression.
  - Spinal decompression.
  - Spinal fusion.

Cauda equina

- Surgical emergency.
- Cauda equina (horse's tail) is a collection of nerve roots (lower lumbar and sacral) at distal end of cord.
- It can present acutely, chronically, or following long-standing lower back problems.
- Causes include large central or paracentral disc herniation (L4/5 or L5/S1), spinal injury neoplasms, tumours, infections (abscess or TB), and haematoma (iatrogenic).
- Key symptoms are dysfunction of bladder, bowel (and sexual function), and saddle or perianal anaesthesia.
- Other symptoms include low back pain, radiation down one or both legs, sensory chances in lower limbs, and weakness.
- Examination should be a thorough lower limb neurological assessment.
- Perianal sensation and anal sphincter tone (bulbocavernosus reflex) are essential.
- Imaging is required urgently if convincing clinical evidence exists. MRI is the modality of choice.
- Treatment once diagnosed is surgical decompression. Referral to an appropriate spinal centre, if not on site, is required urgently.
- Timing of surgery is controversial. Better outcomes are seen if performed <24h following onset.
- Outcomes are variable. If complete cauda equina (urinary retention or incontinence), the prognosis for recovery is poor.

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Paget's disease (osteitis deformans)

Key facts

- Described by Sir James Paget (1876).
- Incidence increases with age (>50y).
- Any bone may be involved. Commonest sites are spine, skull, pelvis and femur, and tibia.
- Autosomal dominant.

Pathological features

- ↑ osteoclastic bone resorbtion, followed by compensatory bone formation, i.e. there is an overall increase in bone turnover.
- Three phases. Lytic, mixed (lysis and formation), and sclerotic.
- The bone is softer, but thickened, and is liable to pathological fracture.

Clinical features

- Most are asymptomatic.
- Diagnosed via an incidental finding on X-ray or raised ALP, whilst investigating other pathologies.
- ↑ thickness of bone may be the only symptom or sign.
- Subcutaneous bones may be deformed, classically the tibia when it becomes 'sabre'-shaped.
- Pain may be present but is unusual. It may represent high turnover at the time or, more likely, a pathological fracture.
- In known Paget's patients, increase in pain must be taken seriously, as it may be a marker of sarcomatous change in the bone.

Investigations

- Serum Ca^{2+} and phosphorus are normal.
- ALP is high (due to ↑ osteoblast activity).
- Urinary excretion of hydroxyproline is high (↑ bone turnover).
- Isotope bone scan shows 'hot spots' in affected areas.
- X-ray shows both sclerosis and osteoporosis. The cortex is thickened and the bones deformed. Pathological fracture is a feature and normal bone architecture is lost, with coarse trabecular pattern.

Complications

- Pathological fractures.
- Osteosarcomatous change (<5%; prognosis very poor).
- High-output cardiac failure may develop due to ↑ vascularity of Paget's bone. Functionally, the bone is acting as an AV fistula.
- Deafness. Bony deformation in the ear causes damage to cranial nerve.
- Osteoarthritis.
- Leontiasis ossea. Thickening of facial bones (rare).
- Paraplegia due to vertebral involvement (rare).

Treatment

- Most patients require no treatment.
- Fractures will heal normally, but bony deformity with a fracture can be a difficult challenge!
- Drugs that reduce bone turnover, such as calcitonin or bisphosphonates, are effective in relieving pain and may also relieve neurological complications such as deafness.

Further reading

The Paget Foundation. Available at: http://www.paget.org/

The great toe

Hallux valgus ('bunions')

- Medial prominence of the first metatarsal head, with lateral deviation of the great toe (hence valgus) due to the pull of the extensors.
- As time passes, a protective bursa develops over the metatarsal head (the 'bunion') and the great toe begins to crowd, or even overlap, its neighbours.

Causes

- Congenital. Often familial, related to metatarsus primus varus where the first metatarsal is angled more medially, i.e. splayed, than usual and is rotated.
- Acquired. The commonest form. Probably due to weak intrinsic muscles due to age. It is not proven to be related to shoes, but there is a higher incidence in shoe-wearing cultures.

Symptoms

- Commonly asymptomatic, even in cases of severe deformity.
- Pain typically at the site of the bunion due to pressure.
- Bursal inflammation.
- Nerve symptoms may be present (compression of digital nerve).
- As the disease advances, symptoms of joint pain may present due to osteoarthritis and subluxation of the joint.
- Lesser toe deformities may be present (hammer toes, calluses).

Investigations

- Weight-bearing AP and lateral views of the foot.
- Measurements of the extent of deformity are made to guide management.
- Commonly calculated are the hallux valgus angle (between long axis of first metatarsal and corresponding proximal phalanx) and the first/second intermetatarsal angle (between the long axis of first and second metatarsals).

Treatment

Conservative

- Correct footwear with a wider toe box and padding to protect bunion.
- This should always be tried and have failed before considering surgery.

Surgical

- *Exostectomy*. Removal of the bunion alone. This is simple but does not remove the underlying deformity and the problem will recur.
- *Distal metatarsal osteotomy*. The bunion is removed and the metatarsal head or neck is cut. The distal fragment is then realigned anatomically and the fracture held with a K wire. There are many types or shapes of osteotomy described, but the commonest eponyms are Mitchell's, Wilson's, and Chevron. This is only suitable for smaller deformities.
- *Proximal metatarsal osteotomy*. This is an osteotomy just proximal to the base of the metatarsal and the metatarsocuneiform joint. Larger bony deformities can be corrected this way. It may be combined with a distal soft tissue release where the lateral constraints by the MTPJ are also released through a small separate dorsal excision.

- *Excision arthroplasty*. Removal of the metatarsal head (Mayo) or base of the proximal phalanx (Keller) can be attempted but is fraught with long-term complications and is only an operation for the elderly.
- *Arthrodesis*. This is suitable for severe deformity and degenerative change and is tolerated well by ♂. ♀ may have a problem with footwear (have to wear flat shoes after). It is normally reserved for salvage surgery.

Hallux rigidus

Degenerate arthritis of the first MTPJ, leading to pain and functional limitation of movement.

Causes

- Not fully determined.
- Congenital. Due to a shortened metatarsal.
- Acquired. Normally traumatic or idiopathic degeneration.

Symptoms

- Pain and swelling of the first MTPJ, with profound stiffness (limited dorsiflexion).
- May irritate on shoes.
- Neurological symptoms due to pressure on the digit between osteophyte and shoe.

Investigations

- AP and lateral X-rays of the foot (demonstrate osteoarthritic changes).

Treatment

- Adolescents/young. Rocker sole to relieve pain.
- Adults.
  - MTPJ replacement (rare).
  - Cheilectomy (excision of dorsal osteophyte and about 25% of dorsal metatarsal head).
  - Arthrodesis.
  - Excision arthroplasty (elderly only).

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Arthroplasty

- The surgical reconstruction or replacement of a malformed or degenerate joint.
- The 1° goal is to relieve pain.
- Increase in mobility and function is 2° aim.

Classification

- *Excision*, e.g. Keller's or Mayo at the first MTPJ.
- *Interposition*. A joint is excised and then a piece of tissue is implanted in the gap to cause a thick scar.
- *Partial (hemi-) or total replacement*. All or one-half of the articular surface is removed and replaced with other material. This has been made possible by the massive advances in both biomaterials and bioengineering, which have produced inert, sterilizable materials of acceptable strength to perform the joint functions.

Example: total hip replacement

Indications

- Osteoarthritis and RA when pain affects sleep, quality of life, and normal daily activities.
- Multiple joint involvement where hip is the worst.
- AVN of the head of the femur with 2° joint degeneration.

Prevention of infection

Deep infection is a potentially devastating complication of hip or any arthroplasty and its incidence should be ≥1% in all units. This is achieved by the following.

- *Ultraclean air systems and exhaust body suits*. The air in a conventional theatre is filtered, so that there are >20 changes/h. By using a unidirectional laminar flow system, 300 or more changes/h with filtration can be achieved. The purpose is to make the number of colony-forming units (CFUs) in the air the minimum possible. Body suits, although cumbersome, provide the best physical barrier between the patient and the surgical team.
- *Prophylactic antibiotics*. These are given IV on the induction of anaesthesia; 10min is usually required for them to penetrate bone to an acceptable level. Broad-spectrum antibiotics are usually used, e.g. cefuroxime 1.5g, co-amoxiclav 1.2g, and gentamicin 80mg being the commonest. Two further doses at 6 and 12h post-operatively are usually given.
- *Strict theatre discipline*.

Procedure

The surgical approach exposes both the femoral head and the acetabulum. The head of the femur is exposed, dislocated, and either reshaped (resurfacing arthroplasty) or, more commonly, removed at the neck. The acetabulum is then deepened and reshaped to allow a cup to be placed (the new 'socket'). A cavity is then created within the cut surface of the femur, going downward to allow a stem to be placed (the new 'ball'). The stem and cup are usually 'grouted' in place with polymethylmethacrylate bone cement, and the two components reduced and stability tested.

Sometimes the cup has a metal shell behind it and components can be hammered in, rather than cemented, and this is known as an 'uncemented hip replacement'. This is more commonly used on the younger patient in the UK but is the implant of choice in the USA. The wound is then closed. Patients are mobilized on day 1 post-operatively, fully weight-bearing, and discharged within 5–7 days usually.

Complications

Operative

- Sciatic nerve injury due to poor technique and overstretching of tissues.
- Dislocation of the prosthesis if incorrectly aligned.
- Profound hypotension can be seen, with absorption of the monomer in the cement causing cardiotoxicity.

Post-operative

- Mortality 1%. This is major surgery.
- Thromboembolic disease (DVT or PE).
- Deep infection (1%).
- Dislocation (4%). Usually due to patient non-compliance with physiotherapy guidelines.
- Aseptic loosening ('wearing out'). Most THRs would be expected to have ≤90% survival rates 10y after surgery.

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National Joint Registry. Available at: https://www.njrcentre.org.uk
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Chapter 17

Plastic surgery

Scope of specialty

Plastic surgery is a constantly evolving surgical discipline, based upon technical exactitude, detailed anatomical knowledge, and innovation. Plastic surgeons have strong aesthetic awareness, but the true scope of their practice is very much broader. In one of the last truly general specialties, plastic surgeons in the UK work and take referrals across surgical remits, including burns, skin cancer, head and neck oncology, facial re-animation, cleft lip and palate and craniofacial surgery, hand surgery, brachial plexus injury, breast surgery, trunk, perineal and urogenital reconstruction, sarcoma, and lower limb reconstruction. They treat cancers, trauma, and congenital conditions.

The ethos of this work is to restore form and function. In pursuit of this goal, techniques have been refined that enable the transfer of tissues around the body as non-vascularized 'grafts' or vascularized 'flaps' that may be 'pedicled' on their anatomical blood supply or revascularized after autologous transplantation by microvascular anastomosis (➔ Pedicled and free flaps: useful examples, pp. 716–17). These techniques allow the reconstruction of some of the most complicated tissue defects, including bone, nerve, muscle, subcutaneous tissues, and skin.

The development of surgical tools and our understanding of vascular anatomy have allowed successful transfer of large tissue blocks, based on vessels that can be smaller than 1mm in diameter. Reconstructions are often planned using CT and MRI scanning prior to surgery, making it possible to shorten the duration of anaesthesia and improve the reliability and quality of the reconstruction. Most recently, it has become possible to precisely shape these tissue blocks intraoperatively using 'thinning' of skin flaps or 'cutting guides'/templates that are custom-designed (or 3D-printed), based on preoperative imaging.

Globally, plastic surgeons collaborate with many specialties to enable oncological treatments and manage congenital abnormalities, trauma, and severe soft tissue infections (SSTIs) across a broad range of conditions. In addition to this work, plastic surgeons have been involved in the development of composite tissue allotransplantation techniques that include facial, abdominal wall, and hand transplantation.

In this chapter, we cover some of the common reasons for referral to plastic surgery and describe some of the common plastic surgery techniques available to address these.

Suturing wounds

Principles

A wound can be closed in the following ways:

- Allowing healing by 2° intention.
- Direct apposition of skin edges by sutures, glue, or staples.
- Skin grafts (➲ Grafts, pp. 710–12).
- Flaps (➲ Pedicled and free flaps: useful examples, pp. 716–17).

Key facts

Meticulous wound closure, particularly of excisional (as opposed to incisional) wounds, is the keystone of plastic surgery. Principles include:

- Orientation of incisions/scar lines along the lines of relaxed skin tension (see Fig. 17.1)
- Careful tissue handling to prevent additional tissue necrosis/fibrosis.
- Appropriate suture choice (absorbable versus non-absorbable; braided versus monofilament).
- Techniques to enable wound closure with small-diameter sutures.
- Appropriate suture technique (to achieve tissue apposition without tension and to balance wound edge eversion with contour).

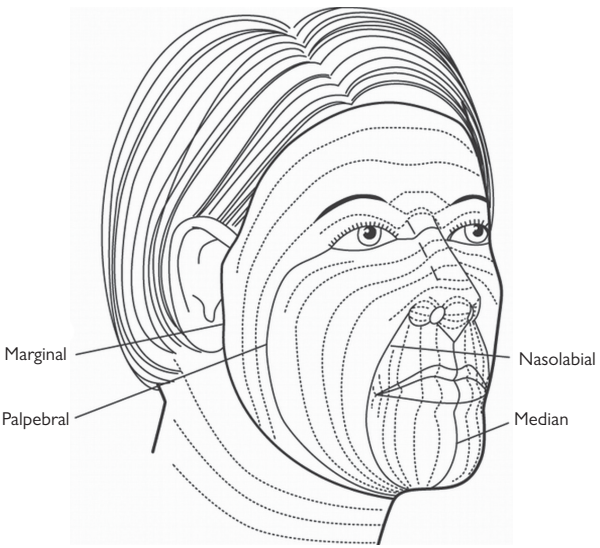

Fig. 17.1 Relaxed skin tension lines (RSTLs).

- Elimination of dead space and reconstruction of anatomical tissue planes (aiming to break up the scar in a vertical plane to prevent tethering to the deeper structures of the wound).
- Fastidious haemostasis without compromise to skin vascularity (to prevent haematoma).
- Appropriate measures to prevent infection.

Considerations for consent

In simple wounds, the common complications of any surgical procedure apply and should be discussed: infection, bleeding/haematoma, numbness or damage to the cutaneous nerve supply, and wound dehiscence/delayed healing. In addition to these, bear in mind the following:

- All wounds leave scars. There are no techniques that are currently available to achieve scar-less wound healing. Research in this area is ongoing.
- Some scars can be very aesthetically displeasing. These are hypertrophic and keloid scars. Wounds on the sternum, deltoid, and face/earlobe are particularly prone to developing this scar type. Patients from Afro-Caribbean, Chinese, and Japanese origin are at higher risk of developing these scars, but the aetiology is not fully understood. Treatments are available to improve 'ugly' scars, but a good surgical technique and management of patient-related factors are the best way to prevent them from developing in the first place (➲ Surgical management of scars, p. 724).

Wound closure technique

This procedure is commonly undertaken in A&E and its principles are applicable to a broad range of surgical specialties.

Before you start

- You will be performing an invasive procedure on an awake patient and it is important to build rapport and trust.
- Perform distal motor, sensory, and vascular examinations to exclude functional injury. In patients with positive signs, wound exploration should be performed in the operating theatre.
- Check the patient's medical history, allergies, and consent.
- Check tetanus status and take appropriate steps (➲ Tetanus, p. 229).
- Review/request X-rays and blood tests, including coagulation screen.
- The first step in wound closure is adequate debridement/irrigation.

Procedure

- Infiltrate the area with field block or regional LA (➲ Practical procedures, pp. 286, 290–2). Try not to infiltrate directly into the wound as this will obscure your subsequent operative view.
- Use a tourniquet where appropriate for the minimum time required. Awake patients are unlikely to tolerate this for >10min. In some patients (sickle cell disease/lymphoedema), a tourniquet may be contraindicated.

- Explore the wound with appropriate magnification and lighting. Make sure that you can see the base. Wounds that extend through the deep fascia are more likely to have a functional component, and in general, further exploration of wounds deeper than this level should be undertaken in the operating theatre.
- If the wound is simple (without a functional component) and amenable to closure, proceed to washout with sterile fluid. A volume of 1L of normal saline is suitable for most wounds.
- Excise or debride the wound to remove all contaminated or necrotic material.
- Consider placing surgical drains to reduce the risk of deep infection.
- Place interrupted stitches (see Fig. 17.2) in appropriate tissue layers.
- Do not stitch fat as this causes fat necrosis.
- Use buried stitches sparingly, just enough to reduce wound tension. The knot can act as a nidus of infection and occasionally these stitches can extrude, causing late complications.
- For interrupted external sutures, tie the knot so the skin edges are just apposed—the wound will swell post-operatively.
- Place the sutures evenly, approximately twice as far apart as they are from the wound margins.
- The distance between the suture and the wound margin should be similar to the thickness of the skin.
- Tailor your wound closure to the patient and their injury. In grossly contaminated/infected wounds (particularly in the hand), it is advisable to wash out, then to leave the wound open (no sutures) to heal by 2° intention. 2° reconstruction/scar revision can be undertaken by plastic surgeons.

Specific considerations

- Wound tape strips can give additional support until dermal healing has occurred.
- Wound glue should be reserved for small, simple wounds or those that are clean and well supported by deep stitches, as it offers little support for the dermis and risks trapping in deep infection.
- Rapidly absorbable stitches are often braided and confer a theoretically higher infection risk. They should be reserved for situations where suture removal may be traumatic or difficult (paediatric patients) and in relatively clean wounds.
- Staples can be used to close wounds quickly but may leave unsightly scarring, so they should be reserved for less aesthetically sensitive areas such as hair-bearing scalp, the torso, or lower limbs.

Post-operative management

- Give the patient appropriate PO analgesia for the nature of their injury.
- Provide a dressing that will keep the wound clean and allow it to breathe until it is healed. Options include semi-occlusive dressings (good for the limbs and trunk) or simple antibacterial ointments (good for highly contoured areas such as the face).
- Dressings can also be used to splint a wound or immobilize a limb during healing.
- Determine the appropriate timing of stitch removal (see Table 17.1).

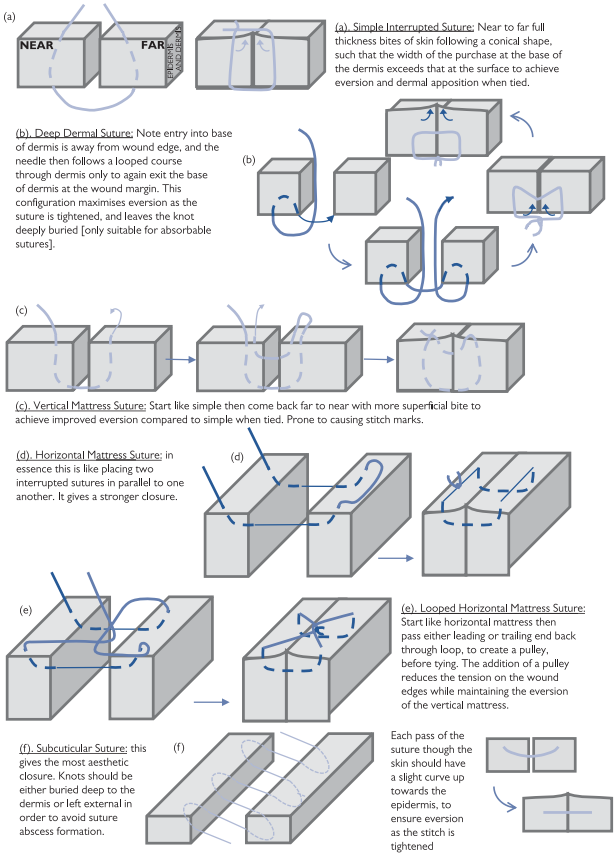

Fig. 17.2 Common suture techniques.

- Arrange an appropriate place for the stitches to be removed or wound checked (hospital dressing clinic/GP practice nurse).
- Consider a course of PO antibiotics as prophylaxis for contaminated wounds.
- For wounds with a high risk of complications, ensure appropriate follow-up with review by medical staff.
- Give the patient written information on how to manage their wound following discharge.
- Provide the GP with written documentation of the procedure performed and future management plans.

Common pitfalls

Even the best surgeons get complications. It is a statistical certainty. Good surgeons should take measures to reduce the incidence of complications through their management of surgical and patient factors.

Wound infection

- Late or inadequate wound washout/debridement.
- Poor selection of prophylactic antibiotics or post-operative dressings allowing subsequent wound contamination.
- High-risk patient factors—infection-prone site of injury (e.g. groin), diabetes, immunosuppression.
- Haematoma.
- Inadequate haemostasis.
- Inadequate elimination of tissue dead space.
- High-risk patient factors—anticoagulant medications, conditions of delayed coagulation.

Adverse scarring

- The 'tramline effect' is caused by epithelial growth into suture tracks that results from leaving stitches in for too long.
- Stitches that are tied too tightly cause tissue ischaemia and lead to 'cross-hatching'.
- Premature removal of stitches may lead to wound dehiscence.
- High-risk patient factors—previous hypertrophic or keloid scars; patients from Afro-Caribbean, Chinese, and Japanese origin; patients <30y or undergoing hormonal change (pregnancy/puberty).

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Table 17.1 Common stitch materials and their uses

Name	Structure	Time until dissolved	Practical use
Coated polygalactin 910	Braided	56–70 days	Deep dermal
Uncoated polygalactin 910	Braided	7–14 days	Cutaneous interrupted/ subcuticular
Poliglecaprone	Monofilament	91–119 days	Deep dermal/ subcuticular
Nylon	Monofilament	• N/A • Easier to handle than polypropylene	Cutaneous interrupted or continuous
Polypropylene	Mono filament	• N/A • Less tissue reaction than nylon	Cutaneous interrupted or continuous. Occasionally subcuticular (requires removal)

N/A, Non-absorbable.

Stitch choice and removal:

Use the finest suture possible to maintain wound closure—5/0 or 6/0 for the face; 4/0 or 5/0 for the hand; and 2/0 to 4/0 for the trunk.

In a low-tension wound closure, non-absorbable sutures may be removed at 5–7 days on the face, 7–10 days on the arm and anterior trunk, and 14 days on the back and lower limb.

Approaches to tissue reconstruction

Principles

Wounds that cannot be closed primarily can often be closed using grafts (➔ Grafts, pp. 710–12) and/or flaps (➔ Flaps: classification, pp. 714–15).

- The reconstructive ladder provides a framework that facilitates the choice of reconstructive technique.
- In the reconstructive ladder, tissue defects are treated using the *least invasive* means possible first. Quality of outcome may be compromised.
- The *reconstructive elevator* differs by treating tissue defects with the **most appropriate** reconstructive technique first.

Reconstructive ladder

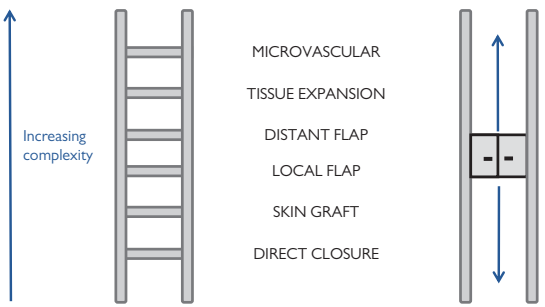
(See Fig. 17.3.)

- Historical approach where tissue defects are addressed primarily by the least invasive method. Reconstructive techniques are arranged hierarchically as 'rungs' on a ladder.
- Start at the lowest 'rung' possible. If the reconstruction fails, progress to the next 'rung' on the ladder to achieve wound closure.

Reconstructive elevator

(See Fig. 17.3.) Borrows on the reconstructive ladder concept, but here:

- The most appropriate reconstruction for a particular tissue defect is performed directly, regardless of invasiveness.
- This minimizes inpatient time and provides patients with the best functional reconstruction for their tissue defect.
- In practice, most modern plastic surgeons use this approach.
- If the first reconstruction fails, the other options on the ladder remain as 'lifeboats' to achieve wound closure.



Reconstructive Ladder: Stepwise progression through unsuccessful techniques until successful reconstruction is achieved.

Reconstructive Elevator: Initial selection of the highest quality reconstructive technique appropriate for that individual case.

Fig. 17.3 The reconstructive ladder.

Grafts

Definition

A graft is a unit of tissue that is completely removed from its original bodily attachment (the donor site). It is fixed to a recipient site and develops a new blood supply from the underlying tissue.

Skin grafts

A skin graft is a piece of skin of variable thickness. Skin grafts can be used to reconstruct tissue defects that have a vascularized base (graftable bed) and can come from various sources:

- *Autograft*. Transfer from one part of a person's body to another part.
- *Isograft*. Transfer between genetically identical individuals.
- *Allograft*. Transfer between individuals of the same species.
- *Xenograft*. Transfer between individuals of different species.

Full-thickness skin grafts (Wolfe grafts)

(See Table 17.2.)

- Contain epidermis plus the entire thickness of dermis.
- Adnexal structures, e.g. hair, are included.
- Harvested by elliptical excision from sites of skin laxity, e.g. post-auricular skin crease, supraclavicular, preauricular, groin, or medial upper arm skin.
- Graft secured with a tie-over dressing, e.g. proflavine-soaked cotton wool, and inspected after a week.
- Donor site primarily closed by suturing.

Split-thickness skin grafts (Thiersch grafts)

(See Table 17.2.)

- Consist of epidermis plus a variable thickness of dermis.
- Harvested by shaving off a layer of skin with a skin graft knife or dermatome. Can be taken from any area of the body (thigh skin most often used—plentiful and easy to access).
- Graft is often fenestrated (to stop blood or serous fluid collecting under it) or meshed (to expand the graft and allow it to contour to the wound bed).
- Graft secured with glue, sutures, or staples, then a non-adherent, compressive dressing. Inspected after 5 days.
- Defect heals by re-epithelialization from skin appendages.
- Donor sites heal by 2° intention.

Graft healing

Stages of graft take

- Adherence (immediate). Fibrin bond between graft and recipient bed.
- Plasmatic imbibition (days 2–4). Graft absorbs fluid and nutrients from bed.
- Revascularization (after day 4). Blood enters the graft, either by flowing directly into the graft vessels (inosculation) or by new vessel ingrowth (neovascularization).

Table 17.2 Split-thickness grafts versus full-thickness grafts

	Split-thickness skin graft	Full-thickness skin graft
Cosmesis	Thin; leaves marked contour defect	Good cosmesis, less contour defect
Contracture	Frequent; this can be an advantage where wound cicatrization is desirable	Less frequent
Availability	Plentiful; can reharvest after 14 days	Limited by skin laxity
Take	Good—low metabolic needs	Needs optimal bed
Donor scar	Minimal—colour change only	Linear scar
Contraindications	• Ungraftable or infected bed • Aesthetically sensitive areas or where contracture will limit function	• Ungraftable or infected bed • Large area to be covered

Reasons for graft failure

- Shearing. Revascularization cannot occur if the graft is mobile.
- Infection. Either of the bed or of the graft tissue.
- Separation of graft from its bed. By haematoma or seroma.
- Ungraftable bed, e.g. bare cortical bone; tendon without peritenon, cartilage without perichondrium.
- Damage to the graft, e.g. poor surgical technique, graft placed upside down.

Other free grafts

Fat, tendon, nerve, bone, cartilage, and fascia can be harvested as free grafts. The potential uses and donor sites are limited only by imagination:

- Fat can be harvested by liposuction and concentrated by centrifugation (Coleman technique) for use in 2° breast reconstruction. The abdomen and thighs are common donor sites.
- Palmaris longus and plantaris tendons are used to secondarily reconstruct tendon defects of the hand.
- The sural nerve is used to reconstruct deficits of the brachial plexus. Harvest leaves a numb patch on the lateral border of the foot.
- Free cortical and cancellous bone is harvested from the iliac crest and used to reconstruct the alveolar defect in cleft palate patients.
- Tensor fascia lata (TFL) can be used to provide static support in the absence of a functional facial nerve.

Composite grafts

More than one tissue type is included in a composite graft. Take follows that for other free grafts with a shorter period of plasmatic imbibition. The vertical depth of tissue grafted is larger, meaning composite grafts are only suitable for defects where the wound bed is optimally vascularized and there is absence of infection. Common examples include:

- Skin, subcutaneous tissue, and cartilage to reconstruct defects of the soft triangle of the nose.
- Re-attachment of a fingertip as a composite graft in a child.
- Nipple share in breast reconstruction.

Stem cell grafts

Stem cell grafts are currently being investigated for use as a 1° regenerative technique and to augment the take of other free grafts.

- Grafted stem cells have the capacity to regenerate tissue primarily, and through the secretion of chemokines, e.g. vascular endothelial growth factor (VEGF), brain-derived neurotrophic factor (BDNF), that stimulate local cells (such as fibroblasts and osteocytes) to support the regenerative process.
- Adipose-derived stem cells (ASCs) are the most commonly used stem cells in plastic surgery.
- ASCs are harvested by liposuction and extracted from the lipoaspirate by a series of collagen digestion, washing, and centrifugation steps. ASCs are mesenchymal in origin but have been shown to be capable of differentiating along multiple cell lineages, including bone, muscle, cartilage, and even nerve.
- ASCs are in phase II trials as a 1° regenerative approach to the treatment of scleroderma of the fingers in Europe (SCLERADEC trial), and their addition to free fat grafts has been shown to enhance volume retention following Coleman fat grafting to the breast.

Further reading

Coleman SR (1997). Facial recontouring with lipostructure. *Clin Plast Surg* **24**: 347–67.

Gentile P, Scioli MG, Orlandi A, Cervelli V (2015). Breast reconstruction with enhanced stromal vascular fraction fat grafting: what is the best method? *Plast Reconst Surg Global Open* **3**: e406.

Guillaume-Jugnot P, Daumas A, Magalon J, *et al*. (2016). Autologous adipose-derived stromal vascular fraction in patients with systemic sclerosis: 12-month follow-up. *Rheumatology* **55**: 301–6.

Flaps: classification

Definition

A flap is a unit of tissue of variable composition which, when transferred from donor to recipient site, brings its own blood supply and intrinsic circulation.

Classification

Broadly, flaps may be classified as local (from tissue adjacent to the defect), pedicled (contains directional blood supply and may be local or regional to the defect), or free (from a distant site and moved to the defect by microsurgical autotransplantation). More specifically, flaps can be classified according to the five 'Cs':

Circulation

By flap blood supply:

- *Random pattern*. No directional blood supply. Survive on blood vessels in dermal and subdermal plexuses which have no specific anatomical pattern. Length-to-breadth ratio is limited to 1:1 (or 4:1 on the face, e.g. most local flaps) (see Fig. 17.4).
- *Axial pattern*. Directional blood supply in angiosomes (Taylor and Palmer)[1]; length-to-breadth ratio can be greatly ↑. Axial flaps include: direct, fasciocutaneous (subclassified by Cormack and Lamberty)[2], musculocutaneous (subclassified by Mathes and Nahai)[3], and venous flaps (subclassified by Thatte and Thatte)[4].

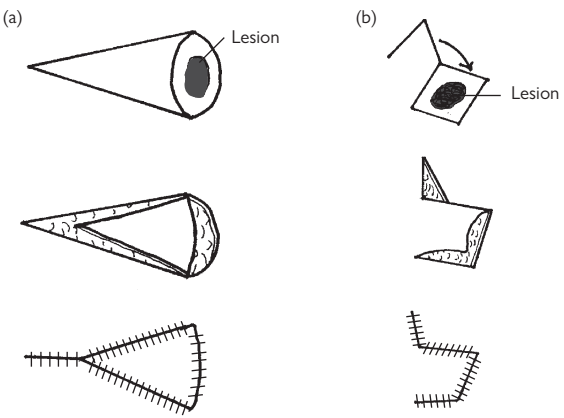


Fig. 17.4 Types of common local flaps. (a) V-Y flap. (b) Rhomboid flap.

Composition

By type of tissue contained within the flap:

- Examples are: cutaneous or adipocutaneous, e.g. deep inferior epigastric artery perforator (DIEP); fasciocutaneous, e.g. anterolateral thigh (ALT); musculocutaneous, e.g. latissimus dorsi; osseocutaneous, e.g. fibula; muscle only, e.g. gracilis; bone only, e.g. deep circumflex iliac artery (DCIA); fascia only, e.g. TFL; or various combinations of these.

Contiguity

By proximity of donor relative to recipient:

- Local, regional, distant, free.

Contour

According to the method of transfer into the defect:

- *Advancement*. The base of the flap advances in the direction of the flap axis (e.g. V-Y local flap).
- *Pivot flaps*. These include transposition, rotation, and interpolation flaps:
  - *Transposition*. Usually a rectangular or square flap that moves from a pivot point laterally to close a defect. The donor site often requires 2° grafting (e.g. fasciocutaneous flap for lower limb).
  - *Rotation*: a semicircular flap that rotates around a pivot point to close the defect, commonly allowing 1° closure of the donor (e.g. scalp rotation).
  - *Interpolation*. Flap passes under or above adjacent skin to reach recipient site, whilst moving about a pivot point (e.g. deltopectoral flap for head and neck).

Crane principle Flap placed into an ungraftable defect for a number of weeks to allow granulation at the base; then the flap is placed back at the donor, converting the recipient site into a graftable bed.

Conditioning

Flaps can be 'delayed' when their blood supply is restricted at the donor, usually weeks prior to inset of the flap to the recipient. This process 'conditions' the flap to hypoxic/ischaemic insult, allowing a larger flap to be raised at the time of definitive reconstruction (e.g. deltopectoral flap).

References

1 Taylor GI, Palmer JH (1987). The vascular territories (angiosomes) of the body: experimental study and clinical applications. *Br J Plast Surg* **40**: 113–41.
2 Cormack GC, Lamberty BG (1984). A classification of fascio-cutaneous flaps according to their patterns of vascularisation. *Br J Plast Surg* **37**: 80–7.
3 Mathes SJ, Nahai F (1981). Classification of the vascular anatomy of muscles: experimental and clinical correlation. *Plast Reconstr Surg* **67**: 177–87.
4 Thatte MR, Thatte RL (1993). Venous flaps. *Plast Reconstr Surg* **91**: 747–51.

Pedicled and free flaps: useful examples

There are myriad reconstructive flaps available. Here are brief descriptions of some of the more commonly used flaps.

Radial forearm

- *Type*: free or pedicled (proximally or distally)/adipofascial, fasciocutaneous, or composite with bone, flexor carpi radialis, or palmaris longus tendons and lateral cutaneous nerve of forearm/superficial radial nerves.
- *Use*: thin flap with multiple uses, commonly used for reconstruction of head and neck and dorsal hand defects.
- *Vascular basis*: can supply skin from entire forearm but usually take less than this. Based on radial artery and its venae comitantes. The cephalic vein can be included in the flap to improve venous drainage. Pedicle length up to 20cm, with vessels 3–5mm in diameter.

Latissimus dorsi

- *Type*: free or pedicled. Muscle only, extended to include fat and fascia, musculocutaneous, or osteocutaneous. The thoracodorsal nerve can be included to give a functional muscle transfer.
- *Use*: the workhorse of breast reconstruction, this flap has multiple uses as it provides a broad, flat muscle with a long pedicle that can be contoured to tissue defects.
- *Vascular basis*: the skin overlying the muscle and for 10cm anteriorly can be used, which is supplied by the thoracodorsal artery and a single venae comitans. Pedicle length up to 15cm, with vessels 2–5mm in diameter.

Rectus abdominis (DIEP/TRAM/VRAM)

There are several variations of this flap, based on composition and circulation—DIEP (no muscle, skin and fat only), transverse (TRAM) or vertical rectus abdominis myocutaneous (VRAM), which differ on orientation of skin island (skin, fat, and ipsilateral rectus abdominis), and rectus abdominis (muscle only) can all share a vascular basis. A muscle-sparing variant of the TRAM/VRAM can also be raised to include a segment of the ipsilateral rectus abdominis muscle and confers less donor morbidity.

- *Type*: free or pedicled, adipocutaneous, musculocutaneous, or muscle only.
- *Use*: popular for breast reconstruction as a free flap and pedicled to difficult defects from nipple to thigh, including sternal dehiscence following cardiac surgery.
- *Vascular basis*: can supply large ellipse of infra-umbilical skin to symphysis pubis. For VRAM, this is vertically oriented over the ipsilateral rectus muscle. Based on the deep inferior epigastric artery and its venae comitantes (branch of external iliac). The rectus abdominis can additionally be raised on the superior epigastric vessels (branch of internal mammary) to increase superior reach as pedicled flap. Pedicle length >7cm, with vessels 2–4mm in diameter.

Anterolateral thigh

- *Type*: free or pedicled, fasciocutaneous, musculocutaneous. The lateral cutaneous nerve of the thigh can be included if a sensate flap is required.
- *Use*: a workhorse flap for trauma, the option of inclusion of a section of the vastus lateralis increases the versatility of this flap, which has minimal donor site morbidity. It can be pedicled from lower abdomen to knee.
- *Vascular basis*: an island of skin 15cm wide and up to 38cm long can be raised over the perforators supplied by the descending branch of the lateral circumflex femoral artery (branch of profunda femoris) and its venae comitantes. Pedicle length up to 7cm, vessel diameter 1.5–2.5mm.

Gracilis

- *Type*: free or pedicled, musculocutaneous or muscle only. A branch of the obturator nerve can be incorporated if a functionalized muscle is required.
- *Use*: an option for small-volume breast reconstruction, the gracilis flap is most versatile in lower limb trauma and as a functionalized muscle (e.g. facial re-animation). It also can be used as a pedicled flap for volume following extended abdominoperineal resection or in cases of vagino-colic fistula.
- *Vascular basis*: an island of skin 8–10cm wide by 20cm long can be raised over the muscle supplied by the medial femoral circumflex artery (branch of profunda femoris) and its venae comitantes. Pedicle length up to 6cm, with vessels 1–2mm in diameter.

Fibula

- *Type*: free or pedicled, osseous, osteocutaneous or osteomusculocutaneous, including a cuff of flexor hallucis longus ± soleus.
- *Use*: as a good source of vascularized bone, the fibular flap is most useful in reconstruction of the mandible but has found use in traumatic and infectious defects of the upper and lower limbs where bone ± a small amount of muscle is required. As a pedicled flap, it can reach the knee proximally and distal tibia/ankle distally.
- *Vascular basis*: an island of skin 8cm wide × 18cm long can be raised over the bone supplied by the peroneal artery (branch of posterior tibial artery) and its venae comitantes. Length up to 2–4cm, with vessels 1.5–4mm in diameter.

Further reading

Buntic R. *Atlas of Microsurgery Techniques and Principles* ((a free online resource, requires registration). Available at: https://www.microsurgeon.org

Strauch B, Vasconez LO, Herman CK, Lee BT (2015). *Grabb's Encyclopedia of Flaps*, 4th edn. Lippincott Williams & Wilkins, Philadelphia, PA.

Burns: assessment

Assessment and management of burns go hand in hand and are simultaneous in practice. They have been divided here only for ease of reading.

Causes Most burns are due to flame or contact with hot surfaces; scalds are commoner in children and the elderly. Chemical, electrical, irradiation, and friction burns are rare.

History

- Find out the exact mechanism, including temperature of water, duration of contact, concentration of chemical, and voltage.
- Record factors suggesting inhalation injury, e.g. burns in a confined space, flash burns.
- Enquire about other injuries.
- Document first aid given so far.
- Document timings of injury, first aid, and resuscitation.

Examination

- *Estimate area of burn*. Do not include areas of unblistered erythema.
- Wallace rule of nines (see Fig. 17.5).
- Patient's hand, including fingers, is ~1% total body surface area (TBSA) in adults.
- Lund and Browder chart (see Fig. 17.6) is the most accurate method.

Estimating depth of burn

- *Epidermal*. Erythema only.
- *Superficial partial thickness*. Pink, wet or blistered, blanches and refills, sensate.
- *Mid-deep dermal*. Blotchy red, wet or blistered, variable blanching, may be insensate.
- *Full thickness. White or charred, leathery, no blanching,* insensate.

Signs of inhalation injury

- Singed nasal hair.
- Burns to face or oropharynx. Look for blistered palate.
- Sooty sputum.
- Drowsiness or confusion due to carbon monoxide inhalation.
- Respiratory effort, breathlessness, stridor, or hoarseness are signs of impending airway obstruction and require immediate intubation.

Features of non-accidental burns injury

Refer to paediatric burns unit if suspected in a child. Features include:

- Delayed presentation.
- History inconsistent or not compatible with injury.
- Other signs of trauma.
- Suspicious pattern of injury, e.g. cigarette burns, bilateral 'shoes and socks' scalds.

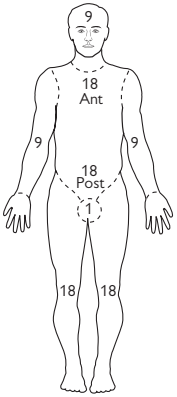
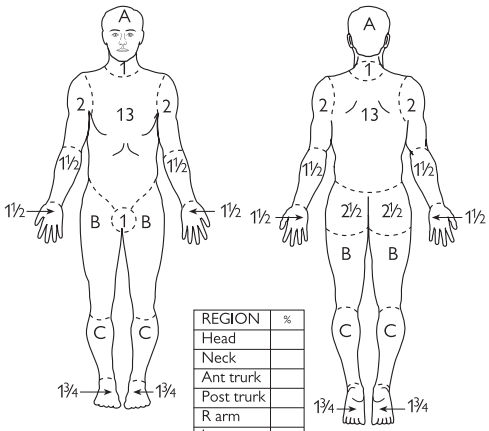


Fig. 17.5 Wallace rule of nines.



REGION	%
Head	
Neck	
Ant trunk	
Post trunk	
R arm	
L arm	
Buttocks	
Genitalia	
R leg	
L leg	
Total	

AREA	0	1	5	10	15	Adult
A = ½ of head	9½	8⅓	6½	5½	4½	3½
B = ½ of one thigh	2¾	3¼	4	3½	3½	4¾
C = ½ of one lower leg	2½	2½	2¾	3	3¼	3½

Fig. 17.6 Lund and Browder chart.

Burns: management

Immediate first aid

- ▶▶ Stop the burning process (do not endanger yourself).
- ▶▶ Cool the wound. Running water at 2–15°C for 20min (beware risk of hypothermia in infants, young children, and adults with >25% TBSA).

- A. Airway maintenance with C-spine control. Intubate if suspected inhalation injury; airway oedema can be rapidly fatal.
- B. Breathing and ventilation.
- C. Circulation with haemorrhage control.
- D. Disability and neurological status.
- E. Exposure and environmental control.
- F. Fluid resuscitation: child, >10% TBSA; adult, >15% TBSA burnt.

Resuscitation

- Two large peripheral IV lines, preferably through unburnt skin.
- Send blood for FBC, U&Es, clotting, amylase, carboxyhaemoglobin.
- Give 3–4mL of Hartmann's solution/kg/% TBSA burnt. Half of this is given over the first 8h following injury, and half over the next 16h.
- Children need maintenance fluid in addition.
- Monitor resuscitation with urinary catheter (aim for urine output of 0.5–1mL/kg/h in adults and 1–1.5mL/kg/h in children).
- Consider ECG, pulse, BP, respiratory rate, pulse oximetry, and ABGs.
- Perform 2° survey.

Referral to a burns unit

(See Box 17.1.) Intubate before transfer if inhalation injury suspected. Give humidified 100% O_2 to all patients. Wash the burn and cover with cling film. Give IV morphine analgesia. Discuss NGT and catheter insertion with burns unit. Give tetanus prophylaxis if required (➲ Tetanus, p. 229). Consider need for emergency surgery prior to transfer (see Box 17.2).

Box 17.1 Criteria for referral to a burns unit

- >10% TBSA burn in adult; >5% TBSA in child.
- Burns to face, hands, feet, perineum, genitalia, and major joints.
- Full-thickness burns >5% TBSA.
- Electrical or chemical burns.
- Associated inhalation injury—always intubate before transfer.
- Circumferential burns of limbs or chest.
- Burns in very young or old, pregnant women, and patients with significant comorbidities.
- Any burn associated with major trauma.

Management of the burn wound

- Superficial dermal burns will heal without scarring within 2–3 weeks as long as infection does not deepen the burn.
- For small burns, outpatient treatment with simple, non-adherent dressings and twice-weekly wound inspection is sufficient.
- Wash burns with normal saline or chlorhexidine.
- Debride large blisters. Elevate limbs to reduce pain and swelling.
- Dress hands in plastic bags to allow mobilization.
- Topical silver sulfadiazine is used on deep burns to reduce risk of infection (but should not be applied until the patient has been reviewed by a burns unit as it makes depth difficult to assess).

Box 17.2 Emergency surgery (escharotomy/fasciotomy)

- *Escharotomy*. Performed for circumferential full-thickness burns to the chest that limit ventilation or to the limbs that limit circulation. Loss of pulses or sensation is a late sign. In the early stages, pain at rest or on passive movements of distal joints indicates ischaemia.
- *Fasciotomy*. Required in compartment syndrome associated with burns. Look for it specifically in burns associated with trauma and high-voltage electrical injuries.

Electrical injuries

- *Low voltage (<1000V)*. Domestic electrical supply. Causes local contact wounds, but no deep injury. May cause cardiac arrest.
- *High voltage (>1000V)*. High-tension cables, power stations, lightning. Causes cutaneous and deep tissue damage with entry and exit wounds. Muscle damage may require fasciotomy.
- Myoglobinuria can cause renal failure. Aim urine output of >75–100mL/h and consider alkalinization and osmotic diuresis.
- ECG on admission for all injuries. Continuous cardiac monitoring for 24h for significant and high-voltage injuries.

Chemical burns

- Treat with copious lavage for at least 30min until all chemical has been removed and skin pH is normal.
- *Acid*. Causes coagulative necrosis; penetrates skin rapidly but is easily removed.
- *Alkali* (includes common household chemicals and cement). Causes liquefactive necrosis so needs longer irrigation (>1h).
- *Hydrofluoric acid*. Fluoride ions penetrate burnt skin, causing liquefactive necrosis and decalcification; 2% TBSA burn can be fatal.
  - Irrigate with water.
  - Trim or remove fingernails.
  - Topical calcium gluconate gel, 10%.
  - Local injection of 10% calcium gluconate.
  - IV calcium gluconate.
  - May need urgent excision of burn.

- Elemental Na, K, Mg, and Li. Do not irrigate initially; they ignite in water. Brush off particles and consider oil lavage.
- *Phosphorus*. Irrigate with water, then debride particles which will otherwise continue to burn. Apply copper sulfate, which turns particles black so they are easier to identify.
- *Bitumen*. Burns by heat; treat by cooling with water. Remove cold bitumen with peanut or paraffin oil.
- *Tar*. Burns by heat. Treat by cooling with water; no need to remove tar as it gradually gets emulsified with topical ointments used for treatment.

Early burns surgery

- Performed for deep dermal or full-thickness burns that are too large to heal rapidly by 2° intention. Usually performed within 72h.
- Excision of burn should be to healthy tissue.
- This may be tangential (with skin graft knife or scalpel) or full thickness (using monopolar diathermy), depending on burn thickness.
- Reconstruction may take the form of grafts and/or flaps.

Surgical dressings/tissue engineering

For large burns, there are a range of options that can either temporize the wound closure or improve the quality of definitive skin grafts in sensitive anatomical areas.

Split skin allograft

- From live-related or live unrelated donor or from cadaveric unrelated donor.
- Usually screened, but small risk of transmission of infection.
- Will ultimately be rejected but can prepare wound bed for subsequent skin graft and prevent ongoing wound losses.
- Cadaveric skin can be fresh frozen, irradiated, or glycerol-preserved.
- *Human amnion*. Used in developing countries as a dressing for superficial partial-thickness (SPT) burns.
- *Porcine xenograft*. An alternative to human amnion.
- *Biobrane™*. Dressing containing silicone film with nylon fabric bound to porcine dermal collagen. Good for SPT burns.
- *TransCyte™*. Similar to Biobrane™ in structure; includes collagen, matrix proteins, and growth factors from neonatal human fibroblasts to encourage healing. Good for SPT burns.
- *Integra™*. A bilaminar dermal template composed of bovine tendon collagen, shark glycosaminoglycan, and silicone. Good for mid-dermal to full-thickness burns as a one- or two-stage technique.
- *MatriDerm™*. A dermal template of bovine dermal collagen and nuchal ligament elastin. It is applied with a split-thickness skin graft as a single stage.
- *Cultured epithelial autograft*. Autologous cells are cultured from a skin biopsy over a period of 1–3 weeks and re-sprayed or re-laid onto the patient. The regenerated skin lacks dermis and its quality is variable if used alone.

Surgical management of scars

Principles

- Scars can be managed non-surgically by observation, massage, pressure, silicone gel or tape, laser, steroid or cytotoxic injection, and radiotherapy.
- Surgical options include excision and re-suture, Z-plasty, W-plasty, and resurfacing by means of tissue import.

Surgical treatment of scars

- *Excision and closure*. For stretched scar or scar with 'tramlines'. New scars usually re-stretch to some extent. Can be performed as 'staged' procedure to minimize tension.
- *Z-plasty* (see Fig. 17.7a, b). Local flap, lengthens and can re-orientate scar into RSTLs (see Fig. 17.1). Good for narrow band scars, e.g. 2° treatment of burn injury across joints.
- *W-plasty* (see Fig. 17.7c). Local flap, breaks up line of scar. Good for scalp to avoid a hairless scar or cheek to break up scar line.
- *Scar release and resurfacing*. Good for wide band scars, e.g. 2° treatment of burns. Resurfacing may include skin grafting or all types of contiguous flap (➲ Grafts, pp. 710–12).

(b)

Angle (Z) degrees	Lengthening
30	25%
45	50%
60	75%
75	100%
90	125%

Fig. 17.7 Surgical techniques for managing scars. (a) Z-plasty. (b) Lengthening according to angle. (c) W-plasty.

Soft tissue hand injuries

History

- Mechanism of injury.
- Dominant hand, occupation, hobbies.
- Medical and smoking history, previous hand injuries, social history.

Examination

Use LA block if needed for pain (check sensation first).

Look Posture of hand and digits. Site of laceration(s) and tissue loss.

Feel Perfusion of hand and digits, pulses. Sensation in distribution of radial, ulnar, median, and digital nerves (see Fig. 17.8). Pain over bones.

Move

- Long extensors extend metacarpophalangeal joints (MCPJs).
- Extensor pollicis longus (EPL) extends thumb dorsal to plane of hand (i.e. up off a table).
- Flexor digitorum profundus (FDP) tendons flex distal interphalangeal joints (DIPJs).
- Flexor digitorum superficialis (FDS) tendons flex proximal interphalangeal joints (PIPJs). Isolate FDS by holding all digits, except the one under examination extended.
- Testing wrist flexors and extensors is unreliable as finger flexors and extensors may mimic function, but pain on movement suggests injury.
- Examine intrinsics and hypothenar and thenar muscles, particularly abductor pollicis brevis (supplied by median nerve) and Froment's sign (for adductor pollicis supplied by ulnar nerve).
- Check stability of joints. Pain or abnormal movement on lateral deviation suggests collateral ligament damage.
- Document injury in relation to flexor/extensor zones (see Fig. 17.9).

Investigations X-ray for fractures of foreign bodies. Photographs.

Treatment

- *Finger pulp injury*. Debride under tourniquet. If there is no bone exposed, it will heal by 2° intention. Exposed bone may need surgery to shorten bone or cover it with a local flap.
- *Subungual haematoma*. Painful bruise under nail. Trephine nail with sterile needle or hot wire to evacuate haematoma.
- *Nailbed injury*. Often with distal phalanx (DP) fracture. Remove nail under tourniquet; irrigate wound; repair nail with absorbable 7/0 suture using loupe magnification. Replace fenestrated nail as splint for eponychial fold.
- *Mallet finger*. Immobilize in splint for 6–8 weeks unless large bony fragment present which may require surgical fixation.
- *Foreign bodies*. Remove organic matter and painful foreign bodies.
- *Lacerations and puncture wounds*.

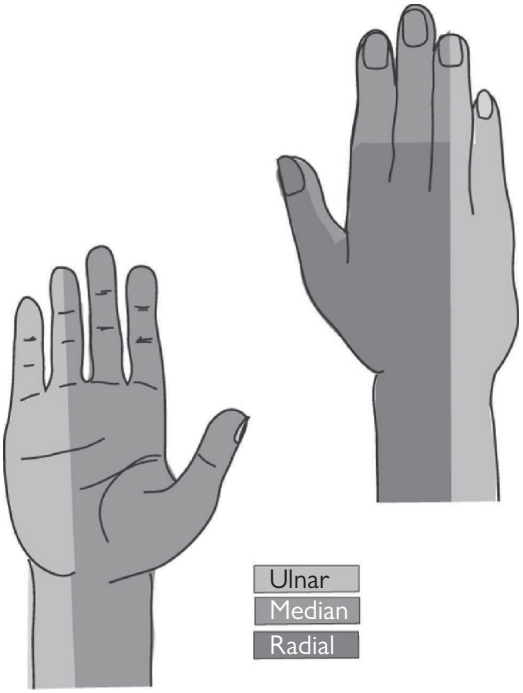


Fig. 17.8 Nerve supply to the hand.

Reproduced with permission from Giele, H. and Cassell, O., Plastic and Reconstructive Surgery, (2008) Oxford University Press.

- Always explore with anaesthetic and tourniquet to determine underlying structural damage.
- Irrigate wounds and debride as necessary.
- Tetanus prophylaxis (➲ Tetanus, p. 229).
- Co-amoxiclav (625mg tds PO) for animal and human bites.
- Repair tendons, ideally primarily within 48h. Post-operative regimes typically involve splints for 6 weeks with physio-directed exercise and 6 more weeks without heavy lifting.
- Repair nerves under magnification. Axonal regeneration progresses at ~1mm/day after 1–3 months from repair.
- Thoroughly irrigate open joints due to risk of septic arthritis.
- Collateral ligaments may need to be repaired and are splinted for around 4 weeks post-repair.

- *Complications*. Haematoma, infection, tendon or ligament rupture, stiffness, painful scars, neuroma, complex regional pain syndrome, scar contracture, cold sensitivity.

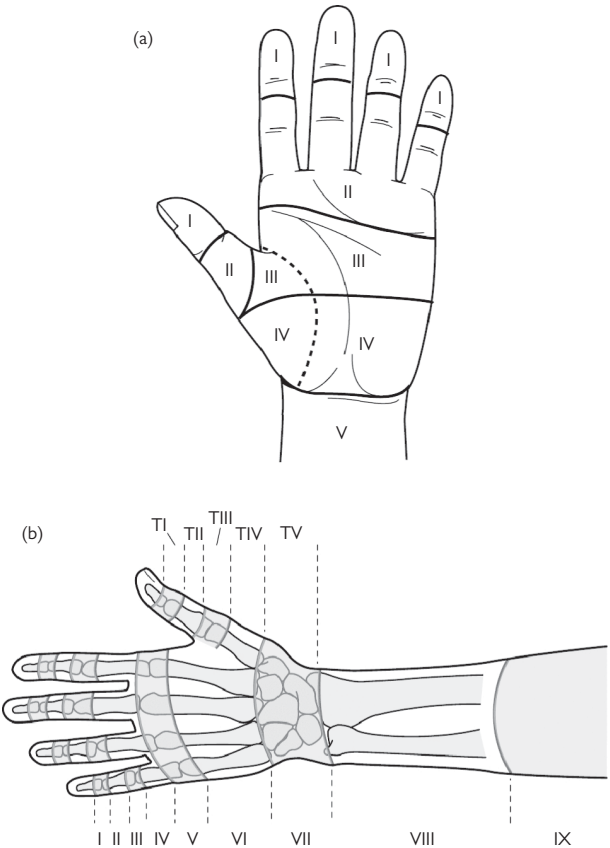


Fig. 17.9 Flexor and extensor tendon zones.

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Hand infections

Key facts

Usually follows a penetrating injury (which may seem insignificant) or a bite.

- *Infecting organisms*. After a penetrating injury, *Staphylococcus aureus* is the commonest, followed by streptococci. Human bites are often also contaminated with *Eikenella corrodens*. *Pasteurella* spp. are common in infected cat and dog bites. Bites carry a high risk of infection—always refer for surgical exploration.
- *Paronychia*. Infection of nailfold. *Candida albicans* causes chronic paronychia and may require excision of crescent of eponychium and topical antifungals. Herpes simplex causes whitlow with vesicles around the nail, but no pus. Avoid surgery in these cases.
- *Felon*. Finger pulp infection.
- *Palmar space infection*. There are four fascial compartments in the palm (web space, hypothenar, mid palm, and thenar). They usually confine infection initially. Pain, swelling, and reduced movement are features. Swelling is often more prominent on the dorsal surface of the hand.
- *Flexor sheath infection*. ▶ The cardinal signs are flexed posture of finger, pain on passive extension, fusiform swelling, and pain along flexor sheath. Requires emergency decompression to prevent damage to the flexor tendons.

Treatment

❗ Delay can be disastrous, resulting in stiffness, contracture, and pain. Any collection of pus must be surgically drained urgently.

Initial treatment

- Tetanus prophylaxis (➲ Tetanus, p. 229).
- Elevation and splintage.
- Broad-spectrum antibiotics (IV co-amoxiclav 1.2g tds for bites unless penicillin allergy) until sensitivities known.
- Plain X-ray may be useful to exclude associated fractures, foreign bodies, underlying osteomyelitis, and evidence of gas-forming infection.

Surgical treatment

- Use a tourniquet, but elevate rather than exsanguinate the limb.
- Send pus swabs and tissue samples for culture.
- Debride and irrigate wounds; fully explore pockets of pus.
- Leave wound open for delayed 1° closure or healing by 2° intention.

Post-operative care

- Continue high elevation. Antibiotics until infection resolved.
- Splint for comfort with wrist extended (0–30°), MCPJs flexed (70°), and interphalangeal joints (IPJs) extended (safe position).
- Mobilize with physiotherapists.

Necrotizing soft tissue infections

Key facts

- Infections that present with pain out of proportion to the injury, soft tissue crepitus, dermal thrombosis, and a rapidly deteriorating clinical picture should be suspected of necrotizing infection.
- The diagnosis is a clinical one; this is a surgical emergency that carries a high mortality (20–25%).
- Popular terms include necrotizing fasciitis, gas gangrene, and Fournier gangrene (specific to perineum and scrotum). ➲Soft tissue and bone infections, pp. 228–9
- Can occur sporadically and in absence of penetrating injury.
- Most at risk are those with local trauma, recent surgery, chronic alcohol and IV drug abuse, and chronic systemic disease.
- Systemic factors are immunodeficiency, HIV/AIDS, steroid use, DM, obesity, and peripheral vascular disease.
- Commonly polymicrobial.

Microbiological classification

- *Type 1* (70–80%). Mixed anaerobes and aerobes, *Clostridia* spp. Indolent clinical presentation.
- *Type 2* (20–30%). Often mono-microbial, skin- or throat-related organisms (group A β-haemolytic *Streptococcus*, *Staphylococcus aureus*). Rapid clinical deterioration.
- *Type 3* (rare). Gram-negative, often marine-related (*Proteus* spp.). Indolent onset, high mortality.
- *Type 4* (rare). Fungal, usually in immunocompromised patients (*Candida* spp., *Zygomycetes* spp.). Rapid clinical deterioration.

Investigations

- ▶▶ No investigation should delay early surgical treatment.
- X-rays/CT may demonstrate subcutaneous gas or a foreign body.
- LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score may have useful positive predictive value in cases of clinical uncertainty, but not to be relied on for diagnosis.
- Test incisions can be performed under LA/GA to inspect the deep fascia directly. 'Dirty dishwater' fluid in the fascial planes represents a positive test.

Treatment

- ▶▶Aggressive fluid resuscitation with invasive monitoring. Patient often require organ support (ITU) as sepsis can develop rapidly.
- ▶▶Prompt broad-spectrum antibiotic therapy.
- Analgesia, including opiate medications.
- ▶▶Early surgical debridement of affected anatomical areas involves wide debridement of skin and subcutaneous tissue ± fascia and muscle.
- Fasciotomies are occasionally indicated when muscle is involved.
- Limb amputation is sometimes indicated to preserve life.
- Debridement is repeated at 24–28h until infection resolved.
- Reconstruction is by grafts and flaps when infection resolved.

Lower limb trauma

Principles

- Joint British Association of Plastic, Reconstructive and Aesthetic Surgeons (BAPRAS) and British Orthopaedic Association (BOA) standards have been published to normalize treatment for Gustillo IIIb or IIIc fractures of the lower limb.
- Involves multidisciplinary approach and includes recommendations for timing and delivery of care that exemplify best practice.

Which patients?

- All patients with severe open fractures of the lower limb.
- *Fractures*. Comminuted tibial fracture with fibula fracture at same level, segmental fracture, bone loss before or after debridement.
- *Soft tissue*. Where direct closure without tension is not possible, there is presence of degloving or muscle injury requiring debridement by wound extension, arterial damage, or wound contamination by marine, sewage, or agricultural material.

Treatment

- EMST (Emergency Management of Severe Trauma)/ATLS (Advanced Trauma Life Support) *Initial assessment*. Treat children the same as adults. Perform EMST/ATLS resuscitation.
- Assess vascular and neurological status of limb systematically; document and repeat regularly, especially following fracture manipulation.
- Perform minimal wound handling; remove gross contamination; photograph, and dress with a saline-soaked gauze/impermeable film.
- IV antibiotics should be administered within 3h of injury (co-amoxiclav 1.2g tds or clindamycin 600mg qds in penicillin allergy) and continued until wound debridement.
- Take orthogonal X-rays of affected limb.
- Reduce fractures and splint limb to include knee and ankle.
- ▶▶ *Immediate surgery*. Proceed directly to theatre in wounds contaminated by marine, agricultural, or sewage matter, vascular injury (maximum warm ischaemia 6h), and compartment syndrome (decompress all four compartments via standardized incisions).
- Make a plan for early transfer to a specialist unit.
- *Early debridement*. Document a combined plastic and orthopaedic surgery plan.
- Perform joint 1° debridement (plastics/orthopaedics) on a normal working day on a scheduled trauma list within 24h of injury unless immediate surgery is indicated.
- Apply a vacuum dressing or an antibiotic bead pouch at 1° debridement until definitive surgery.
- *Definitive surgery*. Within 72h (do not exceed 7 days).
- Continue antibiotics until sooner of definitive surgery/72h).

Further reading

British Association of Plastic, Reconstructive and Aesthetic Surgeons. Standards available freely online at: https://www.bapras.org.uk

Skin cancer

Key facts

- Basal cell carcinoma (BCC), a neoplasm of the basal cells of the epidermis (rodent ulcer), affects 20–40% of Caucasians. It almost never metastasizes but can invade deeply and may therefore be fatal.
- Squamous cell carcinoma (SCC) is a malignant neoplasm of the keratinizing cells of the epidermis or its appendages, affecting 1 in 2000 Caucasians per year.
- Melanoma is a malignant neoplasm of melanocytes, with a lifetime risk of about 1 in 70 for Caucasians. The incidence has doubled over the past 20y.

Clinical features

- The typical BCC is a skin ulcer with a pearly edge and telangiectasia; however, there may not be any of these features. A persistent, itchy, scaly patch in a sun-exposed area may also be a BCC.
- SCC typically presents as an ulcer with a raised, rolled edge but also may take many forms from scaly patch to keratotic horn.
- Melanoma presents with a change in a pre-existing or new mole (naevus).
- ► Remember: **A**symmetry; **B**order irregularity; **C**olour change or variegated colour; **D**iameter >6mm; **E**levation, itch, or bleeding. All these features are suspicious of malignant change in a skin lesion.

Risk factors

- Fair skin and blue eyes (Fitzpatrick skin types 1 and 2).
- Sun exposure, both in adulthood and childhood, especially sunburn.
- Family history.
- Previous skin cancer.
- Immunosuppression, especially post-organ transplantation.
- Xeroderma pigmentosum.
- Radiotherapy.
- Premalignant lesions: Gorlin syndrome and sebaceous naevus of Jadassohn for BCC; Ferguson–Smith syndrome, Bowen's disease, actinic keratosis, and chronic ulcers for SCC; atypical naevus syndrome, giant congenital naevi, and lentigo maligna for melanoma.
- A variety of chemicals, e.g. arsenic and coal, predisposes to SCC and BCC.

Assessment

- History includes sun exposure, previous skin lesions, drug history, and family history.
- Examine the entire skin and palpate draining lymph nodes.
- Dermatoscopy is used to improve accuracy of clinical diagnosis of melanoma.

Management

- Melanomas and SCCs are managed by skin cancer MDTs.

BCC

- Margins of 3–4mm are suitable for well-defined BCCs, but wider margins are used when margins are unclear and in recurrent or morphoeic tumours.
- Moh's micrographic surgery can be used when margins are not clear clinically. This technique is often practised by dermatologists with patients arriving at plastic surgery centres within 24h for reconstruction. It requires advance planning.
- Other treatment modalities include curettage and cautery, cryotherapy, radiotherapy, fluorouracil cream, imiquimod, and photodynamic therapy.
- 95% of lesions are cured by complete excision; 99% with Moh's surgery. Radiotherapy cures 90%.

SCC

- Lesions are excised with a 4–6mm margin, depending on the site and their diameter; 95% of tumours are cured by this treatment.
- Palpable lymph nodes in the draining basin are investigated by FNA, with lymphadenectomy if positive.
- Prognosis depends on diameter of lesion, depth of invasion, and nerve or vessel invasion on histology.
- Radiotherapy can be used as an adjuvant treatment for metastatic tumours or as 1° treatment if the tumour or patient mean that surgery is not possible.

Melanoma

- All suspicious pigmented lesions are biopsied by excision with a 2mm margin to include subcutaneous fat and sent for histological analysis.
- Surgery aims to cure melanoma. Radiotherapy and chemotherapy are used for palliation only. ▶ Wide local excision margins depend on the depth of invasion (Breslow thickness) of the tumour and are typically 1cm for lesions <1mm thick, 2cm for lesions 1–2mm thick, and 2–3cm for lesions >2mm thick.
- 💣 Sentinel lymph node biopsy may be considered, with lymph node dissection of the neck, axilla, or groin if positive.
- Prognosis depends on Breslow thickness, ulceration of the tumour, and lymph node involvement.

Further reading

British Association of Dermatologists. Guidelines for the management of BCC, SCC, and melanoma are available freely at: 🔗 https://www.bad.org.uk

Aesthetic surgery

Definition

Surgery performed on any part of the body with the main purpose of improving cosmesis without physical functional benefit. Considerable psychological benefit is expected in appropriately selected patients.

- Little aesthetic surgery is performed on the NHS in the UK.
- Patients must demonstrate need by engagement with national pathways that set strict criteria for patient selection and include thorough psychological assessment.
- Privately, a vast array of procedures are available (see Table 17.3).
- Patient selection is key to successful aesthetic surgery. Manage patient expectations and engage with thorough informed consent.

Table 17.3 Commonly available aesthetic surgery procedures

Region	Common procedures
Head and neck	• Brow lift • Correction of eyelid ptosis (blepharoplasty) • Correction of prominent ears (otoplasty) • Prominent nose (rhinoplasty) • Facelift • Cheek implants • Chin projection (genioplasty) • Neck lift
Breast	• Breast reduction • Breast augmentation (➔ Breast augmentation, pp. 742–3) • Breast uplift (mastopexy) • Correction of ♂ breast tissue (gynaecomastia)
Upper limb	• Correction of 'bingo wings' (brachiplasty) • Hand rejuvenation
Trunk	Abdominoplasty
Groin	• Thigh lift • Vaginal rejuvenation (vaginoplasty) • Penile rejuvenation (phalloplasty) • Buttock implants
Lower limb	Calf implants
General and non-surgical	• Liposuction (➔ Liposuction, p. 740) • Fat transfer • Hair transplant • Chemical peels • Dermal fillers • Botulinum toxin • Laser (➔ Lasers, pp. 738–9)

Lasers

Definition
Light **A**mplification by the **S**timulated **E**mission of **R**adiation

Principle

- Electrons are created at a cathode and accelerated towards an anode through a lasing medium.
- Passage of the electrons through the lasing medium excites the outer shell electrons in it, stimulating the emission of coherent and collaminated photons of a specific wavelength that can be precisely focused on tissues (Bremsstrahlung radiation).
- The wavelength of the emitted photons determines the target chromophore in the tissue (see Table 17.4).
- Chromophores can be found in blood vessels (oxyhaemoglobin), hair cells (melanin), pigmented lesions (melanin), skin (water), and ink (tattoo) pigments.
- The mechanism of action is by localized tissue destruction through heating. This may be coagulation, vaporization, or selective photothermolysis.[1] Most modern lasers simultaneously treat and cool.
- Intense pulsed light (IPL) is not a true laser, as the light is not coherent, but has use for removal of unwanted hair and some pigmented lesions.

User settings

- Wavelength (defined by lasing medium) (see Table 17.4).
- Beam energy (power). Measure of rate of energy (in joules) per unit time (J/s), measured in watts.
- Pulse duration, also called pulse width, measured in fractions of a second.
- Spot size, measured in cm^2. Larger spot sizes penetrate deeper into tissues.

Table 17.4 Laser types and uses

Lasing medium	Wavelength (nm)	Target chromophore	Use
KTP	532	Oxyhaemoglobin	Vascular lesions
Pulsed dye	585		
Ruby	694	Melanin	Pigmented lesions, hair removal
Alexandrite	755		
Diode	800		
IPL	N/A		
ND:YAG	1064	Oxyhaemoglobin, melanin	Vascular lesions, hair removal
Er:YAG	2940	Water	Skin resurfacing
CO_2	10 600		

CO_2, carbon dioxide; Er, erbium; IPL, intense pulsed light; KTP, potassium titanyl phosphate; ND, neodymium; YAG, yttrium aluminium garnet.

Calculated settings

- The power density is calculated according to the energy delivered per unit area of tissue (W/cm^2).
- The fluence combines power density with total delivered energy (power density; J/cm^2).

Additional functions

- Modern lasers can perform pulse stacking, q-switching, and core treatments that have specific indications.
- q-switched lasers emit photons at a higher power because two full, rather than partial, mirrors are used. They have application in tattoo removal.

Treatment

- Patient selection and management of expectations are key to successful laser treatment.
- The areas and scope of treatment are agreed prior to therapy.
- Following informed consent, the patient and operator wear appropriate protective equipment (protective eyewear and goggles/suction).
- Treatment is often delivered in the outpatient clinic.
- The patient is provided with written post-operative instructions, including wound care advice, and plans are made for follow-up and subsequent treatment as appropriate.

Complications

- *Localized*. Hyperpigmentation, hypopigmentation, erythema, blistering, milia formation, scarring, infection, delayed wound healing, contact dermatitis to topical post-operative medications.
- *Systemic*. Allergic reactions following treatment of tattoo pigment; rhabdomyolysis has been reported as a complication of laser treatment.
- *General*. Ocular damage from inadequate eyewear, operator infection from vaporized lesions (e.g. HPV from CO_2 treatment of cutaneous warts.

Reference

1 Anderson RR, Parrish JA (1983). Selective photothermolysis: precise microsurgery by selective absorption of pulsed radiation. *Science* **220**: 524–7.

Liposuction

Purpose

To reduce the volume of subcutaneous adipose tissue and improve body contour ± tighten skin by mechanical trauma and suction. Removed fat cells will not come back, but the remaining fat cells can still hypertrophy

Indications

- ❗ Cosmetic body contouring (NOT for weight loss!).
- Also indicated to contour flaps, reduce lipomas, harvest fat for transfer, or for ASC procurement; to treat gynaecomastia, lipoedema, and extravasation injuries.

Treatment

- *Infiltration (wetting solution)*. Usually crystalloid, with 1:500 000–1:1 000 000 adrenaline and LA (lidocaine/levobupivacaine). More infiltration reduces post-operative bleeding and improves analgesia. Large volumes can disturb fluid balance. Dry: no infiltration; wet: 100–300mL/treated area; super wet: 1mL/mL of expected aspirate; tumescent: 2–3mL/mL of expected aspirate.
- *Cannula*. Blunt with single or multiple ostium, power-assisted available to achieve reduced operative effort of liposuction.
- *Suction*. Manual or wall suction, can be ultrasound- or laser-assisted.
- Level of aspiration. Superficial or deep (always above deep fascia).

Procedure

- Infiltrate area with wetting solution; make multiple stab incisions to facilitate cannula entry.
- Cannula is passed backwards and forwards in centrifugal rays in multiple levels from stab incision to evenly remove fat.
- Always monitor where the tip of the cannula is in tissue to avoid inadvertent injury to deep structures.
- Keep ostium pointing down to prevent skin dimpling.
- Techniques include pre-tunnelling (creates passages without suction at start), cross-tunnelling (pattern to reduce the risk of creation of contour defects), and feathering (blend treated to non-treated areas).

Post-operative care

- Monitor fluid balance.
- Check Hb and U&Es for 24h if >2L removed.
- Wear compression garments for 6 weeks to 3 months.

Complications

- *Early*. Shock from fluid loss, haematoma, seroma, swelling, thermal injury, friction burns, damage to deep structures, DVT/PE, fat embolus, death.
- *Late*. Contour irregularity, paraesthesiae, discoloration, lax skin.

Breast augmentation

Purpose
To enhance breast size by stretching or filling the skin envelope that lies anterior to the chest wall

Indications

Performed for asymmetry, hypoplasia, involution following childbirth or menopause, and psychological reasons, e.g. self-consciousness or problems with sexual relationships.

❗ Consider the need for a mastopexy (breast uplift) when assessing patients for breast augmentation. Not every patient will be suitable for augmentation alone and an additional mastopexy often has significant financial implications for the patient.

Operative considerations

Incision

- *Inframammary fold*. Good visualization of implant pocket; visible scar.
- *Periareolar*. Semicircular incision at the border of the areolus. Scar fades well, but access is limited. More likely to alter nipple sensation.
- *Transaxillary*. Eliminates scars on breast. Limited access improved by using endoscope. Better for subpectoral implants.
- *Transumbilical*. Only used for saline-filled implants, inserted along a tunnel created superficial to rectus sheath. Endoscope confirms position of implant pocket. Implant inflated once in position.

Position of implant

- *Subglandular*. Under the normal breast.
- *Dual plane*. Under pectoralis major at upper pole and breast tissue at lower pole.[1] Pectoralis major can be 'window-shaded' to provide more or less cover of implant. Risks implant animation but provides reduced rates of capsular contracture and good upper pole coverage.
- *Submuscular*. Good for breast reconstruction. Under pectoralis major superiorly, rectus abdominis fascia inferiorly, and serratus anterior fascia laterally.

Type of implant

- *Size*. Depends on patient's choice. It is virtually impossible to specify a specific cup size preoperatively. A good indication of volume can be achieved by performing the 'rice test' in which the patient fills a bag in their desired bra size with rice whilst wearing it. The rice volume is measured and gives an approximate volume of desired implant size required.
- *Shape*. Round or anatomical. The implant projection (chest wall–nipple), height on chest wall, and width (base) on chest wall can be specified to suit implant pocket.
- *Shell*. Can be smooth or textured. Textured implants have lower rates of capsular contracture, but highly textured implants have been associated with anaplastic large cell lymphoma (ALCL) (➲ 'Implant safety' below).

- *Implant filling*. Saline or silicone, or combination of both (Becker). Saline and Becker implants can be expanding and volume can be fine-tuned post-operatively by administering or removing saline from the implant through an integrated or remote port. These implants may be definitive or temporary pending replacement with a definitive, fixed-volume implant.

Post-operative care

- Usually an overnight stay procedure (longer if drains are used).
- A supportive bra is worn and heavy lifting avoided for 4–6 weeks.

Complications

- *Early*. Haematoma, infection, nerve injury (altering sensation to the nipple), incorrect position of implant.
- *Late*. Capsular contracture, rupture, or deflation; silicone gel bleed.
- Implants have a limited lifespan of up to about 20y. The likelihood is that they will need to be removed or replaced at some time. Patients can usually breastfeed after augmentation. Patients are warned that mammography is technically more difficult, requiring different views, but there is no ↑ risk of breast cancer.

Implant safety

- In 1998, a UK Department of Health Independent Review Group found no link between silicone breast implants and connective tissue or autoimmune diseases.
- This finding was repeated in 1999 by the Institute of Medicine of the US National Academy of Sciences.
- Silicone is an almost universal product and is found at higher concentrations in cow's milk and infant formula than in the breast milk of breastfeeding mothers with implants.
- Breast implant associated-anaplastic large cell lymphoma (BIA-ALCL) has recently been linked to patients with breast implants.
- BIA-ALCL is extremely rare, with an incidence of between 1 in 50 000 and 1 in 300 000. It usually presents >1y post-operatively with a rapidly developing unilateral seroma around the implant. There is an association with highly textured implants. Treatment is by implant removal and capsulectomy.
- BAPRAS has issued guidance that all patients undergoing breast augmentation should be given verbal and written advice about the potential risk of developing BIA-ALCL.[2]
- This has led to the development of a breast implant register.

References

1 Tebbetts JB (2001). Dual plane breast augmentation: optimizing implant-soft-tissue relationships in a wide range of breast types. *Plast Reconst Surg* **107**: 1255–72.

2 British Association of Plastic, Reconstructive and Aesthetic Surgeons. Statement on BIA-ALCL available freely online at: https://www.bapras.org.uk

Chapter 18

Cardiothoracic surgery

Basics

Key revision points: coronary artery anatomy

(See Fig. 18.1.)

- The left main stem (LMS) arises from the ostium of the left sinus of Valsalva, travels between the pulmonary trunk anteriorly and the left atrial (LA) appendage to the left AV groove, dividing after 1–2cm into the left anterior descending (LAD) artery, the circumflex (Cx), and occasionally a third artery (the intermediate). As it provides almost the entire blood supply to the LV, occlusion can be fatal; severe left main disease is known as 'the widow maker'.
- The LAD runs down the anterior interventricular groove to the apex of the heart, usually extending round the apex to the posterior interventricular groove. A variable number of diagonals are given off over the anterior surface of the LV; small branches supply the anterior surface of the RV, and superior septals are given off perpendicularly to supply the anterior two-thirds of the interventricular septum. Occlusions of the LAD result in anterior MI.
- The circumflex originates at 90° from the LMS and runs medially to the LA appendage for 2–3cm, continuing in the posterior left AV groove to the crux of the heart. In left dominant hearts (5–10%), the Cx turns 90° into the posterior interventricular groove to form the posterior descending artery (PDA). In 85–90% of hearts, the PDA arises from the right coronary artery (RCA) (right dominant). About 5% of hearts are co-dominant.
- A variable number of obtuse marginals (OMs) arise from the Cx to supply the posterior LV. The first branch of the Cx is the AV nodal artery, in which 45% course round the LA near the AV groove.
- The RCA arises from an ostium in the right sinus of Valsalva, gives off an infundibular branch and then a branch to the sinoatrial (SA) node, and runs immediately into the deep right AV groove where it gives off RV branches to the anterior RV wall. Occlusions of the RCA result in inferior infarcts and bradycardia. The acute marginal is a large branch which crosses the acute margin of the heart. In right dominant hearts, the RCA reaches the crux of the heart where it turns 90° to form the PDA, which runs in the posterior interventricular groove. Inferior septals, which supply the inferior third of the interventricular septum, arise at 90° from the PDA. The AV nodal artery is given off by the RCA in 55% at the crux.

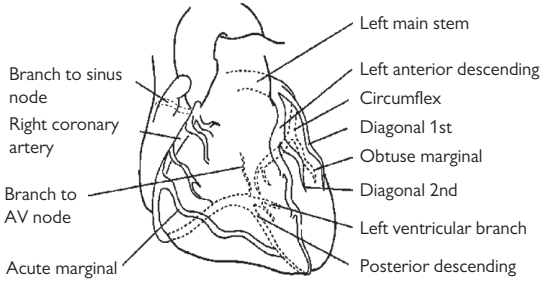

Fig. 18.1 Coronary artery anatomy.

Principles of cardiac surgery

Key facts

- The majority of procedures are CABG operations, followed by aortic valve replacements, mitral valve (MV) repair and replacements, and aortic surgeries.
- Many patients are elderly with multiple comorbidities, but most of the patients should be out of ICU within a day or two and ready to go home in a week.

Preoperative preparation

Meticulous preoperative work-up is essential. All investigations must be checked. Sometimes even small abnormalities can have significant harmful consequences to patient outcomes.

Full history

- Quantify symptoms—New York Heart Association (NYHA) classification for breathlessness/Canadian Cardiovascular Society angina grading.
- Ask about risk factors: family history, smoking, hypercholesterolaemia, HTN, alcohol consumption, previous MI or stroke.
- Other comorbidities (especially COPD, renal failure, PVD, DM). Ask about previous heart surgery, varicose vein surgery, and previous DVT.
- Drugs (aspirin, clopidogrel, and warfarin normally stopped 5 days preoperatively to reduce bleeding) and allergies.
- Valve patients should have had recent dental check.

Full examination

- Feel for pulse and rhythm. Auscultate for heart sounds and murmurs, lungs, and carotid arteries (presence of bruits). Look for signs of heart failure. Active infection is a relative contraindication to valve replacement. Look for surgical scars and deformities.
- Look at conduit—any evidence of varicose veins? Perform Allen's test for radial artery conduit.

Investigations

- All patients should have FBC, U&Es, LFTs, clotting screen, group and save ± cross-match, depending on local policies and type of surgery.
- ECG, CXR, echocardiography.
- Coronary angiograms. All patients undergoing coronary artery surgery and patients aged >40y.
- Carotid duplex in any patient with a history of stroke, TIA, or carotid bruits, or a history of PVD; some centres perform these routinely in patients aged >70y.
- Lung function test. For patients with respiratory diseases, e.g. COPD, pulmonary fibrosis, etc., or with recent smoking history.
- Diabetic patients will require special attention to their blood sugars and tailored treatment for optimal blood sugar control.

Cardiopulmonary bypass

The cardiopulmonary bypass (CPB) machine takes over the function of the heart and lungs by draining the patient's blood circulation from the venous system (IVC, SVC, RA) into the CPB machine where the blood is oxygenated and returned back into the patient's arterial system (usually the ascending aorta). This allows the surgeon to stop the heart and perform surgery on the heart (CABG, valve surgery, etc.) or great vessels (ascending and arch aortic dissection and aneurysm surgery, resection of some tumours invading the great vessels, e.g. renal cell).

This involves:

- Heparinizing the patient so that blood does not clot in the CPB circuit—target activated clotting time (ACT) >480.
- Cannulation:

1. Securing an aortic cannula with a concentric purse string(s) in the ascending aorta.
2. Securing a two-stage venous cannulation in the RA or bicaval cannulation in the SVC and IVC for procedures such as MV/tricuspid valve surgery.

- Connecting both cannulae to the bypass circuit.
- The venous return from the body is siphoned passively into the bypass circuit and then the venous blood is oxygenated and filtered, and can be cooled or warmed, and is pumped back to the patient via the aortic cannula.
- At the end of bypass, heparin is reversed with protamine.

Pathophysiology of CPB

CPB is unavoidable for many operations. It has a major impact on nearly every organ system. Problems associated with bypass include:

- Activation of coagulation and complement cascades.
- Consumption of platelets and clotting factors, causing coagulopathy.
- Microemboli and atherosclerotic emboli from aortic cannulation, which can cause stroke and limb or other end-organ ischaemia.
- ↑ capillary permeability.
- Renal, pulmonary, hepatic, and pancreatic dysfunction.
- Complications of CPB include stroke (atheromatous emboli, hypoperfusion, air, microemboli), systemic inflammatory response syndrome (SIRS), and renal and pulmonary dysfunction.

Myocardial protection

In order to perform cardiac operations, it is necessary to stop (arrest) the heart. This may be achieved through a number of techniques: cardioplegia, fibrillation, and hypothermia. Cardioplegia arrests the heart and protects it from myocardial ischaemia.

Cardioplegia

- Cardioplegia is a K+-rich solution.
- It can be based on blood or crystalloid (blood delivers O_2 better).
- It can be warm or cold (cold may better reduce ischaemic injury).
- It is delivered into the coronary arteries, either anterogradely by inserting a cannula into the aortic root which is clamped distal to the cardioplegia cannula or retrogradely via the coronary sinus vein.
- It can be given continuously or intermittently, every 20min or so.

Post-operative management

Management of five common post-operative emergencies is outlined on ➔ Common cardiac emergencies , pp. 760–1. Most patients are well enough to be extubated within 6h, leave ITU within 24h, and go home within 5 days.

First 6h

- Myocardial function may deteriorate due to ischaemia–reperfusion injury.
- Inotropic support and pacing may be required.
- Patient should be fit for extubation by 4–6h post-operatively.
- Ensure patient has good BP (usually aim MAP >65mmHg).

Days 1–2

- Inotropes and pacing weaned, invasive monitoring lines removed.
- Chest drains removal as per local protocol (usually 2–3h of zero drainage and no air leak).
- Catheter and any epidural removed, patient mobilized.
- PCA morphine reduced to PO analgesia.
- β-blockers should be reinstituted or commenced unless contraindicated, e.g. heart block.
- CABG patients should be on dual antiplatelet (if no contraindications).
- Anticoagulation commenced for patients with mechanical valves.

Days 3–5

- Temporary pacing removed if ECG satisfactory.
- Day 3 CXR to exclude significant effusions or pneumothorax.
- Valve patients should undergo echocardiography.
- Physiotherapists assess exercise tolerance.
- Back to baseline weight, medications stabilized, ready for discharge.

Coronary artery disease

Definition

Narrowing of the coronary arteries caused by atherosclerosis (➔ Atherosclerosis, p. 192).

Incidence

Coronary artery disease (CAD) is the commonest cardiovascular disease. There are >100 000 hospital admissions in the UK due to myocardial ischaemia. In 2015, there were 16 166 cases of isolated CABG (both elective and urgent) in the UK.

Aetiology

- Age, ♂ sex, smoking, ↑ BP, DM, hyperlipidaemia, obesity, family history, stress.

Pathology

Stenoses tend to progress in severity and distribution. Rate of progression is variable and regression of lesions has been observed.

- Narrowings of 50% of cross-sectional area limit coronary flow reserve (the increase in blood flow that occurs to meet ↑ O_2 demand).
- Coronary blood flow at rest is reduced by narrowings of 90%.
- Acute MI is caused by acute total or subtotal vessel thrombotic occlusion. Patients with proximal LAD lesions are particularly at risk (➔ Basics, pp. 746–8 for a description of anatomic territories).

Clinical features

- Angina and/or dyspnoea. Severity is classified using the NYHA score. Dyspnoea implies CCF.
- Class I. Symptoms only with prolonged or strenuous exertion.
- Class II. Symptoms causing slight limitation of ordinary activity.
- Class III. Symptoms with marked limitation of ordinary activity.
- Class IV. Angina occurring even with mild activity or at rest.

Diagnosis

- History and examination.
- ECG may show evidence of old infarcts.
- Exercise treadmill has 97% specificity for exertional angina.
- Coronary angiography is diagnostic and obligatory for planning surgery.
- Myocardial perfusion studies such as thallium scans and MRI are also useful for assessment of myocardial viability.
- CT coronary angiography is increasingly used to screen lower-risk patients

Management

The options are optimal medical therapy and PCI, and surgical revascularization (CABG).

Many large RCTs have been carried out to compare PCI versus CABG and to decide which groups of patients benefit most from surgery.

Indications for surgery

Current guidelines recommend:

- Patients with >50% LMS stenosis.
- Patients with >70% proximal LAD stenosis.
- Patients with >70% disease in all three vessels ('three-vessel disease').
- Three-vessel disease with LVEF >50%.
- One- or two-vessel disease (including proximal LAD stenosis) and LVEF <50%.
- Patients with significant CAD having cardiac surgery for other reasons, e.g. valve replacement.

Coronary artery bypass surgery

- Median sternotomy.
- Harvesting conduits:
  - Long saphenous vein.
  - Left internal thoracic (LITA), a.k.a. mammary artery (LIMA). The LITA is a branch of the left subclavian artery and runs down the inside of the rib cage 2cm lateral to the sternum.
  - Radial artery.
- Conduit is anastomosed to the coronary artery beyond the lesion and then to the ascending aorta. The LITA is usually anastomosed to the LAD, as this has been shown to have survival benefit, compared with vein graft to LAD, in a landmark paper by the Cleveland Clinic in 1986. LITA has been shown to have excellent long-term patency (>90% over 10y). Coronary artery bypass is mostly performed on-pump (with the use of a CPB machine) on the still heart. It can also be performed on the beating heart ('off-pump' without CPB machine).

Complications

Complications (➔ Common cardiac emergencies, pp. 760–1) are more likely with advanced age, poor LV function, renal failure, and COPD. Risk of mortality is calculated using EuroSCORE II.

- Death, 0–1% in low-risk patients.
- Stroke, 1–2% in low-risk patients.
- Re-sternotomy for bleeding or tamponade, 5%.
- Chest infection, AF, wound infection, renal failure.

Valvular heart disease

Mitral regurgitation

- Incidence. MR is the second commonest indication for valvular surgery in Europe.
- Aetiology. MV prolapse (congenital or rupture of chordae/papillary muscles), degenerative, rheumatic disease, endocarditis, connective tissue disorders.
- Clinical features. Acute MR presents with signs of CCF. Chronic MR causes exertional dyspnoea and orthopnoea. Displaced apex beat, soft S1, pansystolic murmur (loudest at apex, radiating to axilla). AF in 80%.
- Diagnosis. CXR shows cardiomegaly. TTE for definitive diagnosis.
- Indications for surgery. Severe MR—symptomatic patients with left ventricular ejection fraction (LVEF) >30%. Asymptomatic patient with LV dysfunction.
- Treatment. MV repair can be performed in the majority of cases of degenerative valve. MV replacement is often required in cases of rheumatic disease, infective endocarditis, and papillary muscle rupture.
- Prognosis. Mortality of untreated severe MR is 6% per year in patients aged 50 and above. Operative mortality is <1% for low-risk cases.

Mitral stenosis

- Incidence. Commoner in Asia and Africa; rare in the Western countries.
- Aetiology. Rheumatic heart disease, degenerative.
- Clinical features. Dyspnoea, bronchitis, haemoptysis, AF, left parasternal heave, tapping apex beat, loud S1, rumbling mid-diastolic murmur at apex.
- Diagnosis. CXR shows splaying of carina (enlarged LA). Echo diagnostic.
- Indications for surgery. Moderate to severe mitral stenosis or MV area <1.5cm^2.
- Treatment. MV replacement.
- Prognosis. Poor once symptoms of heart failure present.

Aortic stenosis

- *Incidence.* The commonest indication for valve surgery. Prevalence increases with age (1.3% in 60–69y old; 9.8% 80–89y old).
- *Aetiology.* Calcific degeneration, bicuspid valve, rheumatic disease.
- *Clinical features.* Triad of angina, syncope, dyspnoea. Sudden death. Slow-rising and low-volume pulse, heaving apex beat, reversed splitting S2, ejection systolic murmur loudest in aortic area radiating to carotids.
- *Diagnosis.* ECG shows LV hypertrophy, TTE diagnostic.
- *Indications for surgery.* Symptomatic aortic stenosis or asymptomatic severe aortic stenosis. Transcatheter aortic valve implantation (TAVI) in patients deemed unfit for surgery.
- *Prognosis.* Without surgery, 50% of patients with angina are dead in 5y, 50% with syncope in 3y, and 50% with dyspnoea in 2y. The UK mortality for isolated aortic valve replacement is 1–2%.

Aortic regurgitation

- Incidence/prevalence. According to the Framingham study, the prevalence of AR is 4.9% (moderate and severe AR around 0.5%).
- Aetiology. Rheumatoid, endocarditis, aortic dissection, Marfan's and other connective tissue disorders, calcific degeneration, trauma.
- Clinical features. Acute AR (endocarditis) presents with signs of left ventricular failure (LVF). Chronic AR often asymptomatic. Later, orthopnoea, fatigue, dyspnoea. Signs of wide pulse pressure, collapsing water hammer pulse, Quincke's sign (nail bed pulsation), Corrigan's sign (visible neck pulsation), De Musset's sign (head nodding), Durozier's sign (femoral diastolic murmur), hyperdynamic displaced apex beat, early diastolic murmur, and Austin Flint mid-diastolic murmur due to regurgitant stream hitting anterior MV cusp.
- Diagnosis. CXR shows cardiomegaly. TTE diagnostic.
- Indications for surgery. Severe AR with symptoms or asymptomatic severe AR with LVEF ≥50%/left ventricular end-diastolic dimension (LVEDD) >70mm/left ventricular end-systolic dimension (LVESD) >50mm.
- Treatment. Aortic valve replacement/aortic valve-sparing procedures for aortic root dilatation.
- Prognosis. Acute AR has poor prognosis. Chronic AR follows a more gradual course. The patient might have a long asymptomatic period, but once symptomatic, may deteriorate quickly.

Options for valve surgery

MV repair (annuloplasty and valvuloplasty)

Results of repair of the aortic valve are unpredictable, so it is usually replaced. The MV is often repaired, with good results.

Mechanical valves, e.g. St Jude mechanical, Carbomedics, ON-X

These are made of pyrolytic carbon. They are mostly bileaflet. Ball-and-cage type, like the Starr–Edwards, is no longer used.

- Advantages. Excellent durability.
- Disadvantages. Thromboembolic, so patient must be warfarinized; patients with bleeding diatheses, women of childbearing age, and patients with very physically active lifestyle or profession may not be suitable for long-term warfarinization.

Tissue valves (xenografts), e.g. Perimount, Hancock

These are made of pig valves (Hancock) or cow pericardium (Perimount), usually suspended on a metal frame covered by a cloth sewing ring.

- Advantages. Patient does not need to be warfarinized.
- Disadvantages. Tissue valves last for 10–15y in the aortic position and 6–10y in the mitral position, depending on the age of the patient; younger patients (<65–70y) will often need a second operation.

Complications

Complications are more likely with advanced age, poor LV function, renal failure, COPD, pulmonary HTN, and additional CABG (➲ Common cardiac emergencies, pp. 760–1).

- Death, 0–3% in low-risk patients.
- Stroke, 5–10% (debris from removing calcified valve, cannulating aorta).
- Re-sternotomy for bleeding or tamponade, 5%.
- Chest infection, AF, complete heart block requiring permanent pacemaker insertion, wound infection, renal failure.
- Prosthetic endocarditis, failure, thrombosis, paravalvular leak.

Key revision points—anatomy of heart valves

- Aortic valve. Tricuspid valve, sitting within bulb of aortic root. Three dilatations called sinuses of Valsalva. Left coronary sinus gives rise to LMS, and right gives rise to RCA. Third known as non-coronary sinus. AV node lies between right and non-coronary cusp. Annulus (where leaflets attach to aorta) is coronal-shaped.
- Mitral valve. Bileaflet valve, lying between LA and LV. Anterior leaflet smaller than posterior leaflet. Leaflets held in place by chordae which attach to two papillary muscles of the LV. Annulus is oval-shaped.

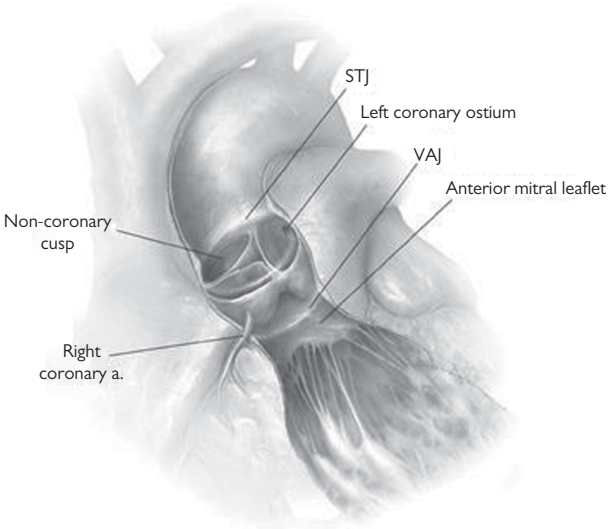

Fig. 18.2 Anatomy of aortic valve. STJ, sinotubular junction; VAJ, ventriculo-aortic junction.

Adapted from Boodhwani M and El Khoury G (2009) Aortic Valve Repair, Operative Techniques in Thoracic and Cardiovascular Surgery 14(4):266–280 with permission from Elsevier.

Cardiothoracic ICU

Commonly used terminology

(See Table 18.1.)

- Instead of referring to systolic and diastolic BP, arterial pressure is usually described using a single figure—the mean arterial pressure (MAP), which is calculated by adding a third of the difference between diastolic and systolic BPs to the diastolic pressure, e.g. the MAP of a patient with a BP of 120/60mmHg is 80mmHg.
- The MAP on its own does not adequately describe cardiac function; a number of other parameters are frequently used.
  - *Cardiac output* (CO). The volume of blood ejected by the heart per minute.
  - *Stroke volume*. The volume of blood ejected by the heart per beat.

Table 18.1 Key haemodynamic formulae and normal values

Cardiac output = SV × HR	
Cardiac index = CO/BSA	
Stroke volume index = SV/BSA	
Mean arterial pressure = DP + (SP – DP)/3	
Systemic vascular resistance = [(MAP – CVP)/CO] × 80	
Systemic vascular resistance index = SVR/BSA	
Pulmonary vascular resistance = [(PAP – PAWP)/CO) × 80	
Pulmonary vascular resistance index = PVR/BSA	
	Normal value
Cardiac output (CO)	4.5–8L/min
Stroke volume (SV)	60–100mL
Body surface area (BSA)	2–2.2m^2
Cardiac index (CI)	2.0–4.0L/min/m^2
Stroke volume index (SVI)	33–47mL/beat/mm^2
Mean arterial pressure (MAP)	70–100mmHg
Diastolic pressure (DP)	60–80mmHg
Systolic pressure (SP)	110–150mmHg
Systemic vascular resistance (SVR)	800–1200dyne-s/cm^5
Central venous pressure (CVP)	6–12mmHg
Systemic vascular resistance index (SVRI)	1970–2390dyne-s/cm^5/m^2
Pulmonary vascular resistance (PVR)	50–250dyne-s/cm^5
Pulmonary artery systolic pressure (PASP)	15–30mmHg
Pulmonary artery wedge pressure (PAWP)	8–14mmHg
Pulmonary vascular resistance index (PVRI)	20–125dyne-s/cm^5/m^2

- CO equals: heart rate × stroke volume.
- *Cardiac index*. This is simply the CO adjusted to take into account the size of the patient and is a more accurate reflection of cardiac function.

Low cardiac output

Much of the initial care after cardiac surgery is aimed at preventing, recognizing, and treating low CO states, as these can lead to organ failure, contribute to sepsis, and cause death, even in 'straightforward' patients. Low CO can be defined as a cardiac index of <2.2mL/min or evidence of end-organ hypoperfusion (e.g. lactic acidosis, oliguria, low mixed venous O_2 saturations).

- Common causes of low CO include:
  - Bleeding (or other causes of hypovolaemia, e.g. polyuria).
  - Tamponade.
  - Arrhythmias.
  - Acidosis, hypoxia.
  - Preoperative cardiomyopathy.
  - Ischaemia and stunning (myocardial recovery from surgery).
- Hypotension can be due to low CO, but even a patient with a high CO could be hypotensive if they were very vasodilated (most commonly due to SIRS or sepsis).
- Sometimes a number of problems may be going on simultaneously and it is easy to miss important problems, so a systematic approach to assessing post-operative cardiac surgery patients is vital.

General assessment of cardiac surgery patients

- Except in emergencies, usually you have time to evaluate every system.
- Review the history. What was the ventricular function preoperatively? What other comorbidities? What operation was done—any problems?
- Cardiovascular.
  - Look at the HR and rhythm, and check the ECG for evidence of ischaemia (ST segment changes), comparing with preoperative ECG.
  - Look at the MAP and CVP. A high CVP is always concerning, suggesting tamponade, ventricular failure, or respiratory problems.
  - Look at the CO, lactate, mixed venous O_2 saturations, and feel the patient's extremities; a warm patient with good peripheral pulses cannot have a low CO. What is the SVR?
  - What inotropes, vasoconstrictors, and antihypertensives are on?
- Respiratory. Is the patient breathing spontaneously or ventilated? Look at the O_2 saturations and a recent blood gas to check the PaO_2, $PaCO_2$, and pH. What does the CXR look like?
- Renal. How much urine is the patient making (ideally >1mL/kg/h)? What is the patient's fluid balance (if it is negative or low, the patient may be hypovolaemic; if it is very high, fluid overload is a concern). Check electrolytes; abnormalities can cause arrhythmias.
- Bleeding. Should be <100mls/h in the chest tubes, with steady fall in the rate. Look at wound sites (remember groin + leg). Is the haematocrit dropping? Check coagulation.
- Assess mental status and focal neurology, analgesia.

Common cardiac emergencies

Poor gases

(➲ Acid base balance, pp. 118–19.)

►► If O_2 sats <85% and falling, get immediate help.

- Increase FiO_2 to 100%; check the pulse oximeter.
- Look at expansion; auscultate the chest, and check PaO_2.

►► If you suspect tension pneumothorax, treat immediately (➲ Tension pneumothorax p. 586 also ➲ Chest drain (intercostal drain/pleural drain) insertion pp. 262–3).

- Suction the endotracheal (ET) tube; check that the patient is not biting on it.
- Check that the drain tubing is patent and drains are on suction.
- Treat bronchospasm with salbutamol 5mg nebulizer.
- Disconnect from the ventilator and hand-ventilate the patient.
- Get a CXR—look for pneumothorax, haemothorax, atelectasis, ET tube position, and lobar collapse.

Poor urine output

(➲ Renal complications, pp. 136–8.)

- Check that the Foley catheter is patent.
- If the patient is hypotensive, treat this first.
- Give a fluid challenge of IV fluids to raise the CVP to 14mmHg.
- If low urine output with rising lactate, this may suggest organ hypoperfusion. Always keep cardiac tamponade in mind.
- If not hypotensive and CVP >14mmHg, give 20mg furosemide IV.

Atrial fibrillation

(See Fig. 18.2.)

- Give 10–20mmol K^+ via central line to get serum K+ 4.5–5.0mmol/L.
- Give empirical 20mmol Mg^{2+} via central line if none given post-op.
- Give 300mg amiodarone IV over 1h in patients with good LV function, followed by 900mg amiodarone IV over 23h.
- In patient with poor LV function, give digoxin in 125-microgram increments IV every 20min until rate control is obtained, up to a maximum of 1500 micrograms in 24h.

Synchronized DC cardioversion for unstable patients (➲ Defibrillation and cardioversion, pp. 268–9).

Bleeding

(➲ Bleeding and coagulation, pp. 232–4.)

- Get immediate help if bleeding is >400mL in 30min.
- Give IV fluids to get CVP 10–14mmHg and systolic BP 80–100mmHg.
- Order further 4U of blood, 2U FFP, and two pools of platelets.
- Send clotting and FBC; request a CXR.
- Transfuse to achieve Hb >8.0g/dL, platelets >100 × 10^9/L, and APTT <40.
- Check ACT—if raised, give further protamine. Consider tranexamic acid.
- Emergency re-exploration is indicated for excessive bleeding.

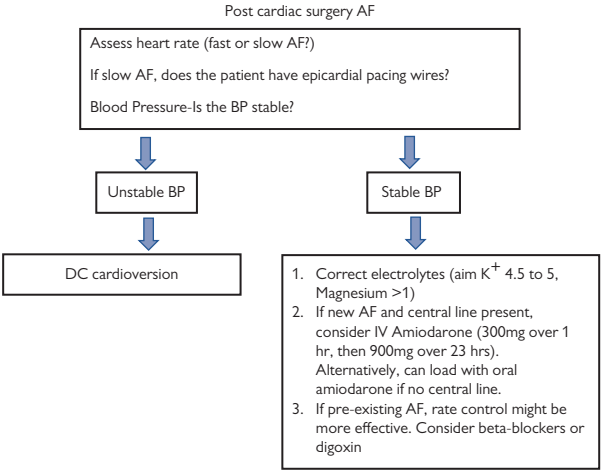

Fig. 18.2 Post-cardiac surgery AF flow chart.

Profound hypotension

(See Fig. 18.3.)

- Get immediate help.
- Quickly assess pulse, rhythm, rate, CVP, O_2 sats, and bleeding.
- Defibrillate VF or pulseless VT; treat AF as above.
- Treat bradycardia with atropine 0.3mg IV or pace.
- Give IV fluids to raise CVP to 12–16mmHg; place bed head down.
- If suspect cardiac tamponade (➔ Cardiac tamponade, p. 587), prepare for re-sternotomy.
- If patient warm and vasodilated, draw up 10 micrograms of metaraminol into 10mL of saline and give 1mL through a central line, and flush.
- If patient still profoundly hypotensive, give 1mL of 1:10 000 adrenaline. However, by this point, cardiac intensivists and cardiac surgeons should be involved. Some centres advise that adrenaline can only be given at the discretion of consultant cardiac intensivists or consultant cardiac surgeons.

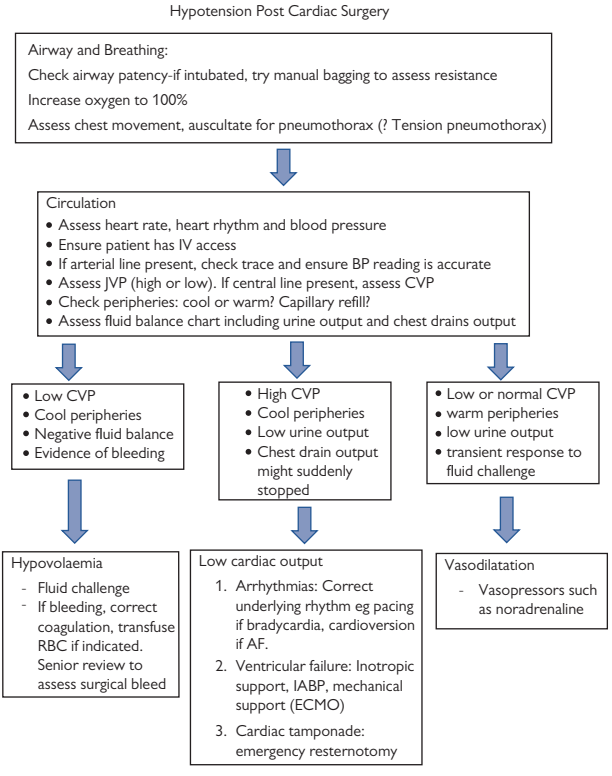

Fig. 18.3 Hypotension assessment and management flow chart.

Lung cancer

Key facts

- Commonest cause of death from cancer in the UK (approximately one-fifth of all cancer deaths).

Risk factors

- Cigarette smoking. Strongly positive association with cigarette smoking (polycyclic aromatic hydrocarbons plus nicotine-related carcinogens).
- Radon exposure.
- Occupational factors. Asbestos, polycyclic aromatic hydrocarbons, arsenic, nickel, silica, coal tar, aluminium production, coal gasification, paints, chromium compounds, bis chloromethyl ether.
- Genetic predisposition and ♂ sex.

Pathology

Lung carcinoma is classified as non-small cell or small cell.

Non-small cell lung cancer (NSCLC)

Squamous cell carcinoma

- 20–30% of all lung cancers.
- Commonest histological type in Europe.
- Two-thirds arise in large airways as an endobronchial mass.
- Slow-growing, late to metastasize.

Adenocarcinomas

- 30–50% of lung cancers. Incidence increasing.
- Usually peripheral tumours (approximately three-quarters).
- Moderate growth with early metastasis.

Large cell undifferentiated carcinoma

- 15% of all lung cancers.
- Peripheral location commoner than central.
- Two subtypes: clear cell and giant cell. Giant cell tumours are uncommon (<1% of lung cancers). Very poor prognosis.

Histological subtypes

- Bronchoalveolar tumours. Highly differentiated adenocarcinoma; 2.5% all lung cancers.
- Adenosquamous carcinomas.

Small cell lung cancer

Neuroendocrine tumours. ~10–15% of lung cancers. Aggressive tumours, not usually amenable to surgical resection. Three subtypes: pure small cell (90%); mixed small and large cell; combined small cell with areas of squamous or glandular differentiation.

Clinical features

- Proximal tumours tend to produce symptoms of major airway obstruction and irritation (haemoptysis, dyspnoea, cough, wheezing, stridor, hoarseness, Horner's syndrome, SVC obstruction, post-obstructive pneumonia, pleural effusion, Pancoast's syndrome).

- Peripheral tumours are often asymptomatic or present with signs and symptoms of pleural or chest wall invasion or pleural effusion (pleuritic chest wall pain, progressive dyspnoea).
- May present with symptoms and signs of metastatic spread. Neurological—headache, blurred vision, nausea, diplopia, ↓ consciousness, ataxia; bony pain and pathological fracture; liver and abdominal pain, anorexia, jaundice, ascites, liver failure, hepatomegaly; adrenal glands—symptoms of Addison's disease.
- Paraneoplastic syndromes.

History and examination

- Ask about: history of tobacco smoking (pack years), asbestos exposure, employment history, weight loss >5% body weight, recent onset of joint pains, change in voice, chest wall pain, back pain.
- Look for: lymphadenopathy, significant weight loss, pleural effusion, localized chest wall pain, clubbing, cutaneous lesions.

Principles of management

NSCLC is relatively resistant to chemotherapy. Curative resection allows the best chance of long-term survival. Surgery is normally offered to all patients with stage I and II disease, along with specific patients with stage III disease (after chemoradiotherapy). Surgical principles of resection are:

- No spillage of cells from 1° tumour during resection.
- Entire tumour must be resected by lobectomy or pneumonectomy, along with intrapulmonary lymph nodes; lesser resections proven to have worse outcome, only considered in high-risk patients.
- All accessible mediastinal lymph nodes should be excised or biopsied to allow complete staging and plan for any adjuvant therapy.
- Frozen section analysis of resection margins to confirm appropriate surgical resection and complete excision of 1° tumour.

Survival

- 1y survival—stage I: ♂ 81% ,♀ 85%; stage II: ♂ 66%, ♀ 69%; stage III: ♂ 42% ♀ 46%; stage IV: ♂ 23%, ♀ 28%.
- Post-operative mortality rate: lobectomy, 2%; pneumonectomy, 6%.

Chemotherapy

Neoadjuvant chemotherapy in stage IB to IIIB NSCLC patients showed significant improvement in overall survival.

Radiotherapy

- Radical. Used in patients unfit or unwilling to undergo surgery. Also used in those with bulky stage IV disease. Survival benefit in non-surgical patients when combined with concomitant chemotherapy.
- Adjuvant. Also used as an adjunct to surgery or as palliation.

Pleural effusion

Box 18.1 Light's criteria

An exudate is characterized by:

- Pleural fluid:serum protein >0.5.
- Pleural fluid:serum LDH >0.6.
- Pleural fluid LDH >200IU.

Key facts

Abnormal amount of fluid within the pleural space. Pleural effusions may be divided into transudates and exudates, according to Light's criteria (see Box 18.1), but the divide is not always clear.

Causes of transudates

- Cirrhosis of the liver.
- Nephrotic syndrome.
- Glomerulonephritis.
- CCF.
- Myxoedema.
- Sarcoidosis.
- Multiple PE.

Causes of exudates

- Neoplasms. Mesothelioma, metastatic disease, 1° lung cancer.
- PE.
- Chylothorax.
- Haemothorax.
- Infectious diseases. Viral and bacterial infections, fungal and parasitic infections, TB.
- GI disease. Pancreatitis, subphrenic abscess, intrahepatic abscess, perforated oesophagus.
- Collagen vascular disease. SLE, Wegener's granulomatosis, Sjögren's syndrome.
- Drug-induced pleural disease.
- Mediterranean fever.
- Rheumatoid disease, sarcoidosis, yellow nail syndrome.
- Asbestos exposure, electrical burns, radiation therapy.
- Trapped lung, post-pericardiectomy.
- Uraemia, urinary tract obstruction.
- Post-myocardial syndrome, Meigs' syndrome.

Clinical presentation

- Small effusions are often asymptomatic. Larger effusions cause cough, chest pain, and dyspnoea.
- ↓ ipsilateral chest expansion, dullness to percussion, ↓ breath sounds over the effusion on auscultation; crepitations may be heard.

Investigations

- Chest radiograph. Small effusions are demonstrated by blunting of the costodiaphragmatic angles; larger effusions produce a fluid level with a meniscus.
- USS. Useful for loculated effusions, helps to localize optimal site for chest drainage.
- CT. Useful when looking at underlying lung and pleural lesions.
- Pleural aspiration cytology, biochemistry, and microbiology. May obtain diagnostic information, helpful when planning treatment.

Management

If possible, treat the underlying cause. Simple pleural effusions due to fluid overload may resolve with diuresis.

- Tube thoracostomy (➲ Chest drain (intercostal drain/pleural drain) insertion pp. 262–3).
- Chemical pleurodesis (tetracycline, blood, talc).
- Surgical abrasion pleurodesis.
- Surgical pleurectomy (open or thoracoscopic).
- Pleuroperitoneal shunt.

Complications

- Infection and empyema.
- Treatment failure with recurrence of pleural effusion.
- Damage to underlying lung parenchyma, leading to prolonged air leak and bronchoalveolar air leak.

Empyema

This is an infected pleural fluid collection, commonly after pneumonia.

- Stage I. Acute exudative phase.
  - Typically occurs 2–5 days after a pneumonia.
  - Accumulation of fluid with low cellular content and viscosity.
  - Characterized by low WCC, LDH, and glucose, and a normal pH.
  - Can be successfully treated with antibiotics only.
- Stage II. Fibrinopurulent phase.
  - Typically occurs 5–14 days after a pneumonia.
  - Turbid or purulent fluid with heavy fibrin deposits.
  - Appearance of simple loculations and septations.
  - May have bacterial invasions and high numbers of polymorphonuclear neutrophils (PMNs) and lymphocytes.
  - Characterized by low pH and glucose and ↑ LDH.
  - Antibiotics and chest tube drainage is required, may need video-assisted thoracoscopic surgery (VATS) decortication.
- Stage III. Chronic organizing phase.
  - Lung trapping by collagen visceral and parietal pleural peel with ingrowth of fibroblast and capillaries.
  - Antibiotics and aggressive decortications, generally by thoracotomy.
  - Bacteriology.

Pneumothorax

Key facts

The presence of air in the pleural space with 2° lung collapse.

Aetiology

- 1° spontaneous pneumothorax.
- 2° spontaneous pneumothorax.
- Post-traumatic and iatrogenic.

Primary spontaneous pneumothorax

Commonly seen in young, tall ♂ smokers. Commoner on the right side. Less than 10% of cases are bilateral. Usually caused by rupture of small subpleural blebs (collections of air <2cm). Usually found at the apex of the upper lobe or the apical segment of the lower lobe. The rest of the lung parenchyma is normal. May also be caused by rupture of bullae (large air-filled spaces).

Presentation

- Dyspnoea, chest pain, cough, tachypnoea.
- Ipsilateral ↓ chest wall movement, hyperresonant hemithorax to percussion, absent breath sounds on auscultation, pleural rub, tachycardia.

Investigations

- PA chest radiograph usually diagnostic.
- CT scan gives an accurate estimate of size of pneumothorax and is useful for assessment of lung parenchyma and contralateral lung.

Complications

- Tension pneumothorax.
- Pneumomediastinum.
- Haemopneumothorax.
- Recurrent pneumothorax.

Management (British Thoracic Society guidelines)

- Observation: for asymptomatic and small pneumothorax <2cm for 1° spontaneous pneumothorax and <1cm for 2° spontaneous pneumothorax. The size of pneumothorax is measured between the lung margin and the chest wall (at the level of the hilum).
- Needle aspiration: >2cm and/or breathlessness in 1° spontaneous pneumothorax; 1–2cm and/or breathlessness in 2° spontaneous pneumothorax.
- Chest drain: unsuccessful needle aspiration or >2cm pneumothorax in 2° spontaneous pneumothorax.

Surgery

According to British Thoracic Society guidelines, thoracic surgical opinion should be sought for the following:

- Second ipsilateral pneumothorax.
- First contralateral pneumothorax.
- Synchronous bilateral spontaneous pneumothorax.

- Persistent air leak (despite 5–7 days of chest tube drainage) or failure of lung re-expansion.
- Spontaneous haemothorax.
- Professions at risk (e.g. pilots, divers).
- Pregnancy.

The aim is to resect the blebs or bullae and obliterate the pleural space with adhesions, either using chemical or abrasion pleurodesis or parietal pleurectomy (apical or full). It may be performed through a mini-thoracotomy or axillary incision, or thoracoscopically.

Recurrence rate

- <2% following surgical pleurectomy via mini-thoracotomy.
- 5% following thoracoscopic procedures.
- 5–10% following chemical pleurodesis.

Secondary spontaneous pneumothorax

Causes

- CF, chronic obstructive airways disease (COAD) and other bullous disease, asthma.
- Interstitial lung disease.
- Infections, including AIDS, mycobacterial, Pneumocystis carinii, bacterial, parasitic, mycotic.
- Malignancy. Bronchogenic carcinoma, metastatic lung cancer (sarcoma and lymphoma).
- Collagen diseases, catamenial, Ehlers–Danlos syndrome, histiocytosis X, scleroderma, lymphangioleiomyomatosis, Marfan's syndrome.
- Rupture of the oesophagus.

Cystic fibrosis Pneumothorax found in 10% of patients. Remember these patients are possible candidates for future lung transplantation when considering management options. Full parietal pleurectomy is a contraindication to lung transplantation.

COAD The commonest cause of 2° pneumothorax. Age usually >50y. Patients often have very little pulmonary reserve. They may not tolerate surgical management and single-lung ventilation. Treatment options are therefore tube thoracoscopy and chemical pleurodesis or long-term tube thoracoscopy.

Infection Cavitating pulmonary lesions rupture into pleural space.

AIDS Usually 2° to Pneumocystis carinii pneumonia. May be presenting feature of AIDS. Most effective treatment is surgical.

Catamenial Age 20–30y. Incidence 3–6% of women. Occurs 2–3 days following onset of menstruation. Right side more commonly affected. Usually small, presenting with dyspnoea and chest pain. Pathogenesis unclear.

Primary spontaneous pneumomediastinum

Uncommon. ♂ affected more frequently than ♀. Occurs following exertion or ↑ intra-abdominal pressure. Commonly associated with cocaine, marijuana, and crack cocaine usage. It is caused by rupture of alveolar sacs with air tracking along the peribronchial and perivascular spaces into the neck.

Presentation

- Sudden onset of chest pain, dyspnoea, dysphagia, and cough.
- Subcutaneous emphysema (neck and chest wall), Hamman's sign.
- Chest radiograph confirms diagnosis.

Management Non-operative, treat expectantly. Emergency surgical decompression very rare.

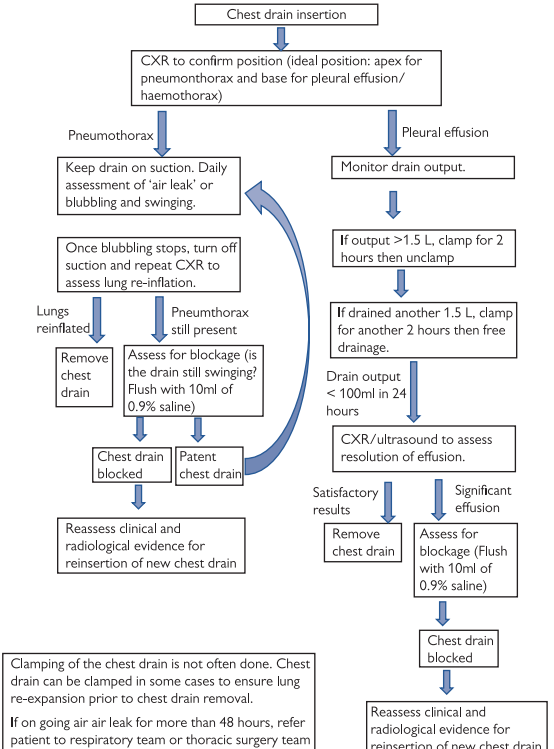

Fig. 18.4 Chest drain management flow chart.

Mediastinal disease

Pericardial effusion and cardiac tamponade

Key facts

- Pericardial effusion is abnormal fluid in the pericardial space; there is normally about 20mL of plasma ultrafiltrate.
  - Pericardial effusion may be acute or chronic.
  - ❗Acute accumulation of fluid can cause cardiac tamponade which is a surgical emergency (see Box 18.2).
  - Effusions commonly result from pericarditis, CCF, metastatic spread to pericardium commonly from lung or breast malignancy, lymphoma or leukaemia, autoimmune disorders, chronic hepatic and renal failure, and infections—specifically HIV.

Clinical features

- Very dependent on the time course. Acute accumulation of a small amount of fluid can cause life-threatening cardiac tamponade, whereas slow accumulations of large volumes of fluid may be well tolerated.
- Chronic pericardial effusion may present with ↓ exercise tolerance, atypical chest pain, orthopnoea, and associated signs of CCF, as well as features of cardiac tamponade.

Management

- Medical management includes diuretics and pericardiocentesis.
- Create a hole in the pericardium or pericardial window so that fluid can drain directly into the pleura via:
  - Thoracotomy or subxiphoid approach.
  - Left VATS approach.

Box 18.2 Cardiac tamponade

❗ Suspect cardiac tamponade if the patient has a history of chest trauma.

- ↓ BP, ↑ JVP (Beck's triad is ↓ BP, ↑ JVP + muffled heart sounds).
- Pulsus paradoxus (exaggeration of the normal ↓ BP with inspiration).
- Progressive tachycardia and dysrhythmias, including SVT, VF, and electromechanical delay (EMD).
- ↓ urine output.
- Excessive widening of the mediastinum on CXR.
- Echocardiography may show clot in pericardium and collapse of the RV in diastole.
- Equilibration of cardiac filling pressures (at cardiac catheterization).

Management

- Emergency pericardiocentesis (➲ Pericardiocentesis, pp. 270–1).
- Emergency thoracotomy or sternotomy.
- Aggressive fluid resuscitation is a temporizing measure.

Thymoma

Key facts

- The thymus is a bilobar structure located in the anterior mediastinum which contains lymphoid tissue. It is the location for maturation of T-cells in early life.
- Thymoma may be benign or malignant.
- Thymectomy is the definitive treatment for myasthenia gravis.

Clinical features

- Thymoma is usually asymptomatic in adults, whereas children often present with thoracic outlet obstruction or upper airway compromise.
- Clinical features of myasthenia gravis are described in ➲ Myasthenia gravis, p. 87.
- Thymoma appears as a smooth mass in the upper half of the CXR.
- CT shows an enlarged thymus, as well as lymph node involvement.

Treatment

- Thymectomy via a median sternotomy or thoracoscopy.

Key revision points—mediastinal anatomy

- Mediastinum is the space between the pleural sacs, below the thoracic inlet and above the diaphragm.
- Superior mediastinum.
  - From thoracic inlet to the line from the sternal angle to T4–5 space.
  - Contains the great vessels, trachea, oesophagus, phrenic nerves, vagus nerves, and thoracic duct.
- Anterior mediastinum.
  - Anterior to pericardium.
  - Contains sternopericardial ligaments, thymus, and lymph nodes.
- Middle mediastinum.
  - Contains pericardial cavity, heart, great vessels, and phrenic nerves.
- Posterior mediastinum.
  - Posterior to pericardium.
  - Contains the oesophagus, descending aorta, azygos veins, thoracic duct, and lymph nodes.

Chapter 19

Peripheral vascular disease

Acute limb ischaemia

Definition Any sudden decrease in limb perfusion that causes a potential threat to viability.

Prevalence One in 6000 of the population.

Causes and features

Acute thrombosis in a vessel with pre-existing atherosclerosis (60% of cases)

- Predisposing factors are: dehydration, hypotension, malignancy, polycythaemia, or inherited prothrombotic states.
- Features suggestive of thrombosis are:
  - Previous history of intermittent claudication.
  - No obvious source of emboli (see below).
  - Reduced or absent pulses in the contralateral limb.

Emboli (30% of cases)

- 80% have a cardiac cause (AF, MI, ventricular aneurysm).
- Arterial aneurysms account for 10% of distal emboli and may be from the aorto-iliac, femoral, popliteal, or subclavian arteries.
- Rarely, acute thrombosis in pre-existing atherosclerosis (see above) will embolize.
- Commonest sites of impaction are the brachial, common femoral, popliteal, and aortic bifurcation ('saddle embolus').
- Features suggestive of embolism are:
  - No previous history of claudication.
  - Presence of AF or recent MI.

Rare causes

- Aortic dissection, trauma, iatrogenic injury, peripheral aneurysm (particularly popliteal), and intra-arterial drug use.

Symptoms and signs (any cause)

Six Ps:

- **P**ain, **p**allor, **p**ulselessness, **p**erishingly cold, **p**araesthesiae, **p**aralysis.

Complications

- Death (20%).
- Limb loss (40%). Severe ischaemia leads to irreversible tissue damage within 6h.

Emergency management

Remember, patients will usually have coexisting coronary, cerebral, or renal disease.

Resuscitation

- Give 100% O_2.
- Get IV access and consider up to 1000mL of crystalloid fluid if dehydrated.
- Take blood for FBC, U&Es, troponin, clotting, glucose, and group and save.
- Request CXR and ECG (look for dysrhythmias).

- Give opiate analgesia (5–10mg morphine IM/IV).
- Call for senior help.

Establish degree of urgency

- Limb viability assessment. Involve senior help early.
  - *Irreversible*. Fixed mottling of skin, petechial haemorrhages in skin, woody hard muscles.
  - *Immediately threatened* ▶▶ Immediate treatment needed. Muscles tender to palpation/swollen, loss of power, loss of sensation.
  - *Marginally threatened*. Prompt treatment after investigation. Pulseless, pale, cold, reduced capillary refill, motor power preserved.

Treatment of all patients

Give heparin (5000IU UFH IV bolus (~70IU/kg) and start an infusion of 1000IU/h) if there are no contraindications (e.g. aortic dissection, multiple trauma, head injury).

- Recheck APTT in 4–6h.
- Aim for a target time of 2–2.5 times the normal range.

Definitive management

Depends on the severity of ischaemia (as above) and there are three broad categories.

- Irreversible (non-salvageable limb). Amputation is inevitable and urgent (but not an emergency).
- Immediate treatment needed (to prevent the systemic complications of muscle necrosis—hyperkalaemia, acidosis, acute renal failure, and cardiac arrest). Consider amputation if ischaemic changes advanced and life-threatening. Surgery to revascularize limb and perform fasciotomies (to prevent or treat compartment syndrome).
- Prompt treatment after investigation. Continue heparinization. Angiogram (stop heparin 4h before) or Duplex USS / CT angiogram to determine cause/location of disease. Thrombolysis, angioplasty, arterial surgery, or combination. Limb may remain viable and functional after period of heparinization alone.

Principles of embolectomy (➲ Femoral embolectomy, p. 864.)

Chronic upper limb ischaemia

Key facts

Upper limb ischaemia occurs less frequently than lower limb ischaemia.

Causes

- Previous trauma or axillary irradiation, leading to arterial stenosis.
- *Atherosclerosis.* As in the lower limb.
- Buerger's disease. Affects small vessels of the hands and feet, principally in smokers, associated with Raynaud's phenomenon, mostly young men, but women may be affected; presents with digital gangrene/ischaemia and may present with acute limb ischaemia in young people.
- Subclavian steal syndrome. Stenosis of subclavian artery proximal to vertebral artery origin; arm claudication causes reversed flow in the vertebral artery/diminished hindbrain perfusion (dizziness/syncope).
- Takayasu's arteritis. Uncommon in Europe; major arch/upper limb vessels affected.
- Thoracic outlet syndrome (see Fig. 19.1).
  - Term used to cover a spectrum of symptoms resulting from compression of the neurovascular bundle (NVB) as it leaves the chest to enter the upper limb, in an area enclosed by the first rib, clavicle, and scalenus anterior muscle.
  - Presents as a variable combination of neural, arterial, and venous symptoms exacerbated by elevation of the limb, with shoulder, arm, or head pain, and paraesthesiae, weakness, or arm claudication. Ninety-five per cent are neurogenic and 5% are arterial or venous manifestations (arm engorgement, swelling with subclavian vein stenosis or thrombosis—Paget–Schroetter syndrome).

Clinical features

- Weakness, cramp or exercise-related pain, and digital ischaemia/gangrene.
- Examine bilateral upper limb pulses, BP in both arms (elevated/at sides), and wrist Doppler pressures.

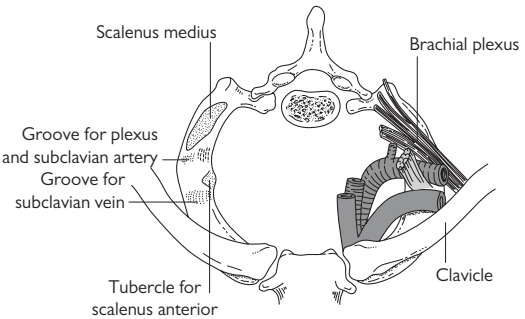

Fig. 19.1 Structures involved in thoracic outlet syndrome.

- Roos test. Arms abducted to 90°, hands up with elbows braced backward, chin elevated, hands serially clenched/opened for 1–2min, positive if pain or weakness in hands or forearms.
- Adson's test. Pulse diminishes or absent on elevation/abduction of arm, with head turned to contralateral side. Reliability improved by using in conjunction with arterial duplex.
- Allen's test. Assesses integrity of the palmar arch and dominant vessel (radial or ulnar).
- Tinel's test. For carpal tunnel syndrome.

Diagnosis and investigation

- Cervical spine and thoracic outlet X-rays, wrist Doppler pressures.
- CT/MRI to look for fibrous bands/ribs and stenoses/occlusions.
- Arterial duplex or angiography to diagnose proximal arterial lesions.
- Duplex or venography for subclavian vein stenosis or occlusion.

Treatment

Thoracic outlet syndrome

- Mild neurogenic problem. Simple analgesia, physiotherapy, and advice on risk factors.
- Surgery (supraclavicular or axillary approach) has good results for those with arterial or venous symptoms/complications.
- Excision of the first rib/band will improve symptoms in over 90%.
- Careful evaluation is needed prior to surgery for pure neurological symptoms, e.g. nerve conduction studies.

Cervical sympathectomy in upper limb disease

Indications

- Palmar hyperhidrosis (less effective and infrequently used for axillary).
- Buerger's disease/small vessel disease with digital ischaemia.

Approach

- Aim is to de-innervate the second and third thoracic ganglia.
- Approach is almost universally thoracoscopic and open approaches have been largely abandoned.

Complications

- Horner's syndrome.
- Pneumothorax.
- Haemorrhage.
- Compensatory truncal hyperhidrosis.
- Frey's syndrome (gustatory sweating).

Axillary hyperhidrosis Treatment of choice is now subcutaneous botulinum toxin A injections to the axillary sweat glands, repeated as necessary, often 6-monthly.

Key revision points—anatomy of the thoracic outlet

Several structures can compress the neurovascular structures:

- Cervical rib. Articulates with C7.
- Scalenus anterior. Aberrant anatomy or scarring/swelling from trauma.
- Costoclavicular ligament.

Chronic lower limb ischaemia

- Atherosclerosis is a generalized disease and has a predilection for the coronary, cerebral, and peripheral circulations (➔ Atherosclerosis, p. 192).
- In the lower limb, it may affect the aorto-iliac, femoral or popliteal, and calf vessel levels, singly or in combinations (see Fig. 19.2).
- Single-level disease usually results in intermittent claudication (IC), and multi-level disease in 'critical limb ischaemia' (CLI).

The Fontaine classification of lower limb ischaemia

- I. Asymptomatic.
- II. Intermittent claudication.
- III. Rest pain.
- IV. Ulcers/gangrene.

Grades III and IV = 'critical limb ischaemia'.

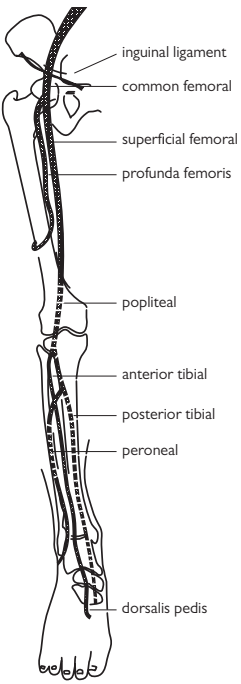

Fig. 19.2 The arterial supply to the leg.

Reproduced with permission from Longmore, M. et al. (2007). Oxford Handbook of Clinical Medicine, 7th edn. Oxford University Press, Oxford.

Intermittent claudication

Key facts

- Affects 4% of people over 55y, mostly men.
- One-third of patients improve, one-third remain stable, and one-third deteriorate.
- 4% require an intervention and 2% result in amputation.
- For risk factors and association see Table 19.1 below.

Clinical features

- Muscular pain on exercise of the affected limb—most commonly in the calf—worsened with increasing levels of exercise, relieved by rest.
- Differential diagnosis.
  - Spinal stenosis. Neurogenic pain caused by drop in distal cauda equina blood flow due to exercise, leading to neurogenic pain.
  - Osteoarthritis, especially of the hip joint.
  - Nerve root entrapment, e.g. sciatica.
  - Popliteal artery entrapment (rare). Due to compression of popliteal artery over medial head of gastrocnemius during exercise. Distal pulses reduced/absent on plantar flexion alone. Treated by surgical release after MRI defines anatomy.

Diagnosis and investigations

- Diagnosis is mostly clinical and not based on imaging.
- ABPI <0.9 in presence of appropriate history is diagnostic.
- In equivocal cases, exercise ABPI may show a post-exercise significant decrease in ABPI coinciding with pain.
- Measure BP, serum glucose, HbA1C, cholesterol, and FBC.
- Imaging is only really indicated if intervention is planned. Duplex ultrasound, CT or MR angiogram, or digital subtraction angiogram may all be used.
- Abdominal ultrasound to rule out aneurysm.

Treatment

Risk factor modification

- 40% of PVD patients have significant coronary or cerebral arterial disease; the mainstay of treatment is aggressive risk factor modification. Stop smoking, oral statin treatment, ↑ exercise, control of BP and serum glucose, antiplatelet therapy.

Table 19.1 Intermittent claudication—risk factors and associations

Risk factors	Associations
Tobacco smoking	Obesity
Hyperlipidaemia	Diet
Diabetes mellitus	Sedentary lifestyle
Hypertension	Gender
Positive family history	Occupation

Endovascular treatment

- Angioplasty ± stent. Excellent results in the aorto-iliac segment (>90% success) and good results in the superficial femoral segment (90% success and 60–80% patency at 2y).
- Usually performed under LA percutaneously as a day case.
- Rarely performed for claudication in the popliteal and tibial segments due to high risk of occlusion.
- Most useful in short stenoses/occlusions.

Surgery

- Used for short-distance claudication, severe lifestyle limitation, and failure/unsuitability of endovascular treatment in the aorto-iliac segments.
- Procedures available are:
  - Aortobifemoral graft. 5y patency of >90%, but carries 2–5% mortality and a risk of impotence. Used for younger patients.
  - Femoro-femoral crossover bypass graft. Used for isolated unilateral iliac disease; 90% 1y patency.
  - Common femoral endarterectomy. Used for isolated common femoral disease; good results and a low complication rate.
  - Femoro-above-knee popliteal bypass. 80% 2y patency with vein or prosthetic graft.
  - Femoro-below-knee popliteal bypass. 70% 2y patency with vein graft.
  - Femoro-distal (below-knee) bypass. 5y patency <35%; usually reserved only for critical ischaemia.
- Most commonly used for long stenoses/occlusions and combined with endovascular treatment.

Critical limb ischaemia

Key facts

- Often progresses to limb loss or progressive tissue loss if it remains untreated.
- High-risk condition, often occurring in high-risk patients with multiple comorbidities.

Clinical features

- European consensus guidelines define critical ischaemia as either rest pain unrelieved by analgesia for >2 weeks and/or tissue loss occurring in the presence of an ankle systolic pressure <50mmHg or toe systolic pressure <30mmHg.
- Rest pain typically worsens at night and during elevation of the limb and is relieved by hanging the limb dependent.
- Arterial ulceration cannot reliably be distinguished from other causes.

Diagnosis and investigations

Diagnosis is clinical. Investigation should aim to:

- Identify and treat risk factors. Treat BP, stop smoking, optimize diabetes control, reduce cholesterol (statins by choice), and use antiplatelet agent (aspirin/clopidogrel by choice).
- Identify location and severity of all arterial stenoses involved. May include:
  - Colour duplex Doppler ultrasound. Zero risk, non-interventional, and often first choice for assessing infra-inguinal arterial disease in particular.
  - CTA. Low risk (radiation and contrast). Usually superior for imaging the aortoiliac segment.
  - Angiography (usually DSA). Interventional, carries risk of arterial injury and renal toxicity of contrast, good for popliteal and distal vessel assessment.
  - MRA. Low risk.

Treatment

- All efforts should be made to revascularize if possible (providing the general condition of the patient allows it).
- General principle is that rest pain will be relieved by dealing with one level of disease only; tissue loss requires the restoration of 'in-line' flow.

Medical care

- Nursing care.
- *Analgesia*. Opiates (e.g. morphine sulfate solution or modified-release tablets).

Endovascular treatments

Angioplasty ± stenting - most successful for short occlusive lesions or stenoses in the aorto-iliac, and superficial femoral arteries. Less successful in popliteal disease and distal disease, but the available techniques are evolving to tackle even very extensive multi-level disease.

Surgery

- Femoro-distal bypass (e.g. to popliteal, tibioperoneal trunk, or anterior or posterior tibial arteries).
- Aorto-iliac bypass ('anatomical') or axillo-femoral or femoro-femoral ('extra-anatomical') bypass.
- Amputation.
  - Usually below knee or above knee in smoking-related atherosclerosis.
  - Distal amputations (toe, forefoot, ankle) may be appropriate in diabetic disease.
- There is little evidence of benefit from treatment with prostacyclin or sympathectomy (surgical or chemical).

Aneurysms

Key facts

- An aneurysm is an abnormal localized dilatation of a blood vessel.
- It may be associated with structural abnormalities of collagen and elastin in the vessel wall.
- Prevalence of AAA: 4% of men aged 65, increasing with age.
- ♂:♀, 5:1.
- Associated with HTN, tobacco smoking, and family history (all associated with atherosclerosis).

Pathological features

Types

- True aneurysms. Contain all three layers of artery wall. May be fusiform (symmetrical dilatation) or saccular. Underlying cause is usually atherosclerosis-related; less commonly associated with infective causes ('mycotic aneurysm') and hereditary connective tissue disease e.g. Marfan, Ehlers–Danlos syndrome (collagen and elastin abnormalities).
- False aneurysms. Do not contain all three layers of vessel wall and often only lined by surrounding connective tissue or adventitia. Usually 2° to penetrating trauma, including iatrogenic injury (e.g. femoral cannulation, surgery).

Sites

- Thoracic, abdominal, and peripheral (iliac, femoral, popliteal, visceral, carotid, or subclavian), cerebral 'berry' aneurysms. The most common site is the infrarenal aorta.

Clinical features

Thoracoabdominal aneurysm

- Classified by Crawford classification (I–IV, dependent on extent of involvement of the thoracic/abdominal aorta): managed in specialist centres.
- Often asymptomatic.
- It is different from aortic dissection—which presents with acute chest or back pain, acute AR, or cardiac failure.
- Diagnosed by widened mediastinum on CXR or on CT/MRI.
- Rupture has high mortality and rare without prior symptoms.
- Elective surgery has up to 20% mortality and risk of paraplegia; 10% require dialysis after surgery.
- Endovascular stenting is a potential treatment of choice due to high risks associated with open surgery.

Abdominal aortic aneurysm (AAA)

- 95% start below the origin of the renal arteries ('infrarenal').
- 15% extend down to involve the origins of the common iliac arteries ('aortoiliac').
- Associated with other peripheral aneurysms (often, popliteal: ▶ remember to check popliteal pulses routinely).
- 5–10% are 'inflammatory' (have gross connective tissue changes around the aortic wall in the retroperitoneum).

- Most are asymptomatic; 40% are detected incidentally (clinical examination, ultrasound, AXR, CT KUB).
- A national ultrasound screening programme exists.(➔ Screening p. 210)
- Six-monthly scans for surveillance if size 4–5.4cm (1% per year risk of rupture).
- Mycotic aneurysms are rare but have a high rupture rate.
- Risk of rupture and mortality increases with increasing aneurysm diameter.
- Surgical intervention is indicated for:
  - AP diameter >5.5cm in fit individuals.
  - Rapid increase in diameter on serial surveillance scans, e.g. >0.5cm in 6 months.

Peripheral aneurysms

- *Iliac*. 2% of patients >70y. Mostly common iliac and asymptomatic. Rarely palpable and rupture may be missed as acute abdomen or renal colic.
- *Femoral*. Mostly asymptomatic pulsatile groin swelling or pain. May present with lower limb ischaemia.
- *Popliteal*. Many asymptomatic and over half are bilateral. May present with acute limb ischaemia. Aneurysm thrombosis is associated with high risk of limb loss. Prophylactic bypass probably best for symptomatic, embolizing aneurysms.
- *Carotid*. Rare and may be bilateral. May present with neurological or pressure symptoms. May present simply as a pulsatile neck swelling. Rarely presents with rupture. Diagnosis with duplex scan.
- *Visceral*. Account for 1% of all aneurysms. Generally small and asymptomatic until rupture. Splenic artery commonest, followed by hepatic and renal arteries.

Treatment of abdominal aortic aneurysm

Aim is to prevent death, as 80% of patients with ruptured AAA will die.

Elective surgery

- Open repair by inlay synthetic graft. May be 'straight' if aneurysm confined to aorta or 'bifurcated/trouser' if there are common iliac aneurysms as well; 3–7% operative mortality.

Endovascular repairs

- Endovascular aneurysm repair (EVAR) with a stent graft. Percutaneous insertion of covered stent to exclude the aneurysmal segment from arterial pressure.
- *Advantages*. Percutaneous technique, reduced early mortality.
- *Disadvantages*. High early re-intervention rate, requires lifelong surveillance, no long-term survival benefits over open repair shown to date.

Ruptured abdominal aortic aneurysm

Causes and features

- Associated with HTN (especially uncontrolled), smoking, family history, and atherosclerosis.
- Rare in patients aged <55.
- Risk of rupture relates to maximum AP diameter.
  - <0.5% per year, <4.0cm diameter.
  - 1% per year, 4–5.5cm.
  - Over 3% per year, >5.5cm.
- Patients with a 'contained leak' with initial haemodynamic stability proceed rapidly to rupture.
- Less than 50% of patients with a ruptured AAA reach hospital alive and the overall mortality of the condition may be as high as 75–95%.
- Outcome is best in the hands of an experienced vascular team (vascular surgeon, vascular anaesthetist, theatre nursing team, two assistants, and ITU) and a rapid transfer from the emergency department to theatre.

Symptoms

- Severe/sudden-onset epigastric and/or back/loin pain.
- History of sudden 'collapse', often with transient hypotension.
- May have history of AAA under surveillance.

Signs

- Cardinal signs are unexplained rapid-onset hypotension, pain, and sweating.
- A pulsatile abdominal mass is not always easy to feel (due to pain and abdominal wall rigidity).

Emergency management

Resuscitation

- ▶▶If the diagnosis is seriously considered, call for senior surgical assistance immediately. Transfer to theatre may be required, even as resuscitation continues.
- ▶▶Alert anaesthetist (and ITU), theatres and blood bank.
- ▶ *Permissive hypotension*. Do not chase a 'normal' systolic BP to reduce the risk of worsening the rupture.
- If the patient is critically hypotensive, consider calling a peri-arrest cardiac emergency.
- IV access via two large-bore cannulae, catheterize, cross-match blood (10U), order FFP and platelets.
- High-flow O_2 via non-rebreathing mask.
- Give modest doses of analgesia (morphine 5–10mg)
- Witnessed verbal consent for surgery may be the only practical way and is acceptable here.

Establish a diagnosis

- If patient stable and diagnosis uncertain, request contrast CT.
- If going to CT, ensure blood is sent for cross-match and IV access has been established before going.

Early management

Ruptures fall into three groups:

- Considered not a candidate for surgery. For analgesia and palliative care. Mortality approximates to 100%. The decision is based on age, physiological status, comorbidities, expressed patient preference, and family wishes.
- Free rupture with collapse and critical condition. All candidates for surgery require emergency transfer to theatre.
- Contained leak. May be stable after initial presentation, but likely to progress to free or complicated rupture unless urgently surgically treated.

Principles of surgery for rupture

- Go straight to the operating table, not the anaesthetic room.
- Repair may be open or endovascular (REVAR)
- If unstable, muscle relaxation/anaesthesia is not started until patient is prepared/draped and surgical team ready to go. LA only may be best for REVAR.
- If haemodynamically stable, central line and arterial line sited whilst waiting for blood to arrive and surgical team in theatre.
- Give antibiotic prophylaxis.
- Proximal neck is controlled rapidly with aortic cross-clamping (open) or an intra-aortic balloon (REVAR). Full fluid expansion with blood can now safely begin.
- Open repair may be with a tube or bifurcated graft.
- REVAR ideally is performed using a bifurcated endograft, although an aorto-uni-iliac endograft may be indicated in hostile anatomy or in a very unstable patient.

Post-operative care

- Transfer to ITU.
- Normalize core temperature.
- Correct clotting and maintain Hb.
- Adequate analgesia and accurate fluid balance.
- Attention to cardiac/renal/pulmonary dysfunction.

Complications

- Death (overall up to 50% of operated cases).
- MI.
- Renal failure.
- Lower limb embolism.
- Gut ischaemia/infarction.
- Abdominal compartment syndrome.

Vascular developmental abnormalities

Key facts

Classified broadly into two principal groups.

Vascular tumours (e.g. haemangiomas)

- All congenital or idiopathic.
- Mostly sporadic but may rarely be part of a familial syndrome (e.g. VHL).
- Pulmonary haemangiomas, commonly seen in hereditary haemorrhagic telangiectasia, are linked to a deficiency in endoglin (endothelial growth factor).

Vascular malformations

- Vascular malformations are histologically categorized as capillary, venous, lymphatic, arteriovenous malformations (AVMs), or mixed-in type, depending on the predominant vessel type affected and subdivided into low- or high-flow varieties.
- AVMs have three main causes.
  - *Congenital.* Origin/cause unknown.
  - *Traumatic.* May follow relatively minor trauma.
  - *Iatrogenic.* Following a variety of surgical/interventional procedures.

Clinical features

- Congenital AVMs are usually evident at birth and the superficial lesion may only represent a part of the overall abnormality.
- Symptoms are dependent on the size, site, and type of vessel affected, and whether the AVMs are high or low flow.

Low flow

- May result in considerable cosmetic deformity if large (e.g. Klippel–Trenaunay—port wine stain + ipsilateral hypertrophy, usually limb).
- Pain may be a feature due to spontaneous thrombosis of some/all of the venous elements.
- Typically, the symptoms are worse after exercise when blood flow is maximized.

High flow

- These are largely asymptomatic, but there may be a detectable venous hum or bruit.
- They may result in local hyperhidrosis, heat, or ulceration, or present with profuse bleeding.
- May lead to high-output cardiac failure if large and untreated.

Diagnosis and investigations

- Colour duplex. Diagnoses lesion, can estimate flow rate, and is useful for follow-up monitoring.
- MRI has replaced CT as the best imaging modality and gives both the extent and related anatomy for complex lesions.
- Angiography is reserved for high-flow lesions when suitability for embolization or surgery is being assessed.

Treatment

- Largely conservative.
  - Congenital AVMs frequently reduce in size with growth of the child and treatment is rarely easy, with recurrence common.
  - Adult AVMs only require treatment for complications or occasionally cosmesis.

Interventional radiology

- Percutaneous or intravascular embolization using wire coils or sclerosant under radiological guidance.
- Risks include:
  - Those of percutaneous puncture (infection, false aneurysm formation, inadvertent embolization of adjacent vessels).
  - Tissue necrosis after successful lesion embolization.
  - Post-embolization syndrome may occur with pain at the site of embolization, accompanied by malaise, fever, leucocytosis, and hyperkalaemia. This usually settles with symptomatic treatment in 24–48h. Due to tissue necrosis and cytokine release.

Surgery

- Small lesions may be excised completely.
- Obliteration of small superficial venous malformations can be undertaken by direct puncture and injecting a sclerosant such as sodium tetradecyl sulfate (STD).
- Open surgery is mostly confined to high-flow lesions after preoperative embolization.

Carotid disease

Key facts

- A cerebrovascular accident (CVA), or stroke, is 'a rapidly developing neurological deficit caused by ischaemia with infarction'.
- A transient ischaemic attack (TIA) is 'an acute episode of focal (cerebral or visual) neurological deficit caused by ischaemia without infarction'.
- CVA is the third commonest cause of death in the UK after coronary heart disease and cancer.
- Incidence—stroke, 200 per 100 000; TIA, 35 per 100 000.
- ~150 000 CVAs occur in the UK per year and ~15% of these are due to atherosclerotic disease of the carotid arteries.

Pathological features

CVA or TIA may arise from disease at the origin of the internal carotid artery (ICA) due to platelet or atheromatous embolization from the plaque surface (usually following plaque rupture).

Clinical features

- Several clinical variants of a classic CVA are recognized.
  - Stroke in evolution. Progressive neurological deficit occurring over hours/days.
  - Completed stroke. The stable end-result of an acute stroke lasting over 24h.
  - Crescendo TIA. Rapidly recurring TIA with increasing frequency, suggesting an unstable plaque with ongoing platelet aggregation and small emboli.
- Carotid bruits are detectable in over 10% of patients aged >60, poor correlation with the degree of stenosis/risk of CVA, and may not arise from the ICA.
- Patients with a significant stenosis may have no audible bruit.

Neurological features

Depend on the territory supplied by the vessel affected by the embolism, the degree of collateral circulation to that territory, and the size/resolution of the embolism.

- Amaurosis fugax. Transient monocular visual loss (described as a curtain coming down across the eye), lasting for a few seconds or minutes—central retinal artery (occlusion can lead to permanent blindness).
- Internal capsular stroke. Dense hemiplegia, usually including the face—striate branches of the middle cerebral artery.
- *Hemianopia.* Loss of vision in one half of the visual field.

Prognosis of patients with TIA

Eighty per cent of TIAs are in the carotid territory. The risk of stroke following a TIA is around 18% in the first year, 10% in the first 90 days, and 4% in the first 24h.

Diagnosis and investigations

- Colour duplex scan. All patients with TIA/CVA within last 6 months.
- MRA or CTA. Used when duplex is inconclusive or difficult due to calcified vessels.

Treatment

Medical management

- Best medical therapy is an antiplatelet agent (e.g. aspirin, dipyridamole), smoking cessation, optimization of BP and diabetes control, and a statin (e.g. simvastatin 40mg daily) for cholesterol lowering, irrespective of baseline cholesterol.
- Acute thrombolysis/mechanical thrombectomy in CT-proven ischaemia indicated in specialized units if detected early.

Surgery

Carotid endarterectomy (CEA)

- Offered to patients with symptomatic >70% stenosis of the ICA or >50% stenosis if recent TIA/CVA and high ABCD2 risk score (age, BP, clinical, duration, diabetes). Men with 50–69% symptomatic stenosis may also benefit from CEA.
- Urgent CEA within 2 weeks now considered for all patients presenting with acute TIA/CVA.
  - ECST (Europe) and NASCET (North America) trials demonstrated ↓ CVA in the first year following CEA, from 18% with best medical therapy to 3–5% with surgery and best medical therapy. No significant benefit to symptomatic patients with <70% stenoses.
  - For Asymptomatic patients, intervention should be considered with >60% stenosis
- Technical details.
  - May be under GA or LA.
  - Incision anterior to sternomastoid.
  - IV heparin prior to trial clamp (if patient awake).
  - Cerebral circulation protected in 10% of awake patients with a shunt (Pruitt/Javid) (without an intact circle of Willis, there is not enough collateral blood flow from the contralateral carotid).
  - Shunt in GA patients, depending on surgeon preference and cerebral monitoring (stump pressure of 50mmHg or transcranial Doppler monitoring of middle cerebral artery blood flow).
  - Patch closure of the arteriotomy common. Eversion endarterectomy technique may avoid the need for a patch.
  - Post-operatively, close monitoring of BP and neurological state.
- Complications.
  - Death or major disabling stroke, 1–2%.
  - Minor stroke with recovery, 3–6%.
  - MI.
  - Wound haematoma.
  - Damage to hypoglossal nerve (weak tongue, moves to side of damaged nerve), glossopharyngeal nerve (difficulty swallowing), facial/neck numbness.

The diabetic foot

Key facts

Foot ulceration is the commonest endpoint of diabetic vascular complications. Diabetics are 15 times more likely to undergo major lower limb amputation than non-diabetics.

Causes and features

- Key features of the diabetic foot are:
  - Ulceration.
  - Infection.
  - Sensory neuropathy.
  - Failure to heal trivial injuries.

Ulceration

- Risk factors for ulceration include:
  - Previous ulceration.
  - Neuropathy (stocking distribution loss and Charcot joints).
  - Peripheral arterial disease—more commonly affects the below-knee calf vessels which are frequently highly calcified, giving rise to falsely elevated ABPI readings or incompressible vessels.
  - Altered foot shape.
  - Callus, indicating high foot pressures.
  - Visual impairment.
  - Living alone.
  - Renal impairment.
- 2° to either large-vessel or small-vessel arterial occlusive disease or neuropathy or a combination of both.
- 45% of diabetic foot ulceration are purely neuropathic in origin, 10% are purely ischaemic, 45% are of mixed neuro-ischaemic origin.

Diagnosis and investigations

Pure neuropathic ulceration

- Warm foot with palpable pulses.
- Evidence of sensory loss, leading to unrecognized repeated local trauma.
- Normal or high duplex flows.

Ischaemic/neuro-ischaemic ulceration

- Foot may be cool.
- Absent pulses.
- Ulcers commonly on toes, heel, or metatarsal head.
- 2° infection may be present with minimal pus and mild surrounding cellulitis.
- ABPIs may be misleadingly high.
- Duplex ultrasound assessment.
- Angiography for suspected critical ischaemia.

Treatment

Prophylactic management

- Best undertaken in a specialist diabetes foot clinic with multidisciplinary input.
- Regular foot inspection for evidence of pressure/ulceration.
- Always use appropriate wide-fitting footwear.
- Attention to nail care with regular chiropody.
- Chiropodist debridement of pressure sites/callus.
- Keep away from heat and do not walk barefoot.

Established ischaemic ulceration

- Treat local or systemic infection.
  - Broad-spectrum antibiotics (local guidelines).
  - Debride obviously dead tissue, including digital amputation.
  - Drain collections of pus.
  - Take plain X-ray for signs of underlying osteomyelitis.
- Consider revascularization if appropriate.
  - Angioplasty.
  - Femoro-distal bypass.
- Consider amputation for failed medical or surgical treatment.
  - Often possible to do limited distal amputations (e.g. transmetatarsal).
  - May be progressive if disease spreads.

The diabetic surgical patient

- Renal disease requires close monitoring of hydration, BP, and renal function.
- Metformin needs to be stopped for 48h before angiography to avoid lactic acidosis.
- Insulin-dependent diabetics starved for any reason require a sliding scale.
- Avoid pressure sores if immobile for any long period with inflatable leg troughs, heel offloading, and prompt attention to any skin breaks.

Amputations

Key facts

- 90% for arterial disease, 10% for trauma, and rarely for venous ulceration, tumour, or deformity.
- Amputation may be a very beneficial treatment for pain, to restore mobility or, occasionally, to save life in trauma or acute limb ischaemia.
- Amputation for arterial disease carries significant mortality and major morbidity.
- The surgical aim is to achieve a healthy stump for a suitable prosthesis and successful rehabilitation.
- Amputees are at the centre of a large team, including surgeons, nurses, physiotherapists, prosthetists, occupational therapists, pain team, counsellors, and the family.

Causes and features

- 'Dangerous': lifesaving.
  - Spreading gangrene, e.g. necrotizing fasciitis, gas gangrene.
  - Extensive tissue necrosis following burns or trauma.
  - Uncontrolled sepsis (diabetic foot) with systemic infection.
  - 1° malignant limb tumours not suitable for local excision.
- 'Dead': vascular events.
  - CLI with unreconstructable disease.
  - Extensive tissue necrosis.
- 'Damn nuisance': neuropathy or deformation. e.g. failed, complicated orthopedic surgery with severely impaired gait.

Level

- The level is chosen according to:
  - Lowest level where tissue is viable for healing.
  - Include as many working major joints as possible to improve function.
  - Be ideally sited between large joints to allow prosthesis fitting.
- Above knee. Most will heal, but only young and fit achieve walking with a prosthesis.
- Through knee. Not favoured traditionally but gives better functional results than above-knee amputation (AKA).
- Below knee. About two-thirds heal and more achieve walking than with AKAs.

Types

- Hip disarticulation. Rarely needed, but indicated for trauma or tissue necrosis above high thigh.
- *AKA.* Bone transected at junction of upper two-thirds and lower third of femur (12–15cm above knee joint), common in end-stage vascular disease.
- Gritti–Stokes (supracondylar AKA). Increasingly popular for bilateral amputees as creates a long stump; especially good for wheelchair-dependent patients.
- Through-knee amputation (TKA). Produces a wide stump, which is difficult for prosthesis fit.
- Below-knee amputation (BKA). Weight-bearing on patellar tendon with good prosthetic fit; good knee function essential.

  - Skew flap is arguably best technique as it produces a better stump for prosthetic fitting.
  - Alternative is a posterior flap, which is bulkier and leads to longer time to mobilization.
  - The tibia is transected 8–10cm distal to the tibial tuberosity and the fibula 2cm more proximally.
  - Post-operative mobilization is early and temporary limb aids can be used when the wound is sound.
- Symes (ankle). Few indications for this in vascular patients and best avoided other than in trauma or diabetics. Prosthetic fitting is difficult and a good BKA is better for walking.
- *Transmetatarsal.* Useful in diabetics or when several toes are gangrenous.
- *Ray.* Used when digital gangrene extends to forefoot, especially useful for diabetics when infection tracks up tendon sheath.
- *Digital.* Usually only for diabetic disease or local trauma.

Treatment

Preoperative care

- Restore Hb levels and correct fluid and electrolyte balance.
- Ensure good diabetes control.
- Cross-match 2U of blood.
- Adequate analgesia (epidural may reduce phantom pain).
- ECG and CXR.
- Optimize cardiac function.
- Prophylactic antibiotics.
- Counselling if available.

Post-operative care

- Pain control with epidural ± PCA.
- Regular physiotherapy to prevent muscle atrophy or contractures, as well as upper limb exercises.
- Early rehabilitation on temporary limb aid.
- Own wheelchair to aid early mobilization.

Complications

- Infection.
- Non-healing of stump.
- Progression of underlying disease and higher-level amputation.
- Phantom limb pain. Due to hypersensitivity in divided nerves, can be helped with gabapentin, amitriptyline, or carbamazepine.
- Failed mobilization. Early regular analgesia and physiotherapy are important.
- Perioperative cardiovascular events in arteriopathic patient.

Vasospastic disorders

Key facts

Many systemic disorders have vasospasm as part of their presentation.

Causes

Rheumatological disease

Often associated with autoimmune disease.

- Systemic sclerosis.
- SLE.
- RA.
- Sjögren's syndrome.
- Dermatomyositis.
- Polymyositis.

Neurological disease

- Reflex sympathetic dystrophy.
- Post-traumatic vasospasm.
- Vibration white finger (due to exposure to handheld vibrating tools in miners, fitters, builders, and platers).

Drug-induced

- α-agonist treatment (ergotamine).

Idiopathic

Raynaud's disease.

- ♂ < ♀.
- Affects 20–30% of young women, with a possible familial predisposition.
- Possibly due to deficiency of a potent vasodilator (a calcitonin gene-related peptide) in the digital nerves, allowing action of unopposed cold stress-induced release of the vasoconstrictor endothelin.

Features

Vasospasm of any cause results in 'Raynaud's phenomenon'.

- Intermittent attacks.
- Initiated with pallor ('white'). Due to local tissue oligaemia.
- Proceeding to cyanosis ('blue'). Due to venous stasis and deoxygenation.
- Followed by rubor ('red'). Due to reactive hyperaemia as blood flow is restored.

Diagnosis and investigations

- Stop all vasoactive treatment 24h prior to assessment.
- After local cooling to 15°C, finger Doppler pressures change (fall >30mmHg significant).
- *Screen.* FBC, U&Es, urinalysis, TFTs, plasma viscosity, rheumatoid factor, autoantibody screen.

Treatment

Medical

- Avoidance of precipitating factors (e.g. outdoor work, smoking).
- Electrically heated gloves/socks.
- Drug therapy used if symptoms are severe enough to interfere with work/lifestyle.
  - Calcium channel blockers (e.g. nifedipine 10mg/day, increasing to 20mg/day tds) may help, but side effects (headache) may limit use.
  - Iloprost (prostacyclin) infusion. Weight-related doses given IV over 48–72h, as tolerated by side effects, for severe pain or impending/actual tissue loss.

Surgical

Sympathectomy. Reserved for patients with failure to respond to medical therapy or 2° complications (e.g. digital ulceration).

- *Lumbar.* Open/laparoscopic/chemical for foot symptoms; effects are mostly short-lived.
- *Cervical.* Mostly now thoracoscopic technique; effects are poor response rate and high relapse rate.

Varicose veins

Key facts

- The venous system of the leg comprises three groups:
  - *Superficial.* Great and small saphenous systems and tributaries.
  - *Deep.* Between the muscle compartments of the legs following the major arteries.
  - *Perforators.* Connecting the superficial and deep systems.
- Blood passes from the superficial to the deep systems via perforators in the calf, and also at the saphenofemoral junction (SFJ), saphenopopliteal junction (SPJ), and mid-thigh perforators (MTPs) which contain one-way valves.
- Varicose veins are tortuous and dilated segments of veins, associated with valvular incompetence.
- Affect 35% of the population.
- ♂ and ♀ almost equal prevalence.

Causes and features

Classification

- Thread veins. Intradermal dilated veins, also called 'flare', 'starburst', or 'broken' veins.
- Reticular veins. Subdermal 1–2mm diameter veins.
- Truncal veins. The great or small saphenous systems.
- Varicose veins. Usually arising from the truncal veins.
- Venous malformations. For example, congenital (Klippel–Trenaunay syndrome).

Causes

- Congenital.
- 1° idiopathic (the majority).
- Acquired.
  - Pelvic masses (e.g. pregnancy, uterine fibroids, ovarian mass, pelvic tumour).
  - Pelvic venous abnormalities (e.g. after pelvic surgery or irradiation, previous iliofemoral DVT).

Clinical features

- *Symptoms.* Pain, aching, itching, heaviness, swelling, oedema, worse at end of day/hot weather/premenstruation, cosmetic concerns.
- *Complications.* Eczema, phlebitis, lipodermatosclerosis, ulceration, or bleeding.

Diagnosis and investigations

- General history and examination. Oedema, eczema, ulcers (usually medial calf), lipodermatosclerosis, atrophie blanche, healed ulceration.
- Visible standing. Cough impulse, thrill, or saphenovarix at SFJ.
- Tap test. Tap downwards over vein from SFJ; impulse should be felt lower down if valves are incompetent (outdated and unhelpful).
- Trendelenburg test. For competence of SFJ, MTP, and SPJ (rarely used now that Doppler/duplex are available).

  - With the patient supine, elevate the leg, empty veins, apply tourniquet high in the thigh, and ask patient to stand.
- Look for venous filling and then release the tourniquet, observing filling of the veins.
  - If controlled by the tourniquet and then rapidly fill on release, the incompetent valve is above the level of the tourniquet, i.e. SFJ.
  - Then repeat twice with tourniquet just above knee and below knee to test the MTP and SPJ, respectively.
- Handheld Doppler (HHD). Listen over SFJ and SPJ, and apply calf compression with other hand and listen for reflux lasting 1–2s.
- Colour duplex. Defines anatomy and incompetence, and portable scanners can be used in clinics. Can be used for all or selectively for recurrent varicose veins, suspected small saphenous vein reflux, known or suspected previous DVT, and mismatch between clinical examination and HHD.

Treatment options

Medical

- Microsclerotherapy, laser sclerotherapy for thread and reticular veins.
- Foam sclerotherapy for truncal and varicose veins.
- Compression stockings.

Surgical

- Local 'stab' avulsions. Deals with varicosities.
- Great or small saphenous vein stripping.
- Thermal ablation (endovenous laser or RFA).
- Non-tumescent, non-thermal (mechanochemical ablation or glue).

Indications for treatment

- Cosmetic.
- For symptoms.
- To prevent complications.
- To reduce risk of recurrent complications.

Complications of intervention

- Bruising (virtually universal).
- Recurrence (50% cases at 10y).
- Haemorrhage (minor or, rarely, major from damaged femoral vein).
- Phlebitis following endovenous procedures.
- Wound infection (commonest in groin).
- Saphenous or sural nerve damage with paraesthesiae (20% numbness, 1% dysaesthesia) (surgery or thermal techniques).
- Damage to major arteries, e.g. femoral (rare).
- Thromboembolism.

Deep venous thrombosis

Causes and features

- Occurs due to abnormalities of the vein wall, blood flow, or constituents of blood (Virchow's triad).
- May be due to vein compression or stasis (immobility, trauma, mass, surgery, paralysis, long distance travel, including airline travel).
- May be due to inherited hypercoagulability (factor V Leiden, protein C, protein S, or antithrombin insufficiency).
- May be due to acquired hypercoagulability (surgery, malignancy, polycythaemia, smoking, hormone replacement therapy, OCP, dehydration).
- Severity may vary from isolated asymptomatic tibial/calf thrombosis to severe iliofemoral segment thrombosis with phlegmasia cerulea dolens (venous gangrene).

Clinical features

- Clinical manifestations may be absent.
- Local features of venous engorgement and stasis.
  - Limb swelling.
  - Pain.
  - Erythema and warmth to touch.
  - Mild fever and tachycardia result from release of inflammatory mediators.
  - H*oman's sign. Calf pain on dorsiflexion of the foot is very unreliable and* should NOT be performed.
- Complications.
  - PE.
  - Venous gangrene (phlegmasia cerulea dolens).

Diagnosis and investigations

Aim to confirm presence and extent of thrombosis (to decide on necessity and type of treatment, risk of embolization).

- D-dimer. If negative, DVT/PE is unlikely.
- Ascending venography. Rarely used now.
- Duplex scan. Investigation of choice; visualizes anatomy, gives extent of thrombosis, and relies on flow of blood and compressibility of vein. Operator-dependent and has lower sensitivity for calf DVT.
- V/Q scan. If suspicion of PE.
- *CTPA*. Most sensitive and specific investigation for suspected PE.

Treatment

- Effective prophylaxis is better than treatment (➲ Prophylaxis—antibiotics and thromboprophylaxis, pp. 98–9).
- Conservative measures. Elevation and good hydration.
- Uncomplicated DVT. LMWH, initially in hospital, may be as an outpatient via a dedicated DVT clinic. Subsequent treatment is with oral anticoagulation with warfarin for 3–6 months.
- Complicated DVT. Initially with IV UFH or LMWH, whilst converting to PO anticoagulation.

- Thrombolysis or surgical thrombectomy are reserved for severe thrombosis with venous gangrene.
- Vena caval filter. Percutaneously inserted via jugular or femoral vein into infrarenal IVC to catch thromboemboli and prevent PE.
  - Used for patients with recurrent PE despite treatment, at risk of major central PE and anticoagulation contraindicated, requiring urgent or major surgery (so cannot be anticoagulated), and major DVT with concomitant CNS injury or major fractures.
  - Risks include air embolism, arrhythmias, pneumo-/haemothorax, IVC obstruction, renal vein thrombosis, retained, misplaced, migrating, eroding, embolizing, or broken catheters/sheaths, complications of insertion (e.g. bleeding).

Chronic venous insufficiency Severe forms are often 2° to extensive or recurrent lower limb DVT (post-phlebitic limb).

Clinical features
- Leg/ankle oedema.
- Varicose/eczema, pigmentation, lipodermatosclerosis.
- Venous ulceration (medial commoner than lateral).
- Venous claudication (rare).

Assessment
- Many (80%) venous disease alone, others mixed with arterial disease.
- History of proven/suspected DVT is common.
- Ulcers present in many patients and 70% are recurrent.

Investigations
- ABPI measurement to assess for arterial disease
- Venous duplex to detect DVT or deep venous incompetence and to look for superficial venous disease.

Treatment
- Elevation, bed rest, and elevation of foot of bed.
- Four-layer bandaging (Charing Cross) if ulceration present and ABPI >0.85. Up to 75% ulcer healing at 12 weeks.
- Graduated compression hosiery (when ulcers healed).
  - Class I. Ankle pressure <25mmHg, prophylaxis.
  - Class II. Ankle pressure 25–35mmHg, marked varicose veins, and chronic venous insufficiency.
  - Class III. Ankle pressure 35–45mmHg, chronic venous insufficiency.
  - Class IV. Ankle pressure 45–60mmHg, lymphoedema.

Surgery
- Skin grafts (split skin and pinch skin grafts).
- Ulcer bed clearance of slough/infection (physical, chemical, larval—maggots, vacuum debridement).
- Surgery for superficial venous disease only (as for varicose veins surgery).
- Role in mixed superficial and deep venous disease is controversial.
- May need arterial revascularization.

Thrombolysis

Key facts

- Introduced over 30y ago.
- Use largely now restricted to DVT, recently occluded synthetic bypass grafts, thrombosed dialysis access, and thrombosed popliteal aneurysms.
- Usually administered as a low-dose intra-arterial infusion or as an adjunct to surgery intraoperatively.

Agents

- *Urokinase.* Expensive, rarely used in the UK other than for dialysis catheters.
- *Streptokinase.* Cheap, but has systemic effects and half-life of 27min. Has side effects of anaphylaxis, fever, and antibody resistance, limiting repeated use. Was widely used for treatment of MI.
- Recombinant tPA. Powerful clot affinity, lower systemic effects and bleeding complications, and half-life <6min.

Indications

- Treatment of acute limb ischaemia with viable limb due to *in situ* thrombosis or embolism not suitable for surgery or extensive distal (calf) thrombosis.
- Treatment of acute surgical graft complications, e.g. prosthetic graft occlusion <2 weeks' duration.
- Treatment of residual thromboembolic disease during reconstructive surgery (intraoperatively).
- Thrombosed popliteal artery aneurysm (allows clearance of distal calf vessels).
- Venous thrombosis (axillary/femoral) may be treatable by venous or arterial lysis, but need to balance risk of haemorrhage and stroke.

Regimen

- Administered via arterial catheter and simultaneous heparin via catheter sheath.
- Regular clinical assessment and coagulation checks are needed with clear protocols. Half-hourly temperature, pulse rate, and BP, as well as foot observations.
- Regular review with repeat angiography.
- ↑ complication rate after 24–36h of infusion.

Contraindications

- ↑ risk of bleeding (haemorrhagic disorders, current peptic ulcer, recent haemorrhagic stroke, recent major surgery, or multiple puncture sites).
- Evidence of muscle necrosis as may result in reperfusion syndrome and multiorgan failure.
- Urgent cases because threatened limb viability if lysis takes too long to work.

Complications

- *Minor.* Allergic, catheter problems (leak, occlusion), bruising, 15% risk of minor haemorrhage.
- *Major.* 5% risk of major haemorrhage or stroke.

Complications in vascular surgery

Complications may occur in the perioperative, early, or late post-operative periods. In general, vascular patients are older and have ↑ cardiac, cerebral, pulmonary, and renal comorbidities. This is due to the associated risk factors of HTN, DM, hypercholesterolaemia, and smoking.

General

Cardiac

(➲ Cardiac complications, pp. 130–2.)

- Atherosclerosis is a systemic disease with a predilection for the cerebral, coronary, peripheral arterial, and renal circulations.
- 40% of patients with PVD have at least two other circulations affected.
- 20% of patients undergoing non-cardiac vascular surgery have evidence of silent myocardial ischaemia.
- 70% of mortality associated with aortic surgery is attributable to perioperative cardiac dysfunction.

Pulmonary

(➲ Respiratory complications, pp. 134–5.)

- Worsened by pre-existing pulmonary disease, smoking, and obesity.
- Ensure adequate analgesia with PCA or epidural and good physiotherapy and early mobilization.

Haemorrhage

(➲ Post-operative haemorrhage, pp. 126–7.)

- *Perioperative.* Bleeding from uncontrolled blood vessels.
- Post-operative. May be due to breakdown of vascular anastomoses. Recognized by acute hypotension, shock, abdominal swelling, and pain. Return to the operating theatre.

Renal failure

- Many vascular patients have pre-existing renal impairment due to renovascular disease, drug treatments, or surgery.
- Acute perioperative risks include dehydration, use of IV contrast, use of NSAIDs, or nephrotoxic antibiotics.

Infection

- Wound infections reduced by prophylactic antibiotics.
- MRSA easy to prevent (hand hygiene), but difficult and expensive to treat.
- Clostridium difficile enteritis (prevented by handwashing) associated with antibiotic mis-/overuse in inpatients.

Specific

Post-declamp shock (reperfusion syndrome)

- Ischaemic extremities during surgery reperfused into circulation.
- Features of acute haemodynamic instability due to release of toxins (K+, myoglobin).
- Prevented by controlled gradual reperfusion, with fluid resuscitation and vasopressor treatment to maintain a good perfusion pressure to the coronary, cerebral, and renal circulations.
- Mannitol often used as a free radical scavenger.

Trash foot
- Embolization of debris to the skin of feet or buttocks after aorto-iliac surgery.
- Avoided by careful surgical technique and distal vessel clamping first.

Swollen limb
- Most are due to reperfusion injury of previously ischaemic limbs.
- Consider investigations for DVT.

Lymphocoele
- Occurs mostly after groin surgery.
- Presents as a fluctuant, non-tender swelling.
- Most will settle spontaneously, although larger collections may be aspirated under **strict** aseptic conditions.
- Rarely requires further surgery to oversew the lymphatics.

Mesenteric ischaemia
- May follow aortic surgery (ischaemic colitis); 2.5% after ruptured AAA surgery and <1% of elective aortic aneurysm surgery due to loss of gut blood supply.
- May present as vague abdominal pain or bloodstained diarrhoea.
- Sigmoidoscopy usually confirms the diagnosis.
- If there is no evidence of peritonitis, then fluids to rehydrate and close observation are required.
- If there is evidence of peritonitis, then an urgent laparotomy is needed with resection of the ischaemic bowel.

Impaired sexual function
- Due to damage to the peri-aortic or hypogastric plexus and underlying vascular disease of the blood supply to the pelvis.

Late complications

Graft occlusion
- Early failure (<30 days). Technical cause; recognized by acute deterioration in symptoms or acute limb ischaemia.
- Late failure. Usually due to intimal hyperplasia, continued smoking, or disease progression; recognized by progressively worsening symptoms, falling ABPI, or duplex scanning showing graft stenosis.

False aneurysm
- Usually 2° to infection or, occasionally, fatigue of graft material (long term). Further surgery is usually required.

Graft infection
- Mostly gut bacteria or coagulase-negative staphylococci; MRSA an increasing problem.
- Can be minimized by prophylactic antibiotics, meticulous technique, and infection control policies on vascular wards.
- Once infected, the graft usually has to be removed and alternative reconstruction required.
- Recognized by signs of low-grade or chronic sepsis (↑ CRP, ↓ Hb, fever).

Aortoenteric fistula

- Rare, usually fatal if not treated.
- Usually follows aortic grafting. Can be years later.
- Presents with small, often overlooked, GI bleeds or anaemia.
- Diagnosis difficult. CT can be helpful; often all other causes of bleeding are negative.
- Treatment by open surgery or endovascular exclusion and antibiotics.

Chapter 20

Transplantation

Basic transplant immunology

Humans have adapted the immune system over generations in order to identify and destroy any 'non-self' cells, which include own cells that have become abnormal. The human leucocyte antigen (HLA) is a cell surface protein that allows recognition of self, and this protein is encoded on an area of the genome called the major histocompatibility complex (MHC). The immune system broadly consists of two major immune responses: (1) a non-specific innate response that involves components that directly recognize and destroy pathogens (e.g. complement system), and proteins that make pathogens more easily recognizable for destruction by immune cells such as neutrophils and macrophages; (2) a targeted adaptive immune response which is more specific and consists of cell-mediated (T-cells) and antibody-mediated (B-cells) immunity. The immune system represents the major biological hurdle in transplantation.

In transplantation, for an allograft to be rejected, the recipient's immune system recognizes the transplanted organ as foreign (allorecognition) and delivers an effector response by which the graft can be destroyed (alloresponse). The 1° role of tissue typing is to establish the ABO blood group and determine the HLA expression of transplant recipients in advance of transplantation.

Antigens

There are two main types of antigens (important in transplantation) that can be recognized for an immune response:

- ABO blood group antigens—A, B, O, and AB. If a patient has a particular blood group antigen, then they are likely to have antibodies to the other blood groups. For example, patients with blood group A will have anti-B antibodies. Transplantation of an ABO-incompatible organ will lead to rapid destruction of the organ, mediated by antibodies against the incompatible blood type.
- HLA antigens—the major target of the immune response in transplantation. There are two classes of HLA: class I and class II. There are a total of six main subgroups of HLA antigens—class I consists of A, B, and C, and class II consists of DP, DQ, and DR. Each subgroups consists of lots of variations, and each individual has two copies of each HLA gene. In kidney transplantation, HLA-A, -B, and -DR have been shown to be the most important antigens in determining outcomes and therefore are the ones used when reporting mismatch between donors and recipients. The maximum number of mismatches possible are six, reported as 2-2-2, and the minimum number is 0, reported as 0-0-0. The greater the number of mismatches, the higher the chances of rejection and poorer outcomes.

Allorecognition

Allorecognition is the identification of antigen as 'non-self'. It is a function mainly of the adaptive immune response but is dependent on priming by an inflammatory response from the innate immune system; such a response occurs in transplantation, both from the inflammatory response to surgery and ischaemia–reperfusion injury in the graft.

Allorecognition depends on two main processes. First, alloantigen must be taken up and complexed with surface HLA molecules on antigen-presenting cells (APCs) that include dendritic cells and macrophages. Second, antigen-specific host T-cell receptors must bind to the peptide fragments of alloantigen complexed with the HLA molecules on APCs. This T-cell binding only activates a response if there is also co-stimulatory signal from the APC.

Alloresponse

Alloresponse refers to the overall immune response which includes the following three main effector pathways:

1. Cellular cytotoxic response (mediated by CD8+ cytotoxic T-cells that target non-HLA-specific graft cells).
2. Humoral response (mediated by alloantigen-specific B-cells that have been activated by CD4+ T-cells, leading to production of antibodies, which, in turn, damage the graft predominantly by activating the complement cascade).
3. Delayed hypersensitivity reaction (mediated by CD4+ T-cells that cause an inflammatory response through activation of macrophages and production of pro-inflammatory cytokines).

B- and T-cell diversity

Lymphocytes are able to identify non-self by the presence of both T-cell receptors (TCRs) and B-cell receptors (BCRs) specific for each alloantigen. There are millions of potential combinations of thousands of TCR and BCR gene segments, allowing the production of a huge number of receptors, each specific to a particular peptide fragment.

A basic glossary of terms in transplant immunology

- Adaptive immunity. Learnt response to specific non-self antigens.
- Allo-. Tissue from genetically different members of same species. Alloantigen. Antigen from genetically different members of same species. Allogenicity. Ability of tissue to provoke an immune response when transplanted into genetically different member of same species. Allorecognition. Recognition of alloantigen as non-self.
- *Antigen.* Cell surface glycoproteins.
- *Antibody.* Specific proteins produced by B-cells in response to non-self antigen, consisting of two light and two heavy chain proteins composing a constant and a variable region. The variable chain region binds with the antigen that triggered the response and the constant region coordinates the cellular response.
- APCs (antigen-presenting cells). These cells ingest and process antigen, then present it bound to surface MHC to T-cells. APCs are most commonly dendritic cells or macrophages.
- B-cells. These cells mature in bone marrow and primarily produce antibodies. In response to antigen, they undergo clonal expansion, triggered and coordinated by T helper cells.
- Cellular immunity. Adaptive immunity mediated by lymphocytes.
- *CD.* Cellular differentiation molecule (followed by a number, e.g. CD4).

- Clonal expansion. Production of large numbers of identical cells.
- Co-stimulatory molecules. Molecule receptors that must be activated, in addition to a main receptor, for a process to happen.
- Direct recognition. Donor APCs present alloantigen to host T-cells.
- HLA (human leucocyte antigen). Human cell surface protein that allows recognition of self, encoded by the MHC.
- Humoral immunity. Immunity mediated by non-cellular components of the immune system such as antibodies or complement.
- Indirect recognition. Host APCs present alloantigen to host T-cells.
- Innate immunity. Rapid inbuilt response to certain non-self proteins.
- MHC (major histocompatibility complex). Glycoproteins expressed on the surface of all cells, coded for by the MHC genes on chromosome 6. Alloantigen must be bound to MHC for T-cells to recognize it. There are two classes of MHC. Class I (HLA molecules A, B, and C) is present on all cell membranes. Class II (HLA molecules DP, DQ, and DR), also known as minor histocompatibility complex, is less allogeneic and is present on only certain cell types.
- *Rejection.* Injury to tissue by host immune response.
- T-cells. These cells mature in the thymus. T-cells that bind to thymic tissue (self) are destroyed. T helper cells that are CD4 +ve coordinate and T cytotoxic cells (CD8 +ve) carry out the response. Regulatory T-cells are also CD4 +ve and reduce the alloreactive response.
- *Tolerance.* A condition of lack of immune response to alloantigen where such a response is expected or has occurred previously.
- *Xenograft.* Tissue transplanted between species.

Immunosuppression and rejection

The goal of immunosuppression is to inhibit the immune response to alloantigen, while preserving the immune response to infection and malignancy. Immunosuppression consists of a short, intense induction phase, followed by a maintenance phase in which immunosuppression dosage is tapered. A careful balance is maintained between therapeutic and toxic doses of immunosuppression; this is generally achieved by combining drugs with different mechanisms of action (see Box 20.1).

Rejection

There are several types of rejection:

- Hyperacute rejection is mediated by preformed antibodies that bind to antigens of ABO blood groups (non-self HLA), causing immediate tissue oedema, haemorrhage, and thrombosis, which can produce a severe systemic reaction like an ABO-incompatible blood transfusion. This should never occur in transplantation due to cross-matching.
- Acute rejection is mediated either by T-cells ('cellular rejection') or newly formed antibodies ('humoral rejection') and results in tissue destruction over days to many months after transplant.
- Chronic rejection is a poorly understood vasculopathy associated with fibrosis of small blood vessels that occurs over years. It is a chronic inflammatory condition which probably has an alloreactive component.

Acute rejection

Diagnosis

Clinical features depend on the organ transplanted and are generally manifested by biochemical evidence of impaired organ function or organ injury (such as elevated transaminase levels in liver transplantation or elevated Cr in kidney transplantation). Systemic immune symptoms are less common, and patients with rejection are often asymptomatic but can present with low-grade fever, malaise, and tenderness over the graft. Diagnosis is made by biopsy, which will also determine whether rejection is cellular or humoral and can identify other causes of graft dysfunction.

Immunosuppression may mask symptoms until rejection is quite advanced, so routine surveillance may be undertaken, especially for those organs where loss of function would be catastrophic. When required, optimal surveillance is by 'protocol biopsies' taken at pre-determined time points such as at 7–10 days after transplantation, with repeat biopsies taken at intervals. Percutaneous imaging-guided biopsy is typically used in liver and renal transplantation, endoscopic biopsy in small bowel and lung transplantation, and transjugular biopsy in heart transplants or liver transplants with uncorrectable coagulopathy.

Management

- Asymptomatic mild rejection may be monitored in some organs but is always treated in renal transplants, as kidneys are very immunogenic.
- The main form of treatment is ↑ immunosuppression.
- Up to 3 days of IV methylprednisolone 500–1000mg/day is typically given as initial treatment for acute rejection.
- Repeat biopsy is performed if no improvement, followed by repeat steroid course if ongoing rejection, or give rescue therapy if severe.

Box 20.1 Immunosuppressive agents

Corticosteroids

Corticosteroids inhibit the immune response at many levels. They decrease production of γ-interferon and ILs that would normally cause upregulation of the lymphocyte response and reduce macrophage function. Side effects include Cushingoid features such as centripetal obesity, thin skin, proximal myopathy, etc.).

Calcineurin inhibitors (CNIs)

Ciclosporin and tacrolimus are CNIs—they inhibit the production of IL-2 by T helper cells, selectively reducing the cytotoxic T-cell response. CNIs are also associated with tremor, which is usually dose-dependent. Nephrotoxicity is a major side effect of CNIs, but they also impair glucose tolerance and ciclosporin is associated with gingival hypertrophy. Tacrolimus is the preferred CNI choice, as it has a better side effect profile.

Mammalian target of rapamycin (mTOR) inhibitors

Sirolimus (rapamycin) and everolimus both inhibit the production of IL-2 by T-cells and thus stop their clonal expansion in a manner similar to CNIs. mTOR inhibitors are not themselves nephrotoxic, but they appear to potentiate the nephrotoxicity of CNIs if given together.

Anti-proliferative agents

These include mycophenolic acid and azathioprine. Mycophenolic acid inhibits purine synthesis in lymphocytes, reducing clonal expansion and lymphocyte counts. Mycophenolic acid comes in two forms—mycophenolate mofetil which can cause severe GI side effects; and mycophenolate sodium which has fewer GI side effects.

Azathioprine reduces lymphocyte production by suppression of purine synthesis. Allopurinol inhibits the metabolism of azathioprine. Bone marrow suppression and pancreatitis are common adverse reactions. Mycophenolic acid is preferred to azathioprine.

Basiliximab

Monoclonal antibody that binds to the IL-2 receptor of T-cells and thus prevent clonal expansion of T-cells. Usually given as induction agent.

Anti-thymocyte globulin (ATG)

Derived from rabbits or horses immunized with T-cells; primarily directed against the T-cell receptor. Systemic inflammatory reactions occur, which essentially depletes T-cells, and is given either as an induction agent or as treatment for severe rejection.

Alemtuzumab

Monoclonal antibody binding to CD52 receptor, leading to depletion of T-cells, B-cells, NK cells, lymphocyte precursors, dendritic cells, and macrophages. It thus reduces all the cells involved in antigen presentation, cellular rejection, and humoral rejection. Usually used as an induction agent. Side effects include bleeding and sepsis.

Belatacept

A new drug which blocks co-stimulation of T-cells by APCs. Not nephrotoxic or diabetogenic. Long-term effects are unknown and trials are still under way.

- Rescue protocols include administration of anti-T-cell antibodies such as anti-thymocyte globulin (ATG).
- Plasmapheresis to remove antibodies is often required in humoral rejection, diagnosed by specific histological features and CD4 positivity in the biopsy.
- Re-transplantation is occasionally performed after graft loss due to refractory rejection but is not performed for certain organs, such as the heart and lung, as results are extremely poor.

Chronic rejection

Chronic rejection is a chronic inflammatory process associated with intimal hyperplasia with dependent ischaemia and fibrosis. The cause is unknown but may be related to the severity of acute inflammatory processes at the time of transplantation such as ischaemia–reperfusion injury and acute rejection. There is no specific treatment available for chronic rejection at the present time, with re-transplantation as the only possible effective option.

The term 'chronic rejection' is often interchanged with other terminology, according to the affected organ, such as chronic allograft nephropathy (kidney) or cardiac allograft vasculopathy (heart).

Side effects of immunosuppression

Side effects of immunosuppression can be related directly to the drugs (see Box 20.1) or due to the dampened immune response caused by them. In general, dampening the immune response leads to more severe infection and the presence of opportunistic infection (similar to that seen in any immunosuppressed patient). These infections can be viral, bacterial, or fungal.

Opportunistic infection

Cytomegalovirus (CMV) is one of the commonest infections seen post-transplant. It can present with non-specific symptoms, such as malaise, lethargy, and fever, or affect specific organs such as the GI tract (CMV colitis) or the lungs (CMV pneumonitis). Diagnosis is made by detection of CMV-PCR DNA copies in the serum and/or evidence of CMV in biopsy samples from the affected organ. CMV infection can be potentially life-threatening and treatment is by PO valganciclovir or IV ganciclovir for severe infections. Prophylaxis for CMV is given to transplant recipients (based on their and the donor's CMV status) for the first 3–6 months post-transplantation (or following a rejection episode), in the form of PO valganciclovir (dose being dependent on eGFR).

Pneumocystis jiroveci is a fungal infection that leads to potentially life-threatening atypical pneumonia. Patients often present with a dry cough and shortness of breath, with minimal CXR signs at presentation, but rapidly deteriorate. Diagnosis is made by bronchoalveolar lavage (BAL) and treatment is with high-dose co-trimoxazole (or occasionally second- or third-line agents). Prophylactic PO co-trimoxazole is given for the first 6 months post-transplantation.

BK virus infection is generally asymptomatic but is a possible cause of failure of renal transplants.

Malignancy

Immunosuppression reduces immune surveillance of tumours, so some are commoner in transplant recipients and others can be more aggressive than would otherwise be the case. Virally mediated tumours and SCC of the skin are especially common.

Post-transplant lymphoproliferative disorder (PTLD) is a condition of B-cell proliferation which may extend to B-cell lymphoma and is due to inhibition by immunosuppressant drugs of the IL-2-dependent mechanism by which T-cells regulate the B-cell proliferation occurring in EBV infection.

Although not a complication of immunosuppression per se, malignancy can also be transmitted from transplant donors to recipients. The risk of this is very small for standard donors (~1 in 2000) but should be considered in unusual cases.

Transplant recipients

Indications for transplantation

Cardiac transplantation

- End-stage heart disease, with a life expectancy of 12–18 months.
- NYHA class III or IV heart failure, refractory to medical or surgical therapy.
- Cardiomyopathy and congenital heart disease.

Lung transplantation End-stage lung disease where conventional therapy is not likely to provide acceptable benefits or satisfactorily improve life expectancy.

Renal transplantation All patients with end-stage renal failure should be considered for renal transplant, unless there are specific contraindications.

Liver transplantation

- Unacceptable quality of life because of liver disease.
- Anticipated 1y mortality >9% without transplant.

Pancreas transplantation

- Usually with or after kidney transplantation for diabetic nephropathy.
- Can be done alone for hypoglycaemic unawareness (consider islets).

Small bowel transplantation (often part of multivisceral)

- Congenital extensive atresia.
- Life-threatening complications of TPN in patients with intestinal failure.
- Loss of central venous access sites in patients with intestinal failure.

Absolute contraindications to transplantation

- Inability to comply with immunosuppression.
- Chronic current systemic infection.
- Irreversible 2° organ failure not appropriate for combined transplant.
- Severe cerebrovascular disease.
- Active untreated malignancy.
- Uncontrolled HIV/AIDS.
- Untreated TB.
- Alcohol or drug addiction.

Relative contraindications to transplantation

- Predicted life expectancy <5y due to comorbidity.
- COPD with forced expiratory volume in 1s (FEV1) <50%, PVR >4 Wood units in heart transplants.
- Chronic renal impairment with GFR <50mL/min, unless candidate for renal transplant, including combined transplants with kidney.
- Diabetes with target organ damage (heart).
- Continued abuse of alcohol or other drugs.
- Lipid disorders refractory to diet or therapy (heart).
- Severe osteoporosis.
- Continued smoking (heart or lung).
- Severe PVD.

Routine investigations in transplant assessment

- A full history and clinical examination.
- CXR, ECG.
- Functional cardiopulmonary assessment if indicated, e.g. CPEX.
- Cardiac catheterization and coronary angiography if indicated.
- Lung function tests.
- MSU, urinalysis, nose swab, and MRSA screen.
- Blood group antibody screen.
- FBC and coagulation profile.
- U&Es, Ca^{2+}, PO_4, LFTs, fasting blood glucose, and lipids.
- Serology for hepatitis B/C, HIV, syphilis, rubella, EBV, herpes, varicella and zoster, CMV, Toxoplasma.
- HLA typing, lymphotoxic antibody screen.
- Assessment of compliance; may include interview with social worker.
- Further organ-specific tests as indicated.

Accepting a patient onto the transplant list

Accepting a patient for transplantation is a multidisciplinary process which varies from organ to organ. Potential kidney and/or pancreas transplant recipients are typically initially assessed by their nephrologist and then referred to a transplant surgeon. Potential recipients of liver, small bowel, heart, or lung transplants are usually discussed in a multidisciplinary meeting of transplant surgeons, anaesthetists, physicians, transplant coordinators, and specialist nurses. The patient is informed of the decision and receives a detailed explanation of the waiting list procedures (including their responsibility to be within contact and available for potential transplant at all times and duty to inform the transplant team of any changes in their health; planned holidays can be permitted by temporary suspension from the list).

Organ donation and allocation

Availability of organs for transplantation

The increasing success of organ transplantation as a modality of treatment for end-stage organ disease has ↑ demand, a problem further exacerbated by widening the criteria for which patients can be considered suitable candidates for transplantation. This has led to an increasing national shortage of organs and efforts are being innovated continuously to increase donation. The legal framework of organ donation for transplantation is regulated by the 'Human Tissue Act 2004' in England and Northern Ireland, 'Human Tissue Act (Scotland) 2006' in Scotland, and the 'Human Transplantation (Wales) 2015 Act' in Wales. These Acts support the 'Opt in' system in which a person actively registers to be on the organ donor register. In Wales and England, the law changed in December 2015 and May 2020, respectively, to bring in an 'Opt out' system, in which an individual who does not want to be considered for organ donation must actively register to 'opt out' of the organ donor register.

Allocation of organs is based on principles that determine a fair division of a limited source. Criteria used for allocation and selection of recipients are based on a combination of several factors, which include: (1) fairness (in which those waiting the longest are allocated the organs first); (2) medical condition (the organ should be given to those in the greatest need; and (3) utility (allocation in such a way that the greatest number of organ life-years are achieved).

Deceased donation

Deceased donors form the majority of organ donors in the UK. Traditionally, the majority have been heart-beating donors after brainstem death (DBD), but with advances in immunosuppression, there is an ever increasing number of non-heart-beating donors after circulatory death (DCD) being used. In addition to this, donors who were considered 'marginal' in the past, including extended-criteria kidney donors (defined as donors over age 60 or donors aged 50–59 with at least two of the following three medical criteria: history of HTN, final pre-procurement Cr above 1.5mg/dL, and CVA as cause of death) are being increasingly used.

Most DCD are controlled donors where life-prolonging treatment is withdrawn after a decision that the overall prognosis means that such treatment is felt to be futile. As such donors are living patients until the time of cardiac arrest and generally lack capacity to consent due to being unconscious, they can only be treated in line with their best interests under common law, restricting the interventions possible to optimize the condition of the transplanted organs.

Potential donors undergo a review of their history and clinical examination, ECG, CXR, ABGs, ABO typing, testing for HIV, hepatitis C/B, and CMV, and tests of organ function (e.g. U&Es, LFTs, echocardiography).

Criteria for cadaveric organ donation

1. Consent (opt in—agreement from next of kin, or deemed consent in Wales).
2. Death certification (via brainstem testing for DBD donors—see Box 20.2; or by neurological assessment for DCD donors).

3. Absence of absolute contraindications: age ≤85; 1° intracerebral lymphoma, all 2° intracerebral tumours; any active cancer with evidence of spread outside affected organ within 3y of donation; melanoma—except completely excised stage 1 cancers; active haematological malignancy; active and untreated TB; definite, probable, or possible case of human transmissible spongiform encephalopathy (TSE), including Creutzfeldt–Jakob disease (CJD) and variant CJD (vCJD); HIV disease (but not HIV infection); and West Nile virus. This list is continually reviewed and updated on the NHS Blood and Transplant (NHSBT) website.

- Organ-specific criteria include the following.
  - Kidney. Donor with acceptable renal function.
  - Heart. Age 1 month to 60y, with no known cardiac disease.
  - Heart–lung. As above; no pulmonary disease or trauma; pO2 and pCO2 levels acceptable on <50% inspired O_2.
  - Liver. No known liver disease, drug addiction, or hepatitis B.
  - Pancreas. Age 10–65y with no diabetes.

Principles of cadaveric organ retrieval

The two fundamental principles of organ retrieval are: (1) to minimize the period of warm ischaemia as much as possible; and (2) to retrieve the organs without any damage. Retrieval of multiple organs is common, so a coordinated approach is needed. Inotropic, volume, and respiratory support is continued until the retrieval teams are ready to start cold perfusion.

- A midline incision from sternal notch to pubis is made.
- A diagnostic laparotomy is performed to assess for any undiagnosed disease, especially malignancy.
- Organs are carefully examined for evidence of trauma and disease. The aim is to retain adequate vascular and visceral cuffs to facilitate later anastomosis. Anatomical variants, especially the vascular supply to the liver, are not that uncommon and can affect retrieval of the liver and pancreas, so these must be carefully assessed.
- As soon as both the abdominal and thoracic teams have completed their assessment and are ready for cold perfusion, IV heparin 200U/kg is given; the supracoeliac aorta is cross-clamped, the ventilator stopped, cold perfusion established through aortic cannulae, and ice slush poured into the abdomen and thorax. The organs are then dissected out and removed once cold.

In DBD donors, the heart is beating and the organs are perfused with oxygenated blood until procurement. Retrieval of organs from DCD donors is different as cardiac arrest has already occurred, so the first priority is to start cold perfusion, with assessment of anatomy and disease done in the cold phase prior to organ retrieval. DCD donors therefore typically have a warm ischaemia time of around 10–20min, whereas DBD donors do not.

Organ preservation

Organ preservation predominantly relies on use of hypothermic temperatures (of around 4°C), the principle of which is to reduce cellular metabolism and O_2 requirements, in order to reduce tissue injury and maintain graft viability. Despite this, there is still 'cold' ischaemic injury and cold ischaemic times must also be kept to a minimum for best outcomes. The organs are preserved in special 'preservation solutions', which contain

appropriate buffers and balanced electrolyte composition (designed to prevent cellular swelling). Two categories exist—extracellular solutions characterized by high Na+ and low K+, such as Bretschneider (HTK), and intracellular solutions characterized by high K+ and low Na+ such as University of Wisconsin solution (UW), considered the 'gold standard' solution of choice.

Box 20.2 Brainstem death testing (UK code)

Essential preconditions

The patient must be in an apnoeic coma, i.e. unresponsive and dependent on a mandatory ventilation mode, following irreversible structural brain damage due to a 'disorder which may cause brain death'.

Drug intoxication, hypothermia, and metabolic or endocrine disturbances must be excluded before brainstem death testing, as these may mimic brainstem death clinically.

Apnoea test

Confirmation of apnoeic coma is performed by ventilator disconnection, with lack of spontaneous respiratory effort despite rising arterial CO_2 tension. Care must be taken to avoid hypoxia which could potentially cause further brain injury in a patient who might not yet be brainstem-dead.

- Ensure systolic BP >90mmHg and adequate intravascular volume.
- Pre-oxygenate with 100% O2 for 10min.
- Check PaCO2 is at least 5.3kPa (40mmHg). If not, give 95% O2/5% CO2 until PaCO2 >5.3kPa.
- Disconnect ventilator and insufflate 100% O2 at 6L/min via an intratracheal catheter passed to the level of the carina.
- Continue disconnection until PaCO2 >6.65kPa (50mmHg), which should occur within 8min.

Apnoea testing should be abandoned if there are cardiac arrhythmias, hypotension, or arterial desaturation. In the UK, apnoea testing must be attempted again before brainstem death is diagnosed.

Clinical tests

The brainstem reflexes should be tested by two experienced doctors, one of whom must be a consultant and neither of whom is a member of the transplant team. They may perform the tests together or independently and must repeat all the tests after a period of at least 2h.

- No pupillary response to light (both direct and indirect reflexes).
- No corneal reflex.
- No vestibulo-ocular reflex. No eye movement on irrigation of tympanic membrane with 20–50mL of ice-cold water.
- No cranial nerve motor responses, e.g. grimacing to pain.
- No gag or cough reflex on deep bronchial suctioning.
- No oculocephalic reflex ('doll's eyes test').

Different methods of organ preservation are used:

1. Cold static storage. Commonest and easiest method which involves storage of organ immersed in preservation solution and packing in ice.
2. Hypothermic machine perfusion. This involves placing the organ on a machine (such as Lifeport), with continuous flushing of preservation solution through the vessels at a low temperature (of around 4°C). This is commonly used for DCD and expanded criteria donor (ECD) kidneys.
3. Normothermic preservation. In recent years, there has been a significant development in normothermic perfusion techniques which restore cellular metabolism and aim to minimize 'cold' ischaemic injury. Normothermic perfusion allows assessment and potential treatment of ischaemic injury prior to implantation and has been particularly successful for marginal organs.

Living donors

These are generally relatives or genetically unrelated, but emotionally connected, individuals (mostly commonly spouses), though undirected living donation to complete strangers is also allowed ('altruistic donation'). Living donation requires meticulous preparation to minimize risk to the donor and exclude coercion or financial reward; potential altruistic donors must also be psychologically assessed.

Kidneys are the commonest transplants from living donors. Donation of a liver lobe is also possible due to the large functional reserve of the liver and its ability to regenerate by hypertrophy; left lobe liver donation from adults to children is especially common.

Living donation of lung lobes is also possible but requires two donors for each recipient. Living pancreas donation using distal pancreatectomy has been described but is not widely used due to potential risks to the donor of diabetes or pancreatic duct leakage.

- All donors undergo blood grouping, tissue typing, and assessment of viral status for hepatitis B/C, HIV, and CMV.
- Tests of organ function, such as isotope split GFR for kidneys, are needed to ensure adequate post-operative function for both donor and recipient.
- General tests of donor fitness are also essential to minimize risk.

ABO-incompatible living donors

(See Table 20.1.) Although ABO incompatibility is normally an absolute contraindication to transplantation as the preformed antibodies will lead to hyperacute rejection, it is possible to desensitize the potential recipient by a preoperative course of plasma exchanges or immunoadsorption to remove the antibody, preceded by an infusion of the anti-B-cell antibody rituximab to prevent antibody regeneration. This treatment is only feasible for living donor transplants, as these are planned operations. Similar treatment can also be given to desensitize patients with preformed antibodies directed against the HLA type of their potential donor (such antibodies can be formed after sensitizing events such as previous transplants, pregnancy, or blood transfusions).

Table 20.1 ABO compatibility for transplants

Recipient blood group	Donor blood group			
	A	B	AB	O
A	Yes	No	No	Yes
B	No	Yes	No	Yes
AB	Yes	Yes	Yes	Yes
O	No	No	No	Yes

To prevent resensitization, care must be taken to avoid accidentally transfusing anti-ABO antibodies when administering blood products after the transplant. Red cell and platelet transfusions should use washed cells of recipient blood group. FFP and cryoprecipitate transfusions must be donor-type if the transplant is donor group A or B to recipient group O or type AB if the transplant is between group A and B; alternatively, recipient-type blood products can be used if screened for low antibody activity.

Where recipient desensitization is not possible, paired exchange may be an alternative where the donor from each pair donates to the recipient of the other pair. Paired exchange requires a large pool of donor–recipient pairs to be successful and is only feasible for kidney transplants.

Heart and lung transplantation

Cardiac transplantation

Assessment for transplantation

- Functional capacity: determine NYHA classification and measurement of maximal O_2 uptake by cardiopulmonary exercise testing (CPET).
- Pulmonary circulation: measurement of PAP, PVR, and transpulmonary pressure gradient (TPG). PAP >60mmHg, PVR >5 Wood units, and TPG >15mmHg are contraindications.

Matching donor to recipient

- ABO compatibility. Donor and recipient must be ABO-compatible; hyperacute rejection occurs in ABO-incompatible patients. Children under 1y can be transplanted despite ABO incompatibility.
- HLA typing. Although the heart is among the least allogeneic organs and an HLA mismatch is not a contraindication to transplantation, HLA-A2 or -A3 mismatch has been associated with chronic rejection and some centres choose to avoid this.
- Size match. Important.

Technique of transplantation

Orthotopic heart transplantation involves transplanting the donor organ into the space vacated by the recipient heart. There are several techniques of orthotopic heart transplantation.

- The most commonly used is the bicaval anastomosis technique. The donor cavae are attached directly to the recipient cavae. This results in less tricuspid regurgitation and better haemodynamic perfomance.
- In the original technique, the RA and LA of donor and recipient are preserved; anastomosing atria to atria is technically less demanding than bicaval anastomosis.
- In the total anastomosis technique, each pulmonary vein is individually anastomosed.
- In heterotopic transplantation, used in 2.5% of heart transplants, the donor heart is retained and the transplanted heart is anastomosed so that it acts to bypass the left heart. The technique is reserved for severe pulmonary HTN.

Post-operative care Monitoring for rejection is done via transvenous endomyocardial biopsy.

Complications

- Early: arrhythmias, bleeding, RV failure, LV dysfunction, CPB-related dysfunction.
- Rejection and graft IHD.
- Hyperlipidaemia and diabetes 2° to immunosuppression.
- Renal failure (similar risk factors to heart failure, perioperative hypoperfusion, nephrotoxic immunosuppression regimes).
- *HTN.* Aetiology poorly understood.
- *Malignancy.* Decrease in T-cell response to EBV as a result of immunosuppression.

Results of cardiac transplantation

- 1y survival is 81%; annual mortality of 4%.
- Half-life for survival is 10y.

Lung transplantation

Indications

- ↑ (>50%) risk of death from lung disease within 2y.
- >80% probability of surviving 90 days after lung transplantation.

Matching donor to recipient

- ABO compatibility. Donor and recipient must be ABO-compatible; hyperacute rejection occurs in ABO-incompatible patients.
- HLA typing. Although an HLA mismatch is not a contraindication to transplantation, improved graft survival is associated with matching HLA-B, HLA-A, and HLA-DR loci.
- Size match. Important.

Technique of transplantation

- Single-lung transplant is performed where the remaining native lung will not compromise graft function or present a hazard; emphysema, asthma, and sarcoid require single-lung transplants.
- Double-lung transplants are performed via a clam-shell incision for CF and bronchiectasis.

Post-operative care

Early post-operative management centres around maintaining a balance between adequate perfusion and gas exchange, whilst minimizing fluid load, cardiac work, and barotrauma. Monitoring for rejection is done by transbronchial biopsy and BAL.

Complications

- *Technical.* Related to bronchial, pulmonary artery, and pulmonary vein anastomoses; phrenic nerve injury (diaphragm paralysis).
- 1° graft dysfunction.
- Cardiovascular complications, e.g. arrhythmias.
- Infections (nosocomial, opportunistic, or acquired). Infection with Pseudomonas spp. is common in CF patients. CMV infection is dangerous.
- Immunological (e.g. rejection).
- Stenoses: arterial stenosis results in pulmonary oligaemia and venous stenosis in pulmonary oedema.
- Tracheal ischaemia may result in leak and mediastinitis.

Results of lung transplantation

- 1y survival is 78%.
- 5y survival is 51%.

Kidney transplantation

Kidney transplantation is the commonest form of organ transplant. A total of 3042 kidney transplants were performed in the UK in 2016–17, of which 1218 were DBD, 887 DCD, and 937 living donor. Despite this number of transplants, there are still around 6000 patients on the transplant waiting list.

Matching donor to recipient

- ABO compatibility. Donor and recipient must normally be ABO-compatible; hyperacute rejection occurs in ABO-incompatible patients, unless desensitization treatment has been performed preoperatively.
- HLA typing. Graft survival is better if there is no more than one mismatch for HLA-A and/or HLA-B and no mismatches for HLA-DR.
- *Children.* Given priority; even small children can receive adult kidneys.

Technique of transplantation

The kidney is normally placed extraperitoneally into the iliac fossa. Both the left and right kidneys can be placed into either iliac fossa. The renal vessels are anastomosed to the external iliac vessels. The common or internal iliac artery can be used if the external is diseased.

- The ureter is anastomosed to the bladder, usually over a stent.
- Preoperative native nephrectomy is only occasionally needed for continued/recurrent urinary infection, TB of the kidney, or massive polycystic kidney disease.

Post-operative care

Early post-operative management centres around maintaining a balance between adequate renal perfusion and BP control.

- Graft function is monitored by serial Cr measurements.
- Early graft failure is usually due to lack of perfusion following an arterial or venous thrombosis, so graft perfusion should be assessed by Doppler ultrasound if there is no immediate graft function.
- Delayed graft function with oliguria or anuria is common early after transplantation, especially if there has been ischaemic injury prior to organ retrieval or a prolonged cold ischaemic time.
- Intermittent haemodialysis may be needed if there is delayed function.
- Polyuria is common once the kidney starts to function until renal tubular function recovers; fluid needs to be replaced to prevent pre-renal failure of the graft.
- Biopsy to confirm suspected rejection is done percutaneously under ultrasound guidance.

Complications

- Infection.
- Rejection (➲ Immunosuppression and rejection, p. 814).
- Renal vein or artery thrombosis may result in loss of the kidney.
- Ureteric stenosis. Treated by ureteroplasty and a stent or surgery.
- Urinary leak often can be managed by urinary catheterization for 6 weeks, followed by cystogram to confirm healing. Re-implantation of the ureter may be required.
- Lymphocele is managed by percutaneous drainage or by laparoscopic or open marsupialization into the peritoneum.

Results of kidney transplantation

- Graft survival for deceased donor kidney transplantation:
  1y: 94%.
  5y: 86%.
- Graft survival for living donor kidney transplantation:
  1y: 98%.
  5y: 92%.
- Patient survival for deceased donor kidney transplantation:
  1y: 96%.
  5y: 88%.
- Patient survival for living donor kidney transplantation:
  1y: 99%.
  5y: 95%.

Pancreas and islet transplantation

Pancreatic transplantation is predominantly performed in patients with type 1 diabetes. However, recently, it has been shown to be of benefit in a select group of patients with type 2 diabetes. There are three main types of pancreas transplants performed: (1) simultaneous pancreas and kidney (SPK)—which accounts for 90% of all pancreas transplants and is indicated for patients with type 1 diabetes and end-stage renal failure; (2) pancreas after kidney (PAK)—performed in diabetic patients who have had a kidney transplant (usually from a living donor); and (3) pancreas transplant alone (PTA)—for patients with type 1 diabetes who have normal kidney function, but life-threatening complications of diabetes (such as recurrent hypoglycaemic unawareness).

Pancreas transplantation is associated with greater morbidity and mortality than kidney transplantation due to ↑ risk of cardiovascular complications in the diabetic population, and therefore, the donor and recipient selection criteria are fairly stringent.

A total of 179 pancreas transplants were performed in 2016–17 in the UK, of which 162 (91%) were SPK and 17 (9%) were either PTA or PAK. Graft and patient survival have substantially improved in recent years (1y graft survival for SPK 87%, patient survival 97%) due to advances in immunosuppression and surgical technique, as well as refinement of donor and recipient selection criteria.

Pancreas transplantation

The transplant operation

The pancreas is usually retrieved as the whole organ with the duodenum attached. It is transplanted either intraperitoneally or extraperitoneally into the RIF using similar techniques to those of renal transplantation.

The dual arterial supply of the pancreas, based on the splenic artery and SMA branches, is provided by forming an arterial Y-graft from a length of common, internal, and external iliac arteries retrieved from the donor. The Y-graft is usually anastomosed to the common iliac artery. Venous drainage is from the portal vein attached to the pancreas, which is generally anastomosed to the recipient's common iliac vein or vena cava.

Drainage of exocrine function is by anastomosis of the attached duodenum either to a convenient portion of the jejunum or to the bladder. Bladder drainage enables monitoring of urinary amylase as a sound marker of pancreatic exocrine function, but this is now less popular due to a high incidence of chemical cystitis from pancreatic exocrine enzymes. Exocrine anastomotic leakage may occur, giving rise to local inflammation, peritonitis, or pseudoaneurysm of the iliac artery.

Post-operative management

Immunosuppression is as for kidney transplantation, although higher levels of immunosuppression, including use of induction antibody therapy such as alemtuzumab or ATG, is common.

Monitoring of pancreas transplants is by serial serum amylase, serum lipase, and blood glucose levels. Immunological damage to the pancreas is usually advanced before changes in blood glucose are recognized.

When the pancreas is transplanted with a kidney, the kidney may be biopsied if rejection is suspected as it usually affects both organs. The pancreas can also be biopsied percutaneously under radiological guidance. Transplant pancreatitis is also a frequent complication, the mild form of which is usually self-limiting, but more severe forms, in the presence of collections and necrosis, may need debridement, drainage, or removal of the graft.

Graft thrombosis of the pancreas transplant occurs more commonly than is seen in kidney transplant (due to lower blood flow through the pancreas). It is therefore routine in some centres to anticoagulate pancreas transplant recipients. Re-laparotomy for exploration of pancreas transplants can be as high as 50%.

Fungal infections are a major problem, so antifungal prophylaxis with PO fluconazole may be administered for the first week to 10 days.

Islet cell transplantation

To avoid the complications associated with the exocrine secretions of the pancreas, transplantation of the pancreatic islets alone is an attractive option. The islets are isolated from the retrieved pancreas and prepared as an infusion to be embolized into the liver via a portal venous catheter inserted by an interventional radiologist. In the UK, 34 pancreatic islet transplants were performed in 2016–17.

Islet cell retrieval is especially feasible from donors with high BMI, as the steatotic pancreas often has a large number of functioning islets, but the pancreas is infiltrated with fat and tolerates ischaemia poorly, leading to severe pancreatitis after solid organ transplantation.

Islet cell transplantation only leads to insulin independence in a minority of patients, but it usually does improve glycaemic control and hypoglycaemia is rare. It is therefore an especially good option for diabetics with hypoglycaemic unawareness.

Complications of islet cell transplantation include portal vein thrombosis and bleeding. Transient elevation of liver enzymes is commonly seen. Acute rejection does occur but is impossible to diagnose. Recurrent infections can increase levels of antibodies, reducing success rates.

Liver transplantation

A total of 946 deceased donor liver transplants were performed in 2016–17 in the UK, of which 814 were whole liver. There were 22 living donor transplants in 2016–17. The liver can be split into the right and left lobes for transplantation into an adult and child simultaneously or to allow living donor liver transplants.

Common diseases suitable for transplantation

- Chronic liver disease—alcoholic liver disease, NAFLD, primary biliary cirrhosis, primary sclerosing cholangitis (excluding cholangiocarcinoma), hepatitis B or C cirrhosis.
- Malignancy such as HCC in a cirrhotic liver (selected cases).
- Fulminant hepatic failure (e.g. acute viral hepatitis, drug reactions, or paracetamol overdose).
- Inborn errors of metabolism (e.g. Crigler–Najjar syndrome type 1).

Clinical indications

(Also see Table 20.2.)

- Acute fulminant liver failure (see Table 20.3).
- Category 1. Expected 1y mortality >9% without liver transplant.
- Category 2. HCC within 'Milan criteria' (see Box 20.3).
- Category 3. Variant syndromes affecting quality of life:
  - Persistent and intractable pruritus.
  - Diuretic-resistant ascites.
  - Hepatorenal syndrome.
  - Hepatopulmonary syndrome.
  - Chronic hepatic encephalopathy.

The transplant procedure

The liver is transplanted on an urgent basis, ideally within 12h of retrieval. The recipient undergoes removal of the native liver and may be placed on veno-venous bypass, and then the new liver is implanted in an orthotopic position, restoring the normal vascular anatomy with the biliary drainage via an end-to-end choledocho-choledochostomy or a Roux-en-Y hepatico-jejunostomy if the recipient bile duct is diseased.

If the recipient has accessory hepatic arteries, the common hepatic artery may be insufficient to perfuse the liver and so arterial conduits can be fashioned from the donor iliac arteries retrieved with the liver.

Post-operative management

Most commonly, immunosuppression is achieved using a combination of tacrolimus, azathioprine, and prednisolone. The liver is less prone to acute rejection than other organs, so immunosuppression can be fairly rapidly tapered after the immediate post-operative phase.

Hepatic artery thrombosis (HAT) is an important complication which occurs in 4% of transplants and requires immediate re-transplantation. It usually presents as metabolic acidosis with rising serum lactate levels. Doppler USS is done as soon as possible after the operation to detect HAT at an early stage. Administration of platelet transfusions increases the risk of HAT. Bile leaks and strictures are common (incidence 5–30%), with a higher

Table 20.2 Monitoring disease progression using Child–Pugh score (patients in class C should be referred for transplantation)

- Child–Pugh class B, 5–6 points
- Child–Pugh class B, 7–9 points
- Child–Pugh class C, 10–15 points

	1 point	2 points	3 points
Bilirubin (micromol/L)	<34	34–51	>51
Albumin (g/L)	>35	28–35	<28
Prothrombin time (seconds prolonged)	1–3	4–6	>6
Ascites	None	Slight	Moderate
Encephalopathy grade	None	1–2	3–4

Table 20.3 King's College criteria for transplantation for acute liver failure

Paracetamol overdose-related acute liver failure	Non-paracetamol-related acute liver failure
Arterial pH <7.3; OR	Prothrombin time >100s; OR
All three of: • Prothrombin time >100s • Creatinine >300micromol/L • Grade III–IV encephalopathy	Any three of: • Bilirubin >300 micromol/L • Jaundice or encephalopathy for >7 days • Prothrombin time >50s • Age <10 or >40y • Drug toxicity

Box 20.3 The Milan criteria for transplantation for hepatocellular carcinoma

- Child's class B or C cirrhosis; and
- Single tumour <5cm or up to three tumours <3cm; and
- Absence of macrovascular portal vein invasion.

incidence seen in split liver transplants. Usually a bile leak or an anastomotic stricture can be treated by ERCP and/or stenting of the bile duct. If not, then a Roux-en-Y hepatico-jejunostomy may be required. For non-anastomotic strictures, there is a worse prognosis and re-transplantation is often required. DCD donor transplants tend to have a higher rate of biliary complications, compared with DBD.

Graft survival has significantly improved over the past 20y and 5y survival is now 80%.

Small bowel transplantation

Small bowel transplantation is rarely performed, compared with other organs, with around 100 per year worldwide. Small bowel may be performed alone, in combination with a liver transplant, or as part of a multivisceral transplant.

The results of small bowel transplantation have improved dramatically in recent years, largely owing to the advances in immunosuppressive therapy. Patient and graft survival at 1y from small bowel and multivisceral transplantation is 80% and 75%, respectively. Some high-volume centres for isolated small bowel transplantation have reported graft survival of as high as 90%.

Indications

The main indication is short bowel syndrome (or intestinal failure) with permanent requirement for parental nutrition. A combined liver and small bowel transplant is required for those patients with short bowel syndrome who have significant liver disease (usually 2° to the hepatotoxic effects of TPN).

Indications for small bowel transplantation are intestinal failure with life-threatening complications of TPN:

- Loss of two or more central venous access sites, including venous thrombosis.
- Life-threatening line sepsis (recurrent line infections or fungal infections).
- Severe liver disease.

Successful small bowel transplantation is dependent on adequate central venous access being available, so potential candidates need to be referred before loss of all access sites.

Small bowel transplantation may also be considered in patients with chronic intestinal failure due to disease conditions where survival on long-term TPN is expected to be poor such as for chronic obstruction, extremely short bowel (<50cm), and end-jejunostomy without colon.

Small bowel transplantation is cost-effective, with lower maintenance costs, compared with home TPN, and additionally, recipients can normally resume full normal activities, with potential return to employment. Quality of life is also better than with home TPN.

Post-operative management

- Feeding tube inserted to introduce early enteral nutrition.
- Serial video zoom endoscopy to assess mucosa. Villous atrophy is early sign of acute rejection.
- Endoscopic protocol biopsies to exclude early acute rejection.
- Diarrhoea is the main presenting symptom of graft dysfunction, whether due to rejection, infection, or ischaemia, and is investigated by endoscopy and biopsy.

Acute rejection remains a major concern. It is common as there is a high population of immune cells in the gut, compared with other organs. Rejection is still associated with high risks, both of graft loss and death, so early diagnosis is key. It is problematic as acute rejection also reduces gut wall barrier function, leading to sepsis, especially in the context of augmented immunosuppression to treat rejection. Sepsis continues to be the leading cause of death following small bowel and multivisceral transplantation.

Standard immunosuppression for small bowel transplantation utilizes alemtuzumab, ATG, or basiliximab as induction therapy, followed by maintenance with tacrolimus monotherapy. Mycophenolate may be added at a later stage to allow reduction in tacrolimus dose and reduce side effects.

Long-term complications of small bowel transplantation include development of PTLD and chronic rejection. In the long term, most foods can be tolerated, although foods high in simple carbohydrates and soluble fibre may cause dumping syndrome symptoms.

Graft-versus-host disease

Graft-versus-host disease is an extremely rare and potentially lethal complication following solid organ transplantation, affecting ~5% of small bowel transplants and <1% of liver transplants. It is commoner in bone marrow transplantation (up to 50%).

Common surgical procedures

Laparotomy

Laparotomy is a surgical incision into the abdominal wall to gain access into the abdominal cavity. It can be performed in the emergency or elective setting. Various incisions can be made, including midline, paramedian, oblique, and transverse. Incision position and size largely depend on organ exposure required to perform the relevant operation.

Preoperative preparation

- Resuscitate the patient.
- Communicate with the anaesthetist, theatre coordinator, and ICU (if required).
- Procedure-specific consent, including relevant general and specific complications (e.g. haemorrhage, intra-abdominal collection, wound infection, thromboembolism, herniation, damage to adjacent organs).
- Calculate Portsmouth-POSSUM (P-POSSUM) or NELA score for morbidity/mortality prediction.
- Also mark intended surgical site, group and save/cross-match, NGT, catheterization, IV antibiotics, and VTE prophylaxis.
- Refer to stoma nurses to mark potential stoma site if time permits.
- Keep patient NBM.

Position and theatre set-up

- Consider patient position on table: generally supine; however, may require Lloyd-Davis position for operations involving pelvic organs, left colon, or anorectum.
- Gel pads (particularly under pressure areas and for long procedures).
- Straps if table tilting required.
- Patient warming, hair removal, and pneumatic calf compression.
- Skin preparation from lower thorax to upper thigh level.
- Sterile drapes ± adhesive plastic drape over skin.

Procedure

Midline laparotomy incision

- Inform anaesthetist prior to initial incision.
- Manipulate the skin taut and make a midline incision continuously with the blade belly. Ensure to curve around the umbilicus.
- Utilize cutting diathermy to go through dermal layer and then coagulation/blend diathermy down to linea alba.
- Apply large wound towels/swabs to both wound edges and lift anteriorly to aid diathermy down to midline linea alba.
- Incise through linea alba and pick up underlying peritoneal layer between two individual clips. Feel the peritoneum in between the clips to ensure no bowel inadvertently caught. If clear, incise the tented peritoneum to enter the abdominal cavity.
- Finger sweep to confirm no adhering underlying structures and then extend midline peritoneal incision with diathermy.

Key steps during laparotomy

- Send any fluid or pus evident on entering the abdomen for microbiological (M,C,&S) ± cytological analysis.
- Pressure-control pack any areas of active haemorrhage.

- Temporarily control bowel perforations with clamps or continuous sutures.
- Explore abdomen and methodically examine organs as clinically required. This may be focused on particular organs, as determined by preoperative radiological imaging.
- Definitively decide on procedure required following abdominal examination.
- Peritoneal lavage to reduce risk of abscess formation and bacterial septicaemia. Use copious amounts of warm saline (at least 2–3L) to irrigate peritoneal cavity.
- Drains: usually closed passive, e.g. Robinson's drain; insert if gross contamination or concern regarding potential complication such as bleeding or bowel re-perforation.

Midline laparotomy closure

- Prior to closure, ensure swab, instrument, and needle check correct.
- Mass closure technique to close the peritoneum and sheath together, using No. 1 or 0 slowly absorbable loop PDS®/non-absorbable nylon on a blunt (safer) or cutting needle.
- Use separate sutures to start at either ends of the wound. Approximate peritoneum and sheath continuously with running sutures along the wound and unite in the middle. Ensure each bite is 1cm apart and also taken 1cm from the fascial edge when suturing.
- The total length of the starting suture should be ~4 times the length of the laparotomy incision.
- Close skin with staples or sutures (subcuticular continuous absorbable Monocryl® 3.0).

Post-operative care

- Thromboprophylaxis: stockings, LMWH, calf compression.
- Inform patient and relatives of operation details and post-operative plan.
- Ensure specific post-operative instructions relayed to recovery staff.
- Clips out 10–14 days.

Complications (specific to laparotomy incision)

- Wound infection, 2–30%, depending on extent of contamination.
- Incisional hernia, 5–30%.

Procedural tips

- Employ retractors to improve exposure during difficult cases, e.g. Omni-Tract® and Goligher® retractors.
- Do not hesitate to extend incision if exposure inadequate.
- If accessing through old laparotomy incisions, excise hypertrophic scars on initial incision. If underlying adherent structures, extend skin incision beyond old incision to go through virgin non-scarred tissue to enter abdomen.
- Repair enterotomies as soon as possible; use PDS® 3.0 seromuscular interrupted sutures.
- Meticulous haemostasis as always.

Establishing pneumoperitoneum

Minimal invasive surgery aims to cause least surgical trauma as possible to patients, compared to 'conventional' open surgery. Benefits include shorter hospital stay, less pain, quicker functional recovery, and superior cosmesis. Pneumoperitoneum induction is the 1° step in performing laparoscopy surgery.

Preoperative preparation

- Consent should include potential conversion to an open procedure. Other specific complications include injury to intra-abdominal structures, port site hernias, wound infection, haemorrhage, and rarely CO2 embolism. Warn about shoulder tip pain post-operatively.
- Group and save. Ensure pregnancy test negative (if applicable).

Position and theatre set-up

- Confirm laparoscopic stack available for use. The stack includes a monitor, light source, insufflator, camera, and possible connected printer. Also ensure you have diathermy and an irrigation system.
- Position the stack appropriately, depending on the procedure.
- Patient supine or Lloyd-Davis for certain pelvic/rectal procedures.
- Use gel pads, patient strapping, body warming, calf compression, and hair removal. Catheterization as appropriate.
- Ensure cables from camera, gas insufflation, and light source are untangled and clipped securely to the sterile drapes.
- Attach diathermy and irrigation systems.
- White balance camera and set on standby mode to turn light off.

Procedure (open Hassan technique)

- Infra-umbilical incision into skin can be curved or vertical, depending on surgeon's preference. Some incise through the umbilicus.
- Deepen until fascial fibres are visualized at base of umbilical stalk.
- Incise the linea alba at this point vertically, whilst elevating the stalk upwards with a Littlewoods' to clear underlying structures.
- Insert a curved artery clip to enter the abdominal cavity (often needs a gentle push through the remaining thin peritoneal layer).
- Insert a 10–12mm laparoscopic port. Connect the insufflation tube and put gas on low flow. If pneumoperitoneum successful, change to high flow and allow insufflation to 12mmHg pressure.
- Insert camera into port and confirm placement.

Port site closure

- Remove ports under vision and release gas by opening ports.
- Close large port incisions (≤10mm) with a fascial suture (use J-needle). Smaller port incisions do not require formal fascial closure.

Post-operative care

- Clear instructions on VTE prophylaxis, oral diet, and antibiotic need.

Complications (specific to inducing pneumoperitoneum)

- Port site infection <5%; port site hernia <2%; visceral injury <1%.

Procedural tips

- Tilt table to aid exposure during operation (e.g. head up and right side up for laparoscopic cholecystectomy).
- Insert fascial stay sutures or box stitch when initially entering abdominal cavity. This will secure port and prevent it from slipping out completely. It also aids closure of the fascia at the end of the procedure.
- Avoid Veress needle as blind and ↑ risk of injuring visceral structures when compared to open Hassan technique.
- Can close skin using suture, clips, or glue.

Diagnostic laparoscopy

Indications

Emergency

- Lower abdominal pain with suspected acute appendicitis or acute gynaecological pathology (e.g. ruptured ovarian cyst).
- Perforated intra-abdominal viscus (perforated peptic ulcer or colonic diverticulum).
- Trauma.

Elective

- Facilitates intra-abdominal biopsy (mesenteric lymph node, omentum).
- Staging laparoscopy as workup for intra-abdominal oncological resection.
- Interval appendicectomy.
- Wide range of elective GI, urological, gynaecological, and endocrine procedures for benign and malignant pathology.

Preoperative preparation

- Always consent for 'laparoscopy and proceed' with potential complications and other procedures (e.g. discovery of a non-viable ovary that needs resecting may result in sub-/infertility).
- Urethral catheterization decompresses the bladder to allow proper assessment of pelvic structures.

Procedure

- Send any fluid or pus evident in the abdomen for microbiological (M,C,&S) ± cytological analysis.
- Diagnostic laparoscopy involves complete examination of the intra-abdominal cavity. Often this is focused examination of part of the abdomen from preoperative imaging.
- If bowel must be handled, do so cautiously with atraumatic Johan graspers.
- Always insert instruments under direct vision to avoid unnecessary injuries.
- When using diathermy, beware of residual heat and coupling.

Inguinal hernia repair

There are various methods to repair an inguinal hernia. The open approach remains the most popular among general surgeons. However, more hernias are being repaired laparoscopically and under LA due to increasing surgical expertise and patient wishes.

Indications

- Symptomatic hernia (emergency repair if incarcerated/strangulated).
- Asymptomatic hernia with high risk of complications.

Preoperative preparation

- Options include GA, LA, or spinal anaesthesia.
- Open repair versus laparoscopic repair (TEP/TAPP).
  - Open approach is operation of choice for 1° unilateral inguinal hernia repairs. Also best option for emergency cases (i.e. strangulated/incarcerated), large inguinoscrotal hernias, previous lower abdominal surgery (hostile scarred tissue), and recurrent hernias following a previous laparoscopic repair.
  - Laparoscopic approach preferred for bilateral (particularly TEP) and recurrent hernias following a previous open repair.
- Consent with complications, including bleeding, infection (wound/mesh), recurrence, chronic groin pain, ischaemic orchitis, urinary retention, and damage to bowel.
- Encourage weight loss and medical optimization (especially for respiratory conditions).
- Mark the site and side of surgery.
- Consider perioperative antibiotics; no consistent evidence to suggest reduction in wound/mesh infection.

Positioning and theatre set-up

- Supine.
- Patient warming and hair removal.
- Ensure full stack and in adequate position for laparoscopic repair.

Procedure

Open (Lichtenstein repair)

- Oblique incision (parallel to Langer's lines) from finger breadth above pubic tubercle extended lateral to deep ring.
- Dissect down to external oblique aponeurosis and identify superficial ring (ligate any large veins on way with Vicryl® 2.0 ties).
- Open external oblique aponeurosis with a slit (use scalpel) parallel to the fibres and extend laterally from the superficial ring with scissors.
- Split superior leaf of external oblique from underlying tissue to allow a finger to be placed directly onto the pubic tubercle. Repeat the same with the inferior leaf and hook up the spermatic cord with your finger and protect in hernia ring/nylon tape.
- Assess for direct (bulge medial to the inferior epigastric vessels) and indirect (at the deep ring and lateral to the inferior epigastrics) hernia sacs.

- For direct hernias, separate the cord from the sac and reduce it. Plicate the posterior wall of the inguinal canal (transversalis fascia) either with continuous or with interrupted sutures from the pubic tubercle towards the deep ring. Approximate the transversalis fascia just above the inguinal ligament to that below the conjoint tendon.
- For indirect hernias, separate the sac from the cord structures. Open the sac and inspect the contents. Reduce the contents back into the peritoneum. Twist the sac and place a transfixion stitch at the base of the sac (Vicryl® 2.0). Cut off the excess sac and reduce.
- For both types of inguinal hernia, customize a polypropylene mesh to cover from pubic tubercle to lateral to the deep ring. Secure the mesh with non-absorbable sutures (e.g. Prolene® 2.0) or tacks.

Laparoscopic TAPP

- Establish pneumoperitoneum.
- Open parietal peritoneum above hernia to create a flap from the ASIS to the medial umbilical ligament.
- Identify cord structures and inferior epigastric vessels.
- Separate sac from cord structures and reduce intraperitoneally.
- Polypropylene mesh placed to cover from pubic tubercle to lateral to the deep ring. Secure with laparoscopic tacker.

Laparoscopic TEP

- Open cut down to sheath inferior and lateral to umbilicus on side of hernia. Use dissecting balloon to create pre-peritoneal space.
- Identify cord structures and inferior epigastric vessels.
- Separate sac from cord and reduce it.
- Insert polypropylene mesh and tack over the pubic tubercle and superiorly.
- Gradually withdraw the gas.

Closure

- For open repairs, close in layers with absorbable sutures.
- Port sites ≤10mm, need musculo-fascial closure.

Post-operative care

- Commence oral diet (unless bowel resected); no heavy lifting for 6 weeks.

Complications (specific to the procedure)

- Groin wound infection, <5%.
- Recurrence, 3% lifetime.
- Groin/scrotal haematoma, 2%.
- Chronic groin pain, 1–2%.
- Ischaemic orchitis, <1%.

Appendicectomy

Laparoscopic appendicectomy has long been the default approach for appendicectomies in the Western world. It facilitates better exposure/access and allows for a preceding diagnostic laparoscopy.

Indications

- Acute appendicitis.
- Diagnostic laparoscopy for acute lower abdominal pain not revealing any other convincing macroscopic pathology.
- Interval appendicectomy following previously conservatively treated appendicitis.
- Appendix mass/mucocele.

Preoperative preparation

- Consent with complications, including pelvic collection, damage to bowel/right ureter/bladder, haemorrhage, and port site herniation. Other procedures may include conversion to open, drain, and bowel resection.
- Pregnancy test for ♀.

Positioning and theatre set-up

- Supine.
- Urethral catheterization to decompress the bladder, particularly in ♀ to allow examination of the pelvic organs.
- Ensure correct stack monitor position—patient right caudal.
- Can either use 5 or 10mm 30° scope.

Procedure

Laparoscopic approach

- Establish pneumoperitoneum.
- Further port site placement in suprapubic and left iliac fossa (see Fig. 21.1).
- Drain any fluid/pus and send for microbiology (M,C,&S).
- Identify appendix and drain any adjacent abscesses.
- Identify appendicular base (follow taenia coli from caecum which merge at appendicular base).
- Divide the mesoappendix from the appendicular tip towards the base with hook diathermy (deskeletonize the appendix).
- Once at the base of the appendix, apply double ligation with two Endoloop® sutures. Apply a further Endoloop® suture 1cm distal to this and divide the appendix in between.
- Some surgeons diathermy the mucosa of the remaining appendicular stump.
- Irrigation and washout with normal saline. Leave a drain in the right paracolic gutter/pelvis if significant pus/contamination present.
- Deliver the appendix via an endoscopic retrieval bag through the umbilical port.

Open approach

- Oblique RIF incision above McBurney's point.
- Open external oblique, internal oblique, and transversus abdominis, splitting the fibres in the direction of their travel without cutting them.
- Open the peritoneum between clips.
- Apply a wound protector to the wound.
- Use digital manipulation and Babcock forceps to deliver the appendix.
- Identify appendicular artery by blunt dissection. Ligate and divide.
- Complete mesenteric division to appendiceal–caecal angle.
- Apply a crushing clamp to the base of the appendix and then release and re-apply just distal to this.
- Transfix or apply a tie to the base of the appendix and cut flush onto the crushing clamp.
- Use a purse string to bury the remaining appendicular stump.

Closure

- Port sites ≤10mm need musculo-fascial closure.
- For open procedures, close in layers with absorbable sutures (peritoneum, each layer of oblique muscle, and skin).
- If free pus in the abdomen, close skin with staples or interrupted sutures, as higher risk of wound infection.

Post-operative care

- Stop antibiotics if no free fluid/pus. If appendix perforated, ensure at least a 5- to 7-day course of antibiotics.
- Oral diet as soon as tolerable.

Complications (specific to the procedure)

- Wound/port site infection, <5% (greater if open and perforated).
- Pelvic collection/abscess.
- Ileus.

Procedural tips

- Tilt table to aid exposure during operation (head down and right side up)
- If retrocolic long appendix, can approach with retrograde dissection.
- Use gentle blunt dissection to free inflamed appendix stuck to parietal peritoneum/adjacent bowel.
- Call gynaecologists to theatre if unexpected gynaecological pathology found.
- If appendicular inflammatory mass discovered, take plenty of pictures and abandon procedure. Treated conservatively with antibiotics and consider interval appendicectomy. Bowel resection may be required if concern for malignancy.

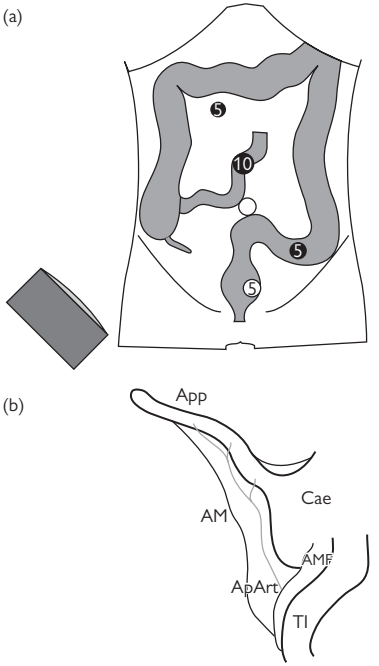

Fig. 21.1 a) Port sites for laparoscopic appendicectomy. b) Anatomy of the appendix as visualised intraoperatively. App., appendix; Cae, caecum; TI, terminal ileum; ApArt., appendicular artery (which travels in the mesoappendix, AM).

Cholecystectomy

Cholecystectomy is commonly performed as an elective day case procedure and laparoscopically. For acute cholecystitis, some advocate early cholecystectomy of a 'hot gall bladder' within 1 week of onset of symptoms as an emergency operation. However, most tend to perform a delayed elective cholecystectomy 6 weeks following initial antibiotic treatment for cholecystitis. This delay allows time for inflammation to settle and reduces procedure-related complications.

Indications

- Symptomatic ultrasound-proven gallstones (biliary colic, acute cholecystitis).
- Gallstone-related pancreatitis (should perform within 2 weeks).
- CBD stones (remove via ERCP/CBD exploration).
- Deteriorating patient with empyema/gall bladder perforation.
- Gall bladder polyps (>10mm, rapidly growing, high risk of cancer).
- Previous acalculous cholecystitis.

Preoperative preparation

- Consent with specific complications, including bile duct injury/bile leak, damage to liver/bowel, residual ductal stones, and persisting symptoms. Other procedures include potential conversion to open (2.5%), on-table cholangiogram, and CBD exploration if relevant.
- Encourage preoperative weight loss and low-fat diet.
- Group and save.
- Review recent LFTs and relevant imaging (ultrasound, MRCP, ERCP).
- No perioperative antibiotics required unless biliary spillage.

Positioning and theatre set-up

- *Supine.* Some surgeons stand between the legs and thus require Lloyd-Davis. Tilt head up and right side up during procedure to improve exposure.
- Ensure correct stack monitor position—patient right cranial.
- Use 10mm 30° scope.

Procedure

- Establish pneumoperitoneum.
- Standard port placement (Umbilical, Right flank, RUQ, epigastric); alternatives include three-port technique and single incision, with multiple instruments placed through one large umbilical port.
- Divide adhesions overlying the gall bladder.
- Dissect to achieve the 'critical view of safety'—the cystic artery and duct are visualized and deemed terminal to the gall bladder.
- Clip the cystic duct (two distal and one proximal on gall bladder neck) and divide in between. Repeat for the cystic artery.
- Cautiously dissect the gall bladder off the liver bed.
- Insert free gall bladder into retrieval bag and remove via umbilical port.

Closure

- Port sites ≤10mm need musculo-fascial closure.

Post-operative care

- Antibiotics if gall bladder inflamed/empyema/perforation.
- Oral diet as soon as tolerable. Home on same day usually possible.

Complications (specific to the procedure)

- Port site infection, <5%.
- Haemorrhage, 2%.
- CBD injury, <1%.

Procedural tips

- Convert to open if laparoscopic approach deemed unsafe (i.e. difficult anatomy, concern for cholecysto-duodenal fistula). Perform Kocher's right subcostal incision to gain access.
- Use left-hand instrument effectively to retract the gall bladder away from the liver. This will demonstrate the plane of dissection when dissecting the gall bladder off the liver.
- If previous midline laparotomy, use a 'visiport' to insert a port under direct vision.
- Use larger clips/Laparoclip® to clip large cystic ducts.
- Laparoscopic ultrasound can assess biliary anatomy and establish the presence of CBD stones.
- Beware duct of Luscha running between the gall bladder.

Perforated peptic ulcer repair

A perforated peptic ulcer is a true surgical emergency, with delays in operative management associated with significant morbidity and mortality. Select patients can be managed conservatively if clinically stable and non-peritonitic. Approach options include traditional open and laparoscopic, depending on surgeon expertise and clinical picture.

Preoperative preparation

- Appropriate imaging, usually CT.
- Resuscitate patient with ICU input.
- IV antibiotics.
- Group and save.
- Consent with specific complications, including intra-abdominal abscess, persistent leak, gastric/duodenal fistula, and mortality. Other procedures (depending on pathology encountered) could include bowel resection and partial gastrectomy.

Positioning and theatre set-up

- Urethral catheterization.
- *Supine.* Tilt table head up. Patient strapping essential.
- If performing laparoscopically, stand between legs to allow for suturing with stack positioned in right cranial position.

Procedure

- *Open:* upper midline between xiphisternum and umbilicus (bile and gas on entry into abdomen confirms diagnosis).
- *Laparoscopic:* establish pneumoperitoneum. Insert four ports overall, with one to retract the liver and other two triangulated to allow suturing.
- Identify site of perforation: gastric, prepyloric, or duodenal (commonest in anterior D1). If no perforation seen, open lesser omentum to assess posterior aspect of stomach.
- Partial gastrectomy with reconstruction required if suspicion of gastric malignancy or large perforated gastric ulcer.
- If benign ulcer perforation, repair with omental patch (see Fig. 21.2). Place at least two or three full-thickness 2.0 or 3.0 absorbable sutures entering and exiting 1cm on both sides of the perforation. Leave these sutures long and untied.
- Dissect an omental tongue and lay over the perforation. Tie the sutures snug to hold the omentum in place, sealing the perforation.
- An alternative option would be to do 1° repair with interrupted sutures.
- Four quadrant peritoneal lavage with at least 4–5L until clear. If performing laparoscopic, use table tilt to help with washout.
- Insert large Robinson's drains (subhepatic/pelvic position).
- Ensure NGT in place and on free drainage.

Closure

- Port sites ≤10mm need musculo-fascial closure.
- Upper midline incision: perform mass closure and close skin with clips/sutures.

Post-operative care

- IV PPIs and consider early *Helicobacter pylori* eradication.
- Free fluids PO initially. May require parenteral nutrition if clinically unstable post-operatively.
- If gastric ulcer, perform OGD in 8 weeks to ensure healing. No need to do OGD for duodenal ulcers.

Complications (specific to the procedure)

- Persistent leak around omental patch, 5%.
- Intra-abdominal abscess.
- Mortality.

Procedural tips

- Ensure omental patch not under tension (higher risk of failure).
- Be wary not to pick up posterior wall with needle when suturing anterior perforation.
- Preoperative optimization with early critical care input.

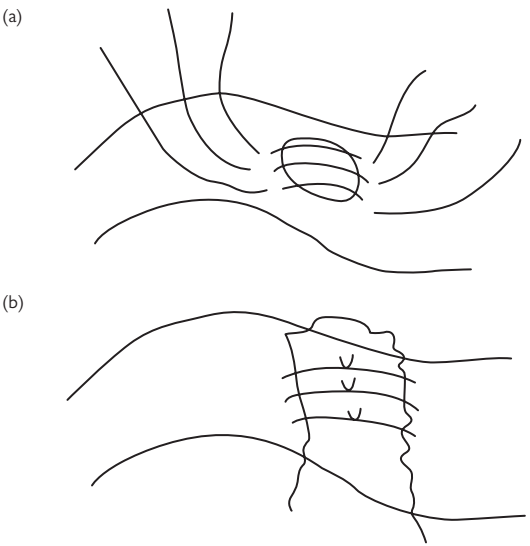

Fig. 21.2 (a) Sutures placed in ulcer edges. (b) Omental patch in place.

Haemorrhoidectomy

Large prolapsing or banding/sclerotherapy-resistant haemorrhoids generally require formal operative excision as a day case procedure. There are various alternative methods to the traditional Milligan–Morgan haemorrhoidectomy, including stapled haemorrhoidectomy and haemorrhoidal artery ligation operation (HALO) which have gained popularity.

Preoperative preparation

Consent with specific complications, including recurrence, pain, faecal incontinence, anal stenosis, and haemorrhage.

Positioning and theatre set-up

- Lithotomy with head down position.

Procedure

Conventional haemorrhoidectomy

- Use Eisenhammer retractor to assess haemorrhoids to be excised and size of remaining skin bridges.
- Apply clip to haemorrhoids to be excised and inject LA with adrenaline into the external haemorrhoid and base.
- Incise the skin around the haemorrhoid with cutting diathermy.
- Deepen the dissection and gently take the haemorrhoid off the underlying internal sphincter.
- Narrow or 'cone' the pedicle whilst dissecting towards the apex.
- Ligate the pedicle.

LigaSureTM haemorrhoidectomy

- Utilizes the LigaSure™ Small Jaw energy device. Results in less heat-related collateral tissue damage.
- Approach with similar steps as a conventional haemorrhoidectomy; however, instead pick haemorrhoidal pedicles between the jaws of the LigaSure™ energy device and activate via the connected foot paddle. This coagulates the tissue to achieve a seal.

HALO

- Uses a Doppler system integrated within a probe to localize haemorrhoidal arteries, which are subsequently ligated superior to the dentate line through a ligation window within the probe.
- Further haemorrhoidopexy can be performed for larger prolapsing haemorrhoids.

Post-operative care

- Home on same day of operation with analgesia and laxatives.
- Review patient in outpatient clinic in 2–4 weeks.

Complications (specific to the procedure)

- 2° haemorrhage at 7–10 days (requiring intervention) <5%, anal stenosis, pain, abscess, faecal incontinence, rectal perforation (with stapled/HALO procedures).

Pilonidal sinus excision

Pilonidal sinus disease requires operative intervention if symptomatic or has previously led to an acute pilonidal abscess. Various operative approaches are available to treating non-infected sinuses, including excision and 1° closure/leave open, Bascom's procedure, and a multitude of flaps (Karydakis, rhomboid, rotational). Excise all tracks evident and if extensive disease, reconstruct with a flap (always avoid a midline suture line).

Preoperative preparation

- Consent with specific complications, including pain, failure to heal, infection, and recurrent sinus disease.
- Antibiotic on induction.

Positioning and theatre set-up

- Prone/jack-knife position with buttocks retracted laterally with tape.
- Shave operative site.

Procedure (excision and primary closure)

- Probe all sinuses to ascertain direction and length of tracks. Can adopt methylene blue dye within tracks to aid operation.
- All sinus disease (single/multiple pits) in an elliptical skin incision.
- Deepen elliptical dissection around tracks with coagulation/blend diathermy. Often dissection will be right down to sacral fascia.
- Meticulous haemostasis.
- Once all sinuses excised, decide whether to close the wound with strong sutures (1° closure) or leave open and allow to heal with 2° intention (can take months). If small wound, it is reasonable to do 1° closure.

Closure

- If wound comes together with minimal tension, close in two layers (deep and superficial). To close the skin, use interrupted mattress sutures (monofilament synthetic).
- If large cavity under sutures, insert closed suction drain.

Post-operative care

- Remove sutures in 2–3 weeks.
- If wound left open, will require initial daily dressing changes.
- Shave wound edges regularly.
- Use silver nitrate to cauterize excess granulation tissue.

Complications (specific to the procedure)

- Wound infection, 5%.
- Recurrent sinus disease, 3–5%.
- Painful scarring, 2%.

Stoma formation

There are various bowel-related stomas, including end and loop ileostomies and colostomies. Ileostomies are spouted on exteriorization, whilst colostomies are flush with the skin. Loop stomas are created to divert bowel contents or protect distal anastomoses. Indications for stomas include management of inflammatory bowel disease, bowel cancer, faecal incontinence, pelvic sepsis, and post-emergency bowel surgery (e.g. bowel ischaemia, colitis).

Preoperative preparation

- Consent with specific complications, including ischaemia, prolapse, retraction, parastomal hernia, stenosis, and high-output stoma.
- Can be performed open or laparoscopically.
- Stoma nurses to mark potential stoma sites preoperatively.
- Group and save.

Positioning and theatre set-up

- Supine or Lloyd-Davis (if rectal surgery performed) position.

Procedure

- A stoma should ideally traverse the musculo-fascial layers of the abdominal wall (commonly the rectus abdominis).
- Excise a circular skin hole at the intended stoma (diameter 2–3cm).
- Deepen dissection down to the anterior rectus sheath with coagulation/blend diathermy.
- Make a cruciate incision in the anterior rectus sheath and use a Roberts forceps to split the rectus abdominis. This exposes the peritoneum, which can be opened between two clips.
- Ensure peritoneal opening can accommodate two fingers. This will allow for delivery of bowel through the trephine without significant strangulation. Now perform the appropriate stoma.

End ileostomy

- Deliver the stapled ileal end with Babcock forceps out to the skin.
- At this stage, close any laparotomy incisions and apply dressings.
- Excise the staple line off the ileum with scissors or a blade.
- Insert absorbable Monocryl® or Vicryl® 3.0 sutures through the skin edge and then take a serosal bite of the ileum 6cm proximal to the distal end. With the same needle, take one or two sequential bites, leading to the final bite at the distal edge of the ileum. Clip this suture and repeat with separate sutures around the stoma site.
- Once all have been inserted, perform an eversion manoeuvre to spout the ileostomy (can use the retraction end of a small Langenbeck retractor to aid eversion).
- Tie all sutures and supplement with further sutures as required.

Loop ileostomy

- Confirm orientation of bowel prior to delivery through the trephine. If need be, mark the distal limb with diathermy or a suture.
- Once delivered, make a transverse anti-mesenteric enterotomy using diathermy. A bridge is generally not required for loop ileostomies.

- As per end ileostomies, place 'spouting' sutures to the proximal limb on the superior edge of the skin incision.
- On the inferior edge of the skin incision, suture the distal lumen flush with the skin (full-thickness ileum and skin bites).
- Tie the sutures and ensure to spout the proximal limb superiorly (see Fig. 21.3).

End colostomy

- Deliver the stapled colonic end with Babcock forceps out to the skin.
- Excise the stapled end and suture the skin edge to the bowel end (full thickness) using interrupted absorbable 3.0 sutures.

Loop colostomy

- Create a mesenteric window adjacent to the bowel wall to insert a stoma-supporting bridge through.
- After delivery, make a longitudinal colostomy and suture full-thickness bowel wall edge to the skin edge. Secure bridge.

Post-operative care

- Commence oral diet unless risk of ileus.
- Regular stoma specialist nurse review of stoma health and patient training.
- Colostomy bridges can be removed generally after 5 days.

Complications (specific to stoma formation)

- Stomal oedema, ~30% (common and usually requires no treatment).
- Ischaemia, <5% (requires surgical treatment).
- Intestinal obstruction (due to tight musculo-fascial opening), 5%.
- Stoma retraction (usually due to inadequate mobilization), <5%.
- Stoma prolapse, <5%.
- Stomal/parastomal hernias (rare acutely), up to 50% long term.

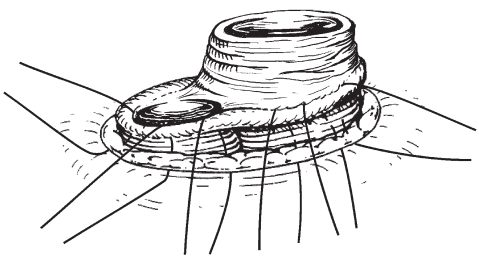

Fig. 21.3 Suture placement for loop ileostomy to achieve proximal spouting.

Small bowel resection and anastomosis

Essential requirements for a successful anastomosis include accurate apposition of the bowel ends, good blood supply, and a tension-free join. Small bowel content spillage must be kept to a minimum when performing an anastomosis.

Indications

- Obstruction with associated non-viable bowel.
- Mesenteric ischaemia.
- Crohn's small bowel disease—stricturing, inflammation.
- Malignancy— 1° or locally advanced large bowel 1°.
- Intussusception.
- Meckel's diverticulum

Preoperative preparation

- Consent with specific complications, including anastomotic leak, stricture, and haemorrhage.
- Can be performed open or laparoscopically.
- Group and save.
- NGT if obstructive symptoms.

Positioning and theatre set-up

- Supine.
- Decide on whether to perform a hand-sewn or stapled anastomosis. A stapled anastomosis will require a linear stapler with potential reloads.

Procedure

- Assess diseased segment of bowel and decide on resection margins.
- Transilluminate the mesentery to demonstrate the vasculature. Decide on mesentery to be excised between bowel resection margins (stay close to bowel for benign disease). With diathermy, gently score the peritoneum over the intended mesenteric excision line.
- Divide the mesentery with diathermy and ligate vessels.
- Milk bowel contents away from the bowel segment to be resected and apply distal and proximal non-crushing bowel clamps.

Single-layer end-to-end anastomosis

- Apply crushing clamps to both ends of the intended bowel resection.
- Place two large packs under the bowel and have suction ready.
- Cut the bowel flush with the crushing clamps using a blade.
- Align the two bowel ends and ensure no large diameter discrepancy.
- Use slowly absorbable PDS® or Vicryl® 3.0 sutures with a round-bodied needle.
- Insert stay sutures at the mesenteric and anti-mesenteric borders and leave untied in clips.
- Insert interrupted sero-muscular (extra-mucosal) sutures along the anterior bowel wall, with 3–4mm apart between the stay sutures.
- Use the stay sutures to rotate the anastomosis and apply interrupted sutures on the posterior aspect in a similar fashion.
- Tie the stay sutures.

Two-layer side-to-side anastomosis

- Lay the two bowel segments side by side for 10cm.
- Using double needle-ended PDS® or Vicryl® 3.0 sutures on a round-bodied needle, insert a posterior sero-muscular continuous suture joining both bowel segments.
- Adjacent to this suture, make 6cm corresponding enterotomies on both bowel segments.
- Place a double needle-ended suture in the middle of the posterior wall. With one end, perform over-and-over full-thickness sutures towards one corner. Repeat the same in the other direction with the other needle end. Keep suture bites 4mm apart.
- Once at the corners, perform 'Connell' sutures to get onto the anterior wall. Complete the anterior wall until both suture ends are in the middle and tie.
- Remove the bowel clamps at this point to test the single-layer full-thickness anastomosis.
- If no leak, complete the anterior sero-muscular layer with the original suture used for the posterior sero-muscular layer.

Closure

- Close any mesenteric windows to prevent internal hernias. Use absorbable sutures and keep bites superficial to prevent damaging mesenteric vessels.
- Loop PDS® 0 for mass closure if laparotomy.
- Close skin with clips or sutures.

Post-operative care

- NGT on free drainage with regular aspirations.
- Clear fluids only until ileus settles.
- Consider parenteral nutrition if prolonged ileus.

Complications (specific to the procedure)

- Haemorrhage, 1–2%.
- Paralytic ileus (common and often prolonged, especially if obstruction prior to operation).
- Anastomotic leak, 3–5%.
- Anastomotic stricture leading to bowel obstruction, 2–3%.

Procedural tips

- Stapled anastomosis with linear staplers can be performed to save time. Leak rates are similar to hand-sewn anastomoses.
- Use extra sutures at the corners (commonest leak site).
- Test your anastomosis by milking small bowel contents through it and insert additional sutures if leak evident.

Right hemicolectomy

Commonly performed laparoscopically by colorectal surgeons. An open approach can be used in emergency cases, locally invading tumours and in a hostile abdomen. An extended right hemicolectomy can be performed for distal transverse colon and splenic flexure pathology.

Indications

- Malignancy or high-grade dysplastic adenomas affecting the caecum, ascending colon, hepatic flexure, or appendix.
- Symptomatic inflammatory conditions (i.e. Crohn's terminal ileitis or right-sided colitis).
- Benign right colonic stricture.
- Right colonic perforation.

Preoperative preparation

- Consent with specific complications, including anastomotic leak, stricturing, ileus, hernia, and damage to ureter/gonadal vessels/duodenum.
- Group and save.
- No bowel preparation required.
- Review all images and preoperative colonoscopies prior to procedure.

Positioning and theatre set-up

- Urethral catheterization.
- *Supine*. Will need tilt for laparoscopic procedures.
- If performing laparoscopically, stand on left of patient with stack positioned in right cranial position. A gel pad and body strapping will be required.

Procedure

Open approach

- Midline laparotomy incision centred on umbilicus.
- Examine the abdomen and assess for micro-metastatic spread not detected on preoperative imaging. Also assess the tumour whilst handling as little as possible.
- Stand on patient's left and retract the caecum medially.
- With diathermy or scissors, dissect up the 'line of Toldt' (peritoneum between the right paracolic gutter and large bowel). Develop the avascular plane between the posterior abdominal wall and mesocolon. Ensure to preserve the right ureter, right gonadal vessels, and the duodenum.
- Just beneath the right gastro-epiploic arcade, divide half the greater omentum. This will enter the lesser sac.
- Clamp, divide, and transfix the ileocolic pedicle vessels close to their origins off the superior mesenteric vessels.
- Also clamp, divide, and transfix the right colic (if encountered) and right branch of middle colic vessels.
- Place crushing clamps at the intended resection margins, with non-crushing clamps proximal and distal to this. An alternative is to use a linear cross-stapler across the ileum and transverse colon.

- Divide the bowel flush with the crushing clamps, and clean the inside of the lumen with swabs soaked in povidone.
- Perform a single-layer end-to-end hand-sewn anastomosis with interrupted PDS® or Vicryl® 3.0 sutures.
- Alternatively, a stapled anastomosis can be performed.
- Close any mesenteric windows.

Laparoscopic approach

- Establish pneumoperitoneum.
- Three or four ports are required, depending on surgeon's preference. Usual port configuration includes 11mm umbilical, 12mm LUQ (for stapler), 11mm LLQ, and 5mm RLQ.
- Dissection can be performed lateral to medial (similar to open approach) or medial to lateral (described below).
- Tilt the table with head down and right side up.
- An energy device (such as Harmonic® scalpel) can be used.
- Identify the ileocolic pedicle by stretching the caecum superiorly.
- Divide the ileocolic vessels with endoclips or vascular staples (do 'high ligation' if resection for malignancy).
- Continue upward dissection, being cautious not to cause duodenal injury. Also bluntly dissect laterally between the colonic mesentery and retroperitoneum, ensuring not to damage the right ureter and gonadal vessels.
- Dissect laterally up the 'line of Toldt', whilst retracting the caecum and ascending colon medially.
- Incise the greater omentum off the proximal transverse colon and hepatic flexure.
- Finally divide the right colic (if encountered) and right branch of the middle colic vessels to fully mobilize the right colon.
- Extend the umbilical port incision to 5cm and place a wound protector into the wound.
- Extract the specimen and perform an extracorporeal ileocolic anastomosis (hand-sewn or stapled).

Closure

- Mass closure to laparotomy/umbilical incision if laparoscopic.
- Port sites ≤10mm need musculo-fascial closure.

Post-operative care

- Enhanced recovery programme.

Complications (specific to the procedure)

- Conversion to open procedure (from laparoscopic), 10–20%.
- Anastomotic leak, 2–5%
- Intra-abdominal bleeding, 5%.
- Port site infection, <5%.
- Paralytic ileus, common.

Wide local excision of breast lesion

Breast-conserving surgery abides by oncological principles for treating breast cancer whilst considering aesthetics. Wide local excisions (WLEs) are used for unifocal carcinoma *in situ* or tumours and generally aim to excise <20% of the ♀ breast.

Mastectomy is performed for large tumours, proportionally large tumours in small breasts, multifocal disease, central tumours, recurrence following previous WLE and radiotherapy, and ♂ breast cancer patients. Always give the patient a choice between breast-conserving surgery and mastectomy.

Preoperative preparation

- Consent with specific complications, including breast haematoma, cosmetic disfiguration, seroma formation, and positive resection margins necessitating further surgery.
- Deep, non-palpable tumours require same-day preoperative wiring by the radiologists. This allows for a wire-guided WLE.
- Axillary node sampling is usually performed as well.
- Group and save.

Positioning and theatre set-up

- Review mammograms and other imaging. If wire-guided WLE, read any reports from the radiologists regarding position of tumour in relation to wire.
- Supine with arm extension to allow access to the axilla.

Procedure

- Use a peri-areolar incision, if possible, for a better cosmetic outcome.
- Mark the lump and the intended incision.
- Removal of skin only required if tumour skin-infiltrating or skin changes evident.
- Incise through skin, subcutaneous fat, and fascia with scissors or a diathermy microdissection needle (Colorado®).
- If wire-guided WLE, follow the wire to localize the tumour.
- Hold a skin flap up with skin hooks to allow deeper dissection.
- Dissect down to pectoralis major fascia. Extend dissection circumferentially around the tumour (take care not to cut into the tumour). Aim to excise a minimum of 1cm normal breast tissue around the palpable tumour.
- Take tumour and dissected tissue off the pectoralis major fascia.
- Ensure to mark the specimen with sutures/clips, depending on the agreed protocol with local pathologists. If any margins are involved, it allows identification of cavity borders that require operative re-excision (the minimum acceptable margin is 1mm, less than this requires re-excision of that border).
- Meticulous haemostasis is vital. Take time on this.
- Apply clips to all borders of the cavity to direct adjuvant radiotherapy.

Closure

- Absorbable sutures to the fascial and subcuticular layers.
- Steristrips over the sutured incision.
- Drains usually not required.

Post-operative care

- Home on same day, usually with breast specialist nurse support.
- Wound check in 1 week.
- MDT discussion once histopathological analysis complete.

Complications (specific to the procedure)

- Breast haematoma, 5%.
- Wound infection, 5%.
- Seroma.
- Keloid scarring.

Femoral embolectomy

Acute embolic occlusion leads to acute limb ischaemia and is a vascular emergency that requires immediate operative intervention if imminent threat to limb viability. If ischaemia present, but no imminent threat, obtain urgent angiography (CT/MRI) to ascertain level and extent of disease. Certain emboli are amenable to catheter-directed thrombolysis.

Indications

- Strong clinical suspicion or radiologically proven embolic occlusion of the distal superficial femoral artery (SFA) or popliteal artery manifesting as acute limb ischaemia.

Preoperative preparation

- Can be performed under GA or LA.
- Consent with specific complications, including haemorrhage, pseudoaneurysm, compartment syndrome, reperfusion injury and limb loss. Other procedures may include thrombolysis, fasciotomies, and proceeding to a bypass procedure.
- Group and save and clotting.
- Single-dose antibiotics on induction.

Positioning and theatre set-up

- *Supine*. Need ability to gain access to groins, so may require table extensions.
- Surgical loupes for operative magnification.
- Resources for on-table angiography if required.

Procedure

(See Fig. 21.4.)

- Assess and document the peripheral vascular status on table prior to starting the procedure. Document time and findings of assessment.
- Oblique groin incision.
- Deepen dissection through fascia and exposure vasculature.
- Expose common femoral artery (CFA), SFA, and profunda femoris (PF) artery. Establish control of all three vessels with slings.
- Transverse arteriotomy stab (size 11 blade) usually close to the origin of the PF.
- Pass an uninflated 4F Fogarty embolectomy catheter into each vessel beyond the level of the emboli. Inflate the catheter balloon gently and withdraw for clot retrieval. Repeat until no further clot to ensure full clot retrieval.
- Flush heparinized saline into each vessel in turn, and apply vascular clamps.
- Once good inflow and backflow achieved, close arteriotomy with 1° closure or with small vein patch if concern about vessel stenosis. Use Prolene® 6.0 sutures with micro-instruments. Can do 1° closure with interrupted or continuous suture.
- Palpate distal pulses to confirm return of circulation. Note time.
- If thrombosis discovered, surgical reconstruction or bypass may be required.
- Four compartment fasciotomies should be performed if high risk of compartment syndrome (i.e. if muscles are tense or prolonged preoperative ischaemia).

Closure

- Absorbable sutures for fascial and subcuticular layers.

Post-operative care

- Regular monitoring of distal limb pulses, perfusion, and pain.
- IV heparin infusion with appropriate APTT monitoring.
- Will need long-term coagulation.

Complications (specific to the procedure)

- Haematoma formation, 5%.
- False aneurysm formation, <1%.
- Reperfusion injury and AKI.
- Distal limb compartment syndrome.

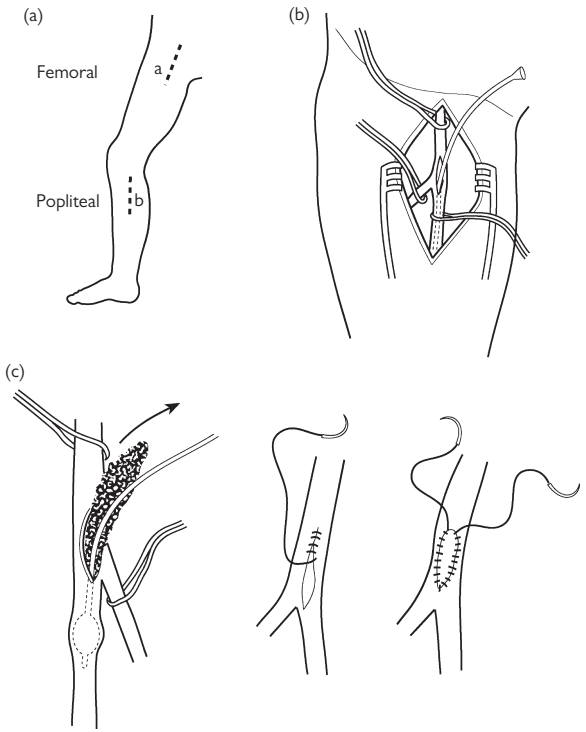

Fig. 21.4 Vessel exposure and control. (a) Incisions for femoral and popliteal artery exposure. (b) Sling control of CFA, SFA, and PF. (c) Femoral embolectomy and closure without and with a vein patch.

Below-knee amputation

Indications

- Acute limb ischaemia with non-viable distal lower limb (disease below popliteal artery level).
- Critical PVD below popliteal artery level unamenable to angioplasty or reconstruction.
- Severe trauma below mid-tibial level.
- Bone/soft tissue tumours and congenital deformities below mid-tibial level.

Preoperative preparation

- Can be performed under GA, or spinal or regional anaesthesia.
- Consent with specific complications, including haemorrhage, infection, failure of stump healing, and phantom limb pain.
- Group and save essential. Cross-match blood when PVD absent.
- Proper assessment of viability of lower limb to ascertain appropriate level of amputation. Preserve the knee joint where possible.

Positioning and theatre set-up

- Supine.
- Mark skin flaps.
- Can use a tourniquet on the thigh if absence of PVD.

Procedure

The two most popular approaches for BKAs are the Burgess posterior flap and the skew flap. The Burgess posterior flap is used more commonly in vascular cases and is described below.

Burgess posterior flap
(See Fig. 21.5)

- Mark flaps, with the anterior flap ~10cm below the tibial tuberosity. The posterior flap is at least double the length of the anterior flap and will provide coverage over the stump.
- Make the incision over the markings and deepen with diathermy or blade. Ensure haemostasis as you dissect. Blood loss can be rapid.
- Incise straight down through muscle and onto bone. Identify anterior tibial and peroneal neurovascular pedicles. Ligate these and ensure to divide the nerves under tension to encourage nerve retraction.
- Use a periosteal elevator to strip the periosteum off the tibia and fibula (go at least 1–2cm above level of transection). Aim to transect the fibula 2.5cm higher than the fibula.
- Tibia and fibula divisions can then be done with an amputation electric saw or a Gigli saw. Bevel the anterior tibia with a rasp.
- Strip the deep posterior muscles off the tibia below the tibial transection level. Continue down to the level of the posterior flap distally.
- Identify the posterior tibial vessels and ligate. Divide the posterior tibial nerve clean and under tension to encourage retraction.
- Trim the soleus and gastrocnemius muscles down to the lower end of the posterior flap. The bulk of the flap should be adequate to cover the bony stump.

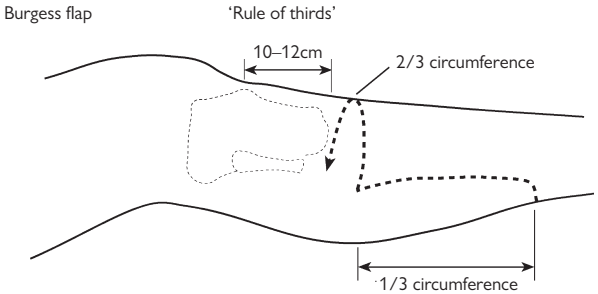

Fig. 21.5 Burgess posterior flap.

- Remove the limb and place on a drape on the theatre floor.
- Smooth over the bone ends with a rasp and bone nibblers. Apply bone wax.
- Bring the posterior flap over the bone and suture it to the deep fascia of the anterior muscle compartment with strong Vicryl® 1 sutures.

Closure

- Close skin with clips or interrupted nylon sutures.
- Apply wound closure strips and a stump bandage.

Post-operative care

- Elevate leg.
- Remove sutures at 14 days.
- Early physiotherapy to knee joint to promote range of movements.
- Prosthetic fitting once stump healed.

Complications (specific to the procedure)

- Flap necrosis, <5%.
- Haematoma, 5%.
- Phantom limb, up to 50%.

Chapter 22

Remote and rural surgery

Remote and rural surgery

For many years, surgeons working in remote and isolated areas have failed to receive the recognition they deserve. Anyone living in a remote and rural area will know of lives saved and diseases cured by locally based surgeons. Delivery of surgical services to remote and rural areas remains an intractable problem in many countries. In 2009, the Surgical Clinics of North America dedicated a single edition to Surgical Practice in Rural Areas. Although mainly directed to practice in the USA, there are descriptions of the situation in Australia and Scotland.[1] Recently, the Royal College of Surgeons of Edinburgh has published a report Standards informing delivery of care in rural surgery[2]; it provides explanations of how surgical services can be provided to remote and rural communities in a safe and appropriate manner.

Rural surgical practice

Recognition of limitations

- To work as a consultant in an isolated area, the surgeon must be resourceful and relish taking on challenges that might, in other settings, be handed to someone else.
- There can be no compromise on the quality of care delivered and the ability to recognize personal limitations is essential.
- There can never be hesitation in transferring care to another hospital if the patient will do better there.
- The rural surgeon can find himself unsupported and in unfamiliar territory; he must have coping strategies to deal with this eventuality.

Skill sets

- The rural surgeon must possess a range of skills appropriate for his environment.
- Training must prepare him to deal with commonly encountered surgical conditions; these will vary from rural hospital to rural hospital.
- Some skills will be specific, but others will be generic, and it is these generic skills that will be called on when unfamiliar situations arise.
- In addition to the skills associated with general surgery, a rural surgeon should be able to perform Caesarean sections and burr holes for evacuation of extradural haematoma, be a skilled GI endoscopist and cystoscopist, and be capable of managing straightforward fractures and joint dislocations and dealing with minor ENT and ophthalmological emergencies.
- A substantial part of a rural surgeon's work is non-operative care, probably more than in larger hospitals, and some conditions they care for could, in other circumstances, be the remit of another specialist.
- Most surgery will be similar to that met in any general surgical practice; it is only rarely that conditions unique to the rural environment, such as ratfish spine injuries in fishermen[3], will be encountered.

Skill maintenance

- The conundrum of how to maintain skills with a low workload is much debated. The volume/quality relationship is well understood for many major procedures and there is little argument that in some circumstances, teams of experienced personnel get better results than individuals who are infrequent practitioners with limited resources. Thus, some patients will be better managed in another hospital than in the rural general hospital (RGH).
- In the rural setting, there is less work to be done because the population is smaller and there are concerns about doing enough to maintain skills. If the surgeon has much experience and a good training, it is probable he will require fewer numbers to maintain his competence.
- There is a difference between the surgeon who does not do many of a procedure when there are plenty of it to be done and the surgeon who does not do many of that procedure but they are all that must be done. The latter surgeon will probably maintain his skills; the former will not.

- Generic skills, such as good dissection technique, safe suturing, and effective anastomosis construction, and extensive anatomical knowledge will be commonly used and form the bedrock of much rural surgery.
- Some surgeons will have included in their contracts the facility to spend time in a larger unit, interacting with colleagues and carrying out surgical procedures alongside them; this will support skill maintenance.

Work practice

- Working in a 'small' rural environment can be daunting and intimidating; the ability of a surgeon to be confident that the way he works is safe and effective is paramount.
- Unlike in bigger hospitals, the rural surgeon is more likely to be doing procedures that others less senior would normally do and unless he enjoys this, he will find the job difficult.
- He must get satisfaction from coming into hospital from home at 3 a.m. to insert a urinary catheter and relieve a man's pain from urinary retention—if he doesn't, he is in the wrong job!
- Patients benefit from the facility to have their 'minor or moderate' procedure carried out close to their own home; so day care and short stay surgery makes up an important part of a rural surgeon's workload.

Peer support

- Smaller hospitals have fewer staff and there is less opportunity for direct peer interaction. At the most, there will be three consultant surgeons in a rural hospital.
- Professional isolation is a commonly perceived problem and mechanisms must be found for supportive and reassuring interactions and conversations with like-minded professionals. This is difficult to achieve, but contact with others by telephone, videolink, and attendance at meetings helps.
- Peer support is another good reason for surgeons to have an arrangement to work for short periods in another hospital.

Continuing professional development (CPD)

- Rural hospitals will have less in the way of postgraduate meetings and courses, placing a greater responsibility on the surgeon to seek other ways of ensuring he can actively involve himself in his own CPD.
- Greater use of online resources and video conferences will be required.
- Study leave allocation must be fully and sensibly used, recognizing that travel times may be greater and allowance must be made for this.
- Cover during leaves of absence needs to be carefully and judiciously managed by colleagues and administrators.

Lifestyle

- Living in a small remote and isolated area has advantages and disadvantages. The ability to live and work in a gold fish bowl is not for everyone.
- The rural surgeon is a big fish in a little pond and along with this comes professional and social responsibilities that might not pertain in an urban environment.

- On-call rotas are more onerous; at best, they will be one in three.
- If there is an internal (prospective rather than locum) cover arrangement, then for 20 weeks of the year, the on-call will be one in two.
- If married with children, meeting the needs of the family is important; getting this part of the job wrong is a cause of people leaving post in remote communities.
- Remuneration should be commensurate with time and commitment spent in the job; with ↑ personal involvement in rural hospitals, it is impossible to squeeze this into ten programmed activities per week.

Rural general hospitals

> 'The Rural General Hospital undertakes the management of acute medical and surgical emergencies and is the emergency centre for the community, including the place of safety for mental health emergencies. It is characterized by more advanced levels of diagnostic services than a community hospital and will provide a range of outpatient, day-case, inpatient and rehabilitation services.'

Delivering for Remote and Rural Healthcare, NHS Scotland 2007 RGHs are a concept of NHS Scotland; they serve small populations of between 20 000 and 30 000 people. They are located on the mainland or on islands. They are >65 miles from the nearest district general hospital, to which the travel time is >90 min.

Individual RGHs differ in the services they provide, but as a minimum, they must offer Level 2+ care that encompasses:

- Immediate care for medical and surgical emergencies.
- Robust arrangements to manage obstetric emergencies.
- Provision for local non-specialist care of seriously ill children.
- Arrangements for safe care of mentally ill patients.
- Essential diagnostic services.
- Effective arrangements for transfer of seriously ill patients.

Core services

To deliver Level 2+ care, RGHs will require a set of core services. These will include but are not be limited to:

- 24h A&E department with nurse-run urgent care for management of minor injury and illness.
- An on-site doctor available day and night.
- Locally based consultant-led services in general medicine, general surgery, and anaesthesia.
- Reliable obstetric service—midwife- or consultant-led.
- A defined system for managing children's health.
- ICUs providing Level 3 care are not available in RGHs, whilst high dependency (Level 2) care may be. Brief Level 3 care for patients prior to transfer must be available.
- Digital radiography, ultrasonography, and CT scanning.
- Defined, but limited, biochemistry and haematology services.
- Cross-matching of blood.
- Upper and lower GI endoscopy.
- Essential cardiac investigation facilities, including stress testing and echocardiography.
- Pharmacy.
- Telemedicine, e-health, and videolinking.

Care pathways

- The acute hospital care pathways for rural patients are a way of providing insight into how patients can access surgical care in remote and rural communities.
- The care pathways described are based on those developed by the Service Models and Care Pathways subgroup of the (Scottish) Remote and Rural Implementation Group (RRIG).
- The conditions ascribed to care pathways are those that should be within the remit of a well-trained rural general surgeon.
- The lists of conditions are not exhaustive. RGH consultant surgeons do not all have the same set of skills, and the ability to manage specific conditions varies between RGHs.
- RGHs have differing visiting arrangements with other hospitals.
- The care pathways are divided into two groups:
  - Non-emergency conditions: seven pathways.
  - Emergency conditions: three pathways.
  - Pathways for malignant disease are considered separately.
- A draft of the care pathways was sent to 62 doctors (13 physicians, 19 surgeons, 16 anaesthetists, one obstetrician, two paediatricians, seven GPs, and four medical directors). They were then validated by two rural physicians, three rural surgeons, the Regional Nursing Advisor, and the Director of Regional Planning of the North of Scotland Planning Group (NoSPG).
- Elements used in the care pathways include:
  - Presenting conditions: non-emergency and emergency.
  - First point of access to health care: primary care practitioner, NHS 24, 999, A&E, and self-referral.
  - Initial specialist referral to RGH, visiting or another consultant.
  - Advice obtained from a visiting or another consultant.
  - Investigations carried out in RGH or another hospital.
  - Management is in the RGH or another hospital.
- Definitions:
  - An RGH consultant is locally based and has the RGH as his/her main place of work.
  - A visiting consultant has his/her main place of work in another hospital but regularly works in the RGH.
  - Another consultant has his/her place of work in another hospital but does not visit the RGH.
  - Another hospital is any (usually larger) hospital than the RGH.

Non-emergency conditions (non-malignant)

Pathway 1

- The patient is managed completely by the rural health care service.

Presentation:	RGH A & E department
Referral:	RGH consultant
Advice	Another consultant
Investigation:	RGH
Management:	RGH

Adult general surgery

Operative care

- Anal fissure, acute and chronic
- Anal fistula (simple)
- Anal warts
- Benign breast lumps
- Bile duct stones
- Cholelithiasis
- Colonic polyps
- Diverticular disease of colon
- Enterocutaneous fistula
- Epididymal cyst
- Haemorrhoids
- Haematuria
- Hernia: inguinal, femoral, incisional, umbilical, and ventral
- Hydrocele
- Gastric outlet obstruction
- Gastrocolic fistula (simple)
- Gynaecomastia
- Lipoma
- Phimosis
- Pilonidal sinus
- Pyogenic granuloma
- Sebaceous cysts
- Sialolithiasis (uncomplicated)
- Skin lesion (excision or biopsy)
- Varicocele
- Varicose veins (simple)
- Volvulus (chronic)

Non-operative care

- Balanitis
- Barrett's oesophagus
- Benign prostatic hypertrophy
- Bladder calculi
- Cystitis
- Epididymo-orchitis
- Faecal incontinence
- Gastritis and duodenitis
- Gastro-oesophageal reflux disease
- Goitre (multinodular)
- Hernia, paraoesophageal
- Oesophageal varices
- Pancreatitis (chronic)
- Peptic ulcer disease
- Peripheral vascular disease
- Prostatitis
- Rectal prolapse
- Renal and ureteric calculi
- Stoma care
- Ureteric calculi
- Venous ulcers

Orthopaedic

- Dupuytren's contracture
- Osteoporotic fractures
- Mal-and non-union of fractures
- Meniscus/knee problems
- Osteoarthritis
- Osteomyelitis
- Rotator cuff syndrome
- Torticollis

Obstetrics and gynaecology

- Bartholin cysts
- Mittelschmerz

Pathway 2

The patient is managed within the rural health care service, with advice from a consultant who either visits the RGH and works in another hospital or works in another hospital but does not visit the RGH.

Presentation:	Primary care
Referral:	RGH consultant
Investigation:	RGH
Advice:	Visiting or another consultant
Management:	RGH

Adult general surgery

Operative

- Anal fistula (complex)
- Bile duct stones
- Carpal tunnel syndrome
- Enterocolic fistula
- Gastrocolic fistula
- Haematuria

Non-operative

- Achalasia
- Aneurysms, other
- Amoebic abscess
- Aortic aneurysm
- Benign prostatic hypertrophy
- Cystitis
- Faecal incontinence
- Intracranial abscess
- Hydronephrosis
- Pancreatitis (chronic)
- Prostatitis
- Rectal prolapse
- Renal and ureteric calculi
- Venous ulcers (chronic)

Orthopaedics

- Bursitis
- Cervical disc disorders
- Dupuytren's contracture
- Internal derangement of knee
- Mal- and non-union of fractures
- Osteomyelitis
- Osteoarthritis
- Patella disorders
- Rotator cuff syndrome

Obstetrics and gynaecology

- Early pregnancy haemorrhage
- Endometriosis
- Mittelschmerz
- Parametritis
- Pelvic cellulitis
- Pyometria
- Salpingitis
- Vesico-vaginal fistula

Pathway 3

The patient is initially managed and investigated within the rural health care service, but on advice from a visiting consultant or a consultant from another hospital, the management is completed in another hospital.

Presentation:	Primary care
Referral:	RGH consultant
Investigation:	RGH
Advice:	Visiting or another consultant
Management:	Another hospital

Adult general surgery
- Achalasia
- Amoebic abscess
- Anal fistula (complex)
- Abdominal aortic aneurysm
- Barrett's oesophagus
- Bile duct stones
- Colonic polyps
- Empyema
- Enterocutaneous fistula
- Gastrocolic fistula
- Gynaecomastia
- Hepatic abscess
- Hiatus hernia
- Hydrocele
- Hydronephrosis
- Intracranial abscess
- Oesophageal varices
- Pancreatitis, chronic
- Pancreatic pseudocyst
- Paraoesophageal hernia
- Peripheral vascular disease
- Post-phlebitic syndrome
- Renal and ureteric calculi
- Sialolithiasis
- Urethral fistula
- Varicocele
- Varicose veins
- Venous ulcers, chronic

Orthopaedics
- Cervical disc disorders
- Dupuytren's contracture
- Mal- and non-union of fractures
- Internal derangement of knee
- Meniscus problems
- Osteomyelitis
- Patellar disorders
- Rotator cuff syndrome
- Torticollis

Obstetrics and gynaecology
- Pyometria
- Vesico-vaginal fistula

Pathway 4

The patient is managed in the rural health care system by a visiting consultant.

Presentation:	Primary care
Referral:	Visiting consultant
Investigation:	RGH and/or another hospital
Management:	RGH

Visiting consultants

This will include conditions that are within the scope of care possible at the RGH. The visiting consultants are contracted by the RGH from another hospital and work regularly in the RGH; if they do not visit, they will fall into the category of another consultant:
- ENT.
- Obstetrics and gynaecology.
- Ophthalmology.
- Oral surgery.
- Orthopaedics.
- Urology.

Pathway 5

The patient is initially managed within the rural health care service by the visiting consultant but is completed in another hospital.

Presentation:	Primary care
Referral:	Visiting consultant
Investigation:	RGH and/or another hospital
Management:	Another hospital

Visiting consultants

This will include conditions that are outwith the scope of care possible at the RGH:

- ENT.
- Obstetrics and gynaecology.
- Ophthalmology.
- Oral surgery.
- Orthopaedics.
- Urology.

Pathway 6

The patient is referred to another consultant in another hospital who plans investigations in either the RGH or another hospital but decides that the management can be carried out in the RGH.

There are very few conditions that follow this care pathway. There will be in place an arrangement whereby these kinds of referrals are permitted to take place. The surgical consultants that come into this category will include:

Presentation:	Primary care
Referral:	Another consultant
Investigation:	RGH and/or another hospital
Management:	RGH

- Breast surgery.
- Cardiothoracic.
- Coloproctology.
- Endocrine surgery.
- Neurosurgery.
- Paediatric surgery.
- Pancreaticohepatobiliary.
- Plastic and burns.
- Upper GI.
- Vascular.

An example of a condition that could be managed by this care pathway would be an elderly man with multiple comorbidities who presents with an expanding AAA. The patient is reviewed by a vascular surgeon in another hospital, and this consultant concludes that operative surgery does not have a role in the man's management. The patient is advised of this and any further care is provided by the RGH.

Pathway 7

The patient is referred to another consultant in another hospital who decides, after investigations carried out in the RGH and/or another hospital, that the management should occur in another hospital.

Patients with major, uncommon, or unusual surgical problems where neither the skill nor the expertise to fully investigate and manage the condition is available in the RGH are referred directly to another consultant in another hospital. There will be in place an arrangement whereby these kinds of referrals are permitted to take place.

Presentation:	Primary care
Referral:	Another consultant
Investigation:	RGH and/or another hospital
Management:	Another hospital

Emergency conditions

The patient arrives at the A&E department as an emergency by ambulance or other means of transport, having telephoned 999 or NHS 111, or as a self referral or sent by primary care.

Pathway 1

Presentation:	RGH A&E department
Referral:	RGH consultant
Investigation:	RGH
Management:	RGH

- Abortion
- Abscess: cutaneous and others
- Aneurysm rupture
- Animal bites
- Appendicitis
- Birth trauma
- Bolus obstruction oesophagus
- Brain injury focal and diffuse
- Burns up to 30%
- Bursitis
- Cellulitis
- Cervical disc disorder
- Cholangitis
- Cholecystitis
- Crush injuries
- Ectopic pregnancy
- Embolism arterial—limb or mesenteric
- Epididymo-orchitis
- Epistaxis
- Foreign bodies in GI tract, ear, eye, GU tract, airway
- Fractures traumatic, stress, osteoporotic, pathological
- Gas gangrene
- Haemarthrosis
- Haematemesis
- Haematuria
- Haemorrhage GI, obstetric, traumatic
- Haemorrhoids (prolapsed or thrombosed)
- Head injury
- Intestinal obstruction and pseudo-obstruction
- Intussusception
- Irreducible hernias
- Ischaemic colitis
- Jaundice obstructive
- Joint dislocations

- Locked knee
- Lymphadenitis (acute)
- Mastoiditis
- Necrotizing fasciitis
- Otitis externa and media
- Ovarian cyst torsion or rupture
- Pancreatitis (acute)
- Paralytic ileus
- Peptic ulcer perforation
- Perianal haematoma
- Perforation abdominal viscus
- Peritonitis (acute)
- Peritonsillar abscess (quinsy)
- Puerperal sepsis
- Pyogenic arthritis
- Pyogenic granuloma
- Pyothorax
- Renal calculi
- Retained placenta
- Rib fractures
- Salpingitis
- Sialolithiasis and sialadenitis
- Skin lacerations
- Testicular torsion
- Tonsillitis
- Torticollis
- Trauma musculoskeletal, intra-abdominal, renal, thoracic
- Ureteric colic
- Urinary retention
- Vascular injury
- Volvulus

Presentation:	RGH A&E Department
Referral:	RGH Consultant
Advice:	Another consultant
Investigation:	RGH
Management:	RGH

Pathway 2

- Abortion
- Abscess hepatic, pancreatic, urethral
- Aneurysm rupture abdominal or thoracic aorta ± dissection
- Brain injury focal and diffuse
- Burns up to 30%
- Cardiac laceration
- Crush injuries
- Joint dislocations (complex)
- Embolism arterial limb or mesenteric
- Epistaxis (severe)
- Extra- and subdural haemorrhage
- Flail chest
- Fournier's gangrene
- Fractures complex
- Haemarthrosis
- Haemopericardium
- Haemopneumothorax
- Haemorrhage gastrointestinal and obstetric
- Internal derangement of knee
- Intussusception (children)
- Ischaemic colitis
- Jaundice obstructive
- Major trauma to liver, lungs, and kidneys
- Mastoiditis
- Necrotizing fasciitis
- Neurological injuries limbs
- Otitis media
- Pancreatitis acute
- Pelvic cellulitis
- Perforated eardrum
- Puerperal sepsis
- Pyogenic arthritis
- Pyometria
- Rib fractures (multiple ± flail chest)
- Sialolithiasis and sialadenitis
- Vascular injuries

Presentation:	RGH A&E department
Referral:	RGH consultant
Advice:	Another consultant
Investigation:	RGH/another hospital
Management:	Another hospital

Pathway 3

- Abortion (complicated)
- Abscess (major)
- Aneurysm rupture abdominal or thoracic aorta ± dissection
- Birth trauma
- Brain injury focal and diffuse
- Burns >30%
- Cardiac laceration/contusion
- Crush injuries (major)
- Joint dislocations (complex)
- Eclampsia
- Embolism limb or mesenteric
- Epistaxis (uncontrolled)
- Extra- and subdural haemorrhage
- Fractures (complex)
- Fracture neck of femur
- Gas gangrene
- Gynaecological (major) problems
- Haemorrhage (uncontrolled) gastrointestinal, obstetric
- Intussusception (children)
- Ischaemic colitis
- Neurological injury limbs
- Obstructive uropathy
- Oesophageal varices haemorrhage
- Pancreatitis acute (severe, Ranson/Imrie >3)
- Rib fractures (multiple ± flail chest)
- Skin loss (major)
- Trauma (major) abdominal, thorax, limbs
- Ulcerative colitis complications
- Vascular injuries limb, neck

Patient transfer

- A cornerstone of care in remote and isolated places is the ability to transfer a patient to a larger, better-resourced hospital if required.
- No rural dweller can have confidence in their local care system if robust transfer arrangements are not in place.
- The transfer system covers both the means of transportation and the place to which the transfer occurs.
- Retrieval systems for neonatal, paediatric, and adult patients with serious injury or illness are required.
- Under the auspices of the Scottish Ambulance Service, a centralized system ScotSTAR (Scottish Specialist Transport and Retrieval), combining all three, has been in existence since 2014.
- Retrieval teams have consultants, trainees and middle-grade doctors, advanced nurse practitioners, and nurses—all trained in transportation of critically ill patients.
- Patients are initially managed and stabilized by the RGH clinical staff; further emergent and urgent care, along with preparation for transfer, is carried out by the retrieval team.

- In the absence of a retrieval team, locally based staff accompany the patient; this can cause a short-lived deficit in available RGH staff. Mechanisms to resolve the logistical problems of how these staff return to base and what insurance cover they have should be in place.
- Fixed wing aircraft and helicopters are often required, but in mainland situations, ambulances can sometimes be just as effective. Some countries make use of high-speed boats for patient transfer.
- There are rare circumstances when weather conditions are so severe that patients cannot be transferred; then RGH staff have to do the best they can, often with guidance from another hospital.

Malignant conditions

- The management of a patient with a malignant or potentially malignant condition must always abide by accepted cancer referral guidelines, irrespective of where he or she lives.
- Rural patients should have the same care as their urban counterparts, with the exception that they may have to be away from home and travel greater distances for that care.
- Rural hospitals make use of MDTs based in larger hospitals. The results of investigations will be available and the best form of management decided according to best practice. In ideal circumstances, MDTs will involve local rural staff (medical and nursing) by some form of electronic communication; well-managed video conferencing is ideal.
- If the surgical procedure required for the patient can be performed in the RGH, then that is where it should be done. Excision of skin cancers and some localized and low-risk soft tissue malignancies, resection of colon cancers, and, in well-defined and agreed circumstances, some breast cancer excisions can be carried out in RGHs.
- If the operative skills or resources are not available in the RGH, two approaches are possible:
  1. The patient's local rural surgeon performs the procedure in another hospital, working alongside other colleagues.
  2. Another surgeon in another hospital carries out the procedure.
- Aftercare can usually be carried out in the RGH.
- Chemotherapy can, under the supervision of either a visiting or other hospital-based oncologist, be carried out in the RGH.
- All radiotherapy will be carried out in another hospital.
- Care of patients presenting late with widespread malignancy (there is some evidence that this occurs more often in remote communities) is supportive or palliative and is provided locally.
- Patients requiring treatment away from home at another hospital should have their travel and accommodation paid for by the health authority which covers their area. Hospitals who regularly serve patients from remote and rural areas often make provision for patient and supportive relative accommodation.
- End-of-life care of patients with malignant disease must ensure that long-distance journeys away from home for consultations or treatment are restricted to those which will be of real value.

Training for remote and rural surgery

- Several attempts to establish training programmes for remote and rural surgery have been unsuccessful.
- Well-intentioned rural surgical trainees usually end up working in a larger hospital.
- Until all training bodies recognize remote and rural surgery as a legitimate and separate surgical specialty, the situation is unlikely to change[4].
- The specialties of rural medicine and rural anaesthesia must also be recognized.
- Training for rural surgery will be predominantly general surgical but will include time learning essential skills in trauma and orthopaedics, urology, obstetrics and gynaecology, otorhinolaryngology, and ophthalmology. Some time will be spent in a neurosurgical centre learning to perform burr holes.

Recruitment and retention

- Hospital human resources departments must make concerted efforts to employ and keep surgeons in rural hospitals.
- A recent European Regional Development funded the Northern Periphery Programme (NPP) project—'Recruit and Retain'— and has produced a business model to help human resources departments develop a more dynamic recruitment and retention strategy for remote and rural areas.
- Eight partners from seven Northern European countries and Canada worked for 3y to find solutions.
- An evidence base of information about living and working in remote areas was generated from over 5000 replies to an online questionnaire, 76 structured interviews, and responses from 19 public sector organizations.
- The evidence was used to develop and pilot 29 products and services designed to aid recruitment and retention in rural areas.
- The NPP 'Recruit and Retain' project published a Business Model with Seven (practical) Steps[5]
- In brief, the Seven Steps describe:
  - Proactive and regular planning for recruitment and relocation of staff with a 'Yearly Wheel'.
  - Proper definition of who is to be looked for, recognizing that recruiting the 'wrong' person can be counterproductive.
  - Provision of honest information about the advantages and disadvantages of working and living in remote and rural areas.
  - Involving local communities in the recruitment and retention process.
  - Putting in place active measures to support staff and their families.
  - Ensuring that the education and training needs of staff are met.
  - Recognition that providing health care to remote and rural populations is expensive and that the necessary money needs to be available.

Locums

- Locums are required to cover leaves of absence and sickness and to fill vacancies before definitive appointments are made.
- Finding suitable locum consultant rural surgeons is difficult, and the ones with the correct skill mix and experience are in short supply.
- Some surgeons make locum employment a career and some can be employed on a regular or planned basis for a particular hospital, providing some continuity.
- The quality of care delivered by a locum consultant can be adversely affected by unfamiliarity with the hospital and staff, inappropriate expectations of what can be done, and an inadequate skill set.
- The Royal College of Surgeons of England[6] produced principles and standards for locum surgeons in this they state:
  - Locum consultant surgeons should be on the specialist register or within 6 months of receiving it.
  - Locum appointments should not be longer than 12 months.
  - Suitable checks should be carried out to ensure suitability.
  - Locum agencies should be NHS professionals or ones which are part of the National Framework Agreement.
  - Locums not on the specialist register should be supervised by a named consultant.
- CPD for locum consultant surgeons is an issue and the necessary appraisal and validation are difficult to structure and organize for someone moving from one job to another.
- The magnitude of the locum problem in rural hospitals must not be underestimated; in a single RGH with 17 consultant posts, a total of 239 different locum consultants were employed over an 8y period (2001–2009); 188 were short-term locums (<3 months) and they were employed on 535 separate occasions[7].

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Surgery in tropical diseases

Medicine in the tropics

Key facts

Although the principles of surgical care are the same in the tropics as in developed countries, there are important differences as resources are limited in the tropics.

- There are major transport problems.
- Treatment is often complicated by underlying disease such as anaemia and malnutrition.
- There are differences in cultural attitudes to disease, e.g.:
  - Stomas are often considered an intolerable burden in some societies for religious or cultural reasons.
  - STDs may carry a greater stigma than in the UK.

Treatment

Surgery on tropical diseases requires flexibility in both clinical management and surgical techniques. Doctors, irrespective of where they work, should be knowledgeable about common tropical diseases and be aware of the possibility of seeing an imported disease in a patient, with the concomitant risks to the patient and the community.

Spending time in tropical medicine

- Find out about where you are going to, the conditions under which you will work, and the local diseases. Be prepared for these conditions. Visit an occupational service before you go to plan antimalarials and vaccinations. Discuss with them options for post-exposure HIV prophylaxis.
- When you arrive, listen to local health care workers. They always know more than you do.
- Your elective is the perfect time to develop a research mind—look and ask why things are being done. Develop a relationship with a more senior doctor and find questions relevant to his/her practice. However, do not expect to get an awful lot done or make any major breakthroughs in just a few weeks.
- Beware 'getting in a bit of practice'. An elective is not designed to allow you to get in a bit of practice doing things to 'the natives' that you are not allowed to do at home. This is a real risk when Western medical students go to under-resourced tropical hospitals. By all means, expand your horizons and use your skills to their limit, but know your limits and always work within them wherever you are.
- You will always be advised to eat only cooked or peeled food. This is clearly impossible. However, when the diarrhoea starts, do not sit on it as you might in more temperate climes. We have twice had to put up drips on medical students who arrived to spend time working with us, having had diarrhoea for several days.
- In regions where dengue or malaria occurs, a fever lasting more than a day requires a visit to a doctor for a blood film or a rapid diagnostic test (RDT) and FBC, despite apparently focal signs such as diarrhoea. D Fever must be taken seriously and could always indicate malaria.

- The most dangerous time during your time in the tropics is likely to be time spent travelling. Think before you get on the bus, boat, or plane. Ask yourself: is this a safe form of transport? Can I minimize the risk in any way? Do not feel ashamed for stopping your journey and getting out of the vehicle if you realize the risk is significant. You might meet someone fascinating …
- Lastly, show respect for the local culture. It is often difficult to appreciate some things and easy to hark back to good things at home. Careless expression of such thoughts can cause great distress to people proud of what they have accomplished.

Typhoid

Key facts

- Caused by *Salmonella typhi* (Gram –ve bacilli). *Salmonella* paratyphi may cause a similar illness (paratyphoid).
- Ingested via contaminated food or water (faecal–oral or urine–oral routes from infected patients or chronic carriers).
- Up to ~21 million cases per year, with 150 000 deaths.
- Endemic in whole regions due to inadequate sewage disposal and contaminated water supplies.
- There are two clinical forms—typhoid and paratyphoid fever—collectively known as enteric fever.
- A surgeon may be the first person to see a patient with typhoid presenting with abdominal features and fever.

Clinicopathological features

- Following ingestion, the bacilli penetrate the intestinal mucosa and multiply within the mesenteric lymph nodes.
- Re-entry into the bloodstream after about 10–14 days causes a bout of 'typhoid fever'.
- Systemic complications ensue due to haematogenous spread.
- Re-infection of the gut occurs through bile or bacteraemic spread and the bacilli localize in Peyer's patches in the lower ileum, which become swollen and red.
- Infection of the bone marrow may cause neutropenia.

Clinical features

- Disease affects older children and young adults, with an incubation period of about 10–14 days.
- 'Relative bradycardia' in patients with high fever does occur.
- Onset is gradual, with swinging high fever, headache, abdominal discomfort, cough, malaise, and anorexia.
- Characteristic stepwise increase in fever occurs over several days and pea-soup diarrhoea may be a feature, although constipation is a frequent presentation.
- In severe cases, mental changes can occur with change in conscious level and psychiatric features.
- Maculopapular ('rose spot') rash may appear during the second or third week, usually on the upper abdominal and lower chest. It fades within 2–3 days.

Complications of typhoid fever

- Paralytic ileus.
- Intestinal haemorrhage.
- Cholecystitis with perforation. Bacilli in the bile can produce a carrier state which can cause isolated outbreaks of typhoid fever ('typhoid Mary' was the cook who infected food wherever she worked).
- Infected aortic aneurysm.
- Phlebitis, especially of the left common iliac vein.
- Intestinal perforation along the anti-mesenteric border of the ileum at the site of Peyer's patches in the long axis of the gut.
- GU typhoid.

- Bone and joint infection.
- Bone marrow suppression
- Myositis and myalgia.
- Parotitis and laryngitis.

Diagnosis

- Widal test is not helpful.
- If available, multiple blood cultures are the best.
- Stool and urine culture with selective enrichment media, e.g. McConkey or deoxycholate citrate agar (DCA).

Treatment

- Prolonged antibiotic treatment IV and PO for up to 28 days. Fever often does not settle for 3–5 days despite effective treatment.
- Resistance to many antibiotics has been described and it is important to use prolonged high-dose antibiotics, including chloramphenicol, ciprofloxacin, cephalosporins, and co-trimoxazole.
- Third-generation cephalosporins may not be available but, if they are, are the mainstay of treatment.
- Treatment of intestinal perforation is surgical, with lavage and closure of the perforation and IV antibiotics.

Prevention

- Immunization with a toxoid conjugate vaccine is the preferred vaccine, given IM.
- An oral vaccine is also available, but neither has 100% protection and does not protect against paratyphoid.
- General food and water hygiene precautions are vital.

Amoebiasis and amoebic liver abscess

Key facts Amoebic liver abscess is a complication of amoebic hepatitis 2° to amoebic dysentery.

Pathological features

- Caused by *Entamoeba histolytica* protozoal infection.
- Cysts ingested from infected water or food.
- Amoebae enter ileal and colonic wall, especially in the ascending colon, multiply, and may enter the portal venous circulation.
- Amoebae that traverse the liver enter the general circulation and systemic effects may occur.
- Amoebae in the liver destroy liver tissue directly, with little or no tissue reaction (no abscess 'rim'). 2° infection may occur. The central 'pus' characteristically appears 'chocolate-coloured' or 'like anchovy sauce' due to liver cell liquefaction containing liver cells and *E. histolytica*. If 2° infection occurs, it may also contain staphylococci, streptococci, or *Escherichia coli*.

Complications

- Infected individuals may be asymptomatic and act as 'carriers'.
- Intestinal amoebiasis producing dysentery and dysenteric colitis and/or amoebic appendicitis. Complications include:
  - Perforation and generalized peritonitis.
  - Haemorrhage, bloody diarrhoea, chronic anaemia of blood loss.
  - Intussusception.
- Hepatic amoebiasis and liver 'abscess'. Complications include:
  - Intraperitoneal rupture with peritonitis.
  - Intrapleural rupture with amoebic empyema.
  - Rupture into an attached loop of bowel.
  - Rupture into the pericardium.
- Extraintestinal amoebiasis may be hepatic or cutaneous.

Clinical features

Intestinal amoebiasis

- Fever, anorexia, weight loss.
- Acute or acute-on-chronic diarrhoea (may be bloody).

Hepatic amoebiasis

- Additional night sweats, rigors.
- Fever, anorexia, weight loss.
- RUQ and lower chest rigidity and tenderness.
- Right shoulder tip pain and right-sided basal changes, including a pleural rub, may also occur.
- Hepatomegaly.

Diagnosis and investigations

- Microscopy and culture of fresh hot stool for amoebae.
- Liver ultrasound to assess number, size, and distribution of abscesses and allow aspiration of pus.
- Amoebic serology—indirect fluorescent antibody test (IFAT)—is present in ~95% of patients with amoebic liver abscess.

Treatment

Medical

- Metronidazole 400–800mg tds for 7–10 days is the best treatment.
- Luminal agent is required for gut clearance—diloxanide furoate is the main treatment. Repeated abscess aspirations if abscess too big or causing symptoms.

Surgical

- Open drainage of abscess that fails to respond.
- Excision of perforated viscera or for uncontrollable haemorrhage.

Anaemias in the tropics

Key facts

- The main causes of anaemias are iron deficiency or 2° to infections and infestations.
- Others causes include:
  - Glucose-6-phosphate dehydrogenase (G6PD) deficiency.
  - Thalassaemias.
  - Sickle cell anaemia.

Sickle cell anaemia

Pathological features

- Substitution of valine for glutamic acid at the sixth position of the Hb gamma chain. Leads to sickle cell anaemia if both genes are affected (homozygotes) or sickle cell trait in single-gene carriers (heterozygotes).
- Sickle cell S-haemoglobin forms crescent-shaped rods when in the reduced state (deoxygenated).
- High incidence in Africa is because heterozygous carriers are more resistant than normal to *Plasmodium falciparum* malaria during early childhood and hence have a degree of 'natural selection' for survival and passing on their genes.

Clinical features

- Vaso-occlusive episodes precipitated by low O2 tension in tissues.
- The abnormally shaped red cells cannot pass through arterioles and capillaries. Infarction, PE, recurrent infections, and arthralgia are common. These episodes of vaso-occlusion may also result from GA.
- Patients with a sickle cell trait have no clinical disabilities but may suffer from sickling episodes when at altitude or flying in unpressurized aircraft. Sickling tendency may lead to splenic infarction.

Complications

Susceptibility to infection, in particular, pneumococcal infection, meningitis, and *Salmonella*. Several types of crisis can occur:

- Painful crisis. Typically affects extremities, bones, and joints.
- Organ infarction, e.g. spleen, renal, brain.
- Sequestration crisis. When sickled erythrocytes are sequestrated in the liver and the spleen, leading to a massive drop in blood volume.
- Aplastic crisis. Loss of red cell production.

Diagnosis and investigations

At-risk patients must be identified before surgical procedures to ensure adequate oxygenation during GA and to prevent dehydration.

- Carry out a 'sickling test' on suspected patients. Add a reducing agent, e.g. CO2, to an unstained drop of blood. Homozygote blood sickles in a few hours. Heterozygote blood takes up to 24h.
- If the sickling test is positive, ensure adequate oxygenation during GA and prevent dehydration.

Treatment

- During a crisis, the main objective is to keep tissue O2 tension high and preventing slow flow in vessels, which promotes sickling. This is achieved by:
  - Providing analgesia.
  - Keeping the patient fully hydrated.
  - Keeping the patient warm (especially the peripheries).
  - Giving supplemental O2 (high flow or even hyperbaric) if possible.
  - Giving antibiotics to prevent 2° infection.
- Following a crisis, give the patient folic acid (5mg per day) to ensure red cell production is not inhibited by inadequate levels.

Malaria

Key facts

- Endemic in Africa, South East Asia, and South America.
- Attempts to eradicate it have been unsuccessful because of economic factors and the development of resistance to drugs.

Malaria can present as an acute abdomen with pain, pyrexia, and vomiting. Consider malaria at all times in patients returning from endemic areas, especially West and Central Africa.

Pathological features

- The causal organisms are:
  - *Plasmodium falciparum*. Malignant tertian malaria.
  - *P. vivax*. Benign tertian malaria.
  - *P. ovale*. Benign tertian malaria.
  - *P. malariae*. Quartan malaria.
- Transmission is by the ♀ anopheline mosquito The mouthpiece (full of organisms) inoculates the skin by repeated bites.
- Parasites enter into red blood cells and divide.
- The incubation period is usually 10–14 days but can be shorter and longer.
- Organisms may be cleared from the circulation by the immune system, but eradication of those within the red cells is rarely possible without drug treatment, and repeated episodes of systemic infection are characteristic of all forms of malaria (e.g. every 3 days = 'tertian'; every 4 days = 'quartan').
- Chronic carriage of organisms may occur with repeated episodes of illness, eventually causing immunosuppression and debility.
- Infected red cells may be removed from circulation by the spleen and liver, which may carry a particularly heavy load of organisms. In chronic cases, the spleen can be massively enlarged.

Clinical features

The fever of malaria is characteristically:

- Intermittent.
- Associated with sweating, chills, and rigors.
- Followed by an afebrile period (not always)

Complications

P. falciparum causes more life-threatening disease and the very young, elderly, and pregnant patients are more at risk.

Late presentation or late diagnosis increases the risk of death.

- ↑ blood viscosity during attacks/'crises' may lead to microvascular infarction of organs such as:
  - Brain (cerebral malaria).
  - Kidney (renal failure with haemoglobinuria—'blackwater fever').
  - Liver (jaundice, including acute hepatic failure).
- ↑ haemolysis of infected red cells, leading to:
  - Haemolytic anaemia.
  - Haemoglobinuria.

Diagnosis and investigations

This is based on a history of exposure (e.g. tourists) and the clinical features with microscopy of bloodstained smear which increases the chance of finding parasites.

- Blood film (thick and thin films).
- RDT.
- FBC may indicate anaemia and thrombocytopenia.
- Imaging of areas of interest: CXR, CT head/abdomen.

Treatment

Medical

Prophylaxis

All visitors to areas of endemic disease *must* be advised to take antimalarial prophylaxis, if indicated, and implement strict bite preventative measures. The recommended regimen depends on the predominant species and the presence of resistant strains.

Uncomplicated malaria

Oral treatment with Riamet® or atovaquone/proguanil are the drugs of choice for *falciparum* malaria. For other forms of malaria, a course of chloroquine and primaquine are the correct treatment (after G6PD testing).

Complicated malaria

- Should always be managed in a hospital with experience and appropriate facilities, if possible.
- Requires ITU or specialist facilities to support patient.
- Begin active rehydration immediately (IV crystalloid), but beware overhydration as ARDS may develop.
- IV drug of choice is IV artesunate 2.4mg/kg stat dose, followed by further doses at 12 and 24h and then every 24h. Most A&E or infectious diseases units should have access to IV artesunate. If no IV artesunate available, use IV quinine 20mg/kg stat dose, followed by 10mg/kg 8-hourly. Do not delay treatment.

Prognosis Untreated vivax malaria may subside in 10–30 days and recur intermittently. Intercurrent infection worsens the prognosis. Untreated falciparum malaria is frequently fatal.

Schistosomiasis (bilharziasis)

Key facts Endemic in many parts of north Africa, the Middle East, and South East Asia. Affects ~250 million people, with deaths exceeding well over 150 000/annum.

Pathological features

- Caused by the helminths *Schistosoma haematobium*, S. mansoni (Africa), or *S. japonicum* (Asia), Rarer forms are *S. mekongi* and *S. intercalatum* (South East Asia, West Africa).
- Infestation occurs from standing/swimming in infected water or showering with infected water. The intermediate hosts are snail (*Bullinus contortus*) that inhabits slow-running water.
- Multiplication of the larvae occurs in the snail; they then become free-swimming and enter humans through the skin. After shedding their tails, they are swept by the bloodstream to all parts of the body. The worms have a particular preference for some sites, according to their species.
- S. *haematobium* has an affinity for the vesical plexus, i.e. they are mostly found in the urinary bladder and ureter. *S. mansoni* and *S. japonicum* have an affinity for the mesenteric veins and biliary tree, i.e. they cause intestinal disease.
- Those that reach the liver develop into ♂ and ♀ worms, living in erythrocytes and when they mature, they leave the liver via the bloodstream to reach the vesical venous plexuses. The worms mate and the ova pass into the urine and faeces where they pass out and infect new water (especially stagnant). After hatching, the larvae enter the snail within 24h.

Clinical features and complications

- Swimmer's itch may occur very early on.
- Katayama fever may occur within 2–6 weeks, causing fever and rash.
- Intestinal worms cause:
  - Intestinal ulcers with bleeding, leading to abdominal pain and distension (due to ascites).
  - Perforation with peritonitis.
  - Pseudopolyps and inflammation, leading to bloody diarrhoea.
  - Chronic malabsorption and malnutrition.
  - Liver fibrosis (periportal) with classical 'pipestem-fibrosis', leading to portal HTN.
- Systemic infection may affect:
  - Brain, causing malaise and fever.
  - Lungs, causing dyspnoea.
  - Spinal cord, causing paralysis.
- Bladder worms produce:
  - Inflammation of mucosa and slough (dysuria, frequency, haematuria).
  - Obstruction of the urinary tract (hydronephrosis).
  - Squamous carcinoma of the bladder which is associated with long-term carriage.

Diagnosis and investigations

- Microscopic examination of an early morning urine or faecal specimen can demonstrate the presence of living eggs.
- Histological examination of a biopsy from the bladder or rectal mucosa can also provide confirmation of infection.
- Antibody testing is more useful as higher yield than microscopic examination.

Treatment

Medical

Praziquantel is the drug of choice. Doses for *S. japonicum* are higher than for *S. mansoni*/*S. haematobium*. A second dose may be required. Praziquantel is not effective in the early phases of infection.

Surgical

Surgical intervention is necessary when complications such as portal HTN, urethral stricture, or peritonitis after perforation develop.

Filariasis

Key facts

- Caused by a range of nematode worms, including *Wuchereria bancrofti, Onchocerca volvulus*, and *Brugia malayi*.
- Widespread in tropical and subtropical areas (India, Africa, China, the West Indies, and Australia).

Clinicopathological features

- Transmitted to humans by the bite of many genera of mosquitoes.
- Wuchererial worms enter the lymphatic system and cause:
  - Acute lymphangitis. Acute, swollen, painful lymphatics and nodes.
  - Chronic lymphadenitis and lymphatic obstruction. Especially of the lower limbs and genitalia—'elephantiasis'.
- Onchocercal worms may reach the eye (especially in African disease) and cause disruption of intraocular tissues—'river blindness'.

Diagnosis Is usually clinical but may also require blood films, especially taken at night-time.

Treatment

Acute lymphadenitis

- Rest the affected part.
- Antibiotics (ampicillin 500mg by IM injection bd for 10 days) are used to treat 2° infection caused by β-haemolytic *Streptococcus* and *Staphylococcus aureus*.
- Specific antifilarial drugs diethylcarbamazine citrate (Hetrazan®, Banocide®)—start at 1mg/kg PO tds for 3 weeks. Other antiparasitic agents like albendazole and ivermectin have been used.
- Surgical drainage of abscesses when they occur.

Chronic lymphoedema ('elephantiasis')

There is no satisfactory operation. Abnormal subcutaneous tissue can be excised and the affected part covered with a split skin graft. One variation is to excise the skin of the leg in long strips and then excise the subcutaneous tissue and apply skin to the denuded tissue. Apply a plaster of Paris dressing. The results are satisfactory, but certainly not cosmetic.

Hydatid disease (echinococcosis)

Key facts Occurs in sheep- and cattle-raising areas of the world, e.g. rural Wales, New Zealand, and not just the tropics.

Clinicopathological features

Pathological features

- Caused by the larval forms of the cestode worms *Echinococcus granulosus* and *Echinococcus multinodularis*.
- Dogs are usually the 1° host, eating infected sheep or cow offal, and the *Echinococcus* parasite, about 1cm long, develops in the dog's intestine.
- It consists of a head and three segments, the last of which contains hundreds of ova. These are passed onto grass, e.g. by defecation.
- Sheep and cattle ingest the ova to complete the normal life cycle. Humans are an incidental 'dead end' host, but the ova penetrate the small intestine and enter the portal circulation.
- 80% of ova thrive in the liver, with the development of hydatid cysts.
- They may also enter the general circulation, forming cysts elsewhere (e.g. kidneys, lungs, brain).

Clinical features/complications

- Infection is usually contracted in childhood but produces symptoms and signs in adult life.
- Commonest presentation is of a liver cyst (either found as a palpable mass, incidentally on CT scanning, or during abdominal surgery).
- Compression of the intrahepatic bile ducts may produce jaundice.
- Rupture of a cyst into the peritoneal cavity causes peritonitis and shock. Cyst fluid also causes a severe allergic reaction with urticaria and eosinophilia if it enters the circulation (either by spontaneous rupture or surgical intervention).
- The prognosis is poor if disseminated disease.

Diagnosis

- Serological tests, in conjunction with clinical findings and imaging, are diagnostic.
- Ultrasound and CT scanning may be used to localize cysts and determine the stage of echinococcosis.
- ERCP may demonstrate connections with, or compression of, the bile ducts.

Treatment

Hydatid cyst management requires expert management from medical and surgical teams experienced in dealing with hydatid disease.

Medical

- Complex—albendazole given for long periods and often in combination with praziquantel, depending on activity of disease and surgical options which is the mainstay of treatment.

Intraperitoneal rupture

- Treat shock.
- Carry out peritoneal toilet.
- Consider control of inflammatory response.

Surgical

- Excision or aspiration of the cyst(s).
- Extreme caution must be taken to prevent peritoneal contamination. Black packs soaked in hypochlorite are placed around the liver to show up any daughter cysts or scolices.
- The cyst is partially aspirated and partially refilled with hypertonic saline which is scolicidal. It is then carefully separated from the liver, and the cavity closed or drained.
- PAIR (percutaneous aspiration, injection, and reaspiration) is useful in some forms of cystic lesions.

Prevention Community hygiene projects to reduce the risk of humans being exposed to infected dog faeces in areas of endemic disease.

Ascariasis

Key facts

- Caused by the nematode roundworm *Ascaris lumbricoides*, a soil-transmitted helminth.
- Common in eastern and south eastern Africa, Sri Lanka, southern and south eastern India, and Bangladesh.
- The incidence of the condition is related to the state of development of the sewage systems.

Clinicopathological features

- Oral infection may occur in children or adults.
- Worms cross the intestinal wall and remain in the GI tract.
- Entry into the circulation may lead to systemic spread to other organs, e.g. liver, lungs, upper aerodigestive tract, blood.
- Intestinal infection causes:
  - Abdominal colicky pain and indigestion.
  - Diarrhoea.
  - Obstructive appendicitis.
  - Intestinal obstruction due to inflammation and impaction of a 'worm mass' or intussusception that may present as a mobile abdominal mass.
  - Protein-losing enteropathy with hypoproteinaemia and bleeding, leading to anaemia.
- Lung infestation may cause severe pneumonitis.
- Bile duct or pancreatic infestation may cause:
  - Bile duct strictures.
  - Liver abscess.
  - Cholangitis and empyema of the gall bladder.

Diagnosis

- Ova are demonstrated by examination of hot fresh concentrated stools.
- A plain AXR or barium meal may demonstrate radiolucent lines within a dense shadow, which represent individual worms.

Treatment

Medical

- Albendazole single dose of 400mg may be enough; mebendazole 100mg bd for 3 days (adults and children >2y). Immobilizes the worms by disrupting their transport systems.
- Piperazine is less often used nowadays.

Surgical

- Abdominal surgery is only indicated for obstruction or peritonitis.
- If intestines are not inflamed, the worm load is squeezed into the caecum and colon where it will be removed by peristalsis. Alternatively, remove it via an enterostomy. Any non-viable gut is resected.
- Biliary infestation may be treated by antispasmodics to allow the sphincter to relax and then by killing the worm load with antihelminthics or removal by ERCP or laparotomy.

Leishmaniasis

Key facts

A spectrum of diseases caused either by the direct effects of infestation by protozoans of the *Leishmania* group or type IV delayed hypersensitivity to the presence of the organisms.

There are over 20 different *Leishmania* spp. and there is variability between so-called old-world and new-world presentations.

Clinicopathological features

- The common route of infection is by bites from infected sand flies.
- The organisms spend their life cycle in the cytoplasm of circulating macrophages.
- Causative organisms include:
  - *Leishmania donovani*. Visceral leishmaniasis ('kala-azar', Indian subcontinent).
  - *L. tropicana, L. mexicana*. Cutaneous leishmaniasis.
  - *L. braziliensis, L. tropica, L. aethiopica*. Mucocutaneous leishmaniasis ('espundia', South America and Africa).

Cutaneous leishmaniasis

- May be a simple sore at the site of fly bite with 1° healing.
- May become a systemic infection if immune response poor.
- Severe type IV hypersensitivity reaction causes multiple skin ulcers ('recidiva').

Visceral leishmaniasis (kala-azar)

- An infection of the reticuloendothelial system with enlargement of the liver, spleen, and lymph nodes.
- May cause splenomegaly and pancytopenia if there is marrow infection.
- 2° dermoid leishmaniasis occurs in failing immunity and is highly infectious.

Mucocutaneous leishmaniasis

- Affects the skin and subsequently the mucous membranes of the mouth and nose, causing nodules and ulceration.
- Large ulcers on the skin of the face and destruction of skin and cartilage are referred to as 'Chiclero's ear'.

Diagnosis

- Bone marrow aspiration and staining with Giemsa/haematoxylin and eosin, looking for Leishman-Donovan bodies in macrophages.
- PCR speciation of biopsy samples.
- Biopsy from affected organ sites with PCR speciation.
- Serological tests may be helpful in visceral leishmaniasis.

Treatment

- Requires specialist advice and support.
- Cutaneous leishmaniasis often heals spontaneously, but when skin lesions are extensive, various compounds have activity—miltefosine and paromomycin have all been used. Sodium stibogluconate has activity but requires intensive monitoring.
- Liposomal amphotericin is treatment of choice in visceral leishmaniasis. In immunocompromised patients, prolonged treatment or recurrent treatment may be necessary.

Trypanosomiasis

Key facts

- Caused by infection with flagellate protozoa of the Trypanosoma group.
- Two main sites of infection are South America and Africa.

Clinicopathological features

- Causative organisms are:
  - *Trypanosoma brucei rhodesiense* (east African). 'Sleeping sickness'.
  - *T. brucei gambiense* (west African). 'Sleeping sickness'.
  - *T. cruzi* (South American). Chagas' disease.
- Transmission is by bites of infected tsetse flies in sleeping sickness or from infected faeces from triatomine bugs ('kissing bug') entering the mouth or eye in Chagas' disease.
- Chagas' disease, in particular, can also be transmitted vertically from mother to child and through blood-borne routes.

Sleeping sickness

- Bite site develops 1° chancre/ulcer.
- Parasitaemia results in acute lymphadenitis (often cervical—'Winterbottom's sign').
- Progressive waves of parasitaemia cause successively less effective immune responses with immunoglobulin M (IgM) and eventual failure of the immune response with widespread systemic parasite infestation.
- CNS infection leads to:
  - Spasm and tremor.
  - Discoordination.
  - Spastic and flaccid paralysis.
  - Unrousable sleep, progressing to coma and eventually death.

Chagas' disease

- Acute flu-like illness following infection. Lymphadenopathy and splenomegaly may occur.
- Eventual systemic parasite infection has a predilection for autonomic and neural crest-derived nervous tissue, e.g.:
  - Enteric nervous system destruction leads to 'megaoesophagus' and 'megacolon'.
  - Myocardial infection causes myocarditis.
  - Cardiac conducting system destruction leads to ventricular aneurysm formation, dysrhythmias, and sudden death syndrome.

Diagnosis

- Wet blood film showing viable parasites, most commonly for *T. rhodesiense*.
- FNAC of lymph nodes and/or lumbar puncture may reveal organisms, especially in *T. gambiense*.
- Chagas' disease is diagnosed clinically and with specific serological tests.

Treatment

Treatment varies for different stages, and American and African trypanosomiasis have completely different approaches.

Medical

- Treatment for African trypanosomiasis is complex and requires specialist management.
- Medications may include suramin for early disease and melarsoprol, a highly toxic compound, for CNS disease.
- New agents are being developed, including fexinidazole.
- Treatment for Chagas' disease includes antiparasitic agents and specialist management of organ disease.

Surgical Rarely required for complications of Chagas' disease (e.g. cardiac or intestinal surgery).

Tuberculosis in the tropics

Key facts

- Caused by *Mycobacterium tuberculosis*.
- In the tropics, chronic malnutrition and associated immunosuppression lead to more acute illness, more rapid advancement of disease, and presentation at a much more advanced stage.
- Intestinal TB is much commoner in tropical presentations.
- HIV co-infection is common.

Clinicopathological features

Infection routes include:

- Infection from food contaminated with the bacilli.
- Swallowed sputum containing bacilli.
- Bacteraemic phase of 1° lung infection.

Features of intestinal disease

- Often severe weight loss—'classical wasting disease'.
- May affect the terminal ileum, mesenteric lymph nodes, omentum, peritoneum, and solid organs related to the GI tract.
- Low-grade fever with night sweats, malaise, anorexia, and weight loss.
- Dull abdominal pain and abdominal distension.
- Ascites.
- Rectal examination may reveal fistulae or fissures.

Pathological types

- *Ulcerative.* Deep, transversely placed ulcers in the direction of the lymphatics that may cause perforation and peritonitis.
- *Hyperplastic.* Fibroplastic reaction, resulting in thickening of the bowel wall along the mesentery and affecting the lymph nodes and omentum, which may lead to malabsorption.
- *Sclerotic.* Associated with strictures in the small intestine, leading to intestinal obstruction.

Diagnosis

- FBC. ↑ WCC (lymphocytosis).
- Barium meal and follow-through may show intestinal strictures or ulcers that may be indistinguishable from those of Crohn's disease, as seen in temperate climates.
- Ultrasound and CT scanning may suggest inflammatory masses (typically in the RIF).
- Intestinal tissue biopsies may demonstrate caseating granulomas.
- Important to obtain biopsies/tissue for microbiological cultures and sensitivity.
- Modern technologies like PCR and whole genome sequencing are being implemented in sometimes even remote areas with high endemicity and financial support from international organizations.

Treatment

Treatment is based on WHO guidelines of standard quadruple therapy, followed by two-drug maintenance treatment for 6–12 months' total duration, dependent on the site of disease.

Certain countries have higher rate of multidrug-resistant TB (MDRTB) or even extensively drug-resistant TB (XDRTB) which require specialist input and management.

Medical

Antituberculous therapy for weight >50kg in fully sensitive patients:

- Rifampicin 600mg daily.
- Isoniazid 300mg daily.
- Ethambutol 10mg/kg daily.
- Pyrazinamide 2g daily.
- After 2 months, change to two-drug regimen of rifampicin/isoniazid for a further 4 months minimum. In patients with CNS disease or mediastinal disease, longer treatment required.

Surgical Laparotomy for peritonitis due to perforation, obstruction, or unresolving inflammatory masses.

- Surgery especially important for diagnostic tissue samples, both histologically and microbiologically.

Leprosy ('Hansen's disease')

Key facts

- Endemic in much of Africa, Southern Asia, the Far East, and South America.
- Affects annually around 250 000 patients.

Clinicopathological features

- Caused by the acid–alcohol-fast bacillus *Mycobacterium leprae*.
- Commonly contracted in late childhood or adolescence, the likely source of infection being nasal discharge from infected patients (rather than open skin lesions).
- Infiltration of nasal membranes leads to very slow systemic spread (incubation period of 3–5y).
- Bacilli eventually infiltrate areas of the body at lower-than-normal temperatures (especially dermis, upper respiratory tract, and peripheral nerves), although central organs (e.g. liver, bone marrow, kidneys, and spleen) may be involved, especially later in the disease.
- Patterns of disease depend on the degree of host cell-mediated immune (CMI) reaction:
  - *Poor CMI*. Lepromatous leprosy (LL). Highly infectious; open ulcerating lesions contain macrophages loaded with bacilli.
  - *Modest CMI*. Dimorphous/indeterminate leprosy (BB). Features of both types (loss of CMI leads to LL, but treatment produces TT-type disease).
  - *Good CMI*. Tuberculoid leprosy (TT). Pronounced lymphocytic infiltration of lesions causing scarring; nerve damage is prominent feature.

Clinical features

Consider the diagnosis of leprosy in any patient who presents with a combination of neural and dermatological disorders.

- Lepromatous leprosy.
  - Dermal changes. Typically widespread hypopigmented and erythematous rash affecting the face, limbs, and trunk.
  - Generalized malaise, fever, and arthralgia.
  - Neural lesions. Often widespread neuritis, followed by nerve thickening and progressive neuropathic tissue injury and ulceration due to anaesthesia.
  - Associated iritis is common.
  - Systemic amyloidosis may occur.
- Tuberculoid leprosy.
  - Dermal changes. Typically focal destruction of melanocytes (hypopigmentation), hair follicles, and sweat and sebaceous glands (dry, hairless, anaesthetic plaques of tissue).
  - Neural lesions. Isolated thickening of nerves (e.g. ulnar, peroneal), with late and relatively limited deformity.

Diagnosis

Diagnosis is based on:
- A history of contact.
- Clinical findings.
- Histological confirmation of M. leprae.

In practical terms, infected tissue is usually obtained by taking a smear with a scalpel blade inserted into the pinched skin of an affected eyebrow or earlobe. The tissue fluid obtained is stained with a modified Ziehl–Neelsen stain.

Treatment

Medical
- Three-drug combination chemotherapy is WHO standard of treatment—for 6–12 months, dependent on whether it is pauci- or multibacillary in diagnosis:
  - Rifampicin.
  - Dapsone.
  - Clofazimine.
- Different regimens may be used in rifampicin-resistant cases and may require longer treatment.

Surgical

Surgical treatment is indicated to correct deformities which may be:
- *1°*. Caused directly by the disease, e.g. thickening of the skin, paralysis of the eyelids, paralysis of the hands and feet.
- 2°. Due to neuropathic injury. Education of the patient on avoidance of injury and self-care is vital to prevent progressive injury since damaged nerves may be permanently anaesthetic.
- Severely damaged limbs may require amputation.

Buruli ulcer *(Mycobacterium ulcerans)*

Key facts

Chronic debilitating disease with high degree of disability affecting skin, muscle, and bone. Reported from 33 countries, including Africa, South America, Western Pacific, and Australia.

Clinicopathological features

- Causative organism is *M. ulcerans*, with unknown transmission.
- May start as a small lump but can progress to ulcerated lesion with large undermining area masking the extent of the disease presentation.
- Over time may lead to deformities and disabilities.

Diagnosis

- Clinical and with PCR-based technology.

Treatment

Medical

- Combination of rifampicin/streptomycin or rifampicin/clarithromycin given for 8 weeks has equal outcome.
- Treatment of disability and deformity and rehabilitation with intensive physiotherapy.

Surgical

- Wound debridement and skin grafting aid speed of recovery/healing.
- May be necessary to help with management of deformity and help with rehabilitation.

Guinea worm infestation (dracunculiasis)

Key facts

This freshwater-borne disease is on the WHO eradication list and in 2017, there were a total of 30 new cases in four countries, compared to 3.5 million people in 1986.

Mycetoma (Madura foot)

Key facts

- First described in 1694, but first reported from Madura in mid-nineteenth century.
- Associated with poor socio-economic background.
- Predominantly from the so-called 'mycetoma-belt', especially Bolivia, Venezuela, Chad, Ethiopia, Sudan, Somalia, Yemen, and India.
- Caused by a chronic infection—either bacterial or fungal.
- May be caused by different species with different colours of spores, but the clinical picture is remarkably uniform.

Clinicopathological features

- The first sign is a painless swelling in the foot that gradually develops multiple sinuses and sometimes discharges purulent material containing the grains of the fungus.
- Local spread may occur if the 1° infection is not treated, leading to deep tissue infection, e.g. (fungal) osteomyelitis.
- Systemic fungal infection is rare.

Diagnosis Microscopy of the discharge shows fungal hyphae or bacteria may be grown on culture.

Treatment

Medical treatment Intensive antibiotic or antifungal treatment, based on organisms and sensitivities.

Surgical treatment

- All the affected areas, including all sinuses, must be excised once treatment has begun.
- Amputation is occasionally necessary if deep osteomyelitis has occurred.

Eponymous terms and rarities

Acanthosis nigricans Pigmentation of the axillary skin associated with breast or gastric cancer.

Achondroplasia Familial dwarfism in which growth of the long bones and skull is defective.

Adenomyomatosis, gall bladder Thickening of the gall bladder wall with occasional intramural sinuses that may partially occlude the gall bladder lumen.

Adiposa dolorosa Multiple lipomas, usually on the arms and trunks, that are occasionally painful.

Adrenogenital syndrome A condition, usually autosomal recessive inheritance, affecting 1 in 5000 to 1 in 15 000 births, characterized by cortisol and/or aldosterone deficiency due to an enzymatic defect in cortisol synthesis, which results in 2° adrenal hyperplasia through loss of feedback on the pituitary gland. Diversion of precursors into the synthesis of other steroids, particularly androgens, results in virilization and ambiguous genitalia (through clitoral hypertrophy) of the ♀ fetus and pseudoprecocious puberty in the ♂. Early closure of the epiphyseal plates leads to short stature. Impaired aldosterone secretion can cause a salt-losing state that requires replacement therapy.

Aerocele The collection of air in one or more tissue layers of the cranium due to injured or inflamed cranial air sinuses.

Albers–Schönberg disease See osteopetrosis.

Albright's hereditary osteodystrophy An X-linked form of pseudohypoparathyroidism characterized by mental retardation, low serum calcium, cataracts, and tetany. Patients tend to be of short stature and have short first, fourth, and fifth metacarpals. Metastatic calcification of the basal ganglia is a feature.

Albright's syndrome/polyostotic fibrous dysplasia A condition thought to be due to disordered bony development, featuring fibrodysplastic bony changes, patchy skin pigmentation, and precocious puberty in girls. Affected bones become soft and deformed from childhood onwards.

Allen's test This assesses the adequacy of the collateral circulation to the hand. Digital pressure is applied to both the radial and ulnar arteries at the wrist and the patient repeatedly clenches a fist. Adequate collateral supply exists if there is complete palmar flushing with 15s of release of each vessel in turn.

Amastia Absence of both breast and nipple. Ninety per cent of patients with unilateral amastia have absent or hypoplastic pectoral muscles.

Amaurosis fugax Episodes of transient blindness due to central retinal artery embolization from carotid vessel disease/proximal vessel atherosclerosis.

Amazia Congenital absence of breast tissue, but not the nipple. It is now known as hypoplasia of the breast, to differentiate it from amastia.

Amyand hernia The presence of a normal or inflamed vermiform appendix within an inguinal hernia.

Angiodysplasia Vascular lesions of unknown aetiology, most frequ-ently found in the right colon, occasionally associated with cutaneous and oral lesions. They occur with increasing age and present with bleeding that may be torrential, but more often as a series of small bleeds.

Angiomyoneuroma (glomus tumour) A small, painful, benign tumour of blood vessels, rarely larger than a few millimetres in size, mainly found in the extremities. Half arise in the digits, predominantly subungually. They are exquisitely painful and tender and appear blue-purple in colour. Treated by excision with a wide margin.

Angiosarcoma A soft tissue tumour of young men and women, producing a hot, bulky tumour, with a tendency to bleed and metastasize to the lungs.

Ankyloglossia Also known as tongue-tie, it is due to a short lingual frenulum. It rarely affects speech, but frenectomy is recommended when food control and oral hygiene are a problem.

Antibioma A hard, oedematous swelling containing sterile pus following the treatment of an abscess with long-term antibiotics, rather than incision and drainage.

Aortoenteric fistula A connection between the aorta and small intestine, resulting in haemorrhage that becomes increasingly frequent and may culminate with exsanguinations; most commonly due to infection of a prosthetic graft, rather than a 1° spontaneous fistula.

Apert's syndrome Occurs in 1 in 160 000 births. Thirty per cent of cases are autosomal dominant. Skull is 'tower-shaped' (oxycephaly), with premature fusion of all the sutures. Mild face aplasia and syndactyly of the middle three fingers occur. Other associations include oesophageal atresia and renal and congenital heart anomalies.

Aphthous ulcers The commonest disorder affecting the oral mucosal membranes. Of unknown aetiology. They are painful and recurrent, and occur most commonly in childhood, rarely in the edentulous. Large ulcers, present for 3 months or more, may mimic carcinomas and should be biopsied if doubt exists.

Arnold–Chiari malformation A hindbrain abnormality where the cerebellum and medulla are found to lie below the level of the foramen magnum. Compression of the foramen of Magendie results in obstructive hydrocephalus in 80–90% of cases. Syringomyelia and spina bifida are commonly associated.

Askanazy cell tumour/Hurthle cell adenoma, thyroid A tumour consisting of featureless granular cells of varying sizes, distributed in the fibrous stroma of the thyroid. They are difficult to differentiate from malignant tumours but are regarded as benign.

Asplenia Absence of the spleen, associated with cardiac anomalies, including situs inversus.

Athelia Absence of the nipple. It is exceedingly rare.

Baker's cyst A central swelling of the popliteal fossa, most evident when the patient stands. It represents a synovial membrane diverticulum, almost always associated with knee joint pathology such as arthritis or torn meniscus.

Balanitis xerotica obliterans A disease of unknown aetiology characterized by keratotic lesions with inflammatory changes, leading to phimosis and occasional meatal stenosis. It has the appearance of a white stenotic band at the end of the foreskin and minor trauma often results in haemorrhage.

Ballance's sign Fixed dullness in the left flank, with shifting dullness best appreciated in the right flank, resulting from intraperitoneal and extraperitoneal bleeding following splenic rupture.

Barrett's oesophagus The presence of columnar-lined mucosa in the anatomical oesophagus; may be due to acid or biliary reflux. It is found in 10% of patients undergoing endoscopy for reflux symptoms. Strictures, ulceration, bleeding, dysplasia, and malignant transformation may occur.

Battle's sign Bruising over the mastoid process following a base of skull fracture that involves the petrous temporal bone.

Bazin's disease See erythrocyanosis frigida.

Beckwith–Wiedemann syndrome A congenital defect of the anterior abdominal wall associated with macroglossia, gigantism, and transient hypoglycaemic episodes.

Beck's triad Combination of hypotension, raised jugular venous pressure, and muffled heart sounds, suggestive of cardiac tamponade.

Bezoars Masses of ingested human hairs (trichobezoars) or indigestible vegetable matter and fibre (bezoars) that form in the stomach and interfere with digestion or may migrate into the small bowel and cause intestinal obstruction.

Bier spots The presence of white patches among the mottled blue–purple appearance of an acutely ischaemic limb that has been in a warm environment for several hours.

Blind loop syndrome Malabsorption due to colonization of a blind-ending segment of bowel by abnormal bacteria that prevent the digestion and absorption of food. Causes include congenital abnormalities (e.g. small bowel diverticula), strictures, or, more commonly, surgical construction of small bowel anastomoses and loops.

Blue naevus Results when embryonic melanocyte migration from the neural crest is arrested in the dermis.

Bochdalek hernia A posterior diaphragmatic hernia where the septum transversum fails to unite with the intercostal part of the diaphragm. It occurs in infants and is characterized by gross herniation of abdominal contents and associated lung hypoplasia.

Boerhaave's syndrome Spontaneous oesophageal rupture following an episode of intense vomiting or retching, characterized by severe upper abdominal and chest pain, tachycardia, tachypnoea, and subcutaneous emphysema.

Bornholm disease (epidemic pleurodynia) Coxsackie B4 virus infection of the pleura and peritoneum, characterized by severe upper abdominal and chest pain (worse on movement and respiration), associated with dyspnoea, pleuritic pain, headache, and sore throat.

Bowen's disease An irregular, reddish brown cutaneous plaque, occasionally ulcerated and commonly found on the trunk. It is an intra-epidermal carcinoma in situ and may develop into squamous cell carcinoma.

Branham's test When a pneumatic tourniquet is inflated around the root of a limb with a suspected arteriovenous malformation, a significant fall in the pulse rate suggests a significant arteriovenous shunt.

Budd–Chiari syndrome Post-hepatic venous obstruction that may result from spontaneous thrombosis, extrinsic compression by tumour, or a web in the vena cava.

Buschke–Lowenstein tumour A rare benign penile 'tumour' caused by human papillomavirus infection with giant tumour growth, but only local tissue destruction; may result in urethral fistula formation.

Cloquet's (Callisen's) hernia A deep femoral hernia that cannot protrude from the saphenous opening as it lies deep to the femoral vessels.

Calot's triangle An essential landmark in laparoscopic cholecystectomy surgery. Its boundaries are the common hepatic duct, cystic duct, and inferior border of the liver.

Campbell de Morgan spots Small red spots that commonly occur on the trunk in middle age and do not blanch. They are of no significance.

Cancer en cuirasse Multiple malignant nodules on the chest wall in breast cancer that mimic the breast plate on a suit of armour.

Caput medusa Engorged veins radiating from the periumbilical region, resulting from extrahepatic portosystemic shunting from portal hypertension.

Carbuncle Multiple adjacent follicular infections with Staphylococcus aureus, commonly seen in diabetics. Treat with flucloxacillin and surgical drainage as required.

Cardiac myxoma A rare 1° cardiac tumour, commonly arising in the left atrium, which can present with either obstruction mimicking mitral stenosis or tumour emboli.

Carnett's test Determines whether an abdominal lump lies intraperitoneally or within the abdominal wall. The patient lies flat and raises his extended legs off the couch. An intraperitoneal lump disappears, whereas one in the abdominal wall persists.

Caroli's disease An anatomical abnormality characterized by intrahepatic cystic changes, with an ↑ risk of bile duct cancer.

Carr's concretions Microscopic calculi within the papilla of the kidney thought to be involved in the pathogenesis and propagation of renal calculi.

Charcot's triad Fever, rigors, and jaundice characteristic of acute cholangitis. Right hypochondrial pain is often an additional feature. This is a serious and potentially fatal condition, caused by ascending infection of the biliary tree associated with partial biliary obstruction.

Chemodectoma A carotid body tumour extending from the carotid bifurcation that presents with a solitary or bilateral lump(s) anterior and deep to the sternocleidomastoid. Characteristically, they can be displaced laterally, but not vertically, and are associated with bruits and thrills in 20% of cases. The risk of malignancy increases with size.

Chilaiditi's syndrome Bowel loops interpositioned between the liver and the diaphragm which, on the chest radiograph, can mimic free air.

Chopart's amputation An amputation made through the tarsal bones.

Churg–Strauss syndrome Affects young and middle-aged adults, often with a history of atopy, asthma, and allergic rhinitis, in which there is marked eosinophilia. Clinical manifestations include peripheral neuropathy, cardiac involvement (heart failure and myocardial infarction), and vascular involvement that affects the stomach, small bowel, kidneys, and central nervous system due to aneurysm formation, thrombosis, and infarction.

Chvostek's sign Hyperexcitability of the facial nerve to local percussion over the parotid gland in patients with a reduced serum calcium concentration. It can also occur in 10% of normal people.

Chylothorax The accumulation of lymphatic fluid (which can have the appearance of pus) within the pleural cavity following thoracic duct trauma (blunt and penetrating injuries or surgical procedures), obstruction by malignant disease (particularly lymphomas and carcinomas of the lung and breast), and congenital defects (usually also associated with ascites).

Codman's triangle Radiographic evidence of periosteal elevation found with osteosarcomas.

Contrecoup injury Injury to the brain on the opposite side of the initial injury, due to transmitted movements of the cerebral tissue within the skull.

Cooper's hernia A rare multilocular deep femoral hernia that enters the thigh via the deep investing fascia.

Corrigan's pulse A collapsing pulse found in the presence of an arteriovenous fistula.

Courvoisier's law A palpable distended gall bladder in a jaundiced patient is more likely to be due to malignant disease obstructing the bile ducts than gallstones (where the gall bladder tends to be fibrotic and contracted).

Craniocleidodysostosis An autosomal dominant disease characterized by partial or complete clavicular aplasia, vertebral and digital deformities, and patent fontanelles.

Craniofacial dysostosis/Crouzon's syndrome A condition characterized by stenotic cranial sutures, maxillary hypoplasia and prognathism, beaked nose, exophthalmos, and mental retardation.

Crigler–Najjar syndrome Pre-hepatic jaundice due to an inability to conjugate bilirubin within the liver. There are two types—autosomal recessive (type I) and autosomal dominant (type II).

Cronkhite–Canada syndrome A triad of gastrointestinal polyps, alopecia, and fingernail atrophy. The changes are not neoplastic, but due to an unidentified deficiency state.

Crueveilhier's sign (saphena varix) It is positive if an impulse is felt at the saphenofemoral junction when the patient stands and coughs.

Cullen's sign Periumbilical bruising seen in acute severe necrotizing pancreatitis or other form of severe intraperitoneal bleed, i.e. ectopic pregnancy and abdominal trauma.

Curling's ulcer Acute gastroduodenal ulceration associated with severe burns.

Curtis–Fitz–Hugh syndrome Severe right hypochondrial pain due to perihepatitis due to Chlamydia trachomatis infection.

Cushing's ulcer Acute gastroduodenal ulceration associated with stress such as severe haemorrhage, myocardial infarct, and multiple trauma, and in critically ill patients.

Dandy–Walker syndrome Congenital absence of the foramen of Magendie, resulting in marked ventricular dilatation. The lateral sinuses appear higher than normal on X-ray because of cerebellar hemisphere widening and the higher attachment of the tentorium cerebelli.

De Garengeot hernia Presence of the vermiform appendix within a femoral hernia.

De Quervain's disease Inflammation around the extensor pollicis brevis and abductor pollicis longus tendons, often associated with thickening of the extensor retinaculum. This results in pain on movement of the thumb and tenderness where the tendons cross the radial styloid.

De Quervain's thyroiditis Self-limiting viral inflammation of the thyroid gland, which usually follows a recent upper respiratory tract infection, characterized by giant cell infiltration.

Dermatomyositis A condition of insidious onset characterized by proximal muscular weakness, pain, and tenderness. There is a characteristic purple skin rash that affects the cheeks and light-exposed areas. Association with occult malignancies of the colon, lung, breast, and genitourinary tract.

Desmoid tumour A locally expanding tumour of mesenchymal tissue often found in the infra-umbilical abdominal wall muscles or intra-abdominal mesenchymal tissue. It commonly affects middle-aged ♀ and requires wide excision. Associated with familial adenomatous polyposis.

Dietl's crisis The passage of large volumes of urine following acute intermittent hydronephrosis. Classically, there is ureteric colic and a palpable, distended kidney. Both resolve after the passage of urine.

Dysplastic naevus (FAMMM) syndrome Dysplastic naevi are considered precursors of malignant melanoma when there is a family history. Solitary lesions in the absence of a family history are not. All patients should avoid excessive sunlight.

Ectopia vesicae (bladder exstrophy) Occurs in 1 in 30 000 live births, more commonly in ♂. There is an open bladder and a defective anterior abdominal wall associated with separated pubic bones and penile epispadias or bifid clitoris. Associated with glandular metaplasia and the risk of squamous carcinoma of the bladder remnant.

Ehlers–Danlos syndrome A rare hereditary collagen disorder characterized by joint hypermobility, hyper-extensible skin and/or vascular/visceral fragility. The most clinically dangerous form, Vascular EDS presents at a young age with spontaneous arterial rupture and/or bowel perforation.

Emphysematous cholecystitis A rapidly progressive infection of the gall bladder due to anaerobic organisms, characterized by air in the wall of the gall bladder and a high risk of perforation.

Empyema—gall bladder A pus-filled gall bladder, resulting from impaction of a gallstone in the neck of the gall bladder.

Encephalocele The protrusion of cranial meninges, cerebrospinal fluid, and brain tissue through an opening in the skull.

Epidermal naevus syndrome The presence of extensive light brown warty lesions in association with skeletal and central nervous system developmental abnormalities.

Epiplocele A hernial sac containing omentum.

Epispadias A rare condition characterized by failure of development of the anterior wall of the lower urogenital tract, affecting the glans and penis alone (1 in 120 000) or the whole urinary tract when it is commonly associated with bladder exstrophy (1 in 30 000). It most commonly affects ♂ and is characterized by the urethra exiting from the dorsal penile surface at varying sites.

Epithelioma of Malherbe Another term for a pilomatrixoma, a red/white subepidermal nodule, frequently calcified and found on the upper body.

Erythrocyanosis frigida Also known as Bazin's disease. It affects healthy ♀ with fat and often hairless legs. Capillary dilatation, alongside arteriolar constriction, results in dusky red/purple blotches that blanch on pressure and rapidly refill. They can be painful. Ulceration and persistent oedema may occur in severe cases.

Erythromelalgia A condition characterized by erythema and pain in the dependent extremities, relieved by elevation. The inappropriate release of local vasodilators has been implicated.

Erythroplasia of Queryat A reddish brown, irregular lesion found on the glans penis, which may ulcerate and crust. It is regarded as a carcinoma in situ and nearly always occurs in uncircumcised patients.

Exophthalmos Proptosis (sticking out of the globe of the eye), lid retraction, conjunctival oedema, and, in severe cases, ophthalmoplegia or optic nerve damage. Affects 2–3% of patients with Graves' disease.

Extradural haematoma The formation of a haematoma in the extradural space, most commonly following a fracture of the parietal or temporal bones, with rupture of the middle meningeal artery or its branches that traverse them.

Fallot's tetralogy Congenital cyanotic heart disease with four features: (1) ventriculoseptal defect; (2) pulmonary stenosis; (3) overriding aorta; and (4) right ventricular hypertrophy. The infant becomes cyanosed on exertion and adopts a classical squatting position, which raises their systemic vascular resistance, thereby increasing pulmonary blood flow.

Familial adenomatous polyposis (FAP) Autosomal dominant syndrome characterized by multiple colorectal and intestinal polyps, as well as other intestinal and mesenchymal lesions.

Felty's syndrome An association between rheumatoid arthritis, splenomegaly, and granulocytopenia, which may be complicated by leg ulcers and recurrent infections.

Finkelstein's test Used to identify cases of stenosing tenosynovitis. The patient places their thumb in their palm and clenches a fist. The examiner pushes the hand into ulnar deviation and, if positive, pain is felt at the radial styloid, radiating down the forearm.

Foster Kennedy syndrome Optic atrophy of one eye and papilloedema in the other, which is due to a frontal tumour blocking the subarachnoid space on the ipsilateral side, but causing papilloedema on the other side because of raised intracranial pressure.

Fournier's gangrene A form of necrotizing fasciitis involving the perineal or scrotal skin, leading to subcutaneous necrosis. Synergy appears to occur between normal non-pathogenic organisms, leading to local vascular thrombosis and necrosis. Associated with uncontrolled diabetes mellitus.

Frey syndrome Gustatory sweating of the cheek following accidental or surgical trauma of the parotid region. It results from cross-regeneration of the transected sympathetic and parasympathetic fibres and develops over about 12 months.

Froment's sign Weakness of the adductor pollicis, following a high ulnar nerve palsy, leads to compensatory overaction of the flexor pollicis longus (innervated by the median nerve) when the patient is asked to squeeze a sheet of paper between the thumb and the index finger.

Galactocele A cystic lesion containing breast milk, occurring in women who suddenly stop breastfeeding.

Gamekeeper's thumb A sprain of the metacarpophalangeal joint of the thumb, leading to rupture of the ulnar collateral ligament. Non-healing leads to chronic instability and weakened pinch grip.

Gardner's syndrome Variant of familial adenomatous polyposis, involving an association between multiple epidermal cysts, intestinal polyposis, desmoid tumours, and osteomas.

Garrod's pad Subcutaneous tissue thickening over the proximal interphalangeal joints that are histologically similar to those found in Dupuytren's disease.

Gaucher's disease A genetic abnormality leading to active storage of abnormal glucocerebrosides in the spleen, resulting in massive childhood splenomegaly.

Gilbert's syndrome Congenitally acquired mild jaundice due to a failure of transport of bilirubin to the liver, which can be precipitated by episodes of starvation. There is an absence of urinary bilirubin, although faecal and urinary urobilinogen levels are ↑. It is of little clinical significance.

Glomus tumour See angiomyoneuroma.

Glucagonoma A tumour of the pancreatic islet cells characterized by mid-maturity-onset diabetes, an erythematous rash that tends to blister and crust, glossitis, and raised glucagon levels.

Gluteal hernia A very rare type of hernia where visceral contents pass through the greater sciatic notch. It is often only discovered during laparotomy for the relief of intestinal obstruction of no obvious cause. Rarely, swelling around the buttock and pain referred along the sciatic nerve occur.

Goodsall's rule Rule for predicting how anal fistulae behave. Anterior fistulae follow a direct path to the anus, whereas posterior fistulae 'horseshoe' to the anus.

Grawitz tumour Adenocarcinoma of the kidney.

Grey–Turner sign Bruising in the flanks resulting from retroperitoneal haemorrhage (e.g. haemorrhagic pancreatitis).

Gynaecomastia The benign growth of breast tissue in ♂. The breast is uniformly enlarged and soft. May be physiological (e.g. maternal oestrogens, oestrogen–androgen imbalance of puberty), hypogonadism (pituitary disorders, androgen blockade), neoplasms (adrenal/gonadotrophic tumours, bronchogenic, renal cell, etc.), systemic disease (hepatic failure, renal dialysis, hypothyroidism), and drug-induced (androgen blockers, oestrogens, cimetidine, spironolactone, ketoconazole, methyldopa, metoclopramide, etc.).

Hamartoma Overgrowths of one (or more) cell type(s) normally found within the organ from which they arise, e.g. neurofibromas.

Hammer toe Hyperextension of the metatarsophalangeal joint and distal interphalangeal joint, with flexion of the proximal interphalangeal joint. This can lead to the development of bursae and calluses. Frequently associated with hallux valgus, overcrowded toes, and diabetic neuropathy.

Hand–Schüller–Christian disease Multiple visceral and lytic skeletal lesions, characteristically also involving the skull, that are associated with diabetes insipidus and exophthalmos.

Hangman's fracture Traumatic disruption of the pars interarticularis of the atlas (C2) following a hyperextension injury.

Hashimoto's disease A diffusely enlarged, painless thyroid gland due to lymphocyte infiltration. Rubbery in nature and often mimicking a multinodular goitre. If enlargement is asymmetrical, other causes must be excluded. Clinically, the patient is euthyroid or mildly hyperthyroid.

Henle–Coenen sign If an arteriovenous fistula is occluded and the distal vessels still pulsate, this indicates that the fistula can be safely treated by ligation.

Hereditary osteodystrophy An X-linked form of pseudohypo parathyroidism characterized by hypoparathyroidism, low serum calcium, mental retardation, cataracts, and tetany. Metastatic calcification of the basal ganglion is also a feature.

Hesselbach's hernia A rare form of external femoral hernia that enters the thigh, lateral to the deep epigastric and main femoral vessels.

Hibernoma A lipoma consisting of brown fat cells.

Hidradenitis suppurativa A chronic, recurrent deep-seated skin infection of the axilla or perineum.

Housemaid's knee Chronic bursitis of the prepatellar bursa from the trauma of repeated kneeling (as in scrubbing floors).

Howship–Romberg sign Pain referred to the inner aspect of the thigh via the genicular branch of the obturator nerve, which may arise from an obturator hernia that strangulates.

Hunner's ulcer Stellate white ulcers within the bladder, resulting from chronic inflammation, that open up on distension and bleed on decompression. Aetiology is unknown and women are more frequently affected. Bladder capacity is reduced and symptoms include urinary frequency and pain on distension, relieved by micturition.

Hydatid of Morgagni Also known as the appendix testis. Remnant of the Müllerian duct found at the upper pole of the testis, situated in the groove between the testis and the epididymis; may undergo torsion.

Hyperhidrosis Excessive sweating of the axillae, palms, and feet, which can be socially embarrassing and distressing.

Hyperostosis frontalis interna (Morgagni's hyperostosis) ↑ density and projection of the frontal bones, affecting the inner table only. Aetiology is unknown. Patients are often asymptomatic.

Hypersplenism A combination of splenomegaly, anaemia, leucopenia, and/or thrombocytopenia, with bone marrow hyperplasia. Splenectomy may be required.

Inspissated bile syndrome Inspissation of bile in the common bile duct during early infancy (usually from haemolysis), resulting in proximal bile duct and gall bladder dilatation.

Insulinoma A rare tumour of pancreatic islet beta-cells, characterized by hypoglycaemic attacks that both are unpredictable and worsen in severity with time. Diagnosis is based on Whipple's triad.

Intraperitoneal rupture of bladder Usually traumatic in origin, from surgical instrumentation or abdominal trauma in the presence of a full bladder.

Jansen's disease An inherited autosomal dominant form of metaphyseal dysostosis, characterized by deafness and extreme dwarfism.

Jefferson's (burst) fracture Disruption of the ring of atlas (C2) following traumatic injury to the neck. Spinal column damage is uncommon as the fragments tend to open outwards.

Kalokerino's sign A filling defect of the fundus of the stomach that mimics a neoplasm. It arises when part of the fundus to the left of the cardio-oesophageal junction is about to herniate through it.

Kanavel's sign Is due to an infected ulnar bursa. Greatest tenderness is elicited in the transverse palmar crease on the ulnar side.

Kantor's string sign Is indicative of Crohn's disease. Involvement of the terminal ileum leads to structuring of the lumen. This gives the radiological appearance of a thread-like structure on barium follow-through.

Kaposi's sarcoma Painless red-brown macules on the limbs and anal and oral mucosa. Occasionally, they may ulcerate. They can be found in the elderly, endemically (e.g. in Africa), and in immunosuppressed patients (e.g. transplant patients, human immunodeficiency virus).

Kartagener's syndrome Bronchiectasis and sterility resulting from abnormal ciliary action.

Kehr's sign Left shoulder pain referred from splenic injury and rupture.

Kenaway's sign A venous hum that is louder on inspiration (on auscultation with the bell of the stethoscope below the xiphisternum), associated with splenomegaly in patients with bilharzial cirrhosis of the liver.

Keratoacanthoma See molluscum sebaceum.

Killian's dehiscence The weak point between the cricopharyngeal and thyropharyngeal muscles through which pharyngeal mucosa can herniate, leading to the formation of a pharyngeal pouch.

'Kiss' cancer Cancer implanted in one area by local contact from another affected site, e.g. cancer of the lip, vulval labium.

Klein's sign Right iliac fossa pain that moves to the left when the patient turns onto their left side. It can be associated with mesenteric lymphadenitis and Meckel's diverticulum.

Klippel–Trenaunay syndrome A condition of the lower limb characterized by congenital varicose veins, deep vein abnormalities, bony and soft tissue deformity, limb elongation, and capillary naevi.

Köhler's disease Osteochondritis of the navicular bone. This can be one of the causes of a painful limp in a child under 5y of age.

Krukenberg tumour An ovarian tumour arising from transcoelomic spread of a primary gastrointestinal carcinoma.

Ladd's bands Persistent fibrous bands between the small bowel mesentery and liver, which can lead to obstruction of the second part of the duodenum. They are commonly associated with incomplete rotation of the bowel.

Laugier's hernia A rare form of femoral hernia that enters the thigh through a defect in the pectineal part of the inguinal ligament.

Li–Fraumeni syndrome An inherited predisposition to cancer thought to be due to mutation of the p53 tumour suppressor genes.

Linitis plastica Also known as leather bottle stomach. Submucosal proliferation of fibrous tissue secondary to carcinoma of the stomach leads to gastric wall thickening and a reduction in stomach volume and plasticity. Because it spreads readily along the mucosa plane and presents late, its prognosis is poor.

Lipodystrophy Excessive fat deposition in the legs that may be mistaken for oedema.

Littre's hernia Abdominal wall hernia (usually groin) containing Meckel's diverticulum.

Livedo reticularis Cyanotic skin mottling due to vasospasm of the arterioles with concomitant capillary dilatation.

McBurney's point Lies one-third of the way along a line drawn from the right anterior superior iliac spine to the umbilicus. It is the classical point of maximal tenderness in acute appendicitis and the centre point for the gridiron (McBurney's) incision used in open appendicectomy.

McMurray's test This is used to identify medial meniscal tears. With the patient supine, the knee is flexed and foot rotated medially and laterally, whilst bringing the knee to 90° of flexion. Discomfort or a click is noted in the presence of a tear.

Mackler's triad Combination of chest pain, vomiting, and subcutaneous emphysema suggestive of Boerhaave's syndrome.

Madelung's deformity Dorsal subluxation of the lower end of the ulna that is congenital or traumatic in origin.

Maisonneuve's fracture The triad of a medial malleolar fracture, spiral fracture of the neck of the fibula, and separation of the distal tibiofibular joint found in severe ankle trauma.

Malgaigne's bulges Bulges seen above the inguinal ligament in thin individuals on coughing or straining. They are variants of normal and do not represent inguinal hernias.

Mallory–Weiss syndrome/tear Haematemesis resulting from prolonged violent vomiting, leading to mucosal tears in the cardia of the stomach.

Marble bone syndrome See osteopetrosis.

Marfan's syndrome A rare autosomal-dominant connective tissue disorder typically characterised by cardiovascular defects (aortic aneurysm/dissection, aortic/mitral regurgitation), skeletal manifestations (tall stature, increased arm-span, arachnodactyly (long fingers), joint hypermobility, scoliosis) and/or lens dislocation.

Marion's disease A rare cause of bladder outflow obstruction in young men due to narrowing of the bladder neck.

Marjolin's ulcer A long-standing venous ulcer that fails to heal, in which squamous cell carcinoma develops.

Maydl's hernia The presence of a double loop of bowel within the neck of a hernia, 'W' in shape, where strangulation of the middle loop can occur.

Medullary sponge kidney Is due to dilatation of the terminal collecting ducts of the kidney, which predisposes to the formation of renal calculi.

Meigs' syndrome Ascites and pleural effusions (usually right-sided) associated with benign ovarian tumours.

Meleney's gangrene A form of necrotizing fasciitis mostly seen after abdominal surgery.

Ménétrier's disease Hypertrophy of the gastric mucosa, most typically proximally, resulting in hypochlorhydria and hypersecretion of gastric juices, leading to protein loss. Patients may present with epigastric discomfort and peripheral oedema. There is no associated ↑ risk of gastric cancer.

Meralgia paraesthetica Numbness and hyperalgesia around the lateral thigh following entrapment of the lateral cutaneous nerve of the thigh as it passes beneath the inguinal ligament.

Mesentericoparietal hernia of Waldeyer An internal paraduodenal hernia that lies medially and inferior to the third part of the duodenum. It may present with recurrent episodes of abdominal pain and vomiting due to small bowel obstruction.

Meyer–Weigert's law In patients with complete ureteric duplication, it is the lower pole ureter that refluxes because its mucosal tunnel through the bladder wall is shorter.

Milia Small, white, superficial facial spots derived from hair follicles. They appear in newborn babies and following skin grafting and dermabrasion, and are treated by expression.

Mills' manoeuvre When the forearm is pronated whilst holding the elbow straight, pain over the common extensor origin is consistent with extensor tenosynovitis (tennis elbow).

Milroy's disease Lymphoedema, presenting from adulthood onwards, resulting from congenital aplasia of the lymphatic trunk.

Mirrizi's syndrome Obstructive jaundice resulting from impaction of a gallstone in the cystic duct, which presses against the common hepatic duct, causing extrinsic compression.

Molluscum contagiosum Small, pale, firm nodules with a characteristic central depression that follow infection with the pox virus. They tend to regress with time, although they can be treated by curettage.

Molluscum sebaceum A solitary skin tumour that grows rapidly over 6–8 weeks and involutes over about 6 months to leave a residual scar. It has the appearance of a dome-shaped lesion with a central keratin-filled crater and can be mistaken both clinically and histologically for a well-differentiated squamous cell carcinoma.

Mondor's disease of the breast Superficial thrombophlebitis affecting the veins of the breast. Initially, there may be tenderness which is followed by fibrosis and contraction, resulting in skin dimpling.

Morgagni hernia A congenital diaphragmatic hernia that presents in early adult life with dyspnoea or as an incidental mediastinal mass. Abdominal contents expand into the anterior mediastinal compartment through a persistent defect in the anterior diaphragm.

Morgagni's syndrome See hyperostosis frontalis interna.

Murphy's sign Pain and tenderness in the right upper quadrant directly beneath the subcostal space upon deep inspiration, whilst the examiner's fingers rest over the edge of the lower thoracic margin at this point. It is due to an inflamed gall bladder and the patient may be unable to fully inspire because of the pain.

Myositis ossificans Ectopic bone formation arising within a haematoma in muscle following soft tissue injury.

Nail–patella syndrome An inherited condition characterized by radial head subluxation, small patellae, and absent or deformed nails.

Narath's hernia A rare form of femoral hernia that extends anterior to the femoral artery beneath its investing fascia.

Nelson's syndrome The presence of skin hyperpigmentation and accelerated growth of a pituitary tumour following bilateral adrenalectomy for pituitary-dependent Cushing's. It results from loss of pituitary feedback.

Nutcracker oesophagus Alternative name for diffuse oesophageal spasm—the presence of long-duration, high-intensity peristaltic contractions in the oesophagus, which may be associated with chest pain and dysphagia.

Obturator sign The aggravation of right iliac fossa pain upon passive internal rotation of the right hip in patients with appendicitis where the appendix lies adjacent to the obturator internus.

Osteogenesis imperfecta An inherited collagen disorder, resulting in fragile bones that fracture easily, blue sclerae, deafness, and soft teeth.

Osteopetrosis (Albers–Schönberg disease, marble bone disease) An inherited disorder of bone, resulting in ↑ bone density, fractures, and anaemia. The recessive form is less severe than the autosomal dominant form.

Osteopoikilosis Infantile patchy long bone sclerosis (found radiologically) associated with yellow skin lesions. It has autosomal dominant inheritance and is of no clinical significance.

Oxycephaly An autosomal dominant condition characterized by stenotic sutures, resulting in a tower-shaped skull, a prominent nose, and a lateral squint. Facial deformities are also common. Mental retardation, optic atrophy, and deafness can also occur.

Painful arc syndrome Shoulder pain occurring between 70° and 110° of abduction due to the passage of an inflamed supraspinatus tendon between the acromion process and the head of the humerus. The subacromial space is narrowest at this point, leading to impingement.

Pancreas divisum Arises when the ventral and dorsal pancreatic buds fail to fuse during embryological development. Consequently, the main pancreatic duct drains via an accessory ampulla. The vast majority of patients are asymptomatic, although it may be one of the causes of chronic pancreatitis.

Panda sign Bilateral black eyes following a head injury, suggestive of a base of skull fracture involving the anterior cranial fossa.

Parkes–Weber syndrome Bony and soft tissue limb overgrowth resulting from multiple arteriovenous fistulae. Lipodermatosclerosis, ulceration, and high cardiac output failure may also occur.

Paterson–Brown Kelly syndrome The association of a pharyngeal web with dysphagia and iron deficiency anaemia. There is an ↑ risk of post-cricoid carcinoma.

Peau d'orange Localized oedema found in breast cancer where the skin of the breast has the pitted appearance of orange skin.

Pendred syndrome Familial association of deafness and goitre with peroxidase deficiency (involved in the synthesis of thyroxine).

Phalen's test The reproduction of discomfort and paraesthesiae of the fingers in the distribution of the median nerve (lateral three and half fingers) when the wrist is held in flexion. It is due to compression of the median nerve as it passes beneath the flexor retinaculum through the carpal tunnel.

Phlegmasia alba dolens White, massively swollen, painful leg following extensive DVT (partially compensated by collaterals). Progression to phlegmasia cerulea dolens may occur.

Phlegmasia cerulea dolens Blue(cyanotic), massively swollen painful limb following extensive DVT. The limb is threatened due to thrombosis of all main venous outflows and collaterals, leading to increased compartment pressures and peripheral cyanosis, with imminent venous ischemia/gangrene. This may progress to venous gangrene as circulatory congestion and stasis occur.

Plagiocephaly Development of an asymmetrical skull arising from the early closure of sutures on one side of the skull.

Plummer–Vinson syndrome See Paterson–Brown Kelly syndrome.

Pneumatosis cystoides intestinalis Gas-filled cysts within the intestinal and mesenteric walls, most commonly affecting the small intestine. These can be seen on plain abdominal X-rays.

Pneumaturia The passage of flatus in urine, which can arise from a colovesical fistula (e.g. diverticular disease, carcinoma, inflammatory bowel disease) or urinary tract infection in diabetics where glucose is fermented by the infecting organism.

Poland's syndrome An association between pectoral muscle abnormality, absence or hypoplasia of the breast, and characteristic hand deformity of hypoplasia of the middle phalanges and skin webbing (synbrachydactyly).

Pott's carcinoma of the scrotum Squamous cell carcinoma of the scrotum, associated with chronic exposure of the scrotal skin to aromatic carcinogens in coal-derived chimney soot. Now more commonly associated with exposure to heavy metals and mineral oils, particularly the ones used in the cotton industry.

Pott's disease of the spine Tuberculosis of the spine, leading to bony destruction and vertebral collapse, which lead to kyphosis and spinal compression.

Pott's peculiar tumour A large trichilemmal cyst, commonly occurring on the scalp, which can ulcerate and resembles a squamous carcinoma (both clinically and histologically). They are benign but may recur after excision. They rarely undergo malignant transformation.

Pott's puffy tumour Osteomyelitis of the skull bones following untreated frontal sinusitis. This results in overlying scalp inflammation and swelling.

Proctalgia fugax Severe recurrent rectal pain in the absence of any organic disease. Attacks may occur at night, after bowel actions, or following ejaculation. Anxiety is said to be an associated feature.

Pseudomyxoma peritonei Disseminated mucinous tumour within the peritoneal cavity. Commonly due to ruptured ovarian or appendiceal mucinous neoplasms. Locally recurrent and potentially fatal, even with heroic surgery and chemotherapy.

Ramsay Hunt syndrome Reactivated varicella-zoster virus which affects the facial nerve, causing ipsilateral paralysis.

Ranula A saliva-containing cyst in the floor of the mouth that has a soft, bluish submucosal appearance.

Raspberry tumour A tender, granulomatous mass arising from the posterior urethral meatus.

Redcurrant jelly stool A description attributed to the bloodstained stool found in intussusception.

Riedel's thyroiditis Thyroiditis characterized by a marked fibrotic reaction, leading to a hard, non-tender thyroid gland. Thyroid function tends to be normal and differentiation from malignant disease can be difficult, as fine needle aspirates tend to be acellular.

Reinke's oedema The presence of generalized oedema of the upper vocal cords in response to noxious stimuli.

Reiter's syndrome The triad of polyarthritis, conjunctivitis, and urethritis as a result of venereal infection, usually chlamydia. The initial attack lasts for 4–6 weeks, although some patients develop chronic symptoms.

Rendu–Osler–Weber syndrome Hereditary haemorrhagic telangiectasia; a rare autosomal dominant condition characterized by the presence of haemangiomas affecting the lips, buccal cavity, nasopharynx, and whole gastrointestinal tract. These may bleed, resulting in episodes of haematemesis, haematuria, melaena, epistaxis, or anaemia that are self-limiting.

Richter's hernia A form of strangulated hernia in which only part of the bowel lumen becomes strangulated, leading to incomplete intestinal obstruction with ischaemia and gangrene of the strangulated part.

Rovsing's sign Pain and tenderness in the right iliac fossa produced by palpation of the left iliac fossa. It may be found in acute appendicitis.

Sabre tibia Occurs in late syphilis where new formation of subperiosteal bone results in bowing.

Saint's triad The association between cholelithiasis (gallstones), hiatus hernia, and diverticular disease.

Scheie's syndrome An autosomal recessive skeletal disorder associated with corneal clouding, cardiac anomalies, and epiphyseal dysplasia.

Scheuermann's disease A disease that predominantly affects young adolescent ♂ where vertebral growth plates are affected by osteochondrosis or aseptic necrosis, resulting in back pain and progressive kyphosis.

Schmorl's node A lucent area with surrounding new bone formation in a vertebral body, resulting from extrusion of the nucleus pulposus into the vertebral body.

Sever's disease Osteochondritis of the posterior epiphysis of the os calcis near the insertion of Achilles tendon.

Shoveler's fracture A stable cervical injury resulting from fracture of the spinous process of C7 due to either trauma or muscular contraction.

Sinding–Larsen's disease Osteochondritis of the distal part of the patella.

Sister Joseph's nodule The appearance of umbilical nodules in the presence of advanced intra-abdominal carcinoma, typically stomach, but also large bowel, ovarian, or occasionally, breast.

Sjögren's syndrome The presence of keratoconjunctivitis sicca, salivary gland involvement (leading to xerostomia, i.e. dry mouth), and rheumatoid arthritis or other mixed connective tissue disorders. 1° Sjögren's is characterized by the first two features, whereas 2° Sjögren's has all three.

Spigelian hernia A rare type of hernia due to defects within the internal oblique aponeurosis as it interdigitates with the anterior and posterior rectus sheath. Peritoneum and visceral contents may herniate through these small defects.

Stevens–Johnson syndrome Also known as erythema multiforme, characterized by ulceration that has a characteristic target appearance and results from drug allergies (particularly to sulfonamides and barbiturates), mycoplasmal infections, or idiopathically. The lesions may be associated with conjunctivitis, tracheitis, and dysphagia.

Stewart–Treves syndrome The development of angiosarcoma in a chronically lymphoedematous limb, in this particular case, following radical mastectomy for breast cancer.

Sump syndrome Due to the collection of stones and debris in the distal common bile duct following choledochoduodenostomy, resulting in epigastric pain, cholangitis, and pancreatitis.

Thrombophlebitis migrans ↑ coagulability of blood associated with visceral cancers, particularly adenocarcinomas.

Tietze's disease Also known as costochondritis. Affects the costal cartilages, resulting in chest pain that can be reproduced by pressure to the affected cartilages.

Tinel's sign Transient finger paraesthesiae that follows percussion of the median nerve proximal to the wrist in patients with median nerve compression due to carpal tunnel syndrome.

Treacher Collins syndrome Also known as mandibulofacial dysostosis. An autosomal dominant condition characterized by abnormal development of theexternal and middle ear leading to hearing impairment, zygoma, and mandibular hypoplasia, parrot-beaked nose, absence of medial lower eyelashes, dental crowding, and abnormal palpebral fissures.

Trichobezoars See bezoars.

Troisier's sign Enlargement of the left supraclavicular lymph node due to advanced metastatic gastric carcinoma.

Trousseau's sign Phlebothrombosis of the superficial leg veins associated with gastric cancer.

Umbolith An umbilical concretion of desquamated skin, which can lead to infection.

Ureterocele A cystic dilatation of the intravesical submucosal ureter. It is often associated with other congenital anomalies, including duplicated ureters.

VACTERL syndrome Spectrum of congenital disorders that tend to co-exist. The association of vertebral (V), anorectal (A), cardiovascular (C), tracheo-oesophageal (TE), renal (R), and limb (L) anomalies. Progesterone and oestrogen intake during early pregnancy has been implicated.

Vermooten's sign Digital rectal examination reveals a doughy, displaced, or absent prostate in the presence of an intrapelvic rupture of the prostatic urethra.

von Hippel–Lindau disease An inherited disorder characterized by cerebellar and spinal cord haemangioblastomas, retinal angiomas, and an ↑ risk of visceral cancers, particularly renal cell carcinoma.

von Recklinghausen's disease (neurofibromatosis type I) Autosomal dominant inherited nodular thickening of nerve trunks, associated with patchy skin pigmentation (*café-au-lait spots*). Malignant transformation of these neurofibromas tends to occur only in this particular subgroup of patients and their prognosis is poor.

von Rosen's sign Congenital dislocation of the hip. Results in a click when the hip is flexed and adducted, then flexed and abducted, as this causes the femoral head to dislocate and relocate.

Waldenström disease Necrosis of the articular cartilage of the head of femur following a slipped femoral epiphysis. This leads to stiffness or complete loss of movement in that hip.

Waterhouse–Friderichsen syndrome Bilateral adrenal cortical necrosis due to septicaemia (meningococcal, pneumococcal, streptococcal), haemorrhage, or burns.

Whipple's triad Fasting hypoglycaemic attacks with blood glucose <2.5mmol/L, relieved by glucose, and associated raised insulin levels. These are characteristic of an insulinoma.

Whitaker test Can be used to assess the degree of ureteric obstruction. Saline is perfused into the kidney via a renal puncture at a rate of 10mL/min and the pressure gradient measured across the ureteric–vesical junction. Ureteric obstruction is indicated by a pressure difference >20cmH_2O. Less than 15cmH_2O is normal.

Youssef's syndrome A ureterovesical fistula that presents with monthly episodes of haematuria.

Zenker's diverticulum A pharyngeal pouch that occurs through the dehiscence of Killian, between the cricopharyngeus and the inferior constrictors of the pharynx. There is usually a history of food sticking and regurgitation. Progressive weight loss, dysphagia, and aspiration pneumonia can also occur.

Zollinger–Ellison syndrome Intractable duodenal ulceration due to elevated levels of circulating gastrin levels. There is an association with multiple endocrine neoplasia type I. Diagnosis is confirmed by acid secretion tests which show elevated basallevels. Secretin challenge elevates gastrin levels in G-cell hyperplasia, but not G-cell tumours.

Chapter 25

Day case surgery

Introduction

The definition of day case surgery is the planned day admission of a patient to hospital for a surgical procedure, after which there is subsequent successful and safe discharge back home on the same day. The main rationale behind day surgery is to get patients discharged home following their operation in a safe and timely manner, instead of spending prolonged periods within hospital as an inpatient. This has significant implications, including reducing hospital stay, hospital-acquired infection, and health care-related costs, whilst also improving patient experience and service efficiency.
Factors to be considered for successful day case surgery include the following.

Surgical factors

- Procedures with low incidence of major complications.
- Short procedures (ideally <2h).
- No significant impairment on oral intake or mobilization.
- Consultant or consultant-supervised operative lists to improve efficiency.

Anaesthetic factors

- Procedures with predictable post-operative recovery course.
- Avoidance of opioid analgesics.
- Local and regional block options.

Patient factors

- Appropriate patient selection taking into account comorbidities, as opposed to arbitrary limits such as American Society of Anesthesiologists (ASA), age, and BMI.
- Thorough preoperative assessment.
- Patient awareness and education in principles of day surgery to enhance patient experience.

Social factors

- Support at home following discharge post-operatively—responsible adult to take patient home and provide support for 24h.
- Suitable home environment for post-operative recovery.

Ultimately, successful day surgery functioning is dependent on motivated patients, an effective day surgery pathway, and enthusiastic day surgery staff.

History of day case surgery

The 'day surgery' concept can be originated to 1909 when James Nicoll (a Glaswegian surgeon) reported a large series of children, all of whom were discharged on the same calendar day of their operations. In 1955, Eric Farquharson (Edinburgh surgeon) published a similar experience with 458 consecutive open inguinal hernia repairs as day cases. Eventually, the first UK day unit was opened at Hammersmith Hospital in 1969 by Professor James Calnan.

The Royal College of Surgeons of England released formal guidelines on day case surgery in 1985 which recommended a target of 50% of all elective surgical procedures to be performed as day cases (only 15% were being performed as day cases at that point in time). Day surgery services rapidly progressed and led to the formation of the British Association of Day Surgery (BADS) in 1989. This initial organization was chaired by Professor Paul Jarrett (general surgeon at Kingston Hospital, Surrey), with a council of anaesthetists and senior nurses.

Aims of the British Association of Day Surgery (BADS)

- Encourage the expansion of day surgery.
- Promote education and high-quality treatment.
- Conduct research and publish findings.
- Organize meetings, seminar lectures, and conferences.
- Provide advice on construction and management of day units.
- Maintain high standards of surgical, anaesthetic, and nursing care.
- Organize a day surgery reference library.

Following further years of success with day case surgery, the UK government strongly supported the model, largely due to the significant financial benefits of treating patients as 'day cases', as opposed to inpatients. In 1990, the Audit Commission (independent external auditor of the NHS) published recommendations, including a 'basket' of 20 surgical procedures deemed suitable for day surgery. This was expanded to 25 procedures in 2001, following consensus with BADS. This 'basket' of procedures were audited across UK hospitals to ascertain day surgery performance status.

There was still vast variation in day surgery practice across the UK at the turn of the millennium, with only 68% of elective operations done on a day case basis nationally. The NHS Modernisation Agency in 2001 recommended further improvement with a target of 75%.

In 2006, BADS produced the concept of a Directory of Procedures, which was not limited to the 'basket' of 25 procedures in assessing individual hospital performance. The directory consisted of procedures across multiple surgical subspecialties and over 200 procedures in the latest edition (sixth edition, 2020). The Directory of Procedures has changed descriptors of day case operations by describing them as 'zero-night stay' procedures. This has been extended to 'one-night stay' and 'two-night stay' for procedures that patients should be discharged within 48h and 72h, respectively. The 'one- and two-night stay' procedures cover operations that need longer recovery periods.

Audit Commission 2001 'basket' of 25 procedures

- Orchidopexy.
- Circumcision.
- Inguinal hernia repair.
- Excision of breast lump.
- Anal fissure dilatation or excision.
- Haemorrhoidectomy.
- Laparoscopic cholecystectomy.
- Varicose vein stripping or ligation.
- Transurethral resection of bladder tumour.
- Excision of Dupuytren's contracture.
- Carpal tunnel decompression.
- Excision of ganglion.
- Arthroscopy.
- Bunion operations.
- Removal of metalware.
- Extraction of cataract with or without implant.
- Correction of squint.
- Myringotomy.
- Tonsillectomy.
- Submucous resection.
- Reduction of nasal fracture.
- Operation for bat ears.
- Dilatation and curettage/hysteroscopy.
- Laparoscopy.
- Termination of pregnancy.

Day surgical unit

The remit of day case surgery has rapidly progressed in recent times with government initiatives and financial pressures on the NHS in the UK. With this, day surgery has continually evolved, including the continual development and infrastructure of the day surgical unit (DSU).

Day surgery patient admissions are pre-planned with full preoperative clinical assessment. The nature of day case admissions differ from inpatient surgical admissions and are more amenable to 'streamlining' with protocols and patient journey pathways. Running day surgery from inpatient wards did not function well, largely due to the chaotic nature of such wards. Nursing staff had to devote more time to acutely unwell patients, and thus a lack of incentive to push for same-day discharge for day case surgical patients.

The concept of dedicated day wards and self-contained DSUs separate from the main hospital building led to significant improvements in same-day discharges for patients undergoing intended day case surgery. This model best works with its own dedicated theatre suites and recovery wards, day surgery care-trained nursing staff, and patient facilities (e.g. parking, waiting area). The most functional units are those 'ring-fenced' against emergency pressures of the main hospital. Opening times should be at least from 7 a.m. to 8 p.m. to maximize the service. Most trusts in the UK now have such DSUs to facilitate day case surgery.

Day surgery pathway

A robust day surgery pathway is essential to the patient journey experience. Each facet of this pathway can be scrutinized at a local trust level to optimize day surgery service delivery. There is ample room for service innovation and protocols to make the pathway more efficient and patient-friendly. Recent examples of this include GPs fast-tracking referrals directly to preoperative assessment clinics, thus eliminating a step of the pathway.

Key steps of day surgery pathway

- Patient listed for a day case procedure from outpatient clinic.
- Nurse-led preoperative assessment (often same day as clinic).
- High-risk anaesthesia clinic if patient deemed high risk (if deemed not suitable for DSU—explore if suitable for inpatient admission, local/regional anaesthesia, or cancel operation).
- Patient given operation date with specific preoperative instructions.
- Day of admission: protocol preoperative checks.
- Operation performed in DSU theatre.
- Discharge with appropriate analgesia and post-operative instructions.
- Follow-up in clinic/over telephone if required.

Leadership and staffing

Every DSU requires a Clinical Lead with a subspecialty interest in day surgery, usually an anaesthetist or a surgeon. This role requires protected time within their job plans and entails developing local guidelines, pathways, and general clinical governance with the unit. A multidisciplinary operational group led by a Day Surgery Manager supports the Clinical Lead and oversees the day-to-day running of the unit. As a cohort, they develop timetables and protocols, solve operational issues, and perform audits (guided by the BADS Directory of Procedures). Day surgery must also be represented at Hospital Board level as a separate entity.

Staff members of a typical DSU (trained in day surgery care)

- Clinical Lead.
- Day Surgery Manager (typically a Band 7 senior nurse).
- Recovery ward sisters and junior nurses.
- Operating department practitioners (ODPs).
- Scrub nurses.
- Administrators.
- Receptionists.
- Housekeeping ancillary staff.

Indicators of quality of service

Day surgery performance at a local level must be audited regularly to assess quality of service delivery and guide commissioning groups. Improvements can be made to achieve the full benefits of day surgery. Quality indicators are vital and help facilitate identification of deficiencies within individual units and allow comparison with other trusts.

Quality measures

- Did not attend (DNA) rate.
- Cancellation on arrival rate—preoperative assessment failure.
- Number of patients treated.
- Nature of procedures undertaken.
- Day case patients who have to stay overnight as a result of previously foreseen circumstances.
- Inpatient emergencies using DSU facilities.
- Day case patients admitted overnight.
- Complication and infection rates.
- Readmission rates within 30 days of original procedure.
- Patient satisfaction of DSU journey.

Patient selection criteria

Most of elective operations in the UK are performed as day cases. Patient selection still remains important in addressing the suitability of patients for day case procedures to allow same-day discharge as intended. Advances in both anaesthetic and surgical techniques have increased the array of procedures that can be performed. Development of minimally invasive surgery has been particularly significant. This has been further supplemented with positive study outcomes on day surgery in patients with a multitude of comorbidities. These factors have strongly influenced changes in patient selection criteria, going away from traditional arbitrary upper limit restrictions in age, ASA, and BMI. Ultimately, the procedure must be deemed safe to perform as a day case with suitable home circumstances and this is judged on an individual patient basis.

A multidisciplinary approach to preoperative assessment best identifies those not suitable for day case surgery. This involves various subspecialties during the patient's journey, including the initial consultation with the surgeon, preoperative assessment with highly trained nurses, and the anaesthetist's review for high-risk patients. Often local multidisciplinary consensus dictates individual hospital guidelines on patient inclusion and exclusion criteria for day surgery.

Age

Traditionally, age was used as an arbitrary exclusion factor (e.g. >75y not suitable for day surgery). There is no longer an upper age limit for day case surgery. Biological age and comorbidities should be considered over chronological age. Thorough preoperative assessment plays a crucial role in assessing suitability and if any thought, formal anaesthetic review should be sought for borderline-risk patients. Benefits of performing day surgery on the elderly include swift return back to similar home surroundings and therefore less post-operative delirium.

Term infants over 1 month of age can safely have day case surgery. Paediatric patients with significant comorbidities or syndromes are generally not suitable for day surgery. Adequate paediatric facilities must be available on site.

BMI

BMI is measured in kg/m^2:

- <18.5: underweight.
- 18.5–24.9: normal.
- 25–29.9: overweight.
- 30–39.9: obese.
- ≤40: morbidly obese.

The incidence of obesity in the UK continues to rise, with an increasing burden on the NHS. In the obese, there are strong associations with heart disease, DM, sleep apnoea, reflux disease, and hiatus hernia. Other anaesthetic implications include difficulty in intubation and IV cannulation. The higher use of IV propofol, compared to older fat-absorbing, volatile anaesthetic drugs, has led to more control over anaesthesia in obese patients.

From an operative perspective, surgery on obese patients is more technically demanding and time-consuming and carries a higher complication risk profile in most procedures.

Calculating BMIs during the preoperative pathway is necessary to identify obese patients, and weight loss must be strongly encouraged at an early stage. Referral to community weight loss programmes should be offered. Post-operative early mobilization is crucial to reduce risk of VTE. The upper limit BMI depends on local day surgery policies, with some units setting limits (e.g. up to a BMI of 35).

ASA

ASA has become an arbitrary factor in patient selection criteria for day surgery. ASA I and II patients have shown similar complication rates to ASA III patients. Knowledge of specific comorbidities during preoperative assessment is more useful to optimize pre-, peri- and post-operative care. Essentially, patients with stable chronic conditions are no longer excluded from day surgery. In fact, early discharge home can result in less disruption to normal daily management of chronic medical conditions.

Hypertension

A common medical comorbidity in the UK. Uncontrolled and severe HTN can lead to ↑ MI and stroke risk during surgery, as well as dreaded post-operative haemorrhage. If BP readings consistently high (>180/110 mmHg), refer back to primary care for control before considering day case surgery.

Diabetes mellitus

Diabetics (particularly insulin-dependent) have high risk of operative morbidity in day surgery. Optimal blood glucose control prior to surgery is vital for reducing complications. HbA1c (glycosylated Hb) of <8.5% implies adequate long-term diabetic control and therefore, surgery is safe. Readings higher than this require primary care/endocrinology input prior to consideration for surgery. Ideally, diabetics should be first on operative lists, particularly if fasted overnight. Also, alternative anaesthetic options should be explored (e.g. regional/local).

Smoking

Associated high risk of respiratory and wound complications. Smoking cessation should be encouraged at an early stage, ideally commenced at least 6 weeks prior to surgery. Effective smoking cessation services must be incorporated into the preoperative workup.

Preoperative assessment

Proper preoperative assessment is an important stage of the patient journey to day surgery. It educates the patient about the pathway to enhance their experience but also optimizes them medically for surgery with same-day discharge. With this assessment, there should theoretically be no 'on the day' cancellations on clinical grounds, therefore avoiding wasted resources within the NHS. The nature of the process allows identification of medical and social factors that may fulfil exclusion criteria. These can subsequently be resolved prior to surgery, e.g. uncontrolled asthma or no overnight support at home prior to procedure.

Essential components to preoperative assessment

- Educate patients about day surgery pathways.
- Provide information (verbal and written) regarding planned procedures and post-operative care to help patients make informed decisions.
- Identify any medical risk factors, promote health, and optimize patients medically prior to their surgery.

Preoperative assessment clinic

Preoperative assessment is ideally run in a self-contained outpatient setting with day surgery-trained specialist nurses. Individual patients are assessed for medical, social, and operative factors to ascertain if suitable for day case surgery. Patients identified with significant comorbidities are subsequently seen in the 'high risk anaesthetic clinic' by consultant anaesthetists for a formal medical review. Decisions made from this clinic can then be fed back to the operating surgeon to make a final decision regarding surgery.

Nurse-led clinics make up the bulk of the preoperative assessment process. In some units, it is possible to get a preoperative assessment unit (PAU) appointment on the same day, following initial surgical consultation where a decision to operate has been made. This prevents a further separate visit to PAU.

Patient self-reporting questionnaires are often implemented as screening tools asking patients for medical and social problems. This represents a point of triage prior to nurse assessment to identify any major issues regarding suitability for day surgery. These questionnaires tend to be general but also should take into account any considerations specific to the intended procedure. Certain hospitals have day surgery-specific proformas or booklets into which all patient-related information goes. This standardizes documentation throughout the day surgery pathway.

PAU nurses often follow local protocols regarding requesting appropriate preoperative investigations. This is dependent on the proposed operation and the patient's medical status. Routine blood tests are generally performed on most patients undergoing surgery; however, specific tests, such as group and save, are procedure-specific. Other tests include ECGs, CXRs, and peak expiratory flow readings and are requested as local guidance.

Patient information

A well-informed patient leads to providing a high-quality day surgery service with good outcomes. Information given to patients about the day surgery experience and their proposed operation should start in the primary care setting at the time of referral to hospital specialists. Information can be reinforced in surgical outpatient clinics and PAU, on the day of admission, and at discharge. This continual flow of information to the patient leads to reduced anxiety and enhanced satisfaction.

Information can be given verbally and in written form. Leaflets and directing patients to official websites at an early stage allow the patient ample opportunities to ask questions and clarify issues throughout the later stages of the day surgery pathway. Some local units have developed procedure-specific consent forms with information about operations and potential complications. They also explain details on preoperative preparation and post-operative care to fully educate the patient for their surgery. These detailed consent forms are usually given unsigned to the patient at the time of initial surgical consultation.

Day of admission for surgery

Most documentation and preparation have been completed by the time the patient enters the DSU on the day of their surgery, due to comprehensive preoperative assessment. For this reason, very little is required of the patient on arrival to the DSU. This significantly reduces anxiety in the immediate preoperative period.

Several units have staggered arrival times to the DSU in order to reduce waiting times for patients on the day of surgery. Fasting protocols are also customized, so afternoon operative lists allow patients to have early breakfast meals.

Anaesthetic considerations

Day surgery anaesthesia requires experience and efficiency, but safe practice to be successful. The service should be predominantly consultant-led. However, anaesthetic trainees require supervised training to increase their competency levels in day surgery.

Anaesthetic technique

Day surgery anaesthesia should allow rapid recovery following the operation, in order to get the patient home on the same day. There are various techniques that can be employed, including GA, LA, and regional anaesthesia, as well as sedation. Anaesthetists have different approaches to day surgery anaesthesia, depending on their experience and personal preferences. Patient stress must be minimized by ensuring smooth induction of, and recovery from, anaesthesia.

Premedication includes any medication administered to the patient prior to entering the theatre for their surgery. Benzodiazepines can be used to alleviate preoperative anxiety. This, in turn, can reduce anaesthetic agent dosages to induce anaesthesia.

Sedation can be used effectively during certain procedures similar to endoscopy. 'Conscious sedation' is optimal in such cases where the patient has depressed consciousness but can still respond to verbal commands. Anaesthetists should be present perioperatively to monitor O2 saturations and BP. Again, benzodiazepines (e.g. midazolam) can be administered to achieve this and may require titrating to ensure minimal patient stress. Use half doses for the elderly to reduce respiratory depression.

Airway management

- Required with GA.
- Either ETT or LMA used.
- ETT used for longer procedures.
- LMA has the benefit of not requiring muscle relaxant and is tolerated at lighter doses of anaesthesia, therefore rapid recovery.
- LMA is not a protected airway; therefore, aspiration can occur. It is avoided in those with severe GORD and the morbidly obese.

General anaesthesia

Drug-induced loss of consciousness during which patients are unarousable. The ideal agent allows rapid induction and recovery from anaesthesia and minimal PONV. Anaesthetists tend to favour total IV anaesthesia (propofol) over volatile inhalation agents for this reason, particularly reduced PONV. It is paramount that the patient rapidly returns to a full cognitive state and can mobilize early following their surgery to ensure they are discharged on the same day. This control and rapid recovery from anaesthesia ultimately leads to quicker turnaround time.

Local/regional anaesthesia

Includes procedures performed with LA infiltration to operative site and peripheral nerve and central blocks (i.e. spinal, epidural). These methods can be used in combination with sedation and GA to optimize post-operative recovery.

Patients must be fully counselled preoperatively about the duration of blocks and possible residual sensory and motor block on discharge. They require written instructions, including protecting the anaesthetized area from inadvertent trauma.

Toxicity from LA can occur and usually manifests as lip tingling, light-headedness, and tinnitus. Severe adverse effects include cardiac arrhythmias and tonic–clonic seizures.

Spinal analgesia can be used for lower limb or rectal surgery. It is advocated in those with significant cardiorespiratory comorbidities. Complications can include post-dural puncture headache, hypotension, epidural haematoma, spinal infection, and damage to the spinal cord.

Analgesia

Adequate analgesia is paramount to the patient's day surgery experience, including post-operative recovery after discharge. The WHO analgesia pain ladder must be adopted when administering pain relief. Generally speaking, regular paracetamol, along with NSAIDs, should be prescribed to all, unless contraindicated. Judicious use of opioids should be adopted due to strong side effects, including PONV. The use of LA and peripheral nerve blocks can further optimize pain control. An example is a TAP block during open inguinal hernia repairs.

Post-operative nausea and vomiting

PONV is best avoided, as opposed to treated. It can be extremely distressing to a patient undergoing day surgery. Those prone to PONV or motion sickness should be prescribed prophylactic antiemetics. Similarly, administration is required with certain procedures associated with a high risk of PONV, including laparoscopic surgery, middle ear ENT operations, and prolonged operations (longer than 1h). Routine IV fluids can reduce PONV and ensure proper hydration.

Typical drugs used include ondansetron, cyclizine, and metoclopramide. These should be administered IV and prescribed as required on drug charts for recovery nurses to use when PONV occurs. It is important to co-administer antiemetics with opioid medication.

Post-operative discharge

Discharge following a day case operation is principally nurse-led and dependent on the patient returning to their preoperative baseline. Many hospitals utilize set protocols for steps to discharge in day case surgery. This results in safe discharge and avoids complications post-discharge.

There are three stages of recovery from surgery and associated anaesthesia. Appropriately trained and experienced day surgery nurses are essential throughout these stages to have an efficient, functional DSU. Emphasis post-operatively should be on early mobilization, oral intake, and pain and PONV control.

Stages of post-operative recovery

First stage—early recovery

- From the end of the operation until the patient regains consciousness and protective neurological reflexes return.
- This stage bypassed if operation done under LA.
- Performed in day surgery recovery area by trained nurses. This allows the anaesthetist to start the next case, whilst recovery nurses care for patients throughout this stage.
- Analgesia and antiemetics can be administered as appropriate.

Second stage—preparing for discharge

- From patient waking up to being ready for discharge from DSU.
- Often the anaesthetist and surgeon will review the patient prior to discharge. Otherwise this stage is heavily nurse-led and protocol-driven.
- Performed on DSU ward usually.
- Early complications become apparent during this stage.
- Eating food is no longer a strict requirement prior to discharge, as can exacerbate PONV. However, tolerance of oral fluids is generally expected.
- Patients who fail to progress will require surgeon and anaesthetist review and likely overnight admission to a hospital ward.

Third stage—post-discharge late recovery

- This occurs following discharge and until the patient has made a full recovery physiologically and psychologically.
- Occurs at home and often takes weeks to months.
- May require interval medical reviews, e.g. wound check, outpatient appointment.

Discharge

Most units have post-operative day surgery protocols with criteria to be met prior to safe discharge. Once declared fit to go home, the patient can be discharged with a responsible adult. Surgeon and anaesthetist review is not required before discharge, unless any issues arise.

Discharge criteria

- Vital signs stable for at least 1h.
- Orientation as to time, place, and person.
- Adequate pain control.
- Understanding the use of oral analgesia supplied, supported by written information.
- Ability to dress and walk (depending on procedure).
- Minimal PONV.
- Minimal bleeding.
- Tolerates oral fluids.
- Passed urine.
- Responsible escort to take patient home.
- Carer at home for the next 24h.
- Written and verbal instructions given about post-operative care, including removal of sutures.
- Follow-up appointment details given to patient.
- Dressings supplied to patient.
- Sick certificate if needed.
- Emergency contact number supplied.

All day surgery patients should be discharged with clear verbal and written information regarding post-operative care at home and potential complications to look out for. A copy of the discharge summary written by the surgical team will detail any specific instructions and follow-up out-patient appointments. In particular, wound care and timing of removal of sutures are essential information to notify the patient to reduce anxiety during their recovery.

Patients having procedures under GA must not drink alcohol or operate machinery for 24h. Driving must be avoided until able to perform an emergency stop comfortably. Following GA, this should be a minimum of 24h.

Surgeons prescribe analgesia for patients to go home with. Most patients receive paracetamol supplemented with NSAIDs or codeine phosphate for breakthrough pain. Other medications commonly prescribed include laxatives, antibiotics, and PPIs (for NSAID cover). Clear instructions should be given regarding adequate dosage and duration of course for these drugs to prevent side effects.

Common day surgery procedures

By no means is this a complete list of day case surgical procedures, but a wide range in various surgical subspecialties.

General surgery procedures

- Inguinal hernia repair—unilateral, bilateral, 1°, recurrent, open, laparoscopic (TEP/TAPP), GA, LA, regional anaesthesia.
- Laparoscopic cholecystectomy.
- Laparoscopic repair of hiatus hernia with antireflux procedure.
- Pilonidal sinus surgery (laying open or suture/skin graft).
- Haemorrhoidectomy (conventional, circular stapling, HALO).

Urology procedures

- TURP.
- Transurethral resection of bladder (TURB).
- Circumcision.
- Orchidectomy.
- Ureteroscopic extraction of ureteric calculus.

Orthopaedic procedures

- Arthroscopy of knee, including meniscectomy.
- Carpal tunnel release.
- Bunion operations.
- Excision of Dupuytren's contracture.
- Excision of ganglion.

ENT procedures

- Myringotomy.
- Tonsillectomy.
- Thyroidectomy.
- Parathyroidectomy.
- Polypectomy of internal nose.

Vascular procedures

- Open varicose vein surgery (ligation of SFJ).
- Endovenous ablation for varicose veins.
- Foam sclerotherapy of varicose veins.
- Creation of AV fistula for dialysis.
- Temporal artery biopsy.

Breast procedures

- Simple mastectomy.
- Wide local excision of lesion of breast.
- Microdochectomy (excision of breast ducts).
- Axillary node sampling.
- Breast lipofilling reconstruction.

Plastics surgery procedures

- Excision of skin lesion ± graft reconstruction.
- Pinnaplasty.
- Excision of nail/nailbed.
- Rhinoplasty.
- 2° tendon repairs.

Gynaecological procedures

- Termination of pregnancy.
- Diagnostic laparoscopy.
- Colposcopy (± biopsy).
- Cone biopsy of cervix uteri.
- Marsupialization of Bartholin cyst.

Ophthalmological procedures

- Extraction of cataract with or without implant.
- Correction of squint.
- Laser coagulation of ciliary body.
- Surgical trabeculectomy or other glaucoma procedures.
- Correction of ectropion.

Day case and minimally invasive surgery

Day surgery procedures

An increasing number of procedures in all aspects of surgery are being performed as day surgery. The key features that make a procedure suitable include:

- Low risk of major complications.
- Predictable recovery period not requiring specialist post-operative therapy or treatment.
- Post-operative analgesia that does not need routine opiates.
- Anaesthetic technique not requiring invasive monitoring, prolonged muscle relaxation, or epidural/spinal anaesthesia.
- Low risk of difficult or unpredictable anaesthetic technique.

Many areas of surgery are now performed routinely as day surgery, including minor and intermediate anorectal surgery, hernia surgery, minor laparoscopic surgery, arthroscopy, and minor endoscopic bladder surgery.

Selection of patients for day case surgery

Most hospitals have well-defined protocols to select patients for suitability for day surgery and most day surgery units conduct their own pre-admission assessment either by telephone or questionnaire. Typical criteria might include:

- Maximum age of 75y (this upper limit has gradually ↑ as familiarity with the procedures has grown).
- Appropriate social support for the patient at home, including transport and a responsible adult to monitor progress.
- No history of more than mild to moderate cardiac or respiratory disease (e.g. uncomplicated asthma or controlled angina).
- Non-insulin-dependent diabetes only (unless for LA procedures).
- BMI below 35 (typically)—higher than this is associated with ↑ risk of anaesthetic and surgical complications.

Minimally invasive surgical procedures

Minimally invasive surgery is becoming commoner in many areas of surgery. It is a broad term that includes many types of procedure and there is much overlap with conventional 'open' surgery and, at the other end of the spectrum, interventional radiological procedures. A useful definition of minimally invasive surgery is a procedure that can be performed by a technique involving fewer or smaller incisions than alternative 'conventional' surgery or under less invasive anaesthetic techniques. This includes most laparoscopic and thoracoscopic surgery (cholecystectomy, gastric fundoplication, colectomy, lobectomy, nephrectomy, adrenalectomy). It also includes flexible and rigid endoscopic procedures (diagnostic and therapeutic colonoscopy, cystoscopy, transurethral prostate surgery, hysteroscopic surgery) and several procedures using specific techniques or equipment (e.g. transanal endoscopic microsurgery, subfascial endoscopic venous surgery).

Advantages of minimally invasive surgery

Many minimally invasive surgical techniques require specific training to perform and utilize expensive equipment and consumables, so surgeons and managers look to minimally invasive surgery to provide benefits to both patients and hospitals. Although some benefits can be achieved by modern post-surgical management, there are demonstrable benefits in different areas.

Patient benefits

- Smaller, fewer, or absent scars.
- Reduced time in hospital.
- Fewer post-operative complications (particularly wound- and respiratory-related).

Surgeon benefits

- Reduced post-operative stay.
- Possible avoidance of the need for interventional anaesthetic techniques such as epidurals.

Hospital benefits

- ↑ bed use efficiency.
- Reduced post-operative complications.

To whom should minimally invasive surgery be offered?

The advantages of minimally invasive surgery give it a wide application.

- *Young patients*. Small scars and short hospital stays are ideal.
- *Elderly*. Reduced post-operative complications and shortened hospital stay are vital in patients who often have multiple comorbidities.
- *Unfit patient*. Easier anaesthetic techniques and reduced surgical stress may reduce the perioperative risk.

Chapter 26

Emergency surgery topics

Acute abdominal pain

Acute abdominal pain is the commonest emergency surgical presentation to hospitals in the UK. Whilst the differential diagnosis is extensive, there are common tools that can be relied upon to arrive at a diagnosis and formulate an initial management plan.[1,2] Be methodical and remember some simple rules.

- Take a proper history and examination; do not work to the diagnosis given to you by the referring doctor.
- Resuscitate the patient properly and give adequate analgesia; this often helps to clarify the diagnosis. There is no reason to withhold analgesia prior to 'senior' clinical examination. With that, early on, try to identify the patient with sepsis and get the patient on the sepsis pathway (➲ Sepsis, p. 172).
- Diagnosing peritonitis is a slow-developing art that requires all your observation skills: watch senior surgeons examine patients. Determine whether it is present and localized or generalized. This will determine the urgency of care.

Causes

As a general rule, if the patient is able to localize the pain (and the doctor is able to localize the tenderness during examination) in their abdomen, then this suggests stimulation of the somatic pain fibres, which, in turn, suggests the disease has progressed to irritate the parietal peritoneum. Whilst the diagnosis gets easier, the urgency increases (see Box 26.1). Early on in the disease, however, the pain is generally non-specific in the midline, following the embryological origin of the diseased organ. In the case of the gut:

- Foregut (stomach, liver, gall bladder, pancreas, spleen, duodenum)—epigastric pain.
- Midgut (small bowel, appendix, right colon)—periumbilical pain.
- Hindgut (left colon, sigmoid colon, rectum)—hypogastric (suprapubic) pain.

Clinical features

Each condition has its own clinical features, but here are some rules to follow.

Symptoms and signs

- Constant pain, gradual in onset, but progressively worsening, suggests an underlying infection or inflammation in an organ.
- Colic is an intermittent pain that is poorly localized and suggests visceral pain (non-somatic) as above. It indicates peristalsis against an obstruction.
- Central and lower abdominal pain in children (under the age of 12) is self-limiting (non-specific) in 70%, from benign gynaecological causes in 25% (girls), and only pathological in 10–20%.
- Severe pain out of proportion with the clinical signs should precipitate suspicion of organ ischaemia which may require urgent surgery to save that organ.
- Pain in the loin or back arises from (at least partially) retroperitoneal structures; consider the pancreas, renal tract, and abdominal aorta.
- ♀ lower abdominal pain arising from gynaecological structures requires careful history to differentiate from other common surgical diseases such as appendicitis.

Box 26.1 Causes of acute abdominal pain arranged according to abdominal region

Right hypochondriac	Epigastric	Left hypochondriac
• Right lower lobe pneumonia/pulmonary embolism	• Pancreatitis • Gastritis • Peptic ulcer • Myocardial infarction	• Left lower lobe pneumonia/pulmonary embolism • Large bowel obstruction
• Cholecystitis • Biliary colic • Hepatitis		
Right lumbar	Umbilical	Left lumbar
• Renal colic • Appendicitis	• Intestinal obstruction • Intestinal ischaemia • Aortic aneurysm • Gastroenteritis • Crohn's disease	• Renal colic • Large bowel obstruction
Right iliac	Hypogastric	Left iliac
• Appendicitis • Crohn's disease • Right tubo-ovarian pathology	• Cystitis • Urinary retention • Dysmenorrhoea • Endometriosis	• Sigmoid diverticulitis • Left tubo-ovarian pathology

Gynaecological causes

- Complications of menstruation: retrograde menstruation, mid-cycle ovulation pain ('Mittelschmerz'), endometriosis.
- Ovarian cyst: bleed into a cyst, rupture, torsion.
- Tubo-ovarian infection, including PID, abscess.
- Ectopic pregnancy, including rupture and bleed.

Emergency management

Resuscitation

- Establish secure IV access.
- If criteria met for sepsis, start sepsis pathway, including early empirical antibiotics. (➲ Sepsis, p. 172).
- Catheterize and place on a fluid balance chart only if hypotensive.
- Give adequate analgesia. If renal pathology is suspected, diclofenac 100mg PR is very effective (avoid in asthma and renal disease). If intra-abdominal pathology is suspected, 5–10mg morphine IV is reasonable. Morphine IV never hides established clinical signs; it often helps to clarify the diagnosis by its anxiolytic effect on patients.
- Send blood for FBC (Hb, WCC), U&Es (Na+, K+), amylase, LFTs, CRP, and group and save.

Establish a diagnosis

The time frame for the diagnosis of acute abdominal pain varies according to the presentation. It is not uncommon for 12–24h of 'masterful inactivity' to be used to allow the diagnosis to be clarified. Young patients with central and mild RIF pain are typical of this sort of management. Do not assume this is normal. Some causes of acute abdominal pain require diagnosis and

management immediately upon admission or within 6–8h or less. Try to be thoughtful in diagnostic tests; many may be requested, but usually only one or two are really useful.

- Blood investigations are very rarely diagnostic. Serum amylase >3 times normal maximum is very highly suggestive of acute pancreatitis.
- Plain abdominal radiographs are very rarely diagnostic, except in obstruction.
- Erect chest radiograph can pick free abdominal air but is pointless in appendicitis.
- Upper abdominal ultrasound is an excellent investigation for suspected hepatobiliary pathology and is first line in a well patient with upper abdominal pain.
- Pelvic ultrasound, transabdominal (TA) or transvaginal (TV), is a good test for excluding gynaecological causes as above, but often misses appendicitis and surgical causes.
- CT scan is often requested in a sick patient after senior review when diagnosis is in doubt or of such severity that early confirmation is required. It should be used sparingly in young people to minimise lifetime accumulation of radiation.

Early treatment

- IV antibiotics are inappropriate without a clear diagnosis; they will suppress, but may not adequately treat, developing infection. The exception is sepsis.
- Until a definitive management plan is established, concentrate on fluid balance, analgesia, thromboprophylaxis, and monitoring vital signs.
- Keep patient NBM if an operation is likely. Remember gastric emptying is delayed in emergency patients, but as a minimum, 2h for clear fluids and 6h for solids are required.
- Communicate your thoughts clearly with the patient and your senior colleagues, and describe your management plan to the nurse in charge of looking after your patient. Many complaints, mistakes, and vital omissions arise from poor communication at this stage.

Decision-making

Whilst the diagnoses are many, the final actions required are remarkably limited (see Box 26.2). Once your initial investigations are back, practise making decisions without having to act on them, using the following chart. Good decisions come with practice and are best made explicit in order for feedback to be useful.[2]

References

1 Royal College of Surgeons of England (2011). *Emergency surgery: standards for unscheduled surgical care*. RCSENG, London.

2 Jessop ZM, Behar N (2014). A tool for training in decision making for emergency general surgery—explicit training is possible through facilitation. *J Surg Educ* **71**: 466–71.

Box 26.2 Decision-making

- Home with no follow-up.
- Home with Hot Clinic review.
- Discharge to another specialty.
- Admit for observation/conservative (non-operative) management.
- Admit for further investigations—operation unlikely.
- Admit for further investigations—operation likely.
- Admit as operation is needed as Expedited (CEPOD* Cat 3: after 24h).
- Admit as operation is needed as Urgent (CEPOD Cat 2b: within 24h).
- Admit as operation is needed as Very Urgent (CEPOD Cat 2a: within 6h).
- Admit as operation is needed as Immediate (CEPOD Cat 1: within 1h).

* CEPOD or NCEPOD, National confidential enquiry into patient outcome and death, provides guidelines on emergency surgical care. (p. 24) The term is also often used in clinical practice synonymously with emergency operating.

Intra-abdominal abscess

Key facts

Intra-abdominal sepsis can present as an intra-abdominal abscess if the sepsis is contained by tissues or anatomy. Common locations are:

- Alongside the organ of origin (e.g. paracolic in diverticulitis, parapancreatic after infected pancreatitis).
- Pelvic (especially after pelvic sepsis such as appendicitis or after generalized peritoneal infection).
- Subphrenic (e.g. after upper GI perforation).

Causes

- Sigmoid diverticulitis (➲ Acute Diverticulitis , p. 992).
- Acute appendicitis (➲ Acute appendicitis, p. 972).
- Severe acute cholecystitis (➲ Acute cholecystitis, p. 976).
- Upper GI perforation (➲ Acute upper GI perforation, p. 970).
- Post-operative, e.g. anastomotic leakage.
- Infected acute pancreatitis (➲ Acute pancreatitis , p. 980).
- Post-trauma.
- Gynaecological.

Clinical features

Depending on the source, the preceding pathology may have specific clinical features, but the development of an abscess gives rise to certain common features, independent of the origin.

Symptoms

- Malaise, anorexia, loss of appetite.
- Localized abdominal pain—constant.
- Sweats and rigors.
- Diarrhoea or constipation.

Signs

- Swinging fever, typically peaks in excess of 38.5°C, occurring twice a day.
- Tachycardia tends to follow the temperature.
- Localized abdominal tenderness, with a possible mass if abscess is in an accessible position and is large enough (e.g. paracolic).

Emergency management

Resuscitation

- Establish large-calibre IV access in a straight vein.
- Catheterize and place on a fluid balance chart only if hypotensive.
- Give adequate analgesia (e.g. 5–10mg morphine IV).
- Send blood for FBC, U&Es, LFTs, CRP, group and save, clotting, and blood cultures.

Establish a diagnosis

- Helical CT scanning is the diagnostic investigation of choice.
- Pelvic ultrasound (TA or TV) is occasionally useful if a pelvic abscess is suspected and CT scanning is to be avoided due to age.

Early treatment

IV antibiotics should be given, according to the most likely underlying diagnosis and organisms. Use established local hospital antibiotic guidelines and expect to change treatment after 48h when cultures available. Do not forget thromboprophylaxis.

Definitive management

- Interventional radiologically guided drainage by ultrasound or CT scanning is preferred, depending on availability. Limitations include multi-loculated abscesses and bowel in the way of drainage.
- Laparoscopy or, failing that, laparotomy and surgical drainage usually indicated if:
  - Radiological drainage not possible or safe.
  - Radiological drainage fails to adequately drain the abscess, as evidenced by ongoing signs of sepsis and rising CRP or the abscess recurs at repeat CT scan.
  - Surgical treatment is required for the 1° underlying pathology, in addition to drainage of the abscess.
- Occasionally, long-term antibiotics are used despite above treatments, guided by microbiology.

Acute peritonitis

Defined as acute inflammation in the peritoneal cavity.

Causes

May be 1° (rare) or 2° (common).

- 1° peritonitis. Typically streptococcal, with probable portal of entry via bloodstream, rather than intra-abdominal organs. SBP, a variant, occurs in cirrhotic and dialysis patients.
- Commonest causes of 2° peritonitis are:
  - Acute perforated appendicitis (➔ Acute appendicitis , p. 972)—commonest cause of peritonitis, especially in under 45s.
  - Acute perforated diverticular disease (➔ Acute diverticulitis, p. 992)—commonest cause in the elderly.
  - Upper GI perforation (duodenal ulcer, perforated gall bladder) (➔ Acute upper GI perforation, p. 970).
  - Perforated tumours (colonic, rarely gastric, small bowel after chemotherapy for lymphoma).
  - Perforated ischaemic bowel, e.g. due to adhesions.
  - Acute pancreatitis, usually inflammatory, rather than infective (➔ Acute pancreatitis, p. 980).
  - Post-surgical intervention, e.g. anastomotic leak, enteric injury.
  - Fulminant colitis, due to infection, UC, or Crohn's disease.
  - Intra-abdominal bleeding.

Clinical features

There are features common to all causes. Additional features suggestive of an underlying cause should also be sought, particularly in the history.

Symptoms

- Anorexia and fever.
- Severe generalized abdominal pain radiating to shoulders and back.
- Abdominal pain worse with movement, coughing, and sneezing.

Signs

- Fever, tachycardia.
- Generalized abdominal tenderness with guarding and rigidity.
- Differential maximal tenderness *may* indicate the possible underlying cause.
- Gentle palpation may allow identification of an underlying mass.

Emergency management

Resuscitation

- Establish large-calibre IV access in a straight vein.
- Catheterize and place on a fluid balance chart.
- Send bloods for FBC (Hb, WCC), U&Es (eGFR), CRP, amylase, clotting, LFTs, and group and save.
- ABGs if shocked or ischaemic bowel/pancreatitis suspected.

Establish a diagnosis

Most causes of acute peritonitis require surgery to correct them, but surgery is contraindicated in acute pancreatitis, except later if complicated or cholecystectomy is required to prevent future attacks.

Initial diagnostic investigations, along with plain AXR and erect CXR, may be sufficient to proceed to surgery to save time. ▶▶ In peritonitis: speedy source control of sepsis = improved survival.

If diagnostic uncertainty exists, then abdominal CT scanning is the investigation of choice. It can often locate the probable source of peritonitis if proper history is given to the radiologist.

The laparoscopic approach is frequently preferred over laparotomy for diagnostic uncertainty, to minimize wound complications, and to speed up recovery. It has become the gold standard in appendicitis.

Early treatment

IV antibiotics are appropriate without a clear diagnosis, as per sepsis guidelines, on an empirical basis (e.g. metronidazole 500mg IV tds + cefuroxime 750mg IV tds).

Definitive management

Acute appendicitis(➲ Acute appendicitis, p. 973)
Upper GI perforation(➲ Acute upper GI perforation, p. 971)

Perforated diverticular disease

- IV antibiotics (e.g. cefuroxime 750mg tds + metronidazole 500mg tds or co-amoxiclav 1.2g tds on its own, with gentamicin added for severe infections).
- Surgical treatment involves resection of the affected segment. Depending on the length of time from perforation, extent of the contamination, and extent of inflammation in the affected segment, the bowel may be anastomosed (1° anastomosis) or the proximal end brought out onto the abdominal wall as an end colostomy ('Hartmann's type' resection). Laparoscopic washout without resection in carefully selected patients also has promising outcomes.
- Perforated tumour surgical resection is required, even if palliative. Ends of bowel may be re-anastomosed or exteriorized as stomas, depending on the circumstances (degree of contamination, underlying pathology).
- Generally, surgeons avoid bowel anastomosis in very sick and elderly patients on inotropic support, as the risk of anastomotic leak is much higher. Stomas are often, but not always, reversible at a later date.

Primary peritonitis or continuous ambulatory peritoneal dialysis (CAPD)-related peritonitis

- A diligent and systematic search before and during surgery is necessary to ensure there is no occult source of perforation as a cause. Self-sealed fish bone perforation is not uncommon and Meckel's diverticulitis will be missed unless looked for.
- 1° treatment is extensive laparoscopic lavage of all quadrants and treatment with appropriate antibiotics (guided by culture results from peritoneal fluid).

Acute upper GI perforation

Causes and features

- Duodenal ulceration.
- Gastric ulceration (usually anterior prepyloric; less commonly anterior body).
- Gastric carcinoma.
- Traumatic, e.g. severe vomiting or high-velocity impact on full stomach.
- Ischaemic (usually 2° to gastric volvulus).
- Post-bariatric surgery such as gastric bypass, sleeve gastrectomy, or gastric band or balloon.
- ERCP complication typically due to unsuspected duodenal diverticulum.

Symptoms

- Acute-onset upper abdominal pain. Severe, constant, worse with breathing and moving; may radiate to back or shoulders.
- Prodrome of upper abdominal pain (in benign or malignant ulceration).
- Copious vomiting and upper abdominal distension suggest volvulus.
- Prodrome of weight loss, vomiting, dyspepsia, and anorexia suggests carcinoma.

Signs

- Generalized peritonitis common ('board-like' generalized rigidity with marked guarding and tenderness).
- Localized upper abdominal peritonitis may occur, especially with previous surgery where adhesions may act to contain the contamination.
- Mild fever, pallor, tachycardia, and hypotension (often profound due to autonomic reaction); typically respond quickly to modest fluid resuscitation.

Emergency management

Resuscitation

- Establish large-calibre IV access; give rapid crystalloid fluid up to 1000mL if tachycardic or hypotensive.
- Catheterize and place on a fluid balance chart.
- Send bloods for FBC (Hb, WCC), U&Es (Na+, K+, eGFR), LFTs (albumin),CRP, group and save, and clotting.

Establish a diagnosis

- Erect CXR (free gas only seen 60% of the time). If the CXR is non-diagnostic, a CT scan is often required to establish diagnosis. Radiologist will need to give IV contrast medium (potentially nephrotoxic) which requires eGFR above 35.

Early treatment

- Once the diagnosis of perforation is confirmed on clinical or radiological grounds, treatment is surgical unless:
  - The patient declines.
  - The patient is considered to have too poor quality of life or is unlikely to survive and supportive care is deemed more appropriate.
- Conservative management (IV PPI, limiting oral intake, active physiotherapy)—has very limited role in management; offers an outcome similar to that of surgery only in cases where the perforation has sealed at the time of presentation, there is no haemodynamic instability, and there are no signs of peritonitis.

Definitive management

Duodenal ulcer

- Laparoscopic or open sutured closure with omental patch.
- Empirical *Helicobacter pylori* eradication after discharge.
- Duodenal exclusion with Roux-en-Y gastrectomy is rarely required for large and obstructing ulcers.

Gastric ulcer

- Sutured closure with omental patch if prepyloric.
- Local excision and sutured closure if body.

Gastric carcinoma

- Partial gastrectomy (usually palliative).

Traumatic

- Sutured closure.

Volvulus with irreversible ischaemia.

- Usually subtotal gastrectomy.

Acute appendicitis

Causes and features

Commonest cause of urgent abdominal surgery in the UK.

- Can affect any age, but uncommon under the age of 4 and over the age of 80. Peak age of incidence is early teens to early 20s.
- It is best classified according to the degree of peritonitis at diagnosis:
  - Mild: well patient with localized peritonitis can wait for surgery overnight.
  - Severe: sick patient with generalized peritonitis ▶▶ requires very urgent surgery, whatever the hour.

Differential diagnosis

- Children.
  - Non-specific abdominal pain, including 'mesenteric adenitis'.
  - Meckel's diverticulitis.
  - Ovarian cyst/menstrual symptoms (peri-menarchal girls).
- Adults.
  - Terminal ileal pathology. Crohn's, Meckel's diverticulitis, gastroenteritis.
  - Retroperitoneal pathology. Pancreatitis, renal colic.
  - Ovarian pathology. Ectopic pregnancy, cyst, infection, menstrual pain.
- Older adults.
  - Ileocaecal pathology. Caecal diverticulitis, caecal tumours.
  - Colonic pathology. Sigmoid diverticulitis (sigmoid flopped over to the right).
  - Ovarian pathology. Cysts, infection, tumours (ruptured dermoid cyst).

Clinical features

- Symptoms.
  - Malaise, anorexia, loss of appetite, and fever.
  - Abdominal pain starts centrally and localizes to the RIF.
  - Abdominal pain caused by coughing and moving.
  - Dysuria can occur in pelvic appendicitis and may be mistaken for UTI.
  - Diarrhoea is a common delayed symptom and may be mistaken for acute gastroenteritis.
- Signs.
  - Fever, tachycardia.
  - Abdominal tenderness. Peritonitis suggests perforation if generalized and severe, and serositis if localized. Often maximal over 'McBurney's point', which is the topographical anatomical location for the base of the appendix.
  - Palpation of the LIF causes pain worse in the RIF (Rovsing's sign).

Complications

- Perforation (can be localized, but more often generalized).
- RIF 'appendix mass' (usually appendicitis with densely adherent caecum, omentum, and ileum, forming a 'mass').
- RIF abscess (usually 2° to perforated retrocaecal appendicitis).
- Pelvic abscess (usually 2° to perforated pelvic appendicitis).

Emergency management

Resuscitation

- Establish IV access and rehydrate rapidly.
- Catheterize and place on a fluid balance chart only if hypotensive or septic.
- Request FBC (Hb, WCC), U&Es, CRP, group and save, clotting, amylase, and LFTs.

Establish a diagnosis

- The diagnosis is a clinical one in most cases. Remember the rule of 3s:
  - Only 3% of patients will have appendicitis with RIF pain, if both WCC and CRP are normal.
  - Also in 3% of patients, CT scan will miss appendicitis.
- CT is appropriate in adults over the age of 65 or if the diagnosis is unclear (such as patients with inflammatory bowel disease) since the differential diagnosis is much wider and appendicitis relatively less likely above the age of 65.
- CT is the best investigation in suspected appendix mass or abscess.
- USS (pelvis) is indicated in young women of childbearing age to exclude gynaecological pathology, not to diagnose appendicitis. It is therefore useless in men.

Early treatment Avoid giving IV antibiotics without a clear diagnosis unless septic. Remember analgesia and thromboprophylaxis. Keep NBM in anticipation of early surgery.

Definitive management

▶ *Acute appendicitis*

Laparoscopic appendicectomy (rather than open incision centred on McBurney's point) is the gold standard approach, especially in women, allowing diagnosis of other pelvic pathology, e.g. PID. Avoids unnecessary appendicectomy, although the appendix is often removed, even if normal-looking, if no other significant pathology is found. A normal appendicectomy rate of 10% is accepted in order not to miss inflamed cases. A normal appendix can only be established after definitive histopathological examination. A single dose of IV antibiotic is required 30min before induction; a full course is only indicated in perforated appendicitis or with significant intra-abdominal evidence of peritonitis with free pus in multiple quadrants. A drain is often left in the latter.

➲ Diagnostic Laparoscopy p. 842; ➲ Appendicectomy p. 846.

Appendix mass or appendix abscess

- May be diagnosed on palpating a mass preoperatively or during examination of the patient under GA (EUA). Preoperative CT scan will help decision-making. It is often an indicator of delayed diagnosis/presentation and is best treated with IV antibiotics (e.g. cefuroxime 750mg tds + metronidazole 500mg tds) and CT/laparoscopic-guided drainage if an abscess is present.
- When symptoms settle without removing the appendix as above, delayed elective (interval) appendicectomy after 3 months is recommended, as the recurrence rate is high.
- If symptoms fail to settle, may need acute appendicectomy, which is likely to be difficult and to require laparotomy.

Key revision points—anatomy of appendicectomy

- Commonly retrocaecal, but may be pelvic, retroileal, or retrocolic.
- Taenia coli of caecum converge at base of appendix and aid location intraoperatively, especially when in difficult locations.
- Has a small mesentery with sole blood supply from appendicular artery (a terminal branch of ileocolic), which may thrombose, causing gangrene.
- Principles of appendicectomy are as follows:
  - Laparoscopic approach preferred.
  - Appendix is carefully located, and inflammation confirmed.
  - The mesentery of the appendix is divided and ligated.
  - The appendix is tied at the base and divided, avoiding further contamination.
  - Peritoneal cavity is washed locally or generally with saline, as dictated by extent of contamination.

Acute cholecystitis

Severe continuous RUQ pain; often radiates to right flank and back, associated with anorexia and pyrexia. Tenderness over gall bladder during inspiration (Murphy's sign).

Complications of acute cholecystitis include:

- *Empyema*. Formation of an empyema of the gall bladder (abscess by virtue of pus being contained in the gall bladder that is obstructed) is rare. Indicated by high swinging fever and severe localized pain.
- *Perforation*. With biliary peritonitis is very rare. Can be acalculous in ICU patients.
- *Gallstone ileus*. Cholecystoenteric fistula formation (may lead to a gallstone entering and obstructing the distal ileum)).
- *Jaundice*. Due to compression of the adjacent CBD by pressure ('Mirizzi syndrome') or by virtue of a gallstone obstructing the CBD.
- *Biliary colic*. Intermittent severe epigastric and RUQ pain, usually associated with nausea and vomiting. Resolves after few hours; tenderness over gall bladder during acute episodes is possible.
- *Chronic cholecystitis*. Repeated episodes of infection causes thickening and fibrosis of the gall bladder.
- *Mucocele*. Stone in neck of gall bladder; bile is absorbed, but mucus secretion continues, producing a large, tense globular mass in RUQ. If infected, this becomes an empyema.

Diagnosis and investigations

- *FBC, U&Es, LFTs,* blood culture, serum amylase—in acute presentations.
- *Erect CXR*. To exclude ulcer perforation or pneumonia as differential diagnosis.
- *Ultrasound*. Investigation of choice; identifies stones, determines wall thickness, and assesses ductal dilatation—due to a stone within the duct (➲ Common Bile Duct Stones, p. 978).
- *HIDA scan*. Rarely used if diagnostic difficulty in clinic.

Early treatment

- IV antibiotics (e.g. co-amoxiclav 1.2g tds or piperacillin/tazobactam 4.5g tds).

'Hot'/emergency cholecystectomy is recommended if the patient's symptoms developed within the preceding 72h and imaging confirms the absence of CBD stones where there is expertise and theatre availability as a one-stop treatment. Otherwise the patient has to wait at least 6 weeks following an episode of acute cholecystitis for elective cholecystectomy.

Definitive surgical treatment

Cholecystectomy ➲ *Cholecystectomy {Chapter 21}*

Majority done laparoscopically; often as a day case. This is the treatment of choice for all patients fit for GA. Indicated for:

- Patients with symptoms deemed to be due to gall bladder stones.
- Asymptomatic patients with gall bladder stones at risk of complications—diabetics, porcelain gall bladder (15–20% associated with carcinoma), history of gallstone pancreatitis, long-term immunosuppressed, sickle cell disease.

Risks of laparoscopic cholecystectomy

- Conversion to open operation, 5–10%.
- Bile duct injury, 0.4%.
- Bleeding, 2%.
- Bile leak, 1%.

NB Risks increase slightly, but acceptably, in 'hot' cholecystectomy.

Non-surgical treatments

Percutaneous drainage of gall bladder

- Done under ultrasound or CT guidance. Transhepatic to avoid biliary peritonitis.
- Used for empyema of the gall bladder in patients unsuitable for emergency cholecystectomy.
- Drain needs to stay for at least 10 days for tract to form.
- After resolution of the infection, the patient may be brought back for elective cholecystectomy.

For patients who are high risk and not suitable for GA

- Repeated courses of antibiotics, with avoidance of fatty foods, for prevention of recurrent attacks of cholecystitis.
- ERCP and sphincterotomy for the prevention of further episodes of gallstone pancreatitis.

Common bile duct stones

Key facts

- Types of stones as per gall bladder stones; 90% not visible on plain X-ray. ➲ Gallstones, p. 412
- CBD stones present in 10% of patients with gallstones.
- Most pass from the gall bladder into the CBD (2° duct stones).
- Rarely form within the CBD (1° duct stones); almost always associated with partial duct obstruction.

Clinicopathological features

Asymptomatic. Usually found incidentally on ultrasound for gall bladder stones.

Obstructive jaundice ➲ *Jaundice, p. 406*

- Usually due to CBD stone causing obstruction; rarely due to stone-induced CBD stricture.
- Anorexia, nausea, itching.
- Dark urine and pale stools.
- Epigastric pain and fever commoner with CBD stones than other cause; due to associated low-grade bile infection.
- A palpable, distended gall bladder is rare with CBD stones.

Courvoisier's law

'If in the presence of jaundice, the gall bladder is palpable, then the cause of the jaundice is unlikely to be due to a stone.'

This is due to the fact that CBD stones originate in the gall bladder which is usually scarred and fibrotic, preventing distension.

- *Ascending cholangitis*. Constant, severe RUQ pain, obstructive jaundice, and high swinging fever ('Charcot's triad').
- *Acute pancreatitis*. 60% of acute pancreatitis in adults in the UK are due to gallstones (➲ Acute pancreatitis, p. 416).

Diagnosis and investigations

Basic tests

- FBC (high WCC in cholangitis and pancreatitis), U&Es, Cr, LFTs (high conjugated bilirubin and ALP), serum amylase (↑ in pancreatitis), and clotting studies (deranged in liver disease and requires correction with vitamin K to enable therapeutic ERCP).

Advanced tests

Ultrasound (transabdominal)

- Best first-line investigation.
- Accuracy low for distal CBD stones, in acute presentations, obesity, and with extensive overlying bowel gas.

MRCP

- Investigation of choice for inconclusive ultrasound result.
- Non-invasive, avoids radiation exposure, highly accurate.

ERCP

- Used diagnostically for patients unable to tolerate MRCP (claustrophobia).
- Mainly reserved for therapeutic interventions:
  - Endoscopic sphincterotomy (ES) and stone extraction or destruction (lithotripsy).
  - Stent insertion for unextractable stones.
- Risks of ERCP (higher with ES):
  - Haemorrhage.
  - Acute pancreatitis.
  - Ascending infection.
  - Perforation (usually retroduodenal, may cause peritonitis).

PTC

- Used for failure of ERCP as therapeutic procedure (often in combination with ERCP).
- Risks include sepsis, tube movement, leakage around the tube, and dehydration.

Treatment

Principles of treatment of CBD stones are as follows.

Emergency treatment of CBD stones

- Indicated in unresolving gallstone pancreatitis and unresolving ascending cholangitis.
- Usually ERCP with stone extraction or stent insertion.
- Occasionally PTC required.

Elective treatment of CBD stones

- Indicated for:
  - All patients having had complications (pancreatitis, cholangitis, obstructive jaundice).
  - All patients with CBD stones prior to cholecystectomy.
- Usually by ERCP or combined ERCP/PTC.
- CBD exploration required at time of cholecystectomy (laparoscopic or open) if ERCP/PTC fail or impossible or as surgeon's preference. Open CBD exploration may require a T-tube to be left in the CBD.

Treatment of persistent CBD stones after cholecystectomy

- Rarely necessary with more accurate preoperative diagnosis and more effective preoperative treatments.
- Stones can be extracted via a T-tube track if present (6 weeks after surgery with radiologically guided basket extraction).
- Post-operative ERCP is rarely required.

Acute pancreatitis

Causes and features

Inflammatory process with cascade of release of inflammatory cytokines (TNF-α, IL2, IL6, PAF) and pancreatic enzymes (trypsin, lipases, co-lipases) initiated by pancreatic injury, but which may develop into full-blown multiple organ dysfunction syndrome (MODS) or SIRS).

Causes

- Gallstones (60%).
- Alcohol (30%).
- Hyperlipidaemia (needs to be very high such as in hereditary hyperlipidaemias).
- Hypercalcaemia (hyperparathyroidism, multiple myeloma).
- Direct damage (trauma, ERCP, post-surgery, CPB).
- Toxins:
  - Drugs, e.g. azathioprine, oestrogens, thiazides, isoniazid, steroids, NSAIDs.
  - Infection, e.g. viral (mumps, CMV, hepatitis B), *Mycoplasma*.
  - Venom (scorpion, snake bites).
- Idiopathic.
- Pancreatic cancer.

Classification/complications

- Oedematous (70%). May be simple or associated with phlegmon formation; transient fluid collections common.
- Severe/necrotizing (25%). Necrosis may be sterile or infected. Persistent, large peripancreatic fluid collections may form ('pseudocyst'), which may become infected and/or cause long-term pain.
- Haemorrhagic (5%).

Clinical features

- Severe epigastric pain radiating to the back.
- Severe nausea and vomiting.
- Fever, dehydration, hypotension, tachycardia (may be frankly shocked).
- Epigastric tenderness associated with guarding and, in severe cases, rigidity which may be generalized.
- Left flank ecchymosis (Grey Turner's sign) and periumbilical ecchymosis (Cullen's sign) in 1–3% of cases with haemorrhagic pancreatitis.

Emergency management

Resuscitation

- Establish large-calibre IV access. Give crystalloid fluid up to 1000mL if tachycardic or hypotensive; may require ongoing fluids IV.
- Catheterize and place on a fluid balance chart.
- Send bloods for FBC (Hb, WCC), U&Es (Na+, K+), LFTs (bilirubin, albumin), amylase, LDH, group and save, and clotting.
- Monitor pulse rate, BP, and urine output (urinary catheter).
- Consider insertion of a central line and manage patient in HDU if haemodynamically unstable or fails to respond to early resuscitation.
- Assess the severity of the attack by the Glasgow Imrie criteria (see Box 26.3).

Box 26.3 Glasgow Imrie criteria

Three or more positive criteria within 48h of admission = severe attack (mnemonic: PANCREAS):

- PaO_2 <8kPa.
- Age >55y.
- Neutrophils/WCC >15 000 × 10^9/L.
- Corrected Ca^{2+} <2mmol/L.
- Raised blood urea >16mmol/L.
- Elevated Enzymes: AST >200U/L, LDH >600U/L.
- Albumin <32g/L.
- Sugar: blood glucose >10mmol/L.

Establish a diagnosis

- *Serum amylase* >1000U. Diagnostic, but may be normal, even in severe cases; elevated amylase may occur in a wide range of other acute abdominal events (intestinal ischaemia, leaking aneurysm, perforated ulcer, cholangitis).
- *Serum lipase*. Remains elevated longer than serum amylase; more specific, but less sensitive.
- *AXR (non-specific findings)*. Absent psoas shadows, 'sentinel loop sign' (dilated proximal jejunal loop adjacent to pancreas because of local ileus), 'colon cut-off sign' (distended colon to mid-transverse colon, with no air distally); may show gallstone and pancreatic calcification.
- *CT may be required* in diagnostic uncertainty or to exclude complications typically after 1 week of disease failing to settle. Shows pancreatic oedema, swelling, and loss of fat planes; may show haemorrhagic or necrotic complications.
- *USS*. Must be done within 48h of admission to identify gallstones in the bile duct.[1]

Early treatment

Patients scoring 3 or more (severe pancreatitis) should be reviewed by intensive care, as these patients have severe pancreatitis and are at risk of deteriorating and requiring organ support. They require aggressive monitoring and therapy:

- Urinary catheter to monitor fluid balance, with aggressive IV fluid replacement.
- NGT and NBM to prevent vomiting and rest bowel.
- IV morphine and antiemetics.
- DVT thromboprophylaxis.
- IV PPIs to prevent stress ulceration.
- Antibiotics in some cases.

Patients scoring below 3 are classified as mild pancreatitis and should be managed expectantly with oral diet, analgesia, and IV fluids, *without* a urinary catheter, NGT, antibiotics, or PPIs.

Urgent ERCP and stone extraction are indicated for proven bile duct stones causing obstruction and resulting gallstone pancreatitis.

Definitive management

Identifying & preventing complications

- Aggressive fluid balance and rehydration, along with effective analgesia.
- IV antibiotics (e.g. IV imipenem tds) are sometimes started in severe cases when there is evidence of infected necrosis. In majority of cases, they have no place.
- Delayed CT scan identifies development of pancreatic phlegmon, collections, necrosis, haemorrhage, or adjacent transverse colonic perforation.
- CT-guided pancreatic aspiration to culture infected necrosis to direct antibiotic therapy.
- Early low-volume enteral feeding in severe pancreatitis is increasingly used to reduce the risk of stress ulceration and bacterial translocation causing sepsis.

Treatment of early complications

- Consider treating all severe cases on HDU/ITU for optimized fluid balance and respiratory, cardiovascular, and renal support.
- Proven infected necrosis: surgical debridement may be required but is associated with a poor prognosis.
- Acute pseudocysts rarely need drainage, unless very large.

Overall outcome

- Mortality is associated with pancreatic necrosis and the presence of sepsis, including MODS.

References

1 UK Working Party on Acute Pancreatitis (2005). UK guidelines for the management of acute pancreatitis. *Gut* **54**: iii1–9.

2 National Institute for Health and Care Excellence (2018). *Pancreatitis*. Available at: https://www.nice.org.uk/guidance/ng104

Acute groin swelling

Causes of chronic testicular swelling are discussed on ➲ Scrotal swellings, p. 466. ➲ Acute testicular pain, p. 488.

Causes and features

- ***Incarcerated groin hernia*** (inguinal or femoral). May or may not be associated with intestinal obstruction; often red, hot, and tender.
- ***Acute epididymo-orchitis*** (in ♂). Tenderness is particularly over the spermatic cord and the epididymis.
- ***Torsion of the testis***. May present with pain in the groin, but unless the testicle is undescended, the tenderness is primarily over the scrotum (and testis). Testicle is tender, swollen, and high-riding. Elevation of the scrotum, unlike in epididymitis, makes the pain worse.
- ***Iliopsoas abscess***. Tenderness primarily below the inguinal ligament; may be fluctuant, associated tenderness in the RIF due to underlying pathology.
- Acute iliofemoral ***lymphadenopathy*** (e.g. from infected toenail). Tender, diffuse swelling; often multiple palpable lumps (nodes).
- Acute ***saphena varix***. Compressible, cough thrill.
- Acute complications of femoral aneurysm, commonly pseudoaneurysm 2° to IV drug abuse or angiography via the groin.

Emergency management

Resuscitation

- Establish IV access; consider giving crystalloid fluid if there is suspicion of a complicated hernia or an iliopsoas abscess.
- Catheterize and place on a fluid balance chart if hypotensive.
- Send bloods for FBC (Hb, WCC) and U&Es (Na+, K+).
- Group and save and clotting if arterial disease or abscess formation is suspected.

Establish a diagnosis

- ►► Torsion of the testis is a true surgical emergency and should not wait long prior to exploration. Colour flow Doppler assessment may be able to confirm the presence of a hyperaemic testis, but unless it is immediately available, it should not delay surgical exploration. If there is a strong clinical history in a young ♂, immediate operation remains the diagnostic investigation of choice.
- If an iliopsoas abscess is suspected, abdominopelvic CT scanning is the investigation of choice.
- If the history is inconclusive and the patient is clinically well with normal bloods, a groin ultrasound can aid diagnosis and subsequent management.

Definitive management

Incarcerated groin hernia

- Repair is indicated for all patients, except those considered unfit for any surgical procedure or those declining treatment.
- It may not be possible to establish if the hernia is inguinal or femoral preoperatively; if so, it is safest to approach as if for an inguinal hernia.
- Femoral hernias may be approached via an infra-inguinal or a trans-inguinal dissection.
  - Infra-inguinal approaches may be limited in exposure if there is necrotic bowel within the hernia requiring resection.
  - A trans-inguinal approach will involve repair of the inguinal canal as well but offers an almost unlimited exposure of the femoral canal from above and allows plenty of exposure for bowel resection.
- Inguinal hernias should be approached through a conventional incision. Repair may require a mesh, although there is an ↑ risk of infection if there is an associated bowel resection.
- Anaesthesia will often need to be general in an emergency repair.
- Laparoscopic approach is possible in experienced hands and has the added benefit of inspecting the bowel intraperitoneally for reversible ischaemia without an unnecessary laparotomy.

Psoas abscess

The underlying cause should be identified as a matter of priority. Incision and drainage of the groin collection may be indicated, but only as part of the overall treatment.

Torsion of the testis

- Once identified, the affected testis should be assessed; if non-viable, a simple orchidectomy is performed and if viable, an orchidopexy is the operation of choice.
- If the diagnosis is confirmed, the contralateral testicle should also be fixed by orchidopexy to prevent subsequent torsion.

Epididymo-orchitis

- Antibiotics PO (e.g. ciprofloxacin 500mg od) for 14 days or IV if severe.

Small bowel obstruction

Key facts

Small bowel obstruction (SBO) is a common acute surgical presentation, with varying underlying causes which direct subsequent management. It can be broadly categorized into paralytic obstruction, where there is abnormal or absent peristalsis (as with post-operative ileus), and mechanical obstruction where the bowel is trying to overcome a physical obstruction

Causes

- Adhesions[1]—post-operative, congenital bands.
- Hernia—acquired, incisional, congenital.
- Tumour—caecal, small bowel (carcinoma, lymphoma, sarcoma).
- Gallstone ileus.
- Foreign body—bezoar, food bolus, parasite.
- Inflammatory conditions—Crohn's disease, TB, post-operative stricture.

Simple obstruction occurs when the blood supply to the affected segment of bowel remains intact. The patient is uncomfortable, but not in severe pain, and there is time to investigate and, if necessary, to operate later.

In strangulated obstruction, the blood supply to a bowel loop is also cut off by way of a congenital band, an adhesion, or a very tight hernial neck. The patient is in severe pain and may be acidotic on ABGs. Very urgent surgery is required within 6h to save bowel and life.

Clinical features

The presentation varies, depending on the level and underlying cause of obstruction and the time course of obstruction.

Symptoms and signs

- Abdominal pain (see above for severity).
- Abdominal bloating with tympanic bowel sounds.
- Vomiting and dehydration.
- Absolute constipation (not even flatus).

Emergency management

Resuscitation

- Establish large-calibre IV access and rehydrate with IV fluids.
- Give adequate analgesia (e.g. 5–10mg morphine IV).
- NGT decompression.
- Catheterize and place on a fluid balance chart.
- Check serial lactate levels for an indication of ischaemic bowel.
- Send bloods for FBC (Hb, WCC), U&Es (K+), CRP, group and save, and clotting.

Establish a diagnosis

- Plain AXR and erect CXR.
- Majority of SBO is due to adhesions from previous laparotomies. Provided the patient is relatively well, with minimal pain, on admission, 100mL of Gastrografin® mixed with 100mL of water can be given via NGT or PO, followed by serial AXRs to 'chase' the Gastrografin®. If after 24h, Gastrografin® has not reached the colon, the obstruction is unlikely to resolve and the patient should be prepared for surgery.

Further delay in the hope of spontaneous resolution is likely to compromise the patient nutritionally and adversely affect post-operative recovery (NASBO trial).

- Abdominopelvic CT is useful in patients with a 'virgin' abdomen with no previous surgery, as the underlying pathology is likely to be discovered such as tumours. In addition, it will also pick up an internal hernia in a bariatric surgery patient some years following gastric bypass surgery with successful weight loss. A 'mesenteric swirl' is a typical radiological description.

A thorough history and clinical assessment are crucial to identify the underlying cause and determine the subsequent management plan. Most obstructing hernias should be obvious and operated on without investigative delays.

Early management

In a stable patient without evidence of strangulation, ischaemia, or perforation, a conservative 'drip and suck' regimen is tried, with administration of Gastrografin® as above. The patient is kept NBM, administered IV fluids, with a large-bore NGT for bowel decompression. The majority of cases of SBO 2° to adhesions resolve within 24h, as evidenced by bowel function or passage of Gastrografin® into the colon on plain AXR.

Surgical management

The specific underlying cause of the obstruction dictates when surgical intervention should be considered.

Surgical intervention within 6h (CEPOD Cat 2a)

- Peritonitis.
- Evidence of perforation.
- Evidence of strangulated or necrotic bowel.

Surgery in 24–48h

- CT finding of significant pathology requiring surgery.
- Non-resolution of SBO with 'drip and suck' and Gastrografin®.

Occasionally, longer periods of wait are tolerated in patients with complex previous operations who are deemed high-risk surgical candidates.

Large bowel obstruction

Key facts

Large bowel obstruction presents in a similar way to SBO, but as the obstruction is distal, vomiting is a delayed symptom. The underlying causes and subsequent management considerations are also somewhat different. Adhesions are never a cause and the colon rarely herniates through the abdominal wall, as it is much less mobile than the small bowel.

Causes

- Colorectal carcinoma.
- Volvulus—sigmoid or caecum.
- Pseudo-obstruction—related to almost all acute medical conditions (see below).
- Faecal impaction.
- Diverticulitis—often recurrent, causing fibrotic stenosis.
- Inflammatory bowel disease—typically Crohn's stricture, but also UC with toxic megacolon.
- Anastomotic stricture—often a very late surgical complication.
- Other malignancies causing external compression by invasion.

Pseudo-obstruction occurs when the patient has symptoms of large bowel obstruction, without any obvious mechanical cause for obstruction. These patients are often either critically unwell or have a predisposing condition such as chronic neurological conditions, acute medical conditions or electrolyte imbalances, orthopaedic trauma, or retroperitoneal malignancy (Ogilvie's syndrome). Supportive therapy, along with treatment of the acute medical condition, usually resolves the pseudo-obstruction.

Clinical features

The specific presentation varies, depending on the underlying cause of obstruction. However, some common features are listed below.

Symptoms and signs

- Abdominal pain.
- Abdominal bloating and distension, with absent or tympanic bowel sounds.
- Absolute constipation.
- Delayed vomiting.

Emergency management

Resuscitation

- Establish large-calibre IV access and rehydrate with IV fluids.
- Give adequate analgesia (e.g. 5–10mg morphine IV).
- NGT decompression.
- Catheterize and place on a fluid balance chart.
- Check serial lactate levels for an indication of ischaemia or peritonitis.
- Send bloods for FBC (Hb, WCC), U&Es (Na^+, K^+), Ca^{2+}, amylase, TFTs, clotting, CRP, and group and save.

Establish a diagnosis

- Plain AXR and erect CXR.
- Abdominopelvic CT is the examination of choice to get a definitive diagnosis and direct subsequent management.
- Occasionally, flexible sigmoidoscopy is used to treat by decompression and diagnose colonic causes of pseudo-obstruction.

On a plain AXR, caecal volvulus shows a characteristic grossly distended caecum, whilst a sigmoid volvulus often shows the classic 'coffee bean' sign. Once suspected, a CT scan is urgently indicated.

Definitive management

The specific underlying cause of the obstruction dictates the management plan.

►► *Emergency surgery (within 6h) indicated in the following situations*

- Generalized peritonitis.
- Evidence of perforation or ischaemia.
- Complete large bowel obstruction, with competent ileocaecal valve.
- Caecal volvulus.

Clinically stable patients found to have underlying colorectal carcinoma should be fully worked up medically and radiologically, prior to being considered for surgery. In patients who are medically unfit for surgery, endoscopic stenting of obstructing lesions should be considered where expertise is available. Another option is surgery to create a defunctioning trephine colostomy in palliative or unfit cases.

Sigmoid volvulus should be treated by flatus tube insertion in the first instance. Colonoscopic decompression should be considered if this fails.

Mesenteric ischaemia

Key facts

An embolus or thrombus involving the mesenteric circulation can cause mesenteric ischaemia in the acute setting, resulting in catastrophic illness and severe pain. A more chronic disease occurs with atherosclerosis, which results in abdominal cramps typically after food—so-called mesenteric angina. Patients at particular risk are older and smokers, have a history of atherosclerosis or cardiovascular disease, or have AF or a recent history of MI. Mesenteric ischaemia is rare and difficult to diagnose and therefore should always be suspected in the above patients with acute abdominal pain.

Clinical features

The clinical presentation of mesenteric ischaemia varies, depending on whether the presentation is acute and severe, or chronic and recurrent in nature. The common features of acute ischaemia are listed below.

Symptoms and signs

- Severe generalized abdominal pain, out of proportion to abdominal findings or observations.
- Nausea and vomiting.
- Diarrhoea ± bloody diarrhoea (redcurrant jelly).

In the early stages of acute ischaemia, physical examination can often be unremarkable. As ischaemia progresses to bowel infarction, the abdomen may become grossly distended, with absent bowel sounds, and peritonism is a late sign.

Emergency management

Resuscitation

- Establish large-calibre IV access and rehydrate with IV fluids.
- Give O2.
- Give adequate analgesia (e.g. 10–20mg morphine IV).
- Catheterize and place on a fluid balance chart.
- ABGs and serial lactate levels.
- Send bloods for FBC (Hb, WCC), U&Es (Na+, K+), CRP, amylase, LFTs, clotting, and group and save.

Establish a diagnosis

- Plain AXR and erect CXR.
- Abdominopelvic CT with contrast is the investigation of choice if the patient has an acute abdomen.
- CTA is the most useful investigation if there is a high level of suspicion of acute mesenteric ischaemia.

An abnormally high or rising lactate in the context of severe generalized abdominal pain increases the likelihood of acute mesenteric ischaemia.

Early management

Following resuscitation, patients in whom there is a high suspicion of acute ischaemia or presenting with peritonism should undergo emergency laparotomy and surgical resection of non-viable bowel, with or without anastomosis of the remaining bowel.

Cases of chronic ischaemia and non-occlusive thrombosis can be managed by non-operative supportive therapy, including anticoagulation or thrombolysis, if indicated.

Acute diverticulitis

Acute onset of LIF pain, nausea, fever, and frequently with loose stools. Usually febrile, with moderate tachycardia and LIF tenderness. Colonic wall shows acute neutrophil infiltration around the inflamed diverticulum and in the subserosal tissues.

Complications

Pericolic/paracolic mass/abscess

Acute diverticulitis may progress to persistent pericolic infection with thickening of surrounding tissues and the formation of a mass. If this suppurates, a pericolic abscess forms. Enlargement and extension of this into the paracolic area leads to a paracolic abscess. The features are those of acute diverticulitis with a swinging fever, fluctuating tachycardia, unresolving abdominal pain, and a tender LIF mass.

Peritonitis

Perforation of a pericolic or paracolic abscess usually leads to purulent peritonitis. Direct perforation of the acute diverticular segment leads to faeculent peritonitis. The features are of acute diverticulitis with high fever, severe abdominal pain, and generalized guarding and rigidity.

Diverticular fistula

Acute infection with paracolic sepsis may drain by perforation into adjacent structures. This is typically the posterior vaginal vault in women or the bladder in either sex. Colovesical fistula leads to recurrent UTI caused by enteric organisms, with bubbles and debris in the urine (pneumoturia and faecuria). Colovaginal fistula leads to faeculent PV discharge.

Stricture formation and obstruction

Chronic or repetitive inflammatory episodes may lead to fibrosis and narrowing of the colon. A history of recurrent diverticulitis with recurrent colicky abdominal pain, distension, and bloating suggests stricture formation.

Diagnosis and investigations

- CT scanning is the acute test of choice, as it can also identify complications, including abscess formation and perforation.
- Colonoscopy is contraindicated during an acute attack but is useful in making a definitive diagnosis after 6 weeks.
- Hb, WCC, and CRP during acute episodes of inflammation.

The Hinchey classification of peritoneal contamination

- Stage 1—pericolic or mesenteric abscess.
- Stage 2—walled-off pelvic abscess.
- Stage 3—generalized purulent peritonitis.
- Stage 4—generalized faecal peritonitis.

Treatment

Medical treatment

- IV antibiotics (co-amoxiclav 1.2g tds, with gentamicin added for severe infections) during acute infective exacerbations.
- Once patient is able to eat, a low-residue diet is recommended for 2 weeks, with high fluid intake and stool softeners to reduce intracolonic pressure and allow non-bulky stools to pass through the inflamed and narrowed segment in sigmoid colon. After this period, the patient will return to a healthy high-fibre diet.
- Recurrent infective episodes may be prevented by prolonged cyclical use of antibiotics and probiotics.
- Significant paracolic abscesses may be drained under CT radiological guidance (Hinchey 2).

Surgical treatment

- Resection is indicated in the acute setting for:
  - Acute inflammation failing to respond to medical management.
  - Undrainable paracolic sepsis.
  - Free perforation—Hinchey 3 and 4.
- Some Hinchey 2 and 3 patients are suitable for laparoscopic washout without resection but require careful observation post-operatively.
- The affected region should be resected (segmental colectomy). The ends may be re-anastomosed if they are healthy and the patient's general condition is suitable. If not, a proximal end colostomy and oversewing of the distal end is usual (Hartmann's-type resection).
- Diverticular strictures and fistulae are managed by surgery in the elective setting.

Acute severe colitis

Causes and features

Any cause of colitis may progress to acute severity. Common causes that may require colectomy include:

- Severe UC (usually pancolitis); occasionally, acute severe colitis is the presentation of UC with no prior history;
- Acute infective colitis (e.g. Escherichia coli O157, Clostridium difficile, Clostridium perfringens toxin A).
- Neutropenic colitis.
- Pseudomembranous colitis (C. difficile-related).
- Progressive Crohn's colitis (usually with a clear prior history).
- Cocaine abuse.

Symptoms

- Diarrhoea (usually bloody with urgency and frequency). 'Constipation' may be an ominous feature, suggesting acute colonic dilatation.
- Abdominal pain (generalized).
- Malaise, anorexia, and fever (systemic inflammatory features).

Signs

- Fever, tachycardia, possible hypotension, shock.
- Abdominal tenderness; peritonitis suggests perforation or impending perforation.

Complications

Any acute severe colitis may develop any of these complications:

- Haemorrhage.
- Hypokalaemia.
- Hypoalbuminaemia.
- Perforation (localized or generalized).

'Toxic dilatation/megacolon' is a term used to describe the situation of acute severe colitis with colonic dilatation of over 5.5cm, usually associated with reduced bowel frequency and impending perforation.

Fulminant severe colitis is defined as: tachycardia (HR >120bpm) or stool frequency >10 times/24h or albumin <25g/dL.

Emergency management

Resuscitation

- Establish large-calibre IV access; give crystalloid fluid up to 1000mL if tachycardic or hypotensive.
- Catheterize and place on a fluid balance chart.
- Send bloods for FBC (Hb, WCC), U&Es (Na+, K+), LFTs (albumin), group and save, clotting, amylase, CRP, and blood cultures.

Establish a diagnosis

- Send 'hot' stools for M,C,&S, as well as microscopy for ova, cysts and parasites. Even known colitics may catch acute infective colitis.
- Plain AXR (looking for colonic dilatation) and erect CXR (looking for free gas).
- Rigid sigmoidoscopy and biopsy, only if the patient is clinically stable.

- Flexible endoscopy may be indicated to establish a diagnosis, as CT may not differentiate between different types of colitis but carries a small risk of perforation.
- Early review by the surgeon if the patient is under a medical team.
- Stool chart is mandatory to monitor progress.

Early treatment

- Give IV hydrocortisone if the diagnosis of UC is likely; if infective colitis is suspected, consider withholding steroids until the M,C,&S results are back.
- Blood transfusion if anaemic.
- Surgery if peritonitis or free gas on CXR.

Definitive medical management

Acute ulcerative colitis

- IV hydrocortisone 100mg qds, converted to PO prednisolone if responding to treatment.
- IV ciclosporin if fulminant/not responding to IV steroids (discuss with Gastroenterologists).
- Regular blood investigations and plain abdominal radiography to monitor treatment.

Infective colitis

- IV or PO metronidazole until organism identified.
- Salmonella, C. difficile—metronidazole.

Neutropenic colitis

- IV antibiotics.
- Bone marrow support.

Patients on high-dose steroids may have a dampened inflammatory response that masks the severity of their acute colitis. Therefore, have a low threshold for considering surgery in these patients.

Definitive surgical management

Surgery is indicated for the following clinical situations:

- Failure to respond to maximal medical therapy.
- Toxic dilatation of the colon.
- Suspicion or evidence of perforation.
- Severe haemorrhage.
- Recurrent attacks of severe colitis.

The operation of choice for any acute severe colitis is usually a subtotal colectomy and end ileostomy formation. The rectum is preserved, and either closed as a rectal stump or brought out as a mucous fistula.

Acute anorectal pain

Causes and features

- Fissure-in-ano. Acute severe, localized, 'knife-like' pain in the anus during defecation. Often associated with deep throbbing pain for minutes or hours afterwards due to pelvic floor spasm. Blood on the paper when wiping (small volume, red-pink streaks or spots).
- Haemorrhoids. Rarely painful, except when they are external and/or acutely thrombosed. Usual symptoms are pain, itching, and bright red bleeding after defecation. Profuse bleeding is rare, but possible in patients with therapeutic or pathological coagulopathy.
- Perianal abscess. Gradual onset, constant localized perianal pain. Associated fluctuant swelling with tenderness and possible discharge. May have associated systemic features of fever, malaise, and anorexia.
- Perianal haematoma. Also known as external thrombosed pile—usually sudden onset; acutely painful with associated perianal purple swelling. It is self-limiting.
- Rectal prolapse. Acute full-thickness rectal prolapse, occasionally causes pain. Obvious large perineal lump with visible dark red/blue mucosa with surface mucus and occasionally some mucosal ulceration.

Emergency management

Establish a diagnosis

- Good inspection and careful DRE are usually all that is required.
- Rigid sigmoidoscopy may be painful and is often unnecessary.
- Flexible sigmoidoscopy is rarely indicated.
- Remember these conditions are painful and often definitive diagnosis occurs during the treatment under GA.

Early treatment

- Give adequate analgesia; opiates may be necessary.
- Topical treatment is highly effective; cool pads, topical LA gels.

Definitive management

- Fissure-in-ano. Mainstay of acute treatment is analgesia and anal sphincter muscle relaxants, e.g. topical GTN 0.4% ointment, diltiazem 2% ointment. LA gels are helpful early in treatment. If creams fail, botulinum toxin injection into internal sphincter under GA paralyses this muscle for 3 months to effect healing.
- Haemorrhoids. If thrombosed, may require bed rest, with continued topical treatment until swelling resolves and spontaneous reduction begins. Acute haemorrhoidectomy is almost always best avoided, due to the risk of over-excision of anal tissue.
- Perianal abscess. Incision and drainage is a surgical emergency, particularly if the patient is diabetic or immunosuppressed.
- Perianal haematoma. Incision to allow decompression of acute haematoma may be necessary if patient wishes, often done under topical LA.
- Rectal prolapse. Swelling may be reduced by cool packs, elevation, and sometimes icing sugar applied to the swollen mucosa as a dessicant! Very rarely requires emergency surgery for ischaemia.

Acute rectal bleeding

Causes and features

Acute rectal bleeding is broadly divided into regions of the colon from which it comes and the blood is typically different according to the origin.

Anorectal

Bright red blood, on the surface of the stool and paper, after defecation.

- Haemorrhoids.
- Acute anal fissure.
- Distal proctitis.
- Rectal prolapse.

Rectosigmoid

Darker red blood, with clots, on surface of stool and mixed.

- Rectal tumours (benign or malignant).
- Proctocolitis.
- Diverticular disease.

Proximal colonic

Dark red blood mixed into stool or altered blood.

- Colonic tumours (benign or malignant).
- Colitis.
- Angiodysplasia.
- NSAID-induced ulceration.

Upper GI bleeding occasionally produces dark red rectal bleeding, but it is usually associated with significant haemodynamic instability when sufficiently large. More typically, it presents with distinctive smelling melaena.

Features (signs)

- Tachycardia and hypotension suggest substantial blood loss with shock.
- LIF tenderness suggests diverticular inflammation with bleeding.

Emergency management

Resuscitation

- Establish large-calibre IV access; give crystalloid fluid up to 1000mL if tachycardic or hypotensive.
- Catheterize and place on a fluid balance chart if hypotensive or severe bleeding suspected.
- Send bloods for FBC (Hb, WCC, platelets), U&Es (Na+, K+), LFTs (albumin), group and save, clotting, and CRP.

Determine urgency and Establish a diagnosis

- First determine if the patient is stable or unstable:
  - if haemodynamically unstable (recent guidelines[1] define this as shock index (SI = HR/SBP) >1, proceed to CT angiography to localize lesion +/- interventional radiology for endovascular intervention
  - if stable and deemed high risk for further bleeding (↑Age, previous LGI bleeding, ↓Hb and haemodynamic parameters)* – admit for inpatient investigation
- If stable and low risk*, outpatient investigation is considered safe.

*The "Oakland" score[1] is a recent scoring system which can be used as a guidelines for the risk of rebleeding.

- Rigid proctosigmoidoscopy should be performed in all cases to exclude a simple anorectal cause.
- Urgent flexible sigmoidoscopy and colonoscopy may be undertaken, though it is a difficult examination and requires bowel prep beforehand. It may confirm an origin and may allow therapeutic intervention (polypectomy, adrenaline injection, heater probe coagulation, APC).
- Urgent selective mesenteric arteriography for severe or persistent bleeding; needs active bleeding of at least 0.5mL/min.
- Urgent gastroscopy should be used to exclude massive upper GI bleeding if suspected in haemodynamically unstable patients.

Definitive management

Anorectal causes

- Most can be controlled by local measures such as phenol injection, banding of piles, suturing, or packing.

Acute colitis

- IV or PO metronidazole if thought to be infective until organism identified.
- IV hydrocortisone 100mg qds if thought to be UC or Crohn's colitis.
- Surgery may be necessary, whatever the aetiology, if bleeding persists (subtotal colectomy and ileostomy formation).

Diverticular disease

- IV antibiotics (cefuroxime 750mg tds + metronidazole 500mg tds).
- Angiographic embolization if bleeding fails to stop and patient not critically unstable for time in radiology.
- Surgery is high risk but may be unavoidable. If the location is known, a directed hemicolectomy may be performed (on-table colonoscopy may be used). If not, a subtotal colectomy is safest.

Angiodysplasia

- Colonoscopic therapy (injection, heater probe, APC) is ideal.
- Angiographic embolization may be possible.
- Right hemicolectomy is occasionally unavoidable.

Undiagnosed source

In case of ongoing bleeding in spite of the usual investigations, further options include: repeat CTA, capsule endoscopy and nuclear medicine studies. Rarely, the patient remains unstable with active bleeding and no cause can be reliably confirmed. Surgical options to deal with this include:

- On-table colonoscopy with washout via colostomy to locate bleeding source.
- Formation of mid-transverse loop colostomy and subsequent targeted hemicolectomy.
- 'Blind' (hemi)colectomy (left if significant diverticular disease present; right if no other cause obvious and angiodysplasia is likely).

Reference

Oakland K., Chadwick G., East JE et al. Diagnosis and management of acute lower gastrointestinal bleeding: guidelines from the British Society of Gastroenterology. *Gut* 2019;68:776-789.

Acute breast pain

Causes and features

Breast origin

- Breast abscess. Acute severe, localized pain in the breast, associated with swelling, redness, and sometimes purulent nipple discharge. Commonest in breastfeeding women. May be due to chronic mastitis/mammary duct ectasia, occasionally recurrent.
- Mastitis. Recurrent intermittent breast pain with swelling, tenderness, and seropurulent nipple discharge. Commonest in smokers; associated with mammary duct ectasia.
- Fibrocystic disease (➲ Benign breast disease, p. 322). Usually recurrent or chronic breast pain, but may be acute isolated episode. Often multifocal and associated with tender, vague swelling or 'lumpiness'.

Non-breast origin

- Musculoskeletal. Often onset after exercise, coughing, or straining, but not always. No associated breast symptoms. Pain usually sharp and precipitated by movement or breathing. Often tender deep to breast tissue and over other chest wall areas, e.g. costochondral junctions in costochondritis (Tietze's disease). May be due to pleural disease—post-pneumonic, post-PE, viral pleurodynia (Bornholm's disease).
- Visceral. May be due to atypical angina or acute coronary syndrome.
- Skin pathology. Such as infected sebaceous cysts, cellulitis, and skin abscess.

Emergency management

Establish a diagnosis

- Good inspection and careful history taking are usually all that is required.
- Imaging is rarely necessary and is often painful if the pathology is 1° breast. Mammography should be avoided due to the breast compression required. Breast ultrasound may help, particularly in diagnosis and targeted aspiration of breast abscess.
- Consider specialist referral or opinion if PE, cardiac ischaemia, or pneumonia suspected. CXR is simple, but often unhelpful.

Early treatment

- Give adequate analgesia. NSAIDs—diclofenac 50mg PO or 100mg PR—are effective in most causes. Opiates may be necessary.
- Breast abscesses may be effectively aspirated for relief of pressure symptoms under LA. Formal incision and drainage is often avoided, especially in lactational abscesses.

Definitive management

- ***Breast abscess***. If lactational, PO antibiotics (including flucloxacillin 500mg tds) and aspirational drainage (often repeated several times on a daily or alternate-day basis). If associated with chronic mastitis, PO antibiotics (to include metronidazole 400mg tds PO or co-amoxiclav 750mg tds PO).
- ***Fibrocystic disease***. NSAIDs (e.g. ibuprofen 400mg as required), G-linoleic acid, danazol, and occasionally tamoxifen.

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