

DNB Emergency Medicine



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question papers



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Volumes

3

Parts

27

Updated till

Oct 2026

Three volumes in this sample

- 1** Volume 1 Parts 1–8: Basic Sciences, Resuscitation and Cardiovascular
- 2** Volume 2 Parts 9–17: Respiratory, GI, Renal, Obstetrics, Paediatrics, Infections, Neurology, Toxicology and Environmental
- 3** Volume 3 Parts 18–27: Endocrine, Haematology, EENT, Skin, Orthopaedics, Psychiatry, Trauma, Procedures and Trials

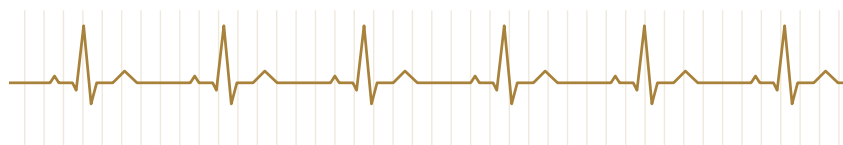
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DNB Emergency Medicine

EXIT EXAM

Solved Previous-Year Question Papers



Free sample from Volumes 1, 2 and 3

The full index of each volume, followed by its opening answers

Volume 1 Parts 1–8 · 216 answers

Volume 2 Parts 9–17 · 195 answers

Volume 3 Parts 18–27 · 216 answers

UPDATED TILL OCTOBER 2026

REVIEWED BY

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DNB (Emergency Medicine)

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DNB (Emergency Medicine)

Introduction

The DNB Emergency Medicine exit examination rewards answers that are accurate, well structured and pitched at the length the marks demand. This book is built around that examination: it gathers the previous-year theory questions and answers each one in the form an examiner expects to read.

What this book covers

The series runs to three volumes and 27 parts, with 627 answers drawn from question papers set between June 2020 and December 2025. Questions asked more than once, in the same words or in slightly different ones, are brought together under a single answer. Every version is printed verbatim at the head of that answer with its session, paper and question number, so you can see at a glance how a topic has been examined and how often.

How the answers are written

Each answer is written from standard textbooks of emergency medicine, anatomy, physiology and allied subjects, and from current guidelines. Sources are listed at the end of every answer with edition and page; figures carry their original figure numbers; and management statements taken from a guideline carry the guideline's name and grade. Where a point could not be found in the sources after a full search, the book says so plainly instead of filling the gap.

The three volumes

Volume	Parts	Answers	Subjects
Volume 1	Parts 1–8	216 answers	Basic Sciences, Resuscitation and Cardiovascular
Volume 2	Parts 9–17	195 answers	Respiratory, GI, Renal, Obstetrics, Paediatrics, Infections, Neurology, Toxicology and Environmental
Volume 3	Parts 18–27	216 answers	Endocrine, Haematology, EENT, Skin, Orthopaedics, Psychiatry, Trauma, Procedures and Trials

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How to read this book

Every answer follows the same layout, so you always know where to look.

Paediatric airway: differences from the adult airway

3×

Describe the anatomical differences between adult and paediatric airway. [6] — May 2024 · P-I · Q3(a)

Asked in: Dec 2020 P-I Q5; May 2024 P-I Q3(a); Oct 2024 P-I Q2(b)

Management basis: the guideline or book that drives management

PEARLS

★ Five or six lines to read the night before the paper

WORKED EXAMPLE

Doses and calculations, with every number recomputed

NOT COVERED IN SOURCE

What was searched, and the closest sourced material

Fig. 16.12 · Schematic (exam diagram) · Our own flowchart

C · High-yield (asked 3× or more)	Page
Topic asked in four sessions	4×

Question, versions and frequency

The heading names the topic and the badge shows how many times it has been asked (1×, 2×, 3×, 4×+). Below it, every version of the question is printed as set, with its marks and session; the *Asked in* line gathers the sessions and suggests the answer length the marks call for.

Management basis strip

Names the source behind the management section. Guideline-derived lines carry a tag such as [AHA 2025] with the guideline’s own grade.

Pearls

The points most worth remembering from the answer, set out for last-minute revision.

Worked example

Drug doses, scores and calculations worked through step by step.

Not covered in source

Marks a point that is absent from the source books and guidelines. The box records the search, and the closest sourced material follows, labelled as such.

Figures and diagrams

Figures are reproduced from the source books with their own numbers and pages. Drawings labelled *Schematic (exam diagram)* are simplified versions you can reproduce in the exam; flowcharts are drawn from the guideline or book text.

Using the index

Section A lists the parts in this volume, Section B lists every question with the sessions it was asked in, and Section C lists the high-yield topics asked three or more times. Section C is a good place to start revising.

DNB Emergency Medicine



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Volume 1

Basic Sciences, Resuscitation and
Cardiovascular

- ✓ Solved previous-year questions
- ✓ Reviewed by DNB (Emergency Medicine) doctors

Paper

1

Parts

8

Updated till

Oct 2026

Eight parts in this volume

- | | |
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| 1 Applied Anatomy | 5 EMS, Disaster, ED Systems & Education |
| 2 Applied Physiology | 6 Resuscitation, Airway & Shock |
| 3 Biostatistics & Research Methodology | 7 Analgesia, Sedation & Wound Care |
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Acute limb ischaemia	Dec '20 PII Q7(a,b,c,d); Apr '23 PII Q9(a)	364

C · High-yield (asked 3× or more)

Answer	Times asked	Page
Spinal cord syndromes; central vs anterior cord syndrome	4×	23
Cerebral circulation, regulation of CBF and autoregulation	4×	89
Physiology of temperature regulation: the hypothalamic thermostat and responses to heat and cold	4×	124
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Child abuse: types, signs and injuries that suggest abuse, evaluation and reporting	4×	171
Risk-stratification scores: HEART, EDACS, ACC/AHA, TIMI, GRACE	4×	333
Paediatric vs adult airway	3×	12
Brachial plexus: labelled diagram	3×	33
Buffers, respiratory and renal regulation of acid–base balance	3×	105
Physiology of haemostasis, the coagulation cascade and approach to the bleeding patient on warfarin	3×	120
Anatomical and physiological changes of pregnancy	3×	128
Physiology of pain perception: pathways and mechanism of transmission	3×	133
Oxygen-haemoglobin dissociation curve	3×	136
Forest plot: salient features	3×	157
Brain (brain-stem) death: definition, prerequisites, clinical tests, ancillary studies and certification	3×	164
Mass-casualty field triage: START, SALT, JumpSTART, colour categories	3×	192
Thromboelastography: normal thromboelastogram and interpretation of waveform components	3×	231
Anaphylaxis: ED treatment incl. vaccine-induced	3×	235
Metabolic alkalosis	3×	238
Antiarrhythmic drugs: classification and mechanisms of action	3×	267
Local anaesthetics: classification, mechanism, indications, contraindications, adverse effects	3×	307
Cardiac biomarkers and high-sensitivity troponin	3×	321
Cardiogenic shock: pathogenesis, evaluation, management	3×	327
Hypertensive AHF and SCAPE	3×	341
Ultrasound in acute dyspnoea: role, BLUE-type diagram and bedside identification of acute heart failure	3×	343

HOW TO USE THESE NOTES

- **One answer, many questions.** Repeats and near-repeats are merged; every merged question is printed verbatim at the head of its answer with an **Asked in** line and a frequency badge (1×, 2×, 3×, 4×+).
- **Management basis strip** (amber) names the guideline or book that drives management; every guideline-derived line carries a tag such as [AHA 2025] or [SSC 2026 R16] with the guideline's own grade.
- **Red box — NOT COVERED IN SOURCE:** the point is absent from the source books and saved guidelines after a full search; the box records the search, and the closest sourced material follows, labelled as such.
- **Figures** are real figures cropped from the source books with their own numbers and pages. Drawings labelled **Schematic (exam diagram)** are simplified diagrams built from a labelled spec, printed beside the original; flowcharts are our own drawings from the guideline or book text.
- **Green box** = pearls for last-minute revision; **blue box** = worked example (every number recomputed).

Part 1 · Applied Anatomy

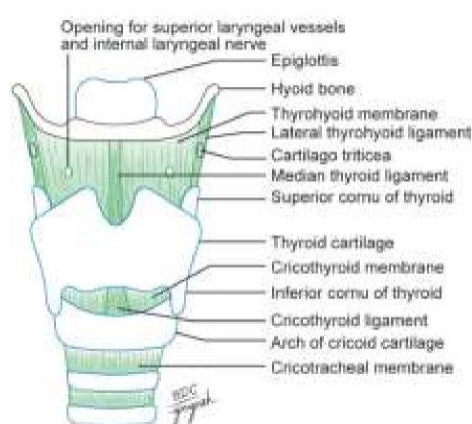
23 answers

Describe anatomy of larynx as relevant for endotracheal intubation. Draw appropriate diagram(s). [3+2] — Oct 2024 · P-I · Q2(a)

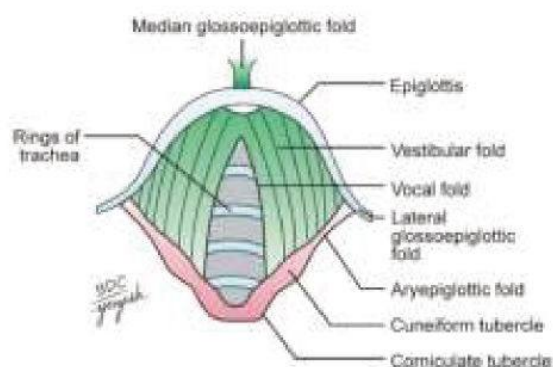
Asked in: Oct 2024 P-I Q2(a) · 3 + 2 → 4-5-mark length · anatomy question: original figures + schematic

Position and framework

- Larynx lies in the anterior midline of the neck from the root of the tongue to the trachea: in front of **C3–C6** in the adult male, higher (**C1–C4**) in children and adult females (BDC).
- Nine cartilages:** unpaired thyroid, cricoid, epiglottis; paired arytenoid, corniculate, cuneiform.
- Thyroid cartilage:** two laminae meeting at ~90° (male) or ~120° (female) to form the **laryngeal prominence**. External laryngeal manipulation (ELM/BURP) is applied here, not on the cricoid (RH).
- Cricoid cartilage:** ring with a narrow anterior arch and a broad posterior lamina. **Cricoid pressure** pushes this ring backwards to compress the oesophagus against the vertebrae (RH).
- Cricothyroid ligament** (thick anterior part of the conus elasticus) joins thyroid and cricoid. Emergency laryngotomy: needle in the midline of the cricothyroid membrane below the thyroid prominence (BDC).



BDC 10e vol. 3 Fig. 16.1, p. 258 — Cartilages and skeleton of the larynx (anterior view).



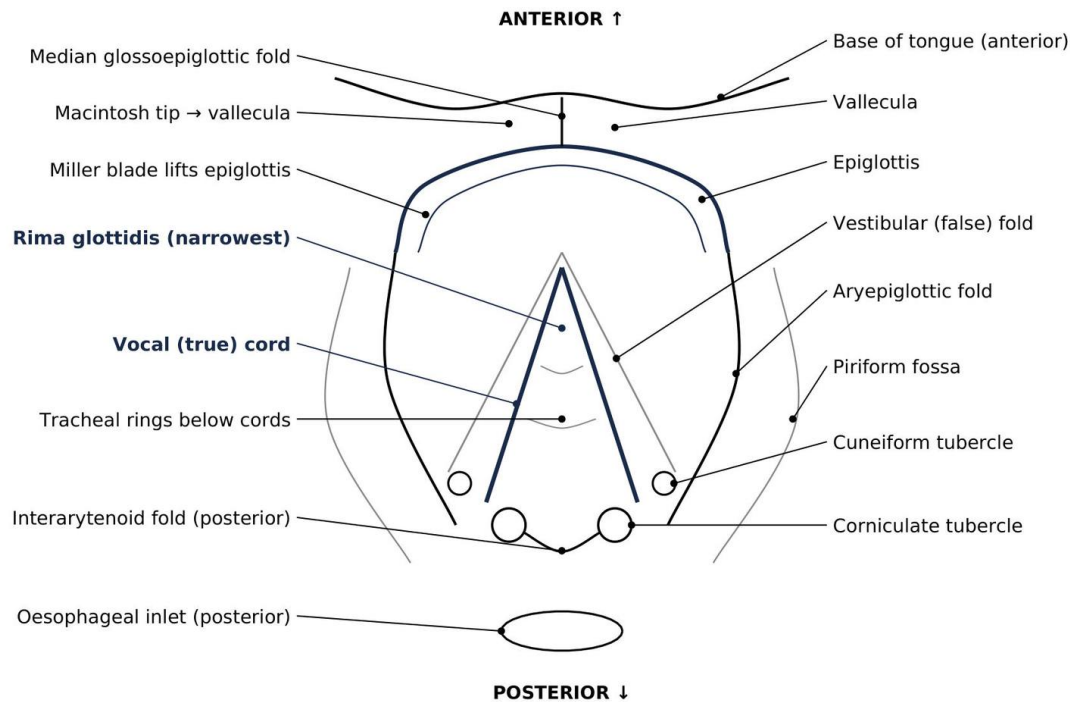
BDC 10e vol. 3 Fig. 16.12, p. 267 — Parts of the larynx seen by (indirect) laryngoscopy.

Epiglottis and vallecula: the laryngoscope blade

- Epiglottis:** leaf-shaped; its lower end is fixed to the thyroid angle by the thyroepiglottic ligament. Its anterior surface is joined to the tongue by the **median glossoepiglottic fold** and to the hyoid by the **hyoepiglottic ligament** (BDC).
- Vallecula** = space between the tongue base and the epiglottis. The **curved (Macintosh)** tip sits here and lifts the epiglottis indirectly through the median glossoepiglottic fold and hyoepiglottic ligament (RH).
- Straight (Miller)** blade goes under the epiglottis and lifts it directly (RH).
- Find the epiglottis first ("**epiglottoscopy**"); the laryngeal inlet lies just below it. A blade inserted too deep shows unfamiliar anatomy (oesophagus) and must be withdrawn (RH).

Inlet, folds and glottis

- Laryngeal inlet** slopes downward and backward. Boundaries: epiglottis (anterior), **interarytenoid fold** (posterior), **aryepiglottic folds** (lateral). The **piriform fossa** lies lateral to each aryepiglottic fold (BDC).
- Vestibular (false) folds** above; **vocal (true) folds** below, running from the thyroid angle to the vocal process of the arytenoid.
- Rima glottidis** (gap between the vocal folds) is the **narrowest part of the adult larynx**; foreign bodies lodge here (BDC).
- Cavity: vestibule (inlet → vestibular folds), ventricle, infraglottic part (vocal folds → lower border of cricoid).
- Posterior cartilages** (arytenoids with corniculate and cuneiform tubercles) are the key landmark. Seeing any arytenoid separates Cormack–Lehane grade II (usually easy) from grade III (only epiglottis seen) (RH).
- A tube railroaded over a bougie commonly catches on the **right arytenoid**: withdraw 2 cm and rotate 90° counterclockwise (RH).



Schematic (exam diagram): the larynx as seen at direct laryngoscopy, anterior at top. Based on BDC 10e vol. 3 Fig. 16.12, p. 267 and RH 8e Fig. 4.2B, p. 64; text BDC3 pp. 259–264, 268; RH pp. 70, 76.

Nerve supply: reflexes and topical anaesthesia

Table 1. Laryngeal innervation and its intubation relevance *Source: BDC 10e vol. 3 pp. 264, 267–268; RH 8e p. 81*

Nerve	Supplies	Relevance
Internal laryngeal (superior laryngeal branch)	Mucosa above the vocal folds (sensory)	Cough/gag reflex; topical anaesthesia of the supraglottis
Recurrent laryngeal	Mucosa below the vocal folds; all intrinsic muscles except cricothyroid	Transtracheal lidocaine (3–4 mL through the cricothyroid membrane) anaesthetises the trachea (RH)
External laryngeal	Cricothyroid only	Tension of the cords
Adductors (lateral cricoarytenoid, transverse arytenoid)	Close the rima	Laryngospasm = persistent adductor contraction; treat with jaw thrust or a paralytic (RH)

PEARLS

- ★ Macintosh → vallecula (indirect lift); Miller → under the epiglottis (direct lift).
- ★ Inlet: epiglottis in front, interarytenoid fold behind, aryepiglottic folds at the sides.
- ★ Adult narrowest point: rima glottidis.
- ★ ELM/BURP on the thyroid cartilage; cricoid pressure on the cricoid ring.
- ★ See the arytenoids before passing the tube.

Sources

- BDC 10e vol. 3, ch. 16 "Larynx", §Location, §Cartilages, §Laryngeal Ligaments and Membranes, §Cavity of Larynx, §Clinical Anatomy, §Muscles, §Nerve Supply, pp. 258–264, 267–268; Fig. 16.1, p. 258; Fig. 16.12, p. 267.
- RH 8e, ch. 4 "Tracheal Intubation", Fig. 4.2, p. 64; §Laryngoscope, Fig. 4.5, pp. 69–70; §Procedure and Technique of DL, p. 76; §ELM, p. 78; §Tracheal Tube Introducer, §Laryngospasm, pp. 80–81; ch. 3 "Basic Airway Management", §Cricoid Pressure, p. 53.

Paediatric airway: anatomical characteristics, differences from the adult airway (including the larynx) and clinical implications

3x

Anatomical characteristics of pediatric airway. [3] — Dec 2020 · P-I · Q5(a)

How does pediatric airway differ from adult airway? [4] — Dec 2020 · P-I · Q5(b)

Describe the anatomical differences between adult and paediatric airway with its clinical implications. [6] — May 2024 · P-I · Q3(a)

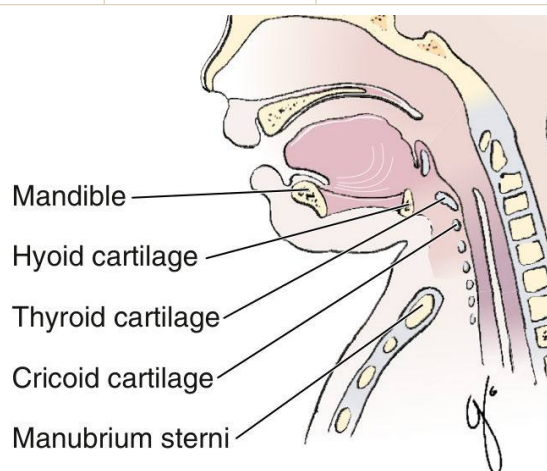
Describe the differences in laryngeal anatomy in adults and paediatric patients. [5] — Oct 2024 · P-I · Q2(b)

Asked in: Dec 2020 P-I Q5(a, b); May 2024 P-I Q3(a); Oct 2024 P-I Q2(b) · 3 + 4 + 6 + 5 (overlapping asks) → 6–7-mark length
· anatomy: original figure + schematic

Anatomical characteristics and differences

Table 2. Child versus adult airway and the clinical consequence *Source: RH 8e Table 4.2, p. 77; TIN 9e Table 113-1 and text, pp. 710–711*

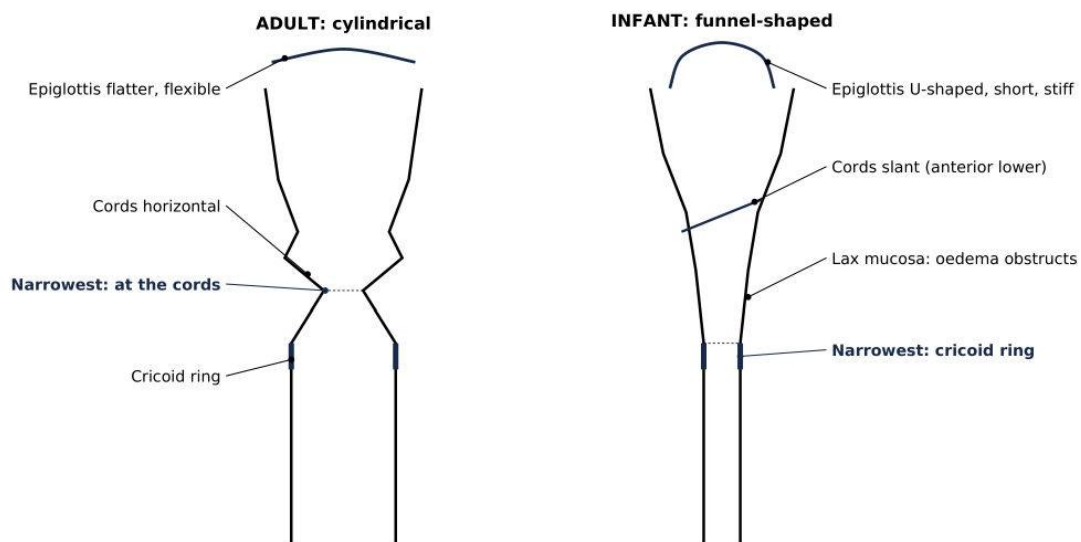
Feature	Child	Adult	Clinical implication
Head / occiput	Proportionately larger (to ~10 y)	Smaller	Child lies naturally sniffing; no towel under the occiput; infant may need a shoulder roll. Floppy head – assistant holds it
Tongue	Relatively larger; small mandible	Smaller	Obstructs when obtunded; hard to displace – jaw thrust, airway adjuncts, straight blade
Tonsils / adenoids	Large, friable	Not a problem	Avoid nasotracheal intubation (bleeding); tissue may plug the tube
Teeth	Easily knocked out	Stable	Risk of aspiration of teeth
Larynx level	C2–C3, appears anterior	C4–C6	Glottis harder to see; straight blade; video laryngoscopy may help
Epiglottis	U-shaped, shorter, stiffer, floppy	Flatter, flexible	Lift it directly with a straight blade
Vocal cords	Concave; anterior end lower (slant)	Horizontal	Tube catches on the anterior commissure – rotate 90° counterclockwise
Narrowest part	Cricoid ring (subglottis)	Between the vocal cords	Tube may pass the cords but not the cricoid → use next smaller size; watch cuff pressure
Trachea	Short	Longer	Main-stem intubation; depth = $0.5 \times \text{age (y)} + 12 \text{ cm}$
Cricothyroid membrane	Small	Larger	Open cricothyrotomy not possible <10 y; needle cricothyrotomy preferred
Lungs / FRC	Small FRC, high O ₂ consumption	Larger	Rapid desaturation; shorter safe apnoea time



RH 8e Fig. 4.14, p. 76 — Sagittal section of the neck of an infant: shorter neck, larynx more cephalad.

Differences in laryngeal anatomy (infant)

- Cavity **short and funnel-shaped**; size about **one-third** of the adult; lumen very narrow (internal diameter 3 mm up to 3 years vs 12 mm in the adult) (BDC).
- Position higher: **epiglottis at C2**, reaching C1 on elevation, so the infant can breathe through the nose while suckling (BDC).
- Cartilages **softer and more pliable**; thyroid cartilage shorter and broader; vocal cords only **4–4.5 mm** long.
- **Lax supraglottic and subglottic mucosa**: swelling quickly obstructs (BDC). The cricoid ring is also the site of croup swelling (TIN).
- Glottic opening, epiglottis and aryepiglottic folds are prominent, soft and mobile; under the blade the oesophageal inlet can mimic the cords (TIN).



Schematic (exam diagram): adult cylindrical vs infant funnel-shaped larynx. Based on RH 8e Table 4.2, p. 77 and Fig. 4.14, p. 76; BDC 10e vol. 3 p. 268; TIN 9e Table 113-1, p. 710.

Table 3. Laryngeal level — the sources differ

RH 8e (Table 4.2)	BDC 10e vol. 3 (p. 258)
Child: opposite C2–C3; adult: C4–C6	Adult male: C3–C6; children and adult females: C1–C4

Physiological differences and implications

- Higher metabolic rate and O₂ consumption with small FRC → **rapid desaturation**, particularly below SpO₂ 90%. Bag-mask ventilate before and during the apnoeic period (TIN).
- **Resistance**: a 25% fall in diameter (4 → 3 mm) halves the area and raises resistance by 200%; crying with turbulent flow raises work of breathing up to 32-fold. Keep the obstructed child calm (TIN).
- Gastric distension (swallowed air, bagging) splints the diaphragm: careful BMV, early oro/nasogastric tube (TIN).
- Larger extracellular fluid compartment: faster onset, shorter action and higher per-kg doses of RSI drugs (TIN).
- Children are far more prone to **laryngospasm**; a chest thrust may momentarily open the cords (RH).

Practical adjustments

- **Blades:** Miller 0 (premature), 1 (term infant), 2 (older child) (RH).
- **Tube size** (≥ 2 y): uncuffed = $\text{age}/4 + 4$; cuffed = $\text{age}/4 + 3$ mm ID; or little-finger nail width or a length-based tape (RH).
- Cuffed tubes are safe in infants and children and reduce tube changes; watch size and cuff pressure [AHA 2025 PALS, cuffed ETT synopsis].
- Position: align the tragus with the shoulder/clavicle; shoulder roll in infants (RH; TIN).

PEARLS

- ★ Child: big head, big tongue, high anterior larynx, floppy U-shaped epiglottis, funnel larynx narrowest at the cricoid.
- ★ Adult: cylindrical larynx narrowest at the cords.
- ★ Straight blade, no occiput towel, shoulder roll in infants.
- ★ Cuffed tube $\text{age}/4 + 3$; uncuffed $\text{age}/4 + 4$; depth $\text{age}/2 + 12$ cm.
- ★ Needle, not open, cricothyrotomy in young children (<10 y, TIN).

Sources

- RH 8e, ch. 4 "Tracheal Intubation", §Tracheal Tubes and Table 4.1, pp. 71–72; §Infants and Children, Fig. 4.14, p. 76; Table 4.2, p. 77; §Laryngospasm, p. 81.
- TIN 9e, ch. 113 "Intubation and Ventilation of Infants and Children", §Physiologic and Anatomic Characteristics, Table 113-1, pp. 710–711.
- BDC 10e vol. 3, ch. 16 "Larynx", §Location and Extent, §Size, p. 258; §Infant's Larynx, p. 268.
- AHA/AAP 2025 Guidelines for CPR and ECC, Part 8 PALS: Use of Cuffed Endotracheal Tubes for Intubation (synopsis).

Anatomy of the coronary circulation

1×

Anatomy of coronary circulation. [5] — Dec 2021 · P-I · Q8(a)

Asked in: Dec 2021 P-I Q8(a) · 4–5-mark length · anatomy question: original figure + schematic

Overview

- Two coronary arteries arise from the **ascending aorta** and run in the **coronary (atrioventricular) sulcus**.
- They fill during **diastole**; the left is larger and supplies more myocardium.
- They are **functional end arteries**: they anastomose, but one cannot compensate for the other after thrombosis.

Table 4. Right and left coronary arteries *Source: BDC 10e vol. 1 pp. 269–272*

Feature	Right coronary artery (RCA)	Left coronary artery (LCA)
Origin	Anterior aortic sinus	Left posterior aortic sinus
Course	Between pulmonary trunk and right auricle → right anterior coronary sulcus → round inferior border → right posterior sulcus → crux; ends by anastomosing with circumflex (60%)	Between pulmonary trunk and left auricle → divides into anterior interventricular (LAD) and circumflex ; circumflex runs round left border in the left coronary sulcus to the posterior sulcus
Branches	Atrial (SA nodal in 60%); right conus (annulus of Vieussens); anterior and posterior ventricular; right marginal; posterior interventricular (septal to posteroinferior 1/3, AV nodal in 60%)	LAD: anterior ventricular, diagonal , left conus, septal (anterior 2/3 septum). Circumflex: ventricular, atrial, SA nodal (40%), left marginal, posterior interventricular (10–20%), Kugel's artery
Territory	RA; most of RV; LV strip by posterior IV groove; posterior 1/3 septum; conducting system except part of left bundle branch	LA; most of LV; RV strip by anterior IV groove; anterior 2/3 septum; part of left bundle branch

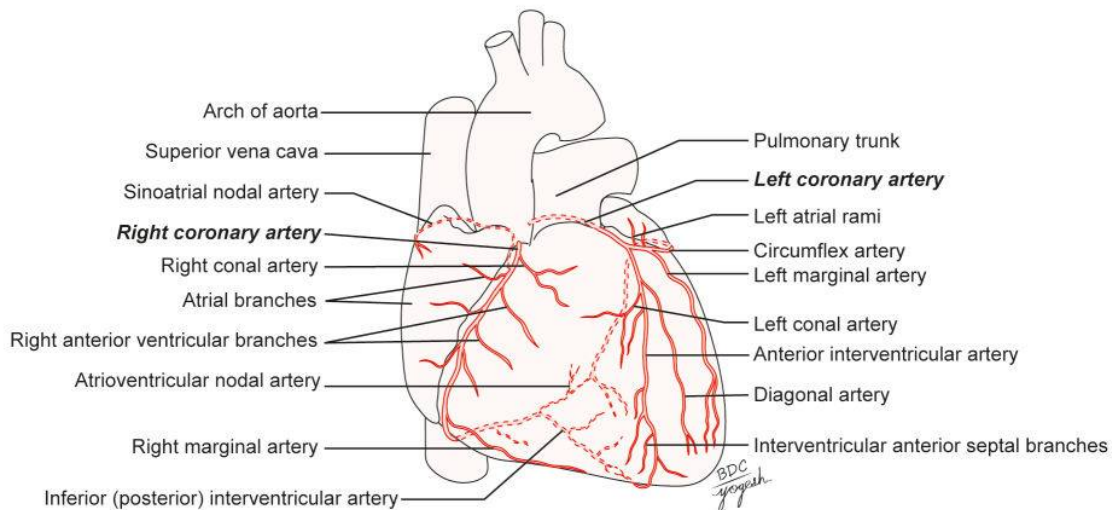
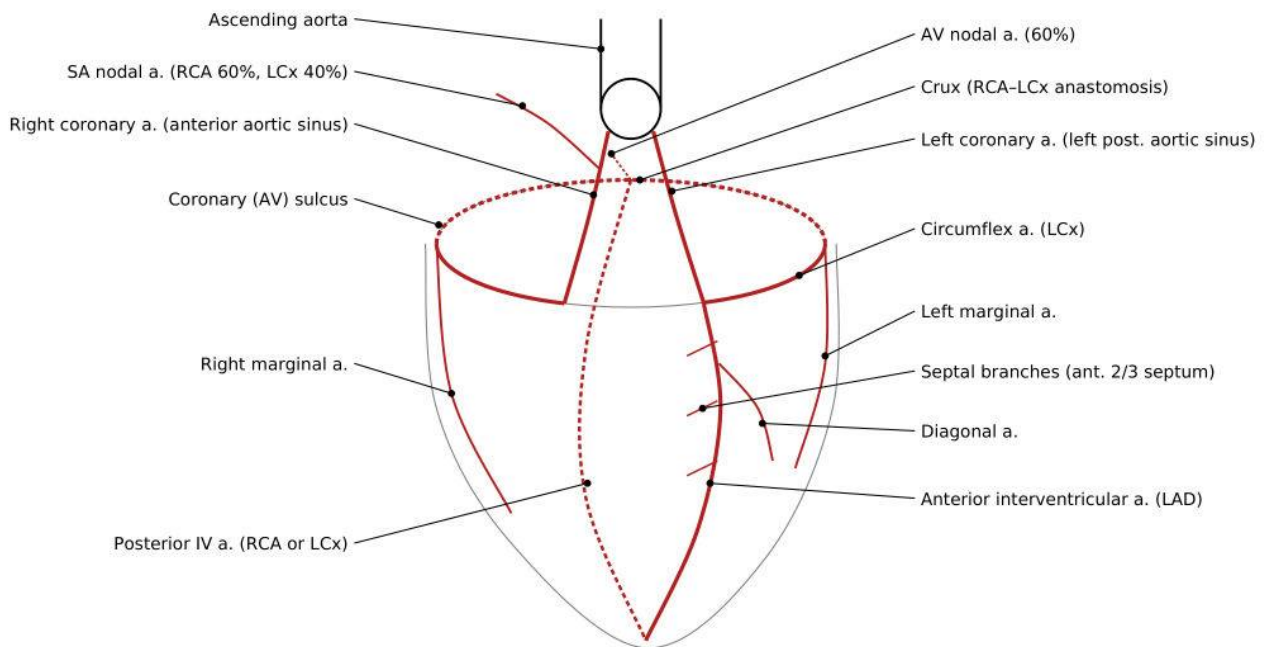


Fig. 18.28: Arterial supply of heart

BDC 10e vol. 1 Fig. 18.28, p. 271 — Arterial supply of the heart (sternocostal view).



Solid = anterior (sternocostal) surface; dashed = posterior (diaphragmatic) surface

Schematic (exam diagram): coronary arteries in the coronary sulcus and interventricular grooves. Based on BDC 10e vol. 1 Fig. 18.28, p. 271, Fig. 18.30, p. 272 and text pp. 269–272.

Dominance, anastomoses and venous drainage

- **Dominance** = the artery giving the **posterior interventricular** branch. Mostly the RCA (**right dominant**); in $\approx 10\%$ the circumflex gives it and the AV nodal artery (**left dominant**).
- **Anastomoses**: in the septum, Kugel's artery near the crux, annulus of Vieussens round the pulmonary trunk. Extracardiac links through vasa vasorum and internal thoracic, bronchial and pericardiophrenic arteries.
- **Veins**: coronary sinus (2–3 cm, left posterior coronary sulcus) → posterior wall of RA. It receives great, middle and small cardiac veins, posterior vein of LV and oblique vein of LA.
- **Anterior cardiac veins** and **venae cordis minimae** open directly into the chambers.

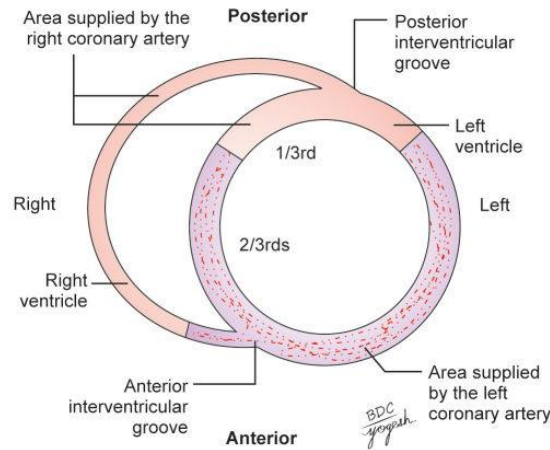


Fig. 18.30: Transverse section through the ventricles showing the areas supplied by the two coronary arteries

BDC 10e vol. 1 Fig. 18.30, p. 272 — Transverse section through the ventricles: areas supplied by the two coronary arteries.

Applied relevance in the ED

- Commonest occlusions: **LAD 40–50%**, RCA 30–40%, circumflex 15–20% (BDC).
- AV conduction is fed by the AV branch of the RCA and the septal perforators of the LAD, so **complete heart block** occurs in inferior and anterior AMI (TIN).
- The **posteromedial papillary muscle** has a single (usually RCA) supply and is the one that commonly ruptures (TIN).
- RV infarction accompanies inferior AMI because the RV is supplied mostly by the RCA (circumflex in left-dominant hearts) (TIN).

PEARLS

- ★ RCA ← anterior aortic sinus; LCA ← left posterior aortic sinus.
- ★ LAD = anterior 2/3 septum; PIV = posterior 1/3.
- ★ SA nodal artery: RCA 60%, circumflex 40%; AV nodal artery from the dominant artery.
- ★ Coronary sinus drains most veins into the RA; anterior cardiac veins and thebesian veins bypass it.

Sources

- BDC 10e (Competency Based), vol. 1 Thorax, ch. 18 "Pericardium and Heart", §Arteries Supplying the Heart, §Right and Left Coronary Artery, §Cardiac Dominance, §Anastomosis, §Clinical Anatomy, §Veins of the Heart, pp. 269–274; Fig. 18.28, p. 271; Fig. 18.30, p. 272.
- TIN 9e, ch. 49 "Acute Coronary Syndromes", §Anatomy, p. 334; §Bradydysrhythmias and AV block, p. 349; §Papillary muscle rupture, p. 350; §Right ventricular infarction, p. 351.

Visual pathway: diagram and lesion sites causing right homonymous hemianopia

1×

Draw, label and locate the lesion in visual pathway in right homonymous hemianopia. [5] — Jun 2022 · P-I · Q10(b)

Asked in: Jun 2022 P-I Q10(b) · 4–5-mark length · draw/label asked: original figure (BDC) + schematic

The visual pathway

1. **Retina:** rods and cones → bipolar cells (1st-order neurons) → **ganglion cells** (2nd order). The temporal field falls on the nasal hemiretina, and vice versa.
2. **Optic nerve:** ganglion-cell axons; strictly a CNS tract (oligodendrocyte myelin, no regeneration). It passes through the optic canal to the middle cranial fossa.
3. **Optic chiasma:** **nasal fibres** (including the nasal half of the macula) cross; temporal fibres stay on the same side.
4. **Optic tract:** winds round the cerebral peduncle and carries ipsilateral temporal + contralateral nasal fibres — i.e. the **opposite half of the visual field**. A few fibres go to the superior colliculus, pretectal nucleus (light reflex) and hypothalamus.
5. **Lateral geniculate body:** six layers (3rd-order neurons). Layers 2, 3, 5 receive ipsilateral fibres; layers 1, 4, 6 contralateral.

6. **Optic radiation** (geniculocalcarine tract) runs through the retrolentiform part of the internal capsule to the visual cortex.
7. **Visual cortex** (striate area 17) lines the calcarine fissure on the medial occipital lobe; the macula has a disproportionately large area near the occipital pole.

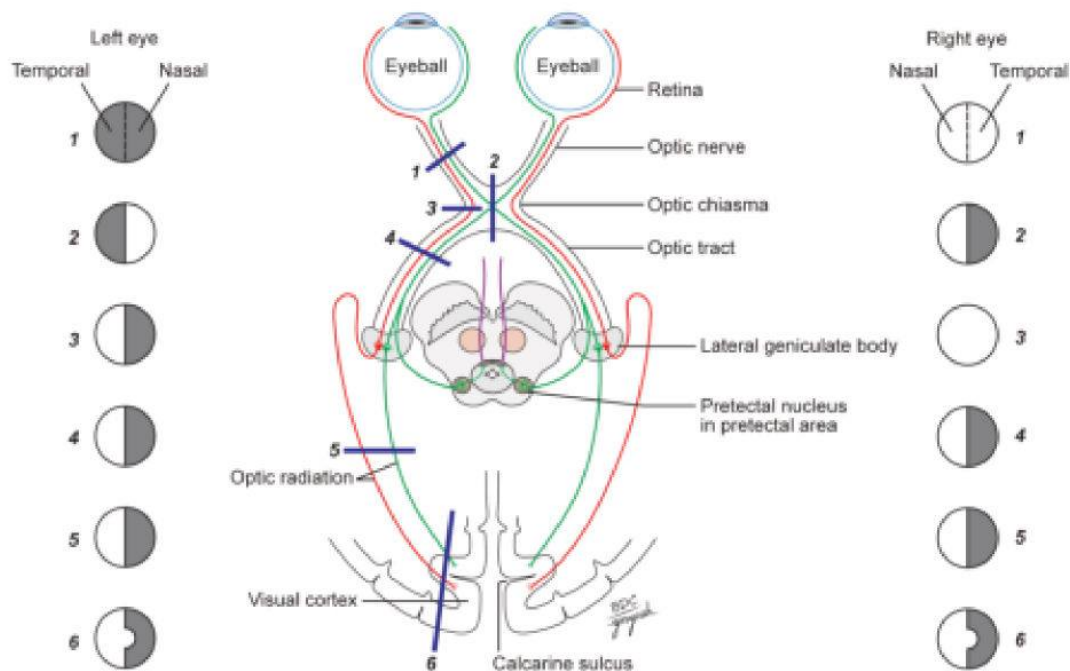
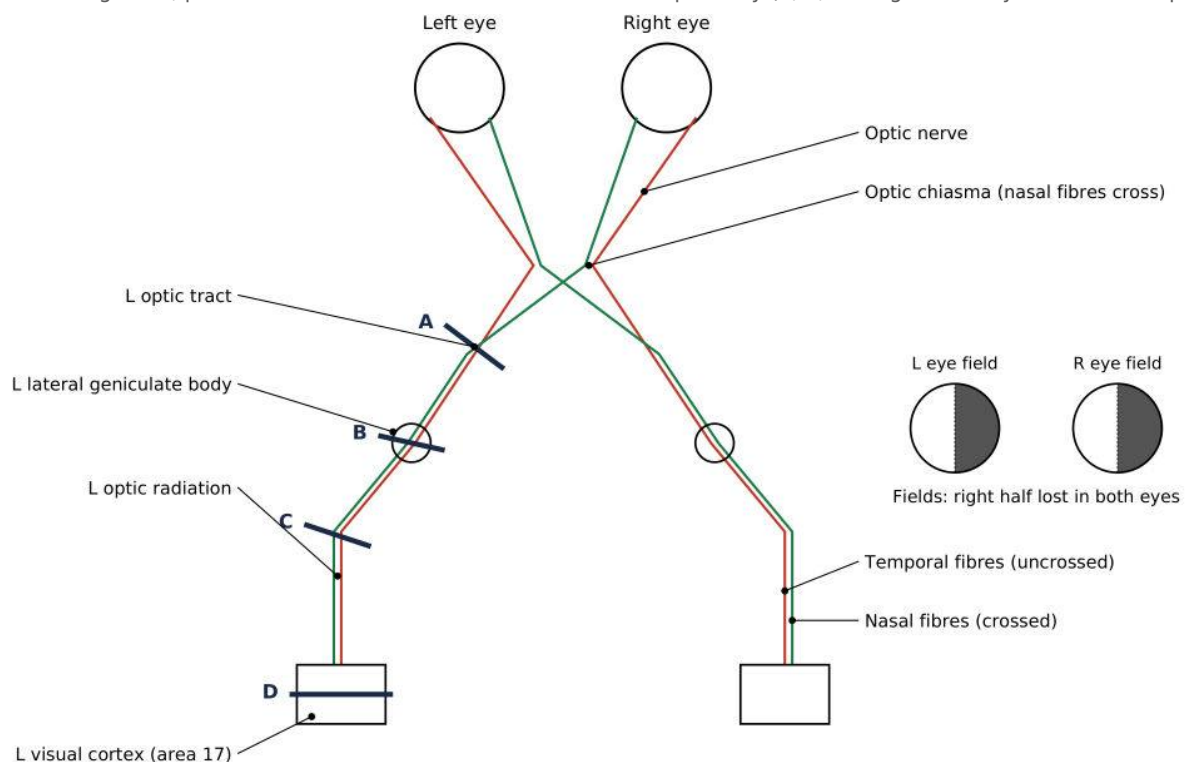


Fig. 6.10: Field defects associated with lesion of visual pathway: (1) Blindness of left eye; (2) Bitemporal hemianopia; (3) Left nasal hemianopia; (4) Right homonymous hemianopia with macular involvement; (5) Right homonymous hemianopia; (6) Right homonymous hemianopia with macular sparing

BDC 10e vol. 3 Fig. 6.10, p. 57 — Field defects with lesions of the visual pathway (4, 5, 6 = right homonymous hemianopia).



Lesion sites A-D (left side) → right homonymous hemianopia

Schematic (exam diagram): visual pathway with lesion sites A-D producing right homonymous hemianopia. Based on BDC 10e vol. 3 Fig. 6.10 (p. 57) and text pp. 54-55; GUY 15e Fig. 52.1 (p. 663).

Locating the lesion in right homonymous hemianopia

- **Homonymous** = the same half of the field lost in both eyes; right homonymous hemianopia = loss of the right half of both fields.

- The right half-field is carried by the **left** temporal and **right** nasal fibres, which travel together only behind the chiasma. The lesion is therefore **retrochiasmal on the left**.

Table 5. Lesion sites for right homonymous hemianopia *Source: BDC 10e vol. 3 pp. 55, 57, 164; TIN 9e p. 1124*

Site (left side)	Field defect	Typical cause
A. Left optic tract	Right homonymous hemianopia with macular involvement (BDC Fig. 6.10, lesion 4)	Destruction of the tract (BDC)
B. Left lateral geniculate body	Right homonymous hemianopia (loss of opposite half-field)	Destruction of the LGB (BDC)
C. Left optic radiation	Right homonymous hemianopia (lesion 5)	Middle cerebral artery stroke — hemianopia with hemiparesis and aphasia (TIN)
D. Left visual cortex	Right homonymous hemianopia with macular sparing (lesion 6)	Left posterior cerebral artery thrombosis (BDC; TIN)

- **For contrast:** optic nerve → blindness of that eye; central chiasma (pituitary tumour) → bitemporal hemianopia; lateral chiasma → nasal hemianopia (BDC).

PEARLS

- ★ Nasal fibres cross at the chiasma; each tract carries the opposite half-field.
- ★ Any homonymous defect = lesion behind the chiasma, on the opposite side.
- ★ Right HH: left tract, LGB, radiation or cortex.
- ★ Macular sparing points to an occipital cortex (PCA) lesion.

Sources

- BDC 10e (Competency Based), vol. 3 Brain-Neuroanatomy, ch. 6 "Cranial Nerves and Special Senses", §Visual Pathway, §Clinical Neuroanatomy, pp. 54–55; Fig. 6.10, p. 57; ch. 14, §Clinical Neuroanatomy (posterior cerebral artery), p. 164.
- GUY 15e, ch. 52 "The Eye: III. Central Neurophysiology of Vision", §Visual Pathways, §Lateral Geniculate Nucleus, §Primary Visual Cortex, pp. 663–664; Fig. 52.1, p. 663.
- TIN 9e, ch. 167 "Stroke Syndromes", §Middle Cerebral Artery Infarction, §Posterior Cerebral Artery Infarction, p. 1124.

Circle of Willis: labelled diagram

1×

Draw and label circle of Willis. [5] — Jun 2022 · P-I · Q4(a)

Asked in: Jun 2022 P-I Q4(a) · 4–5-mark length · draw/label asked: original figure (BDC) + schematic

Definition and position

- The **circulus arteriosus** is a **hexagonal arterial circle** at the base of the brain in the **interpeduncular fossa**.
- It is formed by the two internal carotid and the two vertebral (via basilar) systems — the anterior and posterior circulations (GUY; TIN).

Formation

Table 6. Components of the circle of Willis *Source: BDC 10e vol. 3 pp. 164–165*

Position	Vessel
Anterior	Anterior communicating artery joining the two anterior cerebral arteries
Anterolateral	Anterior cerebral arteries (smaller terminal branches of the ICA)
Lateral	Internal carotid arteries (terminal part)
Posterolateral	Posterior communicating arteries (ICA → PCA, across the crus cerebri)
Posterior	Posterior cerebral arteries (terminal branches of the basilar artery)

- The **middle cerebral artery**, the larger terminal branch of the ICA in line with it, does **not** form part of the circle.

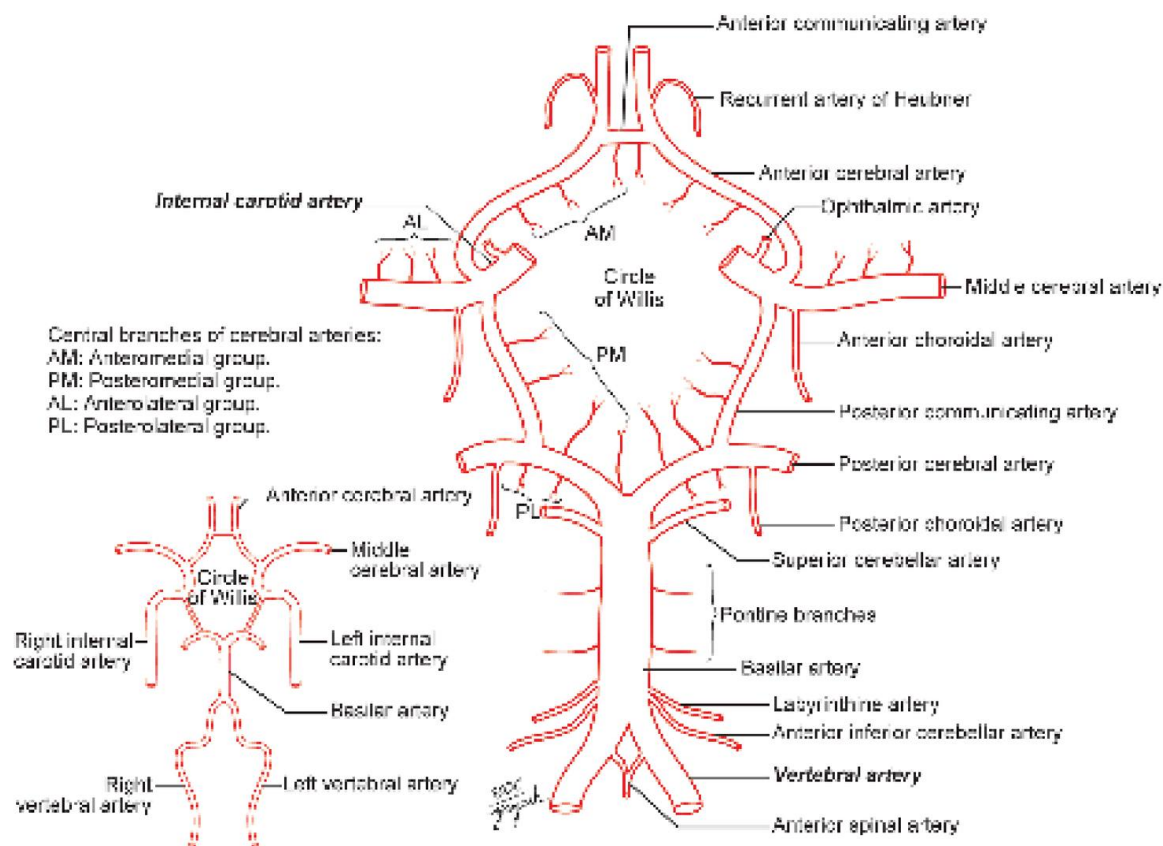
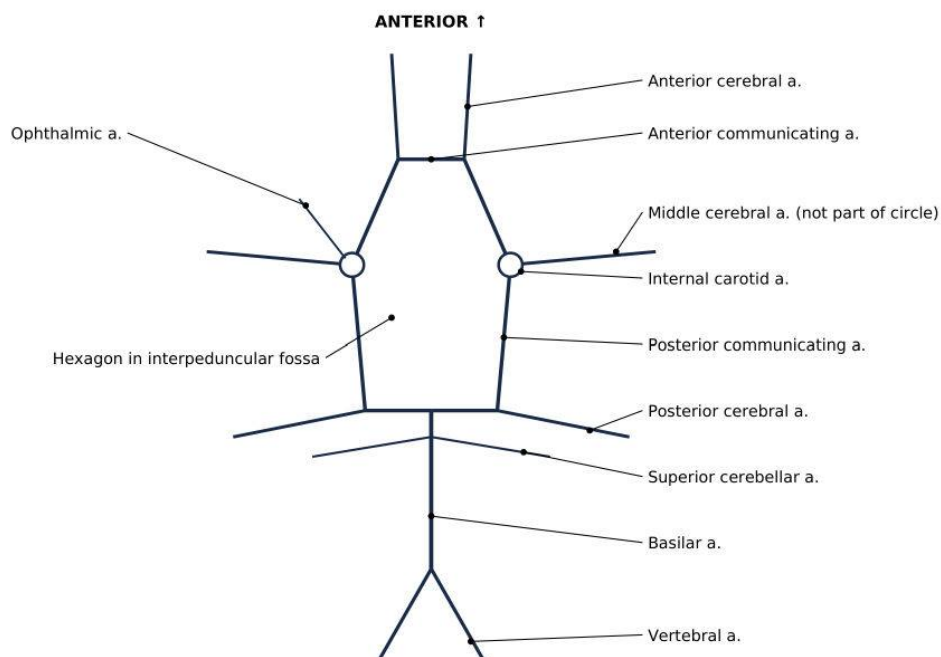


Fig. 14.10: Circle of Willis and the branches of arteries supplying the brain

BDC 10e vol. 3 Fig. 14.10, p. 166 — Circle of Willis and the branches of arteries supplying the brain.



Schematic (exam diagram): circle of Willis, inferior view. Based on BDC 10e vol. 3 Fig. 14.10 (p. 166), Fig. 14.9 (p. 165) and text pp. 164-167.

Branches

- **Cortical** — on the hemisphere surface; they anastomose, so a block gives a small infarct.
- **Central (perforating)** — anteromedial, anterolateral (lateral striate; the largest is **Charcot's artery of cerebral haemorrhage**), posteromedial and posterolateral groups. They supply thalamus, basal ganglia and internal capsule; as **end arteries**, a block gives a large infarct.
- **Choroidal** — anterior choroidal (ICA) and posterior choroidal (PCA).

Functional and clinical significance

- Equalises flow and provides a **collateral route**; normally there is hardly any mixing of right and left streams.
- If one ICA occludes, the other ICA may perfuse both ACAs; if the basilar occludes, each PCA may fill from its own ICA.
- **Berry (saccular) aneurysms** form at bifurcations of the circle; their rupture causes subarachnoid haemorrhage (TIN).
- **Territories**: ACA — medial hemisphere, leg; MCA — most of the lateral surface, face and arm, speech areas; PCA — occipital and visual cortex, medial temporal lobe; vertebrobasilar — brainstem, cerebellum, thalamus (TIN Table 167-1).

PEARLS

- ★ Hexagon: ACoA, ACA ×2, ICA ×2, PCoA ×2, PCA ×2 — the MCA is not part of it.
- ★ Lies in the interpeduncular fossa.
- ★ Central branches are end arteries; cortical branches anastomose.
- ★ Aneurysms sit at the bifurcations → SAH.

Sources

- BDC 10e (Competency Based), vol. 3 Brain-Neuroanatomy, ch. 14 "Blood Supply of Brain", §Internal Carotid Artery, §Circulus Arteriosus or Circle of Willis, §Branches, §Arterial Supply, §Clinical Neuroanatomy, pp. 164–167, 171–172; Fig. 14.9, p. 165; Fig. 14.10, p. 166; Table 14.1, p. 167.
- GUY 15e, ch. 62, §Cerebral Blood Flow, p. 791.
- TIN 9e, ch. 166 "Spontaneous Subarachnoid and Intracerebral Hemorrhage", §Pathophysiology, p. 1114; ch. 167 "Stroke Syndromes", Table 167-1, p. 1120.

Portosystemic anastomoses: labelled diagram and clinical implications

1×

Draw, label and describe the clinical implications of porto-systemic anastomosis. [6] — May 2024 · P-I · Q8(a)

Asked in: May 2024 P-I Q8(a) · 6-mark → 6–7-mark length · draw asked: original figure + schematic

Definition

- **Portosystemic (portocaval) anastomoses** are sites where tributaries of the **portal** vein communicate with tributaries of **systemic** veins.
- They form routes of **collateral circulation** when portal flow through the liver is obstructed.
- **Portal vein**: ≈8 cm; formed by the superior mesenteric and splenic veins behind the neck of the pancreas at L2 (the inferior mesenteric vein joins the splenic vein). It ends at the porta hepatis in right and left branches.
- Portal vein brings 80% of liver blood (hepatic artery 20%). Normal portal pressure ≈5–10 mm Hg; **portal hypertension** = above 10 mm Hg.

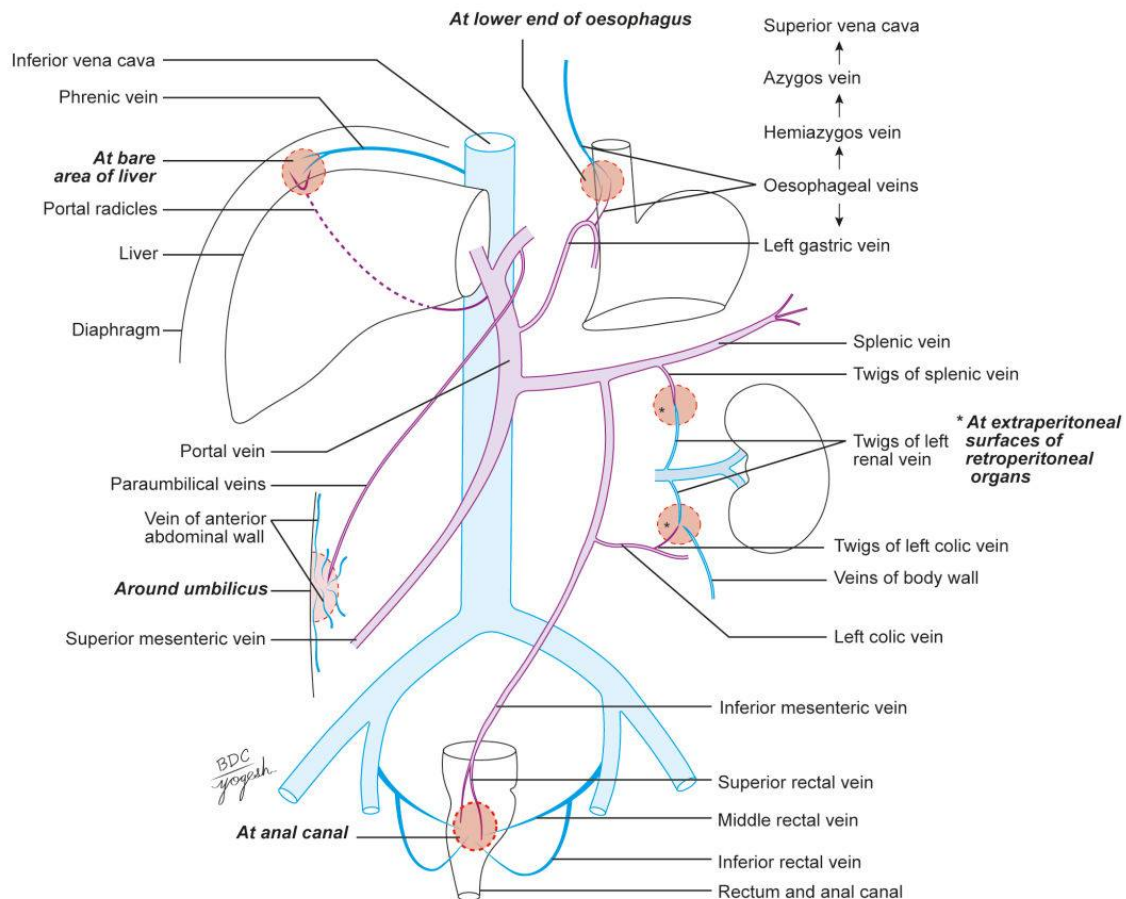


Fig. 21.11: Important sites of communication of portal and systemic veins

BDC 10e vol. 2 Fig. 21.11, p. 295 — Important sites of communication of portal and systemic veins.

PORTAL SIDE	SITE	SYSTEMIC SIDE	IN PORTAL HT
SVC / IVC			
Portal vein → liver			
Left gastric vein	At lower end of oesophagus	Oesophageal vv → hemiazygos → azygos	→ Oesophageal varices
Paraumbilical veins	At bare area of liver	Superior and inferior epigastric veins	→ Caput medusae
Superior rectal vein → IMV	At umbilicus	Middle and inferior rectal veins	→ Haemorrhoids
Portal radicles	At anal canal	Phrenic and intercostal veins	
Splenic and colonic veins	At bare area of liver	Left renal vein	
Left branch of portal vein	Retroperitoneum (extraperitoneal)	Inferior vena cava	
	Ligamentum venosum		

Portal vein = SMV + splenic vein (behind neck of pancreas, L2); IMV → splenic vein

Schematic (exam diagram): the six sites of portosystemic anastomosis with the portal and systemic veins at each, and the result in portal hypertension. Based on BDC 10e vol. 2 Fig. 21.11 (p. 295), Fig. 21.12 (p. 296) and Table 21.1 (p. 295).

Sites

Table 7. Sites of portosystemic anastomosis *Source: BDC 10e vol. 2 Table 21.1, p. 295; p. 294*

Site	Portal tributary	Systemic tributary	Clinical result
Lower (abdominal) end of oesophagus	Left gastric vein	Oesophageal veins → hemiazygos → azygos → SVC	Oesophageal varices ; rupture → haematemesis
Umbilicus	Paraumbilical veins (along ligamentum teres)	Superior epigastric, lateral thoracic; superficial and inferior epigastric; posterior intercostal, lumbar	Caput medusae
Anal canal	Superior rectal → inferior mesenteric vein	Middle and inferior rectal veins → IVC	Haemorrhoids (bleeding per rectum)
Bare area of liver	Portal radicles (central and sublobular veins)	Phrenic and intercostal veins	—
Extraperitoneal tissue of posterior abdominal wall	Splenic and colonic veins	Left renal vein (and veins of the body wall)	—
Ligamentum venosum (patent ductus venosus)	Left branch of portal vein	Inferior vena cava	Congenital anomalies

Clinical implications

- **Causes of portal hypertension:** cirrhosis (vascular bed obliterated), Banti's disease, portal vein thrombosis (BDC).
- **Effects:** congestive splenomegaly, ascites and opening of the collaterals — caput medusae (diagnostic), oesophageal varices, haemorrhoids (BDC).
- **Variceal haemorrhage:** varices cause 59% of upper GI bleeds in cirrhotics and carry high rebleeding and mortality; treat with octreotide, antibiotics, endoscopic banding, and balloon tamponade as a bridge (TIN).
- **Hepatic encephalopathy:** portal–systemic shunting lets ammonia made by colonic bacteria bypass the liver; protein loads and GI bleeding fuel it (TIN). Even reduced liver flow through a portocaval shunt raises blood ammonia (GUY).
- **Therapeutic shunts:** splenic, superior mesenteric or portal vein joined to the IVC or left renal vein (BDC); **TIPS** shunts portal blood to the IVC, lowering portal pressure and variceal bleeding but commonly precipitating encephalopathy (TIN).
- **Streamline flow:** SMV blood goes mainly to the right lobe (toxins, amoebic abscess in the right lobe); splenic and IMV blood to the left lobe (BDC).
- **Bedside:** spider naevi, caput medusae and other surface shunting are signs of chronic liver disease (TIN).

PEARLS

- ★ Six sites: oesophagus, umbilicus, anal canal, bare area of liver, retroperitoneum, ligamentum venosum.
- ★ Left gastric ↔ oesophageal (azygos) = varices; paraumbilical ↔ epigastric = caput medusae; superior ↔ middle/inferior rectal = haemorrhoids.
- ★ Portal hypertension >10 mm Hg (normal 5–10).
- ★ Shunting (natural, TIPS or surgical) → encephalopathy.

Sources

- BDC 10e (Competency Based), vol. 2 Abdomen and Pelvis, ch. 21 "Large Blood Vessels of the Gut", §Portal Vein, §Portosystemic Communications (Portocaval Anastomoses), §Clinical Anatomy, pp. 293–296; Fig. 21.11 and Table 21.1, p. 295; Fig. 21.12, p. 296.
- TIN 9e, ch. 75 "Upper Gastrointestinal Bleeding", §Esophageal and Gastric Varices, p. 495; ch. 80 "Hepatic Disorders", §Pathophysiology, p. 516; §Clinical Features, p. 517; §Hepatic Encephalopathy, p. 520.
- GUY 15e, ch. 71 "The Liver", §Protein Metabolism (urea formation), p. 892.

Spinal cord syndromes (incomplete cord injury); central cord versus anterior cord syndrome

4x+

Discuss spinal cord syndromes. [5] — Dec 2021 · P-II · Q10(b)

Discuss spinal cord syndromes. [5] — Jun 2022 · P-I · Q10(a)

Discuss the incomplete spinal cord syndrome. [5] — Oct 2023 · P-II · Q4(a)

Discuss central cord syndrome versus anterior cord syndrome. [5] — May 2024 · P-II · Q8(b)

Asked in: Dec 2021 P-II Q10(b); Jun 2022 P-I Q10(a); Oct 2023 P-II Q4(a); May 2024 P-II Q8(b) · 5 + 5 + 5 + 5 (overlapping; adds a comparison) → 6–7-mark length · anatomy figure + schematic

Basis — the three key tracts

- Incomplete SCI = some motor or sensory function below the level; produces recognisable syndromes from the tracts damaged.
- **Lateral corticospinal tract** (motor): ~90% of fibres cross in the lower medulla, so cord damage gives **ipsilateral** weakness, spasticity, hyperreflexia, Babinski.
- **Lateral spinothalamic tract** (pain, temperature): fibres ascend 1–2 segments, cross in the anterior white commissure, so loss is **contralateral**, starting 1–2 segments below.
- **Dorsal columns** (vibration, position, fine touch): ascend ipsilaterally, cross in the medulla → **ipsilateral** loss. Light touch travels in both systems, so is lost only if both are damaged.
- **Lamination:** cervical (arm) fibres lie medially, sacral (leg) fibres laterally — the basis of arm-predominant central cord syndrome.
- **Blood supply:** anterior spinal artery supplies the anterior and lateral horns and anterior/part of lateral white columns; posterior spinal arteries supply the dorsal columns.

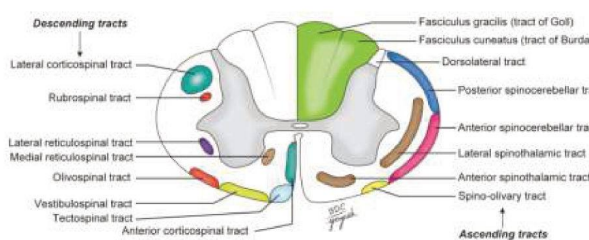
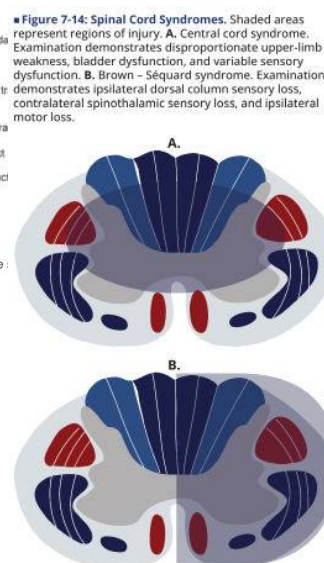
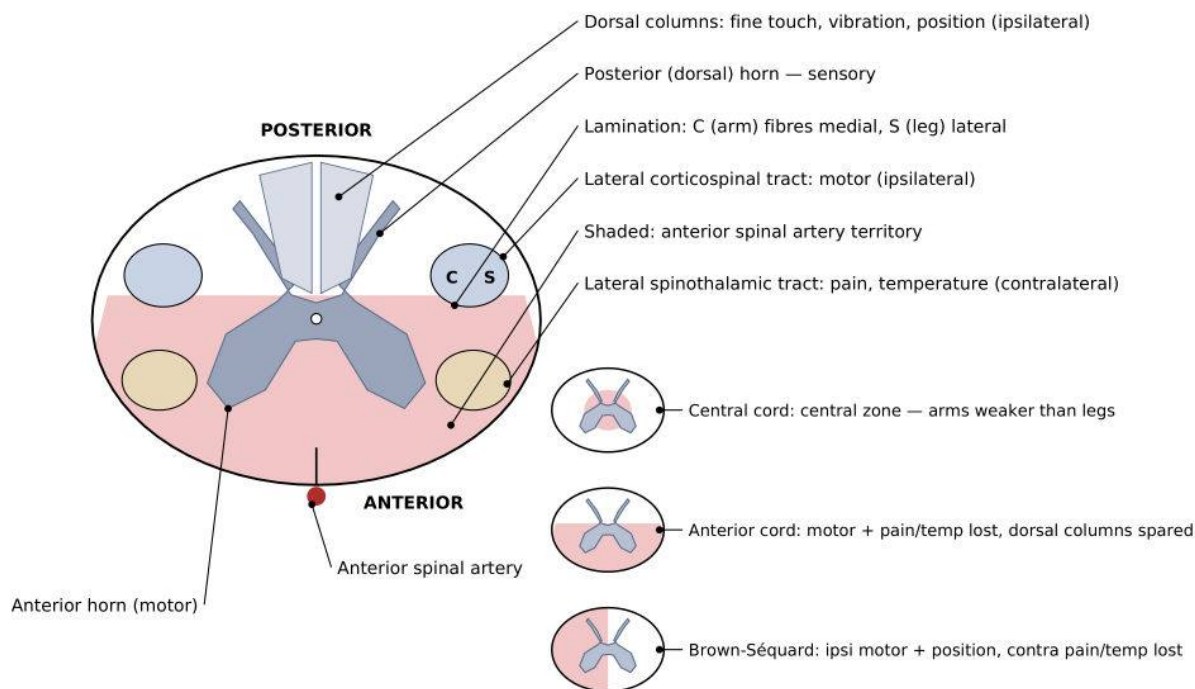


Fig. 5.14: Main descending (motor) tracts in the left half and ascending (sensory) tracts in the right half (transverse spinal cord at mid-cervical region)

BDC 10e vol. 3 Fig. 5.14, p. 37 — Descending (left) and ascending (right) tracts, transverse section at mid-cervical level.



ATLS 11e Figure 7-14, p. 110 — Spinal cord syndromes: (A) central cord, (B) Brown-Séquard.



Schematic (exam diagram): cervical cord cross-section — key tracts, lamination, anterior spinal artery territory, and lesion areas of the incomplete cord syndromes. Based on BDC 10e vol. 3 Figs 5.14 (p. 37) and 5.21 (p. 44), text pp. 37–47; TIN 9e Fig. 258-2 (p. 1707), pp. 1697, 1706, 1709–1710; ATLS 11e Fig. 7-14 (p. 110).

The syndromes

- **Central cord syndrome** (15–25% of SCI; commonest): hyperextension in an older patient with cervical spondylosis/stenosis, or disrupted cord blood flow; damages central grey and the medially placed arm fibres → weakness **arms > legs**, lesser pain/temperature loss (also arms more), position/vibration usually preserved; bladder control mostly kept. Prognosis good.
- **Anterior cord syndrome**: flexion injury (cord contusion, bone fragment, flexion teardrop), anterior spinal artery thrombosis, or an extrinsic decompressible mass → complete paralysis and loss of pain/temperature below the lesion with **dorsal columns spared**. Prognosis poor.
- **Brown-Séquard syndrome** (4%): hemisection, commonly penetrating (also disc, haematoma, fracture) → **ipsilateral** motor loss and loss of position/vibration; **contralateral** pain/temperature loss. Prognosis good.
- **Posterior cord syndrome**: rare after trauma, usually vascular.
- **Cauda equina syndrome** (root, not cord): saddle anaesthesia, bowel/bladder dysfunction, ↓ anal tone, variable leg weakness and ↓ reflexes, sciatica — emergent MRI.

Central cord versus anterior cord syndrome

Table 8. Central vs anterior cord syndrome *Source: TIN 9e pp. 1709–1710; ATLS 11e p. 110*

Feature	Central cord	Anterior cord
Typical patient / mechanism	Older, spondylotic neck; hyperextension (e.g., fall onto the face)	Flexion injury; anterior spinal artery thrombosis
Area damaged	Central grey and medial (arm) fibres of corticospinal and spinothalamic tracts	Anterior two tracts: corticospinal + spinothalamic
Motor	Arms weaker than legs	Complete paralysis below lesion
Pain/temperature	Some loss, arms more	Lost below lesion
Position/vibration	Usually preserved	Preserved
Bladder	Mostly controlled; impaired if severe	—
Prognosis	Good	Poor; look for a decompressible mass

ED points

- Document motor, sensory (including position/vibration) and sacral function early to follow change; spinal shock can mimic a complete lesion.
- Central cord syndrome may occur with a normal CT — MRI if deficits persist (three missed central cord injuries in a large CT study).
- Maintain SMR, MAP targets and avoid hypotension and hypoxia; spine surgery consultation.

PEARLS

- ★ Corticospinal and dorsal columns: ipsilateral; spinothalamic: contralateral.
- ★ Central cord: arms > legs, elderly hyperextension, good prognosis.
- ★ Anterior cord: paralysis + pain/temp loss, dorsal columns spared, poor prognosis.
- ★ Brown-Séquard: hemisection, usually penetrating.

Sources

• TIN 9e, ch. 258 "Spine Trauma", §Spinal Cord Lesions (tracts), pp. 1697, 1706; Fig. 258-2, p. 1707; §Incomplete Spinal Cord Syndromes, Table 258-3, pp. 1709–1710; §Cervical Spine MRI, p. 1711.
• ATLS 11e, ch. 7, §Anatomy (tracts), p. 103; §SCI Syndromes, p. 110; Fig. 7-14, p. 110.
• BDC 10e vol. 3, ch. 5 "Spinal Cord", §Tracts, pp. 37–38; lateral spinothalamic tract, p. 42; dorsal columns, pp. 43–44; Fig. 5.14, p. 37; Fig. 5.21, p. 44; Brown-Séquard, p. 46; blood supply, p. 47.

Anatomy of the neck relevant to trauma

1×

Anatomy of neck relevant to trauma. [5] — Dec 2022 · P-I · Q8(a)

Asked in: Dec 2022 P-I Q8(a) · 5 marks → 4–5-mark length · anatomy question: original figure + schematic

Platysma — the depth landmark

- Thin muscle in the superficial fascia, from the facial muscles to the thorax; it separates **superficial from deep** wounds (TIN; BDC).
- Wounds that do **not** breach the platysma are not life-threatening; wounds that penetrate it are significant and need evaluation (TIN; ATLS).

Triangles

- **Sternocleidomastoid** divides each side into **anterior** and **posterior** triangles (TIN Fig. 260-1).
- **Anterior triangle:** anterior border of SCM, inferior border of mandible, midline — contains most vital structures; subdivided by digastric and superior belly of omohyoid into submental, digastric, carotid and muscular triangles (TIN; BDC).
- **Posterior triangle:** posterior border of SCM, anterior border of trapezius, middle third of clavicle (TIN).

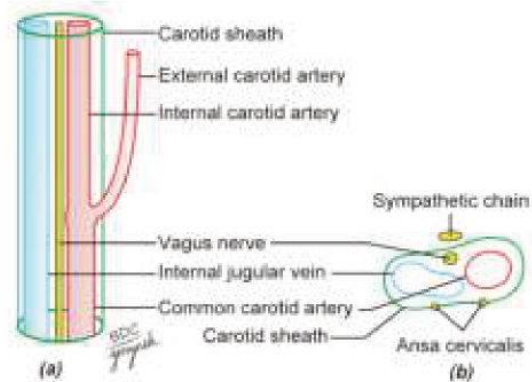
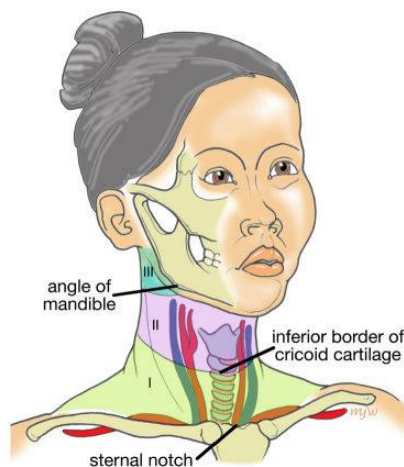
Zones of the anterior neck

Table 9. Zones of the neck and structures at risk *Source: ATLS 11e Table 22-1, p. 310; TIN 9e Table 260-1, p. 1722*

Zone	Boundaries	Structures at risk	Surgical access
I	Clavicles / sternal notch → cricoid cartilage (thoracic inlet)	Major thoracic vessels (subclavian, great vessels), proximal carotid and vertebral arteries, trachea, oesophagus, thoracic duct, lung apices, spinal cord, lower thyroid/parathyroid, thymus	Difficult — may need thoracotomy or sternotomy
II	Cricoid → angle of mandible	Common/internal/external carotids, vertebral arteries, jugular veins, larynx, pharynx, trachea, oesophagus, recurrent laryngeal nerves, spinal cord, upper thyroid	Open surgery; most often injured and most accessible
III	Angle of mandible → base of skull	Distal carotid and vertebral arteries, jugular veins, pharynx, cranial nerves IX–XII, sympathetic trunk	Difficult with open surgery

■ **Figure 22-3: Anatomic Zones of the Anterior Neck.**

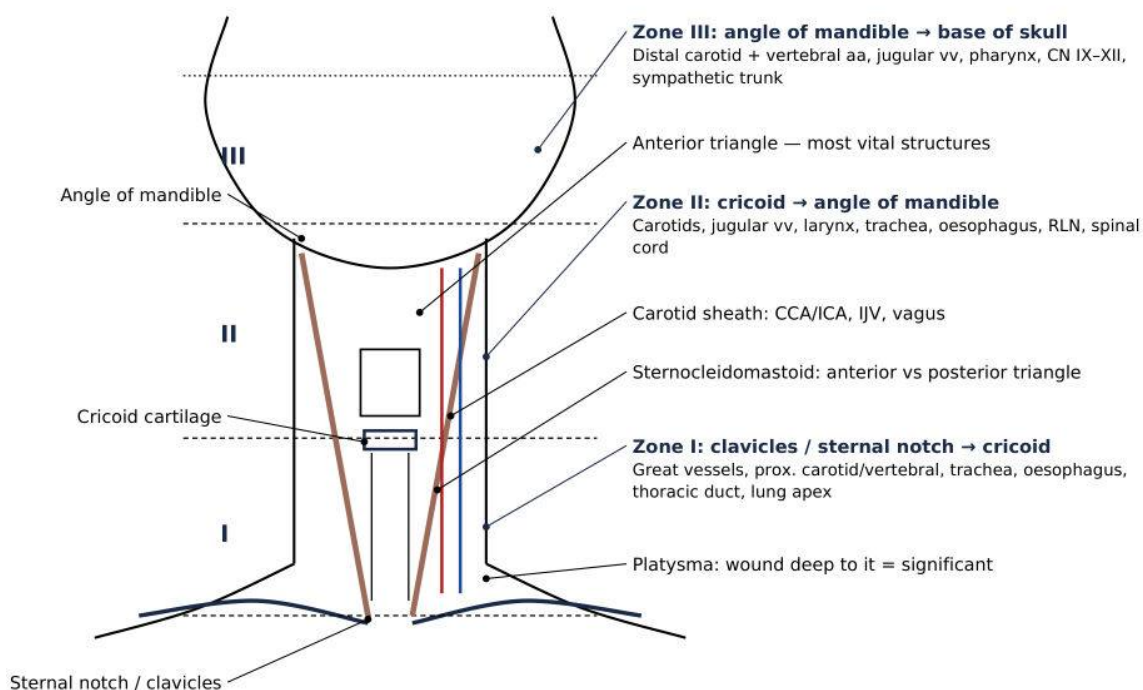
Zone I extends from the clavicles or sternal notch to the cricoid cartilage. Zone II extends from the angle of the mandible to the cricoid cartilage and Zone 3 extends above the angle of the mandible. Note the rich blood supply of the area.



Figs 3.10a and b: Carotid sheath with its contents: (a) Surface view and (b) section view

BDC 10e vol. 3 Figs 3.10a, b, p. 80 — Carotid sheath and its contents.

ATLS 11e Figure 22-3, p. 309 — Anatomic zones of the anterior neck.



Schematic (exam diagram): zones, triangles and key structures of the neck, frontal view. Based on ATLS 11e Fig. 22-3 and Table 22-1 (pp. 309-310); TIN 9e Figs 260-1, 260-2 and Table 260-1 (p. 1722); BDC 10e vol. 3 Fig. 3.10, p. 80.

Fascial layers

- **Deep cervical fascia:** investing layer (encloses SCM and trapezius; roofs both triangles), pretracheal, prevertebral, and the **carotid sheath** (common/internal carotid, internal jugular vein, vagus; formed by pretracheal fascia in front and prevertebral behind) (BDC).
- Fascial compartments may **confine a haematoma** and prevent exsanguination — but an expanding haematoma can compress the airway; the layers also carry infection into the **mediastinum** (TIN). Retropharyngeal and “danger” spaces extend into the posterior mediastinum (BDC).

Trauma relevance

- A wound in one zone may track into another zone or cavity — nearly half traverse several zones; modern evaluation (CTA) treats the neck as one unit and is based on physiology and hard/soft signs (TIN; ATLS).
- Penetrating spine injury rarely needs a collar, which can hide wounds and haematomas (ATLS).

PEARLS

- ★ Platysma breached = significant neck wound.
- ★ Zone I: clavicle–cricoid; II: cricoid–angle of mandible; III: angle–skull base.
- ★ Zone II is most injured and easiest to explore; I and III are hard to reach.
- ★ Carotid sheath: carotid artery, IJV, vagus.

Sources

- ATLS 11e, ch. 22 "Penetrating Trauma", §Penetrating Neck Trauma, Fig. 22-3, Table 22-1, pp. 309–310.
- TIN 9e, ch. 260 "Trauma to the Neck", §Anatomy, Figs 260-1 to 260-3, Table 260-1, pp. 1722–1723; §Selective Management (zones), p. 1725.
- BDC 10e (Competency Based), vol. 3 Head and Neck, ch. 3 "Side of the Neck", §Superficial Fascia, §Deep Cervical Fascia, §Carotid Sheath, pp. 76–80, Figs 3.10a, b; ch. 4 "Anterior Region of the Neck", §Anterior Triangle, p. 93.

Intrinsic muscles of the hand: anatomy, nerve supply, actions and testing

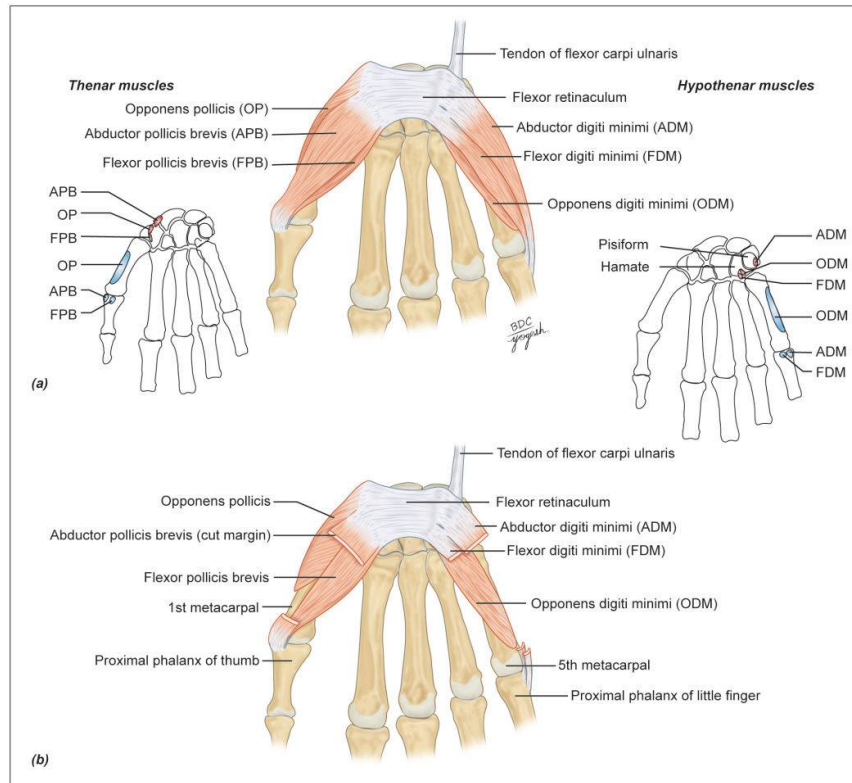
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Anatomy of intrinsic muscles of the hand. [5] — Dec 2021 · P-I · Q5(a)

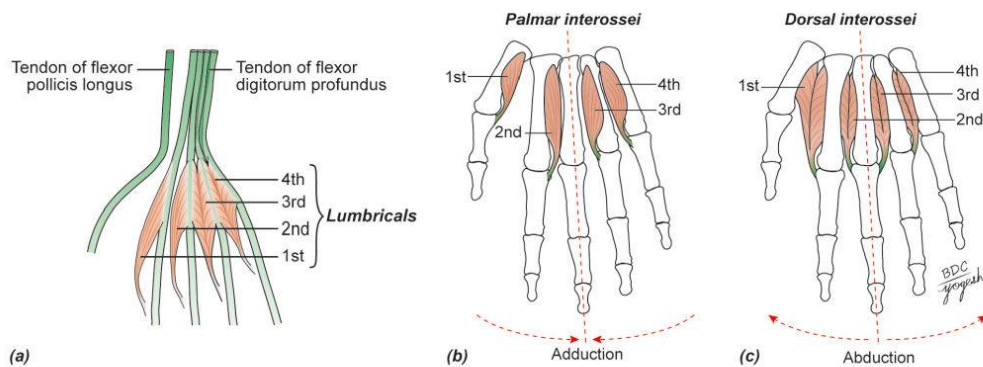
Asked in: Dec 2021 P-I Q5(a) · 4–5-mark length · anatomy question: original figure + schematic

Definition and groups

- Intrinsic muscles have both **origin and insertion within the hand**; they adjust the hand during grip and perform fine, skilled movements.
- **20 muscles** (BDC): thenar 3, adductor pollicis 1, hypothenar 3 + palmaris brevis, lumbricals 4, palmar interossei 4, dorsal interossei 4.



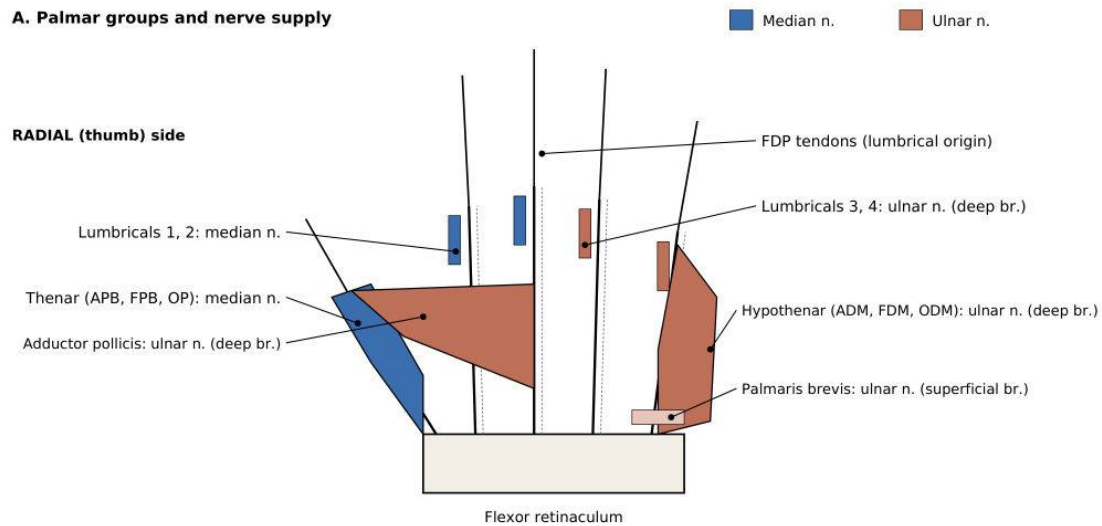
BDC 10e vol. 1 Plate 9.11, p. 118 — Thenar and hypothenar muscles.



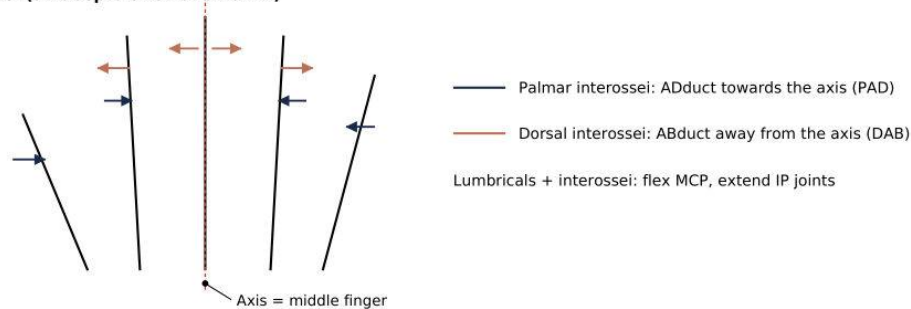
Figs 9.20a to c: Lumbricals and interossei muscles

BDC 10e vol. 1 Figs 9.20a-c, p. 117 — Lumbricals and interossei.

A. Palmar groups and nerve supply



B. Interossei (all deep branch of ulnar n.)



Schematic (exam diagram): intrinsic muscles of the right hand with nerve supply (A) and interosseous actions about the middle finger (B). Based on BDC 10e vol. 1 Plate 9.11, p. 118; Figs 9.20a-c, p. 117; Tables 9.5-9.6, pp. 116-117.

Attachments, nerve supply and actions

Table 10. Intrinsic muscles of the hand *Source: BDC 10e vol. 1 Tables 9.5, 9.6, pp. 116-117*

Muscle	Origin → insertion	Nerve	Action
Abductor pollicis brevis	Scaphoid tubercle, trapezium crest, flexor retinaculum → lateral base of thumb proximal phalanx	Median	Abducts thumb
Flexor pollicis brevis	Trapezium crest, capitate, retinaculum → lateral base of thumb proximal phalanx	Median	Flexes thumb MCP
Opponens pollicis	Trapezium crest, retinaculum → lateral half of 1st metacarpal	Median	Opposition
Adductor pollicis	Oblique head: capitate, bases of 2nd-3rd MC; transverse head: 3rd MC shaft → medial base of thumb proximal phalanx	Ulnar (deep br.)	Adducts thumb
Palmaris brevis	Flexor retinaculum → skin of medial palm	Ulnar (superficial br.)	Wrinkles skin, improves grip
Abductor / flexor / opponens digiti minimi	Pisiform; hook of hamate; retinaculum → base of little-finger proximal phalanx / 5th MC	Ulnar (deep br.)	Abduct, flex little finger; cup the palm
Lumbricals (4)	FDP tendons → radial side of MCP, into dorsal digital expansion	1, 2 median; 3, 4 ulnar	Flex MCP, extend IP
Palmar interossei	Metacarpal shafts → dorsal digital expansion	Ulnar (deep br.)	Adduct towards the middle finger (PAD)
Dorsal interossei (4)	Adjacent sides of two metacarpals (bipennate) → dorsal digital expansion	Ulnar (deep br.)	Abduct from the middle finger (DAB)

- Thumb movements occur at right angles to the other digits because the 1st metacarpal is rotated medially through 90°; **opposition** = flexion + medial rotation.

- Interossei and lumbricals flex the MCP and extend the IP joints through the dorsal digital expansion (lumbrical–interosseous complex).

Table 11. Source disagreement on the number of palmar interossei

BDC 10e (anatomy lead)	TIN 9e
Four palmar interossei; the 1st arises from the base of the 1st metacarpal and inserts on the thumb	Seven interossei in all: three palmar and four dorsal

Clinical testing

- **Pen test** (abductor pollicis brevis, median): palm up, touch a pen held above the palm with the thumb.
- **Opponens pollicis**: touch the tip of the thumb to the bases of the other digits.
- **Dorsal interossei**: spread fingers against resistance; feel the 1st dorsal interosseous.
- **Palmar interossei and adductor pollicis**: grip paper between fingers or between thumb and index finger.
Froment's sign: the thumb IP flexes (FPL, median) to compensate for a weak adductor pollicis (ulnar).
- **Lumbricals and interossei**: flex MCP joints against resistance.
- **ED relevance**: the thenar motor (recurrent) branch of the median nerve is tested by resisted palmar abduction of the thumb; ulnar nerve by finger abduction (TIN).

PEARLS

- ★ Median: three thenar muscles + lumbricals 1–2; ulnar (deep branch): all the others; palmaris brevis by the superficial branch.
- ★ PAD and DAB about the middle finger.
- ★ Froment's sign = weak adductor pollicis (ulnar).
- ★ Lumbricals arise from FDP tendons and have no bony origin.

Sources

- BDC 10e (Competency Based), vol. 1 Upper Limb, ch. 9 "Forearm and Hand", §Intrinsic Muscles of Hand, p. 115; Tables 9.5, 9.6, pp. 116–117; Figs 9.20a–c, p. 117; Plate 9.11, p. 118; §Actions of Thenar Muscles, §Actions of Dorsal Interossei, §Testing of Some Intrinsic Muscles, pp. 118–121.
- TIN 9e, ch. 268 "Injuries to the Hand and Digits", §Intrinsic Hand Muscles, pp. 1783–1784; §Nerve Testing, p. 1787.

Anatomy of the wrist (radiocarpal) joint

1×

Anatomy of wrist joint. [4] — Jun 2020 · P-II · Q5(a)

Asked in: Jun 2020 P-II Q5(a) · 4–5-mark length · anatomy question: original figure + schematic

Type and articular surfaces

- **Synovial joint, ellipsoid variety (biaxial); compound**, as more than two bones take part.
- **Proximal**: inferior surface of the lower end of the radius + **articular disc** of the inferior radioulnar joint (the ulna does not articulate directly).
- **Distal**: scaphoid, lunate, triquetral. The **pisiform** is not part of the joint; it is a sesamoid acting as a pulley for FCU.

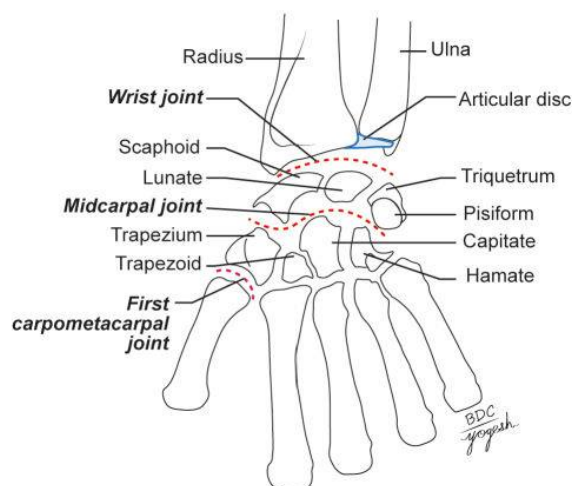
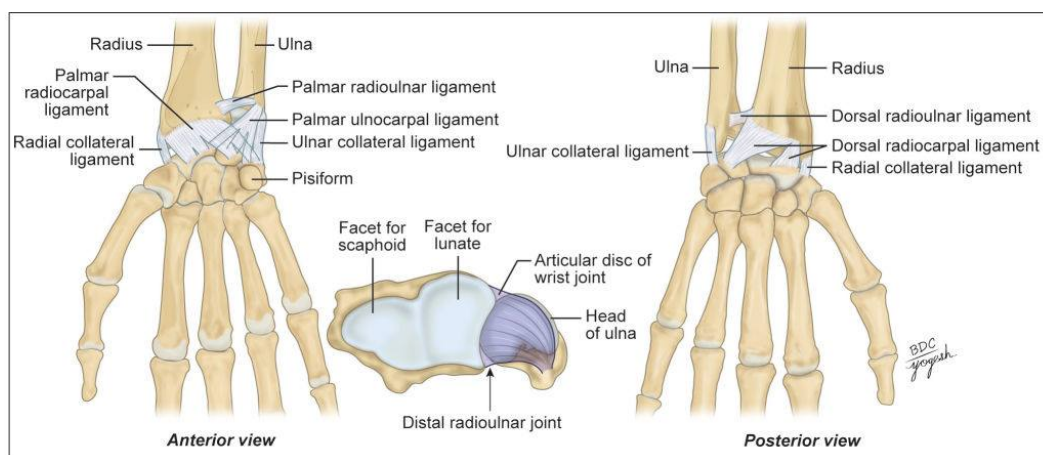


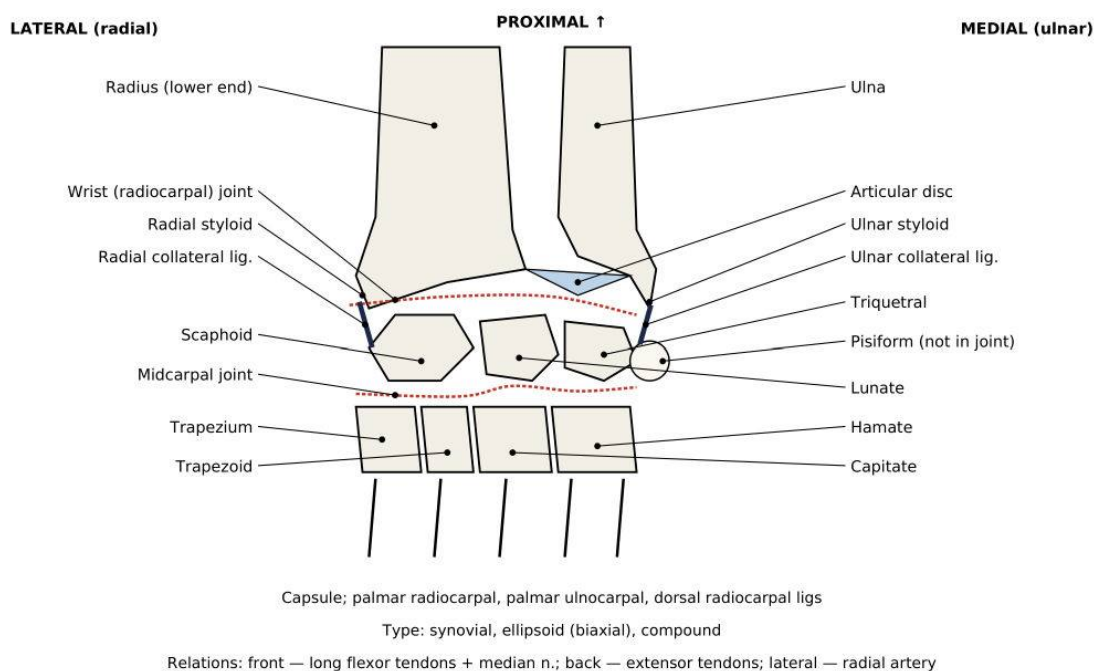
Fig. 10.23: Wrist, midcarpal and 1st carpometacarpal joints

BDC 10e vol. 1 Fig. 10.23, p. 155 — Wrist, midcarpal and first carpometacarpal joints.

Plate 10.6: Wrist joint: Articular surface and ligaments (Fig. 10.24)



BDC 10e vol. 1 Plate 10.6 (Fig. 10.24), p. 156 — Wrist joint: articular surfaces and ligaments.



Schematic (exam diagram): right wrist joint, coronal view. Based on BDC 10e vol. 1 Fig. 10.23, p. 155 and Plate 10.6 / Fig. 10.24, p. 156; text pp. 155-157.

Ligaments, relations, supply

Table 12. Wrist joint: ligaments, relations, blood and nerve supply *Source: BDC 10e vol. 1 pp. 155-156*

Feature	Detail
Capsule	Lower ends of radius and ulna → proximal row of carpal bones
Palmar radiocarpal ligament	Broad band: anterior margin of radius and styloid → scaphoid, lunate, triquetral
Palmar ulnocarpal ligament	Base of ulnar styloid and articular disc → lunate, triquetral
Dorsal radiocarpal ligament	Weaker; posterior margin of radius → dorsum of scaphoid, lunate, triquetral
Radial collateral ligament	Tip of radial styloid → lateral scaphoid; related to the radial artery
Ulnar collateral ligament	Tip of ulnar styloid → triquetral and pisiform (both collaterals poorly developed)
Relations	Anterior: long flexor tendons in their sheaths, median nerve. Posterior: extensor tendons. Lateral: radial artery
Blood / nerve supply	Anterior and posterior carpal arches / anterior and posterior interosseous nerves

Movements

Table 13. Movements of the wrist *Source: BDC 10e vol. 1 p. 157*

Movement	Main site	Principal muscles
Flexion	More at the midcarpal joint	FCR, FCU, palmaris longus
Extension	Mainly the wrist joint	ECRL, ECRB, ECU
Abduction (radial deviation)	Mainly midcarpal	FCR, ECRL, ECRB, APL, EPB
Adduction (ulnar deviation)	Mainly the wrist joint	FCU, ECU

- Range of flexion exceeds extension; adduction exceeds abduction (shorter ulnar styloid).
- Wrist motion is shared almost equally between the radiocarpal and midcarpal joints; the scaphoid links the two carpal rows and is therefore the most injured carpal bone (TIN).

Applied (emergency) relevance

- **Aspiration** from the dorsum between the tendons of EPL and extensor digitorum.
- **Immobilise** in the optimum position of 30° dorsiflexion.
- **Space of Poirier**: weak volar area between the two palmar ligament arcades at the capitoulunate junction; forced dorsiflexion tears here → lunate/perilunate dislocation (TIN).
- Ligament injury to either carpal row can cause volar or dorsal carpal subluxation.

PEARLS

- ★ Ellipsoid, biaxial, compound synovial joint.
- ★ Radius + disc above; scaphoid, lunate, triquetral below; pisiform excluded.
- ★ Extension and adduction mainly at the radiocarpal joint; flexion and abduction mainly midcarpal.
- ★ Aspirate dorsally between EPL and extensor digitorum.

Sources

- BDC 10e (Competency Based), vol. 1 Upper Limb, ch. 10 "Joints of Upper Limb", §Wrist (Radiocarpal) Joint, pp. 155-157; Fig. 10.23, p. 155; Plate 10.6 and Fig. 10.24, p. 156; §Movements, Clinical Anatomy, p. 157; §Joints of Hand (midcarpal joint), p. 158.
- TIN 9e, ch. 269 "Wrist Injuries", §Anatomy, §Carpal Bones, pp. 1794-1795.

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- 9** Respiratory Emergencies
- 10** Gastrointestinal & Hepatobiliary Emergencies
- 11** Renal, Genitourinary, Electrolyte & Acid–Base Disorders
- 12** Obstetric & Gynaecological Emergencies
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Answers

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Updated till

Oct 2026

- 14** Infectious Diseases, Sepsis & COVID-19
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HOW TO USE THESE NOTES

- **One answer, many questions.** Repeats and near-repeats are merged; every merged question is printed verbatim at the head of its answer with an **Asked in** line and a frequency badge (1×, 2×, 3×, 4×+).
- **Management basis strip** (amber) names the guideline or book that drives management; every guideline-derived line carries a tag such as [AHA 2025] or [SSC 2026 R16] with the guideline's own grade.
- **Red box — NOT COVERED IN SOURCE:** the point is absent from the source books and saved guidelines after a full search; the box records the search, and the closest sourced material follows, labelled as such.
- **Figures** are real figures cropped from the source books with their own numbers and pages. Drawings labelled **Schematic (exam diagram)** are simplified diagrams built from a labelled spec, printed beside the original; flowcharts are our own drawings from the guideline or book text.
- **Green box** = pearls for last-minute revision; **blue box** = worked example (every number recomputed).

Part 9 · Respiratory Emergencies

13 answers

Enumerate the causes and evaluation of hypercapnia in ED. [6] — Apr 2023 · P-I · Q6(b)

Asked in: Apr 2023 P-I Q6(b) · 6 marks → 6–7-mark length

Definition and mechanism

- **Hypercapnia** = $\text{PaCO}_2 > 45 \text{ mm Hg } (> 6 \text{ kPa})$. It is caused exclusively by **alveolar hypoventilation**; increased CO_2 production alone never causes it.
- Alveolar ventilation $V_A = (V_T - \text{dead space}) \times \text{rate}$: it falls with a lower rate, smaller tidal volume or larger dead space (ventilated but unperfused lung).
- Alveolar PCO_2 rises in proportion to CO_2 production and in inverse proportion to alveolar ventilation (GUY).
- Hypoxia from low inspired O_2 , anaemia or diffusion block does **not** cause hypercapnia (CO_2 diffuses 20 times faster); hypoxia from hypoventilation does.

Causes

Table 1. Causes of hypercapnia Source: *TIN 9e Table 62-2, p. 427*

Group	Examples
Depressed central drive	Brainstem lesions; opioids, sedatives, anaesthetics; tetanus
Thoracic cage disorders	Kyphoscoliosis, morbid obesity
Neuromuscular impairment	Myasthenia gravis, Guillain–Barré syndrome; organophosphate poisoning, botulism
Intrinsic lung disease (↑ dead space)	COPD
Upper airway obstruction	Foreign body, angioedema, others

Clinical features

- Depend on the absolute PaCO_2 and its rate of rise. Acute rise → raised ICP: headache, confusion, lethargy; then seizures, coma.
- Alveolar PCO_2 60–75 → severe dyspnoea; 80–100 → lethargy, semicoma; 120–150 → anaesthesia and death, as CO_2 now depresses respiration (vicious cycle) (GUY).
- Cardiovascular collapse usually only with acute $\text{PaCO}_2 > 100$; chronic levels > 80 may be well tolerated.

Evaluation in the ED

1. **Recognise:** signs of respiratory failure — altered consciousness, inability to speak, paradoxical breathing, diaphragmatic fatigue.
2. **Confirm:** ABG (or end-tidal CO_2); a venous pH agrees well with arterial pH in COPD and is a screening option.
3. **Acute or chronic?** HCO_3^- rises $\sim 1 \text{ mEq/L}$ per 10 mm Hg PaCO_2 (acute) and $\sim 3.5 \text{ mEq/L}$ per 10 mm Hg (chronic, near-normal pH); acute pH change $\approx 0.008 \times (40 - \text{PaCO}_2)$. Values outside these suggest a mixed metabolic disorder.
4. **A–a gradient:** normal → pure hypoventilation (central drive, neuromuscular, chest wall); raised → lung disease (COPD, pneumonia).
5. **Cause-directed tests:** toxicology history and opioid/sedative screen; neurological examination and **negative inspiratory force** for neuromuscular weakness; peak flow/spirometry for obstruction; chest radiograph, lung US; ECG; electrolytes, glucose.
6. **Severity and disposition:** admit new acidosis, CNS symptoms or acute neuromuscular hypercapnia; chronic stable COPD with normal pH may go home.

WORKED EXAMPLE — Drowsy patient after an opioid overdose, room air: pH 7.16, PaCO_2 70, PaO_2 50 mm Hg

Expected acute pH = $7.40 - 0.008 \times (70 - 40) = 7.16$ → pure **acute** respiratory acidosis. Expected $\text{HCO}_3^- \approx 24 + 3 = 27$ (chronic would be $\approx 24 + 10.5 = 34.5$ with near-normal pH).

$\text{PAO}_2 = 0.21 \times 713 - 70/0.8 = 149.7 - 87.5 = 62.2$; A–a = $62.2 - 50 = 12 \text{ mm Hg}$ (normal) → hypoventilation without lung disease: central depression.

PEARLS

- ★ Hypercapnia = $\text{PaCO}_2 > 45$ = alveolar hypoventilation, always.
- ★ Acute: $\text{HCO}_3^- +1$ per 10; chronic: $+3.5$ per 10.
- ★ Normal A-a + high $\text{PaCO}_2 \rightarrow$ drug, CNS, neuromuscular or chest wall cause.
- ★ Never withhold O_2 to “drive” breathing in chronic lung disease.

Sources

• TIN 9e, ch. 62 "Respiratory Distress", §Clinical Features, §Laboratory Testing, pp. 425–426; §A-a gradient and mechanisms, pp. 426–427; §Hypercapnia, Table 62-2, pp. 427–428; ch. 70 "COPD", §Diagnosis, pp. 468–469; ch. 16, p. 79.
• GUY 15e, ch. 38, §Dead Space, p. 507; ch. 40, § CO_2 in alveoli, p. 525; ch. 42, §Attenuated Effect of CO_2 , p. 544; ch. 43, §Hypercapnia, p. 558.

Type I (hypoxaemic) respiratory failure: ED management

1×

Management of Type I respiratory failure in ED. [5] — Apr 2023 · P-III · Q6(b)

Asked in: Apr 2023 P-III Q6(b) · 5 marks → 4–5-mark length

Definition and mechanisms

- **Hypoxaemia** = $\text{PaO}_2 < 60$ mm Hg (< 8 kPa); in type I failure PaCO_2 is not raised. Mechanisms: V/Q mismatch, shunt, diffusion impairment, low inspired O_2 .
- All except **right-to-left shunt** respond to supplemental O_2 ; shunt (consolidation, atelectasis) is never fully corrected.

Management basis: TIN 9e ch. 62 Respiratory Distress, ch. 28 Noninvasive Airway Management and ch. 29B Mechanical Ventilation (lead); RH 8e ch. 8 for NIV and HFNC procedure. No Appendix-A guideline applies.

Step 1 — airway and oxygen

- Ensure a patent airway; give O_2 to **$\text{PaO}_2 > 60$ mm Hg and/or $\text{SaO}_2 \geq 90\%$** . Assess for signs of failure (altered consciousness, inability to speak, paradoxical breathing).

Table 2. Oxygen and respiratory support — escalation Source: TIN 9e Table 28-3, p. 174; pp. 175, 191–192; RH 8e pp. 170–172, Fig. 8.13

Device	Flow / setting	Delivered FiO_2 / notes
Nasal cannula	2–5 L/min	20–40%
Simple face mask	6–10 L/min	40–60%
Non-rebreather mask	10–15 L/min	Near 100%
High-flow nasal cannula	Up to 60 L/min, heated, humidified	Some positive pressure, ↓ work of breathing; for $\text{SpO}_2 < 92\%$ and $\text{RR} > 22$ with intact drive; avoid in bullous disease/pneumothorax; may improve mortality vs NIPPV or conventional O_2 in nonhypercapnic hypoxaemic failure
CPAP / BiPAP (NIPPV)	Start EPAP 5 (8 if obese); raise EPAP if hypoxic; FiO_2 to SpO_2 86–92% (RH)	Best evidence in cardiogenic pulmonary oedema and immunosuppressed patients; in other hypoxaemic failure only if haemodynamically stable, single-organ failure, with close monitoring
Intubation + ventilation	V_T 6–8 mL/kg IBW, PEEP 5–8, RR 10–20	Titrate FiO_2 to $\text{SpO}_2 < 96\%$ (avoid hyperoxia); ARDS: V_T 6 mL/kg, plateau < 30

Step 2 — monitor and decide on escalation

- **NIPPV contraindications:** arrest or impending collapse, severe upper GI bleed, facial trauma or deformity, upper airway obstruction, inability to cooperate or protect the airway, copious secretions, high aspiration risk (RH Box 8.3); hypotension — use cautiously (TIN).
- Reassess within **30 min** and check ABG at **1–2 h**; persistent tachypnoea or failure to improve → intubate without delay.
- Pure shunt (e.g. large consolidation) will not correct with O_2 alone: needs PEEP to recruit alveoli.

Step 3 — treat the cause

- Pneumonia (antibiotics), cardiogenic pulmonary oedema (CPAP plus specific therapy), PE (anticoagulation), ARDS (lung-protective ventilation), pneumothorax (drainage) — identified by history, examination, ECG, point-of-care US and chest radiograph.
- **Disposition:** HFNC, NIPPV or ventilation → ICU or monitored bed.

PEARLS

- ★ Target $\text{PaO}_2 > 60$ mm Hg / $\text{SaO}_2 \geq 90\%$.
- ★ Shunt = O_2 -refractory hypoxaemia → needs PEEP.
- ★ HFNC up to 60 L/min: first escalation in nonhypercapnic hypoxaemia.
- ★ Reassess NIPPV at 30 min and ABG at 1–2 h.

Sources

- TIN 9e, ch. 62 "Respiratory Distress", §History and Examination, §Treatment, §Hypoxia and Hypoxemia, pp. 425–427; ch. 28 "Noninvasive Airway Management", §Oxygenation, Table 28-3, §NIPPV, pp. 174–175; ch. 29B, Table 29B-3, §Hyperoxia, pp. 191–192.
- RH 8e, ch. 8 "Mechanical Ventilation", §NPPV (Box 8.2, Box 8.3, Fig. 8.13), §High-Flow Nasal Cannula, pp. 169–172.

Point-of-care ultrasound (POCUS) in COVID-19

1x

Use of POCUS in COVID-19 infections. [5] — Jun 2020 · P-IV · Q1(a)

Asked in: Jun 2020 P-IV Q1(a) · 5-mark → 4–5-mark length

NOT COVERED IN SOURCE

Searched TIN 9e, RH 8e, ATLS 11e, GUY 15e, BDC 10e (3 vols), STAT 4e and the whole guideline store (AHA 2025 Parts 1–12 and algorithms, SSC 2026, AHA/ASA 2026, Indian STG/NPCCHH/legal texts) for 'COVID', 'SARS-CoV-2', 'SARS CoV', 'coronavirus', 'pandemic', 'lung ultrasound', 'lung US', 'B-lines', 'POCUS', 'point-of-care ultrasound'; TIN ch. 50, 53, 56, 62 and RH ch. 10, 16 read. COVID-19 lung-ultrasound patterns and protocols are not described. Below: POCUS applications the sources give for the problems a COVID-19 patient presents with.

Closest material: what POCUS answers at the bedside

Table 3. POCUS in the acutely dyspnoeic or shocked patient *Source: TIN 9e ch. 50, 53, 56, 62; RH 8e ch. 16*

Question	POCUS finding	Source
Interstitial / alveolar fluid?	B-lines: vertical ring-down artefacts from the pleural interface, crossing the screen and A-lines; >2 per window is pathological. Bilateral B-lines also occur in bilateral pneumonia , fibrosis and contusion, not only oedema	TIN pp. 355, 370
Heart failure or not?	Lung US separates acute decompensated heart failure from non-cardiac dyspnoea; add LV function and IVC (>2 cm, poor collapse = high CVP)	TIN pp. 370, 426
Other causes of hypoxia	Pleural effusion, pneumothorax, consolidation, tamponade, intravascular volume status	TIN p. 426
Pulmonary embolism / DVT	Lung US sensitivity 85%, specificity 83% for PE; compression venous US sensitivity and specificity 96% for DVT	TIN p. 395
Shock	Ejection fraction, wall motion, dilated RV, pericardial fluid, IVC, B-lines (cardiogenic shock approach)	TIN p. 354
Pericardium	COVID-19 can cause serous, serosanguinous or haemorrhagic pericardial effusion, rare and delayed; MIS-C in children	RH p. 310

Practical points

- Bedside US is an adjunct, not a replacement, for formal echocardiography (TIN).
- Serial bedside scanning avoids moving an infectious patient; infection-control precautions apply to the operator (RH ch. 69).
- Severe COVID-19 is characterised by refractory hypoxia (RH): POCUS helps exclude effusion, pneumothorax, PE and cardiogenic causes before escalating support.

PEARLS

- ★ Bilateral B-lines = interstitial syndrome (pneumonia or oedema) — add heart and IVC to decide.
- ★ Use POCUS to exclude PE/DVT, effusion, pneumothorax and tamponade in the hypoxic patient.

Sources

• TIN 9e, ch. 50 "Cardiogenic Shock", Fig. 50-1, p. 354 and §Echocardiography, p. 355; ch. 53 "Acute Heart Failure", §Ultrasound, p. 370; ch. 56 "Venous Thromboembolism", §Lung US, §Venous US, p. 395; ch. 62 "Respiratory Distress", p. 426.
• RH 8e, ch. 16 §SARS-CoV-2, p. 310; ch. 8 §Lessons Learned in COVID, p. 175.

Haemoptysis: differential diagnosis and approach in the ED

2×

[A 45-year-old presents to emergency department with history of hemoptysis for one day and develops massive hemoptysis in the ED.] Discuss the differential diagnosis of hemoptysis. [4] — Jun 2021 · P-III · Q4(a)

Outline the approach to acute haemoptysis in the ED. [5] — Dec 2025* · P-III · Q9(a)

Asked in: Jun 2021 P-III Q4(a); Dec 2025* P-III Q9(a) · 4 + 5 (differential diagnosis is part of the approach) → 6–7-mark length
• J21 case: 45-year-old developing massive haemoptysis

Definitions and source of bleeding

- **Haemoptysis** = expectoration of blood. **Massive:** definitions range from 100 to >1000 mL/24 h; **600 mL/24 h** is widely accepted. Because even small volumes can asphyxiate, any haemoptysis needs prompt attention.
- **Minor** = self-limited, small volume, no comorbid lung disease, normal oxygenation, ventilation and vital signs, no risk factors for continued bleeding.
- **Bronchial arteries** (high pressure, 1% of lung flow) cause **90%** of haemoptysis; pulmonary arteries (99% of flow, low pressure) rarely cause massive bleeding. Bronchial arteries arise from the thoracic aorta at T5–T6, ectopically in one-third.

Differential diagnosis

Table 4. Causes of haemoptysis Source: TIN 9e Table 63-1 and text, pp. 432–433

Group	Causes
Infectious	Acute bronchitis; tuberculosis (Rasmussen aneurysm in a cavity wall); fungal (aspergilloma); parasitic (paragonimiasis, echinococcus, schistosomiasis)
Structural	Bronchiectasis ; tracheo-arterial fistula (tracheostomy); aortobronchial fistula
Neoplastic	Bronchogenic carcinoma (squamous cell — many massive bleeds); bronchial adenoma
Cardiopulmonary	Pulmonary embolism, pulmonary hypertension, mitral stenosis, heart failure, congenital heart disease, obstructive sleep apnoea
Vasculitis / collagen disease	Goodpasture's, granulomatosis with polyangiitis, SLE, Behçet's, Osler–Weber–Rendu, Takayasu
Iatrogenic / traumatic	Lung biopsy, PA catheter injury, bevacizumab; bronchial rupture, contusion, penetrating trauma
Miscellaneous	Cocaine inhalation, NO ₂ (ice arenas), catamenial, Dieulafoy's disease; anticoagulants
Mimics (pseudohaemoptysis)	Haematemesis (dark stools, nausea, abdominal pain, guaiac-positive stool); epistaxis

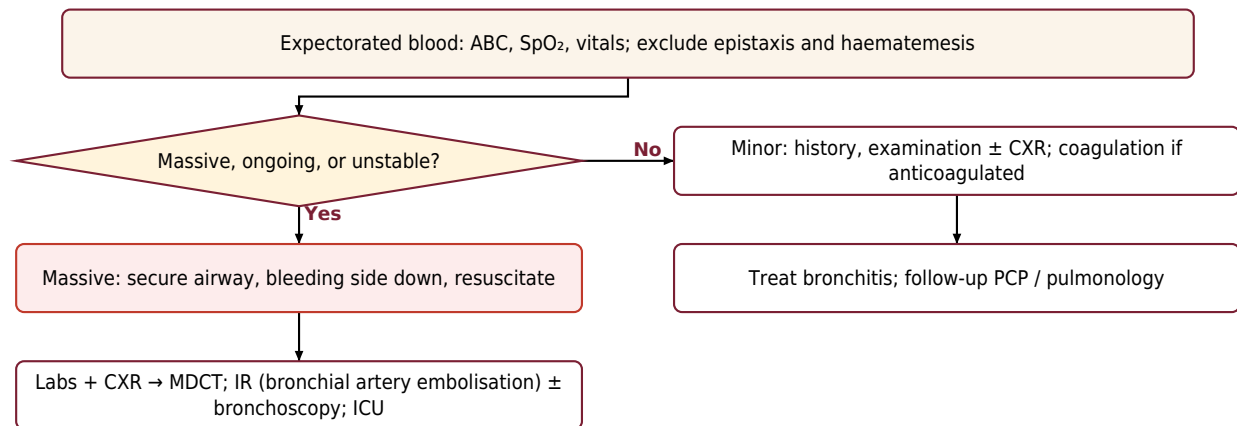
- Commonest causes of **potentially massive** bleeding are parenchymal: TB, mycetoma, neoplasm, bronchiectasis. About 30% were historically cryptogenic.
- **In the 45-year-old (J21 case):** prioritise TB, bronchogenic carcinoma (smoker), bronchiectasis, aspergilloma and PE; check anticoagulant use.

Approach

1. **Life threat first:** airway patency, oxygenation, haemodynamics; judge stability by history, rate of ongoing bleeding, ability to clear blood and comorbidity.
2. **Confirm true haemoptysis:** exclude haematemesis and epistaxis (examine nares and posterior pharynx); blood from lung is bright red.
3. **History:** smoking, VTE, connective-tissue disease, vasculitis; TB exposure or travel/emigration from high-prevalence areas (start respiratory precautions); haematuria or renal failure (Goodpasture's, GPA); cyclical bleeding; cocaine/heroin; anticoagulants; recent bronchoscopy or PA catheter.

4. **Examination:** inspect the sputum; tachypnoea, tachycardia, hypotension, hypoxaemia; difficult-airway features; wheeze, focal ↓ breath sounds (side of bleeding), crackles; murmurs; telangiectasia, petechiae; saddle nose.
5. **Tests:** minor — none unless anticoagulated. Massive/recurrent — metabolic panel, renal function, CBC, coagulation, urinalysis (Hb may be falsely normal for 6 h). **Chest radiograph** first (diagnosis in up to 50%); **multidetector CT** preferred in massive haemoptysis (identifies bronchial arteries, Rasmussen aneurysm, Dieulafoy's).
6. **Disposition:** minor — reassure, treat bronchitis, follow-up with primary care/ENT, pulmonologist if cancer or structural disease suspected. Massive — ICU or tertiary centre (see HEMOPTYSIS-2).

Approach to haemoptysis in the ED



Our own flowchart drawn from the text and Figs. 63-1 and 63-2 of TIN 9e ch. 63, pp. 433–435.

PEARLS

- ★ Bronchial arteries: 1% of flow, 90% of haemoptysis.
- ★ Massive ≈600 mL/24 h, but airway compromise defines danger.
- ★ TB, bronchiectasis, carcinoma, aspergilloma = massive-bleed causes.
- ★ CXR first; MDCT is the key test in massive haemoptysis.

Sources

• TIN 9e, ch. 63 "Hemoptysis", §Introduction, §Pathophysiology, Table 63-1, §Clinical Features, §History, §Physical Examination, §Diagnosis, §Imaging, §Treatment and Disposition, Figs. 63-1, 63-2, pp. 432–435; ch. 67 "Tuberculosis", §Diagnosis (respiratory precautions), p. 452.

Massive haemoptysis: ED management; indications for bronchoscopy and bronchial artery embolisation

2×

[A 45-year-old presents to emergency department with history of hemoptysis for one day and develops massive hemoptysis in the ED.] How will you manage the patient? [6] — Jun 2021 · P-III · Q4(b)

Discuss indications for bronchoscopy and embolization in management of acute haemoptysis. [5] — Dec 2025* · P-III · Q9(b)

Asked in: Jun 2021 P-III Q4(b); Dec 2025* P-III Q9(b) · 6 + 5 (bronchoscopy and embolisation are part of the management) → 6–7-mark length · J21 case

Management basis: TIN 9e ch. 63 Hemoptysis (lead); RH 8e ch. 26 for nebulised tranexamic acid. No Appendix-A guideline covers haemoptysis.

1. Airway and oxygenation

- Ensure airway patency and oxygenation; summon interventional radiology (IR) and cardiothoracic surgery/pulmonology early.
- **Tracheostomy present:** suspect a tracheo-innominate fistula — digital pressure on the anterior trachea against the back of the sternum through the stoma.
- **Intubate** by RSI with a **large-bore ETT** (allows bronchoscopy). Then place the **bleeding lung dependent** to protect the other side.

- Uncontrollable bleeding: **selective intubation of the main bronchus of the non-bleeding lung**, or a **Fogarty catheter (14F/100 cm)** passed beside the ETT to tamponade the bleeding bronchus. Failed intubation → cricothyrotomy.

2. Circulation

- Crystalloid and blood products; correct coagulopathy and thrombocytopenia (both increase recurrence and morbidity).
- Nebulised tranexamic acid** has been used for haemoptysis: 500–1000 mg in adults (250 mg in children <25 kg); optimal dose unknown; it does not seem to share the thromboembolic or seizure risks of IV TXA (RH).

3. Localise and stop the bleeding

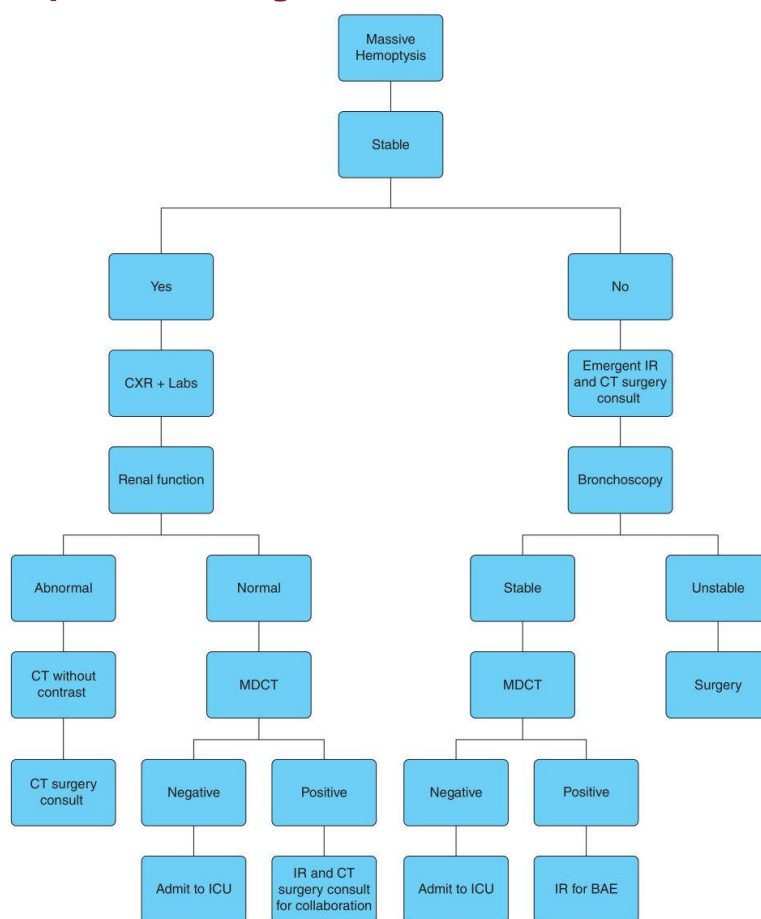


FIGURE 63-2. Algorithm for massive hemoptysis. Determine whether patient is stable based on history provided by patient, rate of ongoing bleeding, ability of patient to clear the blood, and comorbidities. BAE = bronchial artery embolization; CT surgery = cardiothoracic surgery; CXR = chest radiograph; ICU = intensive care unit; IR = interventional radiology; MDCT = multidetector computed tomography.

TIN 9e Figure 63-2, p. 435 — Algorithm for massive haemoptysis (stability judged on history, rate of bleeding, ability to clear blood, comorbidity).

- Best outcomes come from combining **multidetector CT (MDCT)** and bronchoscopy; most recommend MDCT before bronchoscopy. Bronchial arteries — the treatable source — are almost always seen on MDCT.

Table 5. Indications and choice of bronchoscopy *Source: TIN 9e §Bronchoscopy, p. 434; Fig. 63-2, p. 435*

Aspect	Flexible (fiberoptic)	Rigid
Indication	Identify the origin of bleeding; in unstable patients, urgent bronchoscopy (after IR/CT surgery consult) to localise and stabilise	Same, when airway control, suction and local treatment are needed
Anaesthesia	Awake	General anaesthesia or deep sedation
Reach	Peripheral and upper lobes	Cannot fully see upper lobes or periphery
Suction / therapy	Poor suction; no local treatment	Better suction; Fogarty balloon tamponade, epinephrine instillation, ice-water lavage; flexible scope can pass through it
Limitation	Massive bleeding obscures the view for both	

Bronchial artery embolisation (BAE)

- **Indications:** initial and most effective definitive treatment of **massive and recurrent haemoptysis**, followed by bronchoscopy if needed; MDCT-positive bleeding in a stable patient; after bronchoscopic stabilisation of an unstable patient (Fig. 63-2).
- **Risks:** transverse myelitis (spinal cord ischaemia) and pulmonary infarction from non-target embolisation.
- **Surgery** is now reserved for iatrogenic pulmonary artery injury, thoracic trauma, tracheo-innominate fistula, and patients still unstable after bronchoscopy.

4. Disposition

- ICU admission or transfer to a tertiary centre with IR and thoracic surgery.

PEARLS

- ★ Large ETT; bleeding side down; selective intubation of the good lung or Fogarty blocker.
- ★ MDCT before bronchoscopy; rigid scope for suction and local therapy.
- ★ BAE = first-line definitive therapy; watch for spinal cord ischaemia.
- ★ Tracheostomy + bleeding = tracheo-innominate fistula until proved otherwise.

Sources

- TIN 9e, ch. 63 "Hemoptysis", §Pathophysiology, p. 432; §Diagnosis, §Imaging, §Treatment: Severe Hemoptysis, §Airway Control, §Bronchoscopy, pp. 433–434; Fig. 63-2, §Definitive Bleeding Control, §Disposition, p. 435.
- RH 8e, ch. 26 "Alternative Methods of Drug Administration", §Bleeding: Nebulized Tranexamic Acid, p. 487.

Community-acquired pneumonia: aetiology, pathogens by age group and CURB-65

2×

Etiology of community-acquired pneumonia. [2] — Jun 2020 · P-III · Q7(a)

CURB-65 score. [2] — Jun 2020 · P-III · Q7(c)

Enumerate the common pathogens of community acquired pneumonia based on age-group and describe CURB-65 rule. [3+2] — Dec 2022 · P-III · Q1(a)

Asked in: Jun 2020 P-III Q7(a, c); Dec 2022 P-III Q1(a) · 2 + 3 (aetiology and pathogens overlap) + 2 (CURB-65, asked twice)
→ 4–5-mark length

Definition and route of infection

- **CAP** = acute pulmonary infection in a patient not hospitalised or in long-term care ≥14 days before presentation.
- Organisms are inhaled or (more often) **aspirated**; *S. aureus* and pneumococcus may seed haematogenously.
- **Predisposing factors:** aspiration risk (dysphagia, stroke, nasogastric tube, seizure, syncope); bacteraemia risk (vascular devices, alcoholism, extremes of age, neoplasia, immunosuppression, diabetes, renal or liver failure, heart failure); pulmonary disorders (COPD, chest-wall or muscle disorders, bronchial obstruction, viral lung infection).
- **Typical vs atypical:** bacteria provoke intense alveolar inflammation and productive cough; atypical agents (*Legionella*, *Chlamydia*, *Mycoplasma*, viruses) lack a cell wall, cause a milder dry cough and do not respond to β-lactams — they need a macrolide or respiratory fluoroquinolone.

Aetiology

- No pathogen is identified in about **half** of cases. When found: ***S. pneumoniae*** (commonest) > viruses > atypicals (*Mycoplasma*, *Chlamydia*, *Legionella*). Mixed infection in up to 5%.
- **Severe CAP** in healthy adults: *S. pneumoniae* and *Legionella* — empiric therapy must cover both.
- **Special groups:** alcoholics — pneumococcus, *Klebsiella*, *Haemophilus*; COPD/elderly — *H. influenzae*, *Moraxella*; after influenza — *S. pneumoniae*, *S. aureus*, *H. influenzae*; nursing-home — pneumococcus, gram-negative bacilli, *H. influenzae* (MRSA risk); *Pseudomonas* — structural lung disease, high-dose steroids, recent hospital/antibiotics; diabetics — *S. aureus*, gram-negatives, TB.

Table 6. Common pathogens by age group *Source: TIN 9e ch. 128 pp. 811–812; ch. 65 pp. 439–443*

Age group	Common pathogens
Neonates (0–30 d)	Group B streptococci, Klebsiella, E. coli, Listeria; late: S. aureus, S. pneumoniae, S. pyogenes; C. trachomatis if no prenatal care
30 d – 2 y	Mostly viral (RSV, rhinovirus, influenza, parainfluenza, metapneumovirus, adenovirus); S. pneumoniae commonest bacterium; H. influenzae; S. aureus, group A strep, pertussis
2 – 5 y	Viruses (RSV, rhinovirus, metapneumovirus); S. pneumoniae, H. influenzae; Mycoplasma/C. pneumoniae less common
5 – 13 y	Viruses; M. pneumoniae (17%), C. pneumoniae, S. pneumoniae; less often S. aureus, group A strep, Legionella, TB
Adolescents / young adults	As adults; atypicals (Mycoplasma, C. pneumoniae) significant
Adults	S. pneumoniae, viruses, atypicals (Mycoplasma, Chlamydia, Legionella)
Elderly	S. pneumoniae (3× bacteraemia risk), influenza, post-influenza S. aureus/H. influenzae; atypicals less common but occur

CURB-65

Table 7. CURB-65 (score = number of features present) *Source: TIN 9e p. 446*

Letter	Feature
C	Confusion
U	Uraemia: urea >7 mmol/L
R	Respiratory rate ≥30/min
B	Blood pressure: diastolic ≤60 mm Hg
65	Age ≥65 years

- **Score <2** → low mortality (outpatient candidate); **≥3** → may need intensive care.
- **CRB-65** drops urea (usable without labs). Neither is as well studied or validated as the Pneumonia Severity Index; scores support, not replace, judgement.

PEARLS

- ★ Pneumococcus is commonest; half have no pathogen identified.
- ★ Cover pneumococcus and Legionella in severe CAP.
- ★ Children <5 y: mostly viral; 5–13 y: Mycoplasma rises.
- ★ CURB-65 <2 home; ≥3 consider ICU.

Sources

- TIN 9e, ch. 65 "Community-Acquired Pneumonia", Tables 65-1, 65-2, §Pathophysiology, pp. 439–440; §Other Bacterial and Atypical Pneumonias, p. 441; §Special Populations, pp. 442–443; §Disposition, p. 446.
- TIN 9e, ch. 128 "Pneumonia in Infants and Children", §Age-Specific Causes, pp. 811–812.

Pneumonia: clinical and radiological features

1×

Clinical and radiological features in a patient with pneumonia. [4] — Jun 2020 · P-III · Q7(b)

Asked in: Jun 2020 P-III Q7(b) · 4 marks → 4–5-mark length

Symptoms

- Cough (79–91%), fatigue (90%), fever (71–75%), dyspnoea (67–75%), sputum (60–65%), pleuritic chest pain (39–49%). Individual symptoms poorly separate pneumonia from upper respiratory infection.
- **Classic pneumococcal:** sudden fever, a single severe rigor, dyspnoea, bloody (rusty) sputum, chest pain — now uncommon.
- Prodrôme of viral URI; weight loss, malaise, weakness; atypical agents → headache or GI upset; extrapulmonary joint pain, haematuria, rash.
- **Elderly/nursing home:** falls, functional decline, delirium; often afebrile; one-third without leukocytosis.

Signs

- Tachycardia, tachypnoea; **inspiratory crackles** (alveolar fluid), **bronchial breath sounds** (consolidation), dullness with ↓ breath sounds (effusion), rhonchi/wheeze (bronchial congestion). Egophony and pectoriloquy are specific but uncommon.
- **Labs:** leukocytosis, raised bilirubin/liver enzymes, hyponatraemia.
- **Diagnosis** rests on fever, cough and crackles or rhonchi, confirmed by a radiographic infiltrate; physician judgement beats scoring systems in deciding on a radiograph.
- **Mimics** of cough with an abnormal film: lung cancer, tuberculosis, pulmonary embolism, chemical or hypersensitivity pneumonitis, connective-tissue disease, granulomatous and fungal disease.

Radiological features

- Radiograph confirms the diagnosis (fever + cough + crackles + infiltrate) but is **nonspecific** for the organism.
- **Bacterial** → more often unilobar/focal (lobar) infiltrate; **viral/atypical** → diffuse or interstitial; **hilar adenopathy** favours atypical.
- Pleural effusion with any type (parapneumonic in ~25% of pneumococcal). Cavitation → bacterial or TB; abscess → *S. aureus*, *Klebsiella*.
- “Round pneumonia” (pneumococcal, staphylococcal) may mimic a mass. Immunocompromised patients may have no infiltrate; the film can lag or worsen while the patient improves.

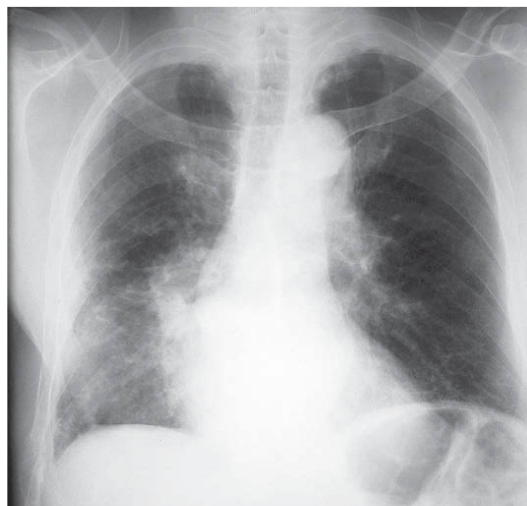


FIGURE 65-1. Lobar pneumonia.

TIN 9e Figure 65-1, p. 441 — Lobar pneumonia.

Table 8. Clinical and radiographic pointers by organism *Source: TIN 9e Table 65-3, p. 440*

Organism	Clinical	Chest radiograph
<i>S. pneumoniae</i>	Sudden fever, rigors, pleuritic pain; rusty sputum	Lobar infiltrate, occasional effusion
<i>S. aureus</i>	Gradual, after viral illness; purulent sputum	Patchy multilobar; empyema, abscess
<i>Klebsiella</i>	Sudden, alcoholics/nursing home; “currant jelly” sputum	Upper lobe, bulging fissure, abscess
<i>H. influenzae</i>	Elderly, COPD; gradual	Patchy basilar; occasional effusion
<i>Legionella</i>	Fever, headache, dry cough, diarrhoea, vomiting	Patchy → consolidation; occasional cavitation, effusion
<i>Mycoplasma</i>	Nonproductive cough, bullous myringitis, headache	Interstitial (reticulonodular), patchy
Anaerobes	Putrid sputum, alcoholics	Dependent consolidation, abscess

PEARLS

- ★ No clinical or radiological finding identifies the organism reliably.
- ★ Lobar = bacterial; interstitial + hilar nodes = atypical.
- ★ Elderly: confusion, falls, no fever.

Sources

Community-acquired pneumonia: ED investigations, management and outpatient antibiotics

3×

Outpatient antibiotic therapy for community acquired pneumonia. [2] — Jun 2020 · P-III · Q7(d)

Discuss investigations and management of community acquired pneumonia in an adult in the emergency department. [2+3] — Dec 2022 · P-III · Q1(b)

Explain the emergency department management of community acquired pneumonia. [5] — Jun 2025* · P-III · Q2(c)

Asked in: Jun 2020 P-III Q7(d); Dec 2022 P-III Q1(b); Jun 2025* P-III Q2(c) · 5 (asked twice) + 2 → 6–7-mark length

Investigations

Table 9. Investigations in CAP — whom and why *Source: TIN 9e pp. 442, 446*

Test	Whom	Purpose / interpretation
Chest radiograph	All suspected	Confirms infiltrate; effusion, cavitation; alternatives (cancer, TB, PE)
Pulse oximetry; ABG	All; ABG if markedly desaturated or severe distress	Oxygenation; $\text{PaO}_2/\text{FiO}_2 \leq 250$ is an ICU criterion
CBC, electrolytes, urea, creatinine, glucose	Patients being admitted	Severity (PSI, CURB-65); ICU criteria (leukopenia, thrombocytopenia, hyponatraemia, BUN >20 mg/dL)
Blood cultures	ICU, leukopenia, cavitation, severe liver disease, alcohol abuse, asplenia, effusion	Yield rises with severity; low yield in outpatients
Sputum Gram stain/culture	Only if a good-quality specimen	Rarely changes therapy
Legionella urine antigen	ICU, alcoholics, travel within 2 weeks	Confirms Legionella
Lactate, other	Suspected sepsis	Lactic acidosis is an ICU criterion

Severity and disposition

- Most CAP does not need admission. **Pneumonia Severity Index** (best studied): classes I–II outpatient (mortality 0.1–0.6%), III individualised (2.8%), IV–V inpatient (8.2–29.2%). **CURB-65** <2 low risk; ≥3 may need ICU.
- **ICU** for sepsis or mechanical ventilation; also markedly raised RR, $\text{PaO}_2/\text{FiO}_2 \leq 250$, multilobar infiltrates, confusion, BUN >20 mg/dL, leukopenia, thrombocytopenia, hypothermia, hyponatraemia, lactic acidosis, asplenia.

Management in the ED

Management basis: TIN 9e ch. 65 (Tables 65-4 to 65-9, based on IDSA guidance). Septic shock from pneumonia is managed per SSC 2026 (see A01).

- Give **antibiotics promptly after diagnosis**; the old 4-hour target is discredited; follow local resistance patterns.
- Oxygen to $\text{SaO}_2 \geq 90\%$ (see RESP_FAILURE-2); fluids and source review if septic.
- **Pregnancy:** start a neuraminidase inhibitor as soon as possible for influenza-like illness (H1N1 deaths fewer and related to time to treatment); varicella pneumonia — IV acyclovir is often started in the ED.
- **Nursing-home and healthcare-associated pneumonia:** cover MRSA (Table 65-7: antipseudomonal β -lactam + fluoroquinolone + vancomycin 15 mg/kg every 12 h).

Table 10. Empiric antibiotic regimens *Source: TIN 9e Tables 65-4 to 65-9, pp. 444-445; text p. 445*

Setting	Regimen
Outpatient, healthy	Clarithromycin XL 1000 mg PO daily × 7 d, or azithromycin 500 mg PO day 1 then 250 mg days 2–5; doxycycline 100 mg BID × 10–14 d (second line)
Outpatient, comorbidity (heart, lung, liver, renal disease; diabetes; alcoholism; malignancy; asplenia) or antibiotics in last 3 months	Levofloxacin 750 mg daily × 5 d or moxifloxacin 400 mg daily × 7–14 d; or amoxicillin–clavulanate 2 g BID (or 3rd-generation cephalosporin) + azithromycin
Inpatient, non-ICU	Levofloxacin 750 mg IV or moxifloxacin 400 mg IV; or ceftriaxone 1 g IV + azithromycin 500 mg IV
ICU	Ceftriaxone 1 g + azithromycin 500 mg IV, or ceftriaxone + levofloxacin 750 / moxifloxacin 400 mg IV. Penicillin allergy: fluoroquinolone + aztreonam 1–2 g or clindamycin 600 mg IV
Pseudomonas risk	Two antipseudomonal agents: piperacillin–tazobactam or cefepime + respiratory fluoroquinolone; or carbapenem + fluoroquinolone/aminoglycoside
MRSA / healthcare-associated risk	Add vancomycin 10–15 mg/kg IV or linezolid 600 mg IV

Outpatient antibiotic therapy — key points

- First line: a **newer macrolide** (azithromycin once daily; clarithromycin fewer GI effects than erythromycin, which causes GI upset in ~25%).
- CDC: reserve **respiratory fluoroquinolones** for intolerance, documented resistance or failure; avoid in myasthenia gravis.
- Comorbidity or recent antibiotics → fluoroquinolone or β-lactam + macrolide/doxycycline; rising macrolide resistance among pneumococci.
- **Course:** improvement in 3–5 days; inpatients switch to oral at about day 3. Advise smoking cessation, alcohol moderation, pneumococcal and influenza vaccination.

PEARLS

- ★ Outpatient healthy: azithromycin 500 mg then 250 mg × 4 d.
- ★ Inpatient: ceftriaxone 1 g + azithromycin 500 mg IV, or respiratory FQ.
- ★ Blood cultures for ICU and high-risk features only.
- ★ PSI IV–V and CURB-65 ≥3 → admit (ICU if criteria).

Sources

• TIN 9e, ch. 65 "Community-Acquired Pneumonia", §Diagnosis, p. 442; §Pneumonia in Pregnant Women, p. 443; §Treatment, §Outpatient Treatment, Tables 65-4 to 65-6, p. 444; Tables 65-7 to 65-9, §Inpatient Treatment, p. 445; §Disposition and Follow-Up, Tables 65-10 to 65-12, p. 446.

Pathophysiology of pulmonary tuberculosis

1×

Describe the pathophysiology of pulmonary tuberculosis infection. [6] — Dec 2025* · P-I · Q1(a)

Asked in: Dec 2025* P-I Q1(a) · 6 marks → 6–7-mark length

Organism and transmission

- *Mycobacterium tuberculosis* is a **slow-growing aerobic rod** that settles where O₂ content and blood flow are high.
- Spread by **aerosolised droplet nuclei**; patients excreting bacilli in sputum or saliva are most infectious. Only about **30%** become infected after a droplet exposure.

Primary infection and latency

1. Bacilli reach the lungs; being highly antigenic, they trigger a rapid immune response. **Alveolar macrophages** invade the infected tissue.
2. Some organisms survive in **regional lymph nodes**; **cell-mediated immunity** contains them.
3. **Granuloma (tubercle)** forms — activated macrophages, T lymphocytes and live bacilli — and is walled off by fibrous tissue; it may undergo **caseation necrosis** and **calcification**. The tubercle also shelters bacterial growth.
4. **Ghon complex** = calcified primary focus with hilar lymph nodes.

5. If containment fails, bacilli spread by **haematogenous, lymphatic or direct** routes to high-O₂, high-flow sites: apical and posterior upper-lobe segments, superior lower-lobe segment, renal cortex, meninges, long-bone epiphyses, vertebrae.
6. Organisms lie **dormant** for years — **latent infection**: tuberculin test positive 1–2 months after exposure; not transmissible.

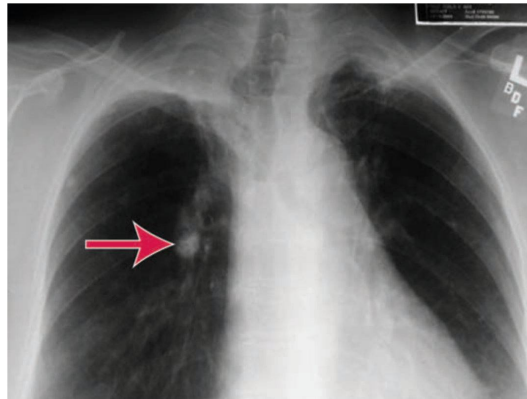


FIGURE 67-1. Primary Ghon complex (arrow).

TIN 9e Figure 67-1, p. 451 — Primary Ghon complex (arrow).

Progression and reactivation

- Only 1–13% of healthy people progress to active post-primary disease; children and HIV patients approach 20%. About 5% progress within 2 years and another 5% later in life.
- **Reactivation** causes 70% of active TB: weakened host defence can no longer contain old haematogenous foci. Risk: HIV (7–10% per year), age >50, solid-organ cancer, leukaemia, transplant, TNF- α antagonists, prednisone ≥ 15 mg/d for ≥ 4 weeks, diabetes, haemodialysis, psoriasis, silicosis.
- Reactivation favours the upper lobe or superior segment of the lower lobe, with or without **cavitation** (10–30% of patients; uncommon in primary disease); fibrocalcific change and pleural capping.
- **Rasmussen aneurysm** — a false aneurysm of a vessel crossing a cavity wall — and neovascularisation of cavity walls cause haemoptysis.

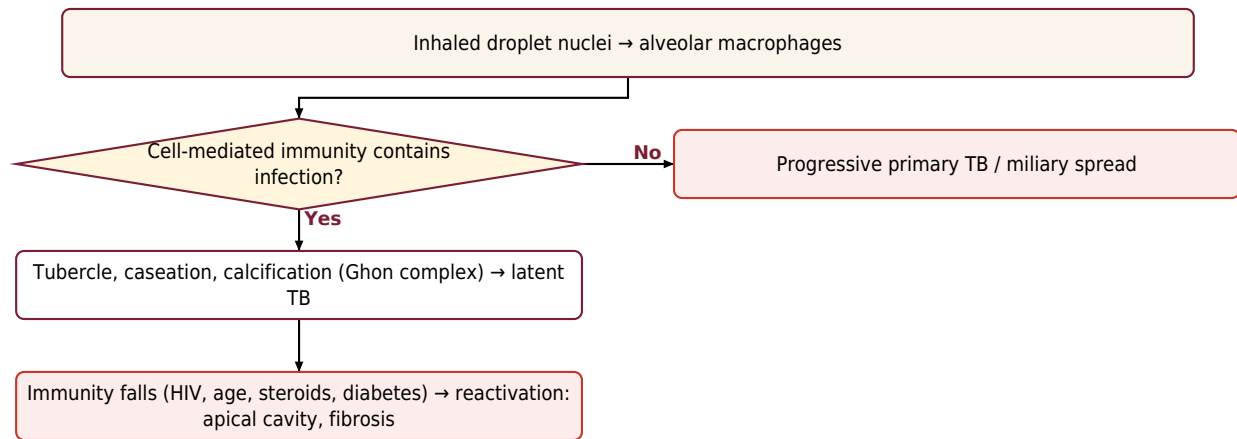
Disseminated (miliary) disease

- Wide **haematogenous spread** in primary or reactivation disease: innumerable 1–3 mm nodules (“millet seeds”). Risk: children, elderly, immunocompromised.
- Primary miliary disease is rapid and severe (multiorgan failure, shock, ARDS); reactivation miliary disease is chronic and nonspecific. Choroidal tubercles are pathognomonic; TB meningitis often seeds after miliary infection.

Effect on lung function

- Untreated, walling-off fails in about 3% of those with disease → spread with destruction and large abscess cavities, then widespread **fibrosis** (GUY).
- Consequences: $\uparrow$ work of breathing with $\downarrow$ vital capacity; $\downarrow$ respiratory membrane area and $\uparrow$ thickness → $\downarrow$ diffusing capacity; abnormal V/Q (GUY). Fibrotic TB constricts the lungs ($\downarrow$ TLC and RV).

Pathogenesis of pulmonary tuberculosis



Our own flowchart drawn from the text of TIN 9e ch. 67, pp. 451–452 and 456.

PEARLS

- ★ Droplet nuclei → macrophages → granuloma (tubercle) → caseation → calcification.
- ★ Ghon complex = calcified focus + hilar nodes.
- ★ Reactivation = 70% of active TB; apical/posterior upper lobe, cavitation.
- ★ Miliary = haematogenous; choroidal tubercles pathognomonic.

Sources

- TIN 9e, ch. 67 "Tuberculosis", §Pathophysiology, §Primary and Latent Infection, Fig. 67-1, p. 451; §Reactivation, §Clinical Features, p. 452; §Chest Radiograph, p. 453; §Miliary Tuberculosis, pp. 456–457. Ch. 63 "Hemoptysis", §Pathophysiology, p. 433.
- GUY 15e, ch. 43 "Respiratory Insufficiency", §Tuberculosis, p. 556; §Maximum Expiratory Flow (constricted lungs), p. 552.

Acute asthma: definition of exacerbation, severity classification and clinical/laboratory parameters of severe asthma

2×

Discuss clinical and laboratory (blood gas and pulmonary functions) parameters in a patient with severe asthma. [3] — Jun 2021 · P-I · Q3(b)

How will you classify severity of bronchial asthma? [3] — May 2024 · P-III · Q6(a)

How will you define acute exacerbation of bronchial asthma? [2] — May 2024 · P-III · Q6(b)

Asked in: Jun 2021 P-I Q3(b); May 2024 P-III Q6(a, b) · 3 + 2 + 3 (severity asks overlap) → 6–7-mark length

Definition of acute exacerbation

- **Asthma** = chronic inflammatory disorder with increased airway responsiveness to multiple stimuli.
- **Acute exacerbation (acute asthma)** = episode of wheezing, breathlessness, chest tightness and cough, often at night or early morning, with **widespread, varying airflow obstruction**; most improve spontaneously or within minutes to hours of treatment.
- **Status asthmaticus** = acute severe asthma not improving with usual doses of inhaled bronchodilators and corticosteroids.

Classification of severity

Table 11. Severity of acute asthma (age >12 years and adults) *Source: TIN 9e Table 69-3 (NHLBI), p. 462*

Severity	Symptoms and signs	Initial PEF (or FEV ₁)	Clinical course
Mild	Dyspnoea only with activity	≥70% predicted/personal best	Prompt relief with inhaled SABA
Moderate	Dyspnoea interferes with or limits usual activity	40–69%	Relief from frequent SABA; symptoms 1–2 d after oral steroids
Severe	Dyspnoea at rest; interferes with conversation	<40%	Partial relief; symptoms ≥3 d after oral steroids
Life-threatening (subset)	Too dyspnoeic to speak; perspiring	<25%	Minimal or no relief; IV steroids, adjuncts; ED/ICU

- Several, not necessarily all, parameters indicate the class. For long-term care TIN uses the chronic categories mild intermittent and mild, moderate or more severe persistent asthma (criteria not tabulated in the sources).

Clinical parameters of severe asthma

- **General:** dyspnoea at rest, inability to speak in full sentences or phrases, perspiring, drowsiness or confusion (impending failure).
- **Signs:** tachycardia, tachypnoea, accessory muscle use, chest retraction, **pulsus paradoxus**; wheeze is unreliable — a **silent chest** means extreme obstruction.
- **High-risk history:** previous severe exacerbation or intubation, ≥2 admissions or >3 ED visits in a year, >2 SABA canisters a month, poor perception of obstruction, low socioeconomic status, illicit drugs, psychiatric or medical comorbidity.
- Look for complications and mimics: pneumothorax, pneumonia, heart failure, upper airway obstruction, vocal cord dysfunction.

Laboratory parameters

Table 12. Pulmonary function and blood gas parameters in severe asthma *Source: TIN 9e pp. 461–463; GUY 15e pp. 553, 556*

Parameter	Severe asthma	Interpretation
PEFR / FEV ₁	<40% predicted (severe); <25% (life-threatening, impending failure)	Key objective measure; repeat at 1 h — the best predictor of admission. FEV ₁ also separates poor effort, restriction, vocal cord dysfunction
FEV ₁ /FVC	Falls from normal 80% to <20% in serious acute asthma	Severe airflow obstruction (GUY)
Lung volumes	↑ FRC and ↑ RV	Air trapping (GUY)
SpO ₂	Low; target ≥90%	Monitors treatment
PaCO ₂	Normally low (hyperventilation); normal or >42 mm Hg is ominous	Extreme obstruction, fatigue, impending ventilatory failure
PaO ₂	Hypoxaemia from V/Q imbalance	Severity
Chest radiograph (selective)	Pneumothorax, pneumomediastinum, pneumonia	Complications/alternatives; abnormal in <1/3 of admitted patients
CBC, ECG (not routine)	Mild leukocytosis; RV strain, P-wave or ST-T changes resolving with treatment	Older patients or heart disease: monitor

- **ABG** is not needed in mild–moderate attacks; reserve it for suspected hypoventilation with CO₂ retention. Capnography's role is unclear (reported differences >10 mm Hg from PaCO₂).

PEARLS

- ★ Severity by PEF: ≥70 mild, 40–69 moderate, <40 severe, <25 life-threatening.
- ★ Normal or rising PaCO₂ (>42) in acute asthma = danger.
- ★ Silent chest, drowsiness, inability to speak = life-threatening.
- ★ Repeat PEF/FEV₁ at 1 h predicts admission.

Sources

• TIN 9e, ch. 69 "Acute Asthma and Status Asthmaticus", §Introduction, §Clinical Features, Tables 69-2, 69-3, 69-4, §Diagnosis and Patient Monitoring, pp. 461–462; Fig. 69-1, p. 463; §Corticosteroids (chronic categories), §Status Asthmaticus,

Acute severe asthma: initial assessment and ED management

Initial assessment and management of acute severe asthma exacerbation. [5] — Oct 2023 · P-III · Q9(a)

Discuss management of acute exacerbation of bronchial asthma. [5] — May 2024 · P-III · Q6(c)

Asked in: Oct 2023 P-III Q9(a); May 2024 P-III Q6(c) · 5 + 5 (repeat) → 4–5-mark length

Initial assessment

- Brief history (onset, triggers, medications, steroid use, previous ICU/intubation), examination (accessory muscles, ability to speak, HR, RR, pulsus paradoxus, silent chest), **PEFR or FEV₁**, SpO₂; ABG only if hypoventilation suspected; chest radiograph if pneumothorax, pneumonia or failure suspected.
- Severe = FEV₁/PEFR <40%; impending arrest = drowsy, confused, exhausted, rising PaCO₂.
- **Repeat assessment** after the first hour (symptoms, examination, PEFR, SpO₂): moderate (40–69%) — SABA every 60 min and oral steroid for 1–3 h, admission decision within 4 h; severe (<40%) — nebulised SABA + ipratropium hourly or continuously, steroids, adjuncts.

Management basis: TIN 9e ch. 69 Acute Asthma and Status Asthmaticus — Fig. 69-1 and Table 69-5 reproduce the NHLBI/NAEPP Expert Panel Report 3 (2007). No Appendix-A guideline covers acute asthma (AHA 2025 Part 10 applies only to cardiac arrest).

Table 13. Drugs for acute severe asthma (adult doses) *Source: TIN 9e Table 69-5, p. 464; text pp. 465–466*

Drug	Dose / route	Notes
Oxygen	To SaO ₂ ≥90%	—
Albuterol (salbutamol)	Neb 2.5–5 mg every 20 min × 3, then 2.5–10 mg every 1–4 h, or 10–15 mg/h continuous; MDI 4–8 puffs every 20 min up to 4 h	MDI + valved holding chamber as effective as nebuliser when well coached
Ipratropium	Neb 0.5 mg every 20 min × 3 (or 8 puffs MDI)	Add to SABA in moderate–severe; no added benefit once admitted
Corticosteroid	Prednisone 40–60 mg PO or methylprednisolone 1 mg/kg IV, within 1 h of arrival	Peak effect 4–8 h; IV no better than oral if absorption normal
Magnesium sulfate	1–2 g IV over 30 min	FEV ₁ /PEFR <25%; monitor BP and reflexes
Epinephrine	0.3–0.5 mg SC/IM every 20 min × 3 (0.5 mg adult for refractory cases)	If unable to use aerosols
Terbutaline	2 µg/kg over 5 min, then 5 µg/kg/h	No proven advantage over aerosol
Ketamine	0.15 mg/kg IV bolus, then 0.25 mg/kg/h	Limited data; induction agent if intubating

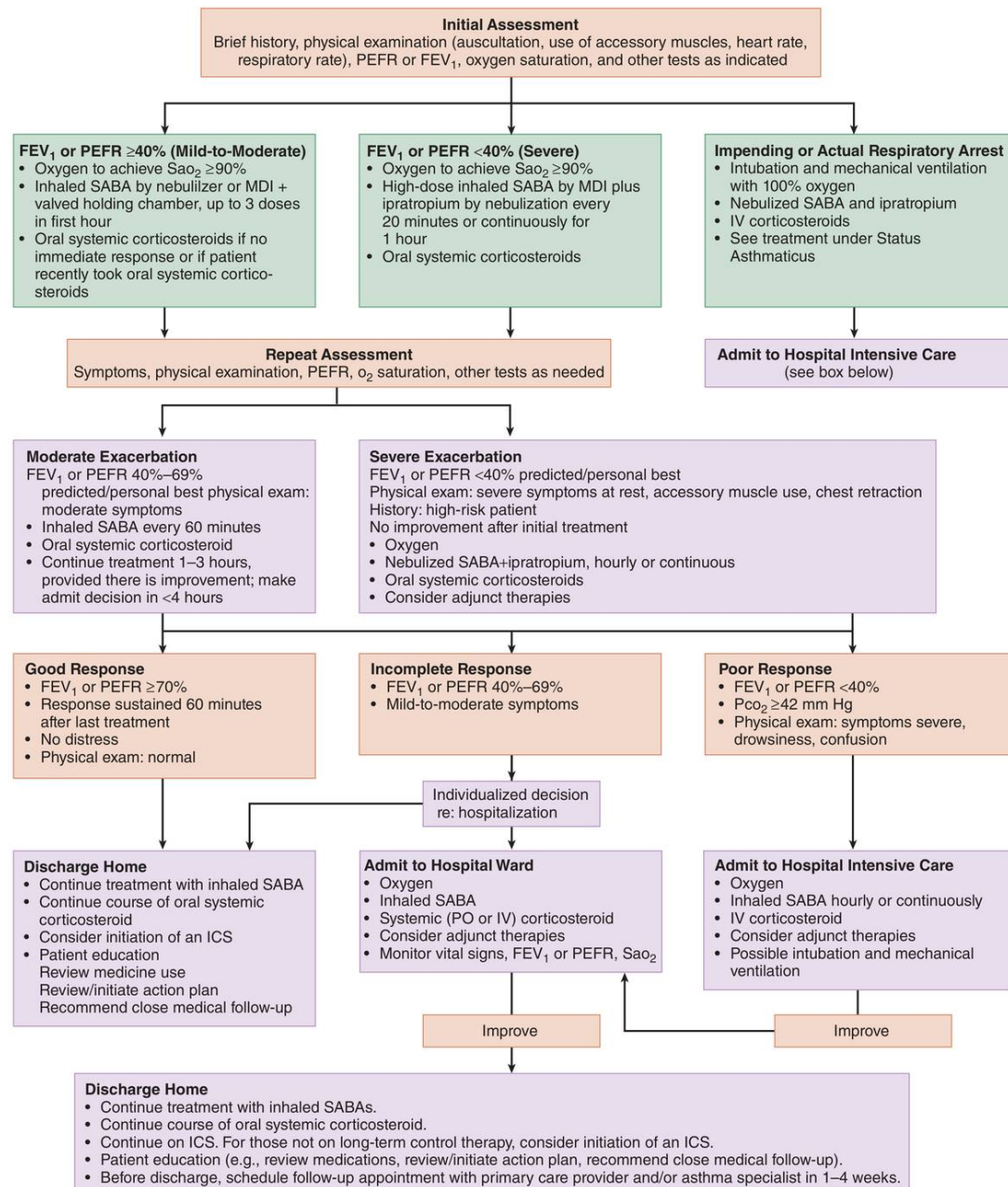


FIGURE 69-1. Management of asthma exacerbations: ED and hospital-based care. FEV₁ = forced expiratory volume in 1 second; ICS = inhaled corticosteroid; MDI = metered-dose inhaler; PEFR = peak expiratory flow rate; SABA = short-acting β_2 -agonist; SaO₂ = oxygen saturation by pulse oximetry. [Source: <http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf> (National Heart, Lung, and Blood Institute; National Institutes of Health; U.S. Department of Health and Human Services: National Asthma Education and Prevention Program, Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma. Publication No. 08-4051. Bethesda, MD, National Institutes of Health, 2007). Accessed November 18, 2014.]

TIN 9e Figure 69-1, p. 463 — Management of asthma exacerbations: ED and hospital-based care (NAEPP EPR-3).

Escalation

- **NIPPV** may avoid intubation; exclude pneumothorax first; not with altered sensorium or near collapse. **Heliox** is an option for non-responders; aminophylline is no longer first- or second-line.
- **Intubate** for progressive hypercarbia or acidosis, exhaustion or confusion. Ventilate with rapid inspiratory flow, low rate (12–14/min), long expiration; target SaO₂ ≥90% and accept hypercapnia (permissive hypoventilation); sedate; watch auto-PEEP, barotrauma, hypotension.

Disposition

- **Good response** (PEFR $\geq 70\%$, sustained 60 min): discharge with SABA, prednisone 40–60 mg/d $\times$ 5–10 d (or dexamethasone 16 mg/d $\times$ 2 d, or depot methylprednisolone 150 mg IM), inhaled corticosteroid, inhaler technique, action plan, follow-up in 1–4 weeks.
- **Incomplete** (40–69%): individualised (observation unit or ward). **Poor** ($<40\%$, $\text{PCO}_2 \geq 42$, drowsy): ICU.

PEARLS

- ★ O_2 + SABA every 20 min + ipratropium + steroid within 1 h.
- ★ Magnesium 1–2 g IV for PEF $<25\%$.
- ★ Rising PaCO_2 , exhaustion or confusion $\rightarrow$ intubate; ventilate slowly.
- ★ Discharge only if PEF $\geq 70\%$ sustained 60 min.

Sources

• TIN 9e, ch. 69 "Acute Asthma and Status Asthmaticus", §Clinical Features, §Diagnosis and Monitoring, pp. 461–462; Fig. 69-1, p. 463; Table 69-5, p. 464; § β -Adrenergic Agents, §Corticosteroids, §Anticholinergics, §Follow-up, §Magnesium, p. 465; §NIPPV, §Ketamine, §Epinephrine, §Mechanical Ventilation, §Agents of Uncertain Benefit, p. 466.

Teaching a patient to use a metered-dose inhaler

1×

How would you teach a patient regarding use of metered-dose inhalers? [3] — Jun 2021 · P-I · Q3(c)

Asked in: Jun 2021 P-I Q3(c) · 3 marks $\rightarrow$ 2–3-mark length

NOT COVERED IN SOURCE

Searched TIN 9e, RH 8e, GUY 15e, ATLS 11e, BDC 10e, STAT 4e and the guideline store for “metered-dose inhaler/MDI” with “technique”, “inhaler technique”, “inhalation technique”, “spacer”, “holding chamber”, “exhale”, “breath-hold”, “shake”; read the TIN index (Asthma, Inhal-, Me- entries; no metered-dose inhaler entry); checked TIN ch. 69 (Acute Asthma), ch. 70 (COPD) and ch. 127 (Wheezing in Infants and Children). The step-by-step inhalation technique is not present. The closest material the sources hold follows.

What the sources say to teach

- **Always use a spacer / valved holding chamber** with the MDI and emphasise its importance; prescribe an age-appropriate chamber (e.g. AeroChamber®) for children.
- With a spacer and optimal use, the MDI delivers the most drug to the target airways — better than a nebuliser; even with optimum technique at most **15%** of the dose is retained in the lungs.
- **Coach the patient:** MDI plus valved holding chamber equals nebulised therapy only with proper technique and coaching by trained personnel; give multiple puffs close together with coaching, then repeat.
- Children <4 years: holding chamber **with facemask**.
- **Teach purpose, teach and check technique** before discharge; reassess technique at follow-up.
- Teach the **peak flow meter**: morning and evening, best of 3 tries, recorded in a diary; give a written **action plan**.
- Reserve nebulisers for patients who cannot use an MDI, when it fails, or at extremes of age.

PEARLS

- ★ Spacer/valved holding chamber with every MDI.
- ★ Teach, then watch the patient demonstrate; recheck at follow-up.
- ★ Facemask with chamber under 4 years.

Sources

• TIN 9e, ch. 69, Table 69-5, p. 464; § β -Adrenergic Agents (aerosol delivery), §Follow-up, p. 465; Table 69-6 (discharge checklist), p. 466.
• TIN 9e, ch. 70, § β_2 -Agonists, p. 469; §Disposition, p. 470; ch. 127 "Wheezing in Infants and Children", §Disposition, p. 808 (and p. 804).

Definition of acute exacerbation of chronic obstructive pulmonary disease. [1] — Oct 2024 · P-III · Q1(a)

Differential diagnoses of acute exacerbation of COPD. [3] — Oct 2024 · P-III · Q1(b)

Treatment of acute exacerbation of COPD. [4] — Oct 2024 · P-III · Q1(c)

Indications and relative contraindications for NIV in acute exacerbation of COPD. [2] — Oct 2024 · P-III · Q1(d)

Acute exacerbation of chronic obstructive pulmonary disease (COPD). [5] — Jun 2025* · P-III · Q4(b)

Asked in: Oct 2024 P-III Q1(a, b, c, d); Jun 2025* P-III Q4(b) · 1 + 3 + 4 + 2 (+ 5, overlapping short note) → 10-mark length

Definitions

- **COPD:** common, preventable, treatable disease with persistent respiratory symptoms and airflow limitation from airway and/or alveolar abnormalities, usually after significant exposure to noxious particles or gases; confirmed by post-bronchodilator $FEV_1/FVC < 0.7$.
- **Acute exacerbation:** worsening of respiratory symptoms **beyond normal day-to-day variation**, usually triggered by infection or an irritant.
- **Triggers:** infection in >75% (up to half bacterial); hypoxia, cold weather, β -blockers, opioids, sedative-hypnotics; GERD a possible risk factor.

Pathophysiology and assessment

- Inflammatory mediators → bronchoconstriction, pulmonary vasoconstriction, mucus hypersecretion; ↑ airway resistance and hyperinflation ↑ work of breathing and CO_2 production → hypercapnia. Exacerbations are chiefly **V/Q mismatch**.
- **Most life-threatening feature: hypoxaemia** ($SaO_2 < 90\%$) — tachypnoea, tachycardia, hypertension, cyanosis, altered mentation. Alveolar hypoventilation → CO_2 retention, respiratory acidosis.
- Tripod posture, pursed-lip breathing, accessory muscles, pulsus paradoxus (>10 mm Hg fall).
- **ABG** is the best acute tool: respiratory failure = $PaO_2 < 60$ mm Hg or $SaO_2 < 90\%$ on air; respiratory acidosis = $PCO_2 > 44$; pH < 7.35 = acute uncompensated component. Venous pH agrees well (screening).
- Acute respiratory acidosis: HCO_3^- rises 1 mEq/L per 10 mm Hg PCO_2 (chronic: 3.5 mEq/L); values outside these ranges suggest an added metabolic disorder.
- Bedside spirometry is usually inaccurate when dyspnoeic. Sputum volume and colour change suggest bacterial infection. CXR, ECG, CBC, electrolytes; troponin, BNP, D-dimer, CT as indicated; theophylline level if used.

Differential diagnosis

Table 14. Critical differential diagnosis of AECOPD Source: *TIN 9e Table 70-3, p. 469; text p. 468*

Diagnosis	Distinguishing features	Caveat
Asthma	Earlier onset, variable symptoms, family history, reversible airflow	Can coexist (asthma–COPD overlap)
Heart failure	Orthopnoea (LR 2.0), JVD, hepatojugular reflux, basal crackles; cardiomegaly/oedema on CXR; BNP <100 unlikely, >500 likely	Can coexist; BNP altered by many conditions
Pulmonary embolism	Risk factors: age, surgery/trauma, prior VTE, cancer, oestrogen; CTPA if intermediate/high probability	20–25% of severe exacerbations with unclear trigger have PE
Acute coronary syndrome	ECG, troponin in those with chest pain or dyspnoea and risk factors	Dyspnoea may be the only complaint
Pneumothorax	Chest radiograph, US or CT	COPD predisposes to spontaneous pneumothorax
Pneumonia	Chest radiograph	Frequently coexists

Treatment in the ED

Management basis: TIN 9e ch. 70 Chronic Obstructive Pulmonary Disease (Tables 70-2, 70-4 to 70-7 derived from GOLD); RH 8e ch. 8 for NIV procedure. GOLD is not an Appendix-A document.

- **Goals:** correct tissue oxygenation, relieve reversible bronchospasm, treat the trigger. Therapy is shaped by mental status, degree of reversible bronchospasm, recent drugs and toxicity (theophylline), previous courses, admissions and intubations, and complications. A patient who does not respond as expected needs prompt re-evaluation for another life-threatening process.

Table 15. ED management of AECOPD Source: TIN 9e Table 70-2, pp. 469–470

Step	Action
Controlled oxygen	Target SaO ₂ 88–92% (PaO ₂ 60–70 mm Hg) by nasal cannula, simple or Venturi mask; higher targets may harm (O ₂ can worsen hypercapnia through V/Q mismatch — use non-rebreathers cautiously). ABG after 20–30 min if SaO ₂ <90% or hypercapnia suspected
Short-acting β ₂ -agonist	First line; every 30–60 min if tolerated (every 20 min speeds FEV ₁ gain but more tremor, palpitations); MDI with spacer and coaching preferred if cooperative; drive nebulisers with air and give O ₂ separately
Ipratropium	0.5 mg (2.5 mL of 0.02%) nebulised or MDI; combine with SABA. No long-acting anticholinergics acutely
Corticosteroid	Oral prednisone 40–60 mg/day for 5–7 days; IV only if unable to take orally or in shock. Hyperglycaemia commonest adverse effect
Antibiotics	If ↑ sputum volume and purulence, fever or infection suspected: azithromycin, doxycycline or trimethoprim–sulfamethoxazole (<i>S. pneumoniae</i> , <i>H. influenzae</i> , <i>M. catarrhalis</i>); antipseudomonal cover if admission/antibiotics in 3 months, prior <i>Pseudomonas</i> , bronchiectasis; 3–14 days
Ventilatory support	NIV early (below); intubation for Table 70-5 indications
Not routine	Magnesium (2–4 g IV), aminophylline (3–5 mg/kg IV); HFNC role unclear

NIV: indications and relative contraindications

Table 16. Noninvasive ventilation in AECOPD Source: TIN 9e Table 70-4, p. 470

Indications (selection criteria)	Relative contraindications (exclusion: any)
Acidosis pH <7.36 / hypercapnia PaCO ₂ >50 mm Hg / oxygenation deficit (PaO ₂ <60 mm Hg or SaO ₂ <90%)	Respiratory arrest; cardiovascular instability (hypotension, arrhythmia, MI)
Severe dyspnoea with respiratory muscle fatigue or increased work of breathing	Altered mental status or uncooperative; high aspiration risk; viscous or copious secretions
	Recent facial or gastro-oesophageal surgery; craniofacial trauma; fixed nasopharyngeal abnormality; burns; extreme obesity

- Early NIV with medical therapy reduces intubation, hospital stay and in-hospital mortality — standard of care in AECOPD without contraindications; started late, the benefit is lost (RH).
- **Settings (RH):** low–high IPAP 10 / EPAP 5 or high–low IPAP 20–25 / EPAP 5; increase pressure support (IPAP – EPAP) for hypercapnia; EPAP 8 if intrinsic PEEP; reassess at 30 min and ABG at 1–2 h — intubate if no improvement.
- **Intubate** for NIV failure or intolerance, arrest, decreased consciousness or agitation, massive aspiration, unclearable secretions, hypotension or haemodynamic instability, persistent hypoxaemia. Ventilate at 10–14/min, tolerate pH ≥7.20, check auto-PEEP.

Disposition

- **Admit:** marked ↑ symptoms (resting dyspnoea), failure of initial therapy, significant comorbidity, new arrhythmia or heart failure, frequent exacerbations, older age, poor home support.
- **ICU:** severe dyspnoea despite treatment, altered mental status, PaO₂ <40 mm Hg and/or pH <7.25 despite O₂ and NIPPV, need for invasive ventilation, haemodynamic instability.
- **Discharge:** home O₂ if needed, MDI with spacer and teaching, short oral steroid course, follow-up within a week; reinforce completion of steroids and antibiotics.
- 25–43% relapse after ED discharge; higher risk with ≥5 ED or clinic visits in the past year, more activity limitation, higher initial respiratory rate, and oral steroid use before arrival.

PEARLS

- ★ AECOPD = symptoms beyond day-to-day variation; >75% infective.
- ★ SaO_2 88–92%; ABG at 20–30 min.
- ★ SABA + ipratropium + prednisone 40–60 mg $\times$ 5–7 d $\pm$ antibiotics.
- ★ NIV for pH <7.36 / PaCO_2 >50 / hypoxaemia — early, reassess at 30 min.
- ★ Unexplained severe exacerbation: think PE (20–25%).

Sources

- TIN 9e, ch. 70 "Chronic Obstructive Pulmonary Disease", §Introduction, §Pathophysiology, §Diagnosis, p. 467; §Acute Exacerbations, §Clinical Features, §Diagnosis, p. 468; Tables 70-2, 70-3, §Treatment, §Oxygen, § β_2 -Agonists, §Anticholinergics, p. 469; §Corticosteroids, §Antibiotics, §NIV and Invasive Ventilation, §Other Therapies, §Disposition, Tables 70-4 to 70-6, p. 470; Table 70-7, p. 471. Ch. 29B, Table 29B-4, p. 192.
- RH 8e, ch. 8 "Mechanical Ventilation", §Acute Exacerbation of COPD, p. 170; §Initiation of NPPV, Fig. 8.13, Box 8.3, p. 171.

Part 10 · Gastrointestinal & Hepatobiliary Emergencies

28 answers

Describe the radiological features on X-ray and ultrasound in a patient with acute abdomen. [5] — Jun 2021 · P-II · Q7(a)

Asked in: Jun 2021 P-II Q7(a) · 5 marks → 4–5-mark length

Principles

- Imaging does not replace history and examination; if surgery is obviously needed, **call the surgeon without waiting for imaging**.
- Plain radiographs should be limited to screening for **obstruction, sigmoid volvulus, perforation** or severe constipation; include the **inguinal region** (incarcerated hernia).
- US: gallbladder, pancreas, kidneys, ureters, bladder volume, aorta — but **not useful for bowel disorders**; operator- and habitus-dependent.

Table 17. Plain radiograph (X-ray) features *Source: TIN 9e pp. 476, 478, 494, 515, 524, 531; pp. 416–417, 858–859*

Condition	X-ray finding	Limitation / note
Perforated viscus	Free air under the diaphragm on upright chest film	Upright chest better than abdominal film, but only ~30% sensitive for small amounts; 60–75% of perforated ulcers show free air
Small bowel obstruction	Dilated loops, air–fluid levels	Signs 6–12 h before symptoms, but absent in up to half; plain films 77% sensitive vs CT 93%
Large bowel obstruction / constipation	Colonic or rectal dilatation ± air–fluid levels	Normal maximum colon 6 cm, rectum 4 cm
Adynamic ileus	Diffusely dilated loops with air in distal colon and rectum	Treated supportively
Gallstone ileus	Rigler's triad: SBO, pneumobilia, ectopic gallstone	CT more dependable
Abdominal aortic aneurysm	Calcified, bulging aortic contour (clearer on lateral view)	~65% calcified; a plain film cannot exclude AAA
Intussusception (child)	Paucity of right-sided gas, “crescent sign”, obstructive pattern	US is first test
Necrotising enterocolitis	Pneumatosis intestinalis , portal venous gas	Pathognomonic; normal film does not exclude
Appendicitis	Nonspecific gas pattern; appendicolith (up to 50% of children)	Plain films not helpful

Table 18. Ultrasound features *Source: TIN 9e pp. 474, 476, 477, 515, 524, 535, 859–860*

Condition	US finding
Acute appendicitis	Non-compressible, blind-ending tube >6 mm ; wall ≥3 mm; hyperaemia; periappendiceal fluid, echogenic fat; appendicolith with shadowing
Acute cholecystitis	Gallstones + sonographic Murphy's sign (PPV 92%); wall >3 mm; pericholecystic fluid; distension >40 mm short axis
Ruptured/leaking AAA	Aorta ≥3 cm (outer wall to outer wall); <3 cm excludes; free fluid on FAST
Haemoperitoneum (e.g. ectopic, splenic rupture)	Free intraperitoneal fluid (FAST); intra- or extrauterine gestational sac on transvaginal US
Urinary tract	Hydronephrosis; distended bladder (volume)
Intussusception	Accuracy near 100% in research settings
Incarcerated hernia	Bowel in sac; free fluid in sac, no peristalsis = incarceration/strangulation
Shock assessment	Cardiac function and IVC size/respiratory variation (resuscitative POCUS)

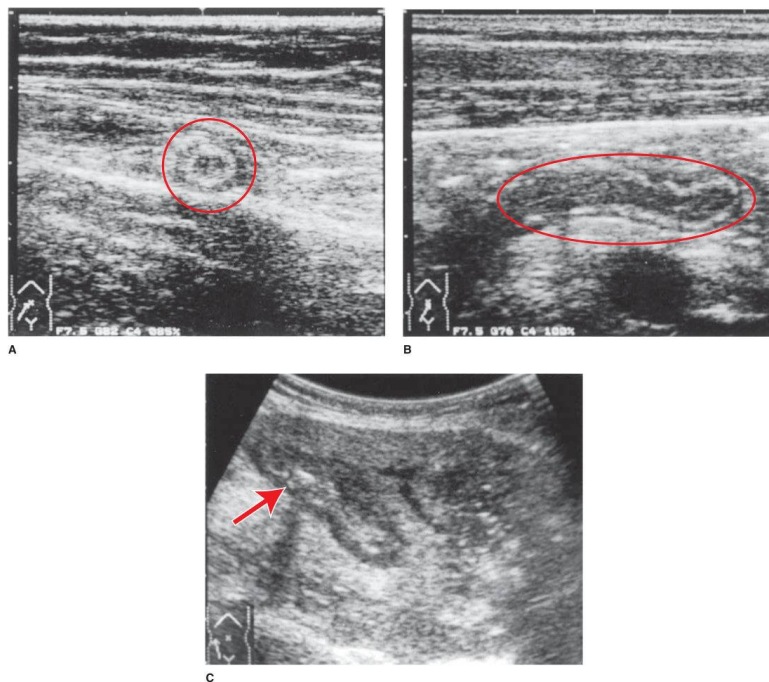


FIGURE 81-1. Ultrasonographic demonstration of acute appendicitis. A noncompressible, inflamed appendix (red circles) is shown in a cross-sectional view (A; 7.5 MHz) and a longitudinal section (B; 7.5 MHz). Mural lamination of the swollen appendix is maintained in the early stages of acute appendicitis. C. An appendicolith (arrow) with acoustic shadowing is demonstrated (5 MHz). [Reprinted with permission from Ma OJ, Mateer JR, Reardon RF, Blaivas M. *Emergency Ultrasound*. 3rd ed. Copyright © The McGraw-Hill Companies, 2014. All rights reserved. Chapter 11, General Surgery Applications, Figures 11-31A&B & 11-33.]

TIN 9e Figure 81-1, p. 525 — US in acute appendicitis: non-compressible appendix (A, B) and appendicolith with shadowing (C).

- **Beyond X-ray and US:** IV-contrast CT is the initial test for suspected AAA rupture or mesenteric ischaemia and shows bowel obstruction with its transition point.

PEARLS

- ★ Upright chest film for free air; still only ~30% sensitive — CT if suspicion persists.
- ★ X-ray views must include the groin.
- ★ US first for biliary disease, AAA, urinary tract, appendicitis in children and pregnancy.
- ★ Appendix >6 mm non-compressible; gallbladder wall >3 mm with Murphy's sign; aorta ≥3 cm.

Sources

- TIN 9e, ch. 71 "Acute Abdominal Pain", §Diagnostic Imaging (Plain Radiographs, Ultrasound, POCUS, CT), pp. 476–477; ch. 60, §Imaging, pp. 416–417; ch. 74 "Constipation", p. 494; ch. 79, §Imaging, Table 79-7, pp. 514–515; ch. 81, §Imaging, pp. 524–525, Figure 81-1, p. 525; ch. 83, §Imaging, p. 531; ch. 84, §Imaging, p. 535; ch. 133, §Necrotizing Enterocolitis, §Intussusception, pp. 858–859.
- TIN 9e, ch. 78, §Perforation, p. 508; Table 71-4, p. 478.

67-year-old with acute upper abdominal pain radiating to the back, pulse 120/min, BP 96/60: differential diagnosis, investigation and ED management

1×

[A 67-year-old patient presents to the emergency with acute onset of upper abdominal pain for the past 4 hours. The pain was non-colicky and radiated to the back. On examination, his pulse was 120/minute and blood pressure was 96/60 mmHg.] Discuss differential diagnosis of abdominal pain in this patient. [5] — Dec 2022 · P-II · Q2(a)

How will you investigate him? [3] — Dec 2022 · P-II · Q2(b)

How will you manage him in the emergency department? [2] — Dec 2022 · P-II · Q2(c)

Asked in: Dec 2022 P-II Q2(a, b, c) · 5 + 3 + 2 → 10-mark length

Reading the case

- Elderly patient, acute severe non-colic pain, **radiation to the back**, tachycardia and borderline hypotension = **pain with shock**: a high-acuity patient — resuscitate and evaluate simultaneously.
- Shock developing rapidly after abdominal pain is usually **intra-abdominal haemorrhage**; SBP falls only after 30–40% blood loss.
- In the elderly, presentations are atypical and fever unreliable; above 80 years mortality almost doubles if the admission diagnosis is wrong.

Differential diagnosis

Table 19. Differential diagnosis — upper abdominal pain radiating to the back with shock *Source: TIN 9e Tables 71-2, 71-4, pp. 476, 478–479; pp. 412, 416, 498, 506–509*

Diagnosis	Supporting features	Key bedside clue / first test
Ruptured/leaking AAA (first to exclude)	Older male, smoker, vascular disease; abrupt severe constant pain to back, flank or groin; only 50% hypotensive; syncope	Bedside US (100% sensitive in Table 71-4)
Acute aortic dissection	Abrupt, worst-ever, tearing pain; abdominal pain commoner in type B; pulse deficit, neurological signs, syncope	CT angiography; widened mediastinum on CXR
Acute (haemorrhagic) pancreatitis	Alcohol or gallstones; severe persistent epigastric pain to the back; vomiting; eased sitting up with knees flexed; Grey Turner/Cullen signs (late, severe)	Lipase (90% sensitive in first 24 h)
Perforated peptic ulcer	NSAIDs, smoking, H. pylori; abrupt severe epigastric pain; rigid abdomen; elderly may lack signs	Upright CXR free air (60–75%); CT 98%
Posterior penetrating ulcer	Rapid-onset mid-back pain from penetration into pancreas	Lipase, CT
Mesenteric ischaemia	AF, vascular disease; pain out of proportion to examination; shock late	Lactate (86% sensitive); CT angiography
Myocardial ischaemia/infarction	Upper midline steady dull pain; diaphoresis; cardiogenic shock	ECG, troponin
Ruptured visceral (splenic/hepatic) artery aneurysm	Epigastric/LUQ pain, then diffuse pain and shock (splenic); RUQ pain, haemobilia (hepatic)	Bedside US for intraperitoneal haemorrhage; CT
Biliary: cholecystitis/cholangitis with sepsis	RUQ/epigastric pain radiating to the right scapula; fever, jaundice	US
Others	Renal colic (only a misdiagnosis trap here), DKA, pneumonia, pulmonary embolism	Glucose, CXR, urinalysis

Investigation

1. **Bedside US immediately** (in the resuscitation bay): aortic diameter (≥ 3 cm = AAA), free intraperitoneal fluid, cardiac function, IVC — identifies AAA and haemorrhage in hypotensive patients.
2. **12-lead ECG** (upper abdominal pain in the elderly) and troponin.
3. **Bloods with the IV line**: CBC with platelets, electrolytes, BUN/creatinine, clotting, **type and cross-match** (haemorrhage suspected); lipase; LFT; lactate (normal in up to 25% of mesenteric ischaemia); glucose; ABG/venous gas.
4. **Upright chest X-ray** for free air (and mediastinal width) when perforation or dissection is suspected.
5. **CT with IV contrast** (CT angiography) only once stable or if US is non-diagnostic: test of choice for AAA rupture, dissection, mesenteric ischaemia; CT shows free air missed on X-ray. **Unstable patients must not leave the ED for imaging.**
6. Urinalysis; if renal colic is suggested in this age group, image the aorta first.

Management in the emergency department

Management basis: TIN 9e ch. 71 (Acute Abdominal Pain) and ch. 60 (Aneurysmal Disease), Table 60-1. No Appendix-A guideline covers this topic.

1. **ABC:** monitor, oxygen 2–4 L/min, **two large-bore IV lines**, bloods drawn at cannulation.
2. **Fluids:** isotonic bolus adjusted to age and cardiac status; cross-matched blood if haemorrhage. If AAA rupture is likely, **permissive hypotension — SBP 80–90 mm Hg** with intact mentation.
3. **Early surgical/vascular consultation** on suspicion — do not wait for imaging; if no vascular service, resuscitate and transfer immediately.
4. **Analgesia:** opioids titrated (do not withhold; avoid severe hypotension and respiratory depression). Antiemetic: ondansetron 4–8 mg IV or metoclopramide 10 mg IV slowly.
5. Nasogastric tube (perforation, obstruction) and urinary catheter (hourly output).
6. **Cause-specific:** perforation or peritonitis → broad-spectrum antibiotics (e.g. piperacillin-tazobactam 3.375–4.5 g IV 6-hourly) + surgery; pancreatitis → fluids and analgesia; dissection → see TIN ch. 59; MI → ACS pathway.
7. **Disposition:** theatre, ICU or transfer; no high-risk elderly patient goes home with an unclear diagnosis.

PEARLS

- ★ Elderly + abdominal/back pain + shock = ruptured AAA until bedside US says otherwise.
- ★ Bedside US, ECG and cross-match come first; CT only for the stable patient.
- ★ Permissive hypotension (SBP 80–90 mm Hg) for suspected AAA rupture.
- ★ Lipase 90% sensitive for pancreatitis; free air in only 60–75% of perforated ulcers.
- ★ Normal lactate does not exclude mesenteric ischaemia.

Sources

- TIN 9e, ch. 71 "Acute Abdominal Pain", §Clinical Risk, §Symptom Treatment, §Laboratory Testing, §Diagnostic Imaging, §Elderly Patients, pp. 473–477; Tables 71-2, 71-3, p. 476; Table 71-4, pp. 478–479; Table 71-5, p. 480.
- TIN 9e, ch. 60, §Symptomatic AAA, §Extremity and Visceral Aneurysms, pp. 416–419, Table 60-1; ch. 59 "Aortic Dissection", §Clinical Features, §Imaging, pp. 412–413; ch. 78, §Diagnosis, §Ancillary Testing, p. 506, §Perforation, p. 508; ch. 79, §Clinical Features, §Diagnosis, pp. 508–509; ch. 76, §Mesenteric Ischemia, p. 498.

Differential diagnosis of right lower quadrant acute abdominal pain (adult male)

2×

Differential diagnosis of right lower quadrant acute abdominal pain in an adult male patient. [6] — May 2024 · P-II · Q2(a)

Enumerate the differential diagnosis of right lower quadrant pain. [5] — Dec 2025* · P-II · Q10(b)

Asked in: May 2024 P-II Q2(a); Dec 2025* P-II Q10(b) · 6 and 5 (same ask) → 6-7-mark length

Approach

- **Appendicitis** is the leading cause and must be considered in anyone with right-sided abdominal, periumbilical or flank pain who still has an appendix.
- Pain from midgut organs (appendix, caecum, ileum) is first **periumbilical** (visceral, T10) and then **localises** to the RLQ when the parietal peritoneum is involved.
- **Referred pain:** ureter and testis share segmental innervation — ureteric colic radiates to the testis, and testicular torsion may present as lower abdominal pain.
- In a male, examine the **hernial orifices, testes and prostate**; in the elderly, think first of the aorta.
- **Life threats first:** leaking AAA, strangulated hernia, testicular torsion, perforation, mesenteric/ischaemic colitis.

Table 20. Differential diagnosis of RLQ pain in an adult male *Source: TIN 9e Figure 71-1, p. 475; Table 81-1, p. 524; Table 71-4, pp. 478–479*

System	Diagnosis	Distinguishing clue
GI	Acute appendicitis	Migration periumbilical → RLQ, anorexia, pain before vomiting, RLQ tenderness, rigidity
GI	Caecal or Meckel's diverticulitis	Incidence rises with age; 50% report a previous similar episode; CT
GI	Crohn's disease (terminal ileitis)/colitis	Appendicitis mimic; CT
GI	Mesenteric lymphadenitis	Appendicitis mimic; CT
GI	Bowel obstruction, caecal volvulus, constipation/ileus	Colicky pain, distension, vomiting
GI	Epiploic appendagitis	Focal pain, not systemically ill; CT oval fatty lesion
GI	Intra-abdominal abscess	CT
GI	Intussusception, malrotation (rare in adults)	Obstructive picture
Hernia	Incarcerated/strangulated inguinal hernia	Tender irreducible groin lump; include groin on films
Vascular	Leaking/ruptured AAA ; ischaemic colitis	Older male, smoker; shock; pulsatile mass; bedside US
Urological	Ureteric calculus	Colicky flank → groin pain, haematuria (85–90%)
Urological	Pyelonephritis	Pyuria on urinalysis
Urological	Testicular torsion (referred pain)	Scrotal examination: high-riding tender testis
Musculoskeletal	Psoas abscess ; abdominal wall/rectus sheath haematoma	Psoas sign; abdominal wall examination
Others	Herpes zoster, regional enteritis	Skin examination; CT

Clues that separate the commonest causes

- Single most useful sign in adult appendicitis: **RLQ pain** (LR+ 7.31–8.46), then rigidity and migration; a prior similar episode makes appendicitis less likely.
- **Psoas sign** (pain on passive right hip extension) and **obturator test** (pain on rotation of the flexed hip) indicate an inflamed retroperitoneal or pelvic process.
- **Urinalysis**: isolated microscopic haematuria supports renal colic; pyuria suggests pyelonephritis — but haematuria or sterile pyuria occurs in appendicitis too.
- **CT abdomen/pelvis** (non-contrast acceptable for appendicitis; non-contrast is preferred for stones) resolves most uncertainty; US for aorta, kidney and testis.

PEARLS

- ★ Appendicitis until proven otherwise — but look at the groin, testis and aorta in every man with RLQ pain.
- ★ Referred pain: ureteric colic → testis; torsion → lower abdomen.
- ★ Microscopic haematuria does not exclude appendicitis.

Sources

- TIN 9e, ch. 71 "Acute Abdominal Pain", §Pathophysiology (Visceral, Parietal, Referred Pain), §History and Physical Examination, pp. 473–474; Figure 71-1, p. 475; Table 71-4, pp. 478–479.
- TIN 9e, ch. 81 "Acute Appendicitis", §Pathophysiology, §Clinical Features, §Diagnosis, §Laboratory Testing, pp. 523–524; Table 81-1, p. 524; ch. 84, p. 532; ch. 93, §Testicular Torsion — Clinical Features, p. 595.

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Brachial plexus injuries: aetiology and clinical features	Jun '20 PI Q6(c); Dec '22 PII Q5(a)	101
Fractures of the hip	Dec '21 PII Q1(a); May '24 PII Q7(a); Oct '24 PII Q2(a)	103
Anterior and posterior cruciate ligament injuries	Oct '24 PII Q2(b)	105
Ottawa and Pittsburgh rules for knee and ankle injuries	Jun '20 PII Q8(c,d); Jun '22 PII Q9(b); Apr '23 PII Q10(b)	107

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Compartment syndrome after crush injury: pathophysiology, investigations, treatment	Dec '20 PII Q9(a,b,c); Dec '25* PII Q3(a)	112
Acute low back pain: causes, red flags, differential diagnosis	Dec '20 PII Q6(a,b); Jun '25* PII Q10(a)	115
Cauda equina syndrome	Dec '20 PII Q6(c); Oct '24 PII Q4(b)	116
Rotator cuff injuries: risk factors, clinical assessment, imaging and management	Jun '20 PI Q6(a); Jun '21 PII Q10(b,c,d); Dec '22 PII Q10(a); May '24 PI Q5(b)	118
Bursal syndromes of the hip and knee	May '24 PII Q6(a)	120
Acute joint pain and monoarthritis: differential, synovial fluid, approach	Jun '21 PII Q8(a,b,c)	122
Septic arthritis of the knee: ED management	Jun '20 PII Q2(a)	125
Acute gout: clinical features and ED management	May '24 PIII Q7(a); Dec '25* PIII Q8(a)	126
Colchicine and corticosteroids in acute gout	Dec '25* PIII Q8(b)	128

Part 23 · Psychiatric & Behavioural Emergencies

Answer	Asked in	Page
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Delirium: definition, causes, assessment (CAM, RASS), evaluation and management in the elderly	Jun '20 PIII Q9(a,b,c); Dec '21 PIII Q4(a); Dec '22 PIII Q5(a,b,c); Oct '23 PIII Q8(a)	134
Anxiety disorders (case with Chvostek and Trousseau signs): classification, ED assessment and management, organic vs primary	Jun '21 PIII Q10(a,b,c)	137
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Role of palliative care in the emergency department	Dec '22 PIV Q10(b)	144
Symptom management in terminal cancer	Oct '24 PIV Q5(b)	145
Grief counselling in the ED and its methods	Jun '20 PIV Q7(a); Jun '22 PIV Q6(b)	146

Part 25 · Trauma (ATLS order)

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Management of haemorrhagic shock in trauma	Jun '21 PII Q1(a); Jun '22 PII Q1(b)	152
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Neurogenic shock vs spinal shock	Dec '20 PII Q1(a); Oct '23 PI Q9(a); Jun '25* PII Q1(b)	157
Shifting a patient with cervical spine injury	Jun '25* PII Q3(b)	158
Hip dislocation and the dashboard injury	Jun '20 PII Q7(c); Dec '21 PII Q3(b); May '24 PII Q7(b); Dec '25* PII Q7(a)	160
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Role of ultrasound and POCUS signs in chest trauma	Dec '20 PII Q4(c); Jun '22 PII Q4(a)	164
Complications of prolonged tourniquet application	Dec '25* PII Q7(b)	166
REBOA: indications, technique, complications	Jun '22 PII Q3(b); Oct '24 PIV Q10(b)	167
Airway challenges in facial trauma	Dec '25* PII Q9(b)	169
Significant clinical features in facial trauma	Oct '24 PII Q7(b)	170
Le Fort fractures	Dec '22 PII Q6(a); Oct '24 PII Q7(c); Jun '25* PII Q4(a)	171
Open pneumothorax	Dec '20 PII Q4(a)	173
Tension pneumothorax: identification and treatment	Oct '23 PII Q3(a)	174
Flail chest	Oct '24 PII Q7(a); Jun '25* PII Q1(c)	175
Rib fractures and acute rib-fracture pain	Dec '20 PII Q4(b); Jun '22 PII Q8(b)	177
Traumatic diaphragmatic injury	Dec '25* PII Q6(a)	179
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Acid–base disorders and coagulopathy in trauma	Dec '20 PI Q6(c); Jun '22 PI Q6(b)	185
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Balanced and haemostatic–hypotensive resuscitation	Oct '24 PI Q5(b); Dec '25* PI Q6(b)	199
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Management of life-threatening cervical spine injury	Dec '21 PII Q10(a)	203
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Paediatric GCS versus adult GCS: key differences	Jun '22 PIV Q4(a)	213
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Canadian CT Head Rule vs New Orleans Criteria	Jun '21 PII Q2(c); May '24 PII Q10(b)	216
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Mannitol and hypertonic saline	May '24 PIV Q6(a)	221
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End-tidal CO2 and capnography	Dec '22 PI Q4(c); Oct '23 PI Q3(b); Oct '24 PIV Q6(a,b,c,d)	265
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High-flow nasal oxygen	Dec '21 PIII Q2(b)	270
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Delayed sequence intubation	Dec '22 PIV Q8(b); Oct '24 PI Q5(a)	282
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Airway ultrasound	Jun '20 PIV Q6(a); Jun '22 PIV Q1(a)	285
Etomidate; ketamine vs etomidate	Dec '20 PIV Q10(b); Dec '25* PI Q8(b)	286
Succinylcholine	Jun '25* PI Q4(b)	288
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Initial ventilator settings in acidosis	Jun '20 PI Q4(b,c); Jun '22 PI Q3(b)	296
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Transcutaneous pacing: equipment, technique, outcomes	Jun '25* PIV Q7(a)	299
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Epistaxis: arterial supply of nasal septum (diagram), aetiology, management	Jun '21 PII Q4(a,b); Dec '22 PII Q8(a); Oct '23 PII Q8(a)	340
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TMJ dislocation: causes and ED management	Dec '21 PII Q5(a)	344
Avulsed tooth: scene care, prehospital care and replantation	Dec '22 PII Q9(b); Apr '23 PII Q6(a); Jun '25* PII Q8(b)	345
Restraints in the ER, including types of restraint for an agitated adolescent	Dec '20 PIII Q10(b); Dec '21 PIII Q5(b)	347

Part 27 • Landmark Trials & Evidence

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Trials of oxygen and blood-pressure targets after ROSC	Dec '22 PIV Q7(a)	353
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WATERFALL trial	Oct '24 PIV Q9(a)	356
E-MOTIVE trial	Oct '24 PIV Q9(b)	357
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CRASH-3 trial	Jun '21 PIV Q10(a); Dec '22 PIV Q7(b)	362
WOMAN trial	Jun '21 PIV Q10(d)	363

C • High-yield (asked 3× or more)

Answer	Times asked	Page
Tumour lysis syndrome: criteria, investigations and ED management	5×	48
Thyroid storm: precipitants, clinical features, Burch-Wartofsky Point Scale and ED management	4×	22
Adrenal insufficiency and acute adrenal crisis: recognition, ED approach, management, hydrocortisone and fluids	4×	25
Acute painful red eye: differential diagnosis and management	4×	54
Periorbital versus orbital cellulitis	4×	57
Acute visual loss: differential diagnosis and management	4×	58
Rotator cuff injuries: risk factors, clinical assessment, imaging and management	4×	118
Acute agitation in the ED: approach, assessment, management, rapid tranquillisation and pharmacological restraint	4×	131
Delirium: definition, causes, assessment (CAM, RASS), evaluation and management in the elderly	4×	134
Hip dislocation and the dashboard injury	4×	160
Ocular ultrasound: technique and utility in trauma	4×	336
Disseminated intravascular coagulation: aetiopathogenesis, diagnosis and ED management	3×	32
Modified Lynch algorithm: approach to the erythematous, life-threatening and undifferentiated rash	3×	75
Stevens-Johnson syndrome and toxic epidermal necrolysis: causes, clinical features and treatment	3×	77
Fractures of the hip	3×	103
Ottawa and Pittsburgh rules for knee and ankle injuries	3×	107
ED treatment of TBI: targets, pearls and pitfalls	3×	154
Neurogenic shock vs spinal shock	3×	157
Le Fort fractures	3×	171
Pelvic fractures: classification, emergency management, binders and embolisation	3×	192
Pathophysiology of TBI; primary and secondary injury; reducing secondary injury	3×	204
NEXUS and Canadian C-spine rules	3×	218
Hard/soft signs of neck injury and penetrating neck trauma protocol	3×	250
End-tidal CO ₂ and capnography	3×	265
Difficult airway assessment and LEMON	3×	274
Physiologically difficult airway	3×	279

Answer	Times asked	Page
Epistaxis: arterial supply of nasal septum (diagram), aetiology, management	3×	340
Avulsed tooth: scene care, prehospital care and replantation	3×	345

HOW TO USE THESE NOTES

- **One answer, many questions.** Repeats and near-repeats are merged; every merged question is printed verbatim at the head of its answer with an **Asked in** line and a frequency badge (1×, 2×, 3×, 4×+).
- **Management basis strip** (amber) names the guideline or book that drives management; every guideline-derived line carries a tag such as [AHA 2025] or [SSC 2026 R16] with the guideline's own grade.
- **Red box — NOT COVERED IN SOURCE:** the point is absent from the source books and saved guidelines after a full search; the box records the search, and the closest sourced material follows, labelled as such.
- **Figures** are real figures cropped from the source books with their own numbers and pages. Drawings labelled **Schematic (exam diagram)** are simplified diagrams built from a labelled spec, printed beside the original; flowcharts are our own drawings from the guideline or book text.
- **Green box** = pearls for last-minute revision; **blue box** = worked example (every number recomputed).

Part 18 · Endocrine & Metabolic Emergencies

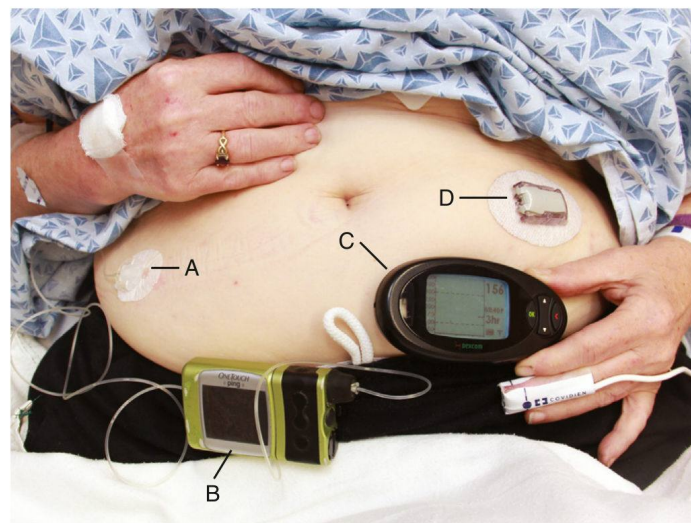
9 answers

Insulin pumps. [5] — Oct 2024 · P-III · Q4(c)

Asked in: Oct 2024 P-III Q4(c) · 4–5-mark length

The device

- A pager-sized pump delivering **rapid-acting insulin** through tubing and a **subcutaneous catheter** as a **basal rate** (generally 0.5–1.5 U/h, variable through the day) plus **boluses** for meals and correction.
- It replaces long-acting injections; the patient refills the reservoir and changes the catheter every **2–3 days**. Some pumps are tubeless and stick to the skin.
- Usual sites: abdominal SC tissue in adults, buttocks in young children; also thighs, hips, upper arms (RH).
- Suits motivated, mechanically adept patients who count carbohydrates and test glucose 4–6 times a day. Benefits over injections: HbA1c $\approx$ 0.5% lower and fewer hypoglycaemic episodes.
- May pair with a **continuous glucose monitor**; the hybrid closed-loop system adjusts dosing from CGM data.



RH 8e Fig. 71.1, p. 1522 — External insulin infusion pump: SC catheter (A), programmable pump (B), CGM receiver (C) and sensor/transmitter (D).

Problems that bring pump users to the ED

- **Delivery failure** — disconnection, empty reservoir, kinked catheter, priming error. Because only rapid-acting insulin is used, **ketoacidosis can follow within an hour or less**.
- Reported adverse events: blood glucose >400 mg/dL (47.3%), <54 mg/dL (23.9%), hospital/ICU admission (15.2%), DKA (8.8%) (RH Box 71.2).
- Infusion-site **cellulitis** and **lipodystrophy**.
- CGM reads interstitial glucose about 10 min behind serum and can be wrong (e.g. acetaminophen): **use capillary or serum glucose for ED decisions**.

Management basis: TIN 9e ch. 223 §Insulin Pumps (no widely accepted ED guideline exists, per TIN); device removal from RH 8e ch. 71.

Table 1. ED management of the patient on an insulin pump *Source: TIN 9e ch. 223, pp. 1422–1423; RH 8e ch. 71, pp. 1521–1522*

Situation	Action
Hyperglycaemia without DKA	Treat as for multiple-daily-injection patients; do not disable the pump . Ask when the reservoir was filled and the set changed, site rotation, water immersion, alarms; check pump on, reservoir, tubing, site. Call the manufacturer's 24-h line; consult endocrinology if malfunction suspected
DKA	Assume a pump problem: disconnect it and start the IV insulin DKA protocol; no IV insulin bolus . Consider SC long-acting insulin at the start. Give long-acting insulin ≥ 1 h before stopping the drip, or restart the pump (fresh insulin, new catheter, diagnostics run) about 1 h before stopping it; check glucose every 30–60 min
Hypoglycaemia	Treat as usual; do not stop the pump (DKA can develop rapidly). Recurrent episodes → suspect malfunction; basal rate may be reduced with endocrinology input
Pump off >1 h (defect, MRI)	Give a dose of rapid- or long-acting insulin
Nil by mouth	Leave the pump on; glucose every 30–60 min
Removal	Peel off the adhesive and embedded catheter together to stop insulin flow (RH)

PEARLS

- ★ Pump = rapid-acting insulin only, so failure → DKA within about an hour.
- ★ DKA on a pump: disconnect, IV insulin protocol, no bolus; overlap with long-acting insulin or restarted pump by 1 h.
- ★ Hyper- or hypoglycaemia without DKA: keep the pump running.
- ★ Never treat on CGM readings; check capillary or serum glucose.

Sources

- TIN 9e, ch. 223 "Type 1 Diabetes Mellitus", §Insulin Pumps, §Insulin Pump Complications, §Continuous Interstitial Glucose Monitoring, §Hyperglycemia/DKA/Hypoglycemia in Patients Using Insulin Pumps, pp. 1422–1423; ch. 226, §Patients With Insulin Pumps, p. 1441.
- RH 8e, ch. 71 "Noncardiac Implantable Devices", §Insulin Infusion Devices (Anatomy, Device Complications, Procedure), pp. 1521–1522; Fig. 71.1 and Boxes 71.1–71.2, p. 1522.

Hypoglycaemia in the ED: clinical features, evaluation (diagnosis) and treatment

2×

Evaluation and management of hypoglycemia in ED. [3+2] — Dec 2021 · P-III · Q5(a)

Describe clinical features, diagnosis and treatment of hypoglycemia in emergency department. [5] — Jun 2025* · P-III · Q7(a)

Asked in: Dec 2021 P-III Q5(a); Jun 2025* P-III Q7(a) · 3 + 2 and 5 (repeat) → 4–5-mark length

Definition and causes

- **Non-diabetic:** clinical definition — symptoms consistent with hypoglycaemia, associated with a low glucose, resolving with glucose. **Diabetic:** plasma glucose ≤ 70 mg/dL (3.9 mmol/L) is the alert threshold.
- Commonest with **insulin and sulphonylureas**; rare with metformin alone, glitazones, glinides or α -glucosidase inhibitors. Risk rises with age, renal failure, poor intake, alcohol, drug interactions.
- Non-diabetics: drugs and alcohol, factitious use, insulinoma or non-islet tumour, sepsis, liver failure, adrenal insufficiency, hypopituitarism.

Clinical features

- **Autonomic** — adrenergic (anxiety, irritability, tremor, palpitations, nausea, vomiting) and cholinergic (sweating, hunger, paraesthesia).
- **Neuroglycopenic** — confusion, lethargy, combativeness, agitation, seizures, coma, **focal deficits that mimic stroke**. Typically pale and diaphoretic.
- **Hypoglycaemia unawareness** (deficient counter-regulation) and β -blockers mask adrenergic warning symptoms.
- Mimics (TIN Table 224-14): stroke, TIA, seizure, head injury, psychosis, sympathomimetic ingestion.

Evaluation and diagnosis

- Check a **bedside glucose in every patient with altered mental status**, prehospital and in the ED.
- Bedside strips are least reliable at extremes; whole-blood values are $\approx 15\%$ below plasma — confirm with a laboratory sample.

- Falsely high POC readings: acetaminophen, ascorbic acid, icodextrin dialysate — use lab glucose if the reading does not fit.
- **Non-diabetic, no hypoglycaemic agent:** draw serum **before dextrose** for insulin, proinsulin and C-peptide.
- **Diabetic on usual sulphonylurea dose:** look for the trigger — interaction, reduced metabolism or excretion. Identify insulin type, timing and meals.

Management basis: TIN 9e ch. 224 §Hypoglycemia (treatment, disposition); ch. 223 §Hypoglycemia in Insulin-Dependent Patients; ch. 146 Table 146-1 for children.

Treatment

Table 2. ED treatment of hypoglycaemia *Source: TIN 9e pp. 966–967, 1422, 1432–1433, 1443*

Situation	Treatment
Alert, able to swallow	15–20 g glucose PO; repeat after 15 min if still low. Sublingual 40% dextrose gel where resources are limited. Not fructose or protein (peanut butter, cheese); then a meal
Altered mental status (adult)	50% dextrose 50 mL IV (25 g) ; repeat after 15 min if needed
No IV access	Glucagon 1 mg IM or SC (acts in 7–15 min; short-lived). Not in glycogen depletion (heavy alcohol use, post-marathon); not for sulphonylurea hypoglycaemia; may cause vomiting
Still unconscious or nil by mouth after correction	Infuse 5% dextrose to keep glucose >100 mg/dL; glucose every 30 min for 2 h for rebound
Child	Dextrose 0.5–1.0 g/kg ; neonate D10 5 mL/kg; infant/child D25 1–2 mL/kg; older child D50 1 mL/kg; maintenance D10 at 6–8 mg/kg/min. Glucagon 0.03 mg/kg IM
Alcoholic or malnourished	Some give thiamine before glucose (Wernicke's prevention)
No response to glucose	Think sepsis, toxin, insulinoma, hepatic failure, adrenal insufficiency

WORKED EXAMPLE — Dextrose doses

Adult: 50 mL of 50% dextrose = $50 \times 0.5 = 25$ g glucose.

20-kg child: $0.5\text{--}1.0$ g/kg = **10–20 g** = 100–200 mL of D10 (0.1 g/mL).

Maintenance for the same child: $6\text{--}8$ mg/kg/min $\times 20 = 120\text{--}160$ mg/min = $7.2\text{--}9.6$ g/h = **72–96 mL/h of D10**.

Disposition

- **Admit:** sulphonylurea, non–short-acting insulin or meglitinide hypoglycaemia; significant long-acting insulin overdose.
- **Discharge:** an isolated accidental episode not caused by oral agents or long-acting insulin, with reliable follow-up, after an **uneventful 4-h observation**.

PEARLS

- ★ Glucose check is part of the primary survey for every altered patient.
- ★ D50 50 mL = 25 g; repeat at 15 min; then feed or run D5W.
- ★ Draw insulin/C-peptide before dextrose in the non-diabetic.
- ★ Glucagon fails in glycogen depletion and is not for sulphonylureas.

Sources

- TIN 9e, ch. 224 "Type 2 Diabetes Mellitus", §Hypoglycemia (Pathophysiology, Clinical Features, Diagnosis, Treatment), pp. 1431–1432; Table 224-14 and §Disposition, p. 1433.
- TIN 9e, ch. 223 "Type 1 Diabetes Mellitus", §Falsely Elevated Capillary Glucose, §Hypoglycemia in Insulin-Dependent Patients, pp. 1420–1422; ch. 146, §Hypoglycemia Treatment, Table 146-1, pp. 966–967; ch. 226, §Treatment, p. 1443.

Management of a patient with sulphonylurea-induced hypoglycemia. [4] — May 2024 · P-III · Q1(b)

Asked in: May 2024 P-III Q1(b) · 4–5-mark length

Why it is different

- Sulphonylureas bind the **sulphonylurea receptor** subunit of the ATP-sensitive K^+ channel on β cells → insulin exocytosis.
- **Long-acting agents** — chlorpropamide, glyburide (glibenclamide), long-acting glipizide — cause most episodes; glyburide carries the highest risk.
- Glucose itself stimulates further insulin release from the drug-primed β cell, so **dextrose alone can aggravate and prolong hypoglycaemia**; hypoglycaemia is more challenging than insulin-induced.
- Precipitants on a usual dose: drug interactions, reduced metabolism or excretion (renal failure), age, poor intake, alcohol.

Management basis: TIN 9e ch. 224 §Hypoglycemia — Treatment and Disposition (sulphonylurea section), pp. 1432–1433.

Management

1. **Confirm** with bedside and laboratory glucose; ABC; look for the precipitant.
2. **Dextrose first:** D50 50 mL (25 g) IV, repeated after 15 min if needed; then oral carbohydrate if awake, or **5% dextrose infusion** to keep glucose >100 mg/dL.
3. **Glucose every 30 min for the first 2 h**, looking for rebound hypoglycaemia; reduce the infusion slowly as glucose holds.
4. **Octreotide** (somatostatin analogue) suppresses insulin secretion immediately and negates the sulphonylurea; believed superior to glucose and diazoxide in preventing recurrence. Give **only after glucose has been started**; consider when the response to dextrose is inadequate.
5. **Glucagon is not recommended** for sulphonylurea hypoglycaemia.
6. **Diazoxide** (inhibits β -cell insulin secretion) for refractory cases: 300 mg as a slow IV infusion over 30 min every 4 h (causes hypotension).
7. Haemodialysis and charcoal haemoperfusion are **not** routinely recommended.
8. **Admit** every sulphonylurea hypoglycaemia for serial glucose monitoring.

Table 3. Octreotide regimens described in TIN 9e Source: TIN 9e pp. 1432–1433 (ideal dose and interval not well defined)

Setting	Dose
After a single hypoglycaemic episode	50–100 μ g SC once
After a second episode	50–100 μ g SC every 6–8 h, or 125 μ g/h IV infusion
Added to standard therapy	50 μ g SC — fewer episodes, higher mean glucose

WORKED EXAMPLE — Octreotide daily totals

50 μ g every 8 h = $50 \times 24/8 = 150$ μ g/day; 100 μ g every 6 h = $100 \times 24/6 = 400$ μ g/day.

IV infusion 125 μ g/h $\times$ 24 h = **3000 μ g (3 mg)/day**.

PEARLS

- ★ Sulphonylurea hypoglycaemia recurs: dextrose drives more insulin release.
- ★ Octreotide 50–100 μ g SC after glucose has started; glucagon is not used.
- ★ Glucose every 30 min for 2 h and admit every case.

Sources

• TIN 9e, ch. 224 "Type 2 Diabetes Mellitus", §Sulphonylureas, p. 1429; §Hypoglycemia (Pathophysiology, Diagnosis, Treatment), pp. 1431–1432; octreotide, glucagon, diazoxide and §Disposition and Follow-Up, p. 1433.

Patho-physiology of diabetic ketoacidosis. [5] — Jun 2020 · P-I · Q2(a)

Describe the pathophysiology of diabetic ketoacidosis Versus hyperglycemic hyperosmolar syndrome. [5] — Jun 2022 · P-I · Q1(a)

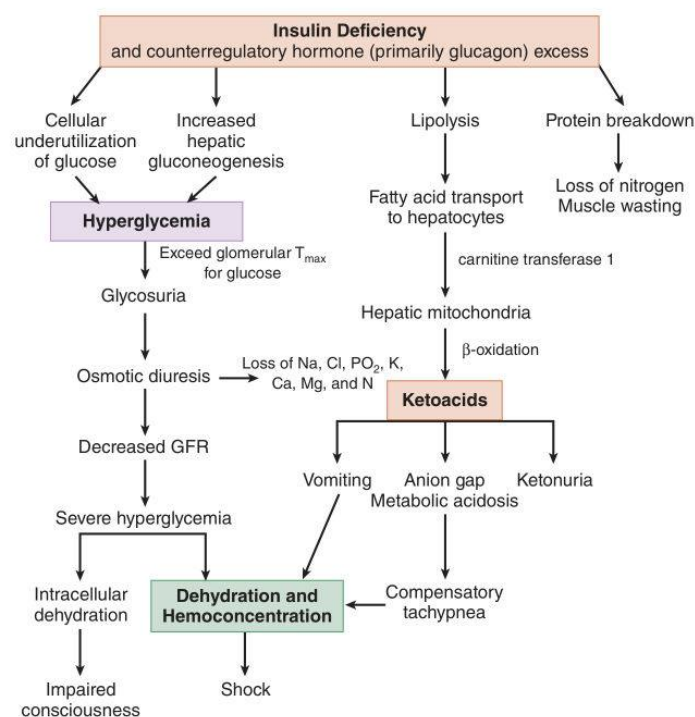
Asked in: Jun 2020 P-I Q2(a); Jun 2022 P-I Q1(a) · 5 + 5 (overlapping) → 6–7-mark length

Normal role of insulin

- Insulin is the only anabolic hormone of the endocrine pancreas. Liver: glucose uptake and glycogen storage, inhibits glycogenolysis and gluconeogenesis. Adipose: lipogenesis, blocks triglyceride breakdown. Muscle: amino-acid uptake and protein synthesis.

DKA — step by step

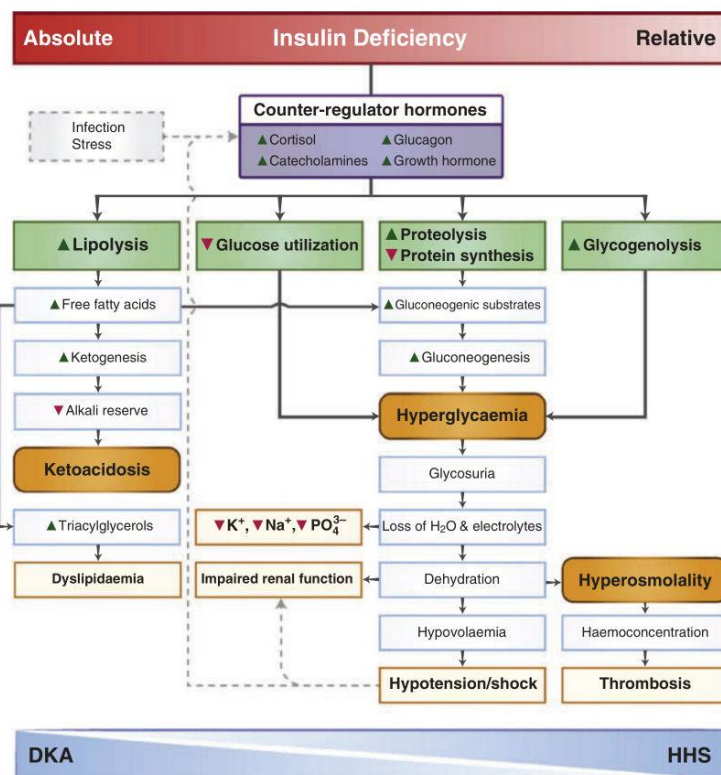
1. **Relative or absolute insulin deficiency + counter-regulatory excess** (glucagon chiefly; catecholamines, cortisol, growth hormone) = cellular starvation despite hyperglycaemia.
2. **Hyperglycaemia:** increased gluconeogenesis and glycogenolysis plus under-use of glucose; glycerol and amino acids from lipolysis and proteolysis feed gluconeogenesis.
3. **Ketogenesis:** free fatty acids travel bound to albumin to the liver, enter mitochondria (carnitine transferase 1) and undergo β -oxidation to β -hydroxybutyrate and acetoacetate ($\text{AcAc} + \text{NADH} \rightarrow \beta\text{HB} + \text{NAD}$); $\text{AcAc} \rightarrow$ acetone. Glycogen depletion favours ketogenesis; low insulin reduces ketone use by brain and muscle.
4. **Anion-gap metabolic acidosis** from ketoacids → Kussmaul breathing; vomiting adds losses.
5. **Osmotic diuresis** once the renal glucose threshold is exceeded → loss of water, Na^+ , Cl^- , K^+ , phosphate, Ca^{2+} , Mg^{2+} → volume depletion and falling GFR, which worsen hyperglycaemia and ketonaemia.
6. **RAAS activation** increases renal K^+ loss; Cl^- retained in exchange for excreted ketoanions → superimposed hyperchloraemic acidosis.
7. **Prostaglandins I_2 and E_2** from adipose breakdown → paradoxical vasodilatation, nausea, vomiting, abdominal pain.
8. **Impaired consciousness** tracks serum osmolality ($>320 \text{ mOsm/kg}$) better than acidosis.



TIN 9e Figure 225-1, p. 1434 — Pathogenesis of DKA from insulin deficiency and counter-regulatory (chiefly glucagon) excess.

HHS — the differences

- Three factors: **insulin resistance and/or deficiency**; an **inflammatory state** (CRP, interleukins, TNF) with counter-regulatory hormones; **osmotic diuresis followed by impaired renal glucose excretion**.
- Typical setting: debilitated, often elderly type 2 diabetic with limited access to water and a precipitating illness; evolves over days to weeks.
- Rising glucose shifts water out of cells (cellular dehydration); a transient GFR rise is followed by diuresis with water loss of **20–25% of body weight (8–12 L in 70 kg)**, then falling GFR that further traps glucose.
- **Little ketosis** because of (1) more endogenous insulin than in DKA, (2) lower counter-regulatory hormones, (3) inhibition of lipolysis by hyperosmolality.
- Lethargy and coma rise linearly with osmolality; focal seizures in up to 15%; venous and arterial thrombosis are common.



TIN 9e Figure 227-1, p. 1444 — Pathophysiology of hyperglycaemic emergencies: the absolute (DKA) to relative (HHS) insulin-deficiency spectrum.

Table 4. DKA vs HHS — pathophysiological contrasts Source: TIN 9e Table 227-3, p. 1445; text pp. 1434–1437, 1443–1446

Feature	DKA	HHS
Insulin deficit	Absolute or severe relative	Relative; resistance; enough insulin to curb lipolysis
Typical patient / tempo	Type 1, young; evolves within 24 h	Elderly type 2, comorbid; days to weeks
Glucose	>250 mg/dL	Usually >600 mg/dL
pH / HCO ₃ ⁻	<7.30 / ≤18 mEq/L	>7.30 / >15 mEq/L
Ketones (βHB, AcAc)	Positive	Negative or small
Anion gap	>12 mEq/L	<12 mEq/L
Serum osmolality	Variable	>320 mOsm/kg
Water deficit	≈100 mL/kg (5–10 L)	20–25% of body weight (8–12 L)
K ⁺ deficit	3–5 mEq/kg	4–6 mEq/kg

PEARLS

- ★ DKA = insulin lack + glucagon excess → ketoacidosis + osmotic diuresis.
- ★ HHS = enough insulin to stop ketosis, not enough to stop hyperglycaemia; dehydration is larger.
- ★ Mental state follows osmolality, not pH.
- ★ Saline-driven ketoanion loss explains the hyperchloraemic phase of DKA.

Sources

- TIN 9e, ch. 225 "Diabetic Ketoacidosis", §Pathophysiology (Insulin, Ketoacidosis), §Clinical Features, pp. 1434–1435; Fig. 225-1, p. 1434; §Volume Repletion, §Potassium Replacement, pp. 1437–1438.
- TIN 9e, ch. 227 "Hyperosmolar Hyperglycemic State", §Pathophysiology, §Clinical Features, §Diagnosis, pp. 1443–1445; Fig. 227-1, p. 1444; Table 227-3, p. 1445; §Potassium, p. 1446; §Disease Complications, p. 1447.

Diabetic ketoacidosis: diagnostic criteria, how it is diagnosed, and complications

2×

Diagnostic criteria for diabetic ketoacidosis. [3] — Jun 2020 · P-I · Q2(b)

Complications of diabetic ketoacidosis. [2] — Jun 2020 · P-I · Q2(c)

How diabetic ketoacidosis is diagnosed? [5] — Oct 2023 · P-III · Q3(a)

Asked in: Jun 2020 P-I Q2(b, c); Oct 2023 P-III Q3(a) · 3 + 2 + 5 (criteria repeat) → 6–7-mark length

Diagnostic criteria

- **Glucose >250 mg/dL (13.9 mmol/L), anion gap >10–12 mEq/L, HCO_3^- <15 mEq/L and pH <7.3** with moderate ketonuria or ketonaemia (TIN ch. 225 text). Table 227-3 lists $\text{HCO}_3^- \leq 18$ mEq/L.
- **Euglycaemic DKA** (glucose <250 mg/dL): pregnancy, young vomiting type 1 diabetics, insulin just taken, alcohol abuse or liver failure, starvation, **SGLT-2 inhibitors**. Diagnose with serum βHB (generally >3 mmol/L).

Table 5. Severity of DKA Source: TIN 9e p. 1435

Severity	Arterial pH	HCO_3^- (mEq/L)	Anion gap	Mental state
Mild	7.25–7.30	15–18	>10	Alert
Moderate	7.00–7.24	10–15	>12	Alert to drowsy
Severe	<7.00	<10	>12	Stuporous to comatose

How DKA is diagnosed in the ED

1. **Suspect at triage:** polyuria, polydipsia, vomiting, abdominal pain, Kussmaul breathing, acetone breath, dehydration, altered sensorium. Fingerstick >250 mg/dL with urine ketones → assume DKA and start fluids.
2. **Bedside:** glucose, urinalysis, **ECG** (hyper-/hypokalaemia; infarction as trigger).
3. **Laboratory:** CBC, electrolytes, urea, creatinine, venous blood gas, phosphate, magnesium, calcium, serum osmolality; cultures as indicated.
4. **Ketones:** quantitative **serum βHB** is preferred. Nitroprusside (Acetest) detects only acetoacetate — it misses βHB , rises paradoxically with treatment, and captopril gives false positives.
5. **Venous pH** replaces arterial (≈ 0.03 lower). PCO_2 lower than expected → primary respiratory alkalosis (pneumonia, PE, sepsis).
6. **Calculate** anion gap, corrected Na^+ and osmolality (worked example). Mixed patterns: hyperchloraemic acidosis in well-hydrated patients; vomiting alkalosis may mask acidosis — then a raised gap is the only clue.
7. **Interpret with care:** K^+ usually normal or high despite deficit; creatinine factitiously raised; WBC >25,000 or bands $\geq 10,000$ suggest infection; lipase can rise without pancreatitis.
8. **Exclude mimics** (TIN Table 225-3): alcoholic and starvation ketoacidosis ($\beta\text{HB} < 3$, $\text{HCO}_3^- > 18$, glucose low/normal), renal failure, lactic acidosis, salicylate, ethylene glycol, methanol; HHS.
9. **Find the precipitant** (Table 225-1): missed insulin, pump failure, infection, MI, stroke, pancreatitis, pregnancy, drugs (steroids, thiazides, antipsychotics), trauma, surgery.

WORKED EXAMPLE — Anion gap, corrected sodium and osmolality (TIN formulas)

Data: Na^+ 128, Cl^- 92, HCO_3^- 8 mEq/L; glucose 540 mg/dL; BUN 28 mg/dL.

Anion gap = $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) = 128 - (92 + 8) = 28 \text{ mEq/L}$ (raised).

Corrected Na^+ = $128 + 1.6 \times (540 - 100)/100 = 128 + 7.0 = 135 \text{ mEq/L}$; with the 2.4 factor (glucose >400) = $128 + 2.4 \times 4.4 = 138.6 \text{ mEq/L}$ → eunatraemic: switch to 0.45% saline after the first bolus.

Osmolality = $2 \times 128 + 540/18 + 28/2.8 = 256 + 30 + 10 = 296 \text{ mOsm/kg}$; effective osmolality (no urea) = $256 + 30 = 286$.

Complications

Table 6. Complications of DKA Source: TIN 9e Table 225-6, p. 1440; text pp. 1438–1440

Related to the acute disease	Related to therapy	Later
Loss of airway; hypovolaemic shock; sepsis; myocardial infarction; DVT in the volume-depleted elderly	Hypokalaemia (most dangerous); hypoglycaemia; hypophosphataemia; ARDS (over-hydration); cerebral oedema	Recurrent anion-gap acidosis; non-anion-gap (hyperchloraemic) acidosis; vascular thrombosis; mucormycosis

- **Cerebral oedema:** mainly young children, new-onset diabetics and adolescents; 4–12 h after therapy (up to 24–48 h). Premonitory: severe headache, incontinence, behaviour change, pupillary or BP change, seizures, bradycardia. Give **mannitol 1–2 g/kg IV** (or 3% saline 5–10 mL/kg over 30 min) before CT.
- **Hypokalaemia:** K^+ falls $\approx 1.5 \text{ mEq/L}$ with therapy → arrhythmia, respiratory paralysis, ileus, rhabdomyolysis.
- **Refractory acidosis** → unrecognised infection or a faulty insulin drip; fluid-unresponsive shock → gram-negative sepsis or silent MI.
- Mortality: sepsis, pulmonary and cardiovascular complications in the elderly; cerebral oedema in children and young adults.

PEARLS

- ★ Criteria: glucose >250, pH <7.3, HCO_3^- <15, AG >10–12, ketones present.
- ★ Order βHB , not nitroprusside ketones; venous pH is enough.
- ★ Euglycaemic DKA: pregnancy, SGLT-2 inhibitors, starvation, alcohol.
- ★ Corrected Na^+ adds 1.6 (2.4 if glucose >400) per 100 mg/dL above 100.

Sources

- TIN 9e, ch. 225 "Diabetic Ketoacidosis", §Clinical Features, §Diagnosis, §Euglycemic DKA, Tables 225-1, 225-2, p. 1435; §Differential Diagnosis, §Laboratory Testing, Table 225-3, p. 1436; §Hypokalemia, p. 1438; §Disease Complications, pp. 1439–1440; Tables 225-4 to 225-6, p. 1440.
- TIN 9e, ch. 227 "Hyperosmolar Hyperglycemic State", §Sodium and §Osmolality formulas, Table 227-3, p. 1445.

Treatment of diabetic ketoacidosis (adult)

1×

Describe the treatment of diabetic ketoacidosis. [5] — Oct 2023 · P-III · Q3(b)

Asked in: Oct 2023 P-III Q3(b) · 4–5-mark length

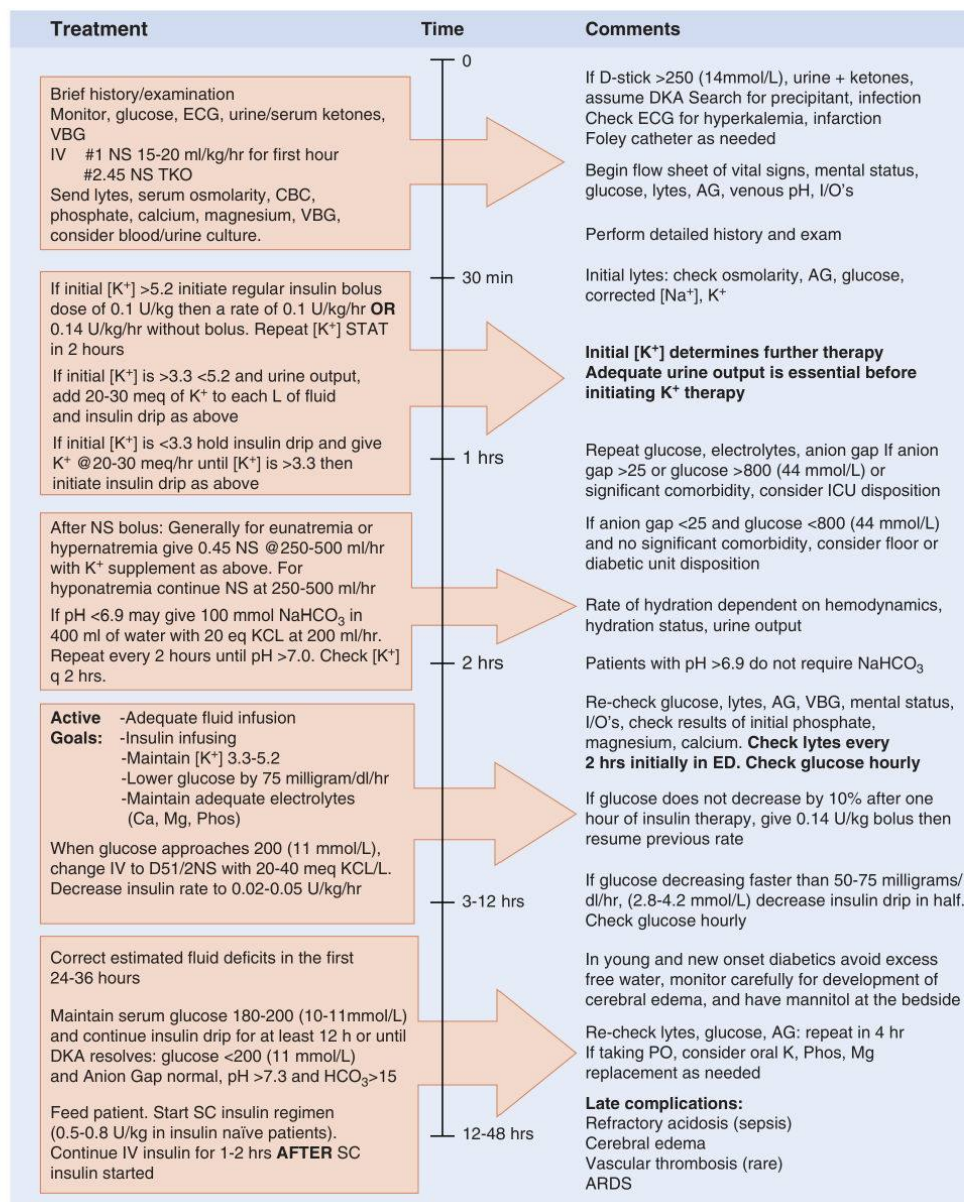
Management basis: TIN 9e ch. 225 "Diabetic Ketoacidosis", §Treatment and Figure 225-2 (timeline for the adult with suspected DKA).

Goals and order of priorities

- Goals: (1) volume repletion, (2) reverse the insulin deficit, (3) correct electrolytes and acid–base, (4) treat the precipitant, (5) avoid complications.
- **Order: volume first, then potassium, then insulin.** Cardiac monitor; one or two large-bore (16–18 G) IV lines. Correct over 24–36 h.
- **Monitor every 2 h:** glucose, K^+ , anion gap, vital signs, consciousness, intake–output (glucose hourly for the first 6 h). Target glucose <200 mg/dL, $\text{HCO}_3^- \geq 18$, venous pH >7.3.

Table 7. Core DKA therapy Source: *TIN 9e pp. 1436–1440; Fig. 225-2, p. 1437*

Component	What to give
Fluids	Isotonic crystalloid 15–20 mL/kg/h in the first hour (NS, Ringer's lactate or a balanced solution). Then NS 250–500 mL/h if corrected Na ⁺ is low, or 0.45% saline 250–500 mL/h if normal or high. Roughly 2 L over 0–2 h, 2 L over 2–6 h, 2 L over 6–12 h (≈50% of the ~100 mL/kg deficit in 12 h). Add dextrose (D5 in 0.45% saline) at glucose ≈250 mg/dL
Potassium	K ⁺ >5.2: none, recheck in 2 h. K ⁺ 3.3–5.2 with urine output: 20–30 mEq per litre , aiming for 4–5 mEq/L. K ⁺ <3.3: hold insulin , give 20–30 mEq/h until ≥3.5. Usual limits 10 mEq/h peripheral, 20 mEq/h central; 100–200 mEq KCl in 24 h
Insulin	Regular insulin 0.1–0.14 U/kg/h by infusion once K ⁺ ≥3.3; bolus optional in adults (0.1 U/kg). Expect glucose to fall 50–75 mg/dL/h; if <10% fall in 1 h, give 0.14 U/kg bolus. At glucose 250 mg/dL reduce to 0.02–0.05 U/kg/h and keep glucose 150–200 mg/dL
Resolution and transition	Glucose <200 mg/dL plus two of: HCO ₃ ⁻ >15, venous pH >7.3, normal anion gap. Feed, start SC insulin (new diabetics 0.5–0.8 U/kg/day), overlap IV and SC by 2–4 h
Bicarbonate	Only if pH <6.9: 100 mEq NaHCO ₃ in 400 mL water + 20 mEq KCl at 200 mL/h for 2 h, repeat until pH >7.0; K ⁺ every 2 h
Phosphate / magnesium	IV phosphate only if <1.0 mg/dL (K ₂ PO ₄ 2.5–5 mg/kg). MgSO ₄ 2 g IV over 1 h if Mg ²⁺ <2.0 mEq/L
Precipitant and disposition	Search for infection, MI. ICU if AG >25, glucose >800 or comorbidity; selected patients (AG <25, glucose <600) to a monitored ward



TIN 9e Figure 225-2, p. 1437 — Timeline for the typical adult patient with suspected DKA.

WORKED EXAMPLE — 70-kg adult, pH 7.0, K⁺ 5.5 mEq/L

First-hour fluid: 15–20 mL/kg × 70 = **1050–1400 mL** isotonic crystalloid.

Insulin: 0.1–0.14 U/kg/h × 70 = **7–9.8 U/h**; after glucose reaches 250, 0.02–0.05 × 70 = **1.4–3.5 U/h**.

Potassium forecast (K⁺ changes ≈0.5 mEq/L per 0.1 pH): correcting pH 7.0 → 7.4 = 4 steps × 0.5 = 2.0 → expected K⁺ 5.5 – 2.0 = **3.5 mEq/L**; start K⁺ once ≤5.2.

Deficits: water 100 mL/kg = **7 L**; potassium 3–5 mEq/kg = **210–350 mEq**.

PEARLS

- ★ Volume → potassium → insulin, in that order.
- ★ Never start insulin with K⁺ <3.3.
- ★ Add dextrose at 250 mg/dL and keep the insulin running until the gap closes.
- ★ Overlap SC and IV insulin by 2–4 h; stopping the drip early causes relapse.

Sources

• TIN 9e, ch. 225 "Diabetic Ketoacidosis", §Treatment, p. 1436; §Volume Repletion, Fig. 225-2, p. 1437; §Potassium Replacement, §Insulin, p. 1438; §Transition, §Hypophosphatemia, §Hypomagnesemia, §Bicarbonate, p. 1439; §Disposition, Table 225-4, p. 1440.

Myxoedema coma (crisis): precipitants, recognition (clinical features) and management

2×

Discuss the precipitating causes, clinical features and management of myxoedema coma. [2+2+2] — Dec 2022 · P-III · Q4(a)

[Discuss the recognition and steps of management of:] Myxoedema coma. [5] — Apr 2023 · P-III · Q9(b)

Asked in: Dec 2022 P-III Q4(a); Apr 2023 P-III Q9(b) · 2 + 2 + 2 and 5 → 6–7-mark length

Definition

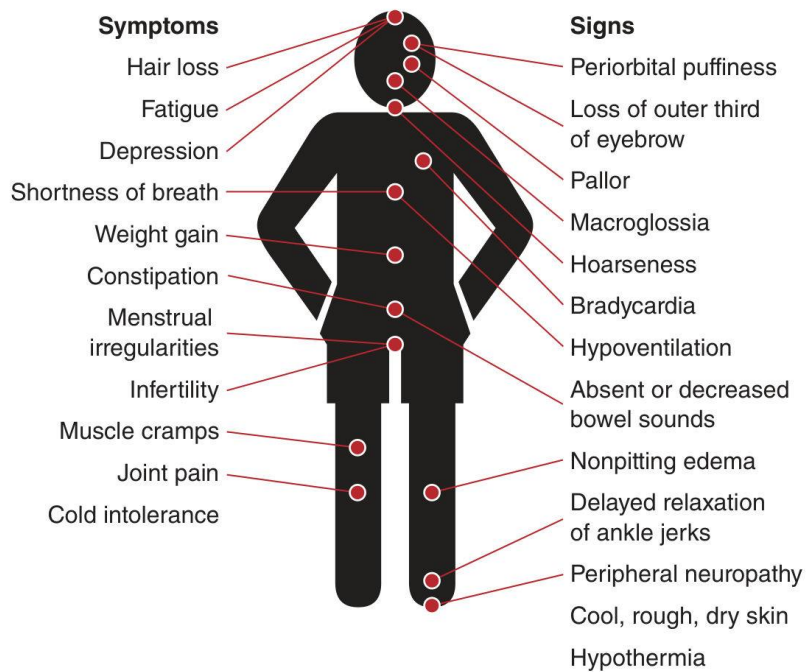
- Metabolic and multi-organ decompensation of uncorrected hypothyroidism with **altered mental status or coma** and **hypothermia (usually <35.5 °C)**. Mortality **30–60%**.
- T₄:T₃ released ≈10:1; T₄ is converted peripherally to T₃, which is 3–4 times more potent (half-lives 7 days and 1 day).

Precipitating causes

- **Infection**, cold exposure, anaesthetic agents, surgery, trauma, burns.
- Myocardial infarction or heart failure, stroke, GI haemorrhage.
- **Drugs:** β-blockers, sedatives, narcotics, phenothiazines, amiodarone; **non-compliance with thyroxine**.
- Also hypoxia, hypercapnia, hyponatraemia, hypoglycaemia (TIN Table 228-3).

Recognition — clinical features

- **Hypothyroid habitus** (Fig. 228-1): periorbital puffiness, loss of outer third of eyebrows, macroglossia, hoarseness, non-pitting oedema, cool rough dry skin, delayed relaxation of ankle jerks.
- **Vital signs:** bradycardia, hypotension (half have SBP <100 mm Hg — leaky capillaries), hypothermia, hypoventilation.
- **CNS:** altered mental status or coma from CO₂ narcosis, hypoglycaemia and reduced clearance of sedatives.
- **Airway/chest:** glottic or vocal-cord oedema, macroglossia; pleural and pericardial effusions.
- **Infection is masked** — fever, tachycardia and sweating may be absent; a **normal temperature suggests infection**.
- From accidental hypothermia: hypothyroid habitus, **no shivering**, pseudomyotonic reflexes (relaxation ≥2× contraction).
- **Diagnosis is clinical**; DDx: sepsis, adrenal crisis, heart failure, hypoglycaemia, stroke, hypothermia, overdose, meningitis.



TIN 9e Figure 228-1, p. 1448 — Signs and symptoms of hypothyroidism.

Table 8. Investigations (do not wait for them to treat) *Source: TIN 9e p. 1449*

Test	Finding
TSH, free T ₄ /T ₃ , cortisol — before treatment	High TSH + low T ₄ = primary; low TSH + low T ₄ = secondary
Electrolytes, glucose	Hyponatraemia (ADH, impaired free-water clearance); hypoglycaemia
ABG	Hypoxaemia, hypercapnia, mixed metabolic and respiratory acidosis
ECG, chest radiograph, bedside US	MI or heart block; pneumonia, effusions, cardiomegaly; pericardial effusion

Management basis: TIN 9e ch. 228 “Hypothyroidism and Myxedema Crisis”, Table 228-3 (ED Treatment for Myxedema Crisis).

Management

1. **ABC:** airway control, oxygen, ventilation for hypercapnia, IV access, cardiac monitoring. Admit to **ICU**.
2. **Hydrocortisone 100–200 mg IV** at the start (stress dose; cortisol drawn first, result not awaited).
3. **IV levothyroxine** on clinical suspicion: **4 µg/kg** (usually 200–400 µg), then **100 µg IV at 24 h**, then **50 µg IV** daily until oral intake; elderly start **100 µg** IV because gut absorption is poor.
4. **IV liothyronine (T₃)** if available — 20 µg then 10 µg every 8 h; ≤10 µg in elderly or coronary disease (arrhythmia, ischaemia). Add to T₄ if haemodynamics or respiratory effort stay poor; monitor ECG.
5. **Supportive:** dextrose for hypoglycaemia; water restriction for hyponatraemia; **passive rewarming**; vasopressors if needed (ineffective without thyroid hormone).
6. **Treat the precipitant:** empiric broad-spectrum antibiotics if sepsis is possible; stop sedatives.
7. Elderly or cardiac patients: start with no more than half the usual T₄ or T₃ dose. Pregnancy: IV levothyroxine is safe.

WORKED EXAMPLE — Levothyroxine loading

70-kg adult: 4 µg/kg × 70 = **280 µg IV** (within the 200–400 µg range); day 2 100 µg IV; then 50 µg IV daily.
Elderly: start at **100 µg IV**.

PEARLS

- ★ Coma + hypothermia + hypothyroid habitus = myxoedema crisis; treat on suspicion.
- ★ Steroid first (hydrocortisone 100–200 mg), then levothyroxine 4 µg/kg IV.
- ★ No shivering and slow-relaxing reflexes separate it from accidental hypothermia.
- ★ A normal temperature here means infection until proven otherwise.

Sources

• TIN 9e, ch. 228 "Hypothyroidism and Myxedema Crisis", §Pathophysiology, §Clinical Features of Myxedema Crisis, p. 1447; Fig. 228-1, Table 228-2, p. 1448; §Diagnosis, §Laboratory Evaluation, §Treatment of Myxedema Crisis, Table 228-3, p. 1449; §Supportive Management, §Disposition, §Special Situations, p. 1450.

Thyroid storm: precipitants, clinical features, Burch–Wartofsky Point Scale and ED management

4 × +

Burch-Wartofsky Point Scale for thyroid storm. [3] — Jun 2021 · P-III · Q8(b)

Management of a patient with thyroid storm. [5] — Jun 2021 · P-III · Q8(c)

Briefly outline the steps in the management of thyroid storm. [5] — Jun 2022 · P-III · Q5(b)

Enumerate Burch and Wartofsky diagnostic parameters for thyroid storm and describe treatment of thyroid storm. [5] — Oct 2023 · P-IV · Q7(a)

Clinical features of thyroid storm. [3] — Oct 2024 · P-III · Q4(a)

Precipitants of thyroid storm. [2] — Oct 2024 · P-III · Q4(b)

Asked in: Jun 2021 P-III Q8(b, c); Jun 2022 P-III Q5(b); Oct 2023 P-IV Q7(a); Oct 2024 P-III Q4(a, b) · BWPS 3 + management 5 + features 3 + precipitants 2 (repeats once) → 10-mark length

Definition

- An acute, severe, life-threatening **hypermetabolic state** of pre-existing hyperthyroidism — adrenergic hyperactivity from excess hormone release or increased receptor sensitivity after a precipitant. Commonest cause Graves' disease. Mortality **8–25%**.
- A **clinical diagnosis**: start treatment on suspicion; hormone levels need not be higher than in uncomplicated thyrotoxicosis.

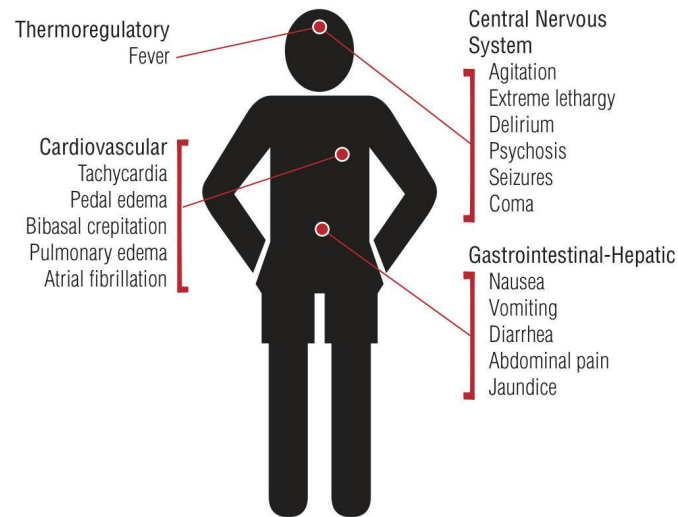
Precipitants

Table 9. Precipitants of thyroid storm *Source: TIN 9e Table 229-4, p. 1452*

Group	Examples
Systemic insult	Infection , trauma, general surgery
Endocrine	Diabetic ketoacidosis, hyperosmolar coma
Drug or hormone	Withdrawal of antithyroid drugs , iodine (e.g. contrast), thyroid palpation, thyroid hormone ingestion
Cardiovascular	Myocardial infarction, stroke, pulmonary embolism
Obstetric	Labour and delivery, eclampsia
Other	Radioactive iodine therapy; unknown in up to 25%

Clinical features

- **Thermoregulatory**: fever — may herald storm.
- **Cardiovascular**: tachycardia (sinus ≈40%), **atrial fibrillation 10–35%**, raised systolic BP with **widened pulse pressure**, high-output failure (pedal oedema, crackles, pulmonary oedema), pleuropericardial rub.
- **CNS**: agitation, extreme lethargy, delirium, psychosis, seizures, coma.
- **GI–hepatic**: nausea, vomiting, diarrhoea, abdominal pain, jaundice.
- Background thyrotoxicosis (Table 229-3): heat intolerance, sweating, weight loss, tremor, lid lag, goitre ± bruit, exophthalmos. Elderly may have **apathetic** thyrotoxicosis.
- **Labs**: leucocytosis, hyperglycaemia, mild hypercalcaemia, raised liver enzymes; a **low cortisol** suggests coexisting adrenal insufficiency.



TIN 9e Figure 229-1, p. 1452 — Clinical features of thyroid storm by system.

Burch-Wartofsky Point Scale (BWPS)

- Seven parameters: **temperature, CNS effects, GI-hepatic dysfunction, heart rate, heart failure, atrial fibrillation, precipitating event.**
- **≥45 highly suggestive** of storm; **25–44 impending**; **<25 unlikely**. Sensitive but not very specific; more sensitive than the Japanese Thyroid Association TS1/TS2 criteria.

TABLE 229-5 Burch and Wartofsky Diagnostic Parameters and Scoring Points for Thyroid Storm	
Diagnostic Parameters	Scoring Points
1. Thermoregulatory dysfunction	
Temperature °C (°F)	
37.2–37.7 (99–99.9)	5
37.7–38.3 (100–100.9)	10
38.3–38.8 (101–101.9)	15
38.9–39.4 (102–102.9)	20
39.4–39.9 (103–103.9)	25
≥40 (≥104.0)	30
2. CNS effects	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, extreme lethargy)	20
Severe (seizures, coma)	30
3. GI-hepatic dysfunction	
Absent	0
Moderate (diarrhea, nausea/vomiting, abdominal pain)	10
Severe (unexplained jaundice)	20
4. Cardiovascular dysfunction	
Tachycardia (beats/min)	
90–109	5
110–119	10
120–129	15
≥140	25
5. Congestive heart failure	
Absent	0
Mild (pedal edema)	5
Moderate (bibasilar rales)	10
Severe (pulmonary edema)	15
6. Atrial fibrillation	
Absent	0
Present	10
7. Precipitating event*	
Absent	0
Present	10

*See Table 229-3.

Scoring system: Score of ≥45: highly suggestive of thyroid storm. Score of 25–44: suggestive of impending storm. Score of <25: unlikely to represent thyroid storm.

Source: Reproduced with permission from Burch HB, Wartofsky L. Life-threatening thyrotoxicosis. Thyroid storm. *Endocrinol Metab Clin North Am* 22: 263, 1993.^{3,5}

TIN 9e Table 229-5, p. 1453 — Burch and Wartofsky diagnostic parameters and scoring points, as printed.

WORKED EXAMPLE — Scoring a febrile thyrotoxic patient

Temperature 39.0 °C (38.9–39.4) = **20**; agitation = **10**; diarrhoea = **10**; HR 125 (120–129) = **15**; pedal oedema = **5**; no AF = **0**; pneumonia (precipitant) = **10**.

Total = 20 + 10 + 10 + 15 + 5 + 0 + 10 = **70** → ≥45: **highly suggestive of thyroid storm**; treat now.

Management basis: TIN 9e ch. 229 “Hyperthyroidism and Thyroid Storm”, Table 229-7 (Treatment for Thyroid Storm).

Management — in order

- **Pregnancy test first**; then **thionamide before iodine** (iodine given ≥1 h later), or iodine fuels new hormone synthesis.
- Treat the precipitant in parallel (infection, MI, DKA); ICU admission.

Table 10. Thyroid storm treatment aims and doses *Source: TIN 9e Table 229-7, p. 1454; text pp. 1453–1456*

Aim	Drug and dose
1. Supportive	O ₂ , monitor; cooling + acetaminophen 325–650 mg every 4–6 h (aspirin contraindicated — frees hormone); IV fluids, saline with 5% dextrose if glucose low; glucose, thiamine, folate
2. Block adrenergic effects	Propranolol 0.5–1 mg IV over 10 min , then 1–2 mg every few hours by vital signs; PO 60–80 mg every 4 h. Or esmolol 250–500 µg/kg load, then 50–100 µg/kg/min (preferred in heart failure). Reactive airways: atenolol/metoprolol or calcium-channel blocker; reserpine or guanethidine only if β-blockade contraindicated
3. Stop new synthesis	Methimazole 20 mg PO every 6 h (60–80 mg/day; avoid in 1st trimester) or PTU 500–1000 mg PO load, then 250 mg every 4 h (1st trimester; hepatotoxic boxed warning). PR or NG if needed
4. Stop release (≥1 h after step 3)	Lugol's 8–10 drops PO every 6–8 h or SSKI 5 drops (250 mg) every 6 h; iopanoic acid 1 g IV every 8 h for 24 h then 500 mg twice daily; lithium 300 mg every 6–8 h if iodine-allergic or thionamide agranulocytosis (not in pregnancy)
5. Block T ₄ →T ₃	Hydrocortisone 300 mg IV, then 100 mg every 8 h , or dexamethasone 2 mg IV every 6 h — also covers relative adrenal insufficiency; improves survival
6. Stop reabsorption	Cholestyramine 4 g every 6 h
7–8. Precipitant; definitive	Treat trigger; radioiodine ablation or surgery later. Plasmapheresis/dialysis as last resort

- **Heart failure:** avoid nitrates; short-acting esmolol is safer. **AF:** rate control (β-blocker, digoxin), anticoagulate; amiodarone is 37% iodine.

PEARLS

- ★ BWPS ≥45 = storm; 25–44 = impending.
- ★ Order: β-blocker → thionamide → iodine 1 h later → steroid → cholestyramine.
- ★ PTU in the first trimester; methimazole otherwise.
- ★ Paracetamol, never aspirin, for fever.

Sources

• TIN 9e, ch. 229 "Hyperthyroidism and Thyroid Storm", §Introduction, p. 1450; §Laboratory Testing, Table 229-3, p. 1451; §Thyroid Storm, §Pathophysiology, §Precipitants, §Clinical Features, Table 229-4, Fig. 229-1, p. 1452; Table 229-5, §Laboratory Testing, §Treatment, p. 1453; Table 229-7, p. 1454; Treatment Aims 2–5, p. 1455; Aims 6–8, §Disposition, §Elderly, p. 1456; §Atrial Fibrillation, §CCF, p. 1457.

Adrenal insufficiency and acute adrenal crisis: recognition, ED approach, management and the role of hydrocortisone and fluids

4 × +

What is the management of adrenal crisis? [5] — Dec 2020 · P-I · Q7(b)

Discuss clinical features and management of a patient with adrenal crisis. [2+2] — Dec 2022 · P-III · Q4(b)

[Discuss the recognition and steps of management of:] Adrenal insufficiency. [5] — Apr 2023 · P-III · Q9(a)

Outline the ED approach to acute adrenal crisis. [5] — Dec 2025* · P-III · Q2(a)

Discuss the role of hydrocortisone and fluid therapy in acute adrenal crisis. [5] — Dec 2025* · P-III · Q2(b)

Asked in: Dec 2020 P-I Q7(b); Dec 2022 P-III Q4(b); Apr 2023 P-III Q9(a); Dec 2025* P-III Q2(a, b) · management 5 + features 2 + recognition 5 + approach 5 + hydrocortisone/fluids 5 → 10-mark length

Definitions and causes

- **Primary** (Addison's): intrinsic adrenal failure → ↓ cortisol, ↓ aldosterone, ↓ androgens; ≈90% of gland lost before symptoms.
- **Secondary:** hypothalamic–pituitary failure of CRH/ACTH → **cortisol deficiency only** (aldosterone preserved).
- **Adrenal crisis:** life-threatening exacerbation when demand rises and output cannot; higher death risk in the old, diabetic or obtunded.

Table 11. Causes *Source: TIN 9e Tables 230-1, 230-2, p. 1458*

Primary	Secondary
Autoimmune (isolated or polyglandular); tuberculosis (commonest worldwide); fungal, HIV/CMV; adrenal haemorrhage/thrombosis (anticoagulants, sepsis, DIC, meningococcaemia — Waterhouse-Friderichsen , antiphospholipid); drugs (etomidate, ketoconazole, mitotane, aminoglutethimide); infiltration (amyloid, sarcoid, haemochromatosis, metastases); bilateral adrenalectomy; CAH	Prolonged glucocorticoid therapy (commonest); Sheehan's syndrome; pituitary/hypothalamic tumour, craniopharyngioma; pituitary surgery, irradiation, head trauma; infiltration; CNS infection (TB, meningitis, fungal, HIV)

Recognition — clinical features

- **Crisis:** severe **hypotension refractory to vasopressors**; severe abdominal pain, nausea, vomiting mimicking an acute abdomen; confusion, lethargy; sepsis may coexist without fever.
- **Suspect** in unexplained hypotension with: steroid use, AIDS, TB, autoimmune disease, severe head injury, chronic fatigue with hyperpigmentation. Precipitants: infection (commonest, especially GI), surgery, burns, trauma, extreme exertion, **stopping chronic steroids**.
- **Cortisol deficit:** weight loss, weakness, lethargy, anorexia, vomiting, diarrhoea. **Aldosterone deficit:** dehydration, salt craving, orthostatic hypotension, syncope. **Androgen deficit** (women): loss of axillary/pubis hair, libido.

Table 12. Primary vs secondary adrenal insufficiency *Source: TIN 9e Table 230-3, p. 1458*

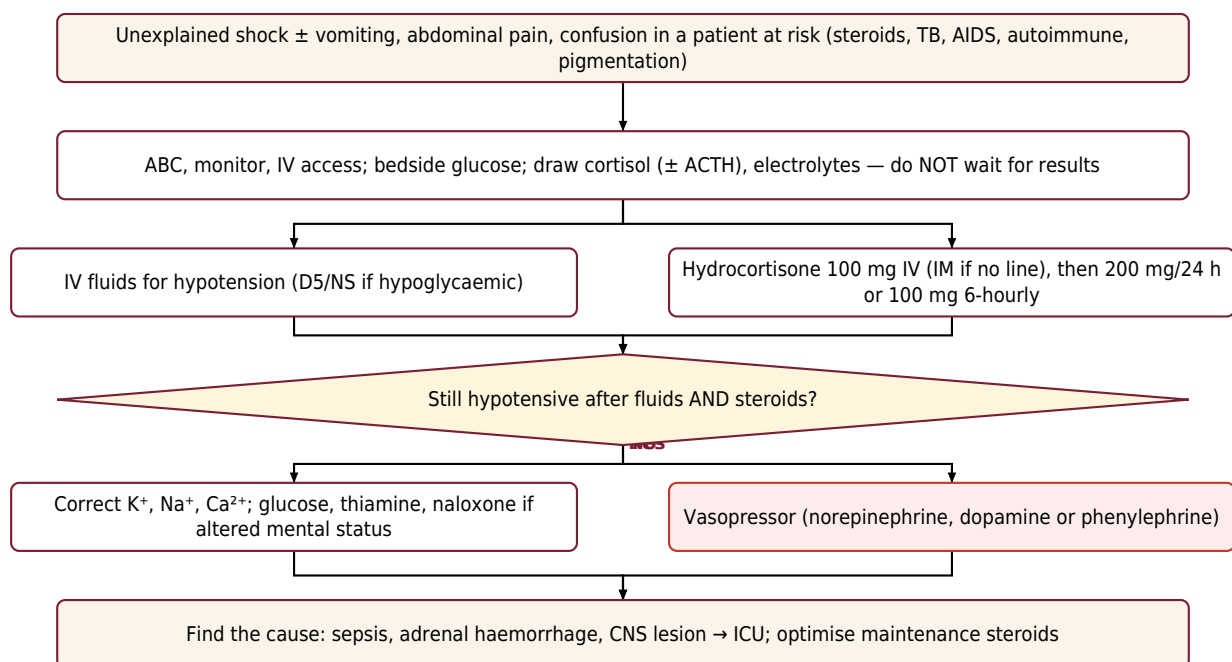
Feature	Primary	Secondary
Volume depletion, hypotension	Marked	Less, unless crisis
K ⁺ / Na ⁺	Hyperkalaemia / hyponatraemia	Hypokalaemia / hyper- or hyponatraemia
Pigmentation	Present (high ACTH)	Absent
Cushingoid look; other pituitary deficits	Absent	May be present
Glucose / cortisol / ACTH	Low / low / high	Low / low / low

Investigations

- **Bedside glucose;** CBC, electrolytes, calcium, cortisol, LFT, ECG (K⁺ effects), urinalysis; CXR (TB, pneumonia); CT abdomen (adrenal haemorrhage); CT/MRI head (secondary causes).
- Expected: hyponatraemia, hyperkalaemia (primary), hypoglycaemia, mild metabolic acidosis, hypercalcaemia.
- Random **cortisol >18 µg/dL** generally rules out insufficiency. Cosyntropin **250 µg IV** stimulation distinguishes types — if steroids cannot wait, give **dexamethasone** (minimal assay cross-reactivity).

Management basis: TIN 9e ch. 230 “Adrenal Insufficiency”, Table 230-4 (Treatment for Adrenal Crisis). Mechanism for the role of hydrocortisone and fluids: GUY 15e ch. 78.

ED approach to suspected acute adrenal crisis



Our own flowchart drawn from TIN 9e Table 230-4 and §Adrenal Crisis, pp. 1458–1459.

Role of hydrocortisone

- **Drug of choice:** **100 mg IV bolus** (IM if no IV), then **200 mg/24 h infusion or 100 mg IV every 6 h**; start immediately — prognosis depends on speed.
- Provides **both glucocorticoid and mineralocorticoid** effect, so it treats primary insufficiency alone (GUY Table 78.1: cortisol mineralocorticoid activity 1.0; dexamethasone ≈ 0).
- In stress a patient with Addison's may need **10 or more times** the normal glucocorticoid output (GUY).
- **Dexamethasone 4 mg** bolus if an ACTH stimulation test is planned — then add **fludrocortisone 100 µg/day** (no mineralocorticoid action).
- **Vasopressors** only after steroids; refractory shock responds to steroid, not to more pressor.
- **Maintenance:** hydrocortisone ≈ 20 mg/day (daily cortisol production) $\pm$ fludrocortisone; stress dose $\approx 3\times$ daily dose for 24–48 h in minor illness; 100 mg bolus in labour.

Role of fluid therapy

- Aldosterone loss $\rightarrow$ renal **Na⁺, Cl⁻ and water wasting** (10–20 g Na⁺/day) $\rightarrow$ low ECF and blood volume, $\downarrow$ cardiac output, **shock**; ADH rise adds hyponatraemia (GUY).
- Give **IV fluids early** with steroids; use **dextrose-containing saline (D5/NS)** when hypoglycaemic (cortisol loss blocks gluconeogenesis).
- Fluid loss is isotonic — replace with **normal saline**; child with salt-wasting crisis: **10–20 mL/kg NS** boluses (TIN ch. 146).
- Saline + hydrocortisone usually corrects hyperkalaemia without insulin–glucose (avoid insulin in infants).

WORKED EXAMPLE — Steroid arithmetic

Crisis, first 24 h: 100 mg bolus + 200 mg/24 h = **300 mg hydrocortisone**.

Mineralocorticoid cover (GUY Table 78.1, cortisol = 1.0, 9 α -fluorocortisol = 125): fludrocortisone 0.1 mg $\times$ 125 = **12.5 mg cortisol-equivalent**; hydrocortisone 300 mg = **300 mg-equivalent** $\rightarrow$ high-dose hydrocortisone needs no added fludrocortisone; dexamethasone (0) does.

Dexamethasone 4 mg $\times$ glucocorticoid potency 30 = **120 mg cortisol-equivalent**.

Sick-day rule: usual 20 mg/day $\times$ 3 = **60 mg/day** for 24–48 h.

Child doses (TIN ch. 146): hydrocortisone 25 mg neonate, 50 mg toddler/school-age, 100 mg adolescent.

Disposition

- Adrenal crisis $\rightarrow$ **ICU**. Discharge only mild insufficiency with known cause and a clear plan; follow-up in 24 h; return if vomiting or unable to keep tablets down.
- Patients on chronic steroids: think adrenal insufficiency in any acute illness; HPA recovery takes about a month or longer. Topical, inhaled, intranasal or PR steroids do not suppress the axis.

PEARLS

- ★ Shock unresponsive to pressors + steroid history or pigmentation = adrenal crisis until proven otherwise.
- ★ Hydrocortisone 100 mg IV/IM immediately, then 200 mg/24 h; do not wait for cortisol.
- ★ Dexamethasone only if a cosyntropin test is planned — add fludrocortisone.
- ★ Fluids: NS, D5/NS if hypoglycaemic; pressors only after steroids.

Sources

- TIN 9e, ch. 230 "Adrenal Insufficiency", §Introduction, §Physiology, §Primary/Secondary Adrenal Insufficiency, p. 1457; §Adrenal Crisis, §Clinical Features, Tables 230-1 to 230-3, p. 1458; §Laboratory Studies, §Treatment, §Disposition, §Special Populations, Table 230-4, p. 1459; §Pregnancy, §Sepsis, p. 1460.
- TIN 9e, ch. 146 "Metabolic Emergencies in Infants and Children", §Hypoglycemia treatment (hydrocortisone doses), pp. 966–967; §Congenital Adrenal Hyperplasia Treatment, p. 971.
- GUY 15e, ch. 78 "Adrenocortical Hormones", Table 78.1, p. 982; §Mineralocorticoid Deficiency, pp. 982–984; §Addison Disease, §Adrenal Crisis, pp. 993–994.

Part 19 · Haematological & Oncological Emergencies

16 answers

Peripheral blood smear examination. [5] — Jun 2020 · P-III · Q5(a)

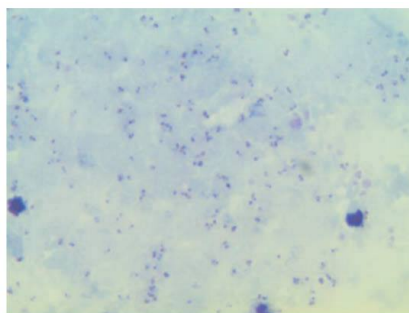
Asked in: Jun 2020 P-III Q5(a) · 4–5-mark length

What it is and when to send it

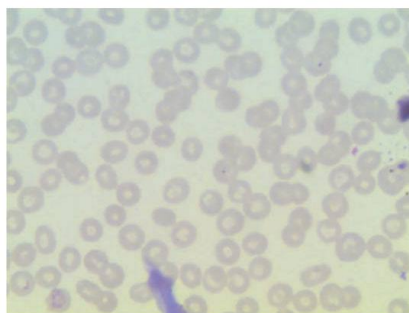
- Direct visualisation of **RBC morphology**, **WBCs** and **platelets**; estimates their quantity and appearance. It is **operator dependent** (TIN Table 232-2).
- **Anaemia**: if there is no evidence of bleeding, review RBC indices, reticulocyte count and peripheral smear (TIN Table 231-3). In children always send a smear and reticulocyte count with the CBC.
- **Thrombocytopenia**: assesses platelet morphology and the other cell lines; detects platelet clumping.
- **Suspected leukaemia** (CBC with differential + smear), suspected **haemolysis**, **HUS/TTP/HELLP** (schistocytes), **hyperviscosity**, and **fever after travel** (malaria films).

Technique (blood films)

- **Thin film** = a standard haematologic smear, **fixed with methanol and stained with Giemsa**: a single sheet of intact red cells, so RBC morphology and parasite species can both be examined.
- **Thick film**: a small drop of blood (**5 µL**) spread to about **1 cm**, dried, then stained **without fixation** (Field's or Giemsa); red cells lyse, so parasitaemia down to about 40/µL is detectable.
- Parasite density is counted per oil-immersion field or per 200 WBC nuclei (thick), or as % of red cells parasitised (thin).
- A thin film can miss parasitaemia below about 1000/µL; if the thin smear is negative a thick smear must be done, and repeated at least twice daily while malaria is suspected.



A



B

FIGURE 159-2. A. Thick and B. thin blood microscopy films from different children with malaria. The thick film shows numerous parasite nuclei, each with a faint blush of cytoplasm, seen either as a ring or as a smudge beside the nucleus. The thin film shows a single early ring stage of *Plasmodium falciparum* (center of field). The absence of dots in the erythrocyte cytoplasm and the normal size and shape of the erythrocyte distinguish this from *Plasmodium vivax* and *Plasmodium ovale*.

TIN 9e Figure 159-2, p. 1060 — A. Thick and B. thin blood films in malaria (single early ring stage of *P. falciparum* in B).

Findings and their clinical correlation

Table 13. Peripheral smear findings examined in emergency practice Source: *TIN 9e Tables 232-2, 237-1; pp. 1059-1060, 1469-1470, 1483, 1487-1491, 1518; ch. 161-162*

Finding	Meaning / associated condition
Spherocytes	Extravascular haemolysis (RBC phagocytosis by macrophages): warm AIHA, hereditary spherocytosis
Schistocytes (fragmented RBCs)	Intravascular haemolysis by shear: TTP, HUS, HELLP, DIC (present but not specific)
Sickle cells	Sickle cell disease; may give a new diagnosis
Abundant target cells (+ few sickle cells)	HbC disease; HbSC disease
Microcytosis, hypochromia, basophilic stippling	β -thalassaemia minor
Heinz bodies (precipitated Hb; crystal-violet stain)	Oxidative haemolysis in G6PD deficiency
RBC agglutination at cool temperature	Cold antibody AIHA
Polychromasia	Continued RBC production when a reticulocyte count is unavailable (SCD crisis)
Rouleaux (RBCs stacked like coins)	Hyperviscosity (macroglobulinaemia, myeloma)
Large, well-granulated, few platelets	Immune thrombocytopenia
Platelet clumping	Pseudothrombocytopenia (EDTA tube) — recount in citrate or heparin tube
Blasts / abnormal WBCs	Leukaemia interfering with coagulation
Ring forms with double chromatin dots, multiple rings per cell, crescent gametocytes	<i>P. falciparum</i> malaria
Intra-erythrocytic ring forms resembling malaria	Babesiosis (Giemsa or Wright stain)
Spirochaetes; sheathed microfilariae (nocturnal smear)	Relapsing fever; lymphatic filariasis

Limitations

- Operator dependent; qualitative more than quantitative.
- A single negative film does not exclude malaria; the first smear is positive in >90% of cases.
- Smear findings are interpreted with the MCV, RDW and reticulocyte count (TIN Figs 231-1 to 231-3).

PEARLS

- ★ Spherocytes = extravascular; schistocytes = intravascular (shear) haemolysis.
- ★ Platelet clumps on smear = pseudothrombocytopenia; repeat in a citrate tube.
- ★ Thin film: methanol-fixed, Giemsa — species and morphology. Thick film: unfixed — sensitivity.
- ★ Rouleaux = hyperviscosity; Heinz bodies = G6PD deficiency.

Sources

- TIN 9e, ch. 231 "Anemia and Polycythemia", §Diagnosis, Table 231-3, pp. 1461-1463; ch. 232 "Hemostasis", Table 232-2, p. 1468; ch. 233 "Acquired Bleeding Disorders", §Thrombocytopenia, §ITP, pp. 1469-1470.
- TIN 9e, ch. 236, §Vaso-Occlusive Pain Crisis, §HbSC, § β -Thalassemia Minor, §G6PD (Fig. 236-3), pp. 1483, 1487-1489; ch. 237 "Acquired Hemolytic Anemia", Table 237-1, §Warm and Cold AIHA, pp. 1490-1491; ch. 240, §Hyperviscosity Syndrome, p. 1518.
- TIN 9e, ch. 159 "Malaria", §Diagnosis, §Thick Blood Film, §Thin Blood Film, Fig. 159-2, pp. 1059-1060; ch. 144, p. 954; ch. 145, p. 956; ch. 161, pp. 1071, 1075; ch. 162, pp. 1085, 1091.



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