General Medicine & Surgery

medical student revision guide

- everything you need to prepare for exams
- diagrams, flowcharts, colour coding and summary boxes
- includes all core topics for medical finals & foundation years
- peer-reviewed with the latest guidelines

Rebecca Richardson
& Ricky Ellis

- Cardiology
- Endocrinology
- Gastroenterology
- Hepato-pancreato-biliary
- Haematology
- Immunology & allergy
- Neurology
- Renal
- Respiratory
- Surgical principles
- The acute abdomen
- Gastrointestinal surgery
- The breast
- Vascular surgery
- Urology
- Critical illness
- Emergency presentations
- Rheumatology
- Trauma & orthopaedics

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"I like the unique style ... It is simple, engaging and easy to read, whatever your learning preferences (I particularly like the colour coding!). It also covers topics comprehensively, making it not only a great aide-memoire but also a very useful everyday book on the ward."

From the Foreword by
Professor Farah Bhatti OBE
Consultant Cardiothoracic Surgeon

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Thank you for your help.

General Medicine & Surgery

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Foreword

A book such as this takes time, effort and dedication from all the contributors who do this alongside their already busy clinical work. It is heartening to see this commitment to supporting the education and advancement of the next generation of doctors and allied health professionals.

I like the unique style which is based on Dr Richardson's personal notes whilst a medical student. It is simple, engaging and easy to read, whatever your learning preferences (I particularly like the colour coding!) It also covers topics comprehensively, making it not only a great aide-memoire but also a very useful everyday book on the ward. The judicious use of references (e.g. NICE guidelines and BMJ Best Practice) also enables readers to look into subjects in depth if needed, without affecting the flow of the text.

Once again, I commend the authors on their remarkable efforts and wish everyone who picks up this book well in their careers.

Professor Farah Bhatti OBE
Consultant Cardiothoracic Surgeon

Preface

I remember it well. Countless evenings sat at my desk, desperately trying to work out what I needed to know, and how I was going to know it. Hours spent trudging through textbooks, soaking up line after line of information, only to be left certain that after a week on my new placement, it would all be forgotten. **Medical school is tough**. The vast volume of knowledge that must be acquired and retained, to achieve the standards expected of a safe and successful doctor, is a daunting task. But as you will find at every stage in your medical career, **help is always available if you know where to look**. For you, I hope this book can be your help. I present it as your trusted companion, a loyal friend that will stand by your side through good times and bad, something to turn to when you need **guidance, reassurance, or simply just a place to start!**

There is no end of resources available for medical students, all designed to make the journey from student to doctor that little bit easier. For me, I found this wealth of information to be just as much of a curse as it was a blessing. Not only was I faced with the overwhelming task of learning it all, but I first had to find and dissect the parts that were relevant to me. **Wouldn't it be easier if everything I needed to know was in the same place?** The simple answer – yes. And that is how I started to write this book.

My first goal when creating the *Medical Student Revision Guides* was to bring together **all the key topics needed for medical school exams and life as a junior doctor** in one readily available place. I did this through summarising a variety of recognised resources, including textbooks, articles and clinical guidelines. This has been **supplemented by the expert knowledge of specialists** in each field, who have reviewed each chapter to ensure it is accurate and reflects the most up-to-date guidance.

My second goal was to do everything I could to help you remember this information. I appreciate everyone is unique in the way they learn, but with most of us relying on **vision as our dominant sense**, it seemed illogical not to utilise its power. As such, I have specifically designed this book with an **extensive use of colour, diagrams and summary tables**, to create a resource that is visually striking and a refreshing change from your current textbooks. The informal 'notes-style' layout and dedicated column that allows for your own annotations on each page, are features that I hope make the content feel **more accessible and easier to engage with**.

It is my sincere hope that you find this book useful, whether it be as your comprehensive revision resource, or a quick reminder of a condition that you come across on the ward. **I am forever grateful for any feedback** that can help me to better help you, so please do leave a review with your honest thoughts.

I wish you luck with your exams, and all the best for your future careers.

Rebecca Richardson

Disclaimer

It is important to note that this book is designed as a revision tool and aide-memoire. It is not intended to give an in-depth understanding of each condition, but rather to focus on the key points that often appear in undergraduate exams. It should not be solely relied upon in clinical situations; please always check the most current and local guidelines before implementing management or administering any treatment.

Every attempt has been made to ensure that the most up-to-date information has been included at the time of writing this book. However, due to the continuously evolving nature of the medical profession, and with variations in clinical practice between hospital Trusts, this cannot be guaranteed. It is therefore advised that you correlate these notes to other resources, and supplement them with your own clinical encounters, to ensure a complete learning experience. Readers should also ensure that they learn all elements of their own medical school curriculum, regardless of whether they are covered in this book.

Acknowledgements

I must firstly say a huge thank you to the team at Scion Publishing for making this book a reality. A special mention to Jonathan and Clare, for your tireless work overcoming layout issues and design problems, to ensure the end product was everything I had hoped it would be.

Secondly, I would like to thank my co-author, Ricky. Your advice and guidance have been invaluable in this process. Your wealth of experience in the field of medical education has undoubtedly benefited the book and its readers, and remains an inspiration for my future work.

My thanks are extended to all those who have contributed time and expertise as a chapter reviewer. I will be eternally grateful that you could see the potential in my work in its early stages and gave up your own time to help me achieve it. The abundance of knowledge and experience you have brought is priceless and will underpin the learning of many future doctors.

Finally, to my loved ones. To Mum and Dad, for the years of selflessness and sacrifice that allowed me the opportunities to achieve all that I have today. To my brother, Chris, whose artistic talent never fails to amaze me, and has inspired the covers and diagrams of both my books. To Nana, Gran, Grandad and Margaret, for the timeless wisdom and unconditional love that only grandparents can provide. And to Martin, for your endless patience, support and encouragement in all that I do.

Thank you.

About the authors

Rebecca Richardson is a junior doctor currently working in the East Midlands. After graduating from the University of Nottingham as one of the top students in her year, with first class honours, she became passionate about helping others to follow in her footsteps. Her revision notes have already helped hundreds of medical students across the UK prepare for their exams. Rebecca has continued her mission to support students and trainees through creating content for a variety of medical education platforms, as well as running regular virtual teaching sessions to help students practise for their clinical examinations.

Ricky Ellis is a Urology Specialist Registrar, Honorary Clinical Senior Lecturer and Research Associate for the Intercollegiate Committee for Basic Surgical Examinations (ICBSE). Ricky is an award-winning trainer and regularly organises teaching courses including the internationally delivered 'Urology Boot Camp for Medical Students'. He is passionate about improving training for medical students and junior doctors and was awarded the prestigious Association of Surgeons in Training and Faculty of Surgical Trainers Silver Suture Award for contributions to and excellence in surgical training.

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General abbreviations

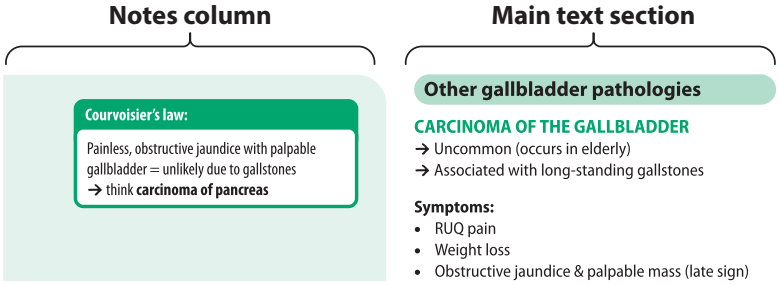
Each chapter begins with a set of abbreviations specific for that chapter.

2ww – 2 week wait
a. – Arterial
ABCDE – Airways, Breathing, Circulation, Disability, Exposure
ABG – Arterial blood gas
ABX – Antibiotics
AXR – Abdominal X-ray
BB – Beta-blocker
BD – Twice a day
BG – Blood glucose
BMI – Body mass index
BP – Blood pressure
ca – Cancer
Ca – Calcium
CBT – Cognitive behavioural therapy
CCB – Calcium channel blocker
CCF – Congestive cardiac failure
CF – Cystic fibrosis
CHF – Chronic heart failure
CI – Contraindication
CK – Creatine kinase
CKD – Chronic kidney disease
CMV – Cytomegalovirus
CN – Cranial nerve
CNS – Central nervous system
CPAP – Continuous positive airway pressure
CPR – Cardiopulmonary resuscitation
Cr – Creatinine
CRP – C-reactive protein
CRT – Capillary refill time
CSF – Cerebrospinal fluid
CT – Computed tomography
CV – Cardiovascular
CVA – Cerebrovascular accident
CVD – Cardiovascular disease
CXR – Chest X-ray
D&V – Diarrhoea and vomiting
d – Day
DBP – Diastolic blood pressure
DDx – Differential diagnosis
DM – Diabetes mellitus
DOAC – Direct oral anticoagulant
DRE – Digital rectal examination
DVLA – Driver and Vehicle Licensing Agency
Dx – Diagnosis
ECG – Electrocardiogram
ECT – Electroconvulsive therapy
EEG – Electroencephalogram
eGFR – Estimated GFR
ENT – Ear, nose and throat
EPO – Erythropoeitin
ESR – Erythrocyte sedimentation rate
FBC – Full blood count
FHx – Family history
G&S – Group & save
GA – General anaesthetic
GCS – Glasgow Coma Score/Scale
GFR – Glomerular filtration rate
GI – Gastrointestinal
GP – General practitioner
h – Hour
Hb – Haemoglobin
HF – Heart failure
HIV – Human immunodeficiency virus
HR – Heart rate
HTN – Hypertension
ICP – Intracranial pressure
Ig – Immunoglobulin
IHD – Ischaemic heart disease
IM – Intramuscular
inc. – Including
IV – Intravenous
IVDU – Intravenous drug user
Ix – Investigation
JVP – Jugular venous pressure
LFT – Liver function test
LHS – Left-hand side
LMWH – Low molecular weight heparin
LOC – Loss of consciousness
m – Month
mcg – Microgram
MCS – Microscopy, culture and sensitivity
MDT – Multidisciplinary team
MI – Myocardial infarction
min – Minute
MND – Motor neurone disease
MRI – Magnetic resonance imaging
Mx – Management
NBM – Nil by mouth
NSAID – Non-steroidal anti-inflammatory drug
N&V – Nausea & vomiting
OCD – Obsessive–compulsive disorder
OD – Once a day
O/E – On examination
OT – Occupational therapist
PCR – Polymerase chain reaction
PE – Pulmonary embolism
PET – Positron emission tomography
PHx – Past history
PNS – Peripheral nervous system
PO – (per ora) Orally
PR – Per rectum
PRN – Pro re nata (as required)
pulm. – Pulmonary
QDS – Four times a day
RA – Rheumatoid arthritis
RBC – Red blood cell
RFT – Renal function test
RHS – Right-hand side
r/o – Risk of
R/V – Review
re – Regarding
RF – Risk factor
RR – Respiration rate
s – Second
SALT – Speech and language therapy
SBP – Systolic blood pressure
SC – Subcutaneous
SD – Standard deviation
SE – Side-effect
SL – Sublingual
SLE – Systemic lupus erythematosus
SOB – Shortness of breath
SSRI – Selective serotonin reuptake inhibitor
Sx – Symptoms
TB – Tuberculosis
TCA – Tricyclic antidepressant
TDS – Three times a day
TFT – Thyroid function test
TIA – Transient ischaemic attack
TNM – Tumour, nodes, metastases
Tx – Treatment
U&Es – Urea & electrolytes
USS – Ultrasound scan
UTI – Urinary tract infection
VBG – Venous blood gas
vit – Vitamin
VTE – Venous thromboembolism
W – Week
WBC – White blood cell
WCC – White cell count
WHO – World Health Organization
y – Year

How to use this book

The underlying principle of this book is to present information in a way that is **eye-catching, clear and easy to remember.** This page will explain some of the **key layout features** that have been used to achieve this.

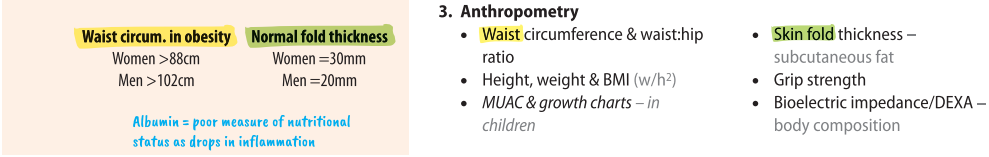
1. Notes column – each page is divided into a main text section, and a tinted notes column. The notes column is used to house additional information, and to provide space for your own notes, should you wish to make any.



2. Text colour

- chapter coloured text – used to expand on a point / provide extra information
- grey text – used for less important information
- red text – used for red flags and emergency points
- blue text – used for extra points / annotations

3. Highlighting – words/phrases that have been highlighted are linked to extra information. Look for another highlight of the same colour on the page to find this information. *The below example uses green highlighting to link 'Skin fold' to the additional information 'Normal fold thickness'.*



4. Box colours – a variety of different boxes are used to display information.

- **Yellow tinted box** – for triggers / risk factors, complications & prognosis
- **Blue tinted box** – for differential diagnoses
- **Red box** – for red flags / emergency points
- **Chapter coloured box** – for all other types of information

Risk factors for carcinoma:
→ **Cirrhosis** (NAFLD, chronic hepatitis)
→ *Aspergillus*
→ Metabolic liver condition
→ Anabolic steroids
→ Parasites

Yellow-tinted

DDx microcytic anaemia: TAILS
- Thalassaemia (α or β-thalassaemia trait)
- Anaemia of chronic disease e.g. renal failure
- Iron-deficiency anaemia
- Lead poisoning
- Sideroblastic anaemia

Blue-tinted

Inflammatory breast cancer
= rare & aggressive subtype
Sx: sudden, red/swollen breast, peau d'orange
→ **no lump**
→ *early axillary spread*
Mx: neoadjuvant chemo, then surgery
DDx: *infection*

Red

Acute coronary syndrome

→ **STEMI, NSTEMI & unstable angina** (not stable angina)

Key differentials:
- Coronary spasm
- Pericarditis/ myocarditis
- Aortic dissection
- MSK (costochondritis)
- PE
- Pneumothorax
- GORD
- PUD

Risk factors
- Smoking
- Hypertension
- Alcohol
- Diabetes mellitus
- FHx
- Previous IHD

BEWARE ATYPICAL PRESENTATION: 'silent MI'
= ACS with no chest pain
Instead: syncope / pulmonary oedema / epigastric pain → in elderly/diabetics/females

Serial troponin: shows continued rise at 6h, **peak at 24h**

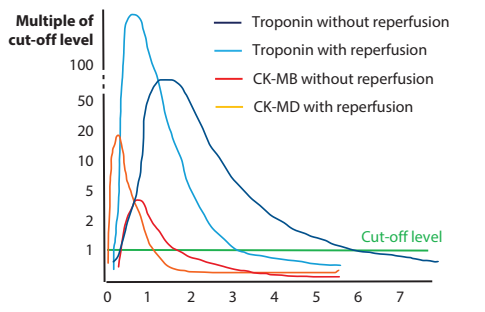


Fig. 1.1

STEMI: complete occlusion
= ST elevation **AND** ↑ troponin
NSTEMI: partial occlusion
= ST depression / T wave inversion **OR** normal ECG **AND** ↑ troponin
UA: partial occlusion
= ST depression/ T wave inversion **WITHOUT** ↑ troponin

ST-elevation criteria:

>1 mm in ≥2 contiguous* **limb leads**
OR
>2mm in ≥2 contiguous* **chest leads**
+ **ST depression in reciprocal leads**

Posterior STEMI:

ST depression in ≥2 chest leads **V1–4**
AND **≥0.5mm** ST elevation in any **V7–9**

*contiguous = represent same territory

Pathogenesis

1. **Atherosclerosis:** fatty deposition in artery wall causes plaque build-up
2. **ACS when:** Plaque rupture → Thrombosis → Vasoconstriction → Ischaemia ± distal thromboembolism

Results in a transmural or subendocardial infarct (circumferential infarct if global hypoperfusion)

Clinical presentation

→ **Severe, *'crushing'* chest pain** (at rest)
- retrosternal, radiating to arm/neck/jaw
- **lasts >20min & NOT RELIEVED by 3× GTN sprays***

*Angina is <20min / relievable

→ **Sympathetic symptoms:** sweating, palpitations, dyspnoea, nausea
- sense of 'impending doom'

On examination

- **Sympathetic Sx:** pallor, sweating, tachycardia
- **Myocardial impairment:** hypotension, ↑ JVP, basal creps, 3rd heart sound, pulmonary/peripheral oedema

Investigations

1. **Bloods:** ↑ lipids, ↓ glucose, clotting
2. **Serial troponin:** on admission + 6–12h later →
3. **ECG:** within 10min of presentation
4. **Other:** CXR, ECHO, angiogram

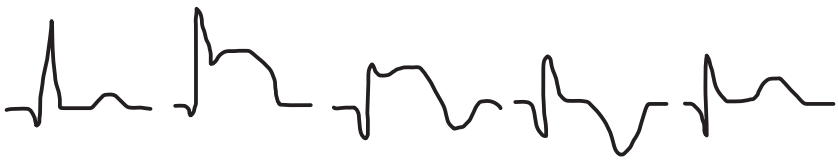
(r/o DDx, assess prognosis & determine best Tx)

Other causes ↑ troponin:
- myocarditis/pericarditis
- **post-PCI**
- ventricular strain / HF
- burns/sepsis
- **PE**
- renal failure

But these show consistently high levels

STEMI ECG CHANGES:

New LBBB + chest pain = assumed STEMI



At onset of pain	Within 1h	Following Tx	24h later	Days/months
normal sinus complex	ST elevation	T-wave inversion Deep Q waves	ST normalised T-wave inversion	deep Q-wave (tissue death)

Fig. 1.2

Widespread concave ST elevation = pericarditis

NSTEMI ECG CHANGES (IF PRESENT):
≥0.5mm ST depression in ≥2 contiguous* **leads**
AND **>1mm T depression** in leads with +ve QRS

Takotsubo: 'broken heart syndrome' (stress cardiomyopathy):
appears with Sx & ECG changes of STEMI but normal angiogram

Management[1]

	NSTEMI/UA	STEMI
Acute	**M**orphine + **m**etoclopramide (5–10mg) **O**xygen (if sats <90%) **N**itrates (GTN spray) **A**ntiplatelets (300mg chewable aspirin + loading dose ticagrelor/clopidogrel/prasugrel) **A**nticoagulant (fondaparinux 2.5mg for 72h)	
	1. **GRACE score:** assess inpatient & 6m mortality 2. **DAPT score:** predicts who will benefit from dual antiplatelet therapy 3. **Angiographic assessment** 4. **Revascularisation options:** PCI, CABG	First line: **primary PCI** ideally if <120min from onset (95% success) **Target 150min call → balloon time** Second line: **thrombolysis** (rarely used as many contraindications)
Long-term	**A** 1. **Lifelong Aspirin:** 75mg OD **A** 2. **Continue 2nd Antiplatelet:** ticagrelor/clopidogrel/prasugrel (for 1y) **BB** 3. **Lifelong Beta-Blocker:** bisoprolol (HR at 60bpm) **A** 4. **Lifelong Atorvastatin** **A** 5. **ACEi:** ramipril 6. **Treat underlying problems:** HF/HTN (BP<140/90)	
Rehab	***Optimise physical, psychological & social functioning + stabilise/slow atherosclerosis*** **Patient education:** about risk factors for CVD **Exercise programme:** gradual return **Return to work:** usually after 2w (depends on job)	

Remember acute management as '*MONA(A)*'

RETURN TO DRIVING:
Angina: if Sx controlled
PCI: 1w
CABG: 4w
ICD: 6m

GRACE score parameters to predict inpatient & 6m mortality

1. Age
2. HR
3. SBP
4. Creatinine
5. Any CHF
6. Cardiac arrest at admission
7. ST segment changes
8. Elevated cardiac markers

Although GRACE score has good predictive value of mortality, it is often not used in practice.

Local logistics determines speed of cardiac catheterisation. STEMI have primary PCI immediately on arrival, NSTEMI and unstable angina have inpatient cardiac catheterisation as soon as feasible, prior to discharge.

NSTEMI: dual antiplatelet therapy
Aspirin + ticagrelor/clopidogrel/prasugrel
→ consider lower dose if high bleeding risk (HASBLED score)
→ consider stopping if going for CABG revascularisation
→ consider GI protection with PPI if >65y or Hx of GORD/PUD

Complications of ACS

D	**Death**	
A	**Arrhythmia**	AF, VF, VT, bradyarrhythmias (VF = most common complication)
R	**Rupture**	Of papillary muscles / ventricular wall / septum (causes MR, VSD & LVF)
T	**Tamponade**	Due to ventricular wall rupture = Beck's triad
H	**Heart failure**	May be due to VSD = **cardiogenic shock**
V	**Valve problem**	Due to papillary muscle dysfunction/rupture = **new murmur**
A	**Aneurysm of ventricle**	Due to weakened ventricular wall
D	**Dressler's syndrome**	Immune-mediated pericarditis (usually 2–3w later)
E	**Emboli**	From thrombus forming over damaged left ventricle wall
R	**Recurrence**	

Beck's triad:
1. ↑JVP
2. ↓BP
3. Quiet heart sounds

Dressler's syndrome:
1. Fever
2. Chest pain
3. Exertional dyspnoea
4. Pericardial effusion

[1] NICE (2020) *Acute coronary syndromes* [NG185]

Pituitary disorders

The pituitary gland sits below the hypothalamus & optic chiasm

Pituitary adenoma: BENIGN

1. **Microadenoma** <1cm
2. **Macroadenoma** >1cm = mass effects
   - **Functioning:** secrete hormones (70–80%)
   - **Non-functioning:** not secretory (20–30%)

Fig. 2.3 Visual fields in bitemporal hemianopia.

(GH-RH) Growth hormone-releasing hormone
(TRH) Thyrotropin-releasing hormone
(CRH) Corticotropin-releasing hormone
(GnRH) Gonadotropin-releasing hormone
(SRIF) Somatotropin release inhibiting factor / somatostatin inhibitor
(DA) Dopamine

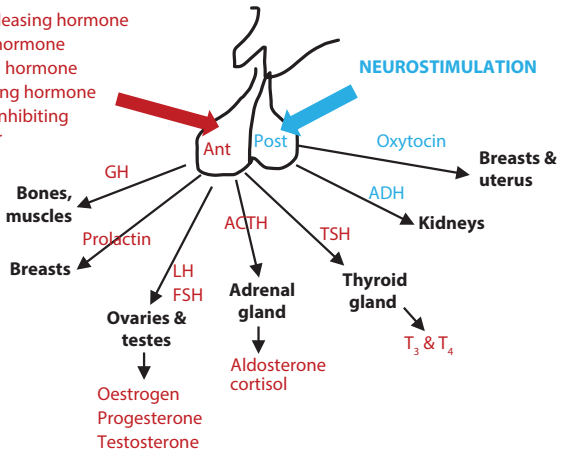


Fig. 2.4 Functions of the pituitary gland.

Mass effects of pituitary adenoma:

- Bitemporal hemianopia = from pressure on **optic chiasm**
- Ocular palsies = from pressure on **cavernous sinus** (CN 3,4,6)
- Headache from ↑ ICP
- Altered appetite/thirst/sleep = from pressure on **hypothalamus**
- **Hypopituitarism** from destruction of functional tissue
- **Hyperprolactinaemia** from stalk effect

'The stalk effect'

→ **Dopamine** released from hypothalamus reaches pituitary gland **via pituitary stalk**
→ Normally dopamine **INHIBITS** prolactin
→ **Damaged/compressed stalk** = ↓ DA = ↓ inhibition = **HYPERPROLACTINAEMIA**

→ *Secretory prolactinoma **plus** compression effects = MASSIVE PROLACTIN ↑ (>10,000)*

Hypopituitarism

Hypopituitarism:

- Sx = vague & non-specific
- General Mx = HORMONE REPLACEMENT
- Ix & Tx of specific cause

Typical order of hormone loss ↓

Hormone lacking	Clinical effects	Management
GH	**Children:** short stature **Adults:** no Sx / abnormal body composition / fatigue	GH analogue
LH/FSH (↓ sex hormones)	**Children:** delayed puberty **Adults:** no Sx / hypogonadism / amenorrhoea	Testosterone (males) or HRT (females)
TSH (↓ T_3/T_4)	Hypothyroidism (cold, weight loss, tired)	Thyroxine
ACTH (↓ cortisol)	Lethargy, weight loss, ↓ glucose, ↓ Na	Hydrocortisone/prednisolone
PROLACTIN	Failed lactation	–
ADH	Diabetes insipidus (polyuria/polydipsia)	Desmopressin

DDx hypopituitarism:

Pituitary	Hypothalamic
• **Non-functioning adenoma** • Metastatic tumour • Surgical resection of gland • Apoplexy (vascular insult)	• Craniopharyngioma • Infarction • Infection • Sarcoid

INVESTIGATIONS:

1. **History & examination:** including visual fields & cranial nerves
2. **Pituitary hormones:**
   - All except prolactin: LOW/normal
   - PROLACTIN
     → 500–1000: (micro-prolactinoma) **or** (non-functioning tumour compressing stalk) **or** (other cause of ↑ prolactin*) → Renal impairment, Antipsychotics, Pregnancy/OCP
     → >1000: (micro-prolactinoma **AND** stalk compression) **or** (macroprolactinoma)
3. **Effector gland hormones:** LOW (T_3/T_4, FSH*, IGF-1, 9am cortisol)
   *FSH should be high if post-menopausal as ↓ oestradiol (no more follicles)

4. **Stimulation tests:** LOW / no response
   - **For ACTH deficiency:** insulin stress test (IST)
   - **For acromegaly:** OGTT → GH suppression
   - **For non-pituitary cause of adrenal disease:** Synacthen test
5. **Imaging:** CT/MRI – determine pathology

When given insulin, blood glucose drops → hypoglycaemia triggers ACTH release – no ↑ ACTH suggests deficiency

When given glucose in OGTT, GH should be suppressed
No suppression suggests acromegaly (check IGF-1 levels)

Secretory/functional adenomas

→ *can produce >1 hormone*

Type of adenoma	Hormonal effect	Symptoms	Treatment
Prolactinoma 35% (F:M = 5:1)	HYPERPROLACTINAEMIA ↑ basal prolactin	• Galactorrhoea (+ female amenorrhoea) • ↓ Fertility, ↓ libido, hypogonadism • **Complications:** osteoporosis	DA agonist* • carbergoline • bromocriptine *SE: pulmonary fibrosis (need CXR)
GH-secreting 20%	ACROMEGALY/GIGANTISM ↑ IGF-1 & glucose	• ↑ **Size** hands/feet slow onset • Coarse facies, wide-spaced teeth • Vision loss • Tired, **headache**, sweaty, ↓ libido • **Complications:** DM, HTN, OSA	1. Surgery: transsphenoidal 2. Radiotherapy 3. Medical • DA agonist • somatostatin analogue
ACTH-secreting 10%	CUSHING DISEASE ↑ cortisol	• ↑ Weight, DM, ↓ libido • Hair, striae, hump	
TSH-secreting rare	HYPERTHYROIDISM ↑ $T_{3/4}$	• ↓ Weight, fatigue • Hot, sweaty, palpitations	

Raised TSH & T4 usually indicates poor compliance with LT4 treatment

LH-/FSH-secreting adenoma = VERY RARE

Oesophageal cancer

→ **UK prevalence:** 14 in 100,000 (increasing due to RFs)
→ **M:F = 2:1**

Squamous cell (20%): upper 2/3 oesophagus
RF = **smoking, alcohol,** Asian, achalasia

Adenocarcinoma (80%): lower 1/3 oesophagus
RF = **smoking, alcohol,** obesity, Barrett's oesophagus (GORD)

Presents late & POOR PROGNOSIS[4]

Stage	5y Survival
1	53%
2	30%
3	16%
4	0%
Overall	17%

Symptoms

- **Progressive dysphagia:** solids → liquids → saliva
- Weight loss & anorexia
- Retrosternal chest pain
- Lymphadenopathy
- ± cough, aspiration, hoarseness

Investigations

- **OGD & biopsy** – **histological grading**
- **CT chest, abdo, pelvis** – **TNM staging**
- Endoscopic USS – more detailed T&N staging
- PET scan – detects metabolically active mets

TNM staging

Tumour	Nodes	Metastases
T_{is} – tumour *in situ* T_{1a} – invades lamina propria T_{1b} – invades submucosa T_2 – invades muscularis propria T_3 – invades adventitia T_4 – invades adjacent tissues	N_0 none N_1 1–2 LNs N_2 3–6 LNs N_3 7+ LNs	M_0 no mets M_1 distant mets

Management[5]

Management requires an MDT approach

Adenocarcinoma	
T_{is} or T_{1a} (N_0, M_0)	Endoscopic mucosal resection/dissection (EMR/EMD)
T_{1b} (N_0, M_0) **or >75yrs**	Surgical resection **or** definitive chemoradiotherapy
T_2, T_3, T_4 (M_0)	Neoadjuvant chemo → surgery + adjuvant chemo
Squamous cell carcinoma	
T_{1a} (N_0)	EMR/EMD
All others (M_0)	Neoadjuvant chemo → surgery + adjuvant chemo
Any histology with mets (M_1) = palliative care	
1. Chemo ± radiotherapy 2. Symptom relief: stents, analgesia	

Complications of upper GI surgery:

- **Weight loss** – need dietetic support
- **Dysphagia** – due to strictures
- **Reflux**
- **Delayed emptying**

SURGICAL OPTIONS:

Ivor Lewis oesophagectomy (2 stage → open or keyhole)

Stage 1 (abdominal):
- mobilise / free stomach from blood supply

Stage 2 (thoracic):
- mobilise & resect affected part of oesophagus
- pass 'free' stomach through hiatus & staple to remaining oesophagus
- pylorectomy to improve gastric emptying post-op

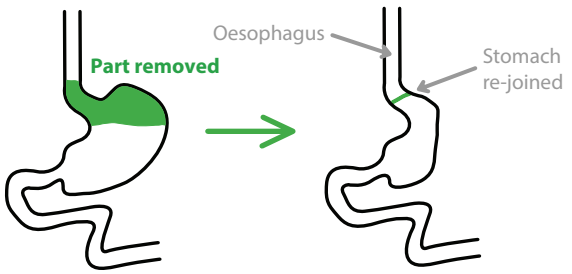


Fig. 3.2 Ivor Lewis oesophagectomy.

NB. Oesophageal surgery = high morbidity & mortality
→ need careful pre-op assessment for suitability

[4] Cancer survival by stage at diagnosis for England, 2019. ONS.
[5] NICE (2018) *Oesophago-gastric cancer* [NG83]

Gastric cancer

*prevalence is increasing globally due to increased prevalence of modifiable RFs

Risk factors:
→ **Internal:** pernicious anaemia, *H. pylori*, polyps
→ **External:** smoking, high salt/nitrate (red meat)
→ **Genetic:** Japanese, HNPCC, Group A blood

Presents late & POOR PROGNOSIS[7]

Stage	5y Survival
1	65%
2	36%
3	24%
4	0%
Overall	20%

→ **UK prevalence*:** 10 in 100,000 → 5th most common cancer worldwide[6]
→ **M:F = 3:1** → **Peak age: 50–70y**

Types

1. **Adenocarcinoma (85%)**
2. **Other (15%)** – lymphoma, leiomyosarcoma, GISTs

Symptoms

- **Epigastric pain / dyspepsia**
- **Early satiety**
- N&V, anorexia, weight loss
- Dysphagia

} often non-specific & mimic PUD

Investigations

- **OGD & multiple ulcer edge biopsies** – **histological grading & location**
- **CT chest, abdo, pelvis** – **TNM staging** (same as oesophageal)
- Endoscopic USS – more detailed T&N staging

Signs O/E

- **Epigastric mass** (50%)
- Virchow's node enlargement
- Hepatomegaly, ascites
- Jaundice, acanthosis nigricans

} only in late stage / metastases

Management[8]

Any histology with mets = palliative care
1. Chemo ± radiotherapy
2. Symptom relief: stents, bypass, analgesia

Adenocarcinoma	
T_{is} or T_{1a} (N_0, M_0)	Endoscopic mucosal resection
T_{1b} (N_0, M_0) or >75y	Direct to surgery
T_2, T_3, T_4 (M_0)	Neoadjuvant chemo → surgery + adjuvant chemo

SURGICAL OPTIONS:

Siewert class	Location	Management
1	1–5cm above GOJ	Oesophagectomy
2	<1cm above or <2cm below GOJ	Oesophagectomy **or** total gastrectomy
3	2–5cm below GOJ	Total gastrectomy
Distal	Near pylorus	Distal gastrectomy

Gastrectomy complications:
- Vit B12 / iron deficiency
- Early satiety / weight loss
- Osteoporosis/osteomalacia

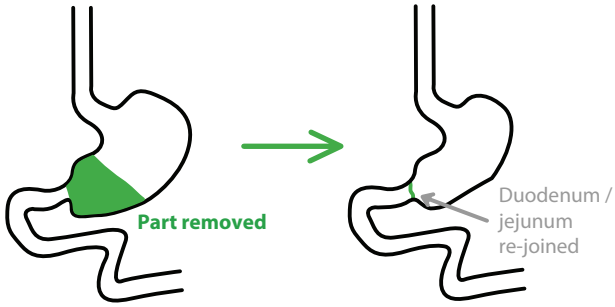


Fig. 3.3 Distal gastrectomy.

Total gastrectomy + Roux-en-Y reconstruction:
1. Resection of stomach
2. Connect proximal jejunum to lower oesophagus
3. Reconnect distal duodenum further down jejunum to allow passage of bile

Distal gastrectomy:
1. Resection of distal part of stomach
2. Various reconstruction options **(Roux-en-Y = better outcome but ↑ risk)**

[6] International Agency for Research on Cancer, WHO 2020
[7] Cancer survival by stage at diagnosis for England, 2019. ONS.
[8] NICE (2018) *Oesophago-gastric cancer* [NG83]

Gallbladder disease

→ Gallbladder **stores** and **concentrates** bile
→ Fatty acids / amino acids in duodenum stimulate **CCK** release
→ **CCK stimulates gallbladder contraction** & bile release

Contents of bile:
- Water (97%)
- Bile salts / pigments
- Cholesterol
- Phospholipids

Investigations

1. **History & abdo exam** + pregnancy test + urine dip
2. **Bloods:** FBC, CRP/ESR, LFTs, clotting (± amylase)
3. **Imaging:**

→ USS (shows GB/duct dilation) → ERCP (Dx & Tx **but invasive**)
→ MRCP (95% sensitive to detect stone) → CT if concerned about tumour

USS = FIRST-LINE imaging

Types of bile stone

1. **CHOLESTEROL** – crystallisation of **excess cholesterol**
2. **PIGMENT** – contain **calcium bilirubinate**
   - **Black:** haemolytic conditions (SCA, malaria)
   - **Brown:** biliary stasis or infection

Risk factors*:

Fat	**F**emale
Fair	**F**ertile
Forty	**F**Hx

*Crohn's & ileal resection = additional RFs as ↓ bile salt absorption

Complications of gallstones

1. **In gallbladder:** biliary colic (cystic duct obstruction), acute cholecystitis, perforation*, carcinoma *needs emergency cholecystectomy
2. **In bile ducts:** biliary colic (common duct obstruction) → ascending cholangitis or acute pancreatitis
3. **In intestine:** gallstone ileus (stone erodes through gallbladder = fistula to duodenum → intestinal obstruction)

AXR shows air in biliary tree

most gallstones = asymptomatic, until complications

Mirizzi's syndrome:
Gallstone impacts in **cystic duct / Hartmann's pouch**
- Extrinsic compression of hepatic duct
- **Obstructive jaundice** without dilation of CBD

Management[4]

OBSTRUCTION → **INFLAMMATION** → **INFECTION**
- *C. perfringens*
- *Klebsiella*
- *E. coli*

BILIARY COLIC
= temporary duct obstruction
Systemically well!

Symptoms:
- **Colicky RUQ / diffuse pain** (visceral)
  - ▸ crescendos (stop if stone moves)
  - ▸ radiates to back/shoulder
  - ▸ worse with food (esp. fatty)
- **± N&V**
- **Obstructive jaundice**

if common duct blocked/Mirizzi's

Investigations:
- **Bloods = normal**
- USS/MRCP (shows dilation/stones)

Management:
- Bed rest, fluid, analgesia, NBM
- Elective **cholecystectomy** (by 6w)

ACUTE CHOLECYSTITIS
= long-term *cystic duct* obstruction causes **inflammation of GB wall**
Systemically unwell!

Symptoms:
Initially: biliary colic

With inflammation: (localised pain)
- Fever & vomiting
- Severe RUQ pain **+ peritonism**

O/E:
Guarding & rigidity + **Murphy's sign**

Investigations:
- **Bloods:** ↑ WCC, CRP, ESR
- USS/MRCP (dilation, thick GB wall)

Management: ABCDE
- IV fluid, analgesia **+ IV ABX**
- Elective **cholecystectomy** (by 1w but ideally within 72h)

ASCENDING CHOLANGITIS
= long-term *common bile duct* obstruction causes **duct infection**

Symptoms:
Charcot's triad:
1. High swinging **fever** (chills + rigors)
2. RUQ pain
3. Jaundice

Reynolds' pentad:
+ 4. Confusion
+ 5. Shock
(ICU admission)

Investigations:
- **Bloods:** ↑ WCC, CRP, ESR
- **LFTs:** obstructive picture

Management: ABCDE + Sepsis 6
- IV fluids, analgesia **+ IV ABX** (e.g. cefuroxime + metronidazole)
- **Emergency ERCP** ± stenting

Key DDx: GORD, PUD, acute pancreatitis, IBD, acute hepatitis, pyelonephritis

Murphy's sign: press over GB – patient has sharp pain during inspiration as peritoneum hits your hand

[4] BMJ Best Practice (2021) *Gallstones, acute cholecystitis, acute cholangitis*

Courvoisier's law:

Painless, obstructive jaundice with palpable gallbladder = unlikely due to gallstones
→ think **carcinoma of pancreas**

Other gallbladder pathologies

CARCINOMA OF THE GALLBLADDER

→ Uncommon (occurs in elderly)
→ Associated with long-standing gallstones

Symptoms:

- RUQ pain
- Weight loss
- Obstructive jaundice & palpable mass (late sign)

Sx occur late (& then mimic chronic cholecystitis)

Management: poor prognosis as late presentation

- Radical cholecystectomy (± liver resection if affected)

CHOLANGIOCARCINOMA (adenocarcinoma of bile ducts / ampulla)

→ Common sites = at ductal confluences
→ Mostly in elderly patients
→ Can be 2° to PSC/IBD

Symptoms:

- Painless progressive jaundice
- Weight loss

Sx occur late & mimic PANCREATIC CANCER

Management: poor prognosis as late presentation

- Whipple's procedure (if operable)
- Palliative stenting (relieves jaundice & helps gastric emptying)

Anaemia overview

Anaemia is defined as Hb <130g/L in men & <115g/L in women

Causes

1. ↓ **RBC production** e.g. IDA (most common), BM disorders, cytotoxic drugs / chemotherapy, CKD, aplastic anaemia
2. ↑ **RBC destruction** e.g. SCD, thalassaemias, G6PD deficiency, autoimmune haemolysis
3. **Blood loss** (rare in children) e.g. vWD, Meckel diverticulum

Signs/symptoms

General signs/symptoms

- Fatigue/weakness
- Pallor (conjunctiva)
- SOB/tachycardia/dizziness

Symptoms occur if Hb <60–70g/L

Specific signs/symptoms

→ **Koilonychia:** IDA
→ **Jaundice:** haemolytic anaemia
→ **Leg ulcers:** sickle cell disease
→ **Tingling fingers/toes:** B12 deficiency

Investigations

- **FBC** – MCV (size of RBC) & MHC (Hb per RBC), reticulocytes
- **Iron studies** – serum iron & ferritin, TIBC
- **Blood film** – size, shape, colour of red cells
- **Serum bilirubin** – high in haemolysis
- **Hb HPLC** or **Hb electrophoresis** – shows amount of each Hb type (HbS, HbA, HbF)

Types of haemoglobin

Fetal (HbF): **2α** chains + **2γ** chains → higher O_2 affinity
Adult (HbA): **2α** chains + **2β** chains → lower O_2 affinity

Adults normally have 2 HbA alleles (HbAA). Patients with haemoglobinopathies such as thalassaemia or SCD have at least one abnormal allele, causing non-HbA haemoglobins (e.g. HbSS, HbAS)

Key questions to ask:

1. Isolated anaemia or pancytopenia? (pancytopenia suggests BM failure)
2. What is the MCV?

All patients are screened for anaemia before surgery

→ If **<100g/L** check haematinics and replace if low
→ If **<60g/L** as above, but transfuse if active bleeding

DDx microcytic anaemia: TAILS

- **T**halassaemia (α or β-thalassaemia trait)
- **A**naemia of chronic disease e.g. renal failure
- **I**ron-deficiency anaemia
- **L**ead poisoning
- **S**ideroblastic anaemia

DDx macrocytic anaemia: ABCDEF

- **A**lcohol & liver disease
- **B**12 deficiency
- **C**ompensatory reticulocytosis (blood loss)
- **D**rugs (cytotoxic)
- **E**ndocrine (hypothyroidism)
- **F**olate deficiency

DDx normocytic anaemia: CHARMD

- **C**hronic disease (↓ iron + ↑ ferritin)
- **H**aemolysis
- **A**cute blood loss
- **R**enal anaemia (causing low EPO levels)
- **M**arrow disorder
- **D**eficiencies combined (iron + B12)

Anaemia from reduced RBC production

Must r/o GI bleed in older patients

Iron-deficiency anaemia

→ ***most common anaemia worldwide*** *(especially menstruating females)*

Causes:

- **Inadequate intake** – Fe-deficient diet e.g. vegetarian
- **Malabsorption** – coeliac, gastrectomy
- **Increased requirements** – pregnancy
- **Chronic blood loss** – menorrhagia / GI bleed

NB. Ferritin will be raised in inflammation

Diagnosis:

1. **FBC:** ↓ MCV = microcytic
2. **Iron studies:** ↓ serum iron & ↓ serum ferritin, ↑ TIBC (body tries to ↑ Fe uptake)
3. **Blood film:** abnormally shaped, small, hypochromic (pale) RBCs

Management[1]:

1. **Determine cause:** thorough Hx & exam → r/o serious causes (e.g. GI malignancy)
   - OGD/colonoscopy, anti-tTG, menorrhagia Hx
2. Treat underlying cause
3. Dietary advice
4. Oral iron supplements e.g. ferrous sulphate/fumarate
   - TDS until Hb normal then minimum 3m OD
   - Can use IV iron if oral not tolerated/absorbed

NB: if no response to Tx consider Ix for other causes (esp. malabsorption)

Dietary advice for IDA

High Fe foods:

- Red meat, liver
- Pulses, beans, peas
- Leafy, green veg
- Oily fish
- Fortified cereals
- Dried fruit / nuts

Vit C (fruit & veg) helps Fe absorption

Foods to avoid:

- Excess cow's milk (only 10% Fe is absorbed)
- Tannin (tea) (inhibits Fe absorption)

B12 & folate deficiency

→ *causes pernicious anaemia*

Causes of B12 deficiency:

- **Low dietary intake** – vegan/vegetarian
- **Malabsorption** (in terminal ileum) – e.g. Crohn's, gastrectomy
- **Low intrinsic factor** – e.g. autoimmune (pernicious anaemia)

Causes of folate deficiency:

- **Low dietary intake**
- **Malabsorption** (in duodenum/jejunum) – e.g. coeliac, jejunal resection
- **Increased requirements** – pregnancy, haemolytic anaemia

Diagnosis:

1. **FBC:** ↑ MCV = macrocytic – often >125
2. **Blood film:** hypersegmented neutrophils (>5 lobules) & tear-drop cells
3. **Iron & B12 studies:** ↓ B12, ↓ serum folate, ↓ cobalamin
4. **Intrinsic factor antibodies**

Management[2]:

B12 = eggs, fortified cereals, dairy
Folate = broccoli, peas, brown rice

- If not pernicious anaemia, **dietary advice**
- IM B12 & folic acid (5mg OD) supplements (4m)
- If pernicious anaemia: **lifelong IM B12** replacement (hydroxocobalamin)

Intrinsic factor binds B12 in stomach to enable absorption in the terminal ileum

B12 = coenzyme needed for folate conversion
Folate = needed for RBC synthesis

B12 deficiency can cause peripheral neuropathy, subacute spinal cord degeneration & angular cheilitis

If B12 AND folate deficient, must replace B12 first to avoid subacute combined degeneration of the spinal cord

RBC aplasia

→ *failure of RBC synthesis*

Causes:

- **Diamond–Black anaemia** = rare, congenital → raised MCV ± short stature, abnormal thumbs
- **Transient erythroblastopenia** = triggered by viral infection in children
- **Parvovirus B19** – infects young RBCs – only causes RBC aplasia in children/adults with inherited haemolytic anaemia

Diagnostic results in RBC aplasia: ↓ Hb, ↓ **reticulocytes,** normal BR, Coombs test –ve

[1] BNF Treatment Summary – *Anaemia, Iron Deficiency*
[2] BNF Treatment Summary – *Anaemia, Megaloblastic*

Spinal cord tracts

Corticobulbar tract innervates each cranial nerve bilaterally except for:

1. **Facial nerve (CN VII):**
→ Forehead = bilateral representation
→ Branch to lower face = unilateral representation
→ UMN lesion spares frontalis

2. **Hypoglossal (CN XII):**
→ Each half of tongue supplied by contralateral corticobulbar tract
→ Lesion causes contralateral weakness, so tongue deviates **towards** weak side

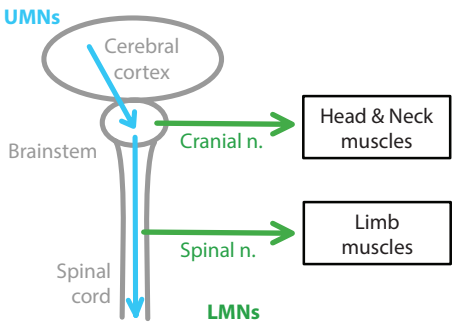


Fig. 7.3 Pathway of UMNs & LMNs.

***Pyramidal weakness:**
Upper limbs: extensor muscles weaker than flexors
= **results in** flexed elbow & wrist
Lower limbs: flexor muscles weaker than extensors
= **results in** extended knee/ straight leg

Bulbar palsy: **LMN lesion** causing dysarthria = weakness of muscles supplied by CN IX, X, XII
→ **Nasal speech / dysarthria**
→ **Hyporeflexia:** jaw jerk & gag reflex absent
→ **Tongue:** weak & wasted & fasciculations

The descending spinal tracts

Pyramidal tracts = voluntary control (cerebral cortex → brainstem & spinal cord)

Corticospinal: voluntary movement of **contralateral body**	**Corticobulbar:** voluntary movement of **face & neck**
→ Originate in **motor cortex** → **Decussate in medullary pyramids** → Synapse with **LMNs** in **spinal cord**	→ Originate in **motor cortex** → Terminates in **brainstem** → Synapse with **cranial nerves**

The ascending tracts

Dorsal column medial lemniscus (DCML)	Spinothalamic	Spinocerebellar
Fine touch, vibration, proprioception **(contralateral)**	Pain & temperature **(contralateral)**	Unconscious proprioception **(ipsilateral)** (awareness of position & movement of body parts in space without conscious thought)

Upper motor neurones vs. lower motor neurone lesions

UMNs: motor cortex → internal capsule → brainstem → spinal cord
LMNs: anterior horn cell → nerve root → peripheral nerve → NMJ → muscle

	Upper motor neurone lesion (brain + spinal cord)	Lower motor neurone lesion (peripheral nerves)
Signs	• Hypertonia • Hyper-reflexia • **Pyramidal** weakness* • Clonus • +ve Babinski • Spastic gait	• Hypotonia • Hypo-reflexia • **Proximal/distal** weakness • Wasting • Fasciculations
Causes	• Stroke / brain tumour • Spinal tumour / injury • Cerebral palsy / MS	• Peripheral neuropathy (DM, alcohol, drugs) • Polio (anterior horn cells) / Guillain–Barré • MND • Myasthenia gravis

Facial weakness

	Upper motor neurone lesion	Lower motor neurone lesion
Signs	• **Contralateral** weakness • **Frontalis spared**	• **Ipsilateral** weakness • **Whole face**
Causes	• Stroke / brain tumour • Subdural haematoma • MS	• Bell's palsy • GBS (usually bilateral) • Infection (HSV, CMV, EBV, Lyme disease) • Trauma

Pseudobulbar palsy: bilateral **UMN lesion** causing dysarthria = damage to medullary cranial nerves
→ **'Donald Duck' speech** (spastic dysarthria)
→ **Hyperreflexia:** jaw jerk & gag reflex increased
→ **Tongue:** weak & spastic

Dermatomes

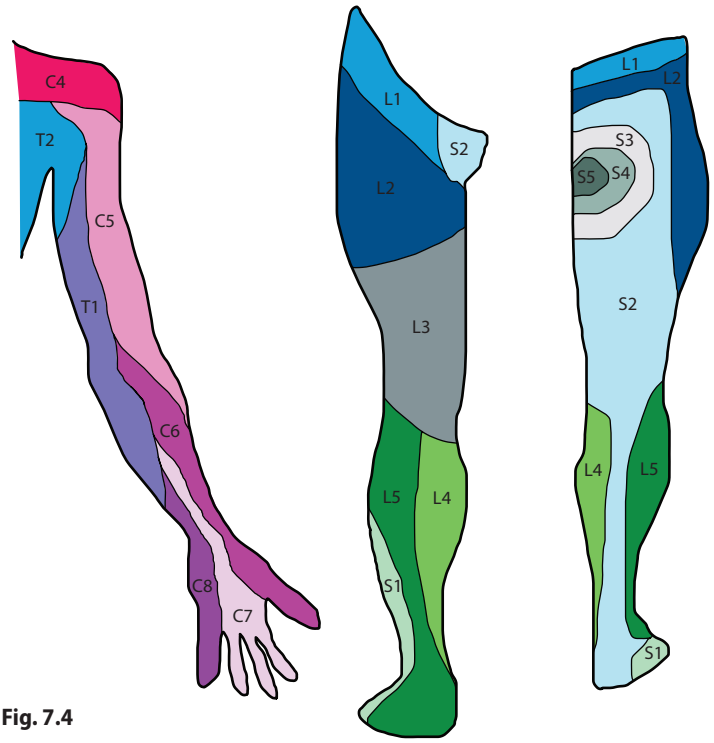


Fig. 7.4

Myotomes

Nerve root	Motor function
C5	Elbow flexion
C6	Wrist extension
C7	Wrist flexion, finger extension, elbow extension
C8	Finger flexion
T1	Finger abduction
L1,2	Hip flexion
L3	Knee extension
L4	Ankle dorsiflexion
L5	Knee flexion, big toe extension
S1	Knee flexion, ankle plantarflexion, big toe flexion

Deep tendon reflex	Nerve roots
Biceps + brachioradialis	C5, C6
Triceps	C7, C8
Knee jerk	L3, L4
Ankle jerk	S1, S2

Focal peripheral nerve lesions

Lesion	Symptoms	Sensory loss	Motor loss	Causes
Median nerve (C5–T1) Carpal tunnel	• Pain/tingling in hand • **Thenar muscle wasting**	Dorsal & palmar aspect of lateral 3.5 digits	• Middle & index fingers **(L)** • Thenar muscles **(OAF)** Lumbricals Opponens Abductor pollicis brevis Flexor opponens	• Repetitive motion • DM/thyroid • RA/OA • Pregnancy
Ulnar nerve (C8–T1) Cubital tunnel	• Pain/tingling in hand • Weak grip • **Ulnar claw (paradox)**	Dorsal & palmar aspect of medial 1.5 digits	**All small hand movement except LOAF (interossei/lumbricals)** • Finger abduction & adduction • Thumb adduction • Little & ring finger flexion • Wrist flexion	• Elbow trauma • Idiopathic
Radial nerve (C5–T1)	• **Wrist drop**	Dorsal hand & 1st web space	**Brachioradialis:** Wrist extension **Extensor digitorum:** Finger extension	• Humerus fracture
Brachial plexus (C4–T1)	• Erb's palsy (weakness, sensory loss & muscle atrophy)	Shoulder & arm	**Deltoid, biceps, brachialis, rotator cuff, serratus anterior**	• Idiopathic • Trauma • Damage from assisted delivery at birth • Compression • Inflammatory
Axillary nerve (C5–C6)	• Deltoid wasting	Regimental badge area	**Deltoid:** shoulder abduction	• Proximal humerus fracture
Common peroneal nerve (L4–S2)	• **Foot drop**	Lateral calf & dorsum of foot	**Tibialis anterior:** dorsiflexion & eversion	• Sporting injury (direct trauma near knee) • Fibular head fracture • Habitual leg crossing

Chronic kidney disease

= progressive & irreversible deterioration of renal function (>3m)

Factors increasing risk of needing RRT

- Younger age
- Lower eGFR
- Male gender
- Higher ACR

Signs/symptoms:

Early stages = **asymptomatic**

- **Anaemia:** pallor, fatigue
- **Bone disease:** osteomalacia/fractures
- **Hyperkalaemia:** palpitations, dizziness, chest pain
- **Fluid overload:** peripheral oedema, SOB
- **Early uraemia:** fatigue, anorexia, N&V, pruritus, hiccups
- **Late uraemia:** confusion
- **Metabolic acidosis:** ↑ HR & RR, vomiting, fatigue, headache
- **Other:** sexual dysfunction = common

** CKD associates with cardiovascular events & AKI **

Investigations:

1. **Urine dip & BP**
   - → Urine protein–creatinine ratio for nephrotic syndromes
   - → Urine albumin–creatinine ratio (ACR) for CKD
2. **Bloods:**
   - → U&Es – **compare to previous eGFR**
   - → Bicarbonate – acid–base balance lost
   - → Hb – normocytic anaemia
   - → PTH (↑ if bone disease & also ↑ ALP)
   - → Glucose (for DM)
   - → ANA, ANCA, complement
3. **USS kidney** (kidneys may be small)
4. **Renal biopsy** – if still unsure of cause
5. **CXR** – for pulmonary oedema

Pathogenesis of renal bone disease:

↓ **Vit D activation** in kidneys = ↓ Ca absorption = **2° hyperparathyroidism** (↑ PTH)

↑ PTH causes ↑ **osteoclastic activity** = ↑ Ca resorption from bone to restore serum Ca **= leaves bone weak**

In kidney transplant patients, parathyroid gland may become autonomous in spite of normal renal function = **tertiary hyperparathyroidism**

Classification

→ *use to identify those most at risk of needing RRT*

Base on 2 factors:
1. GFR
2. Albuminuria

			Persistent albuminuria		
			A1	A2	A3
			<3mg/mmol	3–30mg/mmol	>30mg/mmol
GFR (ml/min/1.73m²)	**G1**	≥90			
	G2	60–89			
	G3a	45–59			
	G3b	30–44			
	G4	15–29			
	G5	<15			

Table based on KDIGO AKI staging system.

Causes of deteriorating GFR

Diabetes mellitus (38%)	Glycation of efferent arteriole = ↑ pressure & sclerosis
Hypertension (25%)	Thickened walls of afferent arteriole = hypoperfusion
Chronic glomerulonephritis (16%)	Inflammation & damage to vessels
Chronic pyelonephritis	Urinary reflux or recurrent infection
Obstructive uropathy	Back-up of pressure = hydronephrosis & damage → neurogenic bladder, BPH, malignancy, stones
Polycystic kidney disease (PKD)	→ **Auto dominant** form presents in adults → Fluid-filled cysts press on nephrons = atrophy → Sx: back pain, headaches, haematuria, HTN

→ **Risk factors for CKD decline:** HTN, DM, smoking, infection, NSAIDs/ACEis

Management of CKD[4]

RISK REDUCTION of CVD: lifestyle factors are important
1. **BP control:** ACEi/ARB if proteinuria (BP<140/90 **or** <130/80 if diabetic)
2. **Cholesterol control:** statin
3. **Comorbidity control:** diabetes control
4. **Stop smoking**
5. **Weight management**

Advise low salt & phosphate diet

TREAT/MANAGE COMPLICATIONS
1. **Anaemia** – IV iron + EPO stimulating agents (r/o B12/folate deficiency first)
2. **Bone disease** – vit D & calcium supplements if deficient, phosphate binders
3. **Oedema** – careful fluid monitoring ± diuretics

[4] NICE (2021) *Chronic kidney disease* [NG203]

MANAGE MEDICATIONS

1. **Stop drugs** that worsen glomerular function / acute nephrotoxics
2. **Alter dose** of medications if GFR is low e.g. stop metformin if GFR <30

REFER TO NEPHROLOGY: if GFR <30 or ↓ >15 in 1y **or** 5y risk of needing RRT >5% (using kidney failure risk equation[5])
→ assess & manage symptoms/complications & prep for RRT

Renal replacement therapy options

ensure patients are fully informed of all options & supported in decision-making

DIALYSIS: usually started around eGFR 10, unless there are complications

	Haemodialysis	Peritoneal dialysis
Method	Blood pumped out of body through 'artificial kidney'	Dialysate solution infused into peritoneal cavity (peritoneum acts as filtering membrane)
Access	AV fistula / semi-permanent jugular or subclavian catheter	Catheter into peritoneum
Complications	Site infection, **hypotension**, air embolus, N&V, endocarditis	Peritonitis, catheter problems, hernia, fluid retention, weight gain
Frequency	3 × 4h sessions each week	**Continuous ambulatory** = 4 × 20min each day while active **Automated** = overnight (3–5 exchanges over 8–10h)

KIDNEY TRANSPLANT: gold standard for those with **end-stage CKD**
→ **but** only 40% of patients with CKD 5 are suitable

Who is suitable?
- Generally fit for general anaesthetic
- At least 5y left to live
- No underlying malignancy
- No other significant comorbidities
- Good vascular supply to legs

+ a suitable match is found:
→ ABO, tissue type, age & gender

+ Need to assess suitability of donor:
urine dip, U&Es, USS & general fitness

Process:
→ Transplant placed in **iliac fossa** & anastomoses of vessels made
→ Usually leave native kidney in place

Post-transplant treatment:
→ Lifelong immunosuppression = tacrolimus or ciclosporin **plus** azathioprine/MMF
→ 6m of steroids to prevent acute rejection e.g. prednisone
→ Aspirin, antihypertensives, PPI, bone protection

Complications:
- **Immediate:** graft thrombosis, ureteric leak/obstruction, bleeding, rejection
- **3–6m:** rejection, HTN, ileus, infections (urinary or respiratory)
- **Long-term:** cancer, interstitial fibrosis (2° to ciclosporin/tacrolimus), cardiac disease, infections

Ethical issue: waiting list of >4500 (avg. wait = 3y)

Transplant rejection: 10–20%
→ **Often asymptomatic** (concern if rise in Cr)
→ **RFs:** non-concordance, drug interactions, poor match
→ **Tx:** IV methylprednisolone & ↑ immunosuppressants

Points to consider for transplant:

Pros	Cons
• ↑ Longevity (80% 10y survival) • ↑ Quality of life (free from dialysis)	• Medication burden • Immunosuppressive SEs • Frequent hospital visits

[5] Major, *et al*. (2019) The Kidney Failure Risk Equation for prediction of end stage renal disease in UK primary care. *PLOS Medicine*, 16:e1002955

Pleural effusion

Types of fluid
Empyema: pus (infection)
Chylothorax: lymphatic fluid
Haemothorax: blood (trauma)
Fluid: transudate or exudate

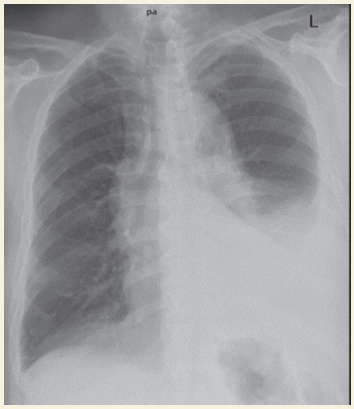


Fig. 9.2 Left-sided pleural effusion.

Accumulation of fluid in pleural cavity

Symptoms

May be asymptomatic
- SOB ± pleurisy
- Sx of underlying cause

Signs

- ↓ Expansion
- Stony dull percussion
- Absent breath sounds
- ↓ Vocal resonance / tactile fremitus
- Tracheal deviation away if massive

Investigations

1. **CXR** – blunted CPAs, homogenous consolidation (only detect if >300ml)
2. **USS** – identify location & volume
3. **Pleural tap (US guidance)***
   - Microbiology: MCS
   - Clinical chemistry: protein, LDH, glucose, pH
   - Cytology: 80% sensitive for malignancy
4. **Further Ix if no obvious cause:**
   - Pleural biopsy – r/o malignancy/TB
   - CT with contrast – shows pleural thickening

*1–2 intercostal spaces below upper border of effusion

Types of pleural effusion

	Transudate	Exudate
Pathophysiology	**Movement of fluid from circulation → pleural space** ↑ Capillary hydrostatic pressure **or** ↓ Capillary oncotic pressure	**Production & secretion of fluid into pleural space** ↑ Capillary permeability
Causes	• Congestive heart failure (↑ hydrostatic pressure) • Renal failure (↓ oncotic pressure ↓ **albumin**) • Liver failure (↓ oncotic pressure ↓ **albumin**) • Hypothyroidism	**Inflammation:** trauma, RA, sarcoid, SLE **Infection:** pneumonia, TB **Infarction:** PE, post-MI (Dressler's) **Malignancy:** 25% of effusions **Medication:** MTX, amiodarone, phenytoin
Presentation	**Bilateral**	**Unilateral**
Fluid	**Lower protein, lower LDH** <25g/L protein + ↑ LDH	**Higher protein, higher LDH** >30g/L protein + ↑↑ LDH
Management	**Usually improve with Tx of underlying cause** → Usually do not need tapping/drainage	**Pleural tap all with pneumonic illness or suspected malignancy + effusion** Treat cause & drain if moderate/large

LIGHT'S CRITERIA
used to distinguish transudate & exudate if protein 25–35g/L

	Transudate	Exudate
Pleural:serum protein	**<0.5**	**≥0.5**
Pleural:serum LDH	**<0.6**	**≥0.6**
Pleural fluid LDH	**<2/3 upper limit of normal**	**>2/3 upper limit of normal**

Specific management of pleural infection (empyema)[2]

1. **Pleural tap: pH <7.2, low glucose, high LDH**
   - Simple parapneumonic infection = straw colour with no orgs
   - Loculated empyema = pockets of semi-solid pus
2. **IV ABX** for minimum 2w
3. **Chest drainage:** if frank pus / organisms cultured / pH <7.2 / no improvement
4. **Decortication (VATS: video-assisted thoracoscopy):** remove restrictive layer of fibrous tissue → if long-standing pus / thickened pleura

Sx of empyema = effusion + FEVER

Organisms causing empyema:
Strep. milleri, H. influenzae, E. coli, Staph. aureus, Pseudomonas

[2] BMJ Best Practice (2021) *Pleural effusion*

Pneumonia = signs of respiratory tract infection + new shadowing on CXR

Community-acquired pneumonia	Hospital-acquired pneumonia
Primary or secondary to lung disease	**>48h** after admission or **within 10d** of discharge
• ***Strep. pneumoniae*** **(80%)** • ***H. influenzae*** **(more common in COPD)** • *Mycoplasma pneumoniae** (younger patients) • *Chlamydia pneumoniae** (elderly patients) • *Legionella pneumoniae** *Atypicals • **Viral:** RSV, influenza, Covid-19 (15%)	• **Gram-negatives:** ▸ Klebsiella ▸ *E. coli* ▸ *Pseudomonas* • MRSA / *Staph. aureus*

Aspiration pneumonia: in stroke, neuromuscular disease, ↓GCS

Risk factors for pneumonia:
- Immunocompromised
- Hospitalised
- Chronic lung disease
- Elderly/young/male
- Alcoholic/smoker/IVDU

Clinical presentation

- **Fever,** rigors, malaise, anorexia
- **Productive cough** – rusty sputum / haemoptysis
- **Dyspnoea**
- Pleuritic chest pain

On examination

- **↑RR, ↑HR, ↑temp**
- **↓O_2 sats** / cyanosis
- **↓GCS** / delirium – if elderly
- **Signs of consolidation**
  - **↓expansion**
  - **coarse crackles**
  - pleural rub, bronchial breathing
  - **dull percussion**
  - **↑vocal resonance**

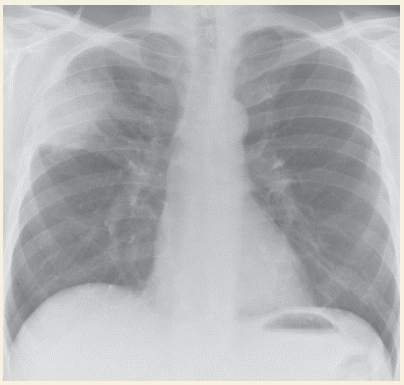


Fig. 9.3 Right upper zone consolidation.

Investigations[3]

- **Bedside:** basic obs, urine dip
- **Bloods:**
  - ▸ FBC, U&Es, CRP, LFT, glucose
  - ▸ ABG
  - ▸ Atypical serology
- **Microbiology:**
  - ▸ Sputum & blood cultures (guide ABX choice)
  - ▸ Urine antigens (atypical orgs)
  - ▸ Throat swab (if suspect viral)
- **Chest X-ray:** consolidation = **DIAGNOSTIC**

Risk stratification: CURB65 score

C	Confusion	MMSE ≤8 **or** disorientated
U	Urea	>7mmol/L
R	Resp rate	≥30/min
B	BP	SBP <90 or DBP <60
65	Age	>65y

Mild: 0–1 = home Tx
Mod: 2 = hospital Tx
Severe: ≥3 = consider ICU

Management[3]

1. **Conservative:** analgesia, O_2, antipyretics, IV fluids, chest physio
2. **Antibiotics:** follow local antimicrobial guideline (see table below for common examples)

		1st line	If penicillin allergy	Duration & route
CAP	**Mild**	Amoxicillin 500mg TDS	Doxycycline	5d PO
	Mod	Amoxicillin 500mg TDS + clarithromycin 500mg TDS	Doxycycline + clarithromycin	7d PO
	Severe	Co-amoxiclav + clarithromycin	Levofloxacin + vancomycin	10d IV
HAP	**Mild**	Doxycycline PO		5d PO
	Severe	Co-trimoxazole PO		5–7d IV
Aspiration		Amoxicillin + **metronidazole**		5–7d IV

3. **Long-term:** smoking cessation advice, influenza vaccine if high risk

Complications of pneumonia:
- Pleural effusion
- Resp. failure
- **Sepsis**
- Empyema
- Lung abscess
- Bronchiectasis
- Pneumothorax
- Lobe collapse

Repeat CXR after 6w to ensure resolution & no underlying malignancy / lung abnormalities

[3] BMJ Best Practice (2021) *Community acquired pneumonia*

Postoperative care

Summary of postoperative problems

Immediate	Delayed
• Primary haemorrhage • Shock • Low urine output • Pain • N&V • Confusion/delirium	• Infection (pneumonia, UTI, wound) • Pressure sores • Paralytic ileus • Secondary haemorrhage (7–10d post) • Acute MI (in first 72h) **Immobilisation:** • DVT, PE, stroke – give LMWH/aspirin • Urinary retention & AKI • Chest infections – in elderly/ventilated • Atelectasis • Pressure sores & muscle wasting

Common sources of post-op pyrexia & infection

Chest (infection)
Catheter (UTI)
Cut (infection)
Cannula (infection)
Calves (DVT)
Central line (infection)
Collections (abdo/pelvis)

Start SEPSIS 6 if potential sepsis (qSOFA score ≥2 / clinical judgement)

1–3d: respiratory
3–5d: urinary
5–7d: wound/abscess/leak

Ix: blood cultures, CXR, urine dip / MCS, cannula site, surgical wound swab, CT/USS of surgical site

Anastomotic leak

AETIOLOGY: leak of luminal contents from a surgical join → important complication of **GI surgery**

RISK FACTORS:

Patient risk factors	Surgical risk factors
• Medication (steroids/ immunosuppressants) • Smoking/alcohol • DM, obesity, malnutrition	• **Emergency surgery** • **Longer intra-operative time** • Oesophageal–gastric or rectal anastomosis • Peritoneal contamination (by free pus or faeces)

CLINICAL PRESENTATION: 5–7d post-op (often due to ischaemia)
- Abdo pain ± peritonism
- **Fever**, tachycardia, new atrial fibrillation

→ consider in any patient **failing to progress** post GI resection

INVESTIGATIONS:
- FBC, CRP, U&Es, LFT, clotting, VBG
- Group & save
- **CT with contrast = diagnostic**

MANAGEMENT:
- **SEPSIS 6** (IV ABX)
- Larger leaks may need drainage or laparoscopic exploration / surgical intervention

Intra-abdominal abscess

RISK FACTORS:
- Intra-abdominal infection (e.g. appendicitis, diverticulitis)
- Recent intra-abdominal surgery

CLINICAL PRESENTATION:

CT abdo with contrast = diagnostic

- Fever
- Anorexia/N&V
- Abdominal pain
- Altered bowel habit / prolonged ileus

MANAGEMENT:
- IV ABX + drainage (+ send fluid for culture)
- May need to return to theatre

Paralytic ileus

AETIOLOGY: **reduced intestinal motility** → very common following abdo/pelvic surgery

RISK FACTORS:

Patient risk factors	Surgical risk factors
• Increased **age** • **Electrolyte derangement** (e.g. Na^+, K^+ and Ca^{2+}) • Neurological disorders (e.g. dementia/Parkinson's)	• Use of **anti-cholinergic/opioid** medication • **Pelvic/abdominal surgery** • Extensive **intra-operative intestinal handling** • Peritoneal contamination (by free pus or faeces)

CLINICAL PRESENTATION:
- Failure to pass faeces/flatus
- Abdo distension & **absent bowel sounds**
- N&V

Ix are done to rule out more serious pathologies

MANAGEMENT:
- IV fluids & daily bloods (electrolytes)
- Encourage mobilisation
- **Review analgesia (opiates)** in conjunction with pain team
- Consider if need to be NBM ± NG tube (remove stomach contents to reduce vomiting)

INVESTIGATIONS:
- FBC, CRP, U&Es
- Electrolytes (Ca^{2+}, PO_4^-, Mg^{2+})
- **Consider imaging:** AXR ± CT (to rule out other pathology e.g. obstruction)

Post-op haemorrhage

Primary bleed – within intra-operative period → resolved during operation & close monitoring post-op
Reactive bleed – within 24h of operation → usually a missed vessel / slipped ligature
Secondary bleed – 7–10d post-op → usually erosion of a vessel 2° to infection

More detail on haemorrhagic shock in Chapter 16: Critical illness

SIGNS/SYMPTOMS: tachycardia, tachypnoea, dizziness, reduced urine output (NB: hypotension = **late sign**)

INVESTIGATIONS: thorough examination for signs of bleeding/swelling/discolouration/tenderness/peritonism

MANAGEMENT:
1. **ABCDE** → IV fluid resuscitation + direct pressure on bleeding site if visible
2. **Urgent senior review** → may need to return to theatre
3. **Urgent blood transfusion** – if moderate/severe bleeding (activate major haemorrhage protocol if necessary)

Urinary retention

Risk factors for urinary retention:
- >50y
- Spinal/epidural
- Neuro comorbidity
- Paralytic ileus
- Pelvic/uro surgery
- Opiates, antimuscarinics
- Infection/sepsis
- Constipation

PRESENTATION:
- Reduced output
- Suprapubic mass/pain

INVESTIGATIONS:
- USS bladder (residual volume)
- Kidney function: eGFR, U&Es

MANAGEMENT:
- Withdraw causative agents
- **Catheter**

Work-up for AKI:
- Fluid status
- FBC, CRP, U&Es, LFT
- Urine dip
- USS KUB

General overview

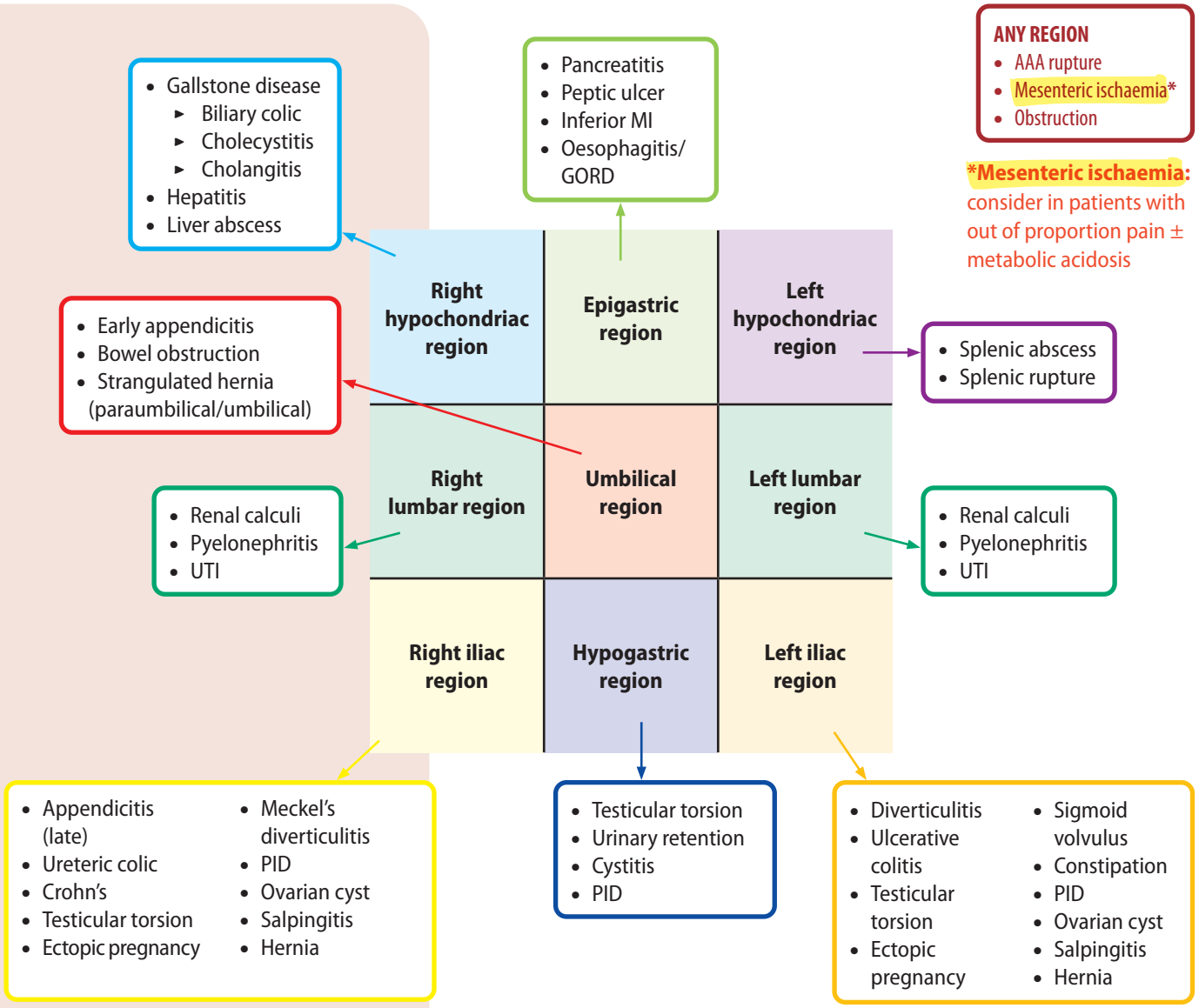


Fig. 11.1 This diagram should be used as a guide to the differentials of acute abdominal pain. It is important to note that any of the mentioned pathologies can present with pain in any area of the abdomen, and thus it is important to correlate location of pain with other clinical signs and investigations, and not to rule out differentials based on the location of pain alone.

Medical causes of acute abdominal pain

- DKA
- UTI
- Basal lobe pneumonia
- Poisoning/overdose
- Addison's disease
- Hypercalcaemia
- Spontaneous bacterial peritonitis
- Mesenteric adenitis
- Constipation

Patterns of pain

Type of pathology			
Inflammatory	**Obstructive**	**Perforation**	**Visceral**
Peritonitic pain → fever, tachycardia → ↑WCC, ESR, CRp	**Colicky pain (waves of pain)** → vomiting, constipation → distension, tinkling sounds	**Sudden localised then peritonitic pain** → **shock** (↑HR, RR, ↓BP) → N&V	**Poorly localised pain (referred)** → specific Sx e.g. jaundice

Peritonitic pain = worse on inspiration & movement → lie still, shallow breaths, **rigidity & guarding**

Surgery

Investigations & management

INVESTIGATIONS:

- **Obs:** BP, HR, RR, sats, temp, ECG
- **Pregnancy test:** in all females of child-bearing age
- **Bloods:** FBC, U&Es, LFT, CRP, amylase, glucose, clotting, ABG (lactate)
- **Urinalysis:** protein, nitrates, leukocytes, blood, glucose, pH
- **Imaging:** USS, erect CXR, abdo CXR, CT
- **Specialist tests:** MRCP/ERCP, MRI, barium swallow, OGD, colonoscopy
  → **not always indicated**

NB: Not all acute abdominal pathologies present with pain – often absent in the elderly, children, diabetics & pregnant women

Amylase can be raised in some pathologies other than pancreatitis e.g. perforated duodenum

Erect CXR: may show air under diaphragm; **however**, a negative erect CXR does **not** exclude pneumoperitoneum

INITIAL MANAGEMENT: early senior input if concerned

- **ABCDE + targeted Mx of suspected cause**

Things to consider:

- Nutritional and feeding status – keep NBM if surgery likely soon or vomiting
- Hydration and fluid balance – IV or oral fluid maintenance, consider catheter
- Are antibiotics indicated? – signs consistent with infection
- Investigations to guide/aid management – blood cultures, ABG, G&S, CXR, ECG
- Is blood transfusion / major haemorrhage protocol required?
- Is theatre / surgical intervention needed? – NBM, consent, book theatre slot

IMAGING INDICATIONS:

X-ray	Abdo USS	CT
• Obstruction • Toxic megacolon • Foreign body	• Biliary pathologies • Kidneys, ureter, bladder • Gynae pathologies • Appendix (in a female patient, no role in male)	• AAA/vascular • Malignancy/mass • Complications of obstructions • Appendix if >50y

CONSIDERATIONS FOR SURGERY:

Patient health:

- Anaesthetic review
- Comorbidities/frailty
- **Ceiling of care:** ReSPECT form / DNACPR
- **Morbidity/mortality calculation** e.g. P-POSSUM score

ReSPECT = Recommended Summary Plan for Emergency Care and Treatment, a patient-held form

Patient wishes:

- Current wishes
- Advance directives / ReSPECT forms
- Discussion with family

NB: the decision to operate is a complicated one and many factors should be considered.

See *Chapter 16: Critical illness* (and *Chapter 10: Anaesthetics* in the companion *Clinical Specialties* book) for further details.

Colorectal cancer

↳ most = adenocarcinomas

→ **3rd most common cancer**
→ 2nd most common cause of cancer deaths
→ **Peak age:** >60y
→ M>F
→ **1 in 30 risk**

Risk factors

- **Polyps** (UC, HNPCC, FAP) → identify & remove
- **Age** (90% are aged >50 y)
- **Diet** – low fibre, high fat, processed meat
- **Smoking/alcohol/obesity**
- **IBD** (UC especially)
- **Genetics** (FAP & HNPCC / Lynch syndrome)
- **FHx**

Polypectomy + follow-up endoscopy (complications = bleeding & perforation)

Familial adenomatous polyposis (FAP):

Auto dominant mutation in *APC* gene

→ **Hundreds of polyps**
→ Presents at **35–45y** (consider if <50y with colorectal cancer)
→ Colonoscopy every **2y** from **25 to 75y**

HNPCC / Lynch syndrome:

Auto dominant mutation in DNA repair genes

→ Cancers of colon, endometrium, ovary, stomach, bladder, brain, skin
→ Presents **>40y** (consider if <50y with colorectal cancer)
→ Colonoscopy every **2y** from **25 to 75y**

NB: non-polyposis condition (no polyps present)

Amsterdam criteria to Dx

Bowel cancer screening programme[3]:

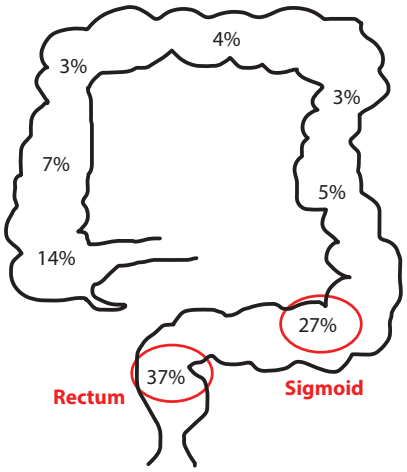
qFIT (quantifiable faecal immunochemical test)
→ if >120mcg/g refer for colonoscopy to remove any polyps

Every 2y from the age of 50y

May present as an acute emergency:

- Bowel obstruction
- Bowel perforation
- Extreme pain

Clinical presentation



Liver = common site of colorectal cancer metastases

Fig. 12.4 Sites of colorectal cancer.

Right side	Left side	Rectal
= Late presentation • **Weight loss / fatigue** • **IDA** ± Abdo pain/mass	• **Weight loss / fatigue** • **Change in bowel habit** • Obstruction • Blood-streaked stools ± Abdo pain/mass	• **Weight loss / fatigue** • Tenesmus • Bright PR bleed • **DRE:** palpable mass

→ 20% present with signs of **disseminated disease** e.g. liver mets = jaundice

Investigations

1. **DRE**
2. **Bloods** – FBC (for IDA), LFTs, U&Es
3. **Tumour marker:** CEA (not specific – use for monitoring)
4. **Colonoscopy & biopsy = gold standard**
5. **MRI** – if rectal cancer
6. **CT (chest, abdo, pelvis)** – for TNM staging

Classification & staging

Duke's criteria or **TNM staging**

Stage	Duke's stage	Features	5y survival[5]
1	A	Mucosa only	90–95%
2	B1	Into muscularis	80–85%
	B2	Through wall	
3	C1	T2 + nodes	60–65%
	C2	T3 + nodes	
4	D	Distant mets	<11%

Red flags for 2ww referral[4]:

1. ≥40y with unexplained weight loss & abdo pain
2. ≥50y with unexplained rectal bleeding
3. ≥60y with altered bowel habit **or** IDA
4. Occult blood present when faeces tested

Also consider if:

- Rectal or abdominal mass
- ≤50y with rectal bleeding and any of:
  - Abdominal pain
  - Altered bowel habit
  - Weight loss
  - IDA

[3] Bowel cancer screening programme (www.gov.uk/guidance/bowel-cancer-screening-programme-overview)
[4] NICE (2015, updated 2021) *Suspected cancer: recognition and referral* [NG12]
[5] Cancer survival by stage at diagnosis for England, 2019. ONS.

Management of colorectal cancer[6]

→ **colectomy + 'en bloc' lymph node removal** ± chemotherapy

Different levels of resection: ● = location of tumour

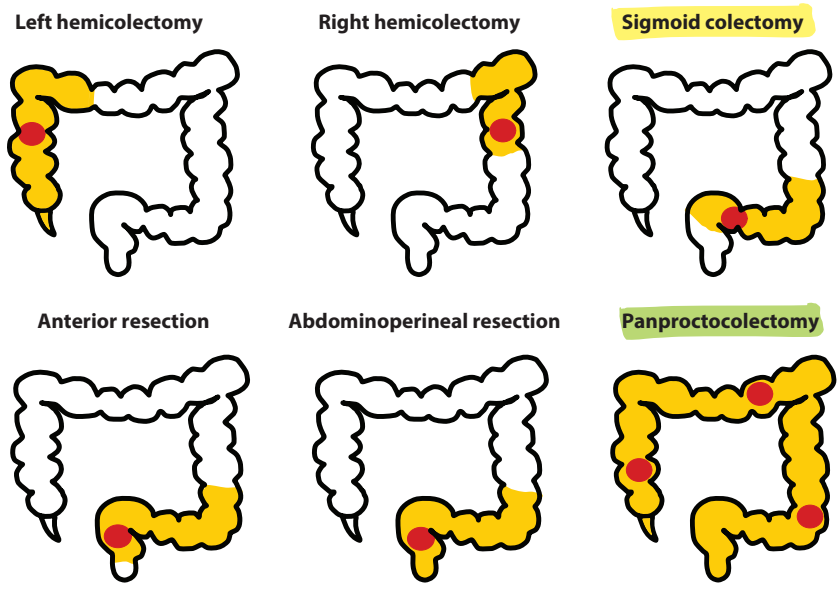


Fig. 12.5

MANAGEMENT OF OBSTRUCTING COLON CANCER: emergency

1. ABCDE
2. Analgesia + NG tube decompression
3. CT scan if no small bowel distension = competent ileocaecal valve = BAD
4. Endoscopic stenting **or** colectomy (often with stoma) → colectomy type depends on obstruction location

Management of rectal cancer[6]

1. **Total mesorectal excision** (removal of rectum)
   - **Low tumours <5cm above anus:** abdominoperineal excision (resect + stoma)
   - **Higher tumours >5cm from anus:** anterior resection (resect + anastomosis)
2. **± Chemotherapy ± radiotherapy**

Colorectal cancer follow-up

Follow-up for 5y post-surgery

1. **CEA** – check every 6m
2. **CT CAP** – annually
3. **Colonoscopy** – at 1 & 5y

20–30% recurrence

MDT input
including stoma nurse / specialist nurse

Lymph drainage:

→ Ascending & transverse drain to **superior mesenteric LNs**
→ Descending & sigmoid drain to **inferior mesenteric LNs**
→ Rectum (above pectinate line) to **internal iliac LNs**

Hartmann's
= sigmoid colectomy with colostomy (stoma formation)

Panproctocolectomy
= if synchronous cancer or inflammatory bowel disease

Stoma formation:

Elective or emergency

Decision depends on:
- location of resection
- indication for operation
- patient comorbidities
- patient lifestyle/preference

[6]NICE (2020, updated 2021) *Colorectal cancer* [NG151]

Chronic peripheral arterial disease

Causes

1. **Atherosclerosis:** DM, smoking, age, male, HTN, obesity, inactivity, cholesterol
2. **Vasculitis:** Buerger's disease → common in **young, heavy smokers**
3. **Fibromuscular dysplasia** → non-inflammatory arterial wall thickening
4. **Other:** cystic adventitial disease, iliac endofibrosis, popliteal artery entrapment

Pathogenesis: endothelial dysfunction → inflammation → macrophages → fatty streaks → plaque → rupture → platelet adherence → thrombus

Fontaine classification:

Asymptomatic
↓
Intermittent claudication
↓
Ischaemic rest pain
↓
Ulceration/gangrene

Signs and symptoms

	Intermittent claudication	Ischaemic rest pain	Peripheral neuropathy Often coexists alongside PAD in DM
Pathology	Insufficient perfusion **during exercise**	**Constant** insufficient perfusion	Damaged **peripheral nerves**
Symptoms	**Cramping muscle pain** → on exercise (limits walking*) → relieved by rest → reproduced walking **same distance**	**Continuous, severe, burning/aching** → worse at night → relieved by dangling leg out of bed → relieved by walking on a cold floor	**Tingling/numbness** ± pain → glove & stocking distribution → **no relief** dangling foot / cold floor → prone to wounds = infection risk
Signs	**Claudication distance* • Absent/weak peripheral pulses • Cold, pale, hairless legs • Buerger's angle <20° (angle leg raised before pallor)		• Peripheral pulses present • No pallor with Buerger's

Infection more likely if coexisting PAD

Leriche syndrome: occluded distal aorta, iliac & femoro-popliteal vessels
→ bilateral buttock/thigh pain ± erectile dysfunction

Ischaemic rest pain >2w + ABPI <0.4 or TP <30mmHg = **critical limb-threatening ischaemia (CLTI)**
can cause development of **ulcers & gangrene** (↑ risk if DM) & needs **urgent Ix & Tx** to prevent limb loss

DDx of intermittent claudication

- **Spinal stenosis**
- **Venous claudication**
  - **bursting** pain from **start** of walking
  - relieved by **elevation**
- **Sciatica** = shooting pain
- **Popliteal artery entrapment**
  - normal pulses
  - younger, active, Sx when exercising

Intermittent claudication	Spinal stenosis
Fixed claudication distance	Leg pain may be present at rest ± back pain
Not precipitated by ↑ time standing	Worsened by ↑ time standing
Worse walking uphill	Better walking uphill / bending forward
O/E: absent peripheral pulses	**O/E:** pulses present but **neurological** Sx

Investigations

→ needs full cardiovascular screen

1. **History & examination:** peripheral vascular exam + BP, ECG
2. **Bloods:** FBC, lipids
3. **ABPI ± treadmill test**
4. **Duplex USS:** show site & degree of stenosis = non-invasive
5. **MR/CTA:** image larger aorto–iliac vessels = **invasive**

$$\text{ABPI}_{\text{right}} = \frac{\text{right ankle pressure}}{\text{highest brachial pressure}}$$

ABPI	Interpretation[9]	Action
>1.3	Calcification (diabetes)	TBPI (toe BPI)
1–1.3	Normal	No action
0.4–0.9	**Mild–moderate PAD** (intermittent claudication)	**Routine** referral
<0.4	**Severe PAD** (rest pain)	**Urgent** referral
<0.3	**Impending gangrene**	

Management[10]

MEDICAL

Action is based on severity of symptoms

- **Lifestyle:** stop smoking, weight loss, exercise programmes (weekly for 3m)
- **Optimise comorbidities:** HTN, DM, cholesterol (high dose **statin**)
- **Antiplatelets:** 75mg clopidogrel (or 2.5mg rivaroxaban BD + 75mg aspirin)
- **Vasoactive drugs:** consider use in claudicants e.g. naftidrofuryl or cilostazol

[9] Cronenwett JL & Johnston KW (2014) *Rutherford's Vascular Surgery*. 8th edition. Elsevier Health Sciences
[10] European Society for Vascular Surgery (2019) *Clinical practice guideline on management of chronic limb-threatening ischemia*

Surgery

Common sites of atherosclerosis

- Coronary arteries
- Major branches of the aortic arch
- Visceral branches of abdo aorta
- Terminal abdominal aorta + branches

*Good collateral blood supply so symptoms only likely to occur if ≥2 vessels affected

Risk factors for chronic mesenteric vascular occlusive disease: as for all arterial disease (+ typically females >60y)

Complications:

- Malabsorption / weight loss
- Bowel infarction

*If not suitable for endovascular intervention, endovascular intervention failed, or young with complex non-atherosclerotic lesions

Presentation of gut ischaemia:

Triad of Sx: clinical diagnosis

1. Out of proportion pain
2. Diarrhoea &/or vomiting
3. Source of embolus e.g. AF

SURGICAL → consider in more severe cases or where medical treatment has failed

- Percutaneous transluminal angioplasty (PTA) or stenting
- Surgical reconstruction (bypass graft)
- Amputation = **last resort**

Chronic mesenteric vascular occlusive disease

→ *atherosclerosis of **SMA, coeliac trunk & IMA***, causing inadequate perfusion for digestion → 'gut claudication'*

CLINICAL PRESENTATION: (at least 3m duration)

- **Postprandial epigastric pain:** 10min to 3h after eating
- Gross weight loss & fear of eating (but appetite unaffected)
- Non-specific GI symptoms: altered bowel habits, N&V
- ± other vascular comorbidities e.g. HTN, DM, smoking etc.

INVESTIGATIONS: diagnosis of exclusion (extensive GI examination needed to r/o other causes)

- History & examination: may have **epigastric bruit & tenderness**
- Routine bloods: often normal → may be nutritional deficiencies
- Duplex ultrasound of mesenteric vessels
- **CT angiography** → diagnostic

MANAGEMENT[11]: urgent if significant weight loss, diarrhoea ± continuous pain

SMA is the most important vessel to keep patent

1. **Modify risk factors:** diet & exercise, stop smoking
2. **Antiplatelet** (75mg clopidogrel) **& high dose statin** (e.g. 80mg atorvastatin)
3. **Percutaneous mesenteric stenting:** less invasive than surgery
4. **Surgery:** bypass graft*

Other causes of gut ischaemia

ACUTE OCCLUSIVE MESENTERIC ISCHAEMIA: due to embolism
Sx: severe, sudden abdominal pain
RF: AF, vascular disease
Ix: negative D-dimer can rule out → do not use lactate to diagnose or rule out
Mx: surgical embolectomy ± retrograde stenting; may need bowel resection

NON-OCCLUSIVE MESENTERIC ISCHAEMIA (NOMI): hypoperfusion causes ischaemia despite patent mesenteric vessels
Sx: severe abdominal pain
Causes: vasopressors, cocaine, abdominal compartment syndrome (ACS), dialysis, severe burns, cardiac surgery
Mx: surgical/endovascular revascularisation ± treatment of ACS with decompressive laparotomy

ISCHAEMIC COLITIS: acute, transient reduction in blood supply to large bowel
Sx: severe pain ± bloody stools
Causes: low BP / shock, thromboembolism, ↑ age, cocaine
Mx: supportive ± colectomy if peritonitic

[11] European Society for Vascular Surgery (2017) *Clinical practice guideline on management of the diseases of mesenteric arteries and veins*

Bladder outlet obstruction

Common causes

→ BPH
→ Prolapse
→ Post-incontinence surgery
→ Bladder calculi
→ Urethral strictures
→ Malignancy (bladder/urethral/prostatic)
→ External compression
→ Neurological disease

Clinical presentation

1. **Lower urinary tract symptoms** (LUTS)
2. **Acute urinary retention:** suprapubic pain, palpable bladder, anuria — rapid onset
3. **Chronic urinary retention:** LUTS, renal impairment, palpable bladder, overflow incontinence, large residual urine volume

Voiding symptoms	Storage symptoms
Hesitancy Poor flow Post-void dribbling Dysuria (sensation of incomplete emptying)	**F**requency **U**rgency ± **u**rge incontinence 'FUN' **N**octuria (bedwetting / overflow Incontinence)
Present in **obstruction**	Present if **bladder dysfunction** (can be 2° to chronic obstruction)

Bladder calculi

PATHOGENESIS: crystallisation of minerals in urine if it becomes concentrated (if incomplete emptying / urine stasis)

Often uric acid or calcium stones (or struvite if UTI-related)

CAUSES:

Outlet obstruction	Neurogenic retention	Other
• BPH / prostate carcinoma • Bladder tumour • Urethral stricture • Prolapse (in women)	• Stroke / spinal cord injury • Spina bifida • Diabetes • Augmentation cystoplasty	• Infection (UTIs) • Dehydration • Foreign body / catheter • Passage of renal calculi into bladder

CLINICAL PRESENTATION:

- **LUTS:** voiding & storage
- **Dysuria & haematuria** at end of stream
- Lower abdo pain

± recurrent UTI

INVESTIGATIONS:

- **Hx & exam:** abdo & pelvic + DRE
- **Bloods:** FBC, CRP, U&Es, Ca, PO_4, urate, glucose, VBG (HCO_3)
- **Urine dip & MCS** – r/o infection
- **Basic imaging:** USS
- **Specialist imaging:** CT KUB

MANAGEMENT[7]:

Transurethral cystolitholapaxy

1. Cystoscope passed up urethra to identify stones
2. Crush stones or fragment with laser / pneumatic device
3. Bladder irrigation to remove fragments

OR Open cystolithotomy = surgical removal of bladder stones via a lower abdominal incision

[7] European Society of Urology (2021) *Guidelines on bladder stones*

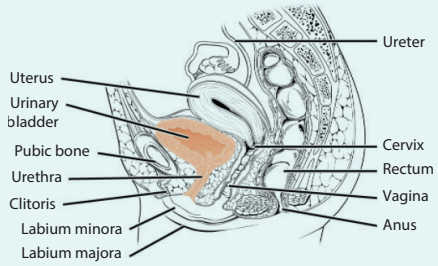


Fig. 15.4 Female genitourinary anatomy.

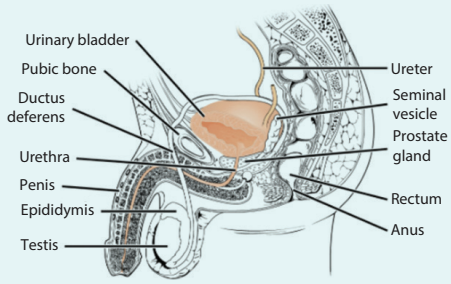


Fig. 15.5 Male genitourinary anatomy.

IPSS: score/questionnaire to determine how much symptoms impact daily life

Management of acute urinary retention:

1. **ABCDE**
2. Bloods: FBC, U&Es (deranged U&Es = HPCR → need USS KUB & urology R/V)
3. **Catheter** (3-way if haematuria)
4. Full Hx and examination (including DRE & neuro exam)
5. Monitor fluid and electrolyte balance

Bladder calculi increase risk of bladder cancer

Benign prostatic hyperplasia (BPH)

→ *50% of over 50s*
→ *affect transitional zone of prostate*

PATHOGENESIS: occurs **with age** due to **androgens** stimulating **increase in number of prostate cells**

CLINICAL PRESENTATION:
- **Voiding symptoms** ± secondary storage symptoms
- ± **Haematuria**
- **Enlarged, smooth prostate** on DRE
- **Acute retention** (sometimes occurs = rapid & painful)

INVESTIGATIONS:
1. **History & examination:** IPSS score & **remember DRE**
2. **Urinalysis:** dip & MCS
3. **Uroflowmetry:** <10ml/sec suggests obstruction
4. **Bloods:** including PSA
5. **Transrectal USS + biopsy:** definitive diagnosis

MANAGEMENT[8]: depends on symptom severity

Mild	Moderate	Severe
1. Reassurance 2. Lifestyle (fluid intake) 3. Follow-up	**Pharmacotherapy** 1. **Alpha blockers (e.g. tamsulosin)** = relax smooth muscle → **SEs:** retrograde ejaculation, postural hypotension, dizziness, headache 2. ± **5-alpha-reductase inhibitors (e.g. finasteride)** = inhibits testosterone → DHT conversion → **pros:** ↓ prostate size → **cons:** take 6w to 6m to work → **SEs:** loss of libido, impotence 3. ± **Anti-cholinergics (e.g. oxybutynin/ solifenacin)** In combination with above to treat 2° storage LUTS	**Surgery:** 1. **TURP** = gold standard 2. **Other:** e.g. laser/steam treatment 3. **Prostatic artery embolisation** 4. **Self-catheterisation / long-term catheter**

Chronic urinary retention

Chronic obstruction can lead to **chronic urinary retention** → gradual stretching of bladder over years (painless)
- **Symptoms:** asymptomatic or LUTS, palpable bladder, overflow incontinence, large residual volume of urine
- **Complications:** UTIs, calculi, high pressure chronic retention (HPCR) → renal impairment & hydronephrosis

Possible complications of TURP:
Acute: bleeding, UTI

Chronic: retrograde ejaculation, impotence, incontinence, bladder neck stenosis / urethral stricture

[8] NICE (2010, updated 2015) *Lower urinary tract symptoms in men* [CG97]

Electrolyte abnormalities

Hyperglycaemia (DKA & HHS)

DIAGNOSIS:

DKA	HHS
1. **Hyperglycaemia:** random BG >11mmol/L 2. **Ketosis:** capillary ketones >3mmol/L or urinary ketones ++ 3. **Acidosis:** HCO_3^- <15 or pH <7.35	1. **Profound hyperglycaemia:** random BG >30mmol/L 2. **No ketosis & pH >7.3** 3. **High osmolality:** >320mosmol/kg

MANAGEMENT OF HYPERGLYCAEMIC HYPEROSMOLAR STATE (HHS)[1]

1. **ABCDE** & confirm Dx with lab results & osmolality (VBG)
2. **IV FLUIDS** 0.9% saline (infusion over 24h)
3. **Do not start insulin** (until rate of fall in glucose is <5mmol/L per hour)
   → then give fixed rate insulin at **half dose of** DKA (0.05 units/kg/h)
4. **K^+ replacement with KCl** if K^+ drops to <5.5mmol/L
5. **Prophylactic anticoagulation** – LMWH for full duration of admission

MANAGEMENT OF DIABETIC KETOACIDOSIS (DKA)[2]

1. **ABCDE**
2. **Confirm Dx with lab results** (VBG)
3. **Consider HDU if:**
   - CBK >6mmol/L
   - HCO_3^- <5mmol/L
   - pH <7.0
   - GCS <12

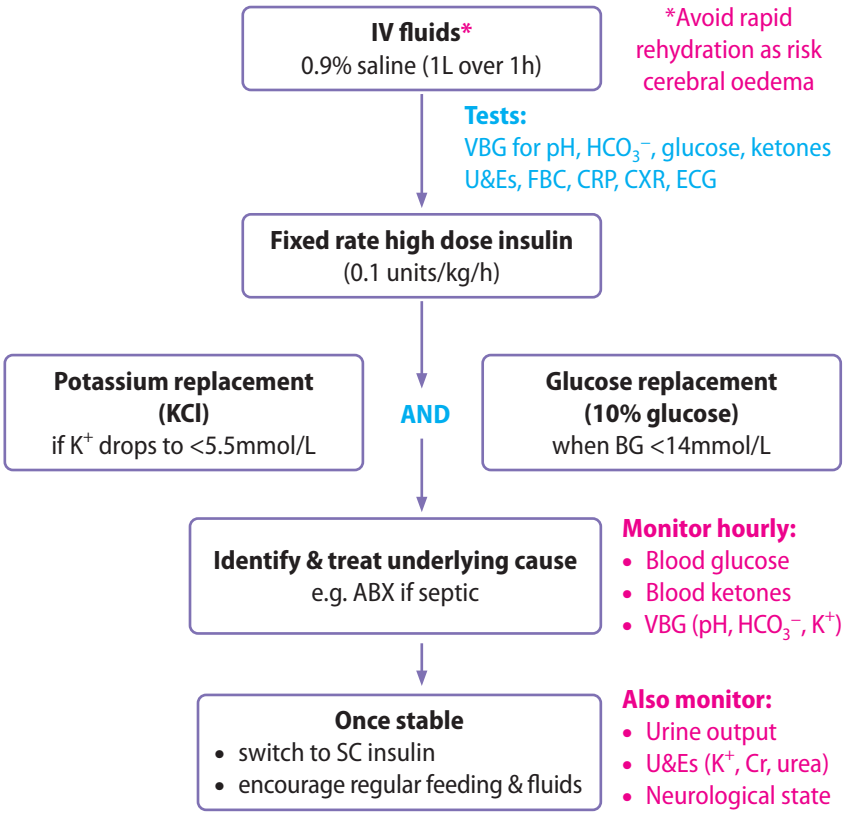


Fig. 17.2 DKA management in adults.

[1] Diabetes UK (2012) *The management of hyperosmolar hyperglycaemic state (HHS) in adults with diabetes*
[2] Diabetes UK (2021) *The management of diabetic ketoacidosis in adults*

Hypoglycaemia[3]

Hypoglycaemia = blood glucose <4mmol/L

CAUSES:

- Reduced oral intake
- Intense exercise
- Binge drinking
- Taking too much insulin (if diabetic)

SYMPTOMS:

- Irritable, anxious, confused
- Hungry or nauseous
- Shaky, dizzy, light-headed
- Seizures if severe

MANAGEMENT:

1. Oral rapid-acting glucose **or** IV glucose 100ml 20% / IM glucagon 1mg
2. Long-acting carbohydrate e.g. toast (once CBG >4mmol/L)
3. **Look for cause** (document, monitor and get specialist review if needed)

Hyperkalaemia

CAUSES:

- CKD / renal failure
- Drugs (ACEis/ARBs, NSAIDs, spironolactone, potassium supplements)
- Burns / trauma / tissue injuries
- Hormonal disorders e.g. Addison's

Hyperkalaemia = K^+ >5.2mmol/L
Severe hyperkalaemia = K^+ >6mmol/L

SYMPTOMS:

- Chest pain / palpitations
- Dizziness/weakness
- Abdominal pain / vomiting

INVESTIGATIONS:

- U&Es
- ECG – tented T waves, flattened P wave, shortened QT interval

MANAGEMENT:

1. 10ml 10% calcium gluconate
2. IV insulin + 25g glucose
3. Salbutamol nebs
4. Treat cause

[3] NICE BNF – *Treatment summary: Hypoglycaemia*

Neck & back pain

Red flags: need urgent MRI

- New onset in <20y or >55y
- Constant night pain
- Progressive motor weakness
- Thoracic back pain
- Saddle anaesthesia
- Bladder/bowel incontinence
- Hx of trauma/cancer
- Unexplained weight loss
- Fever
- Steroid use
- Recent infection

Differentials of neck/back pain

Structural	Inflammatory	Destructive	Metabolic	Referred pain
• Mechanical • Disc prolapse • Spinal stenosis • Spondylolisthesis	• Spondyloarthropathies • Sacroiliitis • Polymyalgia rheumatica	• Malignancy (1° or 2°) • Infection (discitis, osteomyelitis, TB)	• Osteoporosis • Osteomalacia • Paget's	• Major viscera • Uro-genitary • Aorta • Hip

Cervical radiculopathies

CERVICAL SPONDYLOSIS → cervical radiculopathy caused by age-related degenerative changes to spine

- Ageing causes **disc degeneration** (dehydration & flattening) = ↓ shock absorption
- Results in bony changes of vertebrae → **osteophyte development**
- Osteophytes **'pinch' nerve roots** as they leave spinal canal
- **May develop into myelopathy**

Symptoms:

- Pain in neck (radiating down arm as a 'dull ache'/'toothache') → **'brachial neuralgia'**
- Tingling/numbness in one dermatome
- ± Weakness in one arm

On examination:

- Pain reproduced with **lateral neck flexion towards affected side**
- **Motor signs:** modest upper muscle weakness ↳ 'Spurling sign'
- **Sensory signs:** reduced pin-prick sensation discrimination (in one dermatome)
- **LMN signs:** hyporeflexia, hypotonia

Diagnosis: usually clinical

→ **Neurological examination:** myotomes, dermatomes, reflexes
→ **MRI** if no improvement / considering surgery

Management: mostly self-limiting in 6–12w

1. **Conservative:**
   - Rest, physiotherapy, analgesia (NSAIDs or neuropathic – TCAs)
   - Hard collar for neck immobilisation
2. **Surgical:**
   - ACDF (anterior cervical discectomy & fusion) if persistent/worsening/severe Sx

CERVICAL DISC PROLAPSE = seen in those aged 30–40y; usually Hx of mild neck trauma

Symptoms:

- Pain in neck
- Tingling/numbness/paraesthesia in one dermatome of arm
- ± weakness in one arm

Management:

1. **Conservative:** rest, physiotherapy, analgesia (NSAIDs or neuropathic – TCAs)
2. **Surgical:** microdiscectomy

Investigation:

→ **Neurological examination:** myotomes, dermatomes, reflexes
→ **MRI** → recommended for cervical spine (assess need for surgery)

Radiculopathies = conditions where 'pinched' **nerve roots** cause **pain, paraesthesia, weakness** in a **dermatomal distribution (unilateral)**
→ **LMN signs** (hyporeflexia, hypotonia)

Myelopathies = conditions where compressed **spinal cord** causes **pain, paraesthesia, weakness bilaterally** + other neurological symptoms
→ **UMN signs** (hyperreflexia, hypertonia, spasticity)

Upper limb dermatomes	
C5	'Regimental **badge area**'
C6	**Thumb** & index finger
C7	**Middle finger**

Cervical myelopathy

CERVICAL CANAL STENOSIS → cord compression = more serious than cervical radiculopathy

Causes:

- **Age-related degeneration:** osteophyte formation & ligament hypertrophy
- **Disc bulging/herniation:** consider in younger patients

Affects middle-aged/elderly

Symptoms: gradual onset so late presentation & Dx

- **Gait abnormalities:** spastic & ataxic
- **Loss of fine motor skills** → difficulty fastening buttons, writing etc.
- Later = tingling in fingers (may be misdiagnosed as carpal tunnel syndrome) ± sphincter dysfunction

On examination:

- **Gait abnormalities:** spastic & ataxic
- Wasting on shoulder girdle muscles
- UMN signs: spasticity, clonus, +ve Hoffman's/Babinski, hyperreflexia, hypertonia

Investigations: X-ray & MRI

Management:

Surgical intervention: laminectomy → recommended as progressive deterioration

Hoffman's sign: flick middle finger & watch for reflexive movement of index/thumb

→ **positive in UMN pathology** e.g. spinal cord compression

Lumbar radiculopathies

LUMBAR DISC PROLAPSE = common in those aged 25–55y

Pathology:

- With age = increased risk of prolapse through defect in surrounding annulus fibrosus
- Results in **compression of nerve roots**

Lower limb dermatomes	
L4	**Inner shin**, below knee
L5	**Buttock**, lateral calf, big toe
S1	Post. thigh & **calf**, little toe

Symptoms: often onset during lifting/bending/twisting

- Stabbing lower back pain → radiating down the leg/buttock
- Numbness/tingling (in one leg)

Usually **posterolateral** herniation of disc, therefore compresses **nerve root below** i.e. herniated L5/S1 disc will compress S1 nerve root

On examination:

- Pain reproduced with **straight leg raise**
- **Motor signs:** modest lower muscle weakness (usually unilateral)
- **Sensory signs:** reduced pin-prick sensation discrimination (one dermatome)
- **LMN signs:** hyporeflexia, hypotonia
- May be **scoliosis** due to paravertebral muscle spasm

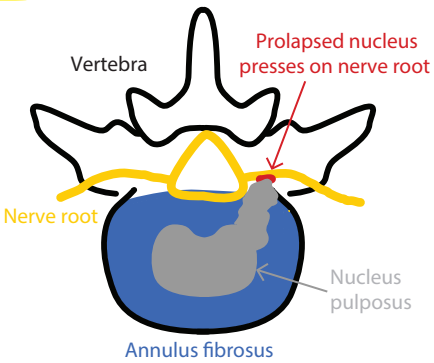


Fig. 18.9 Anatomy of disc prolapse.

Sciatica = pain along path of sciatic nerve (radiates down to toe) due to disc prolapse, stenosis or osteophytes affecting **nerve roots L4–S1**

Discs are named after vertebra above & below e.g. L4/5 or L3/4

Diagnosis: usually clinical

- **Neurological examination:** myotomes, dermatomes, reflexes
- **MRI** if no improvement / considering surgery

Management: 90% self-limiting in 6–12w

1. **Conservative:** rest, physiotherapy, analgesia (NSAIDs or neuropathic – TCAs)
2. **Surgical:** if no improvement in 6w
   - Nerve root block (under fluoroscopic guidance)
   - Microdiscectomy (remove piece of prolapsed disc)

Complications of surgery: Nerve damage, CSF leak, infection, haemorrhage, back pain

Musculoskeletal malignancy

***Urgent referral if red flags:**
1. 2ww for USS
2. **If USS worrying/unclear:** 2ww cancer pathway (MRI & biopsy)

If no worrying features:
observe or biopsy + histology

Soft tissue swellings

Benign	Malignant
• <5cm or slow-growing • Painless • Superficial to fascia • Well-circumscribed • Homogeneous appearance	• >5cm or rapid increase in size* • Painful* • Penetrates fascia (deep within muscle)* • Irregular border / not contained • Heterogeneous radiological appearance
e.g. lipoma, leiomyoma, schwannoma	e.g. sarcoma

Primary bone tumours

	Osteosarcoma (most common)	**Chondrosarcoma**	**Ewing's sarcoma**
Epidemiology	→ Children/adolescents (M>F) → 2nd peak in old age (in Paget's / post radiotherapy)	Adults >50y	Children/adolescents (rare)
Common sites	Distal femur, proximal tibia & humerus	**Flat bones:** pelvis, scapula, rib	**Diaphysis of bones**
Associated Sx	Lung mets	Pain, mechanical Sx, pathological fractures	Systemic upset (weight loss, fever, ↑ ESR)
Treatment	**1.** Chemotherapy **2.** Surgery (+ more chemo)	**Not sensitive to chemo** **1.** Surgery	**1.** Chemotherapy **2.** Surgery
5y survival	**No mets at Dx:** 60–65% **Mets at Dx:** 20%	**Low grade:** 90% **High grade:** 60%	50%

Multiple myeloma

→ malignancy of plasma cells of bone marrow: *proliferation of* ***one type of plasma cell*** *producing* ***one type of Ig***

SYMPTOMS:

- Bone pain (commonly backache)
- Fractures of long bones & vertebral collapse
- **Hypercalcaemia** (due to increased osteolytic activity)
- Anaemia, neutropenia, thrombocytopenia
- ± Renal impairment & recurrent infections

Average age: >70y

RF: Afro-Caribbean

INVESTIGATIONS:

- **FBC:** anaemia, ↓ WCC, ↑ ESR/CRP
- **U&Es, LFTs:** deranged renal function in 20%
- **Bone profile:** hypercalcaemia
- **Plasma electrophoresis:** ↑ monoclonal Igs
- **Urine microscopy:** Bence Jones proteins (monoclonal light chains)
- **X-rays:** punched-out osteolytic lesions ('pepper-pot')
- **Bone scintigraphy:** may have cold spots
- **Bone marrow biopsy:** >10% clonal plasma cells

Poor prognosis:
6y median survival

MANAGEMENT:

- **Supportive for complications** e.g. anaemia, renal failure
- **Localised disease:** radiotherapy
- **Widespread disease:** chemotherapy

Metastatic bone disease

→ *most common cause of destructive bone disease in adults*

→ **70% in axial skeleton (mostly in spinal column)**

SYMPTOMS:

- **Bone pain** (unremitting, dull ache → **night pain**)
- **Pathological fractures** (sudden worsening of pain)
- Hypercalcaemia
- Spinal cord compression
- **B Sx:** weight loss, night sweats

ON EXAMINATION:

- Bony tenderness
- Swelling
- Reduced ROM in joints
- Lymphadenopathy
- Anaemia
- Neurological deficit

NB: if no Hx of cancer, consider primary bone tumour

INVESTIGATIONS: find primary source

- **FBC:** anaemia, ↓WCC, ↑ESR/CRP
- **U&Es, LFTs, TFTs, bone profile:** hypercalcaemia
- **Tumour markers:** PSA for prostate cancer
- **Myeloma screen:** electrophoresis + urinalysis
- **X-rays:** lytic or sclerotic lesions
- **CT/MRI:** for primary tumour (chest, abdo, pelvis)
- **Bone scintigraphy:** hot spots
- **CXR**
- **CT-guided biopsy:** if uncertain of Dx

MANAGEMENT:

1. **Find primary source:** X-ray, CT, MRI, bone scan
2. **Supportive**
   - Pain relief
   - Bone protection: splints/bisphosphonates (assess fracture risk)
3. **Chemo/radiotherapy**
4. **Surgery:**
   - Stabilisation if risk of fracture in long bones / vertebrae
   - Arthroplasty if joint involvement

Common origins of bone mets:
PB-KTL ('lead kettle')

Prostate	Kidneys
Breast	Thyroid
	Lung

Differentials:

- Multiple myeloma
- Lymphoma
- Bone infection

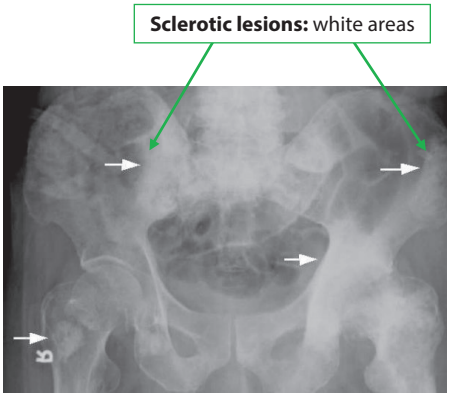


Fig. 19.1

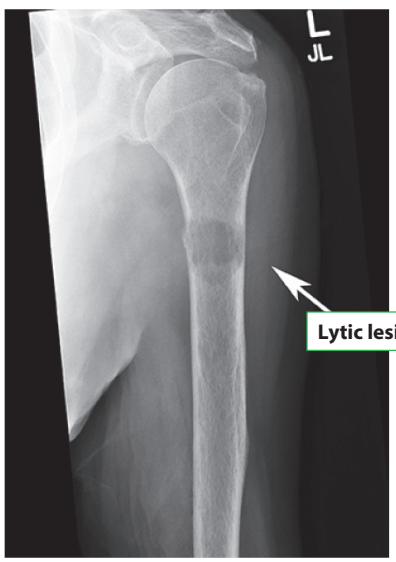


Fig. 19.2