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& POCUS



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MODEL ANSWERS



GUIDELINE-
GRADED

REVIEWED BY

Dr. Mukul Sharma · Dr. Sandeep Mishra

DrNB Critical Care

Anchored to Irwin & Rippe's Intensive Care Medicine 9e and current guidelines

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Critical Care *Medicine*

SOLVED PREVIOUS-YEAR QUESTIONS



SAMPLE EDITION

Selected chapters from Volume I and Volume II



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Dr. Mukul Sharma and Dr. Sandeep Mishra (DrNB Critical Care)

PUBLISHED BY

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SOURCES

Irwin and Rippe's Intensive Care Medicine, 9th edition (2024), and guideline documents read from their official sources (see the Guideline register). Questions: NBEMS Critical Care Medicine theory papers (24 papers, up to April 2026).

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Contents of this preview

Volume I · Part 1 — Procedures, POCUS and Airway

pp. 18–60

1.1 POCUS: lung ultrasound, basic echo views and evaluation of dyspnoea	p. 19
1.2 RUSH and FALLS protocols; POCUS in fluid resuscitation	p. 24
1.3 Sedation and analgesia: drugs, titration scores and bispectral index	p. 29
1.4 Arterial pressure waveform	p. 32
1.5 Difficult and physiologically difficult airway	p. 35
1.6 Percutaneous tracheostomy: anatomy, methods, ultrasound/bronchoscopic guidance...	p. 40
1.7 Tracheo-innominate fistula	p. 45
1.8 Intercostal drain: applied anatomy and drainage systems	p. 48
1.9 High-quality CPR	p. 52
1.10 Defibrillation and synchronized cardioversion	p. 55
Part 2 opener — Monitoring and Applied Physiology (answer list)	p. 59

Volume II · Part 11 — Ventilation and ECMO

pp. 127–176

Part 11 opener (answer list)	p. 127
11.1 Respiratory failure and bedside non-invasive pulmonary monitoring	p. 128
11.2 ARDS: Berlin definition, severe ARDS and refractory hypoxaemia	p. 132
11.3 ARDS: effects on the kidney and cardiovascular system	p. 136
11.4 Methods of setting PEEP in ARDS	p. 139
11.5 Prone position ventilation	p. 141
11.6 Mechanisms of hypoxaemia	p. 144
11.7 Approach to hypercarbia	p. 146
11.8 Ventilator-induced lung injury	p. 150
11.9 Modes of mechanical ventilation and their classification	p. 153
11.10 APRV	p. 158
11.11 Closed-loop ventilation	p. 161
11.12 Heart–lung interactions during mechanical ventilation	p. 162
11.13 ECMO modalities and non-cardiac indications	p. 165
11.14 Inter-hospital air transport of ARDS patients to ECMO centres	p. 168
11.15 High-flow nasal cannula	p. 172

Index

PEARLS — what wins marks

- **One answer, many questions:** repeats and near-repeats are merged into one answer written to the most demanding version; the other identifiers are listed under "Also asked as".
- **Management basis strip** (amber) heads every Management section and names each source; guideline lines end with a source tag and grade, e.g. [SSC 2026; strong, moderate]; [I&R] marks lines from Irwin and Rippe where no guideline addresses the point.
- **Figures and schematics:** figures with a book figure number are real crops from Irwin and Rippe 9e; diagrams captioned "Schematic (drawn for these notes)" are drawn by us from facts in the answer, the textbook or a registered guideline, with the source named in the caption. The e-book has no printed pagination, so the textbook is cited by chapter, section and figure.
- **ISCCM documents** could not be read from official sources during this build (journal site blocks automated reading); where one exists it is listed in Index C as identified but not read, and management falls to the next level of the hierarchy.

A. Parts of the whole set

Part	Title	Answers	Questions	Location
Part 1	Procedures, POCUS and Airway	10	21	Vol 1 · p. 18
Part 2	Monitoring and Applied Physiology	14	26	Vol 1 · p. 59
Part 3	Ethics, Communication and End-of-Life Care	8	15	Vol 1 · p. 101
Part 4	Shock, Fluids, Sepsis and Trauma	18	42	Vol 1 · p. 132
Part 5	Surgical, Obstetric and Organ-Donor Critical Care	12	21	Vol 1 · p. 185
Part 6	Infections and Antimicrobials	19	39	Vol 1 · p. 236
Part 7	Haematology, Transfusion and Onco-Critical Care	15	37	Vol 1 · p. 306
Part 8	Toxicology and Envenomation	6	9	Vol 2
Part 9	ICU Organisation, Quality and Endocrine Emergencies	13	17	Vol 2
Part 10	Neurocritical Care	9	24	Vol 2
Part 11	Respiratory Failure, Ventilation and ECMO	28	52	Vol 2
Part 12	Cardiovascular Critical Care	14	20	Vol 2
Part 13	Kidney, Acid-Base and Electrolytes	15	32	Vol 2
Part 14	Gastrointestinal, Hepatic and Nutrition	20	37	Vol 2
Part 15	Research Methodology, Biostatistics and Landmark Trials	18	28	Vol 2
	Total	219	420	

B. Contents of Volume 1, question by question

#	Answer	Questions answered (marks)	Page
Part 1	Procedures, POCUS and Airway		p. 18
1.1	POCUS: lung ultrasound, basic echo views and evaluation of dyspnoea	Paper I · October 2023 · Q7 (10) Paper I · October 2025 · Q4 (10)	p. 19
1.2	RUSH and FALLS protocols; POCUS in fluid resuscitation	Paper III · January 2025 · Q6 (10) Paper I · December 2022 · Q7(a) (5)	p. 24
1.3	Sedation and analgesia: drugs, titration scores and bispectral index	Paper I · January 2025 · Q9(a) (6) Paper I · January 2025 · Q9(b) (4) June 2019 · Q2(a) (5)	p. 29
1.4	Arterial pressure waveform	Paper I · December 2022 · Q7(b) (5)	p. 32
1.5	Difficult and physiologically difficult airway	June 2019 · Q7(b) (5) Paper I · October 2023 · Q9 (10)	p. 35
1.6	Percutaneous tracheostomy: anatomy, methods, ultrasound/bronchoscopic guidance, complications	June 2019 · Q6(a) (5) Paper I · June 2022 · Q1(a) (4) Paper I · June 2022 · Q1(b) (3) Paper I · June 2022 · Q1(c) (3) Paper III · May 2024 · Q9 (10)	p. 40
1.7	Tracheo-innominate fistula	Paper I · April 2026 · Q8(b) (5)	p. 45
1.8	Intercostal drain: applied anatomy and drainage systems	Paper I · October 2023 · Q1 (10)	p. 48
1.9	High-quality CPR	Paper II · June 2022 · Q8(a) (5)	p. 52
1.10	Defibrillation and synchronized cardioversion	Paper I · October 2023 · Q10 (10) Paper II · June 2022 · Q8(b) (5) Paper I · May 2024 · Q4(a) (4)	p. 55
Part 2	Monitoring and Applied Physiology		p. 59
2.1	Pulse oximetry and perfusion index	Paper I · December 2022 · Q10(b) (5) Paper I · May 2024 · Q5(b) (4)	p. 61
2.2	Cardiac output monitoring: principles and non-invasive methods	Paper I · October 2023 · Q3 (10) Paper III · October 2025 · Q7(a) (7)	p. 64
2.3	Capnography	Paper I · June 2022 · Q3(a) (5)	p. 68
2.4	Haemoglobin–oxygen dissociation curve	Paper I · April 2026 · Q8(a) (5)	p. 71
2.5	Auto-PEEP (intrinsic PEEP)	Paper III · April 2023 · Q2 (10) June 2019 · Q10(a) (5) Paper I · June 2022 · Q7(a) (5)	p. 73
2.6	Driving pressure and mechanical power	Paper I · June 2022 · Q7(b) (5) Paper III · January 2025 · Q5(b) (5)	p. 77
2.7	Oesophageal/transpulmonary pressure and electrical impedance tomography	Paper I · April 2023 · Q3 (10) Paper I · December 2022 · Q4(a) (5) Paper III · October 2025 · Q3 (10) Paper III · December 2022 · Q8(b) (5) Paper III · April 2023 · Q9(b) (5)	p. 79
2.8	Ventilator scalars and loops (incl. volume–pressure loop)	Paper III · October 2023 · Q5 (10) Paper III · June 2022 · Q7(a) (5)	p. 83
2.9	Time constant in respiratory mechanics	Paper I · April 2026 · Q1(b) (5)	p. 88
2.10	ICP monitoring: techniques	Paper I · June 2022 · Q2(b) (5) Paper I · May 2024 · Q2(a) (4)	p. 90
2.11	Monro–Kellie doctrine	Paper I · May 2024 · Q2(b) (3)	p. 93
2.12	Jugular venous oximetry	Paper I · May 2024 · Q2(c) (3)	p. 94
2.13	Cerebral autoregulation	Paper I · December 2022 · Q9(b) (5)	p. 95
2.14	Transcranial Doppler and neuromonitoring of cerebral oedema in stroke	Paper III · January 2025 · Q4(a) (5) Paper III · October 2025 · Q10 (10)	p. 97
Part 3	Ethics, Communication and End-of-Life Care		p. 101
3.1	Artificial intelligence and machine learning in critical care	Paper III · April 2023 · Q9(a) (5) Paper I · May 2024 · Q9(a) (4)	p. 103
3.2	End-of-life care in the ICU	Paper III · October 2023 · Q6 (10) Paper II · April 2023 · Q8(a) (5) Paper II · May 2024 · Q6(a) (3)	p. 106
3.3	Advance medical directive and DNR orders in India	Paper III · December 2022 · Q6(a) (5) Paper III · January 2025 · Q7(b) (5)	p. 111

PART 1**Procedures, POCUS and Airway****Table 1.** Answers in Part 1

#	Answer
1	POCUS: lung ultrasound, basic echo views and evaluation of dyspnoea
2	RUSH and FALLS protocols; POCUS in fluid resuscitation
3	Sedation and analgesia: drugs, titration scores and bispectral index
4	Arterial pressure waveform
5	Difficult and physiologically difficult airway
6	Percutaneous tracheostomy: anatomy, methods, ultrasound/bronchoscopic guidance, complications
7	Tracheo-innominate fistula
8	Intercostal drain: applied anatomy and drainage systems
9	High-quality CPR
10	Defibrillation and synchronized cardioversion

Paper I · October 2025 · Q4 · 10 marks · Long

Discuss the utility of POCUS in the evaluation of dyspnoea in the emergency department. Write the specific findings that can guide management. Make different diagrams for specific findings. [3+2+5]

Also asked as: Paper I · October 2023 · Q7 “Describe in brief about lung USG and basic views of echocardiography. [5+5]”

1. What is being asked

- **Point-of-care (critical care) ultrasonography:** any ultrasound application performed by the treating intensivist at the bedside — **image acquisition, interpretation and immediate application** to the plan are all done by the same clinician.
- For the breathless patient it combines **lung ultrasonography (LUS)**, **basic critical care echocardiography (CCE)** and vascular (DVT) scanning — the “whole-body ultrasonography approach”.
- Why it matters: LUS can **replace the standard chest radiograph** and improves diagnostic accuracy in acute respiratory failure compared with standard clinical testing; it is immediately available, cheap, repeatable and radiation-free.

2. Equipment and scanning technique (lung)

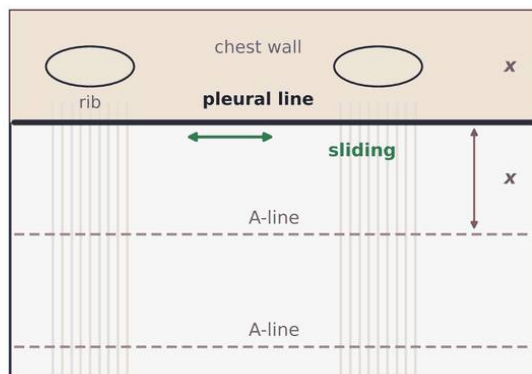
- **Probes:** 3.5–5.0 MHz phased-array or curvilinear for pleura and deeper structures; 7.5–10 MHz linear for lung sliding and detailed pleural morphology.
- Probe held like a pen, **longitudinal plane, perpendicular to the pleura**, orientation marker **cephalad** (cephalad structures to screen left).
- Correct orientation gives a **horizontal pleural line**; tilt the probe where the pleura is curved.
- **Zones:** anterior (sternum → anterior axillary line), lateral (anterior → posterior axillary line), posterior (posterior axillary line → spine).
- Simplest protocol (Lichtenstein): **three predefined points per hemithorax** — anterior, lateral, posterolateral.
- **Posterolateral scanning is essential** in the supine patient — effusion and consolidation collect dependently; press the probe into the mattress angled anteriorly, or roll the patient.
- Rib edges form a hyperechoic curve; the **pleural line lies 0.5–1 cm below the rib margin** regardless of body habitus.

3. Lung ultrasound signs

Table 2. Basic lung ultrasound signs and their meaning (Irwin and Rippe 9e, ch. 11)

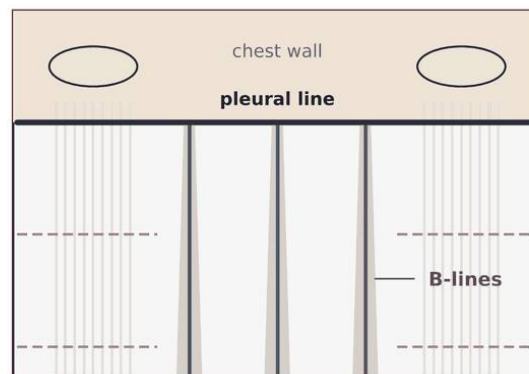
Sign	What is seen	Meaning
Lung sliding	Shimmering pleural line moving with respiration	Visceral pleura apposed to parietal pleura — rules out pneumothorax at that site
Lung pulse	Pleural line moving with cardiac pulsation	Also rules out pneumothorax at that site
Lung point	Intermittent respirophasic appearance of sliding against a background of absent sliding	Confirms pneumothorax: specificity 100%, sensitivity 66%
A-lines	Horizontal reverberation lines below the pleural line, spaced equal to probe–pleura distance	With sliding = normal aeration ; without sliding = pneumothorax or pleurodesis
B-lines	Vertical, hyperechoic, arise from pleural line, reach screen bottom, move with sliding, efface A-lines	Thickened interlobular septa or filled alveoli; >2 per field significant (except dependent bases); also rule out pneumothorax
Consolidation	Tissue-like echogenicity similar to liver ± air bronchograms	Airless lung of any cause; dynamic bronchograms suggest (not prove) pneumonia
Shred sign	Irregular deep border between consolidation and aerated lung	Pneumonia pattern
Pleural effusion	Usually anechoic space between pleurae; compressed lung floating in it	Effusion with compressive atelectasis

- Absence of sliding is **not diagnostic** of pneumothorax — also pleurodesis/pleural symphysis, **mainstem intubation**, bronchial occlusion (mucus plug, clot, tumour), and low-VT ventilation in ARDS.
- Consensus definition: a positive zone has **at least 3 B-lines in a single frame**; diffuse interstitial syndrome = multiple B-lines in **at least two zones per hemithorax**. [ILC-LUS 2026, D3_1.1–D3_1.4; Delphi consensus, no GRADE]

A. Normal aeration: A-lines + lung sliding

A-lines: reverberation of the pleural line, equally spaced, x apart (x = probe-pleura distance)

Sliding + A-lines = normal aeration; excludes pneumothorax at that site

B. B-lines (alveolar-interstitial pattern)

vertical, hyperechoic, arise at the pleural line, reach the screen bottom, efface A-lines

>2 per field significant (except dependent bases); B-lines also exclude pneumothorax at that site

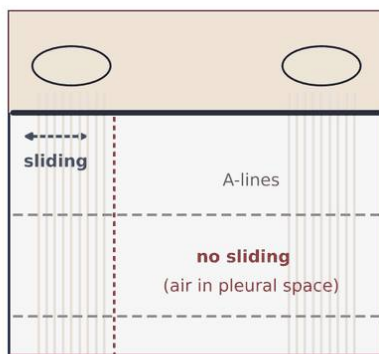
Profuse bilateral anterior B-lines + smooth pleural line = cardiogenic pulmonary oedema

Focal / segmental / lobar / unilateral B-lines = pneumonia; patchy B-lines + irregular pleura = primary lung injury

Schematic (drawn for these notes) — lung ultrasound screen patterns — A, normal aeration (equally spaced A-lines, lung sliding); B, B-lines and their distribution in pulmonary oedema, pneumonia and primary lung injury. Facts per Irwin and Rippe 9e, Ch 11 Lung Ultrasonography, §Findings of LUS and §Pattern Analysis of LUS. Not to scale.

C. Pneumothorax

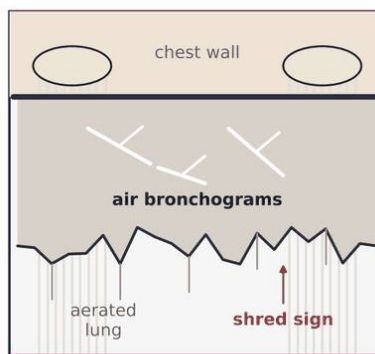
A-lines + absent sliding + lung point



Intermittent respirophasic appearance of sliding against absent sliding, entering from the screen edge.
Specificity 100%, sensitivity 66%

D. Consolidation (pneumonia)

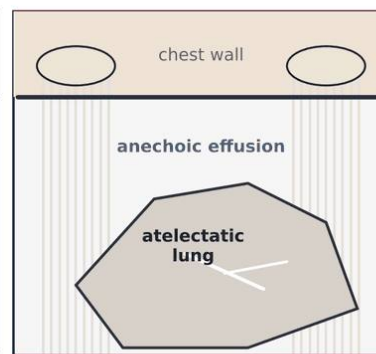
tissue density, similar to liver



Linear pleural interface; irregular deep border with comet-tail artefacts = shred sign; no volume loss

E. Pleural effusion

anechoic space between the pleurae



Compressive atelectasis: lung floats in the effusion, with respirophasic or cardiophasic movement; static air bronchograms

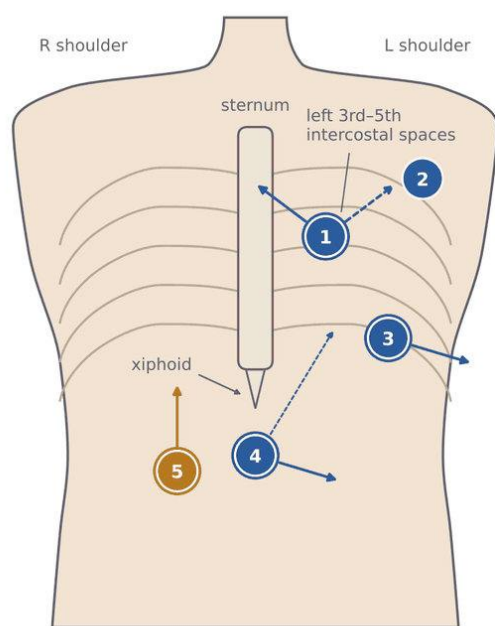
Absent sliding alone is NOT diagnostic of pneumothorax: pleural symphysis / pleurodesis, mainstem intubation, bronchial occlusion (mucus plug, clot, tumour) and low-tidal-volume ventilation in ARDS also abolish it

Schematic (drawn for these notes) — lung ultrasound findings that guide management — C, pneumothorax (absent sliding, A-lines, lung point); D, consolidation with air bronchograms and shred sign; E, pleural effusion with floating atelectatic lung. Facts per Irwin and Rippe 9e, Ch 11 Lung Ultrasonography, §Lung Point, §Consolidation, §Alveolar Consolidation Pattern. Not to scale.

4. Basic critical care echocardiography — the five views

Table 3. Basic CCE views (ACCP/SRLF statement as described in Irwin and Rippe 9e, ch. 16)

View	Probe position / marker	Assesses	Pitfall
Parasternal long axis (PSL)	Left 3rd–5th intercostal space beside sternum; marker to right shoulder	EF, RVOT, LV walls, AV/MV (colour Doppler), LA size, descending aorta, pericardium	Cannot size the RV; off-axis cut makes LV look hyperdynamic
Parasternal short axis (PSS), mid-ventricular	Rotate 90° clockwise from PSL; marker to left shoulder	EF, segmental LV function, LV/RV size, septal kinetics, pericardium	Elliptical LV = off-axis → false septal flattening
Apical four-chamber (AP4)	LV apex; marker at 3–4 o'clock; septum vertical, central	EF, RV/LV size ratio, RA/LA size, TV/MV, pericardium	Hardest view; counterclockwise rotation underestimates RV
Subcostal long axis (SCL)	Below xiphoid towards left shoulder; marker 3–4 o'clock	EF, RV/LV size, septum, pericardium	Translational artefact with respiration; often best view on ventilator and during CPR pulse checks
IVC longitudinal	From SCL rotate to 12 o'clock and angle right, or right mid-axillary coronal through liver	Volume responsiveness (diameter change)	Mistaking aorta for IVC; off-axis and translational artefact



(anterior view: patient's right is on the reader's left)

Five basic echo views

- 1 Parasternal long axis (PSL)**
beside sternum, left 3rd intercostal space, moving down to 4th–5th; marker to right shoulder
- 2 Parasternal short axis (PSS)**
rotate 90° clockwise from PSL; marker to left shoulder; mid-ventricular (papillary) level
- 3 Apical four-chamber (AP4)**
at the LV apex; marker 3–4 o'clock; septum vertical in the centre of the screen
- 4 Subcostal long axis (SCL)**
just below xiphoid towards left shoulder; marker 3–4 o'clock; patient supine
- 5 IVC long axis**
from SCL rotate counterclockwise to 12 o'clock and angle to the right (or right mid-axillary, coronal plane)

Solid arrow = index-mark direction of the probe (dashed in 1 = PSS marker after 90° clockwise rotation; dashed line in 4 = beam towards left shoulder). 12 o'clock = towards the head; 3 o'clock = patient's left.

Schematic (drawn for these notes) — transducer positions and index-mark directions for the five basic critical care echocardiography views (PSL, PSS, AP4, SCL, IVC). Facts per Irwin and Rippe 9e, Ch 16 Critical Care Echocardiography, §Basic Critical Care Echocardiography (each view subsection). Not to scale.

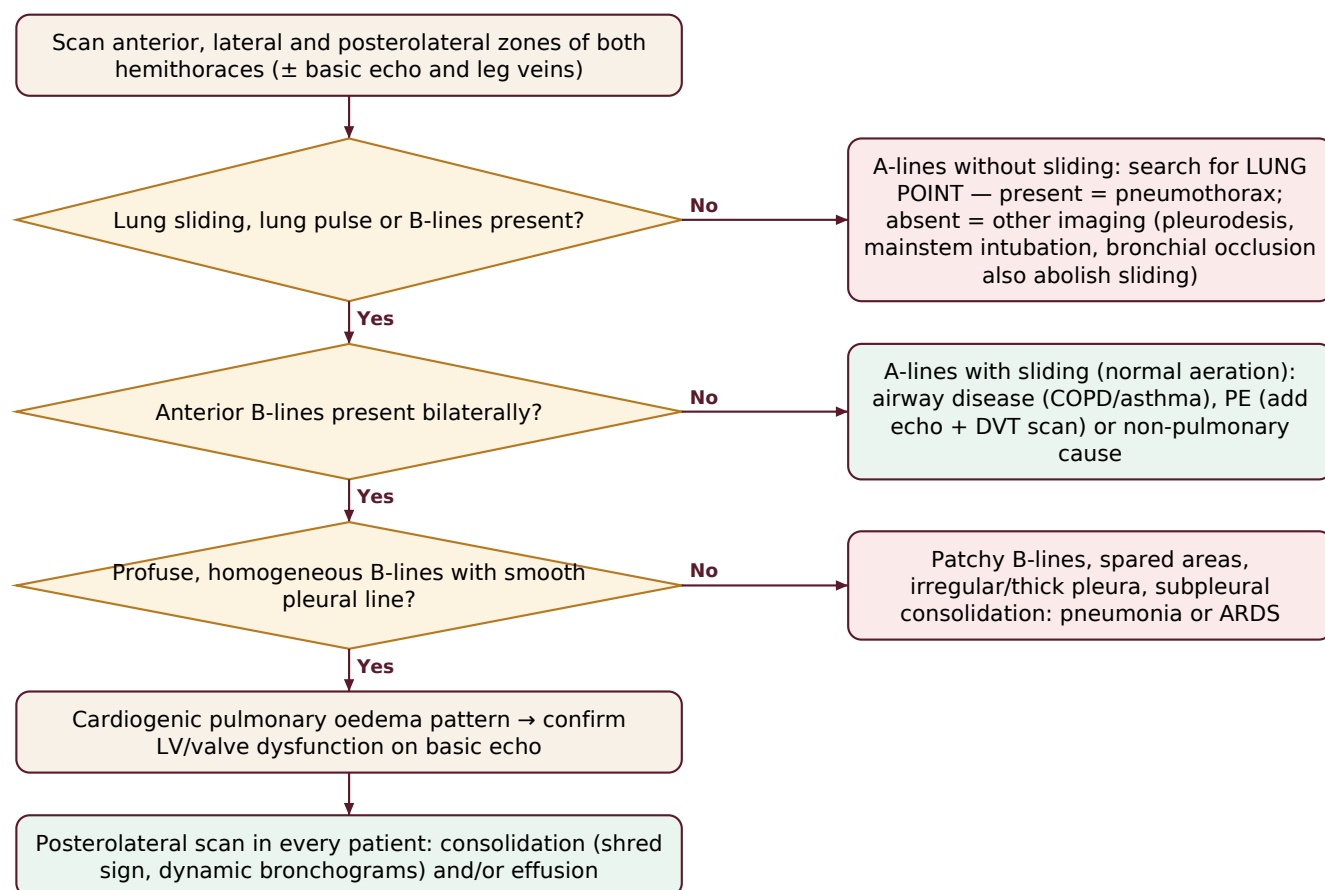
- Machine: portable 2D unit with **2–5 MHz phased-array** probe; colour Doppler recommended, spectral Doppler not mandatory for basic CCE.
- Competence in basic CCE needs **a minimum of 30 acceptable studies** (consensus).
- Limits of basic CCE: no stroke volume, PA pressure or quantitative valve data; cannot reliably exclude severe regurgitation — know when to ask for a full study or TEE.

5. Pattern analysis in acute dyspnoea / respiratory failure

- Generalised A-lines with sliding** (normal aeration) — excludes alveolar, interstitial and pleural disease; think of:
 - Airway:** COPD or asthma
 - Vascular:** pulmonary embolism
 - Non-pulmonary:** neurological, neuromuscular, metabolic, toxic

- **A-lines + absent sliding** → suspect pneumothorax; **lung point confirms**; no lung point → other imaging. Lung pulse, B-lines or consolidation exclude pneumothorax at that interspace.
- **Profuse bilateral anterior B-lines + smooth pleural line** → cardiogenic pulmonary oedema (CPE); requires the linear probe to judge the pleural line.
- **Focal, asymmetric B-lines, irregular pleural line** → primary lung injury (pneumonia, ARDS); segmental/lobar/unilateral B-lines are characteristic of pneumonia.
- **ARDS vs CPE** (Copetti): normal area coexisting with B-lines in one interspace — 100% of ARDS, 0% of CPE; pleural line >2 mm, coarse or with subpleural consolidation — 100% ARDS vs 25% CPE.
- **Consolidation**: pneumonia = tissue density without volume loss, shred sign, dynamic air bronchograms; hypoechoic well-defined area within it = necrosis/abscess.
- **Atelectasis**: static bronchograms; complete bronchial occlusion → no mobile bronchograms; compressive atelectasis floats in effusion.
- Unilateral profuse B-lines with smooth pleura: rare unilateral CPE from asymmetric mitral regurgitation.
- **Pulmonary embolism**: LUS + echo + DVT study reduce unnecessary CT angiograms; a definite alternative diagnosis on ultrasound excludes PE with high certainty.
- **COVID-19**: multifocal separate/confluent B-lines, irregular pleura, small consolidations, spared A-line areas; translobar consolidation and effusion are uncommon and suggest another process.

Flowchart. Lung ultrasound approach to the breathless patient



Drawn from Irwin and Rippe 9e, ch. 11 §Pattern Analysis of LUS; ILC-LUS 2026 Domain 2–3 statements.

SOURCE NOTE — BLUE-protocol

Irwin and Rippe 9e cites the BLUE-protocol (ch. 11, ref. 4) but does not describe it. Its profiles below are taken from the protocol author's description (Lichtenstein, Ann Intensive Care 2014), registered for this Part; the textbook's own pattern analysis above is the primary basis.

- **A-profile**: anterior sliding + A-lines; with DVT → PE (sensitivity 81%, specificity 99%); without DVT or PLAPS ("nude profile") → severe asthma/COPD.

- **B-profile:** anterior sliding + lung rockets → haemodynamic pulmonary oedema (sensitivity 97%, specificity 95%).
- **A'-profile** (A-profile without sliding) + lung point → pneumothorax.
- **B'-, A/B-, C-profiles and A-V-PLAPS** → pneumonia. Overall accuracy **90.5%** for six diseases seen in 97% of ED dyspnoea; takes about **3 minutes**. [Lichtenstein 2014]

6. Specific findings that guide management

MANAGEMENT BASIS

International evidence-based recommendations for point-of-care lung ultrasound — 2025 focused update (ILC-LUS, Intensive Care Med 2026; Delphi consensus, GRADE not applied) and the 2012 recommendations (Intensive Care Med 2012). No ISCCM document on POCUS was found. Lines tagged [I&R] are from Irwin and Rippe 9e, ch. 11, 13, 16, 17.

Table 4. Ultrasound finding → diagnosis → management cue in acute dyspnoea

Finding	Diagnosis	Management cue (source)
Absent sliding + A-lines + lung point	Pneumothorax	LUS signs can be sufficient indication to proceed with immediate drainage [ILC-LUS 2026, D2_2.3]; LUS also guides tube removal timing [I&R]
Bilateral homogeneous B-lines, smooth pleura; poor LV function	Cardiogenic pulmonary oedema	Absence of diffuse interstitial syndrome helps rule out pulmonary oedema [ILC-LUS 2026, D3_3.2]; treat as heart failure
Patchy B-lines, spared areas, abolished sliding, subpleural consolidations	ARDS	LUS is the first-line diagnostic tool for ARDS [ILC-LUS 2026, D3_4.2]; LUS score tracks PEEP response [I&R]
Tissue-like consolidation, shred sign, dynamic bronchograms	Pneumonia	Supports antibiotic decision; resolution can be tracked by LUS [I&R]
Anechoic pleural space, floating lung	Pleural effusion	Site selection for thoracentesis/drain under ultrasound [I&R ch. 13]
Normal LUS + dilated RV ± DVT	Pulmonary embolism	Proceed to confirm/treat PE; massive PE is one of the life-threatening causes basic echo identifies [I&R ch. 16]; RV dilatation points to PE [ACEP RUSH]
Pericardial effusion with RA systolic or RV diastolic collapse, plethoric IVC	Tamponade	Pericardial drainage; small IVC makes tamponade very unlikely [I&R ch. 17]
Normal LUS, no DVT, no PLAPS	Asthma/COPD exacerbation	Bronchodilator pathway (A-profile "nude") [Lichtenstein 2014]

- **Extubation:** Rouby LUS score (0 = A-lines, 1 = spaced B-lines, 2 = confluent B-lines, 3 = consolidation/effusion; 6 interspaces per side; maximum 36) — rise of **>4 points** during a 30-min SBT, or post-SBT score **>17**, predicts extubation failure. [I&R]
- **PEEP titration:** fall of 8 points in the LUS score from PEEP 0 to 15 → lung volume gain >600 mL (cannot separate recruitment from overdistension). [I&R]
- **Chest tube removal** for pneumothorax: confirm re-expansion, clamp, re-scan — superior to chest radiography. [I&R]
- **Tamponade echo:** mitral E-wave respirophasic variation >30% is characteristic; chamber collapse is suggestive, not diagnostic. [I&R ch. 17]
- Lung ultrasound **rules out pneumothorax more accurately than supine anterior CXR** and may be the better initial study in the critically ill. [ILC-LUS 2026, D2_6.2–D2_6.3, carried forward]
- When the lung point cannot be found, use **CXR or CT**. [ILC-LUS 2026, D2_2.2]
- LUS integrated with clinical assessment **shortens time to correct diagnosis**. [ILC-LUS 2026, D3_3.4]

7. Limitations and pitfalls

- Patient factors: dressings, chest tubes, obesity, poor positioning; hyperinflation (ventilation, COPD) blocks windows.
- Physics: air and bone reflect or attenuate the beam — aerated lung itself is never seen, only artefacts.
- Operator: commonest failure is poor gain, depth, frequency and orientation; competence needs supervised scanning and an image library.
- B-lines and consolidation are **non-specific** — LUS identifies the pattern, the clinician determines the cause.

- Documentation of serial examinations is difficult in busy ICUs.

8. Recent advances (as stated in the sources)

- Ultraportable probes connected to phones/tablets with one multifrequency transducer for cardiac, abdominal and vascular imaging. [I&R ch. 1]
- Transesophageal lung ultrasonography (TELUS) images the lower lobes and posterolateral pleura during critical care TEE. [I&R ch. 11]
- ILC-LUS 2025 focused update: 83 consensus statements; its figures show an aeration score in 12 zones and a congestion score by a 28-scan antero-lateral technique. [ILC-LUS 2026]

PEARLS — what wins marks

- LUS signs: sliding/pulse/B-lines exclude pneumothorax; **lung point confirms it** (specificity 100%, sensitivity 66%).
- Profuse bilateral B-lines + smooth pleura = **cardiogenic oedema**; patchy B-lines + irregular pleura + spared areas = **ARDS/pneumonia**.
- Normal aeration (A-lines + sliding) in dyspnoea → COPD/asthma, PE or non-pulmonary cause.
- Five basic echo views: **PSL, PSS, AP4, subcostal, IVC**; RV size from AP4/subcostal, not PSL.
- Probe choice: phased-array 2–5 MHz (heart), 3.5–5 MHz (lung), 7.5–10 MHz linear (pleural line).
- Rouby LUS score: max 36; rise >4 during SBT or post-SBT >17 predicts extubation failure.
- Trap: absent sliding is not pneumothorax by itself — check for mainstem intubation and pleurodesis.

Textbook: Irwin and Rippe 9e, ch. 1 "Point-of-Care Critical Care Ultrasonography": §Introduction, §Machine Requirements, §Competence, §Limitations. Ch. 11 "Lung Ultrasonography": §Basic Principles, §Machine Requirements, §Performance of LUS, §Findings of LUS (Lung Sliding, Lung Point, A-Lines, B-Lines, Consolidation), §Pattern Analysis of LUS, §Clinical Applications (ARDS vs CPE, Extubation, PEEP, PE, Chest Tube Removal), §LUS and COVID-19, §TELUS. Ch. 16 "Critical Care Echocardiography": §Basic Critical Care Echocardiography (five views, utility, pitfalls, limitations). Ch. 17 "Pericardiocentesis", §Assessment for Pericardial Tamponade. Ch. 13 "Chest Tube Insertion and Care", §Utility of Ultrasonography.

Guidelines: ILC-LUS, Volpicelli G, Gargani L, et al., "International evidence-based recommendations for point-of-care lung ultrasound: 2025 focused update of the 2012 recommendations", *Intensive Care Med* 2026;52:1415–1446, statements D1–D4 (Domains 5–7 not readable). doi:10.1007/s00134-026-08487-2. ILC-LUS, Volpicelli G et al., "International evidence-based recommendations for point-of-care lung ultrasound", *Intensive Care Med* 2012;38:577–591. doi:10.1007/s00134-012-2513-4. Lichtenstein DA, "Lung ultrasound in the critically ill", *Ann Intensive Care* 2014;4:1 (BLUE-protocol description; not a guideline). doi:10.1186/2110-5820-4-1.

Paper III · January 2025 · Q6 · 10 marks · Long

Describe the "RUSH" (Rapid ultrasound in shock) and the "FALLS" (Fluid administration limited by lung sonography) protocols for the bedside ultrasonographic evaluation of a patient of SHOCK in the ICU. [5+5]

Also asked as: Paper I · December 2022 · Q7(a) "Role of POCUS in fluid resuscitation. [5]"

1. Why ultrasound in shock

- Shock is often multifactorial (vasoplegia, transient cardiomyopathy, venous underfilling) and its type may not be obvious clinically. [I&R ch. 28]
- The five basic CCE views allow rapid categorisation of shock as **cardiogenic, obstructive, hypovolaemic or distributive**, and early identification of tamponade, major valve failure, severe LV dysfunction or massive PE — this justifies **early basic CCE in every patient in shock**. [I&R ch. 16]

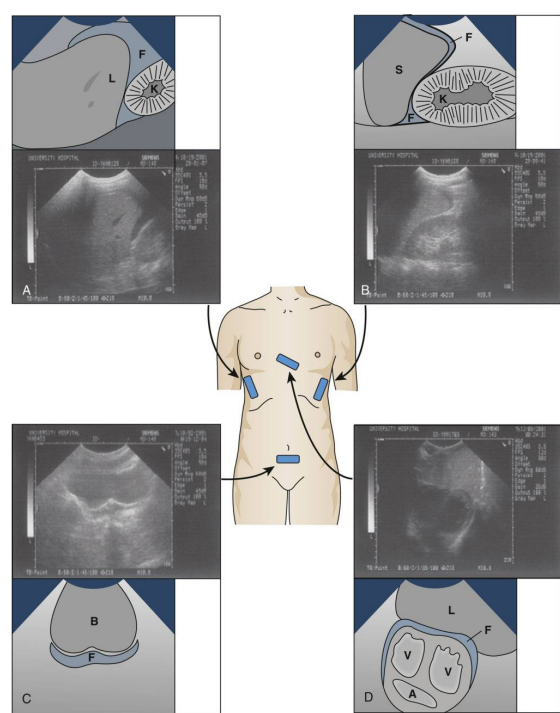
- A structured POCUS protocol raised diagnostic accuracy for undifferentiated shock from **60.6% to 85%** and changed initial management in up to 50%. [ACEP RUSH]

2. RUSH — Rapid Ultrasound for Shock and Hypotension

- Original protocol remembered as **HI-MAP**: **H**eat, **I**VC, **M**orison's pouch (FAST), **A**orta, **P**neumothorax. [ACEP RUSH]
- Expanded "**pump, tank, pipes**" version adds pleural effusion, interstitial oedema (lung) and lower-limb DVT. [ACEP RUSH]
- Comprehensiveness depends on patient stability, time and clinical suspicion; findings are integrated and compared with the expected profile of each shock type; repeat examinations. [ACEP RUSH]

Table 5. RUSH components — what to look for (ACEP Sonoguide; textbook detail from Irwin and Rippe 9e)

Component	Views	Key findings and numbers
Pump — heart	PSL, PSS, AP4, subcostal (I&R ch. 16)	Pericardial effusion/tamponade: RA systolic or RV diastolic collapse, IVC distended, mitral E variation >30% (I&R ch. 17); ACEP: mitral inflow variation >25% or tricuspid >40%. Contractility: hyperdynamic → distributive/haemorrhagic; hypodynamic → cardiogenic; EPSS >7 mm = low EF. RV:LV >1:1, TAPSE <16 mm, McConnell sign → RV strain (PE)
Tank — IVC	IVC longitudinal, ~2 cm from RA junction	Small, collapsible IVC → hypovolaemia; large IVC with little variation → obstructive or cardiogenic. ACEP gives no numeric cut-off; IVC alone can mislead
Tank — FAST / pleura	Hepatorenal, splenorenal, pelvic views; dependent thorax (I&R ch. 36)	Free fluid = intra-abdominal bleeding (RUQ detects as little as 200 mL — ACEP); pleural fluid in haemorrhagic shock = haemothorax
Tank — lung	Anterior 3rd–4th intercostal space, midclavicular line	Absent sliding (~95% sensitive) + lung point (~100% specific) = pneumothorax; diffuse bilateral B-lines = pulmonary oedema
Pipes — aorta	Infrarenal aorta, outer wall to outer wall	AAA >3 cm; presumptive rupture considered when >5 cm; aortic root >3.8 cm or haemopericardium → type A dissection
Pipes — veins	Compression ultrasound of leg veins	DVT (~96% sensitivity/specificity); positive DVT raises PE sensitivity of echo from 32% to 90%; negative scan does not exclude PE



Irwin and Rippe 9e, Figure 23.2 (ch. 23): the FAST examination — right upper quadrant (hepatorenal), left upper quadrant (splenorenal), pelvic and subxiphoid cardiac windows with the free-fluid locations (F).

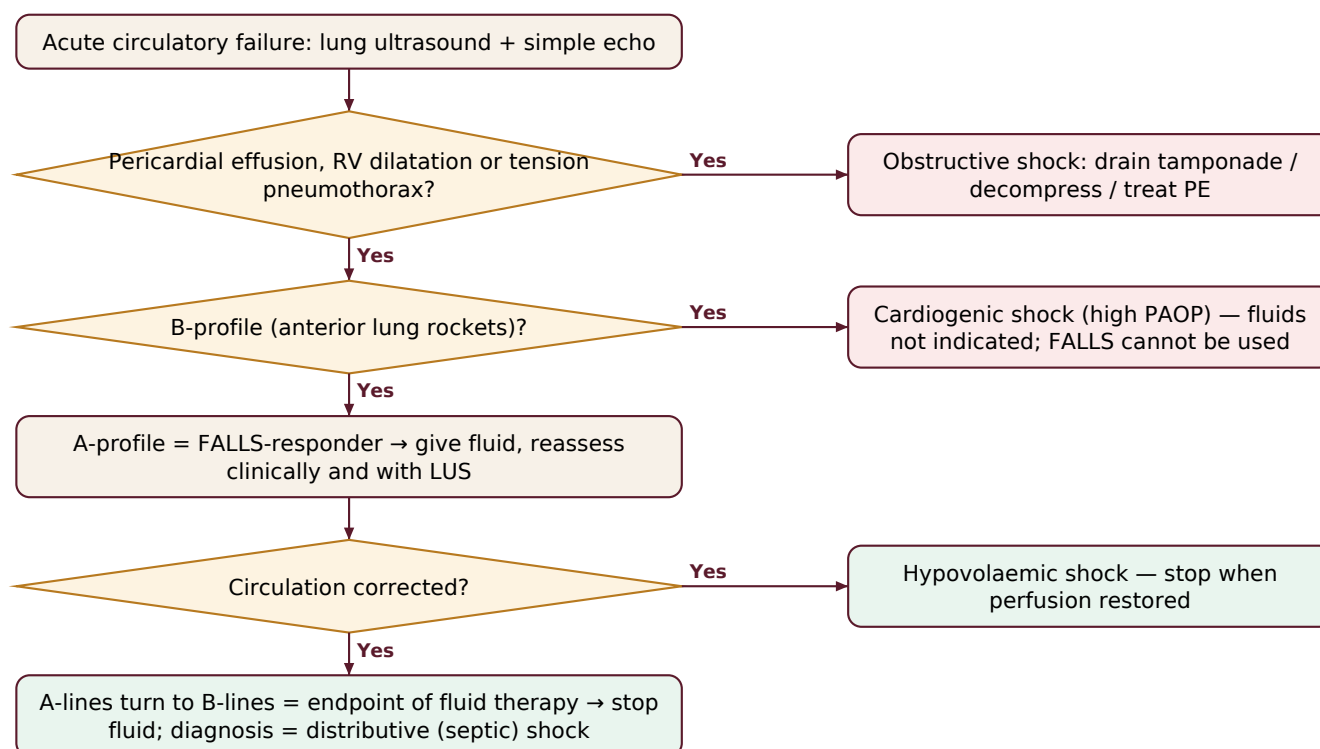
Table 6. Shock phenotypes on RUSH (ACEP Sonoguide; Irwin and Rippe 9e, ch. 16, 17, 36)

Shock type	Pump	Tank	Pipes
Hypovolaemic	Hyperdynamic LV, small atria	Small collapsible IVC; ± free fluid / haemothorax	Ruptured AAA as a cause
Cardiogenic	Hypodynamic contractility	Large IVC, little variation; generalised bilateral B-lines (oedema)	—
Obstructive	Tamponade, or dilated RV (PE)	Large IVC; absent sliding + lung point (tension pneumothorax)	DVT (PE)
Distributive	Hyperdynamic contractility	May show an infective source (e.g. FAST fluid); FALLS: A-profile → fluid until B-lines	—

3. FALLS — Fluid Administration Limited by Lung Sonography

- Adapts the BLUE-protocol to **acute circulatory failure**; makes a **sequential search** for obstructive, cardiogenic, hypovolaemic and distributive shock. [Lichtenstein 2014]
- Step 1 — **Obstructive**:
 - Substantial pericardial effusion (tamponade)
 - RV dilatation (suggesting PE in this context)
 - Tension pneumothorax (A'-profile + lung point)
- Step 2 — **Cardiogenic (left heart)**: defined by low cardiac output and **high PAOP**; in the **absence of a B-profile**, such cardiogenic shock can be discounted.
- Step 3 — remaining causes are **hypovolaemic and distributive**: patients with an **A-profile** (dry lungs) are "FALLS-responders" who can, and mostly must, receive fluid.
- Hypovolaemic shock improves with fluid; if no improvement occurs, fluid eventually enters the lung — the cause is then **distributive (septic) shock**.
- Endpoint**: the change from **A-lines to B-lines** marks the end of fluid therapy — B-lines appear at a PAOP of about **18 mm Hg**; interstitial oedema precedes alveolar oedema and is clinically silent, before gas exchange worsens.
- A **static** parameter usable at the start; **cannot be used if a B-profile is present on admission**.

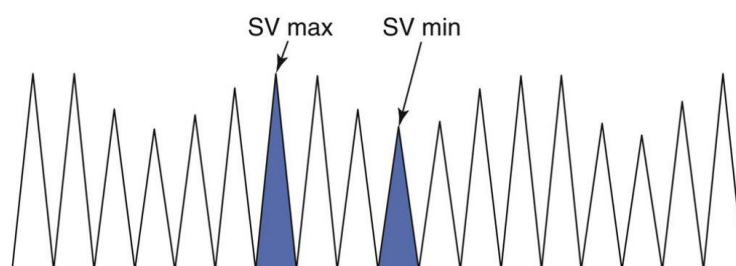
Flowchart. FALLS-protocol sequence



Drawn from the FALLS-protocol description, Lichtenstein, Ann Intensive Care 2014 (not a guideline). Main path = "No" at the first two diamonds.

4. Role of POCUS in fluid resuscitation

- Decision to give fluid should rest on **objective assessment of fluid responsiveness**, not empiric trial and error, given the harm of excessive fluid balance. [I&R ch. 28]
- **Static CVP and PAOP** are poor predictors of fluid responsiveness. [I&R ch. 163, 164]
- **IVC**: diameter and its respiratory change are used to judge volume responsiveness; small IVC or respirophasic variation suggests hypovolaemia. [I&R ch. 16, 36]
- IVC pitfalls: mistaking aorta for IVC, off-axis cut, **translational artefact** on the ventilator; IVC size unreliable with **intra-abdominal hypertension**. [I&R ch. 16, 51]
- In pregnancy POCUS has used **IVC <1.5 cm with significant respiratory variation** as volume responsive (not validated). [I&R ch. 164]
- Echocardiographic SV variability, IVC distensibility, PPV and **passive leg raise** all show reasonably high accuracy for predicting fluid responsiveness — whether they improve outcome is unproven. [I&R ch. 163]
- **VTI** (velocity–time integral) $\times$ LVOT area estimates stroke volume; Δ VTI with PLR or fluid tracks response. [I&R ch. 28]
- Hyperdynamic LV, small atria and increased EF on the subxiphoid view suggest hypovolaemia. [I&R ch. 36]
- **Lung ultrasound as a safety limit**: appearance of new B-lines warns of interstitial oedema (FALLS endpoint). [Lichtenstein 2014]
- Ultrasound also detects the **source** (FAST fluid, haemothorax, pneumonia) and contra-indications to fluid (tamponade needs drainage; RV failure, cardiogenic shock). [I&R ch. 16, 36]
- Serial scans show treatment response, which may appear on ultrasound sooner than in vital signs, and help avoid harmful aggressive fluid in cardiogenic shock. [ACEP RUSH]



Irwin and Rippe 9e, Figure 28.2 (ch. 28): stroke volume variation — maximum and minimum stroke volumes over the respiratory cycle; larger variation predicts volume responsiveness in the passively ventilated patient.

MANAGEMENT BASIS

Surviving Sepsis Campaign 2026 international guidelines (Crit Care Med 2026) for the fluid and haemodynamic recommendations; the guideline states that the use of critical care ultrasound was not evaluated. Protocol content: ACEP Sonoguide RUSH (2021) and Lichtenstein 2014 (FALLS). No ISCCM document on shock ultrasound was found. Lines tagged [I&R] from Irwin and Rippe 9e.

Table 7. Fluid-resuscitation recommendations that POCUS is used to implement (SSC 2026)

Recommendation	Strength; certainty
Sepsis-induced hypoperfusion or septic shock: at least 30 mL/kg IV crystalloid in the first 3 h	Conditional; low
Use dynamic measures to guide fluid resuscitation over physical examination or static measures alone	Conditional; low
After 30 mL/kg with persistent hypoperfusion: liberal or restrictive strategy based on patient and system factors	Conditional; low
Capillary refill time to guide resuscitation as an adjunct to other perfusion measures	Conditional; low
Serial lactate to guide resuscitation	Conditional; low
Crystalloids first-line; balanced crystalloids over 0.9% saline	Strong; moderate / Conditional; moderate
Active fluid removal after the acute resuscitation phase	Conditional; very low
Initial MAP target 65 mm Hg ; norepinephrine first-line	Strong; moderate / Strong

5. Limitations

- Operator-dependent; image quality limited by habitus, dressings, hyperinflation. [I&R ch. 1, 16]
- IVC indices need a passive patient and an accurate longitudinal cut; misleading if used alone. [I&R ch. 16; ACEP RUSH]
- FALLS endpoint is static and unusable when B-lines are present at baseline (ARDS, CPE, fibrosis). [Lichtenstein 2014]
- Retroperitoneal bleeding is poorly seen — high false-negative rate; go to CT/angiography when suspected. [I&R ch. 36]
- Aortic dissection is poorly detected by POCUS (specific but insensitive). [ACEP RUSH]

PEARLS — what wins marks

- RUSH = **HI-MAP** (Heart, IVC, Morison's pouch, Aorta, Pneumothorax) → expanded **Pump–Tank–Pipes**.
- Hyperdynamic heart + small collapsing IVC = hypovolaemia; large non-collapsing IVC = cardiogenic/obstructive.
- FALLS order: **obstructive** → **cardiogenic** → **hypovolaemic** → **distributive**.
- FALLS endpoint: **A-lines turning into B-lines** (≈PAOP 18 mm Hg) — stop fluid.
- No B-profile = left-heart cardiogenic shock discounted.
- SSC 2026: dynamic measures over static (conditional, low); 30 mL/kg in 3 h (conditional, low).
- Trap: IVC is unreliable with intra-abdominal hypertension and with translational artefact on the ventilator.

Textbook: Irwin and Rippe 9e, ch. 16 "Critical Care Echocardiography", §Inferior Vena Cava Longitudinal View, §Clinical Applications of GDE (life-threatening causes; categorisation of shock). Ch. 17 "Pericardiocentesis", §Assessment for Pericardial Tamponade. Ch. 23 "Paracentesis and Diagnostic Peritoneal Lavage", Fig. 23.2. Ch. 28 "Minimally Invasive Hemodynamic Monitoring", §Key Points, §Estimating Cardiac Output (VTI), §Pulse Contour Analysis — Clinical Utility, Fig. 28.2. Ch. 36 "Resuscitation From Shock Following Hemorrhage", §Utility of Ultrasonography (FAST, haemothorax, retroperitoneal bleed). Ch. 51 "Abdominal Compartment Syndrome", §Cardiovascular Effects. Ch. 163 "ARDS", §Pharmacological Interventions (fluid responsiveness). Ch. 164 "Acute Respiratory Failure in Pregnancy", §Diagnostic Testing.

Guidelines: ACEP Sonoguide, Stolz LA, Shah A, "Rapid Ultrasound for Shock and Hypotension (RUSH)", 10 Nov 2021, [acep.org/sonoguide/advanced/rush](https://www.acep.org/sonoguide/advanced/rush) (society educational statement). Lichtenstein DA, "Lung ultrasound in the critically ill", *Ann Intensive Care* 2014;4:1, §FALLS-protocol. doi:10.1186/2110-5820-4-1. Surviving Sepsis Campaign, Prescott H et al., "International guidelines for management of sepsis and septic shock 2026", *Crit Care Med* 2026, haemodynamic statements (SCCM guideline page). doi:10.1097/CCM.0000000000007075.

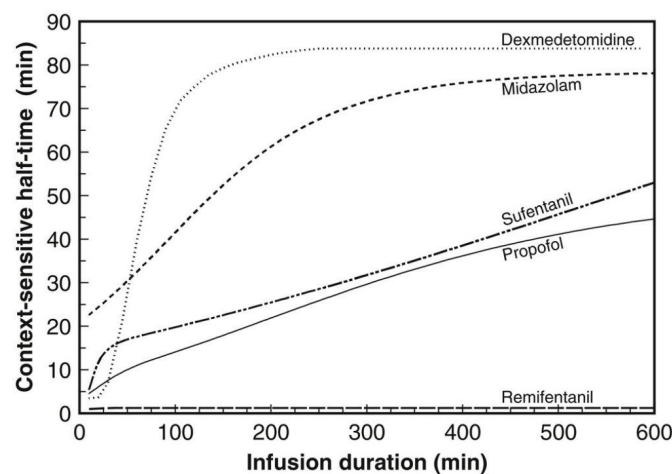
Paper I • January 2025 • Q9(a) • 6 marks • Intermediate

Discuss the common drugs used for sedation and analgesia in ICU patients. [6]

Also asked as: Paper I • January 2025 • Q9(b) “Describe any two scoring systems used for titration of such agents in critically ill patients. [4]” • **FNB • June 2019 • Q2(a)** “Bispectral index.”

Why it matters

- Under-treatment → pain, anxiety, agitation, self-removal of tubes, myocardial ischaemia, ventilator dyssynchrony, hypoxaemia.
- Over-sedation → delirium, pressure injury, nerve compression, prolonged ventilation, VAP and possibly PTSD.
- Critical illness alters pharmacokinetics: hypoalbuminaemia raises free drug, renal/hepatic failure slows clearance, ageing increases sensitivity to opioids and benzodiazepines.
- **Context-sensitive half-time** (time for plasma level to fall 50% after stopping an infusion) predicts offset after infusions.



Irwin and Rippe 9e, Figure 2.1 (ch. 2): context-sensitive half-times of dexmedetomidine, midazolam, sufentanil, propofol and remifentanil against infusion duration.

Common drugs — analgesics

Table 8. Analgesics in ICU (Irwin and Rippe 9e, Table 3.1 and ch. 2–3)

Drug	Front-load / bolus	Increment or infusion	Key points / cautions
Morphine	0.08–0.12 mg/kg	0.03 mg/kg every 10 min	Histamine release → hypotension, bradycardia, bronchospasm; M6G accumulates in renal failure
Fentanyl	1–3 µg/kg	0.5–2.00 µg/kg/h	Rapid onset; accumulates with infusion; chest-wall rigidity with large rapid bolus
Remifentanyl	0.25–1.00 µg/kg	0.05–2.00 µg/kg/min	CSHT ≈4 min regardless of duration; esterase metabolism unchanged in liver/renal failure; pain on stopping
Hydromorphone	0.02 mg/kg	25–50 µg/kg every 10 min	As morphine; dosing errors
Ketamine	0.2–0.5 mg/kg	0.5–2.00 mg/kg/h	Analgesia with preserved airway reflexes; delirium, ↑myocardial O ₂ demand
Acetaminophen	—	<4 g/day (2–3 g/day in cirrhosis on long-term use)	Opioid-sparing; earlier extubation, less delirium and PONV
NSAIDs	—	—	Cut opioid use 30–50% but GI, renal, CV risks — not routine

Common drugs — sedatives

- **Propofol** (GABA): bolus 1–2 mg/kg, infusion 100–200 µg/kg/min for anaesthesia (reduce 20–50% in elderly); faster emergence than midazolam; **no analgesia**; CSHT ≈10 min after 1 h; hypotension (negative inotropy + vasodilation); lipid vehicle supports bacterial growth; propofol infusion syndrome with prolonged high doses (ch. 125, 151).
- **Midazolam**: bolus 0.05–0.15 mg/kg, infusion 0.25–1.5 µg/kg/min (Table 2.3); 95% protein bound; accumulates in obesity, elderly, renal failure (active metabolites); recovery delayed up to 24 h; **benzodiazepines carry the highest delirium risk**.
- **Dexmedetomidine** (α_2 -agonist, locus coeruleus): bolus 1 µg/kg over 10 min, infusion 0.003–0.012 µg/kg/min (ch. 2) — Table 8.4 gives 0.2–1.2 µg/kg/h; sedation **without ventilatory depression**, opioid-sparing; bradycardia, hypotension; avoid in heart block; shorter ventilation than midazolam (5.1 vs 6.8 days).
- **Ketamine**: dissociative; bronchodilator; preserves hypoxic pulmonary vasoconstriction; meta-analyses show no ICP rise when adequately sedated and ventilated; emergence phenomena.
- **Etomidate**: haemodynamically neutral; blocks 11 β -hydroxylase — infusion not recommended (prolonged adrenal suppression).

Scoring systems for titration (two described)

- **Behavioral Pain Scale (BPS)** — earliest, most tested tool for sedated patients:
 - 3 domains: facial expression, upper-limb movement, compliance with ventilation
 - Each 1–4 → total **3 (no pain) to 12 (maximum pain)**
- **Critical-Care Pain Observation Tool (CPOT)**:
 - 4 domains: facial expression, body movement, muscle tension, compliance with ventilation
 - Each 0–2 → total **0 to 8**
- **Richmond Agitation–Sedation Scale (RASS)**: assesses sedation and agitation (POSS assesses opioid-induced sedation); used in parallel — know whether the goal is analgesia or sedation. [I&R ch. 3]
- Self-report (NRS 0–10 preferred; VAS 10-cm line) is used whenever the patient can communicate.

Five-Point Global Scale	None A little = 1 Some = 2 A lot = 3 The worst = 4
Verbal Quantitative Scale	0.....5.....10 None Worst imaginable
Visual Pain Analog Scale	No Worst Pain Pain Place a mark on the line

Irwin and Rippe 9e, Figure 3.1 (ch. 3): self-report pain scales — five-point global, verbal quantitative (0–10) and visual analog scales.

Bispectral index (BIS)

- **Principle**: single forehead sensor with multiple EEG electrodes; processed signal gives a **0–100 integer** corresponding to level of consciousness.
- Uses: depth of anaesthesia, sedation during **neuromuscular blockade** (with Patient State Index), sleep depth; adjunct to clinical examination when titrating propofol to unconsciousness.
- Limits: overlap of cut-offs between sleep stages and sedated states; electrode detachment and movement artefact; reliability, cost-effectiveness and outcome benefit in ICU unproven.

Management (titration strategy)

MANAGEMENT BASIS

SCCM PADIS guideline (Crit Care Med 2018) and its 2025 focused update (Crit Care Med 2025). No ISCCM sedation guideline was found. Doses are from Irwin and Rippe 9e (PADIS statements read give no doses except ketamine).

Newer: PADIS 2025 — dexmedetomidine suggested over propofol when light sedation and/or reducing delirium are the highest priorities (conditional, moderate certainty).

Table 9. Key PADIS recommendations

Recommendation	Strength; evidence	Source
Assessment-driven, protocol-based, stepwise pain and sedation management	Conditional; moderate	PADIS 2018
Treat pain before a sedative is considered (analgesia-first)	Good practice statement	PADIS 2018
BPS and CPOT have the greatest validity for pain in patients unable to self-report; vital signs are not valid pain indicators	Ungraded statement	PADIS 2018
Light (vs deep) sedation in ventilated adults	Conditional; low	PADIS 2018
Propofol or dexmedetomidine over benzodiazepines	Conditional; low	PADIS 2018
Dexmedetomidine over propofol when light sedation/less delirium are priorities	Conditional; moderate certainty	PADIS 2025
Acetaminophen as opioid adjunct	Conditional; very low	PADIS 2018
Low-dose ketamine 0.5 mg/kg IV push then 1–2 µg/kg/min as adjunct	Conditional; very low	PADIS 2018
BIS best suited for titration during deep sedation	Ungraded statement	PADIS 2018

- Daily sedation interruption or nurse-protocolised targeted sedation can achieve light sedation; arousal for DSI defined as **RASS –1 to +1**. [PADIS 2018]
- Opioids at the lowest effective dose for procedural pain. [PADIS 2018; conditional, moderate]
- Neuropathic pain: gabapentin, carbamazepine or pregabalin with opioids. [PADIS 2018; strong, moderate]
- Regional analgesia reduces opioid needs when feasible; give a bowel regimen to all on opioids. [I&R]
- Neuromuscular blockers have no sedative or analgesic action — never use them to treat movement from inadequate analgesia. [I&R]

PEARLS — what wins marks

- Analgesia first, then light sedation (RASS –1 to +1 on DSI) — PADIS.
- BPS 3–12 (3 domains); CPOT 0–8 (4 domains); self-report NRS when possible.
- Propofol or dexmedetomidine over benzodiazepines; dex preferred if light sedation/delirium priority (2025).
- Remifentanyl CSHT ≈4 min; midazolam accumulates; benzodiazepines → most delirium.
- BIS = processed EEG 0–100; useful under paralysis/deep sedation, not routine.
- Trap: propofol and midazolam give no analgesia; NMBAs give neither.

Textbook: Irwin and Rippe 9e, ch. 2 "Anesthesia for Bedside Procedures": §Pharmacokinetic Considerations (Fig. 2.1), §Hypnotics (Tables 2.2–2.3; Propofol, Etomidate, Ketamine, Midazolam, Dexmedetomidine), §Opioids, §Practical Considerations. Ch. 3 "Management of Pain in the Critically Ill": §Evaluation of Pain and Sedation (Fig. 3.1), §Subjective and Objective Pain Assessment, §Medical Management, §Pharmacologic Treatments (Table 3.1). Ch. 4 "Therapeutic Paralysis", §Monitoring of NMBAs. Ch. 8, Table 8.4. Ch. 183 "Sleep Issues in the ICU", §Bispectral Index.

Guidelines: SCCM, Devlin JW et al., "Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU", Crit Care Med 2018;46(9):e825–e873 (SCCM guideline page). doi:10.1097/CCM.0000000000003299. SCCM, Lewis K et al., "A focused update to the clinical practice guidelines for ... PADIS", Crit Care Med 2025;53(3):e711–e727. doi:10.1097/CCM.0000000000006574.

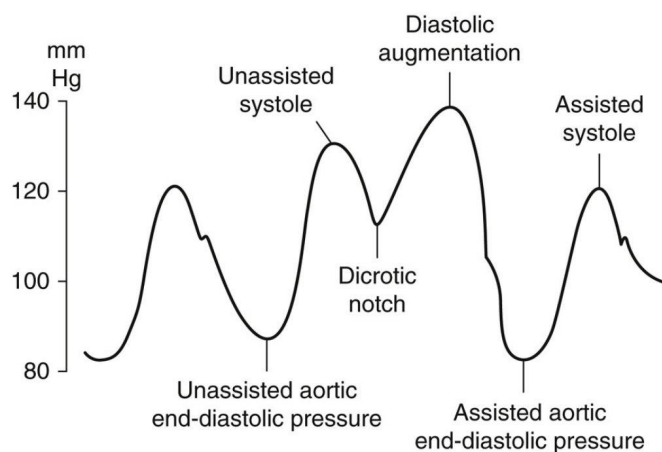
Paper I • December 2022 • Q7(b) • 5 marks • Intermediate

Explain arterial waveform with diagram. [5]**Principle**

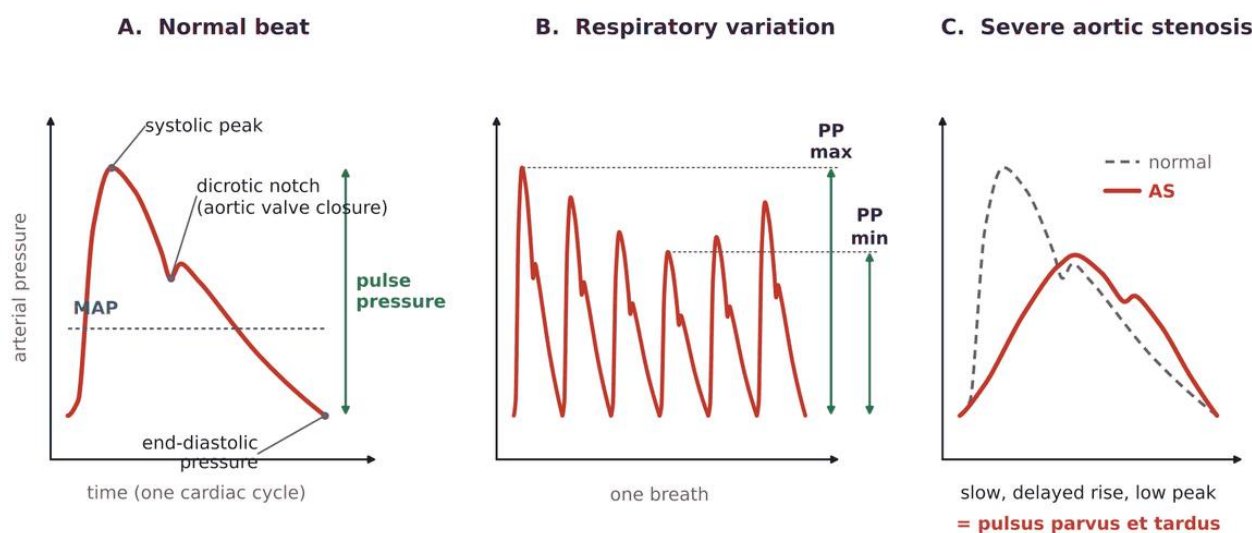
- Intra-arterial pressure is transmitted through a **fluid-filled, non-compliant tubing** to a transducer that converts mechanical displacement into an electrical signal, which is amplified and displayed beat-to-beat.
- System components:
 - Intra-arterial catheter (20-G polyurethane reduces tip thrombosis)
 - Short, stiff tubing with stopcocks
 - Transducer (Wheatstone bridge)
 - Continuous flush device with bag pressurised to **300 mm Hg**
 - Monitor (processor, amplifier, display)
- Arterial lines measure the **end-on pressure** of the arterial pulse; non-invasive methods report the external pressure needed to occlude flow — oscillometric readings may differ by ≥ 10 mm Hg in the critically ill.

The waveform and what it shows

- Each beat shows a **systolic peak**, a **dicrotic notch** marking aortic valve closure, and the **end-diastolic pressure** (Fig. 192.4).
- **MAP** is the value used for decision-making in most critically ill patients.
- Waveform inspection can diagnose:
 - ECG lead disconnection
 - Aortic valve disease — e.g. slow, delayed upstroke (pulsus parvus et tardus) in severe AS
 - Effect of arrhythmias/ectopics on perfusion
 - Respiratory swings — **pulsus paradoxus**
 - Fluid responsiveness (SPV, PPV) in ventilated patients



Irwin and Rippe 9e, Figure 192.4 (ch. 192): aortic pressure tracing with intra-aortic balloon counterpulsation (1:2) — unassisted systole, dicrotic notch (aortic valve closure), diastolic augmentation, and unassisted vs assisted end-diastolic pressures.

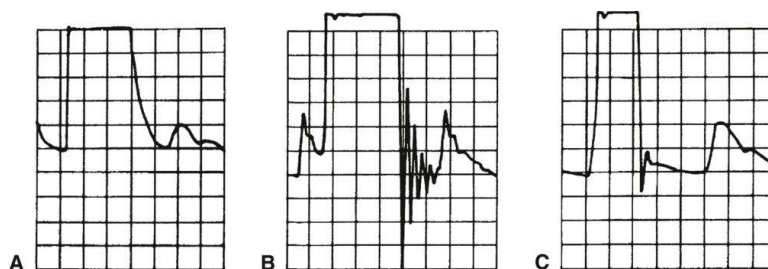


Pulse pressure variation = (PP max – PP min) ÷ mean of the two; valid in passively ventilated patients

Schematic (drawn for these notes) — arterial pressure waveform — A, one beat (systolic peak, dicrotic notch at aortic valve closure, end-diastolic pressure, MAP, pulse pressure); B, respiratory variation of pulse pressure in a passively ventilated patient (PP max, PP min); C, severe aortic stenosis (slow, delayed upstroke — **pulsus parvus et tardus**). Facts per Irwin and Rippe 9e, Ch 7 Arterial Line Placement and Care; Ch 28 (PPV); Ch 193 Valvular Heart Disease, §Aortic Stenosis. Schematic, axes unnumbered.

Dynamic response: natural frequency and damping

- The catheter–tubing–transducer is an **underdamped second-order system**; fidelity depends on **natural frequency** and **damping coefficient**.
- Arterial waveforms have a fundamental frequency of **3–5 Hz**; system natural frequency should ideally be **>20 Hz** ($\geq 5 \times$ highest input frequency) to avoid resonance and systolic overshoot.
- Natural frequency < 7.5 Hz distorts the trace whatever the damping; at 24 Hz a damping coefficient of 0.15–1.1 is acceptable; peripheral monitoring typically 10–20 Hz with damping 0.5–0.7.
- Natural frequency rises with shorter, stiffer, wider, less dense tubing; damping rises with longer, narrower tubing, more viscous fluid, air and clot.
- Fast-flush (square-wave) test**: briefly open the flush (300 mm Hg) → square wave then oscillations; optimal = **one undershoot, a small overshoot, then the patient's waveform**. Repeat at least every 8 h, after any opening of the system, with haemodynamic change, or when the trace looks damped.



Irwin and Rippe 9e, Figure 7.1 (ch. 7): fast-flush test — A, overdamped; B, underdamped; C, optimal damping.

Table 10. Damping errors (Irwin and Rippe 9e, ch. 7, 27)

Feature	Overdamped	Underdamped	Optimal
Fast-flush response	Slow return, no oscillation	Multiple oscillations	One undershoot, small overshoot
Effect on reading	Blunted trace, underestimates systolic	Resonance, systolic overshoot	True pressures
Causes	Large air bubbles, kinks, clot, loose connection, deflated pressure bag, vasoconstriction	Long tubing (commonest), small air bubbles, large/long cannula, hypertension, COPD; less with deeper sedation	—
Fix	Straighten, flush distal stopcock, replace tubing — avoid flushing clot/air proximally	Shorten tubing, 20-G short cannula, remove bubbles	—

Zeroing, levelling and calibration

- **Zero:** open transducer stopcock to air (atmospheric reference). **Level:** transducer at the heart — **4th–5th intercostal space, mid-axillary line**.
- Improper zeroing/levelling is the **single most important source of error**; transducer below the reference reads falsely high, above reads low.
- Re-zero with every position change, major BP change and every 6–8 h (zero drift); calibration check with a manometer at 100/150/200 mm Hg — ± 5 mm Hg acceptable.
- Radial and femoral MAP are interchangeable in most patients, but radial may under-read in vasopressor-dependent sepsis.

Respiratory variation: SPV, PPV, SVV

WORKED EXAMPLE — Pulse pressure variation (definition from Irwin and Rippe 9e, ch. 28)

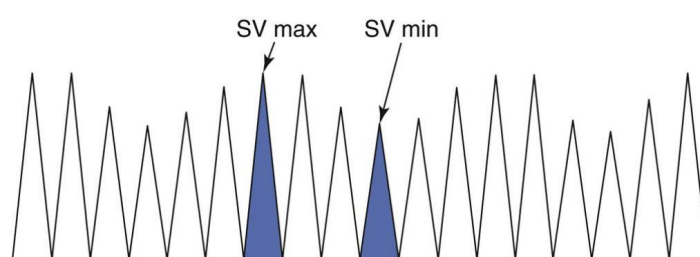
Sedated, volume-controlled patient in sinus rhythm. Over one breath: maximum pulse pressure 52 mm Hg (130/78), minimum pulse pressure 40 mm Hg (110/70).

Step 1 — $PPV = (PP_{\max} - PP_{\min}) \div \text{mean of the two} = (52 - 40) \div 46$.

Step 2 — $PPV = 12 \div 46 = 0.26 \rightarrow 26\%$.

Step 3 — $\geq 13\%$ predicts response with $\sim 90\%$ accuracy; $\leq 9\%$ predicts non-response; 9–13% is the "grey zone" (Cannesson). Meta-analysis thresholds: PPV 12.5%, SVV 11.6% (moderate accuracy).

Step 4 — valid only in passively ventilated patients, usually V_T 8–10 mL/kg, regular rhythm.



Irwin and Rippe 9e, Figure 28.2 (ch. 28): stroke volume variation from pulse contour analysis — SV max and SV min over the respiratory cycle.

- Unreliable with **low tidal volume (<6 mL/kg)**, spontaneous breathing/pressure support, patient movement, and arrhythmias (AF gives large PPV independent of volume).
- Pulse contour analysis ($SV = \text{area under curve} \div \text{impedance}$) gives continuous CO; calibrated (transpulmonary thermodilution, PiCCO) or autocalibrated (FloTrac) — inaccurate in vasodilated states.

Clinical use

MANAGEMENT BASIS

Surviving Sepsis Campaign 2026 (Crit Care Med 2026) for monitoring and targets; technical lines [I&R] from Irwin and Rippe 9e, ch. 7, 27, 28.

- Septic shock: **either invasive or non-invasive** BP monitoring. [SSC 2026; conditional, very low]
- Initial **MAP target 65 mm Hg** [SSC 2026; strong, moderate]; age ≥65 years: MAP 60–65 mm Hg. [SSC 2026; conditional, low]
- Use **dynamic measures** (e.g. PPV, SVV) to guide fluids over static measures. [SSC 2026; conditional, low]
- Insert arterial lines only when needed and remove promptly; one justified when ≥2 blood gases a day are needed. [I&R]
- Arterial catheters colonise at rates similar to CVCs — treat like central lines; femoral CRBSI 1.92 vs radial 0.25 per 1000 catheter-days. [I&R]

PEARLS — what wins marks

- Trace = systolic peak, **dicrotic notch (aortic valve closure)**, end-diastolic pressure; decide on MAP.
- Fast-flush: optimal = one undershoot + small overshoot; test every 8 h.
- Underdamped (long tubing) → systolic overshoot; overdamped (air, clot, kink) → blunted trace.
- Zero to air, level at 4th–5th ICS mid-axillary line — commonest error source.
- PPV = (PPmax – PPmin)/mean; ≥13% responsive, ≤9% not (grey zone 9–13%).
- Trap: PPV invalid with $V_T < 6$ mL/kg, spontaneous breaths or AF.

Textbook: Irwin and Rippe 9e, ch. 7 "Arterial Line Placement and Care": §Indications (Table 7.1), §Equipment, Monitoring, Techniques, and Sources of Error (natural frequency, damping, fast-flush test, zeroing and levelling), Fig. 7.1; §Infections. Ch. 27 "Routine Monitoring of Critically Ill Patients", §Direct Invasive Blood Pressure Measurement, §Site Selection, §Conclusions. Ch. 28 "Minimally Invasive Hemodynamic Monitoring", §Pulse Contour Analysis (Background, Clinical Utility, Table 28.2), Fig. 28.2. Ch. 192 "Mechanical Circulatory Support", Fig. 192.4. Ch. 193 "Valvular Heart Disease", §Aortic Stenosis — Physical Examination.

Guidelines: Surviving Sepsis Campaign, Prescott H et al., "International guidelines for management of sepsis and septic shock 2026", Crit Care Med 2026, haemodynamic statements (SCCM guideline page). doi:10.1097/CCM.0000000000007075.

Paper I · October 2023 · Q9 · 10 marks · Long

Describe about physiological difficult airway in critically ill patients. [10]

Also asked as: FNB · June 2019 · Q7(b) "Difficult airway. [5+5]"

1. Definition

- Every critically ill patient needing airway management is evaluated for the ability to **mask ventilate, intubate, place a supraglottic airway and perform a surgical airway**.
- A difficult airway may be **recognised (anticipated)** or **unrecognised** at the pre-intubation evaluation.
- **Physiologically difficult airway:** the term itself is not used in Irwin and Rippe 9e; this answer groups under it the physiological states the textbook links to peri-intubation harm — **severe hypoxaemia and haemodynamic collapse** (ch. 8), **RV failure/pulmonary hypertension** (ch. 174) and **acidaemia** (ch. 121) — which make induction, apnoea and positive-pressure ventilation dangerous even with normal anatomy.

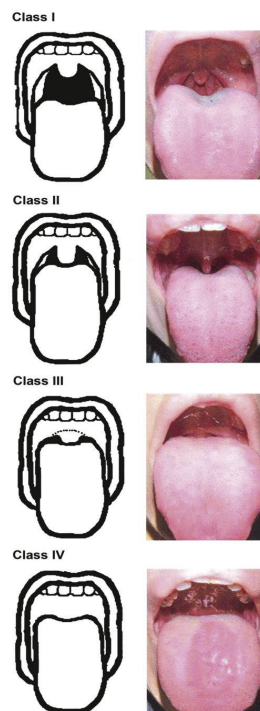
2. Why it matters in the ICU

- Emergency intubation outside the OR has complication rates of about **28%** on wards and in ICUs.
- In the ICU the most frequent complications are **severe hypoxaemia and haemodynamic collapse**; acute respiratory failure and shock are independent risk factors.
- On general wards: multiple attempts and oesophageal intubation predominate.
- Supervision by a trained attending cut immediate complications from **21.7% to 6.1%**; non-anaesthesiology trainees need about **50 supervised intubations** for 90% competence.

3. Anatomically difficult airway — prediction

Table 11. Bedside predictors (Irwin and Rippe 9e, ch. 8)

Problem	Predictors	Comment
Difficult laryngoscopy/intubation	Mallampati II–IV; mouth opening <40 mm (two fingers); thyromental distance ≤7 cm (<3 fingers); high-arched palate; neck flexion–extension ≤80°	Low positive predictive value; negative tests predict easy intubation; only ~30% assessable in emergencies
MACOCHA score (ICU)	Mallampati III/IV = 5; sleep apnoea = 2; 1 each: reduced C-spine mobility, mouth opening <3 cm, coma, SpO ₂ <80%, non-anaesthesiologist	Maximum 12; score >4 → >20% difficult intubation
Difficult mask ventilation	Neck radiation changes (most significant), male sex, sleep apnoea, Mallampati III/IV, obesity, beard; or ≥2 of: age >55, BMI >26, beard, edentulous, snoring	Influences choice of primary approach
Anatomical causes	Mandibular/maxillary hypoplasia, prominent incisors, face/neck injury, acromegaly, tumours, previous neck surgery, C-spine disease, TMJ arthritis	Consider awake technique
Ultrasound	Skin-to-epiglottis distance; locates cricothyroid membrane; gastric fluid	Adjunct when MACOCHA cannot be done



Irwin and Rippe 9e, Figure 8.7 (ch. 8): modified Mallampati classes I–IV (sitting, head extended, tongue protruded, no phonation).

	1	2		3	4
Original Cormack and Lehane system	Full view of the glottis	Partial view of the glottis or arytenoids		Only epiglottis visible	Neither glottis nor epiglottis visible
View at laryngoscopy					
Modified system	1 Same as for original Cormack and Lehane system	2a Partial view of the glottis	2b Arytenoids or posterior part of the vocal cords only just visible	3 Same as for original Cormack and Lehane system	4 Same as for original Cormack and Lehane system

Irwin and Rippe 9e, Figure 8.11 (ch. 8): modified Cormack and Lehane grades of laryngeal view — 1, 2a, 2b, 3, 4.

4. The physiologically difficult airway — components and mechanisms

- **Hypoxaemia:**
 - Short safe apnoea time; preoxygenation (denitrogenation of FRC) less effective in acute lung injury
 - NIPPV, HFNC or both outperform standard preoxygenation for SpO₂ before, during and after intubation
 - Bag-mask ventilation between induction and intubation lowers severe hypoxaemia vs an apnoeic approach
- **Hypotension / shock:**
 - Positive-pressure ventilation lowers preload in hypovolaemia
 - Induction agents vasodilate (propofol ↓MAP); laryngoscopy may cause vasovagal bradycardia/hypotension
 - Haemodynamically unstable patients may need little or no anaesthesia
- **Right-ventricular failure / pulmonary hypertension:**
 - Very high risk of collapse in the peri-intubation period from induction vasodilation and reduced preload
 - Ventilation raises RV afterload (lung volume, PEEP) and reduces RV preload; hypercapnia raises PA pressure
 - Ventilate with low V_T, low PEEP, avoid permissive hypercapnia
- **Severe acidaemia:**
 - Salicylate poisoning: sedation/intubation can cause acidaemia → rapid salicylate tissue distribution and toxicity (ch. 121)
- **Aspiration risk / full stomach:**
 - Gastric ultrasound identifies a fluid-filled stomach; partial recumbent/reverse Trendelenburg may reduce regurgitation; cricoid pressure no longer recommended in non-pregnant patients
- **Raised ICP / coronary disease:**
 - Laryngoscopy without adequate anaesthesia → tachycardia and hypertension, harmful with high ICP or CAD

5. Preparation

- Intubation bag/cart; 100% O₂, bag-valve-mask, Yankauer suction, laryngoscopes (Macintosh, Miller), **video laryngoscope**, bougie, stylets, LMA/intubating LMA, cricothyrotomy kit, capnography.
- **ICU intubation bundle:** preoxygenation with NIPPV ± HFNC, **two operators**, RSI, capnography, lung-protective ventilation after intubation, fluid loading unless contraindicated, early sedation and vasopressors prepared.
- Difficult airway response teams — one hospital recorded no airway deaths over 5 years.

Table 12. Induction and paralytic agents — haemodynamic choice (Irwin and Rippe 9e, Table 8.4)

Drug	IV dose	Onset	Haemodynamic effect / caution
Etomidate	0.2–0.3 mg/kg	20–50 s	Minimal CV effect — useful in hypovolaemia, low EF, AS; adrenal suppression debated
Ketamine	0.5–2.0 mg/kg	30–60 s	↑HR, ↑MAP (catecholamine release); hypotension if catecholamine-depleted; bronchodilator
Propofol	1.0–2.5 mg/kg	<60 s	Myocardial depression, ↓↓MAP — avoid/reduce in shock
Midazolam	0.02–0.2 mg/kg	30–60 s	Mild ↓MAP
Fentanyl	1–2 µg/kg	60–100 s	Blunts pressor response; lowers induction dose
Succinylcholine	1–2 mg/kg	45–60 s	Hyperkalaemic arrest in burns, spinal injury, immobility — risk rises after 14 ICU days
Rocuronium	0.6–1.2 mg/kg	60–90 s	Same intubating conditions as succinylcholine at 0.9–1.2 mg/kg; reversible with sugammadex

6. Management

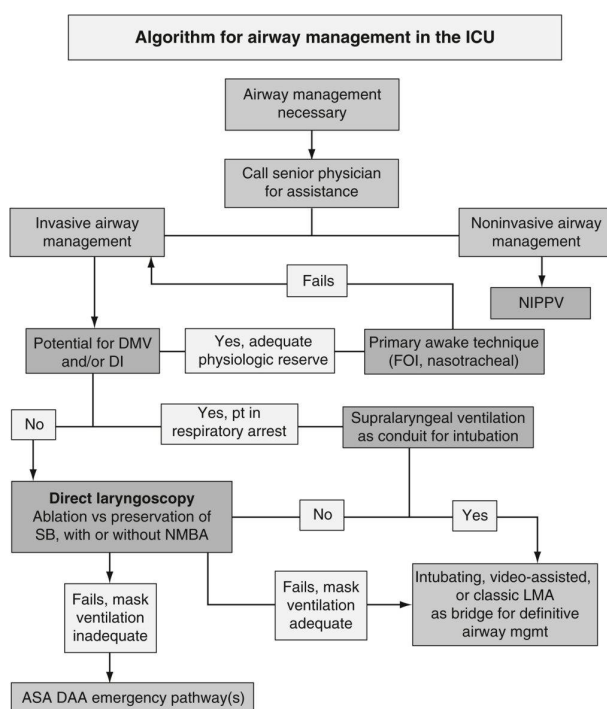
MANAGEMENT BASIS

SCCM clinical practice guidelines for rapid sequence intubation in the critically ill adult (Crit Care Med 2023). No ISCCM airway document was found. The AIDAA 2016 ICU intubation guideline (Indian J Anaesth), DAS 2018 critically-ill intubation guideline (BJA), ASA 2022 difficult-airway guideline and the 2024 international Delphi consensus on the physiologically difficult airway (Intensive Care Med) were identified but could not be read from an official source in this build and are not cited. Algorithm and techniques: Irwin and Rippe 9e, ch. 8 [I&R].

Table 13. Peri-intubation recommendations (SCCM RSI 2023)

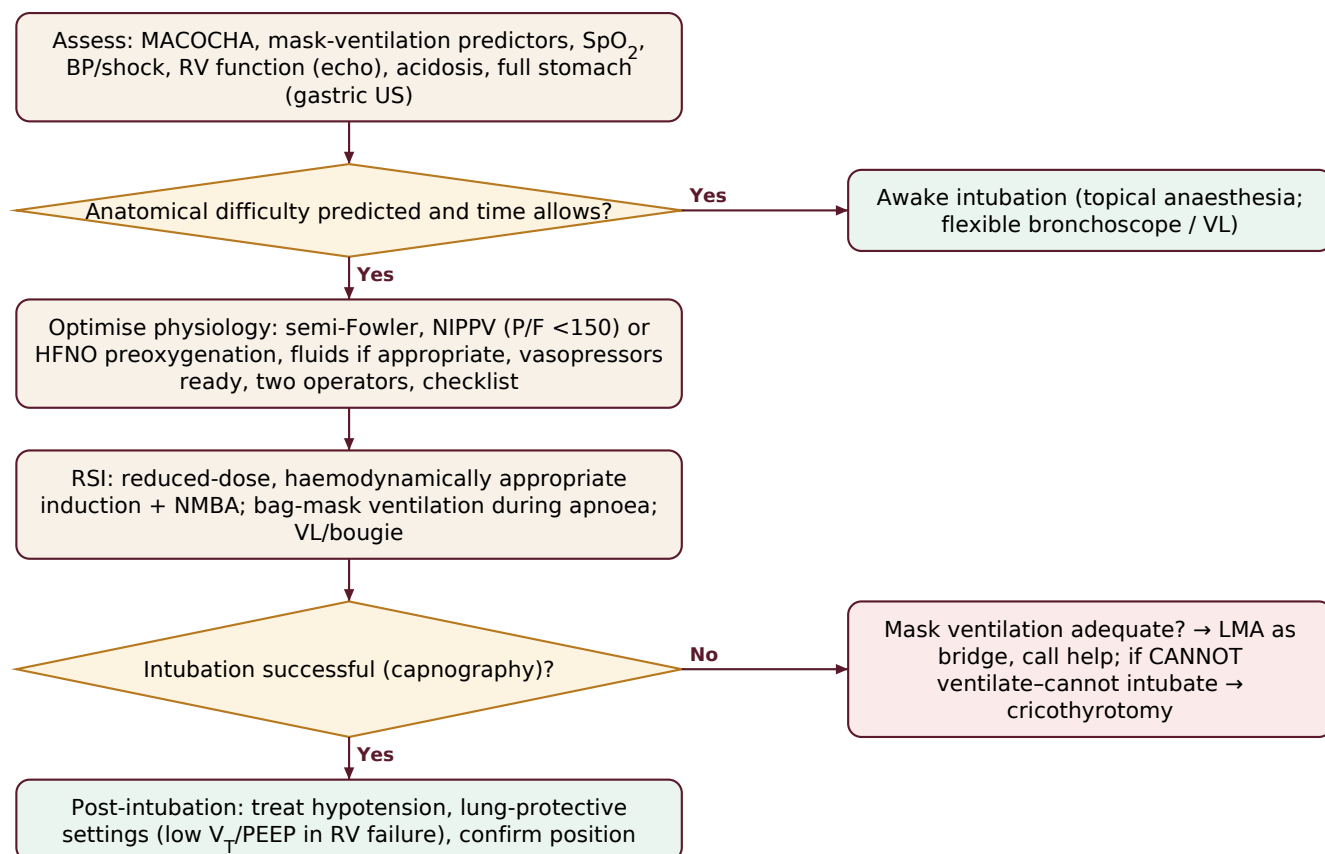
Recommendation	Strength; certainty
Head-and-torso-inclined (semi-Fowler) position during RSI	Conditional; very low
Preoxygenate with HFNO when laryngoscopy is expected to be challenging	Conditional; low
Preoxygenate with NIPPV in severe hypoxaemia ($\text{PaO}_2/\text{FiO}_2 < 150$)	Conditional; low
Medication-assisted preoxygenation to improve preoxygenation	Conditional; very low
NG decompression when benefit outweighs risk	Best practice statement
Peri-intubation vasopressors	Insufficient evidence — no recommendation
Give a sedative-hypnotic when an NMBA is used	Best practice statement
No difference between etomidate and other induction agents	Conditional; moderate
No routine corticosteroids after etomidate	Conditional; low
Give an NMBA when an induction agent is used	Strong; low
Either rocuronium or succinylcholine when no contraindication to succinylcholine	Conditional; low

- **Resuscitate before you intubate** (physiological optimisation): preoxygenate with NIPPV/HFNC, volume-load if appropriate, vasopressors drawn up and ready; reduce induction doses; coadministered opioids lower the induction dose. [I&R]
- Preoxygenation: 100% O₂ at high flow via tight mask for **3½–4 min**; extending to 8 min gives no clinically relevant gain. [I&R]
- Hypotension after intubation: pre-intubation volume loading and immediately available vasopressors; bradycardia responds to **atropine 1 mg IV**. [I&R]
- Anticipated anatomical difficulty → **awake tracheal intubation** after topical anaesthesia (DL, VL, LMA, flexible bronchoscopic oral/nasal, retrograde, lighted stylet or surgical airway). [I&R]
- Topicalisation: lidocaine 4% aerosol 6 mL; transtracheal lidocaine 4% 160 mg; superior laryngeal block 2 mL of 1–1.5% lidocaine below greater cornu of hyoid; watch for lidocaine toxicity. [I&R]
- Poor view: hockey-stick stylet, **BURP**, light wand, **gum elastic bougie**, video laryngoscope (insert tube from the right at 90° if difficult). [I&R]
- Unrecognised difficult airway with adequate mask ventilation → call for experienced help and follow the algorithm. [I&R]
- **Cannot ventilate—cannot intubate** → immediate rescue: **LMA/supraglottic device** or **emergency cricothyrotomy** (needle cricothyrotomy with 14-G catheter and jet ventilation, or scalpel). Ultrasound helps locate the cricothyroid membrane. [I&R]
- Suspected C-spine injury: in-line stabilisation; VL improves view but not first-attempt success (except Airtraq); flexible bronchoscopy or retrograde intubation for maxillofacial trauma. [I&R]
- Confirm tube with **capnography** (or colorimetric detector, oesophageal detector bulb, ultrasound lung sliding); bronchoscopy is the only certain method; tip several cm above carina (T4); 21 cm (women)/23 cm (men) at incisors. [I&R]



Irwin and Rippe 9e, Figure 8.12 (ch. 8): algorithm for airway management in the ICU (modification of the ASA difficult airway algorithm) — non-invasive vs invasive airway, primary awake technique, supralaryngeal ventilation as conduit, emergency pathway.

Flowchart. Stepwise approach — physiologically and anatomically difficult airway



Based on SCCM RSI guideline 2023 and Irwin and Rippe 9e, ch. 8 Management of the Difficult Airway, Fig. 8.12.

7. Complications

- During intubation: hypoxaemia, hypotension, hypertension, arrhythmias (ventricular in 5–10%), bradycardia, aspiration, dental/airway trauma, oesophageal intubation, delayed CPR.

- Tube in place: obstruction, kinking, dislodgement, endobronchial migration; cuff pressure $> \sim 32$ mm Hg (capillary pressure) → ischaemia, erosion into innominate artery or oesophagus.
- After extubation: laryngospasm, oedema/stridor (cuff-leak test specific but moderately sensitive; pre-extubation steroids reduce stridor), vocal cord palsy, tracheal stenosis. Difficult airway — extubate over an **airway exchange catheter**.

8. Recent advances (from the sources)

- Video laryngoscopy, ultrasound for airway assessment (skin-to-epiglottis distance), gastric US in the safety checklist. [I&R]
- Sugammadex reversal makes rocuronium more attractive than succinylcholine. [I&R]
- SCCM 2023: NIPPV for P/F < 150 , HFNO for expected difficult laryngoscopy, semi-Fowler positioning. [SCCM RSI 2023]

PEARLS — what wins marks

- Evaluate four things: **mask ventilation, intubation, SGA, surgical airway**.
- MACOCHA > 4 → $> 20\%$ difficult intubation (Mallampati III/IV = 5 points).
- Physiologically difficult airway = **hypoxaemia, hypotension, RV failure, acidaemia** — resuscitate before intubating.
- Preoxygenate: NIPPV if P/F < 150 , HFNO if difficult laryngoscopy (SCCM 2023, conditional, low).
- Always give an NMBA with the induction agent (SCCM 2023, strong, low); rocuronium 0.9–1.2 mg/kg $\approx$ succinylcholine.
- CICV → LMA or cricothyrotomy immediately.
- Trap: succinylcholine → hyperkalaemic arrest after burns, spinal injury, prolonged immobility (> 14 ICU days).

Textbook: Irwin and Rippe 9e, ch. 8 "Airway Management and Endotracheal Intubation": §Key Points, §Preintubation Evaluation (MACOCHA, Fig. 8.7), §Education and Intubation Management (bundle), §Equipment, §Anesthesia Before Intubation (Table 8.4), §Techniques of Intubation (preoxygenation, Fig. 8.11), §Management of the Difficult Airway (Fig. 8.12), §Cricothyrotomy, §Cervical Spine Injury, §Complications (Table 8.5), §Extubation, §Utility of Ultrasonography. Ch. 121 "Salicylate Poisoning", Key Points. Ch. 174 "Pulmonary Hypertension in the ICU", §Mechanical Ventilation.

Guidelines: SCCM, Acquisto NM, Mosier JM, Bittner EA et al., "Society of Critical Care Medicine clinical practice guidelines for rapid sequence intubation in the critically ill adult patient", Crit Care Med 2023;51(10):1411-1430 (SCCM guideline page).

Paper III • May 2024 • Q9 • 10 marks • Long

Discuss ultrasound-guided percutaneous tracheostomy (PCT). What are the advantages and disadvantages of bronchoscopic guided PCT? [6+(2+2)]

Also asked as: FNB • June 2019 • Q6(a) "Percutaneous dilatational tracheostomy." • Paper I • June 2022 • Q1(a) "Describe about percutaneous tracheostomy: Applied anatomy including arterial and venous supply. [4]" • Paper I • June 2022 • Q1(b) "Describe about percutaneous tracheostomy: Enumerate various methods. [3]" • Paper I • June 2022 • Q1(c) "Describe about percutaneous tracheostomy: Complications. [3]"

1. Definition and indications

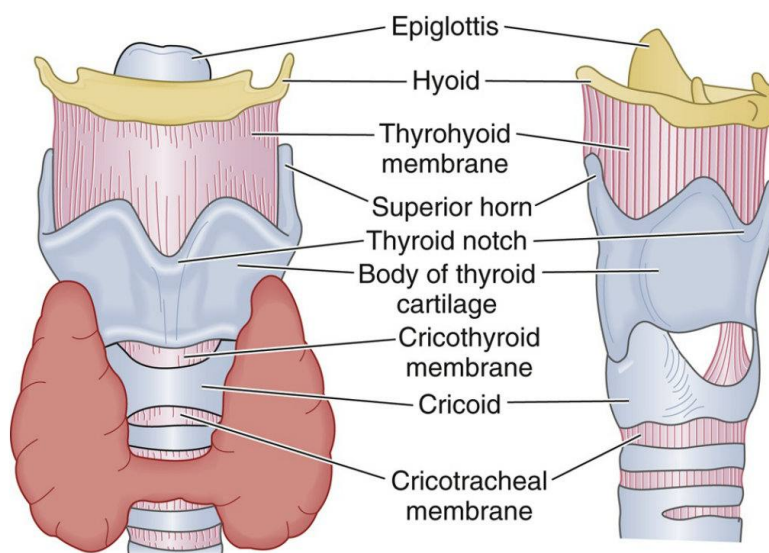
- **Percutaneous dilatational tracheostomy (PDT):** bedside creation of a tracheal stoma by **guidewire placement through the anterior tracheal wall followed by dilatation over the wire** — first described by Ciaglia (1985).
- Usually made between the **cricoid and first ring or first and second rings**; open surgical tracheostomy (OST) between the **second and third rings**.
- Indications (three categories):
 - Bypass **upper airway obstruction**
 - Access for **removal of secretions**
 - **Prolonged ventilatory support** — the commonest ICU indication
- Prevalence in ICU patients: **8% to $> 30\%$** ; tracheostomy is the airway of choice beyond 10–14 days of ventilation.

Table 14. Translaryngeal tube vs tracheostomy for prolonged ventilation (Irwin and Rippe 9e, Table 9.2)

	Advantages	Disadvantages
Translaryngeal tube	Reliable in emergencies; no surgical complications; lower initial cost	Airway colonisation, unplanned extubation, laryngeal injury, tracheal stenosis, sinusitis (nasal), discomfort
Tracheostomy	Avoids laryngeal injury, easier nursing, mobility, secure airway, comfort, speech, oral hygiene, faster weaning, less nosocomial pneumonia	Procedural complications (Table 9.3), colonisation, cost, scar, tracheal/stomal stenosis

2. Applied anatomy (J22-1-1a)

- **Larynx:** thyroid and cricoid cartilages are palpable; the **cricothyroid ligament/membrane** joins them; the **cricoid is the only complete ring** and is attached to the first tracheal ring by the cricotracheal ligament.
- **Cricothyroid space:** only 7–9 mm high, smaller than most tracheostomy tubes (OD 10 mm); lies 2–3 cm below the laryngeal prominence.
- **Trachea:** about **15 cm** long, C-shaped cartilages; oesophagus behind; **thyroid gland in front of the first few rings**; carina at T4.
- **Recurrent laryngeal nerve** supplies all intrinsic laryngeal muscles except cricothyroid — at risk with misplaced tubes.
- The **pleural cupola** extends into the neck (more in emphysema and children) → pneumothorax risk.
- Ultrasound: tracheal rings look like a "**string of black pearls**"; skin-to-trachea distance and midline are measured.



Irwin and Rippe 9e, Figure 8.1 (ch. 8): anterior and lateral anatomy of the larynx — hyoid, thyrohyoid membrane, thyroid cartilage, cricothyroid membrane, cricoid, cricotracheal membrane and thyroid gland over the upper trachea.

SOURCE NOTE — blood supply

Irwin and Rippe 9e describes the vessels that matter for tracheostomy (below) but not the named arterial and venous supply of the trachea itself; searched ch. 8, 9, 182 and full text for "blood supply", "arterial supply", "inferior thyroid artery", "bronchial artery" with the trachea — not present. No registered document covers it, so it is not stated.

- **Arteries at risk:**
 - **Innominate (brachiocephalic) artery** — rises to about the **6th ring** in front of the trachea; anomaly in 1–2%; a **high-riding innominate artery** needs open tracheostomy
 - Aberrant **carotid, brachiocephalic, right subclavian or thyroid artery, aortic arch** — severe PDT haemorrhage
 - **Cricothyroid artery** crosses the upper part of the cricothyroid membrane in the midline
 - Late bleeding sites: common carotid, superior and inferior thyroid arteries, aortic arch
- **Veins at risk:**
 - **Anterior jugular veins** near the field
 - **Transverse jugular vein** crossing the midline just above the jugular notch
 - Large midline veins (may contraindicate PDT); innominate vein (delayed haemorrhage)

- **Thyroid isthmus:** commonest source of major procedural bleeding; divided/ligated in OST.

3. Methods (J22-1-1b)

Table 15. Methods of tracheostomy (Irwin and Rippe 9e, ch. 9, 182; SRLF/SFAR 2018 rationale)

Method	Essence	Comment
Open surgical (OST)	Horizontal/vertical incision, strap muscles separated, isthmus mobilised or divided, window or Bjork flap at rings 2–3	OR or ICU; preferred in high-risk anatomy/physiology
PDT — multiple dilator	Original Ciaglia technique	—
PDT — single dilator	Guidewire + dilatation (all PDT variants)	Lower failure rate than rotating dilation; more minor bleeding and tracheal ring fractures
PDT — guidewire dilating forceps	Guidewire + dilatation	Single dilator has fewer minor complications than forceps or balloon (SRLF)
PDT — rotating dilation	Guidewire + dilatation	Higher failure rate than single dilator
PDT — balloon dilation	Guidewire + dilatation	—
Translaryngeal tracheotomy	—	Higher failure and complication rate
Cricothyrotomy	Incision through cricothyroid membrane	Emergency airway; replace by tracheostomy within 48–72 h; subglottic stenosis 2–3%

- OST vs PDT meta-analyses: **no difference in mortality or major complications**; PDT faster, fewer stomal infections ($\pm$ less bleeding); procedure-related mortality <1% with both.
- PDT advantages: easier timing, no OR or transport, cheaper, better cosmesis, tight stoma with less infection and bleeding.
- **Prefer OST** when: $\text{FiO}_2 > 0.60$ or PEEP >10, complicated intubation, non-palpable cricoid or cricoid <3 cm above the sternal notch; obesity with thick pretracheal fat; large goitre; abnormal airway; need for a second physician to manage ventilation/circulation; uncorrectable bleeding diathesis.

4. Timing and contraindications

MANAGEMENT BASIS

French expert-panel guidelines on tracheotomy in the ICU (SRLF/SFAR, Anaesth Crit Care Pain Med 2018; GRADE). No ISCCM tracheostomy guideline was found. Lines tagged [I&R] from Irwin and Rippe 9e, ch. 9, 10, 182.

Table 16. Examinable difference on timing

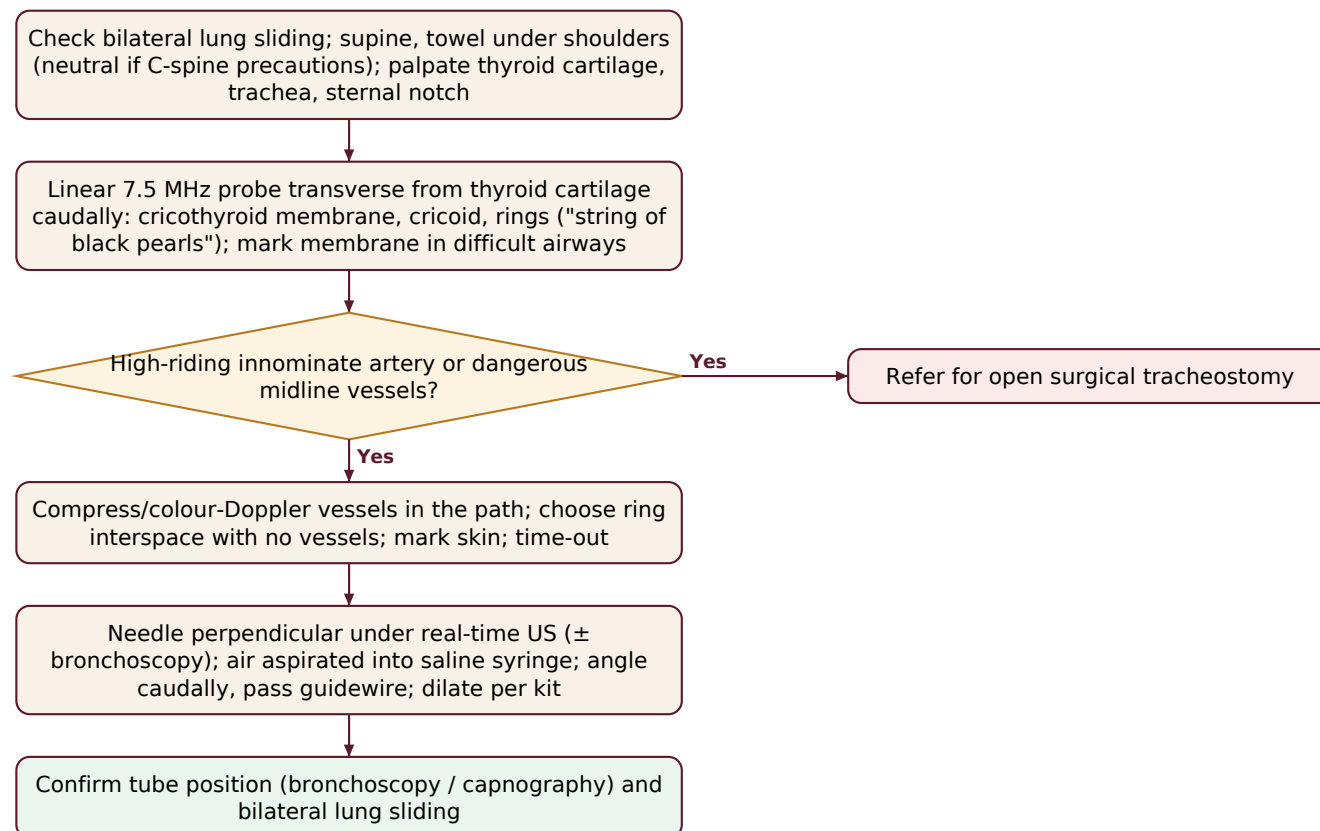
Point	Irwin and Rippe 9e (ch. 9)	SRLF/SFAR 2018
Earliest timing	Benefits of "early" tracheostomy insufficient to recommend it before 7–10 days of intubation; earlier (within 1 week) in multiple trauma, head injury with poor GCS, spinal trauma, burns	Should not be performed before the fourth day of mechanical ventilation (R1.3, GRADE 1+)
Contra-indications	No absolute; relative — uncorrected coagulopathy, high PEEP, abnormal upper-airway anatomy, BMI >30	Avoid in high-risk situations (R1.4, expert opinion): haemodynamic instability, ICP >15 mm Hg, $\text{PaO}_2/\text{FiO}_2 < 100$ with PEEP >10, platelets <50,000/mm ³ and/or INR >1.5

- Tracheotomy proposed for **prolonged weaning** after multidisciplinary discussion. [SRLF 2018, R1.1–R1.2; expert opinion]
- **Percutaneous tracheotomy is the standard method** in ICU patients. [SRLF 2018, R2.1; GRADE 1+]
- PDT should probably be preferred as the standard method. [SRLF 2018, R2.3; GRADE 2+]
- Pre-test: if not already on low PEEP, a trial of supine position with PEEP 5–7.5 cm H₂O shows whether the patient tolerates loss of positive pressure during the exchange. [I&R]
- Correct coagulopathy (including uraemia) — PDT has been done safely in refractory liver coagulopathy and on dual antiplatelets/UFH in cardiac ICU. [I&R]

5. Ultrasound-guided PCT — technique (M24-3-9)

- **Probe:** high-frequency linear (7.5 MHz) transducer, transverse then longitudinal over the anterior neck.
- **Advantages:** identifies aberrant vessels that preclude PDT, selects the ring interspace (especially in obesity or low cricoid), measures skin-to-trachea depth, finds the midline, and real-time guidance improves **first-pass success and needle accuracy**; keeps the tube below the first ring (less subglottic stenosis).
- Post-procedure ultrasound for **bilateral lung sliding** can obviate a chest radiograph for pneumothorax.
- Cervical ultrasound should probably be performed with percutaneous tracheotomy; Doppler US increases success and reduces immediate complications if the operator masters it. [SRLF 2018, R3.3; GRADE 2+]
- Disadvantages/limits: skill and training needed; does not see the posterior tracheal wall — combine with bronchoscopy to prevent posterior wall puncture and confirm tube position.

Flowchart. Ultrasound-guided percutaneous tracheostomy (sequence in Irwin and Rippe 9e, ch. 182)



Main path = "No" at the decision diamond.

6. Bronchoscopic-guided PCT — advantages and disadvantages

Table 17. Fibreoptic bronchoscopic guidance during PDT

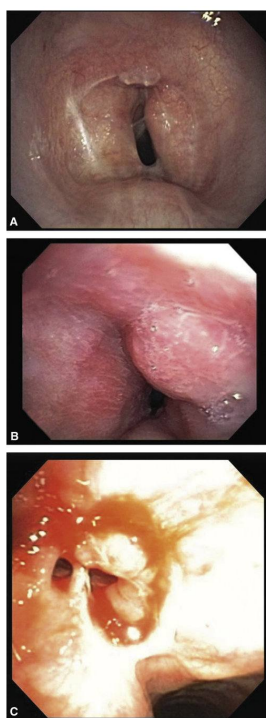
Advantages	Disadvantages
Locates the point of incision [SRLF 2018]	Bronchoscope occupies >50% of the ETT lumen → auto-PEEP approaching 20 cm H ₂ O, risk of volutrauma, hypoventilation [I&R ch. 10]
Directly visualises every stage — needle entry, wire, dilatation [SRLF 2018; I&R ch. 182]	Transient fall in PaO ₂ of about 25% in ventilated patients; FiO ₂ must be raised to 100% [I&R ch. 10]
Ensures needle placement between 1st–2nd or 2nd–3rd rings [I&R ch. 182]	ICP rises ≥50% in 88% of head-injured patients despite deep sedation/paralysis (CPP often unchanged) [I&R ch. 10]
With US, prevents posterior tracheal wall puncture; confirms tube position — no radiograph needed [I&R ch. 182]	Non-randomised series suggest (non-significantly) more complications, with methodological bias [SRLF 2018]
Single RCT: 47% (95% CI 23–64%) fewer early complications [SRLF 2018]	Expired volumes must be monitored continuously while the scope is in the tube [I&R ch. 10]

- Fiberoptic bronchoscopy should probably be performed **before and during** percutaneous tracheotomy. [SRLF 2018, R3.1; GRADE 2+]
- A laryngeal mask should probably **not** be used during PDT in ICU (more conversions and complications). [SRLF 2018, R3.2; GRADE 2-]
- No antibiotic prophylaxis. [SRLF 2018, R3.4; expert opinion]

7. Complications (J22-1-1c)

Table 18. Complications by timing (Irwin and Rippe 9e, Table 9.3 and text)

Timing	Complication	Recognition / action
Immediate (0–24 h)	Haemorrhage (minor ~12.5%, major up to 5%), tube displacement, hypoxia/hypercapnia, loss of airway, pneumothorax (0–5%), pneumomediastinum, surgical emphysema (~5%), arrhythmia, hypotension, tracheal ring fracture (9.6% of PDT), posterior wall/oesophageal injury	Pack, elevate head, haemostatics; displaced fresh stoma → oral reintubation ; US/CXR for pneumothorax
Intermediate (day 1–7)	Persistent bleeding, tube obstruction (mucus, blood), displacement, atelectasis, wound infection (<2%)	Remove inner cannula and suction; if still blocked remove outer cannula
Late (>day 7)	Tracheo-innominate fistula (<1%), tracheal stenosis (some degree 40–60%; 3–12% need intervention), tracheomalacia, tracheo-oesophageal fistula (<1%), aspiration, swallowing/speech deficits, tracheocutaneous fistula	Herald bleed → tracheoscopy; stenosis → dilatation/laser/resection; TEF → repair or stents



Irwin and Rippe 9e, Figure 182.2 (ch. 182): post-tracheostomy tracheal stenosis — A, A-shaped stenosis with fractured cartilage; B, complex stenosis with granulation above the stoma; C, simple web-like stenosis.

8. Post-procedure care

- Cuff pressure **15–20 cm H₂O**, checked daily with a manometer [I&R]; SRLF rationale: not to exceed 30 cm H₂O, checked every 8 h. [SRLF 2018, R4.1]
- Humidify inspired gas. [SRLF 2018, R4.2; expert opinion]
- **No routine tube changes** [SRLF 2018, R4.3; expert opinion]; avoid change before the tract matures (7–10 days) — if essential, change over a guide with intubation equipment ready. [I&R]
- Obturator at the bedside; inner cannula in use; dressing changes at least twice daily; no routine suctioning. [I&R]
- Deflate the cuff when breathing spontaneously [SRLF 2018, R5.2; GRADE 2+]; multidisciplinary decannulation protocol [R5.1].
- 40–65% of tracheostomised patients aspirate (73–77% silently) — no routine oral feeding in ICU. [I&R]

PEARLS — what wins marks

- PDT = wire through anterior tracheal wall + dilatation; Ciaglia (1985); site cricoid–1st or 1st–2nd ring.
- OST ≈ PDT for mortality and major complications; PDT fewer stomal infections.
- Timing: not before day 4 (SRLF 2018, GRADE 1+); textbook: no benefit before 7–10 days.
- US before/during PDT: finds aberrant vessels (high-riding innominate → OST) and ring level (R3.3, GRADE 2+).
- Bronchoscopy before and during PDT (R3.1, GRADE 2+): 47% fewer early complications vs cost of auto-PEEP, hypoxaemia, ICP rise.
- Innominate artery at ~6th ring; transverse jugular vein above jugular notch; thyroid isthmus bleeds.
- Displaced fresh tracheostomy (<7–10 days) → orotracheal intubation, not blind reinsertion.

Textbook: Irwin and Rippe 9e, ch. 9 "Tracheostomy": §Indications (Tables 9.1–9.2), §Contraindications, §Timing, §Cricothyrotomy (Anatomy, Complications), §Open Surgical Tracheostomy, §Percutaneous Dilational Techniques, §Utility of Ultrasonography, §Tubes and Cannulas, §Postoperative Care, §Complications (Table 9.3). Ch. 8 "Airway Management", §Concise Anatomy (Larynx, Trachea), Fig. 8.1. Ch. 10 "Bronchoscopy", §Percutaneous Dilational Tracheostomy, §Complications, §Contraindications, §Procedure — Mechanical Ventilation. Ch. 182 "Interventional Pulmonary in the ICU", §Percutaneous Dilational Tracheostomy, §Ultrasonography Guidance (technique steps), §Real-Time Ultrasound Guidance, Fig. 182.2.

Guidelines: Trouillet JL, Collange O, Belafia F et al. (SRLF/SFAR), "Tracheostomy in the intensive care unit: guidelines from a French expert panel", *Anaesth Crit Care Pain Med* 2018;37:281–294, Recs R1.1–R5.3 and rationale (full text, sfar.org). doi:10.1016/j.accpm.2018.02.012.

Paper I · April 2026 · Q8(b) · 5 marks · Intermediate**Tracheo-innominate fistula. [5]****Definition**

- **Tracheo-innominate artery fistula (TIF):** erosion of the tracheostomy tube **tip or cuff** through the anterior tracheal wall into the **innominate (brachiocephalic) artery**, producing often fatal haemorrhage.
- A **late** complication of tracheostomy (Table 9.3, >day 7); can also follow prolonged translaryngeal intubation when cuff ischaemia erodes anteriorly.

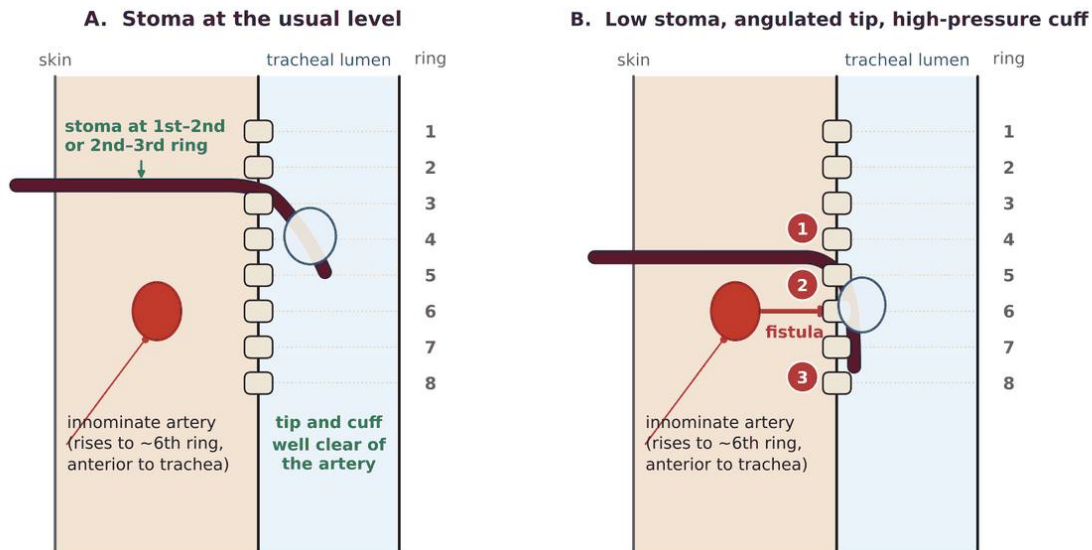
Why it matters

- Once reported to cause **50% of tracheostomy bleeding occurring >48 h** after the procedure; now <1% of cases since low-pressure cuffs.
- **85% occur within the first month**; reported up to 7 months.
- Mortality approaches **100%** without definitive treatment, even with emergency surgery.

Pathogenesis and risk factors

Table 19. Mechanisms and risk factors (Irwin and Rippe 9e, ch. 8, 9, 182)

Factor	Mechanism
Excessive cuff pressure	Cuff pressure above capillary pressure (~32 mm Hg) → mucosal ischaemia, ulceration, cartilage dissolution, erosion into the artery
Angulation of the tube tip against the anterior trachea	Pressure necrosis of the anterior wall
Low stoma (below 2nd–3rd rings)	Innominate artery rises to ~6th ring — tube tip/cuff lies close to it
Anomalous/high-riding innominate artery (1–2%)	Artery directly in front of the trachea
Infection, malnourishment, steroids	Weaken local tissues



Fistula can form at three tracheal sites: 1 stoma (pressure necrosis; stoma too low, below the 4th ring), 2 cuff (pressure necrosis from excessive inflation pressure), 3 cannula tip (excessive angulation of the cannula). Sagittal view, not to scale; tube drawn as a single line.

Schematic (drawn for these notes) — sagittal relation of the tracheostomy tube to the innominate artery — A, stoma at the 1st–2nd or 2nd–3rd ring with tip and cuff clear of the artery; B, low stoma, angulated tip and high-pressure cuff abutting the artery at ~6th ring, with the three fistula sites. Facts per Irwin and Rippe 9e, Ch 9 Tracheostomy, §Tracheoinnominate Artery Fistula; Ch 175 Managing Hemoptysis, §Pathogenesis; Ch 182 §Percutaneous Dilatational Tracheostomy. Not to scale.

Clinical features

- **Herald (sentinel) bleed:** initial haemorrhage is usually **not exsanguinating** — classically 2–4 weeks after tracheostomy.
- **Pulsation of the tracheostomy tube** signals potentially fatal positioning.
- Then sudden massive bleeding from the stoma/tube with airway flooding and shock.
- Any late post-tracheostomy bleeding needs TIF excluded — other late causes are granulation tissue or minor sources.

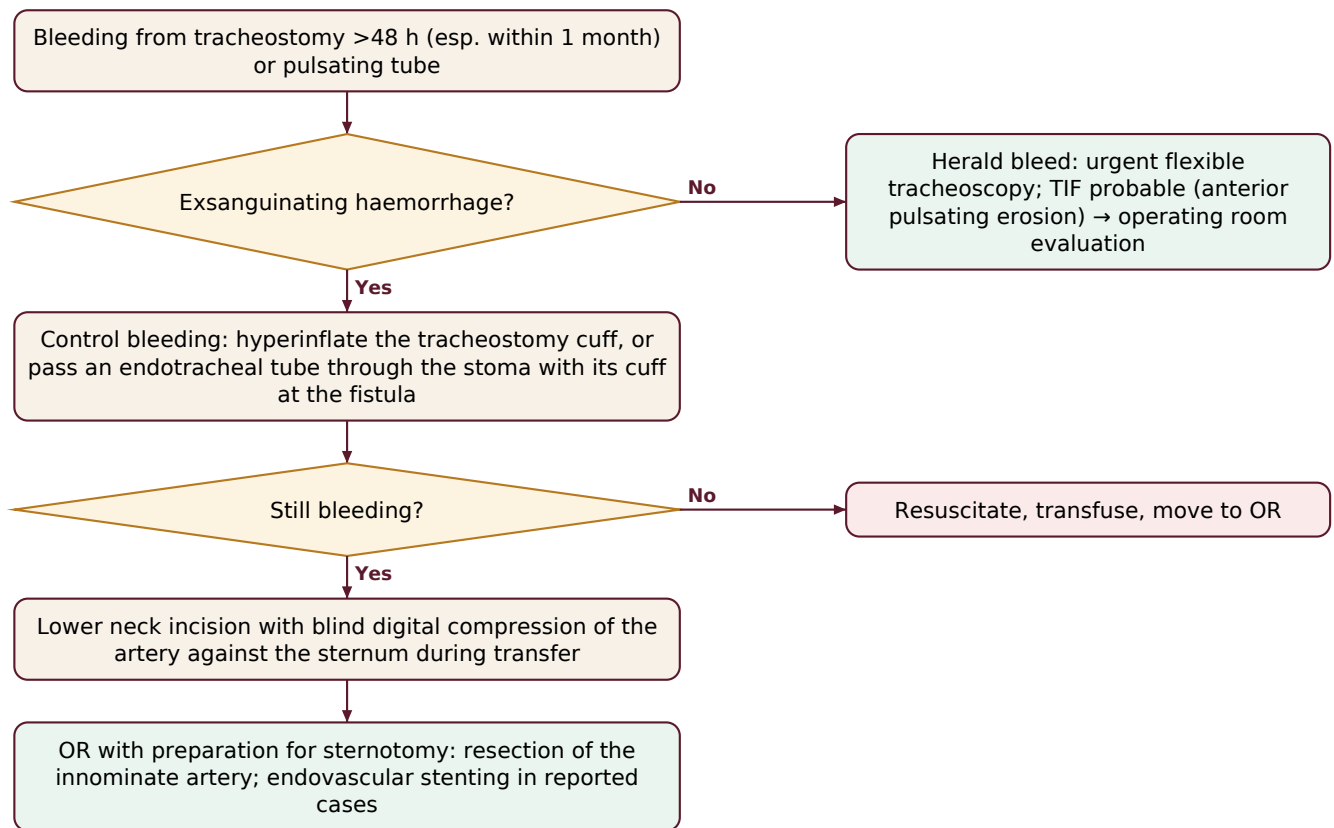
Investigations

- **Flexible tracheoscopy (bronchoscopy) promptly** for any herald bleed — findings of probable TIF: minimal tracheitis with **anterior pulsating erosion**.
- Blood in tracheal secretions after intubation suggests anterior erosion into vessels → urgent bronchoscopy examining mucosa under the cuff.
- CT angiography may be indicated depending on the tracheoscopy findings.

Management

MANAGEMENT BASIS

Irwin and Rippe 9e, ch. 9 §Tracheoinnominate Artery Fistula and ch. 182 (no society guideline addresses TIF management; none found). All lines [I&R].

Flowchart. Suspected tracheo-innominate artery fistula

Drawn from Irwin and Rippe 9e, ch. 9.

- Definitive: historically **resection of the artery**; several reported successes with **endovascular stenting**. [I&R]
- Evaluate in the operating room and prepare for possible **sternotomy**. [I&R]

Prevention

- Place the stoma between the **1st–2nd or 2nd–3rd rings**; avoid low placement. [I&R ch. 182]
- Pre-procedure neck **ultrasound** to exclude a high-riding innominate artery (→ open tracheostomy). [I&R]
- Low-pressure cuffs; cuff pressure **15–20 cm H₂O**, checked daily. [I&R]
- Correct tube length and angle; avoid neck hyperflexion and tube traction. [I&R]

PEARLS — what wins marks

- TIF = tube tip/cuff erodes anterior trachea into innominate artery; <1% now.
- 85% within the first month; classic herald bleed at 2–4 weeks.
- Pulsating tracheostomy tube = danger sign.
- Herald bleed → immediate flexible tracheoscopy → OR.
- Massive bleed: hyperinflate cuff → ETT via stoma with cuff at fistula → digital compression via lower neck incision → sternotomy/resection or stent.
- Mortality approaches 100% untreated.

Textbook: Irwin and Rippe 9e, ch. 9 "Tracheostomy": §Complications (Table 9.3), §Hemorrhage, §Tracheoinnominate Artery Fistula, §Tubes and Cannulas, MCQ 1 rationale. Ch. 8 "Airway Management", §Endotracheal Tube Cuff; §Complications While the Tube Is in Place. Ch. 182 "Interventional Pulmonary in the ICU", §Percutaneous Dilatational Tracheostomy, §Technique for Use of Ultrasound. Ch. 175 "Managing Hemoptysis", §Pathogenesis (tracheoarterial fistula).

Paper I · October 2023 · Q1 · 10 marks · Long

Describe about applied anatomy related to intercostal drain insertion and critical appraisal for drainage system based on number of chambers. [5+5]

1. Introduction

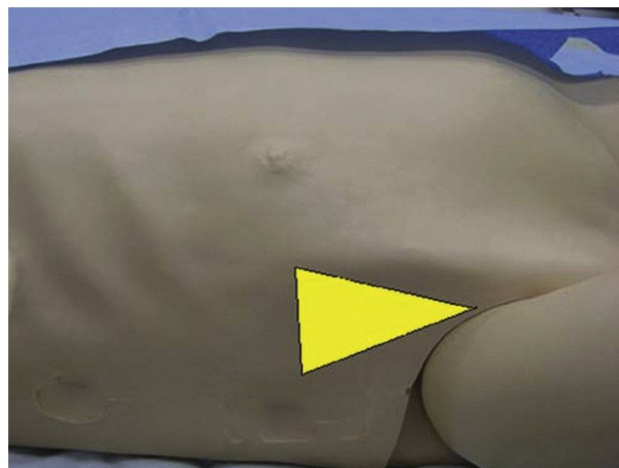
- **Tube thoracostomy:** placement of a sterile tube between the ribs into the pleural space to evacuate air or fluid into a **closed collection system**, restoring negative intrathoracic pressure and lung expansion.
- Indications (Table 13.1):
 - **Pneumothorax** (commonest) — primary, secondary, traumatic, iatrogenic, on positive-pressure ventilation
 - Haemothorax
 - Empyema
 - Chylothorax
 - Pleural effusion (transudate/exudate)
- Immediate decompression for symptomatic, large or expanding pneumothorax, or any pneumothorax on mechanical ventilation.

2. Applied anatomy and physiology of the pleura

- Pleural space: potential space with **0.1–0.2 mL/kg** lubricating fluid; lymphatics clear up to **20 mL/h per hemithorax** (70-kg adult).
- Elastic recoil gives subatmospheric pleural pressure of **–5 to –10 cm H₂O**.
- **Parietal pleura carries somatic pain fibres** — infiltrate it fully with local anaesthetic.
- The **pleural cupola** rises into the neck (higher in children and emphysema). [ch. 9]

3. Applied anatomy of the chest wall for insertion

- **Triangle of safety:** anterior border of **latissimus dorsi**, lateral border of **pectoralis major**, a line at the horizontal level of the **nipple**, apex **below the axilla**.
- Usual site: **4th or 5th intercostal space, anterior axillary line**; alternative for pneumothorax decompression: **2nd intercostal space, midclavicular line** — avoided in adults because of thick pectoral muscles.
- **Intercostal neurovascular bundle runs along the inferior border of each rib** → enter just over the **superior border of the lower rib**.
- Clamp advanced **<1 cm** beyond the pleura to avoid intrathoracic organ injury; finger sweep confirms free space and absence of symphysis.
- Recognised misplacements — **subdiaphragmatic** (liver, spleen), cardiac perforation, lung parenchyma — are avoidable by using ultrasound for all insertions.
- Needle tension decompression: 14- or 16-G catheter, 2nd intercostal space, midclavicular line.

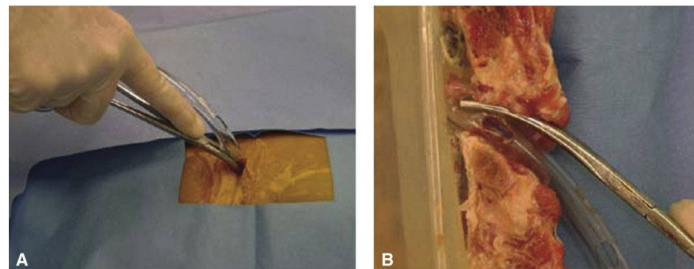


Irwin and Rippe 9e, Figure 13.5 (ch. 13): patient positioned with the arm flexed over the head; the triangle of safety is shown in yellow.

- A **posterior approach should be avoided** — the neurovascular bundle may not be covered there. [BTS 2023]
- Doppler screening of intercostal vessels with the same probe can avoid them; insert directly above a rib. [BTS 2023]

4. Insertion technique (blunt dissection)

- Equipment: chlorhexidine, drapes, **1% lidocaine (up to 30–40 mL)**, scalpel, two Kelly clamps, 24–36F tubes, filled drainage system, silk suture (Table 13.2).
- Tube size: 24–28F for air; 32–36F for effusion; 36–40F for blood or pus (I&R); small-bore (<14F) suitable for most indications including empyema, larger bore in unstable trauma. [BTS 2023]
- Supine, side elevated, arm over head; time-out; wheal **two finger-breadths below** the chosen space to create a subcutaneous tunnel.
- 2-cm transverse incision; anaesthetise periosteum and parietal pleura (aspirate before each injection); confirm air/fluid by needle aspiration.
- Blunt dissection with Kelly clamp over the upper border of the lower rib; finger sweep; tube guided in — **apically for air, dependently for fluid**; all holes inside the pleura.
- Confirm by condensation/fluid; suture (horizontal mattress allows closure at removal); connect to drainage system; chest radiograph.
- Small-bore 8.5F or pigtail catheters by Seldinger technique are effective for pneumothoraces. [I&R]

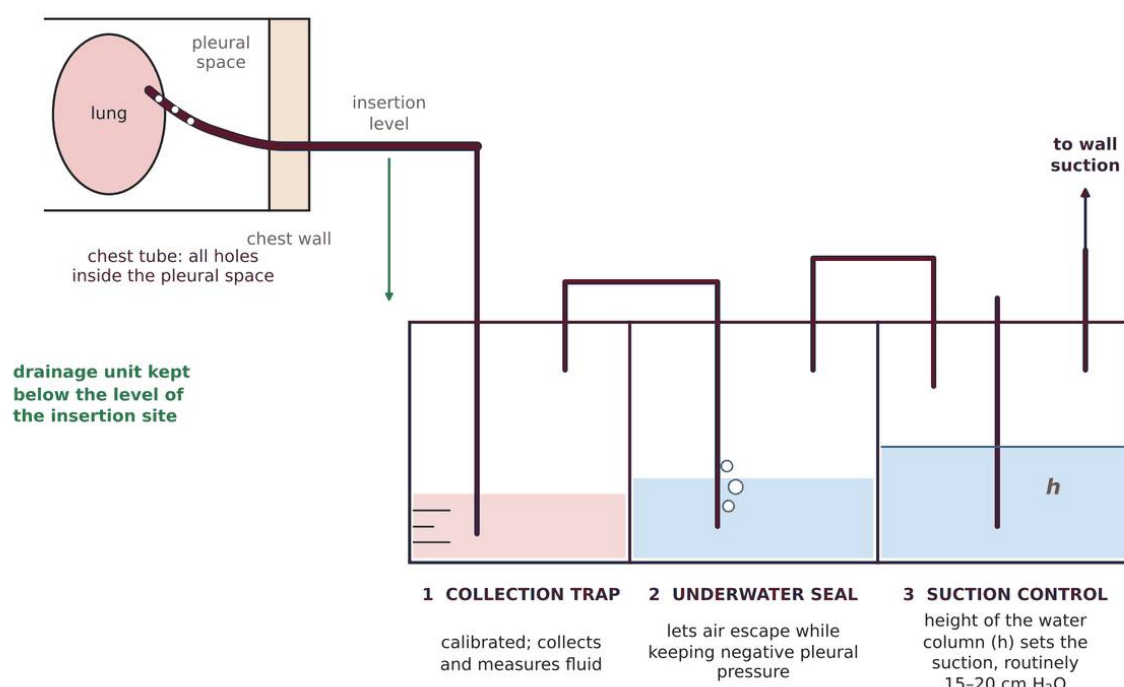


Irwin and Rippe 9e, Figure 13.7 (ch. 13): A, clamp penetrating the intercostal muscle; B, chest tube grasped with a Kelly clamp and guided by a finger through the incision.

5. Drainage systems — critical appraisal by number of chambers

Table 20. Functions of the chambers of a standard (three-chamber) system (Irwin and Rippe 9e, ch. 13)

Chamber	Function	Appraisal (sourced points)
Collection trap (calibrated)	Collects and measures fluid	Measures output — tube for fluid removed when drainage <200 mL/24 h
Underwater seal	Allows air to escape while maintaining negative pleural pressure (one-way valve)	No fluctuation in the water-seal chamber is used as a sign that the leak has ceased [I&R]; a bubbling tube must not be clamped; enough water to cover the tube end; bottle kept below the insertion site [BTS 2023]
Suction control (regulator)	Limits applied suction — set by height of the water column	Routinely 15–20 cm H₂O , kept for the duration of any air leak; BTS: low-pressure, high-volume suction, maximum –20 cm H ₂ O, prescribed by the team; routine suction at initial pneumothorax drainage not advised (re-expansion oedema)



Schematic (drawn for these notes) — three-chamber chest drainage system — chest tube from the pleural space to (1) the calibrated collection trap, (2) the underwater seal and (3) the suction regulator whose water-column height h sets the suction, connected to wall suction. Facts per Irwin and Rippe 9e, Ch 13 Chest Tube Insertion and Care, §Chest Tube Management and Care; BTS 2023 (unit kept below the insertion site). Not to scale.

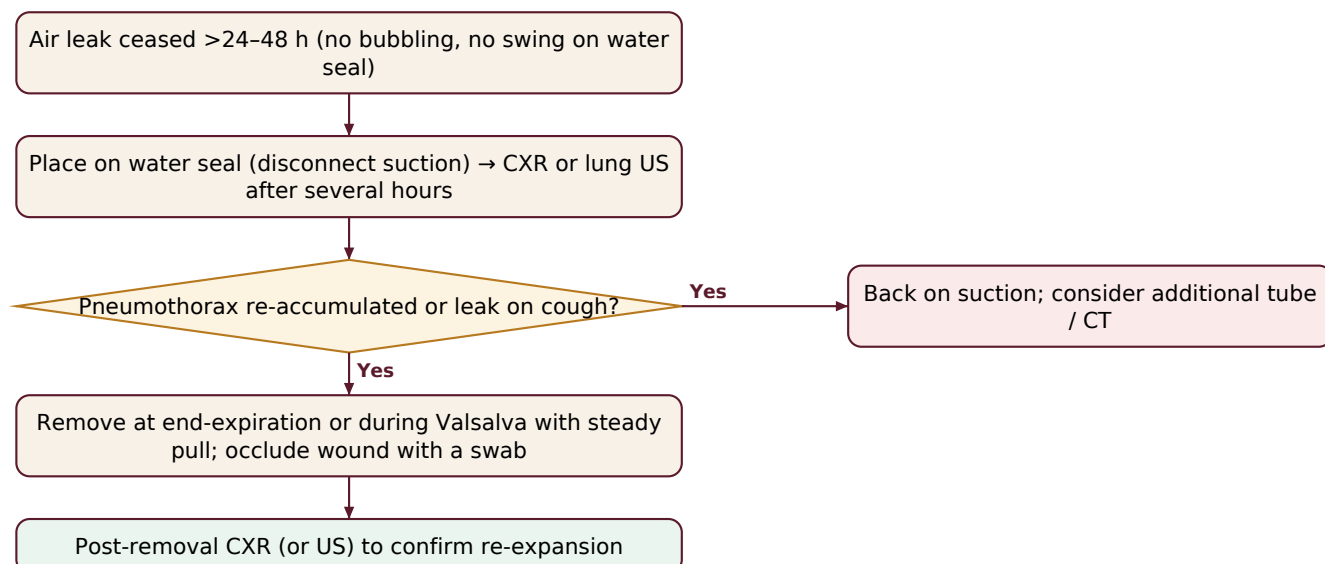
- **Large air leaks** (bronchopleural fistula): compared with water seal, **20 cm H₂O suction** increased air evacuation by all four PDUs tested; **40 cm H₂O** added nothing. [I&R ch. 176]
- At leaks of 4–5 L/min some PDUs become impractical; Pleur-Evac handles up to 34 L/min (impractical >28 L/min from bubbling in the suction chamber); only the low-pressure, high-volume **Emerson pump** handles massive leaks. [I&R ch. 176]
- In BPF use the **least suction that keeps the lung inflated** — negative pressure can transmit to airways (ventilator autotriggering) and increase fistula flow; escaping gas is part of minute ventilation (~25% in ARDS). [I&R ch. 176]
- **Digital suction devices** are an alternative way to deliver thoracic suction. [BTS 2023]
- Heimlich (flutter) device: ambulatory management/discharge [BTS 2023]; small Heimlich valve allows lung re-expansion after thoracentesis-related pneumothorax. [I&R ch. 12]

6. Care of the drain

MANAGEMENT BASIS

British Thoracic Society Clinical Statement on pleural procedures (Thorax 2023). No ISCCM document on chest drains was found. Lines tagged [I&R] from Irwin and Rippe 9e, ch. 13 and 176.

- Ultrasound guidance is **mandatory before pleural fluid procedures** (except emergencies). [BTS 2023]
- Check the tube and system **at least daily**; tight, taped connections; no kinks or dependent loops; **never clamp** a tube on continuous drainage. [I&R]
- **Clamping a bubbling chest tube should be avoided** unless under specialist pleural supervision; clamp promptly if repetitive cough or chest pain during fluid drainage. [BTS 2023]
- Routine milking, stripping and irrigation are discouraged; dressing every 2–3 days. [I&R]
- Daily CXR not recommended — image when breathlessness, desaturation or surgical emphysema occur. [I&R]
- Dislodged tube is never re-advanced; replace at a **different site**. [I&R]
- Persistent pneumothorax: increase suction; then additional tube; then CT. [I&R]

Flowchart. Removal of a chest tube placed for pneumothorax

Based on Irwin and Rippe 9e, ch. 13 §Chest Tube Removal and §Utility of Ultrasonography; BTS 2023 removal statements. Fluid: remove when <200 mL/24 h. Main path = "No".

7. Complications

Table 21. Complications (Irwin and Rippe 9e, Table 13.3 and text)

Complication	Prevention / action
Placement into lung, liver, spleen, heart, retroperitoneum	Triangle of safety, finger sweep, ultrasound for all insertions
Intercostal vessel laceration	Stay over the upper border of the rib; aspirate before injecting
Kinked, too deep or inadequately advanced tube; unresolved/recurrent pneumothorax; persistent effusion	Check position on imaging; reposition/replace at new site
Empyema	Asepsis
Re-expansion pulmonary oedema	Avoid routine suction at initial pneumothorax drainage [BTS 2023]
Overall complications ~9% in blunt trauma; major 1.7%	—

PEARLS — what wins marks

- Triangle of safety: **latissimus dorsi, pectoralis major, nipple line, apex below axilla**; 4th–5th ICS AAL.
- Neurovascular bundle runs under each rib → go over the upper border of the lower rib.
- Three chambers: **collection, water seal, suction control**; suction 15–20 cm H₂O (BTS max –20).
- Water seal must cover the tube end; bottle always below the patient.
- Never clamp a bubbling drain (BTS 2023); never re-advance a dislodged tube.
- Remove: no leak 24–48 h + water-seal trial; fluid <200 mL/24 h.
- Large BPF leak: 20 cm H₂O suction better than water seal; 40 adds nothing.

Textbook: Irwin and Rippe 9e, ch. 13 "Chest Tube Insertion and Care": §Pleural Anatomy and Physiology, §Indications (Table 13.1), §Pneumothorax, §Contraindications, §Technique (Table 13.2; Figs. 13.5, 13.7), §Complications (Table 13.3), §Chest Tube Management and Care, §Chest Tube Removal, §Related Systems, §Utility of Ultrasonography. Ch. 176 "Pleural Diseases of the Critically Ill Patient", §Bronchopleural Fistula — Drainage Systems. Ch. 12 "Thoracentesis", §Procedure. Ch. 9 "Tracheostomy", §Pneumothorax and Pneumomediastinum.

Guidelines: British Thoracic Society, Asciak R, Bedawi EO, Bhatnagar R et al., "BTS Clinical Statement on pleural procedures", Thorax 2023 (statement text, brit-thoracic.org.uk). doi:10.1136/thorax-2022-219371.

Paper II · June 2022 · Q8(a) · 5 marks · Intermediate

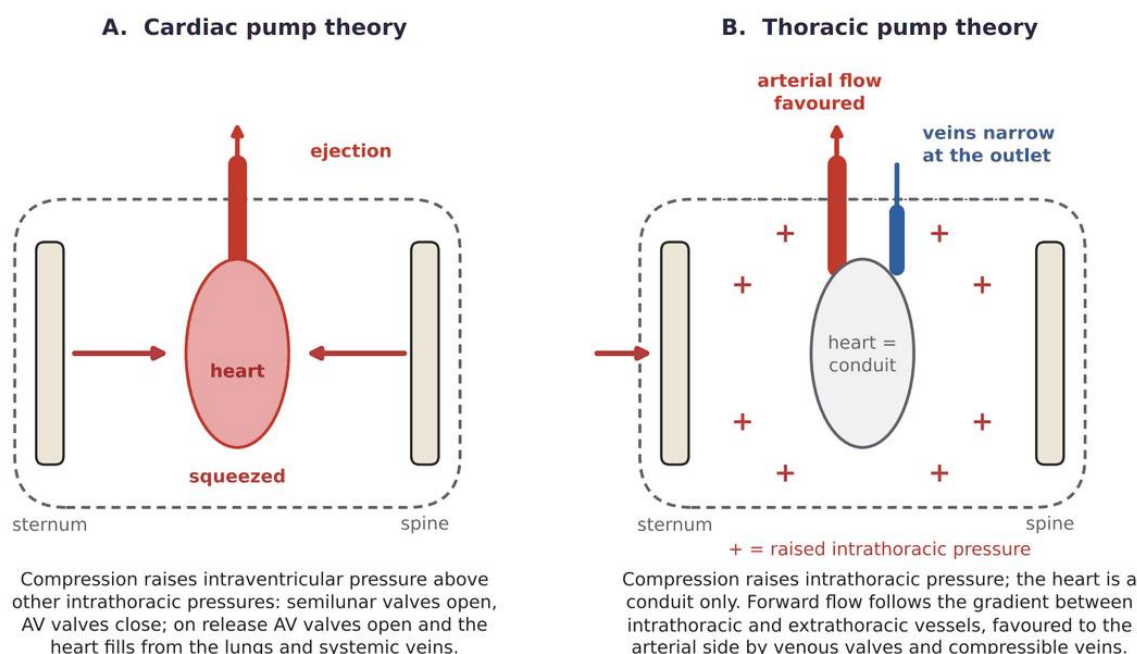
Good quality CPR. [5]

Definition and why it matters

- **High-quality CPR:** minimising interruptions (especially around shocks), adequate compression depth and rate, full chest recoil and avoidance of excessive ventilation.
- Even good CPR produces only **25–33% of normal cardiac output**.
- Survival falls with each minute without ROSC; interruption of CPR lowers the chance that VF converts to another rhythm.
- Seattle data: **43%** of patients in VF discharged if CPR began within 4 min and defibrillation within 8 min.
- In-hospital arrest survival to discharge about **15%**.

Mechanism of blood flow

- **Cardiac pump theory:** heart compressed between sternum and spine; AV valves close, semilunar valves open.
- **Thoracic pump theory:** rise in intrathoracic pressure drives flow; heart acts as a conduit; supported by pressure, angiographic and most echo studies.
- Cardiac pump may predominate in children, thoracic pump in adults.



Which mechanism dominates may vary between patients: cardiac pump in children, thoracic pump in adults.

Schematic (drawn for these notes) — the two theories of blood flow during chest compressions — A, cardiac pump (heart squeezed between sternum and spine; semilunar valves open, AV valves closed); B, thoracic pump (raised intrathoracic pressure; heart a conduit; flow favoured to the arterial side). Facts per Irwin and Rippe 9e, Ch 14 Cardiopulmonary Resuscitation, §Efficacy. Not to scale.

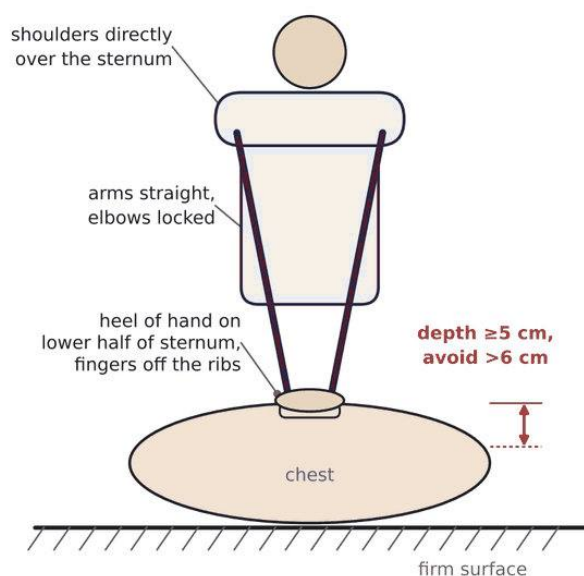
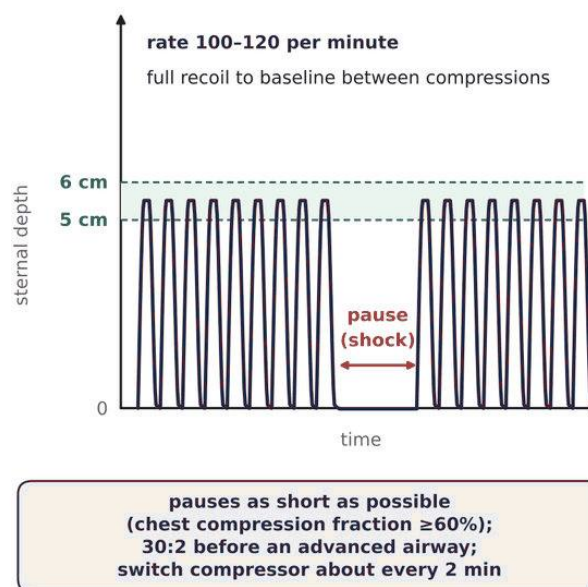
Components of high-quality CPR**MANAGEMENT BASIS**

2025 American Heart Association Guidelines for CPR and ECC — Part 7 (Adult BLS) and Part 9 (Adult ALS), cpr.heart.org. No ISCCM CPR guideline was found. Irwin and Rippe 9e (ch. 14) reflects the 2020 AHA guidance.

Newer: AHA 2025 — rate 100–120/min and depth at least 5 cm but not >6 cm, versus "approximately 100/min" and 4–5 cm in Irwin and Rippe 9e.

Table 22. High-quality CPR targets (AHA 2025)

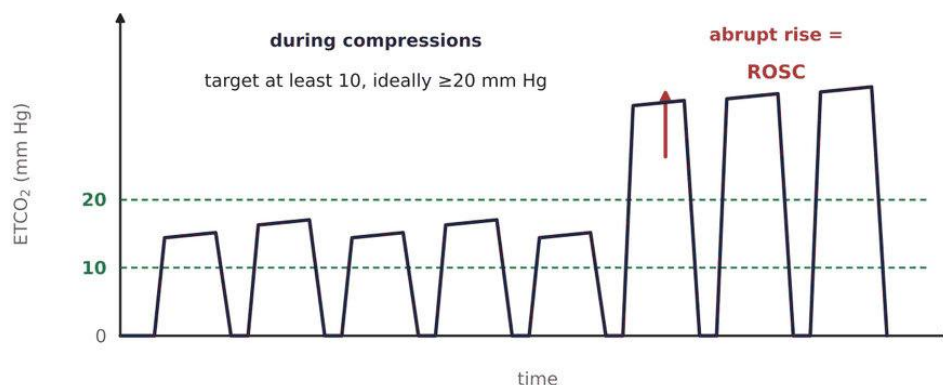
Component	Target	COR, LOE
Rate	100–120/min	2a, B-NR
Depth	At least 5 cm (2 in) in an average adult; avoid >6 cm (2.4 in)	1, B-NR
Recoil	Complete chest-wall recoil — do not lean between compressions	2a, C-LD
Interruptions	Pre- and post-shock pauses as short as possible; chest compression fraction $\geq 60\%$	1, C-LD / 2b, C-LD
Hand position	Heel of one hand on the centre of the chest, other hand on top; dominant hand on sternum may be considered	1, C-LD / 2b, C-LD
Position/surface	Firm surface, supine; torso at about the level of rescuer's knees; partial CPR board in IHCA gives no benefit	2a / 1, B-NR / 3: No Benefit
Ratio	30:2 before an advanced airway	2a, B-NR
Breaths	Visible chest rise; each over 1 s; avoid hypo- and hyperventilation	2a / 2b / 3: Harm
Rotation	Switch compressors about every 2 min	2a, B-R
Feedback	Real-time audiovisual feedback devices	2a, B-R
Pulse check	No more than 10 s	1, C-LD
Physiological monitoring	Arterial pressure or ETCO ₂ may be used; ETCO ₂ at least 10 mm Hg, ideally ≥ 20 mm Hg	2b, C-LD (target in supportive text)

A. Technique**B. Compression targets (adult)**

Schematic (drawn for these notes) — high-quality chest compressions — A, rescuer position and hand placement (Irwin and Rippe 9e, Ch 14 §Basic Life Support); B, depth-time trace against the targets: rate 100–120/min, depth at least 5 cm but not >6 cm, full recoil, shortest possible pauses, chest compression fraction $\geq 60\%$ [AHA 2025]. Not to scale.

Table 23. Examinable change

Point	Irwin and Rippe 9e (ch. 14)	AHA 2025
Rate	Approximately 100/min	100–120/min
Depth	2 in (4–5 cm); 1½–2 in in Table 14.2	At least 5 cm, not >6 cm
ETCO ₂	Goal >10 mm Hg	At least 10, ideally ≥ 20 mm Hg



Schematic (drawn for these notes) — end-tidal CO₂ during CPR — low values during compressions (target at least 10 mm Hg, ideally ≥ 20 mm Hg) and an abrupt rise at return of spontaneous circulation. Facts per Irwin and Rippe 9e, Ch 14 §Basic Life Support (Monitoring Effectiveness) and AHA 2025 Part 9. Schematic, not to scale; amplitudes after ROSC are illustrative.

Technique points (Irwin and Rippe)

- Heel of hand on the **lower half of the sternum**, fingers off the ribs, elbows locked, shoulders over the sternum; equal compression and relaxation time. [I&R]
- Compressions before breathing assessment (C-A-B); interruptions only for essential interventions (intubation, defibrillation) — intubation pause ≤ 10 s; supraglottic airways placed without stopping compressions. [I&R]
- Breaths over 1 s, tidal volume ~ 600 mL; unintended hyperventilation (18–30/min) is common and harmful. [I&R]
- Abrupt ETCO₂ rise (without bicarbonate) indicates ROSC; arterial diastolic pressure approximates coronary perfusion pressure but no adult target is defined. [I&R; AHA 2025]
- Team leader directs rather than performs procedures; rapid response teams lower in-hospital arrest rates. [I&R]
- Interposed abdominal compression CPR is an option for in-hospital arrest with trained staff; mechanical devices not shown to improve outcome. [I&R]

Complications of compressions

- Rib and sternal fractures, costochondral separation, flail chest, pneumothorax, haemothorax, haemopericardium, lung contusion, fat/marrow embolism, liver/spleen/stomach laceration — minimised by correct hand position and avoiding excessive depth; gastric distension with mask ventilation.

PEARLS — what wins marks

- Push hard (≥ 5 cm, ≤ 6 cm), push fast (100–120/min), full recoil, minimal pauses (CCF $\geq 60\%$), no hyperventilation.
- 30:2 without advanced airway; switch compressor every 2 min; pulse check ≤ 10 s.
- ETCO₂ ≥ 10 mm Hg (ideally ≥ 20); sudden rise = ROSC.
- CPR gives only 25–33% of normal cardiac output — every pause matters.
- Audiovisual feedback devices: reasonable (2a, B-R).
- Trap: the book's "100/min, 4–5 cm" is superseded by AHA 2025.

Textbook: Irwin and Rippe 9e, ch. 14 "Cardiopulmonary Resuscitation": §Efficacy, §Experimental and Alternative Techniques (Table 14.1), §Standard Procedures and Team Effort, §Basic Life Support (Chest Compressions, Two-Rescuer CPR, Complications, Monitoring Effectiveness; Table 14.2), §ACLS — Airway and Ventilatory Support, Circulatory Support.

Guidelines: American Heart Association, "Part 7: Adult Basic Life Support" and "Part 9: Adult Advanced Life Support", 2025 AHA Guidelines for CPR and ECC, Circulation 2025 (cpr.heart.org; volume/pages not shown on the pages read).

Paper I • October 2023 • Q10 • 10 marks • Long**Describe about defibrillation - devices and indications with doses. [4+6]**

Also asked as: Paper II • June 2022 • Q8(b) “Role of synchronized cardioversion. [5]” • **Paper I • May 2024 • Q4(a)** “Compare and contrast between defibrillation and cardioversion. [4]”

1. Definitions

- **Defibrillation:** termination of **VF** (and pulseless VT) with an **unsynchronised** shock.
- **Cardioversion:** restoration of sinus rhythm in arrhythmias **other than VF** — electrical (DC shock **synchronised to the QRS**) or pharmacological.
- Synchronisation avoids a shock on the **vulnerable part of the T wave**, which can trigger VF.

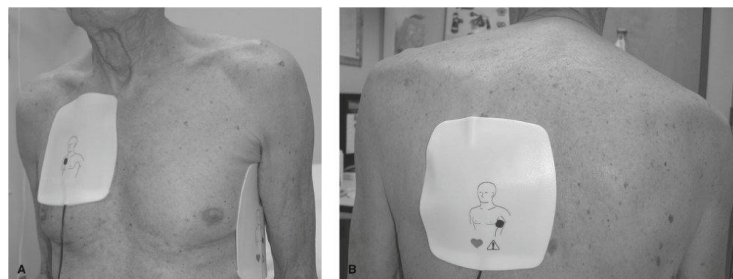
2. Physiology

- Shock causes synchronous depolarisation of myocardium; on repolarisation organised pacemaker activity can emerge.
- **Critical mass hypothesis:** silencing most of the myocardium leaves too little to sustain fibrillation.
- Success depends on energy, waveform, myocardial refractory state and **transthoracic impedance**.
- Impedance falls with firm pad pressure, clipping hair, avoiding breast tissue, shocking in expiration, larger pads; the first shock lowers impedance for later shocks.

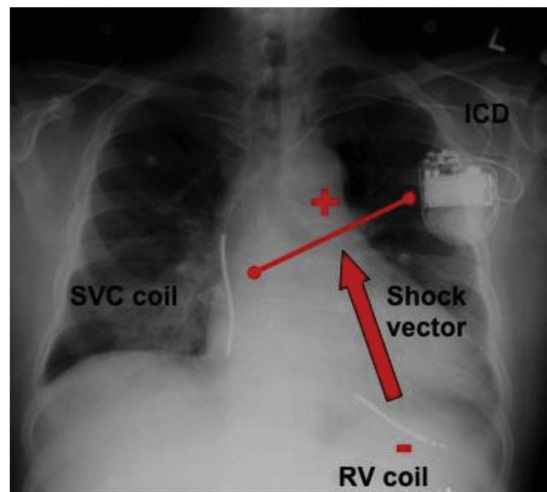
3. Devices

Table 24. Devices for defibrillation and cardioversion (Irwin and Rippe 9e, ch. 14, 15, 196, 197)

Device	Features	Notes
Manual external defibrillator — monophasic	Higher energy needed (360 J)	Largely replaced by biphasic
Manual external — biphasic	Lower defibrillation threshold, less energy, fewer burns	Superior to monophasic for AF cardioversion; preferred for arrest
Automated external defibrillator (AED)	Automatic or semi-automatic	Early AED use improves out-of-hospital survival; does not reduce operator diligence
Paddles vs self-adhesive pads	Paddles: lower impedance, burns rare; pads: burns common	One study: paddles better for AF cardioversion
Implantable cardioverter–defibrillator (ICD)	Dual-coil RV lead + active can; shock vector RV coil → SVC coil and can	Electrical storm = ≥3 VT/VF episodes in 24 h
Subcutaneous ICD (S-ICD)	Generator left anterior–midaxillary, lead tunnelled subcutaneously	Avoids vascular access
Wearable defibrillator	Non-implanted	Option at discharge for selected patients after ventricular arrhythmia (ch. 196)
Double sequential defibrillation	Near-simultaneous shocks from two defibrillators	Usefulness not established



Irwin and Rippe 9e, Figure 15.1 (ch. 15): self-adhesive pads — A, anterior and lateral positions; B, posterior pad (the second pad then goes anteriorly).



Irwin and Rippe 9e, Figure 197.9 (ch. 197): ICD shock vector from the RV coil to the SVC coil and active can.

4. Indications

- **Defibrillation (unsynchronised):**
 - VF and pulseless VT — the definitive treatment, as early as possible
 - Polymorphic VT — high-energy (maximum setting) unsynchronised shock [AHA 2025]
- **Synchronised cardioversion:**
 - Any tachyarrhythmia causing **haemodynamic instability** (hypotension, altered mentation, chest pain, heart failure)
 - Stable WCT or NCT when vagal manoeuvres and drugs fail or are contraindicated
 - Elective cardioversion of AF/flutter (after anticoagulation strategy)
- Do **not** shock **sinus tachycardia** in hypotension from non-cardiac causes.
- Caution: digitalis toxicity, hypokalaemia (VT/VF after shock), sinus node disease (post-shock bradycardia).

MANAGEMENT BASIS

2025 American Heart Association Guidelines for CPR and ECC — Part 9 Adult Advanced Life Support (cpr.heart.org). No ISCCM document found. Device, technique and anticoagulation lines [I&R] from Irwin and Rippe 9e, ch. 14–15 (2020 AHA-based).

Newer: AHA 2025 — AF: initial synchronised energy at least 200 J (2a, B-R); atrial flutter: 200 J may be reasonable (2b, C-LD) — higher than the textbook's 120–200 J (AF) and 50–100 J (flutter).

5. Energy doses

Table 25. Initial energy — textbook vs current guideline

Rhythm	Irwin and Rippe 9e (Table 15.3 and text)	AHA 2025
VF / pulseless VT	Biphasic 120–200 J (device-specific; 200 J if unknown); monophasic 360 J	Manufacturer's recommended dose first (2b, C-LD); higher energy for later shocks may be considered (2b, B-R); biphasic preferred (2a, B-R); single shocks, not stacked (2a, B-NR)
VT with pulse (monomorphic)	Biphasic 50 J (50–100 J for unstable WCT, escalating); monophasic 100 J	Synchronised cardioversion for unstable WCT (1, B-NR); no joule value given
Polymorphic VT	—	Maximum manufacturer setting, unsynchronised
Atrial fibrillation	Biphasic 120–200 J (Table: 200 J); monophasic 100–200 J	At least 200 J (2a, B-R)
Atrial flutter	Biphasic 50 J (50–100 J); monophasic 50–100 J	200 J may be reasonable (2b, C-LD)
SVT (narrow complex)	Biphasic 50–100 J	Synchronised cardioversion for unstable NCT (1, B-NR); no joule value given

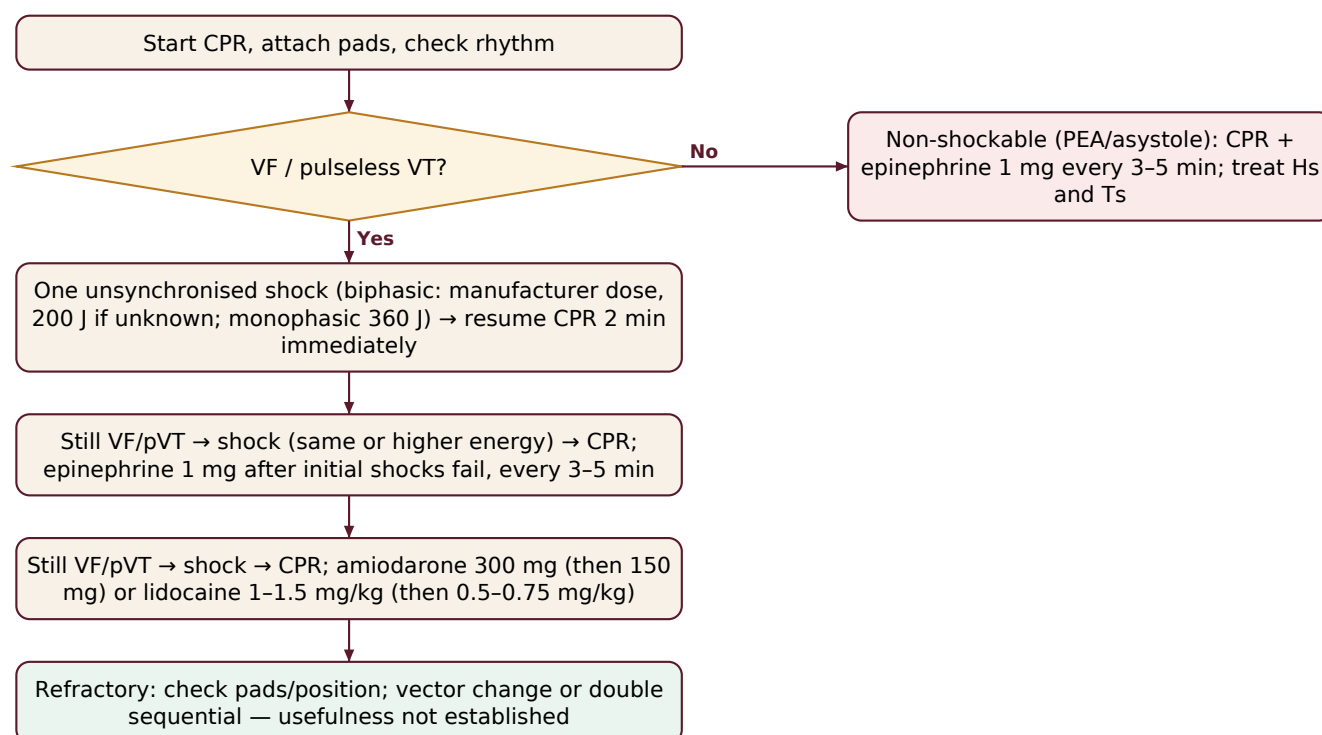
6. Procedure of synchronised cardioversion (J22-2-8b)

- Consent; **fasting 6 h solids / 2 h clear liquids** when elective; continuous rhythm monitoring; 12-lead ECG before and after.
- Sedation/analgesia with short-acting drugs: **midazolam + fentanyl**, or **propofol** with an anaesthetist; goal minimal or no response to voice.
- Pads anterolateral or anteroposterior (heart between electrodes); AP preferred with a pacemaker/ICD and possibly for AF with monophasic devices; never over a device.
- Select **SYNC**, confirm sync markers on QRS (change lead if QRS small), choose energy, charge, clear, shock; escalate if no change.
- **Sync is deselected after each shock** on most devices — reselect before the next.
- Resistant AF: change pad position, firmer pressure, larger pads, shock in expiration, give a class III drug (sotalol, ibutilide, dofetilide, amiodarone) then repeat.
- Pad pressure raised cardioversion success from 87% to 93%, and repositioning to 96%. [AHA 2025 supportive text]

7. Anticoagulation around cardioversion of AF/flutter [I&R]

- Pericardioversion thromboembolism: **5.3%** without vs **0.8%** with anticoagulation.
- AF <24–48 h: TEE not required; peri-cardioversion heparin, direct thrombin inhibitor or factor Xa inhibitor acceptable.
- AF >48 h: **TEE** to exclude thrombus, or anticoagulate **≥3 weeks before**; continue **4 weeks after**.
- Pharmacological cardioversion: dofetilide, flecainide, ibutilide, propafenone, amiodarone, quinidine — no sedation needed, lower success.

Flowchart. Shockable cardiac arrest — defibrillation sequence



Based on Irwin and Rippe 9e Table 15.4 and AHA 2025 Part 9 recommendations.

8. Defibrillation vs cardioversion — compare and contrast (M24-1-4a)

Table 26. Defibrillation vs synchronised cardioversion (Irwin and Rippe 9e, ch. 15; AHA 2025)

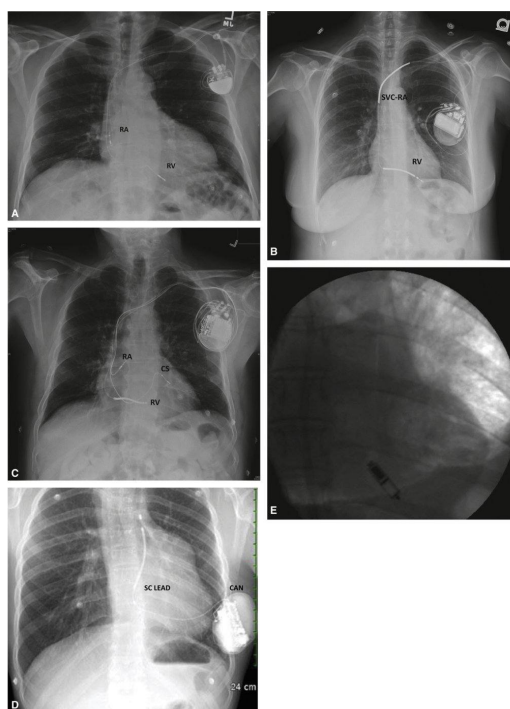
Feature	Defibrillation	Synchronised cardioversion
Rhythm	VF, pulseless VT (polymorphic VT)	AF, flutter, SVT, VT with pulse
Timing of shock	Unsynchronised — immediate	Synchronised to R wave — avoids T-wave shock
Patient state	Pulseless, unconscious — emergency	Pulse present; unstable (urgent) or stable (elective)
Sedation / consent	None	Consent and sedation when time allows; fasting if elective
Energy	High: biphasic 120–200 J / monophasic 360 J	Lower for SVT/flutter (textbook 50–100 J); AF ≥200 J (AHA 2025)
Anticoagulation	Not applicable	AF/flutter >48 h: TEE or ≥3 weeks before, 4 weeks after
Device setting	Sync OFF	Sync ON — reselect after each shock
Main risks	Burns, post-shock arrhythmias	VF if unsynchronised, thromboembolism, post-shock bradycardia, sedation complications

9. Complications

- **Burns** and pain at pad sites — commoner with pads and higher total energy; less with biphasic.
- **Thromboembolism** after AF/flutter cardioversion (also theoretical with LV apical thrombus).
- **Arrhythmias**: transient sinus arrest/bradycardia (unmasked sinus node disease); VT/VF with digitalis toxicity or hypokalaemia.
- **Myocardial damage**: transient ST elevation; CK-MB rise in 10% at high energy but no troponin rise — clinically significant injury unlikely.
- Implanted devices: reprogramming, lead-tip injury, threshold rise — pads away from the generator; interrogate after any external shock.

10. Special situations

- **Pregnancy**: performed in all trimesters without obvious fetal harm; monitor fetal heart rhythm.
- **Precordial thump**: last resort only in a pulseless patient when no defibrillator is available; may trigger VF; never with a pulse.
- **Pre-excited AF**: avoid AV-nodal blockers; procainamide or ibutilide, or immediate cardioversion if unstable.



Irwin and Rippe 9e, Figure 197.1 (ch. 197): chest radiographs of implanted devices — dual-chamber pacemaker, ICD with coils, cardiac resynchronisation device and subcutaneous ICD.

PEARLS — what wins marks

- Defibrillation = unsynchronised (VF/pVT); cardioversion = synchronised to QRS (all other tachyarrhythmias with pulse).
- Biphasic preferred (AHA 2025, 2a); VF: manufacturer dose or 200 J biphasic, 360 J monophasic; single shocks.
- AF: ≥ 200 J (AHA 2025, 2a, B-R); flutter 200 J (2b) — textbook lower values superseded.
- Unstable WCT or NCT → synchronised cardioversion (COR 1, B-NR).
- Re-select SYNC after every shock.
- AF >48 h: TEE or 3 weeks anticoagulation before, 4 weeks after.
- Trap: never cardiovert sinus tachycardia; pads never over an implanted device.

Textbook: Irwin and Rippe 9e, ch. 15 "Cardioversion and Defibrillation": §Introduction, §Physiology of Arrhythmia and Shock, §Indications and Contraindications, §Clinical Competence (Table 15.1), §Methods (Patient Preparation, Shock Waveforms, Electrodes, Using the Defibrillator; Tables 15.2–15.4; Fig. 15.1), §Wide/Narrow Complex Tachycardia, §AF and Flutter (Electrical Cardioversion, Anticoagulation, Pharmacologic Cardioversion, Resistant AF), §Complications, §Miscellaneous Topics. Ch. 14 "Cardiopulmonary Resuscitation", §Defibrillation; §PEA and Asystole. Ch. 196 "Management of Common Arrhythmias", §Arrhythmias in Stress-Induced Cardiomyopathy. Ch. 197 "Management of Cardiac Devices in the ICU", Figs. 197.1, 197.9; §Electrical Storm.

Guidelines: American Heart Association, "Part 9: Adult Advanced Life Support", 2025 AHA Guidelines for CPR and ECC, Circulation 2025 (cpr.heart.org) — defibrillation, cardioversion (WCT, NCT, AF/flutter) and drug recommendations.

PART 2

Monitoring and Applied Physiology

Table 27. Answers in Part 2

#	Answer
1	Pulse oximetry and perfusion index
2	Cardiac output monitoring: principles and non-invasive methods
3	Capnography
4	Haemoglobin–oxygen dissociation curve
5	Auto-PEEP (intrinsic PEEP)
6	Driving pressure and mechanical power
7	Oesophageal/transpulmonary pressure and electrical impedance tomography
8	Ventilator scalars and loops (incl. volume–pressure loop)
9	Time constant in respiratory mechanics
10	ICP monitoring: techniques
11	Monro–Kellie doctrine
12	Jugular venous oximetry
13	Cerebral autoregulation
14	Transcranial Doppler and neuromonitoring of cerebral oedema in stroke

PART 11

Respiratory Failure, Ventilation and ECMO

Table 69. Answers in Part 11

#	Answer
1	Respiratory failure and bedside non-invasive pulmonary monitoring
2	ARDS: Berlin definition, severe ARDS and refractory hypoxaemia
3	ARDS: effects on the kidney and cardiovascular system
4	Methods of setting PEEP in ARDS
5	Prone position ventilation
6	Mechanisms of hypoxaemia
7	Approach to hypercarbia
8	Ventilator-induced lung injury
9	Modes of mechanical ventilation and their classification
10	APRV
11	Closed-loop ventilation
12	Heart–lung interactions during mechanical ventilation
13	ECMO modalities and non-cardiac indications
14	Inter-hospital air transport of ARDS patients to ECMO centres
15	High-flow nasal cannula
16	ROX index and HACOR score
17	Helmet NIV
18	Weaning failure and weaning-induced cardiac dysfunction
19	Ventilator-induced diaphragm dysfunction and diaphragm ultrasound
20	ICU liberation (ABCDEF) bundle and post-intensive care syndrome
21	Oxygen delivery devices
22	Nebulisers
23	Early mobilisation and chest physiotherapy
24	Hyperoxia in critical illness
25	Hyperbaric oxygen therapy
26	Community-acquired pneumonia: severity, ICU management, steroids, newer antibiotics
27	Frailty and geriatric critical care
28	Hypotension on VA-ECMO

Paper I · April 2023 · Q5 · 10 marks · Long

Define respiratory failure. Discuss the common non-invasive techniques for monitoring pulmonary function at bedside in an ICU. [2+8]

1. Definition of respiratory failure

- The respiratory system has two functions: **ventilation** (elimination of CO_2) and **oxygenation** (formation of oxy-haemoglobin); respiratory failure is failure of one or both.
- Irwin and Rippe categorise respiratory failure into two overlapping types:
 - Lung failure** — pure gas-exchange failure, manifested by **hypoxaemia**; commonly ARDS or cardiogenic pulmonary oedema.
 - Pump failure** (= ventilatory failure) — manifested by **hypercapnia and hypoxaemia**; commonly CNS depression (overdose, anaesthesia) or respiratory muscle fatigue/weakness.
- Respiratory failure in COPD is usually a **combination** of lung failure (lost alveoli and vascular bed → V/Q mismatch and shunt) and pump failure (mechanical disadvantage, impaired muscle/neural function).
- Extrapulmonary causes act by **hypoventilation** — the failure is always hypercapnic, with a normal or near-normal A–a gradient.

NUMERIC CUT-OFFS — SOURCE NOTE

Irwin and Rippe 9e gives no numeric $\text{PaO}_2/\text{PaCO}_2$ definition of respiratory failure (searched "respiratory failure is defined", "type 1/type 2 respiratory failure", "PaO₂ less than 60"). Cut-offs below are from the BTS Guideline for Oxygen Use in Adults (Thorax 2017), §3.2–3.3.

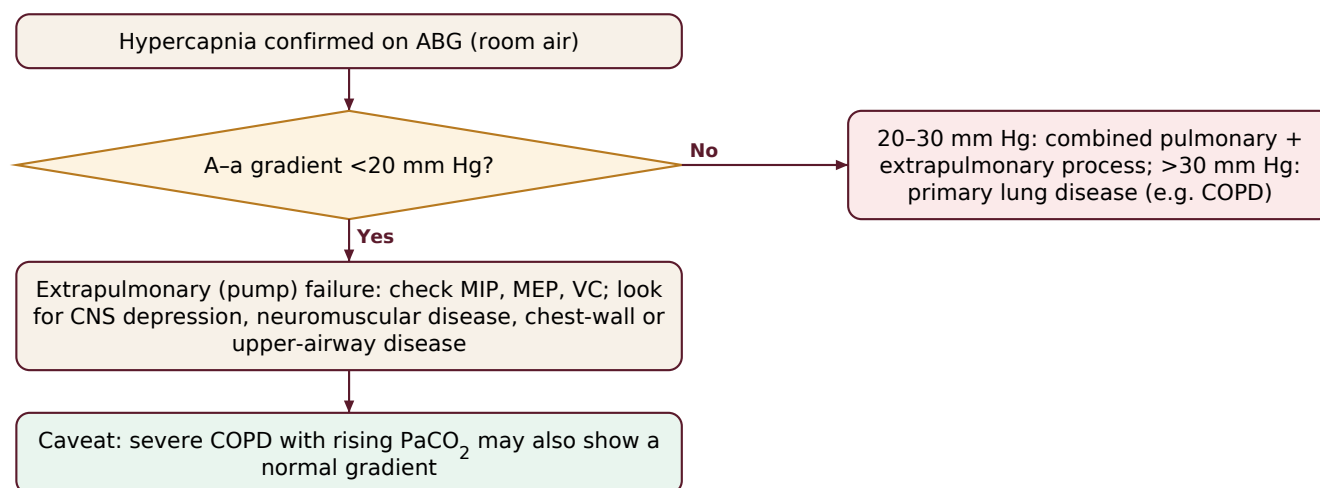
Table 70. Types of respiratory failure — numeric definitions (BTS 2017, §3.2–3.3)

Type	Definition	I&R equivalent
Hypoxaemia	$\text{PaO}_2 < 60$ mm Hg (8 kPa) or $\text{SaO}_2 < 90\%$ ("most authors")	—
Type 1 (hypoxaemic)	$\text{PaO}_2 < 8$ kPa (60 mm Hg), $\approx \text{SaO}_2$ 90%, with normal or low PaCO_2	Lung failure
Type 2 (hypercapnic)	PaCO_2 above normal 4.6–6.1 kPa (34–46 mm Hg), even if SpO_2 is normal; acidosis = pH < 7.35 (§3.4)	Pump (ventilatory) failure

2. Mechanisms and bedside classification

- ICU hypoxaemia/hypercapnia arise from **hypoventilation, diffusion impairment and V/Q mismatch** (true shunt is the extreme of V/Q mismatch).
- Hypoventilation: $\text{PaCO}_2 = (\text{VCO}_2/\text{V}_A) \times k$ — CO_2 rises as alveolar ventilation falls.
- Hypercapnia with A–a gradient < 20 mm Hg** on room air is, with few exceptions (severe COPD), diagnostic of extrapulmonary failure; 20–30 mm Hg suggests a combined process; >30 mm Hg points to primary lung disease.
- Clinical estimation of hypoxaemia is **exceptionally unreliable**; respiratory rate is often recorded inaccurately (>20% error in more than one-third of ICU observations) — hence objective monitoring.
- Hypercapnia should be suspected with anxiety, dyspnoea, lethargy, headache, **asterixis**, myoclonus, seizures or altered sensorium; normal subjects tolerate PaCO_2 75–80 mm Hg acutely, chronic retainers often 90–100 mm Hg.

Flowchart. Bedside localisation of hypercapnic respiratory failure (A-a gradient on room air)



Drawn from Irwin and Rippe 9e, ch. 165, §Diagnosis — General Considerations; §Differential Diagnosis.

3. Overview — non-invasive bedside monitors

Table 71. Non-invasive techniques for monitoring pulmonary function (Irwin and Rippe 9e, ch. 27, 29, 11, 165, 168)

Technique	What it measures	Key strength	Key limitation
Impedance pneumography (ECG leads)	Respiratory rate, approximate V_T	Cheap when ECG already in use	Misses obstructive apnoea; only 4% of respiratory alarms clinically relevant (one study)
Respiratory inductive plethysmography	RR, % change in V_T , FRC change, paradox	Detects obstructive apnoea, asynchronous breathing	Needs calibration; drift on ventilated patients
Pulse oximetry	SpO_2	Within 2% of SaO_2 under ideal conditions	Unreliable <70%; dyshaemoglobins, low perfusion, dark skin
Capnography	$EtCO_2$, waveform, RR	Tube position, apnoea, CPR quality, dead space	Not a substitute for $PaCO_2$
Transcutaneous PO_2/PCO_2	$PtcO_2$, $PtcCO_2$	Tracks arterial values in healthy adults	Reflects local perfusion in shock
Ventilator mechanics	P_{plat} , C_{rs} , R_{rs} , auto-PEEP, ΔP , $P_{0.1}$	Breath-by-breath, no blood sample	Needs passive patient for occlusions
Strength/effort indices	MIP, MEP, VC, RSBI	Predict hypercapnia and weaning	Effort dependent
Lung ultrasound / EIT	Aeration score; regional ventilation	Repeatable, radiation-free	Cannot separate recruitment from overdistension (LUS)

4. Respiratory rate, tidal volume and minute ventilation

- Primary parameters to monitor: **respiratory rate, tidal volume/minute ventilation, oxygenation and ventilation.**
- **Impedance monitors:** use ECG leads; sense impedance change with thoraco-abdominal motion; alarms for high/low rate. May count chest-wall movement against an obstructed airway as breaths → poor for obstructive apnoea; inaccurate in mobile patients.
- **Respiratory inductive plethysmography (RIP):** two elastic bands (above xiphoid and abdomen) with wire coils; changes in cross-sectional area → rate and % change in V_T .
- RIP extras:
 - Detects obstructive apnoea and paradoxical/asynchronous breathing (common in early weaning).
 - Shows FRC change — effect of PEEP, bronchodilator response, estimate of auto-PEEP.
 - Must be calibrated against a known volume; only about two-thirds of V_T estimates within 10% on ventilated patients.
- Other: capnography (accurate RR), pneumotachometer (needs intubation/tight mask), surface EMG (no V_T , poor in obese/oedematous adults); emerging — bed/pillow sensors, acoustic monitoring, photoplethysmography.

5. Pulse oximetry

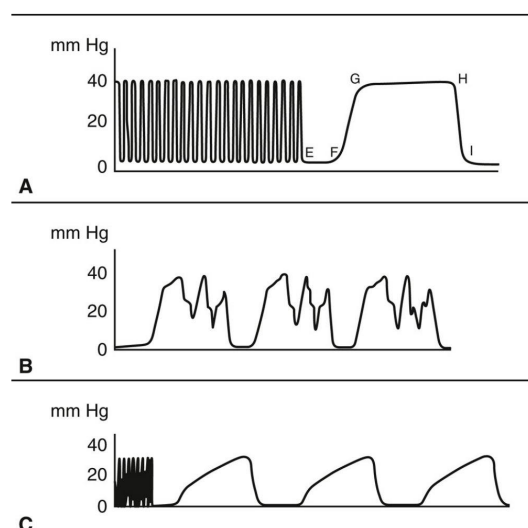
- Principle: oxy-Hb absorbs less **red** (~660 nm) and slightly more **infrared** (~910–940 nm) light than reduced Hb; the pulsatile (arterial) component — only 1–5% of signal — gives SpO₂ from the red:infrared absorption ratio.
- Accuracy usually within **2%** of SaO₂ (ideal conditions); meta-analysis within 5%; regard as **unreliable when SpO₂ <70%** (calibrated on volunteers).
- Errors: severe hypoxaemia, dyshaemoglobinaemia, low perfusion, skin pigmentation (occult hypoxaemia 3× more often in Black patients), motion, ambient light, lipids, nail polish.
- **COHb** read as ~90% oxy-Hb → falsely high SpO₂; **MetHb** biases reading toward 85–90%; **methylene blue** falsely lowers SpO₂; bilirubin does not affect two-wavelength oximeters.
- Response is slower for finger/toe than ear, cheek or glossal probes; a plethysmographic waveform helps recognise artefact.
- SCCM considers pulse oximetry (or transcutaneous O₂) **essential monitoring** for all ICU patients on supplemental O₂; not useful for prompt detection of oesophageal intubation (desaturation lags in pre-oxygenated patients).

Table 72. Conditions adversely affecting oximetry (Irwin and Rippe 9e, Table 27.1)

Poor signal	Falsely lowers SpO ₂	Falsely raises SpO ₂
Probe malposition, no pulse, motion	Nail polish, dark skin	Elevated carboxyhaemoglobin
Vasoconstriction, hypothermia, hypotension	Ambient light, elevated serum lipids	Elevated methaemoglobin, ambient light
—	Methylene blue, indigo carmine, indocyanine green	Hypothermia

6. Capnography (EtCO₂)

- Usually **infrared absorbance** (CO₂ peak ≈4.28 μm), response ~100 ms; mass spectrometry is slower (45 s–5 min) and costly.
- **Mainstream:** in-line, near-instant, needs intubation or tight mask. **Sidestream:** aspirates 100–300 mL/min, delay and clogging, but usable in non-intubated patients (e.g. procedural sedation).
- Waveform: phase I (inspired, ~0 CO₂) → phase II (rapid rise, dead space + alveolar gas) → phase III (alveolar plateau; EtCO₂ = last 20% of exhalation) → phase IV (inspiratory down-stroke).
- Normal PetCO₂ **35–45 mm Hg**; in health EtCO₂–PaCO₂ difference ±1.5 mm Hg; widens with dead space or low perfusion.
- Uses: confirm ETT placement, apnoea/disconnection alarms, CPR quality (>10 mm Hg) and ROSC, fluid responsiveness (≥5% rise with passive leg raise), airflow obstruction (increased α angle), dead-space rise in PE.
- **Not a substitute for PaCO₂** in the critically ill: in one study EtCO₂ differed from PaCO₂ by 13 (mainstream) and 9.7 mm Hg (sidestream).



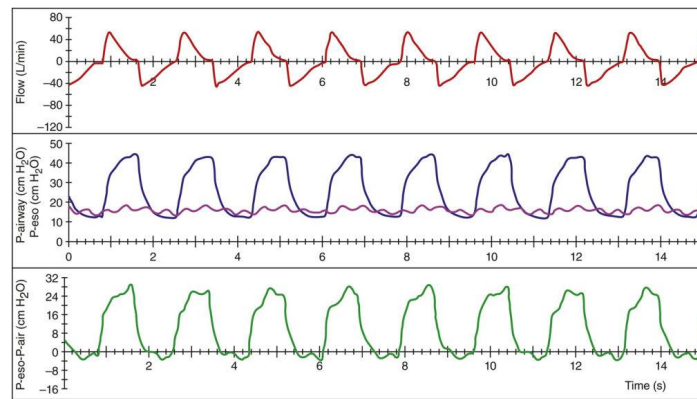
Irwin and Rippe 9e, Figure 27.1 (ch. 27): A, normal capnogram (EF inspiration, FG dead-space washout, GH alveolar plateau, end-tidal value at H); B, erratic breathing; C, obstructive airway disease with sloping plateau.

7. Transcutaneous and regional gas monitoring

- **PtcO₂/PtcCO₂**: in healthy adults track arterial values — typically ~10 mm Hg lower (O₂) and 5–23 mm Hg higher (CO₂).
- In shock, local values reflect **regional perfusion** as well as gas exchange (low flow → low local PO₂, high local PCO₂).
- Related perfusion monitors (gastric tonometry, sublingual capnometry) predict outcome but have not improved it.

8. Ventilator-derived respiratory mechanics

- **End-inspiratory occlusion** (passive patient, constant flow) → Pplat; PIP – Pplat = resistive load; $R_{rs} = (PIP - Pplat)/\text{flow}$.
- **Static compliance** $C_{st,rs} = V_T/(Pplat - PEEP_t)$; elastance is its reciprocal; normal $C_{rs} \approx 70 \text{ mL/cm H}_2\text{O}$ (ch. 166).
- **End-expiratory occlusion** → total PEEP; $PEEP_i = PEEP_t - \text{set PEEP}$; persistent end-expiratory flow on the flow trace signals auto-PEEP.
- **Driving pressure** (Pplat – PEEP) — most predictive ventilatory variable for ARDS mortality (RR 1.41 per ~7 cm H₂O).
- **Dead space** (V_D/V_T , modified Bohr equation; volumetric capnography at the Y-piece) — rises in ARDS, COPD and with excess PEEP; serial values correlate with ARDS outcome.
- **P0.1** (airway occlusion pressure at 0.1 s) — index of drive; –3.5 cm H₂O suggested cut-off for high effort.
- Oesophageal pressure (balloon) separates lung and chest-wall mechanics and gives transpulmonary pressure — minimally invasive (Fig. 29.4).



Irwin and Rippe 9e, Figure 29.4 (ch. 29): flow, airway pressure (P-air) with superimposed oesophageal pressure (P-eso), and their difference (estimated transpulmonary pressure) during pressure-control ventilation.

9. Respiratory muscle strength and weaning indices

- **MIP (NIF)**: normal about –80 cm H₂O; hypercapnia from weakness rarely before MIP falls to ≤30% predicted; MIP less negative than –30 cm H₂O predicts hypercapnia (best objective predictor).
- **MEP <40 cm H₂O** → ineffective cough, aspiration, atelectasis, pneumonia.
- **VC <1 L or <15 mL/kg** — associated with hypercapnia, less sensitive than MIP; in myasthenia, VC <20 mL/kg, MIP less negative than –30 or MEP <40 cm H₂O warn of impending failure.
- **RSBI (f/V_T)**: originally <105 breaths/min/L (commonly <100) favours successful discontinuation; RR >38/min plus RSBI >100 lowers the chance of success.
- Signs of fatigue: rising RR with falling V_T , **abdominal paradox**, respiratory alternans.

10. Bedside imaging — lung ultrasound and EIT

- **LUS score** (6 interspaces per hemithorax, 0–3 each, max 36): 0 = A-lines with sliding, 1 = separated B-lines, 2 = confluent B-lines, 3 = consolidation/effusion.
- Rise in LUS score >4 points during a 30-min SBT, or post-SBT score >17, predicted extubation failure.
- PEEP 0 → 15: fall of 8 points ≈ >600 mL gain in lung volume — but recruitment cannot be separated from overdistension.
- **Electrical impedance tomography**: 16–32 chest electrodes → 2-D impedance map of regional ventilation, collapse and overdistension; approved for regional ventilation monitoring; PEEP titration still in development.

PEARLS — what wins marks

- Lung failure = hypoxaemia; pump (ventilatory) failure = hypercapnia + hypoxaemia (I&R).
- BTS: type 1 $\text{PaO}_2 < 60$ mm Hg with normal/low PaCO_2 ; type 2 $\text{PaCO}_2 > 46$ mm Hg (normal 34–46).
- Hypercapnia + A–a gradient < 20 mm Hg = extrapulmonary failure.
- Pulse oximeter: within 2%; unreliable $< 70\%$; COHb falsely high, methylene blue falsely low.
- EtCO_2 normal 35–45 mm Hg; never a substitute for PaCO_2 in the critically ill.
- MIP less negative than -30 cm H_2O , VC < 15 mL/kg, MEP < 40 cm H_2O = danger thresholds.
- RSBI < 105 (commonly < 100) breaths/min/L; LUS rise > 4 points during SBT predicts extubation failure.

Textbook: Irwin and Rippe 9e, ch. 29 "Respiratory Monitoring During Mechanical Ventilation", §Gas Exchange (Basic Physics, Dead Space), §Respiratory Mechanics (Compliance and Elastance, Intrinsic PEEP, Driving Pressure, Esophageal Pressure Monitoring, Electrical Impedance Tomography, Airway Occlusion Pressure, Maximal Inspiratory Force), Fig. 29.4. Ch. 27 "Routine Monitoring of Critically Ill Patients", §Respiratory Monitoring (Impedance Monitors, RIP, Pulse Oximetry, Table 27.1, Capnography, Fig. 27.1), §Noninvasive Tissue Perfusion Monitoring. Ch. 165 "Extrapulmonary Causes of Respiratory Failure", Key Points, §Diagnosis, §Clinical Features of Hypercapnia, §Differential Diagnosis. Ch. 168 "Discontinuation of Mechanical Ventilation", §Understanding the Problem, §Predictive Indices. Ch. 173 "Critical Care of Acute Exacerbations of COPD", §Severity Scoring. Ch. 11 "Lung Ultrasonography", §Prediction of Extubation Failure, §Measurement of Lung Recruitment With PEEP. Ch. 166, §Evaluating Respiratory System Mechanics.

Guidelines: British Thoracic Society, "BTS Guideline for Oxygen Use in Adults in Healthcare and Emergency Settings", Thorax 2017;72(Suppl 1) — §3.2 (hypoxaemia, type 1), §3.3 (hypercapnia, type 2), §3.4. doi:10.1136/thoraxjnl-2016-209729.

Paper III · December 2022 · Q1(b) · 5 marks · Intermediate**Outline management of severe ARDS with evidence. [5]**

Also asked as: Paper III · December 2022 · Q1(a) "Critically analyze Berlin definition of ARDS. [5]" · Paper I · December 2022 · Q6(b) "Causes of refractory hypoxemia in ARDS. [5]"

A. Berlin definition — what it says

- ARDS: **non-cardiogenic pulmonary oedema and hypoxaemia** after direct or indirect lung injury; "a syndrome of inflammation and increased permeability ... that cannot be explained by, but may coexist with, left atrial or pulmonary capillary hypertension."
- Berlin (2012) replaced the AECC (1994) definition, whose problems were **timing**, **poor inter-rater reliability of the chest radiograph** and the effect of **PEEP on the P:F ratio**.

Table 73. Berlin definition (Irwin and Rippe 9e, ch. 163; ESICM 2023, Domain 1) and the 2024 Global definition

Criterion	Berlin 2012	Global definition 2024
Timing	Acute (median onset 1–2 days; $> 90\%$ within 5 days of the insult — I&R epidemiology)	Acute onset or worsening within 1 week of a predisposing risk factor
Imaging	Bilateral opacities on CXR or CT, not explained by effusion, lobar collapse or nodules	CXR, CT or lung ultrasound (bilateral B-lines and/or consolidations)
Origin of oedema	Not fully explained by cardiac failure; may coexist with it	Not exclusively or primarily cardiogenic
Oxygenation	$\text{P:F} < 300$ on $\text{PEEP} \geq 5$ cm H_2O (ventilated patients)	$\text{P:F} \leq 300$ or $\text{SpO}_2:\text{FiO}_2 \leq 315$ ($\text{SpO}_2 \leq 97\%$)
Non-intubated	Excluded — not on positive pressure	Included on HFNO ≥ 30 L/min or NIV/CPAP ≥ 5 cm H_2O
Severity	Mild 201–300; moderate 101–200; severe ≤ 100	Same P:F bands; $\text{SpO}_2:\text{FiO}_2 > 235$ –315, > 148 –235, ≤ 148
Resource-limited	Not addressed	Neither PEEP nor minimum O_2 flow required

Critical analysis of the Berlin definition**Strengths [I&R]**

- Term "**ALI**" **abolished** → a continuum of mild, moderate and severe ARDS.
- Minimum PEEP standardises the P:F ratio (ESICM: "the patient receives at least 5").
- Severity bands were **validated**: rising category → higher mortality and longer ventilation in survivors.

- CT allowed for the imaging criterion; P:F <300 identifies patients early, before progression.

Weaknesses

- Does not establish the **cause of hypoxaemia** (alveolar damage) or prove **increased permeability**; no bedside test of permeability exists. [I&R]
- Against the histological gold standard (**diffuse alveolar damage**), clinical definitions miss some cases. [I&R]
- Chest radiograph: low inter-observer agreement (atelectasis, effusion, lower-lobe disease); "moderate-to-poor reliability". [I&R; ESICM 2023]
- P:F still depends on ventilator settings and FiO₂; oxygenation indices were historically weak outcome predictors. [I&R]
- Requires positive pressure — patients on HFNO "do not meet the Berlin definition"; unusable where ABG, CXR or ventilators are unavailable. [Global 2024; ESICM 2023]
- Broad inclusion "may dilute severity of illness among patients labeled as ARDS". [ESICM 2023]
- Stabilisation period before measuring P:F is undefined ("the length of this period remains uncertain"). [ESICM 2023]

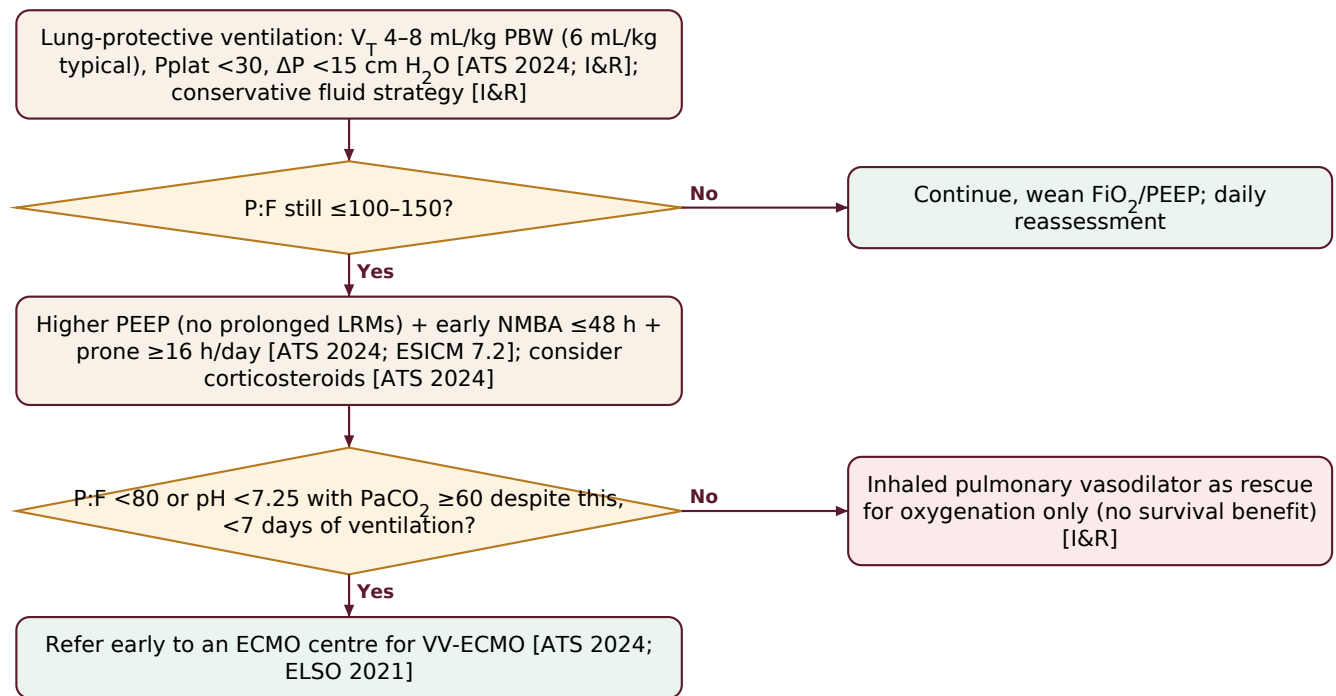
B. Management of severe ARDS (P:F ≤100) — with evidence

MANAGEMENT BASIS

ATS Clinical Practice Guideline update on ARDS (Am J Respir Crit Care Med 2024), ESICM ARDS guidelines (Intensive Care Med 2023), Surviving Sepsis Campaign 2026 (sepsis-associated ARDS). No ISCCM ARDS guideline was found (search 8 Oct 2026). ESICM Domains 8–9 (NMBA, ECLS) could not be read. Trial results and rescue therapies from Irwin and Rippe 9e, ch. 163, 166 [I&R].

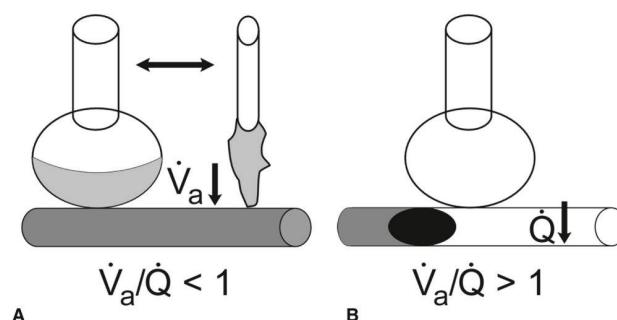
Table 74. Key recommendations for severe ARDS and the trials behind them

Intervention	Recommendation (grade)	Evidence named in I&R
Low V _T + plateau limit	V _T 4–8 mL/kg PBW, Pplat <30 cm H ₂ O [ATS 2024; strong, moderate]; 4–8 mL/kg [ESICM Rec 5.1; strong, high]	ARDSNet 6 vs 12 mL/kg: mortality 31.0% vs 39.8%
Prone position	>12 h/day in severe ARDS [ATS 2024; strong, moderate]; P:F <150, early, ≥16 consecutive h [ESICM 7.1–7.2; strong, high]	PROSEVA: 28-day HR 0.39, 90-day HR 0.44
Higher PEEP	Higher PEEP without LRMs, moderate–severe ARDS [ATS 2024; conditional, low–moderate]; ESICM 6.1 — no recommendation	Briel meta-analysis: 10% RRR in moderate–severe ARDS
Recruitment manoeuvres	Against prolonged LRMs (PEEP ≥35 cm H ₂ O >60 s) [ATS 2024; strong, moderate]; [ESICM 6.3; strong, moderate against]	LOVS: less refractory hypoxaemia, no mortality benefit
NMBA	Early (<48 h) severe ARDS (P:F ≤100), ≤48 h [ATS 2024; conditional, low]; intermittent boluses over infusion [SSC 2026; conditional, moderate]	ACURASYS ↓28/90-day mortality; ROSE (>1000 pts) no benefit
Corticosteroids	Suggest for ARDS [ATS 2024; conditional, moderate]; no agent/course specified; harm if started >2 weeks	DEXA-ARDS: more VFDs, lower 60-day mortality
VV-ECMO	Selected severe ARDS [ATS 2024; conditional, low]; when conventional ventilation fails, experienced centres [SSC 2026; conditional, low]	EOLIA 35% vs 46% (P = .09); CESAR
HFOV	Against routine use [ATS 2024; strong, high]	OSCILLATE stopped early (47% vs 35%)

Flowchart. Escalation ladder for severe ARDS

Based on ATS 2024 Recs (NMBA, PEEP, ECMO selection), ESICM 2023 Recs 7.1–7.2, Irwin and Rippe 9e, ch. 163.

- **Permissive hypercapnia** tolerated to achieve low V_T; avoid bicarbonate buffering without clear rationale; caution in raised ICP, pregnancy, active myocardial ischaemia. [I&R]
- PBW: men 50 + 0.91 (height cm – 152.4); women 45.5 + 0.91 (height cm – 152.4). [I&R]
- **Conservative fluid strategy** (FACTT): no mortality difference, but better lung function and fewer ventilator days without more non-pulmonary organ failure. [I&R]
- **Inhaled NO/prostacyclin**: improve oxygenation, no mortality benefit; reserved as rescue; iNO linked to AKI and methaemoglobinaemia. [I&R]
- Failed therapies: surfactant, β-agonists (ALTA, BALTI-2), statins (SAILS, HARP-2), ω-3 fatty acids, activated protein C. [I&R]
- Insufficient evidence for routine APRV; HFOV is clearly not first-line and is now rarely used in adults, even as salvage. [I&R]

C. Causes of refractory hypoxaemia in ARDS

Irwin and Rippe 9e, Figure 163.3 (ch. 163): A, fluid-filled and collapsed alveoli with intact blood flow — physiological shunt (V_a/Q <1); B, microthrombus obstructing flow to a ventilated alveolus — dead space (V_a/Q >1).

Table 75. Causes of refractory hypoxaemia in ARDS

Cause	Mechanism	Clue / response
Intrapulmonary shunt (flooded or collapsed alveoli)	Perfused, unventilated units; extra FiO_2 never meets shunted blood	Little rise in PaO_2 with FiO_2 ; $\text{PaO}_2 \leq 60$ on $\text{FiO}_2 \sim 1.0 \approx 40\%$ shunt (ch. 169)
Loss of hypoxic pulmonary vasoconstriction	Blood diverted to poorly ventilated regions	IV/enteral pulmonary vasodilators increase shunt (ch. 174)
Derecruitment / inadequate PEEP	Cyclic collapse, falling end-expiratory lung volume	Responds to PEEP/recruitment (if recruitable)
Overdistension from excessive PEEP	Compresses capillaries → West zone 1 dead space; ↑PVR	Rising V_D/V_T , stress index >1
Microthrombi, vasoconstriction	Dead space ("wasted ventilation")	High minute-ventilation need
RV failure / acute cor pulmonale	↑PVR → RV pressure overload; can open the foramen ovale → right-to-left shunt (ch. 174)	Echo: dilated RV, septal shift
Patient–ventilator dyssynchrony, high effort	Double triggering, P-SILI	Waveform analysis; sedation/NMBA
Barotrauma, secretions, tube problems	Pneumothorax; ↑resistance	Rising PIP – Pplat or Pplat; ultrasound, suction
Fluid overload, new VAP	More oedema/consolidation	Falling compliance, new infiltrate

PEARLS — what wins marks

- Berlin: acute, bilateral opacities (CXR/CT), not fully cardiac, P:F <300 on PEEP ≥ 5 ; mild 201–300, moderate 101–200, severe ≤ 100 .
- Berlin weaknesses: no permeability test, CXR unreliable, excludes HFNO/non-intubated, unusable in resource-poor settings → Global definition 2024 ($\text{SpO}_2:\text{FiO}_2$, LUS, HFNO ≥ 30 L/min).
- Severe ARDS core: V_T 4–8 mL/kg PBW, Pplat <30, prone >12 h (ATS) / ≥ 16 h (ESICM).
- ATS 2024: steroids (conditional, moderate), NMBA ≤ 48 h in early P:F ≤ 100 , higher PEEP without LRMs, VV-ECMO in selected (P:F <80; pH <7.25 + $\text{PaCO}_2 \geq 60$; <7 days).
- PROSEVA HR 0.39; ARDSNet 31% vs 39.8%; EOLIA 35% vs 46% (NS); HFOV harmful (OSCILLATE).
- Refractory hypoxaemia = shunt until proved otherwise; also think RV failure with PFO, derecruitment, overdistension, pneumothorax.

Textbook: Irwin and Rippe 9e, ch. 163 "Acute Respiratory Failure Due to Acute Respiratory Distress Syndrome and Pulmonary Edema": §Definition, §Radiographic Findings, §Epidemiology, §Pathophysiology (Fig. 163.3), §Management (Mechanical Ventilation and Low Tidal Volumes, Recruitment, PEEP, APRV, High Frequency Ventilation, ECMO, Prone Positioning, Fluid Management, Corticosteroids, Neuromuscular Blockade, Pulmonary Vasodilators, Surfactant, β -Agonists, Statins), Table 163.2. Ch. 166, §Heart–Lung Interaction, §Clinical Evidence (EOLIA, CESAR). Ch. 29, §Dead Space Measurements. Ch. 169, §Choice of Oxygen Delivery Device. Ch. 174 "Pulmonary Hypertension in the ICU", §Physiology of the Pulmonary Circulation and Right Ventricle.

Guidelines: American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official American Thoracic Society Clinical Practice Guideline", Am J Respir Crit Care Med 2024;209(1):24–36 — new recommendations (corticosteroids, VV-ECMO, NMBA, PEEP, recruitment manoeuvres) and carried-over 2017 recommendations. doi:10.1164/rccm.202311-2011ST. ESICM, Grasselli G, Calfee CS, Camporota L, et al., "ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies", Intensive Care Med 2023;49:727–759 — Domain 1 text; Recs 3.1, 4.1–4.3, 5.1, 6.1–6.4, 7.1–7.3 (Domains 8–9 not readable in this build). doi:10.1007/s00134-023-07050-7. Matthay MA, Arabi Y, Arroliga AC, et al., "A New Global Definition of Acute Respiratory Distress Syndrome", Am J Respir Crit Care Med 2024;209(1):37–47 (consensus definition, ungraded). doi:10.1164/rccm.202303-0558WS. Surviving Sepsis Campaign (SCCM/ESICM), Prescott H, Antonelli M, Alhazzani W, et al., "International guidelines for management of sepsis and septic shock 2026", Crit Care Med 2026 — ventilation statements C8–C11 (SCCM guideline page). doi:10.1097/CCM.0000000000007075. ELSO, Tonna JE et al., "Management of Adult Patients Supported with VV ECMO: Guideline from ELSO", ASAIO J 2021 — indications. doi:10.1097/MAT.0000000000001432.

Paper I • October 2025 • Q2(a) • 5 marks • Intermediate**Acute respiratory distress syndrome (ARDS): Its impact on the renal system. [5]**

Also asked as: Paper I • October 2025 • Q2(b) "Acute respiratory distress syndrome (ARDS): Its impact on the cardiovascular system. [5]"

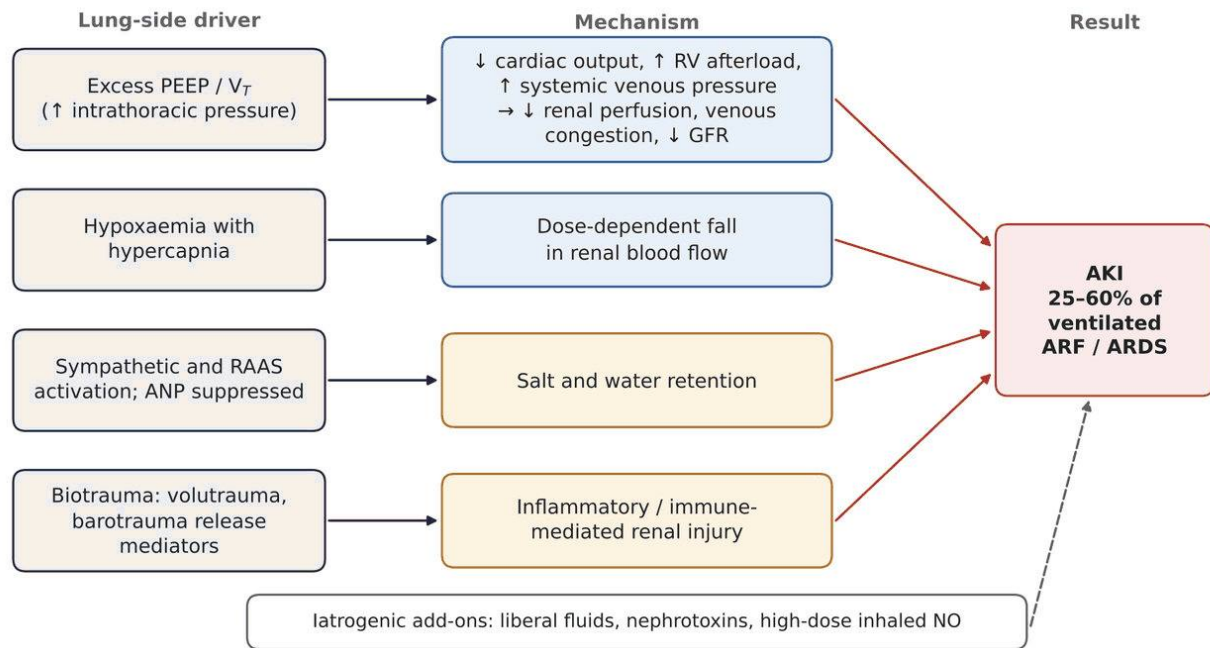
(a) Impact of ARDS on the renal system**Epidemiology** [ADQI 21]

- AKI occurs in **25–60%** of mechanically ventilated patients with ARF/ARDS; invasive ventilation is strongly associated with AKI (pooled OR **3.16**, 95% CI 2.32–4.28), which "often occurs early after institution of IMV".
- AKI is consistently and independently associated with **mortality** in this population.
- The greatest danger in ARDS is progression to **multiorgan failure**; VILI can initiate injury that leads to multiorgan dysfunction. [I&R]

Mechanisms — "multi-factorial ... haemodynamic, neurohormonal and immune-mediated" [ADQI 21]

Table 76. How ARDS and its ventilation affect the kidney (ADQI 21 consensus; I&R where tagged)

Pathway	Mechanism	Renal consequence
Haemodynamic	Excess PEEP/ V_T → ↑intrathoracic pressure → ↓cardiac output, ↑RV afterload, ↑systemic venous pressure	↓Renal perfusion, venous congestion ; ↑renal interstitial hydrostatic pressure → ↓GFR and O_2 delivery
Gas exchange	Hypoxaemia ($ SaO_2$ 83–87%) with hypercapnia	Dose-dependent fall in renal blood flow ; $ SaO_2$ 88–90% altered renal function in ARDS
Neurohormonal	Sympathetic and renin–angiotensin–aldosterone activation; suppressed ANP	Salt and water retention
Biotrauma	Volutrauma/barotrauma release pro-inflammatory mediators (TNF- α receptors, IL-6, IL-8 fall with low V_T — I&R)	Inflammatory/immune-mediated AKI
Right heart failure	ARDS pulmonary hypertension and RV failure (I&R ch. 174)	Systemic venous congestion
Iatrogenic	Liberal fluids, nephrotoxins, high-dose iNO (iNO renal impairment — I&R)	Additional AKI

ARDS / ventilation and the kidney: pathways to AKI

Schematic (drawn for these notes) — Pathways from ARDS and its ventilation to AKI: haemodynamic effects of excess PEEP or tidal volume, hypoxaemia with hypercapnia, neurohormonal activation, biotrauma, and iatrogenic add-ons; AKI occurs in 25–60% of ventilated patients with ARF/ARDS. Facts per the ADQI 21 consensus report on lung–kidney interactions (Intensive Care Med 2019); Irwin and Rippe 9e, Ch 163 where tagged.

- Reverse direction: AKI increases susceptibility to respiratory failure and may **delay weaning** (fluid overload, inflammation, immune dysfunction); clinical mechanistic data are sparse. [ADQI 21]

MANAGEMENT BASIS

ADQI 21 consensus report on lung–kidney interactions (Intensive Care Med 2019) — grades as printed; ATS 2024 / ESICM 2023 for lung-protective ventilation; I&R ch. 163 for fluid strategy evidence.

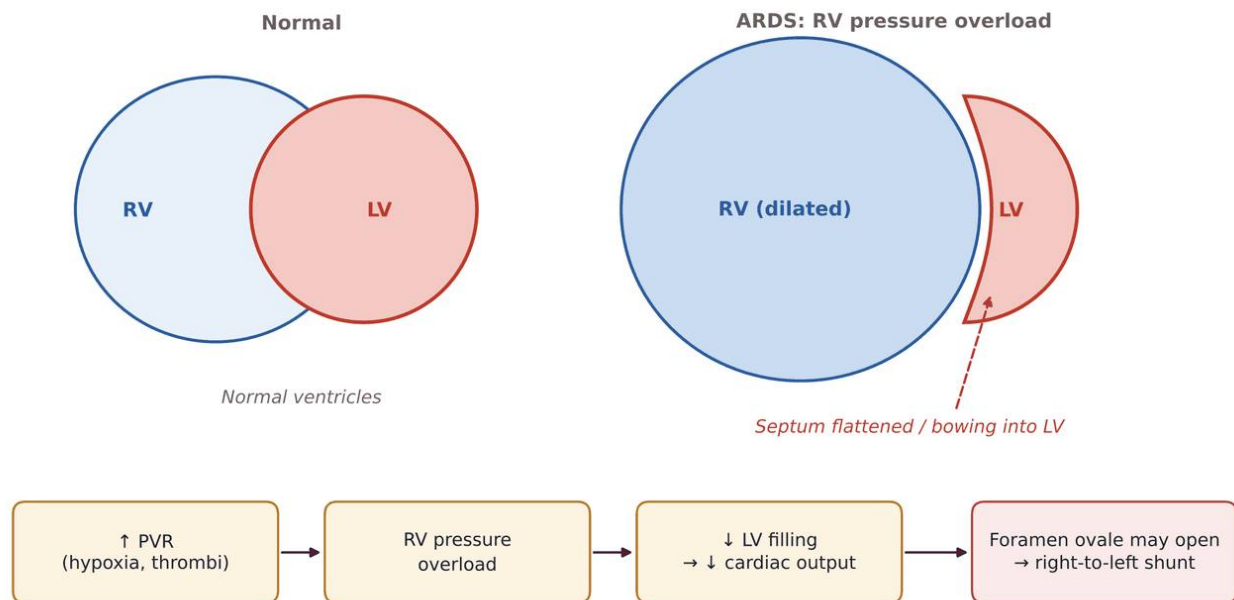
- Monitor tidal volumes and ventilation pressures and apply **lung-protective ventilation** to reduce new or worsening AKI. [ADQI 21; 1C]
- Monitor and treat **hypotension and venous congestion** in ventilated patients. [ADQI 21; 1B]
- Conservative fluid management** with selected diuretics or ultrafiltration (RRT) in ventilated patients with AKI. [ADQI 21; 2C]
- Avoid, if possible, interventions associated with AKI — liberal fluids, nephrotoxins, high-dose iNO. [ADQI 21; 2B]
- At least **daily serum creatinine** and regular urine output in severe ARF/ARDS. [ADQI 21; 1B]
- Low V_T in ARDSNet was associated with fewer AKI days [ADQI 21]; FACTT conservative strategy improved lung function without more non-pulmonary organ failure. [I&R]

(b) Impact of ARDS on the cardiovascular system

- Pulmonary hypertension** complicates 73–92% of ARDS, almost always mild–moderate (severe in only 7% in one study); magnitude tracks severity of lung injury. [I&R ch. 174]
- Causes of raised PVR: hypoxic vasoconstriction, **micro- and macrothrombi**, endothelial injury; later fibrocellular obliteration and vascular remodelling. [I&R ch. 163, 174]
- About 25% of ARDS patients have echocardiographic **RV failure** (acute cor pulmonale); incidence has fallen with low- V_T ventilation. Prognostic significance uncertain. [I&R]
- RV pressure overload → **ventricular interdependence** (septum bows into LV in diastole) → ↓LV filling and cardiac output; may open the foramen ovale → right-to-left shunt and hypoxaemia. [I&R ch. 174]
- Mechanical ventilation: ↑RV afterload (via lung volume) and ↓RV preload; in severe ARDS excessive V_T or PEEP impairs output mainly by **impeding RV ejection**. [I&R ch. 166, 174]
- PEEP up-titration in ARDS: mPAP 25 → 28 mm Hg, PVR 310 → 385 dyn·s·cm⁻⁵; hypercapnia also raises PA pressure. [I&R ch. 174]

- For the LV, raised pleural pressure reduces afterload; withdrawal of PEEP may decompensate a poor LV. [I&R ch. 166]
- Sepsis-associated ARDS: shock and fluid needs compete with the "dry lung" goal; fluid overload worsens ARDS ("iatrogenic salt water drowning"). [I&R ch. 38, 163]
- Pro-coagulant state: high rates of venous and arterial thrombosis in COVID-19 ARDS, linked to multiorgan failure and death. [I&R ch. 163]

Ventricular interdependence in ARDS with RV pressure overload (short-axis, schematic)



Schematic (drawn for these notes) — Ventricular interdependence in ARDS: raised PVR (hypoxia, thrombi) causes RV pressure overload, the septum bows into the LV, LV filling and cardiac output fall, and the foramen ovale may open with right-to-left shunt. Facts per Irwin and Rippe 9e, Ch 174 (pulmonary hypertension in ARDS) and Ch 163. Short-axis schematic, not to scale.

Table 77. Cardiovascular effects and protective measures (Irwin and Rippe 9e)

Effect	Driver	Protective measure [I&R]
Pulmonary hypertension	Hypoxia, thrombi, vasoconstriction, PEEP, hypercapnia	Limit V_T /PEEP; avoid excessive hypercapnia
Acute cor pulmonale / RV failure	High PVR + positive pressure	Prone positioning lowers $PaCO_2$ and RV afterload
Low cardiac output	RV ejection impeded; ↓ venous return	Reassess PEEP/ V_T ; dynamic preload indices
Hypoxaemia via PFO	RV pressure overload	Treat RV load
Shock + cor pulmonale	Combined failure	V-A ECMO preferred over V-V (ch. 166)

- Inhaled pulmonary vasodilators reduce PA pressure and improve oxygenation but not survival; IV/enteral pulmonary vasodilators increase intrapulmonary shunt. [I&R ch. 174]

PEARLS — what wins marks

- AKI in 25–60% of ventilated ARF/ARDS; ventilation OR 3.16 for AKI (ADQI 21).
- Kidney hit by: ↓ cardiac output + venous congestion (PEEP), hypoxaemia/hypercapnia, SNS/RAAS activation, bio-trauma, nephrotoxic co-therapy.
- ADQI 21: lung-protective ventilation (1C), treat hypotension/venous congestion (1B), daily creatinine + urine output (1B), conservative fluids (2C).
- Pulmonary hypertension in 73–92% of ARDS; RV failure ~25%.
- Positive pressure ↑ RV afterload, ↓ RV preload; ventricular interdependence ↓ LV output.
- Prone position unloads the RV; ARDS + cor pulmonale/shock → V-A ECMO.

Textbook: Irwin and Rippe 9e, ch. 163, §Pathophysiology, §Management — Low Tidal Volumes (cytokines), Fluid Management, Pulmonary Vasodilators, Statins; ch. 166, §Ventilator-Induced Lung Injury (Definition), §Heart-Lung Interaction,

§ECLS Systems (V-V ECMO); ch. 174 "Pulmonary Hypertension in the Intensive Care Unit", §Classification/Etiology (ARDS), §Physiology of the Pulmonary Circulation and RV, §Pathology, §Treatment — Mechanical Ventilation; ch. 38 "Management of Sepsis", §Fluid Therapy.

Guidelines: Acute Disease Quality Initiative, Joannidis M, Forni LG, Klein SJ, et al., "Lung-kidney interactions in critically ill patients: consensus report of the ADQI 21 Workgroup", Intensive Care Med 2019 — consensus statements and graded recommendations. doi:10.1007/s00134-019-05869-7. American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official American Thoracic Society Clinical Practice Guideline", Am J Respir Crit Care Med 2024;209(1):24–36 — new recommendations (corticosteroids, VV-ECMO, NMBA, PEEP, recruitment manoeuvres) and carried-over 2017 recommendations. doi:10.1164/rccm.202311-2011ST.

Paper III · May 2024 · Q10(a) · 5 marks · Intermediate

Various methods of setting PEEP in ARDS patients. [5]

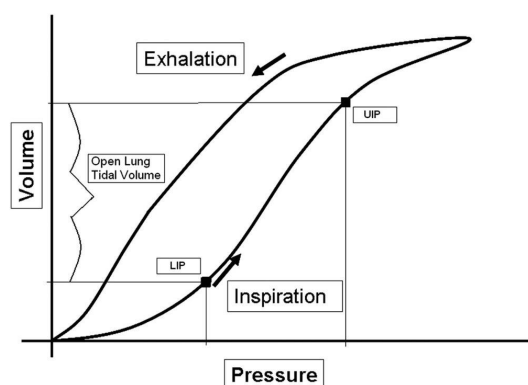
Why PEEP, and why it is controversial

- PEEP retards alveolar derecruitment, redistributes lung water, raises FRC and PaO_2 , and limits **atelectrauma**; too much PEEP causes **overdistension**, $\uparrow$ dead space and haemodynamic compromise.
- Recrutable lung varies widely between patients — PEEP set without knowing recruitability may offset its benefits.
- Irwin and Rippe: no consensus on how to choose the level — PV curves, stress index, oesophageal manometry, high-PEEP table or other methods; "the optimal level of PEEP remains elusive".

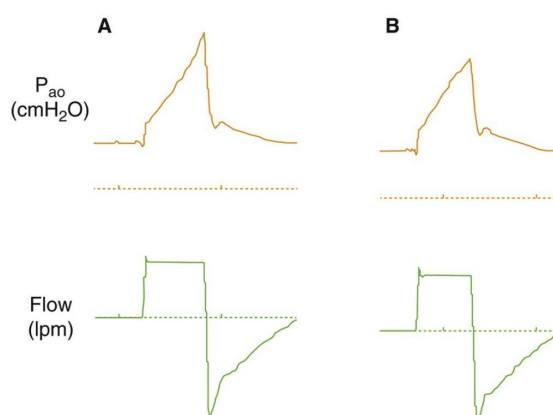
Methods of setting PEEP

Table 78. Methods of setting PEEP in ARDS (Irwin and Rippe 9e, ch. 29, 163, 166, 11)

Method	How it is done	Target	Evidence / limitation
PEEP/ FiO_2 table (lower or higher)	PEEP coupled step-wise to FiO_2 requirement	Oxygenation goal	Higher-PEEP table (Brower 2004, 549 pts): no benefit in mortality or VFDs
Least PEEP for oxygenation	Lowest PEEP giving adequate PaO_2	Adequate oxygenation	"Low PEEP" camp; may permit atelectrauma
Plateau-pressure directed (ExPress)	Raise PEEP until Pplat 28–30 cm H_2O at V_T 6 mL/kg	Pplat 28–30	No mortality gain; $\uparrow$ VFDs, organ-failure-free days, oxygenation and compliance
Open-lung (LOVS)	Recruitment manoeuvres + higher PEEP	Keep lung open	Less refractory hypoxaemia/rescue therapy; no mortality benefit
Oxygenation response	Increase PEEP and watch P:F	P:F rise ≥ 25 mm Hg	Responders had lower mortality (marker of recruitability)
Static P–V curve	Super-syringe, multiple occlusion or low-flow inflation	PEEP just above LIP ; stay below UIP	LIP/UIP hard to identify; changes not specific for collapse
Stress index	Shape of P_{aw} -time curve during constant-flow inflation	SI ≈ 1 (0.95–1.05); >1 overdistension, <1 tidal recruitment	Small studies only
Oesophageal pressure (P_L)	Pes estimates pleural pressure $\rightarrow$ transpulmonary pressure	End-expiratory P_L 0–6 cm H_2O allowed; mortality lowest near 0	EPVent-1 better oxygenation; EPVent-2 (200 pts) no difference
Best compliance / driving pressure	Choose PEEP giving highest C_{rs} / lowest ΔP	$\Delta P < 15$ cm H_2O	ΔP change correlates with mortality better than oxygenation change
Imaging — LUS, EIT, CT	Aeration score / regional collapse vs overdistension	Reaeration without overdistension	LUS fall of 8 points $\approx >600$ mL; cannot detect overdistension; EIT still in development



Irwin and Rippe 9e, Figure 29.5 (ch. 29): pressure-volume curve with lower and upper inflection points (LIP, UIP) bounding the compliant segment proposed as the "open lung" tidal volume; deflation limb shows hysteresis.



Irwin and Rippe 9e, Figure 166.12 (ch. 166): constant-flow volume assist-control — A, stress index >1 (concave-upward pressure rise, overdistension); B, stress index = 1 (linear rise, stable compliance).

Physiological notes on each approach

- **Higher PEEP benefit depends on severity:** Briel meta-analysis — 10% relative risk reduction in moderate–severe ARDS, trend to harm in mild ARDS. [I&R]
- **P–V curve:** injured lung shows an S-shaped inflation curve; low-flow P–V curves accurately predicted CT-measured recruitment (Chiumello). [I&R]
- **Transpulmonary pressure:** Pes excursions track pleural pressure well, absolute values less so; individualises PEEP when chest-wall elastance is high (chest-wall oedema, abdominal distension). [I&R]
- **Recruitment manoeuvres:** transient oxygenation gain, may cause hypotension and desaturation; benefit short-lived unless followed by adequate PEEP. [I&R]
- In **obstructive disease**, external PEEP $\approx$ 75% of auto-PEEP lowers work of triggering without worsening hyperinflation. [I&R]
- Measure total PEEP by end-expiratory occlusion; record vascular pressures at end-expiration, not by reducing PEEP. [I&R]

MANAGEMENT BASIS

ATS Clinical Practice Guideline update on ARDS (2024); ESICM ARDS guidelines (2023), Recs 6.1–6.4; Surviving Sepsis Campaign 2026 (sepsis-associated ARDS). No ISCCM document found.

- Suggest **higher PEEP without lung recruitment manoeuvres** rather than lower PEEP in moderate–severe ARDS (higher-PEEP strategies in the trials: PEEP/FiO₂ table, PEEP to a maximal safe Pplat, titration to maximal compliance). [ATS 2024; conditional, low–moderate]
- No recommendation for or against routine higher PEEP/FiO₂ titration. [ESICM 2023, Rec 6.1; no recommendation, high level of evidence of no effect]
- No recommendation for or against PEEP titration guided principally by respiratory mechanics (incl. oesophageal pressure) vs PEEP/FiO₂. [ESICM 2023, Rec 6.2; no recommendation, high level of evidence of no effect]

- Against prolonged high-pressure recruitment manoeuvres [ESICM Rec 6.3; strong, moderate]; against routine brief high-pressure manoeuvres [ESICM Rec 6.4; weak]; against prolonged LRMs (PEEP ≥ 35 cm H₂O for >60 s) [ATS 2024; strong, moderate].
- Sepsis-associated moderate–severe ARDS: higher over lower PEEP [SSC 2026; conditional, moderate]; recommend against incremental PEEP titration strategy [SSC 2026; strong, moderate].

PEARLS — what wins marks

- No single best method: PEEP/FiO₂ tables, Pplat-directed (28–30), P–V curve (above LIP), stress index (≈ 1), oesophageal P_L (near 0), best compliance/ ΔP , LUS/EIT.
- Higher PEEP helps moderate–severe ARDS (Briel 10% RRR), may harm mild ARDS.
- Oxygenation response ≥ 25 mm Hg P:F to PEEP = recruitable lung, lower mortality.
- EPVent-2: oesophageal-pressure PEEP no better than empirical high PEEP.
- ATS 2024: higher PEEP without LRMs (conditional); never prolonged LRMs (PEEP ≥ 35 >60 s; strong).
- Stress index >1 = overdistension → lower PEEP or V_T.

Textbook: Irwin and Rippe 9e, ch. 163, §Management — Recruitment, Positive End-Expiratory Pressure, Table 163.2; ch. 29, §Pressure Volume Curves (Static, Semistatic, Clinical Use; Figs. 29.5–29.6), §Stress Index, §Esophageal Pressure Monitoring, §Electrical Impedance Tomography; ch. 166, §Positive End-expiratory Pressure, §Evaluating Respiratory System Mechanics (Fig. 166.12), §VILI — Prevention; ch. 11 "Lung Ultrasonography", §Measurement of Lung Recruitment With PEEP.

Guidelines: American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official American Thoracic Society Clinical Practice Guideline", Am J Respir Crit Care Med 2024;209(1):24–36 — new recommendations (corticosteroids, VV-ECMO, NMBA, PEEP, recruitment manoeuvres) and carried-over 2017 recommendations. doi:10.1164/rccm.202311-2011ST. ESICM, Grasselli G, Calfee CS, Camporota L, et al., "ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies", Intensive Care Med 2023;49:727–759 — Domain 1 text; Recs 3.1, 4.1–4.3, 5.1, 6.1–6.4, 7.1–7.3 (Domains 8–9 not readable in this build). doi:10.1007/s00134-023-07050-7. Surviving Sepsis Campaign (SCCM/ESICM), Prescott H, Antonelli M, Alhazzani W, et al., "International guidelines for management of sepsis and septic shock 2026", Crit Care Med 2026 — ventilation statements C8–C11 (SCCM guideline page). doi:10.1097/CCM.0000000000007075.

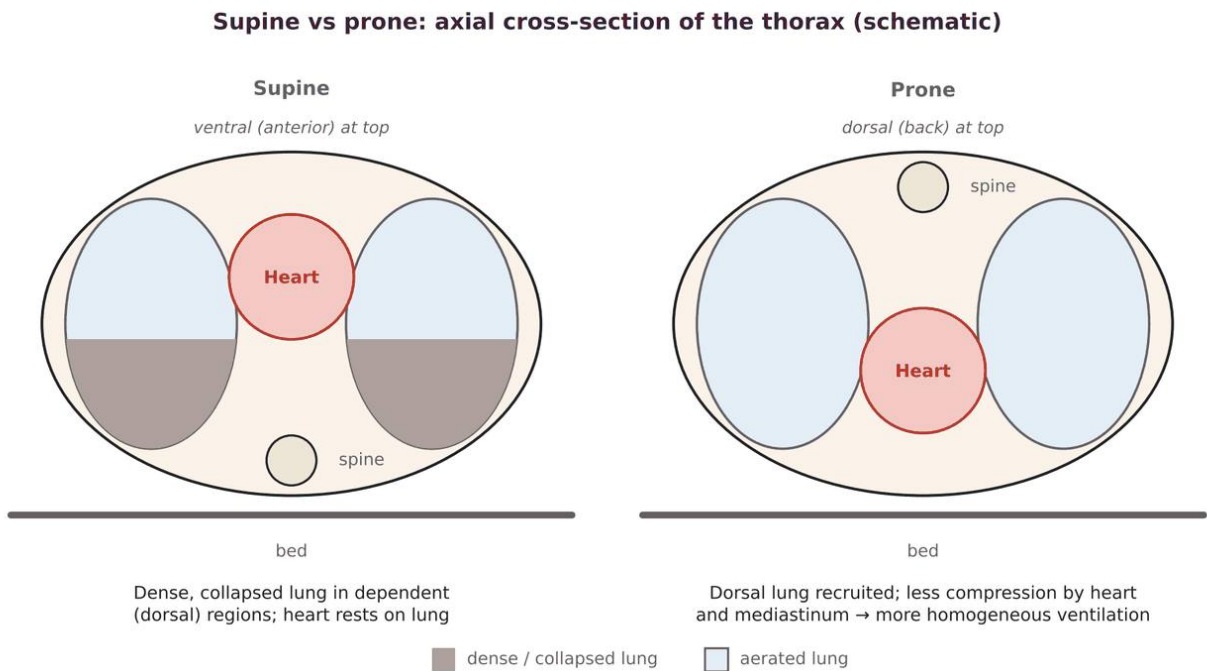
Paper II · January 2025 · Q5(b) · 6 marks · Intermediate

Describe the evidence-based merits and de-merits of proning in ARDS patients in ICU. [3+3]

Also asked as: Paper II · January 2025 · Q5(a) "Discuss the principles of prone position ventilation. [4]"

(a) Principles of prone position ventilation

- Improves oxygenation in hypoxic respiratory failure — known since the mid-1970s.
- **Recruits dorsal (previously dependent) lung** — ventilation improves in dorsal regions.
- Mechanism is **not mainly gravitational redistribution of perfusion**: prone position has limited gravitational effect on regional perfusion in normal or injured lung.
- Benefit comes from **regional changes in chest-wall mechanics** and **reduced compression of lung by the heart and mediastinum** → more homogeneous ventilation and better V/Q matching.
- In ARDS the dense, collapsed lung is concentrated in gravitationally dependent regions (CT) — the target of proning.
- Haemodynamic principle: prone positioning **reduces RV afterload**, lowers PaCO₂, improves echocardiographic RV pressure overload and can boost cardiac output.
- Contributes to lung protection — listed with dyssynchrony correction, paralysis and sedation as protective against VILI in selected ARDS.
- Survival benefit is seen when combined with **low V_T** (6 mL/kg IBW, Pplat <30) and **long sessions** (PROSEVA 16 consecutive hours).



Schematic (drawn for these notes) — Supine versus prone axial thorax: dense, collapsed lung lies in the dependent dorsal regions when supine; prone position recruits dorsal lung and reduces compression of lung by the heart and mediastinum. Facts per Irwin and Rippe 9e, Ch 163 and Ch 166 (prone positioning). Schematic, not to scale.

(b) Evidence-based merits

Table 79. Trials and data on prone positioning (Irwin and Rippe 9e, ch. 163, Table 163.2; ch. 83, 166)

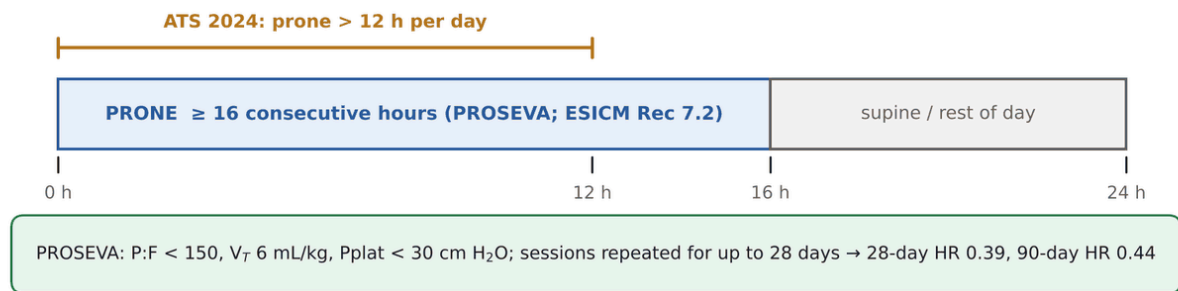
Study	Population	Result
Gattinoni 2001 (304)	ALI/ARDS, no low- V_T mandate	Better oxygenation, no mortality benefit; post hoc benefit in most severe
Guerin 2004 (791)	Hypoxaemic ARF	No 28/90-day benefit; safety concerns
Mancebo 2006 (136)	Severe ALI, prolonged prone	Mortality 43% vs 58% ($P = .12$, NS)
PROSEVA 2013 (466)	$P:F < 150$; 16 h/session for up to 28 days; V_T 6 mL/kg, $P_{plat} < 30$	28-day HR 0.39; 90-day HR 0.44
Meta-analysis	All severities	Benefit only with $V_T < 8$ mL/kg IBW
COVID-19 cohort (62 vs 199)	Severe COVID ARDS	Death HR 0.61 (0.46–0.80); higher SpO_2 and $P:F$
Awake proning (single centre)	Non-intubated COVID-19	Oxygenation sustained in 50% 1 h after resupination (≥ 3 h prone)

- Merits summarised: ↓**mortality in moderate–severe ARDS**, better oxygenation and V/Q , RV unloading, lower $PaCO_2$, lung protection, compatible with ECLS (may improve oxygenation and hospital mortality during ECLS). [I&R]
- In TBI with ARDS, a dedicated series found improved oxygenation with proning. [I&R ch. 40]

(b) Demerits and limitations

- **High reported complication rates** → experts limit proning to severe hypoxaemia, in centres with expertise. [I&R]
- Requires a **team of trained clinicians** to turn a ventilated patient. [I&R ch. 83]
- Earlier trials (without low V_T , short sessions) improved oxygenation but **not survival**; benefit absent with $V_T > 8$ mL/kg. [I&R]
- Raised ICP reported in some neurocritical-care series — caution in brain injury. [I&R ch. 40]
- Unstable or rapidly deteriorating **non-intubated** patients should not stay prone — rescue breaths and resuscitation are difficult. [I&R ch. 83]
- Monitoring artefacts: continuous glucose sensors may read wrongly during prone ventilation. [I&R ch. 136]
- Proning during ECLS needs larger trials before routine recommendation. [I&R ch. 166]

Prone-positioning session targets (per 24-h day)



Schematic (drawn for these notes) — Prone-session targets over a 24-h day: at least 16 consecutive hours (PROSEVA; ESICM 2023 Rec 7.2) against the ATS 2024 statement of more than 12 h per day; PROSEVA entry criterion P:F below 150 with VT 6 mL/kg and Pplat below 30 cm H₂O. Facts per Irwin and Rippe 9e, Ch 163 Table 163.2; ESICM 2023; ATS 2024. Time axis schematic.

MANAGEMENT BASIS

ESICM ARDS guidelines (2023), Recs 7.1–7.3; ATS ARDS guideline (2024, 2017 recommendation carried over); Surviving Sepsis Campaign 2026. No ISCCM document found.

Table 80. Guideline recommendations on prone positioning

Recommendation	Grade	Body
Prone vs supine for moderate–severe ARDS (P:F <150 mm Hg, PEEP ≥5)	Strong; high level of evidence in favour	ESICM 2023, Rec 7.1
Start early after intubation ; prolonged sessions (≥16 consecutive hours)	Strong; high level of evidence in favour	ESICM 2023, Rec 7.2
Awake prone positioning for non-intubated COVID-19 AHRF	Weak; low level of evidence in favour	ESICM 2023, Rec 7.3
Awake proning for non-COVID AHRF	No recommendation; no evidence	ESICM 2023, Rec 7.3
Prone >12 h per day in severe ARDS	Strong; moderate certainty	ATS 2024 (from 2017)
Prone >12 h daily in sepsis with moderate–severe ARDS	Conditional; moderate	SSC 2026

- A trial of proning should precede ECMO; ECMO "should not be an alternative to proning". [ELSO 2021]

PEARLS — what wins marks

- Mechanism: dorsal recruitment, chest-wall mechanics, less cardiac/mediastinal compression — **not** perfusion redistribution.
- PROSEVA: P:F <150, 16 h sessions, V_T 6 mL/kg → 28-day HR 0.39.
- ESICM: P:F <150, early, ≥16 h (strong, high); ATS: >12 h/day (strong, moderate).
- Benefit requires low V_T (<8 mL/kg IBW).
- Demerits: complications, trained team, ICP rise, not for unstable awake patients.
- Prone also unloads the RV and lowers PaCO₂.

Textbook: Irwin and Rippe 9e, ch. 163, §Management — Prone Positioning, Table 163.2; §Radiographic Findings; ch. 166, §VILI Prevention, §Heart-Lung Interaction, §Supportive Therapies during ECLS; ch. 174, §Treatment — Mechanical Ventilation; ch. 83 "COVID-19", §Management; ch. 40 "Traumatic Brain Injury", §Mechanical Ventilator Management; ch. 136, §Glucose Monitoring.

Guidelines: ESICM, Grasselli G, Calfee CS, Camporota L, et al., "ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies", *Intensive Care Med* 2023;49:727–759 — Domain 1 text; Recs 3.1, 4.1–4.3, 5.1, 6.1–6.4, 7.1–7.3 (Domains 8–9 not readable in this build). doi:10.1007/s00134-023-07050-7. American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official American Thoracic Society Clinical Practice Guideline", *Am J Respir Crit Care Med* 2024;209(1):24–36 — new recommendations (corticosteroids, VV-ECMO, NMBA, PEEP, recruitment manoeuvres) and carried-over 2017 recommendations. doi:10.1164/rccm.202311-2011ST. Surviving Sepsis Campaign (SCCM/ESICM), Prescott H, Antonelli M, Alhazzani W, et al., "International guidelines for management of sepsis and septic shock 2026", *Crit Care Med* 2026 — ventilation statements C8–C11 (SCCM guideline page). doi:10.1097/CCM.0000000000007075. ELSO, Tonna JE et al., "Management of Adult Patients Supported with VV ECMO: Guideline from ELSO", *ASAIO J* 2021.

doi:10.1097/MAT.0000000000001432.

Paper II · June 2022 · Q7(a) · 5 marks · Intermediate**Mechanism of hypoxemia. [5]****Definition**

- **Hypoxaemia:** $\text{PaO}_2 < 60 \text{ mm Hg (8 kPa)}$ or $\text{SaO}_2 < 90\%$ — the definition used by most authors. [BTS 2017, §3.2]
- Clinical estimation of hypoxaemia is **exceptionally unreliable** — measure $\text{SpO}_2/\text{PaO}_2$. [I&R]
- Arterial O_2 content: $\text{Ca} = (1.34 \times \text{Hb} \times \text{SaO}_2) + (\text{PaO}_2 \times 0.003)$. [I&R]

Mechanisms (Irwin and Rippe 9e, ch. 29, 165)**1. Hypoventilation**

- Inadequate alveolar ventilation: $\text{PaCO}_2 = (\text{VCO}_2/\text{V}_A) \times k \rightarrow \text{PaCO}_2$ rises and alveolar PO_2 falls.
- Causes: drugs, neurological impairment, muscle weakness/fatigue; all extrapulmonary failure.
- Hallmark: **normal A–a gradient** ($< 20 \text{ mm Hg}$) — the PaO_2 fall simply mirrors the PAO_2 fall; corrected by ventilation and supplemental O_2 .

2. Diffusion impairment

- Inadequate O_2 transfer across the alveolar–capillary membrane: **thickened membrane** or **high cardiac output** (short transit, e.g. sepsis).
- Hypoxaemia readily corrected by supplemental O_2 ; clinical importance in the ICU debatable.
- PaCO_2 rarely affected — CO_2 is highly soluble.

3. Ventilation–perfusion (V/Q) mismatch

- **Most common cause of hypoxaemia in the ICU.**
- Low V/Q units ($\text{V/Q} \rightarrow 0$) produce hypoxaemia; usually overcome with supplemental O_2 .
- Spectrum: $\text{V/Q} \rightarrow 0$ approaches **true shunt**; $\text{V/Q} \rightarrow \infty$ is **dead space** (raises PaCO_2 / minute-ventilation need rather than causing hypoxaemia).
- Normal regulator: **hypoxic pulmonary vasoconstriction**; impaired in ARDS and by high FiO_2 in COPD.

4. Shunt (extreme V/Q mismatch)

- Blood passes perfused, unventilated alveoli (or intracardiac right-to-left) and mixes with arterial blood without gas exchange.
- Shunt equation: $Q_s/Q_t = (\text{Cc} - \text{Ca})/(\text{Cc} - \text{Cv})$.
- **Not corrected by raising FiO_2** — shunted blood never meets alveolar gas; little effect on PaCO_2 .
- $\text{PaO}_2 \leq 60 \text{ mm Hg}$ on $\text{FiO}_2 \approx 1.0$ (non-rebreather) $\approx$ right-to-left shunt of $\sim 40\%$ of cardiac output (ch. 169).
- Examples: ARDS (flooded/collapsed alveoli), pneumonia, atelectasis distal to completely occluded airways (asthma), PFO opened by RV pressure overload.

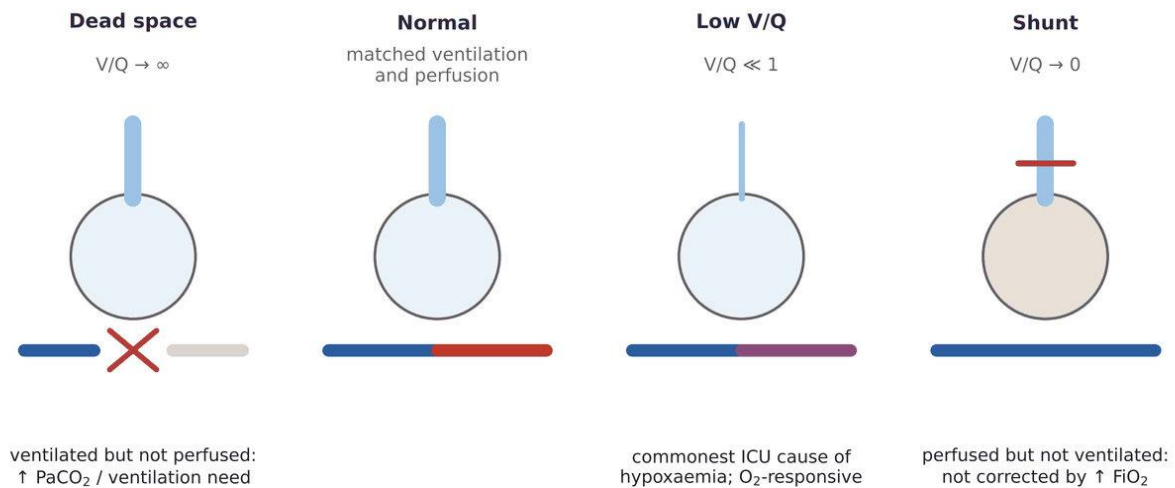
5. Low inspired PO_2 (from the alveolar gas equation)

- PAO_2 depends on FiO_2 and barometric pressure: at altitude barometric pressure falls (565 mm Hg at 8000 ft; Table 177.4) while O_2 fraction stays 20.95% $\rightarrow$ lower alveolar PO_2 ; A–a gradient normal.

Table 81. Distinguishing the mechanisms

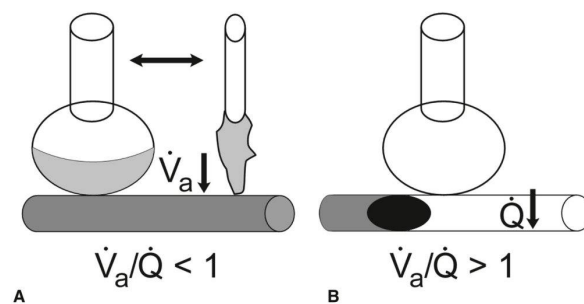
Mechanism	A–a gradient	PaCO_2	Response to $\uparrow \text{FiO}_2$	ICU example
Hypoventilation	Normal	$\uparrow$	Good	Opioid overdose, GBS, myasthenia
Low inspired PO_2	Normal	—	Good	Altitude; aircraft cabin
Diffusion impairment	$\uparrow$	Usually normal	Good	Interstitial thickening, high-output sepsis
V/Q mismatch	$\uparrow$	Normal or $\uparrow$ (dead space)	Good	COPD, asthma, pneumonia
Shunt	$\uparrow\uparrow$	Normal (little effect)	Poor	ARDS, lobar collapse, intracardiac shunt

Ventilation-perfusion spectrum (one alveolar unit each)



Capillary: blue = mixed venous blood in; red = fully oxygenated; purple = incompletely oxygenated; light-blue tube = airway

Schematic (drawn for these notes) — Ventilation-perfusion spectrum for one alveolar unit: dead space ($V/Q \rightarrow \infty$), normal, low V/Q and shunt ($V/Q \rightarrow 0$), with the response of each to raised FiO_2 . Facts per Irwin and Rippe 9e, Ch 29 and Ch 165 (mechanisms of hypoxaemia). Not to scale.



Irwin and Rippe 9e, Figure 163.3 (ch. 163): A, shunt — fluid-filled and collapsed alveoli still perfused ($\dot{V}_a/\dot{Q} < 1$); B, dead space — microthrombus stops flow to a ventilated alveolus ($\dot{V}_a/\dot{Q} > 1$).

WORKED EXAMPLE — A-a gradient (alveolar gas equation, Irwin and Rippe 9e, ch. 29)

$\text{PAO}_2 = (\text{P}_{\text{atm}} - \text{P}_{\text{H}_2\text{O}}) \times \text{FiO}_2 - \text{PaCO}_2/\text{RQ}$, with P_{atm} 760, $\text{P}_{\text{H}_2\text{O}}$ 47 mm Hg, $\text{RQ} \approx 0.8$ (the e-book prints the PaCO_2/RQ term with a "+"; it is subtracted).

Case 1 — opioid overdose on room air: PaCO_2 70, PaO_2 50 mm Hg. $\text{PAO}_2 = 713 \times 0.21 - 70/0.8 = 149.7 - 87.5 = 62.2$; $\text{A-a} = 62.2 - 50 = 12$ mm Hg → normal → pure hypoventilation.

Case 2 — pneumonia on room air: PaCO_2 32, PaO_2 55 mm Hg. $\text{PAO}_2 = 149.7 - 40 = 109.7$; $\text{A-a} = 54.7$ mm Hg → V/Q mismatch/shunt.

If PaO_2 stays ≤ 60 mm Hg on $\text{FiO}_2 \approx 1.0$ → shunt $\approx 40\%$ of cardiac output.

Applied points

- Hypercapnia with A-a gradient 20–30 mm Hg → mixed pulmonary + extrapulmonary; >30 → primary lung disease. [I&R]
- Severe COPD with rising PaCO_2 may narrow the A-a gradient to normal — a normal gradient does not exclude COPD. [I&R]
- High FiO_2 in COPD worsens hypercapnia mainly by releasing hypoxic vasoconstriction ($\uparrow$ dead space), plus Haldane effect and mild $\downarrow$ drive. [I&R]

PEARLS — what wins marks

- Five mechanisms: hypoventilation, low inspired PO_2 , diffusion impairment, V/Q mismatch, shunt.
- Commonest in ICU = V/Q mismatch; the one that does not respond to O_2 = shunt.
- Normal A–a gradient = hypoventilation or low inspired PO_2 .
- $\text{PaO}_2 \leq 60$ on $\text{FiO}_2 \sim 1.0 \rightarrow \sim 40\%$ shunt.
- Diffusion defect rarely raises PaCO_2 (CO_2 highly soluble).
- A–a gradient: $\text{PAO}_2 = 713 \times \text{FiO}_2 - \text{PaCO}_2/0.8$ at sea level.

Textbook: Irwin and Rippe 9e, ch. 29, §Gas Exchange — Basic Physics of Gas Exchange (hypoventilation, diffusion, V–Q mismatch, shunt equation, O_2 content and alveolar gas equations); ch. 165, Key Points, §Diagnosis, §Differential Diagnosis; ch. 163, §Pathophysiology, Fig. 163.3; ch. 169, §Choice of Oxygen Delivery Device; ch. 173, §Management — Oxygen; ch. 174, §Physiology of the RV; ch. 177 "Gas Embolism Syndromes", Table 177.4; ch. 27, §Pulse Oximetry.

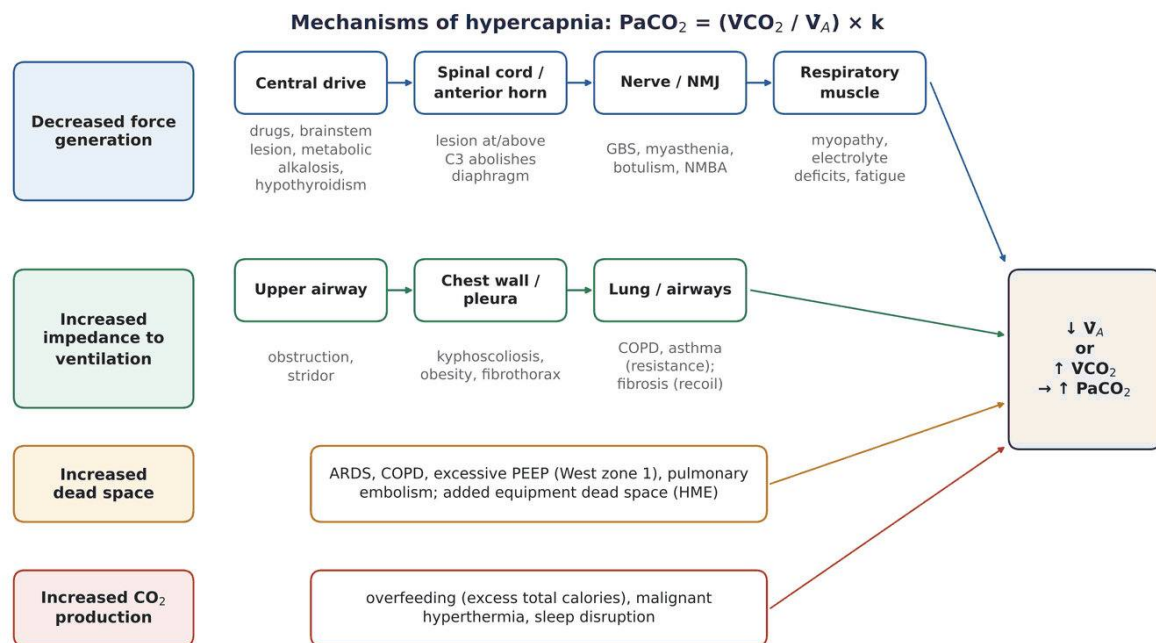
Guidelines: British Thoracic Society, "BTS Guideline for Oxygen Use in Adults in Healthcare and Emergency Settings", Thorax 2017;72(Suppl 1), §3.2. doi:10.1136/thoraxjnl-2016-209729.

Paper II • October 2023 • Q6 • 10 marks • Long**Describe about approach to a patient with hypercarbia. [10]****1. Definition**

- **Hypercapnia:** PaCO_2 above the normal range of 4.6–6.1 kPa (34–46 mm Hg); such patients have **type 2 respiratory failure** even if SpO_2 is normal. [BTS 2017, §3.3]
- Acidosis = $\text{pH} < 7.35$. [BTS 2017, §3.4]
- Governing equation: $\text{PaCO}_2 = (\text{VCO}_2/\text{V}_A) \times k$ — PaCO_2 rises when alveolar ventilation falls or CO_2 production rises. [I&R]
- Alveolar ventilation falls with low minute ventilation or a high **dead-space fraction** (V_D/V_T). [I&R]

2. Mechanisms

- **Decreased force generation** (extrapulmonary, ch. 165):
 - Reduced central drive — drugs, structural brainstem lesion, metabolic alkalosis, hypothyroidism, primary alveolar hypoventilation, central sleep apnoea.
 - Impaired transmission — spinal cord (lesion at/above C3 abolishes diaphragm and intercostals), anterior horn cell, neuropathy, neuromuscular junction.
 - Respiratory muscle weakness or fatigue — myopathies, electrolyte deficits.
- **Increased impedance to ventilation:**
 - Pulmonary — airflow resistance (COPD, asthma), elastic recoil (interstitial fibrosis).
 - Extrapulmonary — chest wall/pleura (kyphoscoliosis, obesity hypoventilation, fibrothorax) or upper airway obstruction.
- **Increased dead space:** ARDS, COPD, excessive PEEP (West zone 1), pulmonary embolism; added equipment dead space (heat–moisture exchanger).
- **Increased CO_2 production:** overfeeding (excess total calories, not carbohydrate proportion), malignant hyperthermia (CO_2 production resistant to $\uparrow$ minute ventilation), sleep disruption ($\uparrow \text{VCO}_2$).
- **Iatrogenic:** high-concentration O_2 in COPD ($\downarrow$ hypoxic vasoconstriction $\rightarrow \uparrow$ dead space, Haldane effect, mild $\downarrow$ drive); **permissive hypercapnia** during lung-protective ventilation.



Schematic (drawn for these notes) — Mechanisms of hypercapnia: $\text{PaCO}_2 = (\text{VCO}_2 / \text{V}_A) \times k$. Decreased force generation along the neuromuscular chain, increased impedance to ventilation, increased dead space and increased CO₂ production each lower alveolar ventilation or raise CO₂ output. Facts per Irwin and Rippe 9e, Ch 165 (Tables 165.1–165.4) and Ch 29.

Table 82. Causes of hypercapnic respiratory failure by compartment (Irwin and Rippe 9e, Tables 165.1–165.4)

Compartment	Examples	Pointer
CNS (drive)	Sedative/opioid overdose, myxoedema, metabolic alkalosis, brainstem tumour/infection, Ondine curse, central sleep apnoea	Depressed consciousness, pupils, needle marks; normal MIP
Spinal cord / anterior horn	Trauma (C3, C5–C6), myelitis, tetanus, strychnine, ALS, polio	Level of injury; spasms (tetanus)
Peripheral nerve / NMJ	GBS, CIP, phrenic palsy, diphtheria, tick paralysis, porphyria, myasthenia, Lambert–Eaton, botulism, organophosphates, prolonged NMBA	Areflexia, ptosis, bulbar weakness, orthopnoea
Muscle	Dystrophies, myositis, periodic paralysis, ↓PO ₄ , ↓/↑Mg, ↓K, NMBA–steroid myopathy	Proximal weakness, raised CK, weaning failure
Chest wall / pleura	Kyphoscoliosis (curve ≥120°), obesity hypoventilation, flail chest, fibrothorax, ankylosing spondylitis	Deformity, restriction on PFTs
Upper airway	Epiglottitis, laryngeal oedema (anaphylaxis, post-extubation), foreign body, vocal cord palsy, tumour, OSA	Stridor, flow–volume loop
Lung / airways	COPD, asthma, fibrosis, ARDS (dead space)	A–a gradient >30 mm Hg

3. Immediate assessment and stabilisation

- Suspect hypercapnia with anxiety, dyspnoea, lethargy, headache, **asterixis**, myoclonus, seizures, delirium or coma; acute PaCO_2 75–80 mm Hg is tolerated before consciousness falls, chronic retainers up to 90–100. [I&R]
- ABC; assess consciousness, airway protection, secretions, haemodynamics — these decide NIV vs intubation (Table 167.4). [I&R]
- Controlled oxygen:** start 0.5–2 L/min nasal cannula or Venturi 0.24–0.28; titrate to $\text{PaO}_2 \geq 55$ mm Hg with ABG every 30 min for 1–2 h; expect PaCO_2 to rise 5–10 mm Hg. [I&R]
- In COPD exacerbation target SpO_2 **88–92%**, avoiding hyperoxaemia (indiscriminate O_2 ↑ mortality and intubation). [I&R ch. 173]
- Never stop O_2 abruptly** when CO_2 narcosis is possible — PaO_2 falls below the pre-oxygen level. [I&R]
- Do not delay ventilatory support while waiting for blood gases if work of breathing is clearly excessive. [I&R ch. 167]

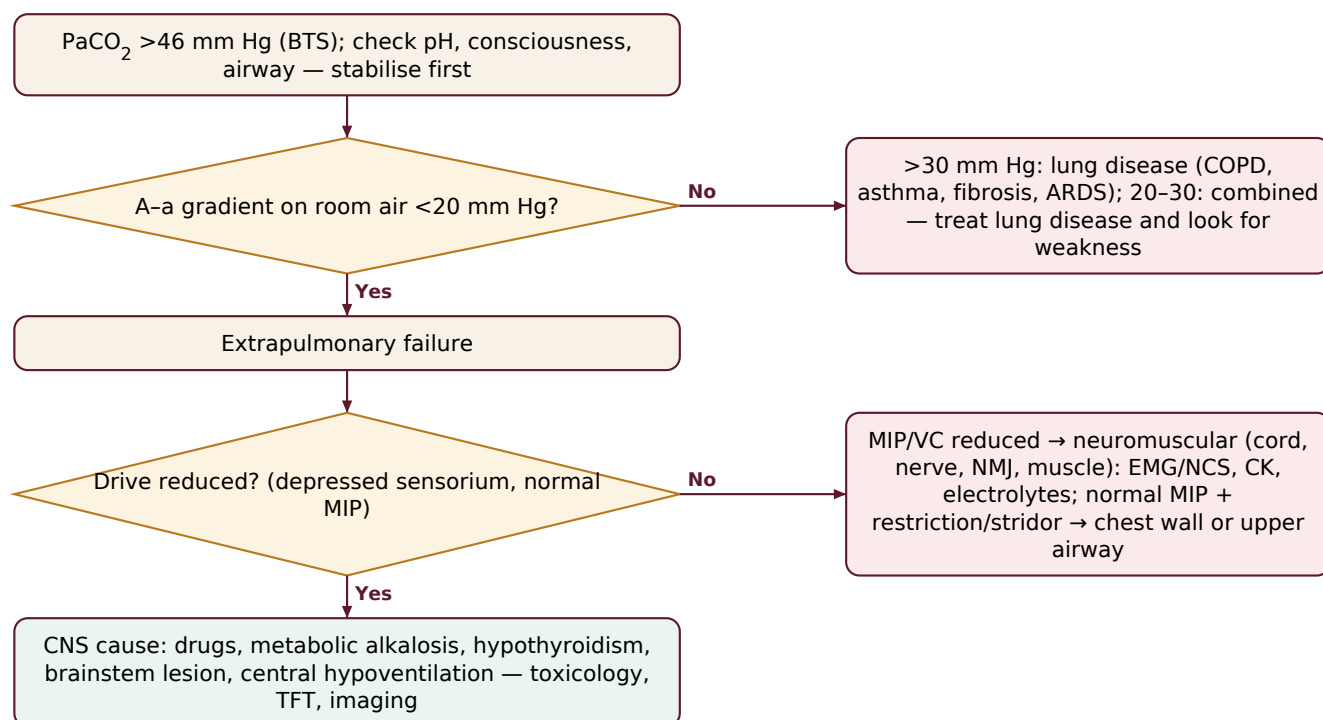
4. Focused history and examination

- Drugs/toxins (sedatives, opioids, aminoglycosides, organophosphates), recent surgery or steroids (myasthenic crisis), shellfish ingestion, wound (botulism, tetanus), tick exposure. [I&R]
- Pattern of breathing: rising RR with falling V_T , **abdominal paradox** (diaphragm failure), respiratory alternans (fatigue). [I&R]
- **Orthopnoea** with supine paradox → bilateral diaphragmatic paralysis; dyspnoea upright → C5–C6 quadriplegia. [I&R]
- Bulbar signs, ptosis, fatigability, fasciculations, areflexia, myxoedema, kyphoscoliosis, massive obesity, stridor or wheeze. [I&R]
- Sleep history (snoring, hypersomnolence) — OSA; 10–20% of OSA patients have awake hypercapnia (often with COPD or obesity). [I&R]

Table 83. Investigations in hypercapnia (Irwin and Rippe 9e, ch. 165, 27, 29)

Test	Finding	Interpretation
ABG on room air + A–a gradient	<20 / 20–30 / >30 mm Hg	Extrapulmonary / combined / primary lung disease
MIP, MEP	MIP less negative than –30 cm H ₂ O; MEP <40	Muscle weakness causing hypercapnia; weak cough
Vital capacity	<1 L or <15 mL/kg (<20 mL/kg in MG = warning)	Neuromuscular failure (less sensitive than MIP)
Electrolytes, Ca, PO ₄ , Mg, K; TSH; CK	Low PO ₄ /Mg/K; hypothyroid; ↑CK	Reversible muscle causes
Toxicology; RBC cholinesterase	Drug/organophosphate	Specific antidote
CT/MRI brain or spine, CSF	Brainstem/cord lesion; ↑protein without cells (GBS)	Central or cord cause
EMG/NCS, repetitive stimulation, AChR antibodies	Demyelination (GBS), axonal (CIP), decrement (MG), increment (LEMS)	Neuromuscular diagnosis
Fluoroscopy (sniff), Pdi, phrenic studies	Absent/paradoxical motion; low Pdi	Diaphragm paralysis
Flow–volume loop, laryngoscopy	Fixed/variable, intra/extrathoracic	Upper airway obstruction
Polysomnography	Apnoeas with/without effort	OSA vs central apnoea
Capnography; transcutaneous PCO ₂	Trend only	Not a substitute for PaCO ₂

Flowchart. Diagnostic approach to a patient with hypercapnia



Drawn from Irwin and Rippe 9e, ch. 165 (§Diagnosis, §Differential Diagnosis, Tables 165.1–165.4); threshold from BTS 2017 §3.3.

5. Management

MANAGEMENT BASIS

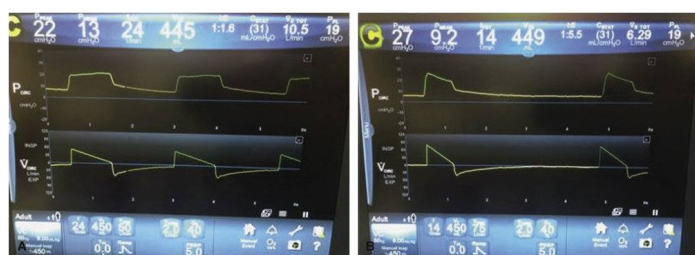
ATS Clinical Practice Guideline on noninvasive respiratory support (Am J Respir Crit Care Med 2026); BTS oxygen guideline (Thorax 2017); ELSO VV-ECMO guideline (2021) for extracorporeal support. Oxygen titration, NIV selection, ventilator settings and cause-specific therapy from Irwin and Rippe 9e, ch. 165, 167, 169, 173, 166 [I&R]. No ISCCM document was searched separately for this topic; the ISCCM NIV guideline (2020) is registered as NOT READ (root register).

Non-invasive ventilation

- **NIV via facemask** over standard oxygen for acute hypercapnic respiratory failure. [ATS 2026; strong, moderate]
- HFNC as an alternative to NIV for hypercapnic failure with **mild acidaemia (pH >7.25)**, provided monitoring and prompt escalation to NIV are available. [ATS 2026; conditional, low]
- Start NIV if $\text{PCO}_2 > 6 \text{ kPa}$ (45 mm Hg) and $\text{pH} < 7.35$. [BTS 2017, section G]
- RCT selection criteria: dyspnoea, $\text{RR} > 24/\text{min}$, accessory muscles/abdominal paradox, **pH < 7.35 but > 7.10, $\text{PaCO}_2 > 45$ and < 98 mm Hg**. [I&R, Table 167.3]
- Contraindications: arrest, haemodynamic instability, severe upper GI bleed, inability to protect airway, excessive secretions, facial trauma/burns, agitation, undrained pneumothorax, multiorgan failure. [I&R, Table 167.4]
- Hypercapnic coma (GCS < 8) is **not** an absolute contraindication — success equals non-comatose patients. [I&R]
- **1–2-hour checkpoint:** rising pH and improving mentation predict success; if not improving, intubate promptly. NIV failure ~20% in trials. [I&R]

Invasive ventilation [I&R ch. 173, 166]

- Indications: reduced consciousness, uncontrolled secretions, need for sedation/controlled ventilation, worsening hypoxaemia or acidosis despite O_2/NIV , haemodynamic instability.
- Obstructive disease: limit **minute ventilation (<10 L/min)**, prolong expiration, V_T 5–8 mL/kg, assist-control initially; aim for the patient's baseline PaCO_2 , not normal.
- Monitor auto-PEEP and Pplat; external PEEP $\approx 75\%$ of auto-PEEP eases triggering; hypotension after intubation from hyperinflation — fluids and reduce rate/ V_T .
- Neuromuscular/chest-wall failure: normal-lung settings; chest-wall restriction — limited V_T , minimum PEEP.



Irwin and Rippe 9e, Figure 173.1 (ch. 173): reducing rate from 24 to 14/min and raising inspiratory flow from 50 to 75 L/min changes I:E from 1:1.6 to 1:5.5; in A expired volume does not return to zero — air trapping and intrinsic PEEP.

Cause-specific therapy [I&R, Tables 165.1–165.5]

- Hypothyroidism — cautious thyroid replacement; electrolyte deficits (PO_4 , Mg, K) — replete.
- GBS — plasmapheresis or IVIG (effective within 4 weeks of onset); myasthenic crisis — plasmapheresis; organophosphates — atropine, pralidoxime; botulism — antitoxin, debridement.
- Drug-induced neuromuscular block — stop the drug; IV calcium gluconate may shorten recovery.
- Upper airway obstruction — establish an airway first; OSA — nasal CPAP/bilevel; kyphoscoliosis — NIV; high cervical cord with intact phrenics — phrenic pacing.
- Overfeeding — reduce total calories; check for excessive ventilator/equipment dead space.

Extracorporeal CO_2 removal / ECMO

- V-V ECCO₂R: ~1 L/min circuit flow removes all metabolic CO_2 ; may suit COPD exacerbation or status asthmaticus when its risks (mainly bleeding) are outweighed; case series show early extubation and ambulation. [I&R]
- VV ECMO indicated for hypercapnic respiratory failure with **pH < 7.25** despite optimal conventional ventilation ($\text{PaCO}_2 \geq 60 \text{ mm Hg}$ cited); ECMO also listed for severe asthma. [ELSO 2021]

- **Permissive hypercapnia** in ARDS: tolerated with sedation; avoid bicarbonate buffering; caution in intracranial hypertension, pregnancy, myocardial ischaemia. [I&R]

6. Consequences of hypercapnia to anticipate

- $\uparrow \text{PCO}_2 \rightarrow$ cerebral arterial vasodilation $\rightarrow \uparrow \text{ICP}$ (vicious cycle with reduced drive); avoid permissive hypercapnia without ICP monitoring after drowning-related brain injury. [I&R ch. 155, 171]
- Raises pulmonary artery pressure ($\pm$ PVR, $\downarrow$ RV ejection fraction). [I&R ch. 174]
- With hypoxaemia, reduces renal blood flow dose-dependently. [ADQI 21]

PEARLS — what wins marks

- Hypercapnia = $\text{PaCO}_2 > 46$ mm Hg (BTS); $\text{PaCO}_2 = \text{VCO}_2 / \text{V}_A \times k$.
- Four mechanisms: $\downarrow$ drive/force, $\uparrow$ impedance, $\uparrow$ dead space, $\uparrow \text{CO}_2$ production.
- A–a gradient < 20 mm Hg + hypercapnia = extrapulmonary; > 30 = lung disease.
- MIP less negative than -30 cm H_2O is the best predictor of hypercapnia from weakness.
- Controlled O_2 : Venturi 24–28%, $\text{PaO}_2 \geq 55$, SpO_2 88–92% in COPD; never stop O_2 abruptly.
- NIV facemask: strong, moderate (ATS 2026); pH $< 7.35 > 7.10$; reassess at 1–2 h.
- Ventilating obstruction: minute ventilation < 10 L/min, long expiration, target baseline PaCO_2 .

Textbook: Irwin and Rippe 9e, ch. 165 "Extrapulmonary Causes of Respiratory Failure": Key Points, §Pathophysiology (Tables 165.1–165.4), §Diagnosis (Clinical Features of Hypercapnia, Decrease of Normal Force Generation, Increased Impedance), §Differential Diagnosis, §Treatment (Table 165.5). Ch. 29, §Basic Physics of Gas Exchange, §Dead Space. Ch. 167, §Selection Guidelines (Tables 167.3–167.4). Ch. 169, §Choice of Oxygen Delivery Device. Ch. 173, §Severity Scoring, §Management (Oxygen, Ventilatory Support, NIV, Invasive MV, Ventilator Settings, Fig. 173.1). Ch. 166, §Ventilator Settings according to Disease Type, §ECLS Systems (ECCO2R), §Clinical Evidence. Ch. 168, §Managing Discontinuation Failure. Ch. 185, §Malignant Hyperthermia. Ch. 155, §Elevated ICP. Ch. 174, §Treatment — Mechanical Ventilation.

Guidelines: American Thoracic Society, Goel NN, Ferreyro BL, Pitre T, et al., "Noninvasive respiratory support for adult patients with acute respiratory failure: an official ATS Clinical Practice Guideline", *Am J Respir Crit Care Med* 2026;212(9):2135–2158 — acute hypercapnic respiratory failure recommendations. doi:10.1093/ajrccm/aamag302. British Thoracic Society, "BTS Guideline for Oxygen Use in Adults in Healthcare and Emergency Settings", *Thorax* 2017;72(Suppl 1) — §3.3, §3.4 and recommendation section G (NIV). doi:10.1136/thoraxjnl-2016-209729. Extracorporeal Life Support Organization, Tonna JE, Abrams D, Brodie D, et al., "Management of Adult Patients Supported with Venovenous Extracorporeal Membrane Oxygenation (VV ECMO): Guideline from the ELSO", *ASAIO J* 2021;601–610 — indications, contraindications, configurations, rest settings, transfer. doi:10.1097/MAT.0000000000001432 (also.org PDF). Acute Disease Quality Initiative (ADQI 21), Joannidis M et al., *Intensive Care Med* 2019. doi:10.1007/s00134-019-05869-7.

Paper III · January 2025 · Q5(a) · 5 marks · Intermediate

What are the various types of "ventilator-induced lung injury" (VILI) in patients on prolonged invasive mechanical ventilation? [5]

Definition

- **VILI:** functional and structural lung alterations that occur as a **direct consequence of mechanical ventilation**.
- Usually perpetuates and worsens injury in damaged lungs, but can **initiate injury in healthy lungs** and lead to **multiorgan dysfunction**.
- Main drivers: excessive mechanical **stress and strain**; the effective distending pressure is the **transpulmonary pressure** ($P_{\text{alv}} - P_{\text{pl}}$).

Types of VILI

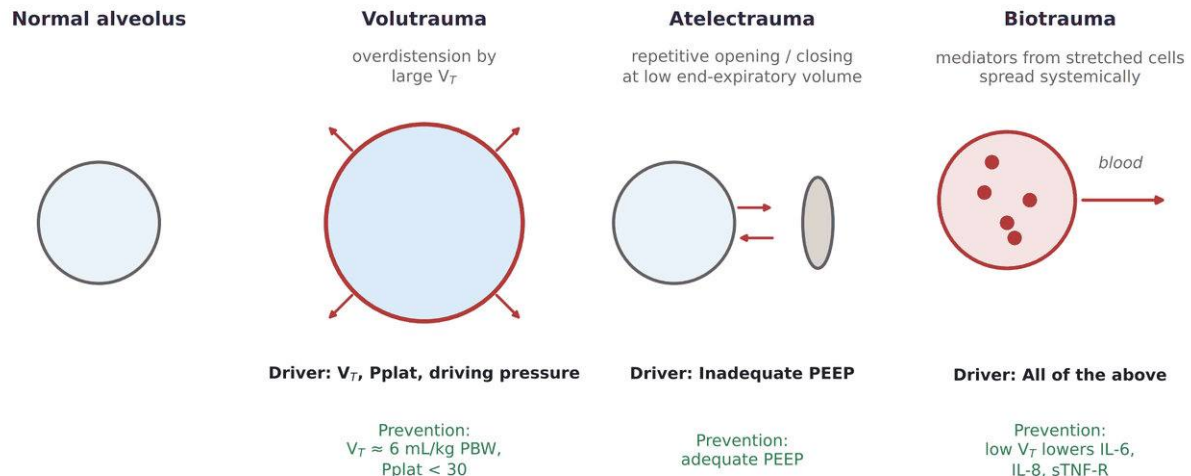
Table 84. Types of ventilator-induced lung injury (Irwin and Rippe 9e, ch. 166, 29, 163)

Type	Mechanism	Ventilator determinant	Prevention
Barotrauma (macroscopic)	Extra-alveolar air: pneumothorax, pneumomediastinum, pneumoperitoneum, subcutaneous emphysema, cystic spaces	Peak pressure, PEEP, V_T , minute ventilation, dyssynchrony (association controversial)	Limit Pplat; in obstruction, avoid hyperinflation
Volutrauma	Overdistension of the "baby lung" by large V_T	V_T , Pplat, driving pressure	$V_T \approx 6$ mL/kg PBW, Pplat <30
Atelectrauma	Repetitive opening/closing of airways and alveoli at low end-expiratory volume	Inadequate PEEP	Adequate PEEP
Biotrauma	Mediators from stretched cells activate epithelium, endothelium and leucocytes → systemic spread	All of the above	Low V_T lowers IL-6, IL-8, sTNF-R
P-SILI (effort-dependent)	Strong efforts → large pleural swings in dependent lung → overstretch, tidal recruitment	High drive, dyssynchrony	Monitor effort; PEEP; sedation, NMBA, prone
Energy (mechanical power)	Stress, strain, rate and strain-rate combined (J/min)	High RR, V_T , pressures	Limit power (unproven)
Secondary factors	Temperature, respiratory acidosis, pulmonary vascular pressures	—	—

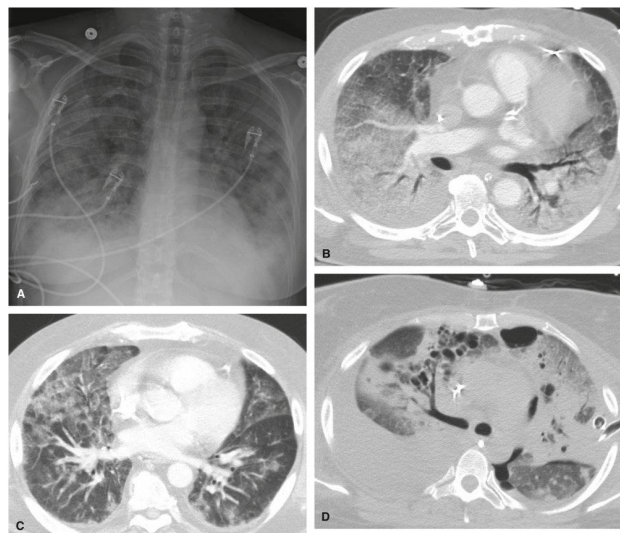
Pathophysiology (step by step)

- ARDS lung is heterogeneous — dependent consolidation with small aerated "**baby lung**"; V_T goes preferentially to compliant regions → overdistension of remaining normal lung.
- **Microscopic injury**: disrupted alveolar–capillary barrier, impaired surfactant, distal airway injury, inflammatory mediator release, hyaline membranes, pulmonary oedema.
- Mechanical ventilation itself alters surfactant structure and biophysics.
- Low V_T in the ARDS Network trial reduced soluble TNF- α receptor I and caused a greater fall in IL-6 and IL-8 — evidence that biotrauma links VILI to multiorgan failure.
- P-SILI: lung injury appears proportional to the magnitude of effort.
- **VIDD** (diaphragm counterpart): over-assistance myotrauma (atrophy), under-assistance, eccentric contractions in expiration, longitudinal atrophy with high PEEP; mitochondrial dysfunction and proteolysis.

Microscopic mechanisms of ventilator-induced lung injury (alveolar view)



Schematic (drawn for these notes) — Microscopic mechanisms of ventilator-induced lung injury: volutrauma (overdistension by large V_T), atelectrauma (repetitive opening and closing at low end-expiratory volume) and biotrauma (mediators from stretched cells spread systemically), with their ventilator drivers and prevention. Facts per Irwin and Rippe 9e, Ch 166 §Ventilator-Induced Lung Injury; Ch 29; Ch 163.



Irwin and Rippe 9e, Figure 163.2 (ch. 163): ARDS — A, chest radiograph; B–C, CT with patchy consolidation and ground glass; D, severe disease with dense consolidation, effusion and sequelae of barotrauma (pneumatoceles, persistent pneumothorax despite two chest tubes).

Barotrauma — recognition

- Air dissects along the bronchovascular interstitium (parenchymal cysts, perivascular halos) → mediastinal and subcutaneous emphysema → pneumothorax. [I&R ch. 172]
- Any pneumothorax on ventilation must be presumed to be **under tension** — immediate tube thoracostomy; ultrasound detects it rapidly. [I&R ch. 172]

Prevention

MANAGEMENT BASIS

ESICM ARDS guidelines (2023) Rec 5.1 and ATS ARDS guideline (2024) for tidal volume and plateau pressure; remaining measures from Irwin and Rippe 9e, ch. 166, 29, 163 [I&R].

- Low tidal volume **4–8 mL/kg PBW**. [ESICM 2023, Rec 5.1; strong]
- Limit V_T (4–8 mL/kg PBW) and **Pplat < 30 cm H₂O**. [ATS 2024; strong, moderate]

- $V_T \approx 6$ mL/kg PBW with $P_{plat} < 30$ reduced ARDS mortality by 22% — standard of care; higher P_{plat} acceptable with stiff chest wall. [I&R]
- Keep $\Delta P < 15$ cm H_2O (≤ 14 had better outcomes); ΔP change correlates with mortality better than oxygenation change. [I&R]
- Adequate PEEP against atelectrauma; oesophageal pressure gives true transpulmonary pressure (large trial negative). [I&R]
- Monitor drive and effort routinely: $P_{0.1}$, end-expiratory occlusion (up to 5 s) ΔP_{aw} , diaphragm thickening fraction on ultrasound; P_{es} -time product is the gold standard. [I&R]
- Correct dyssynchrony; prone positioning, NMBA and adequate sedation in selected ARDS. [I&R]
- Diaphragm protection: modest inspiratory effort, synchronous cycling, avoid post-inspiratory effort; if lung and diaphragm goals conflict, **protect the lung**. [I&R]
- Accept permissive hypercapnia (avoid in intracranial hypertension, perhaps pregnancy). [I&R]

PEARLS — what wins marks

- VILI = macroscopic (barotrauma) + microscopic (volutrauma, atelectrauma, biotrauma) + P-SILI.
- Transpulmonary pressure, not airway pressure, is the true distending pressure.
- V_T 4–8 (≈ 6) mL/kg PBW, $P_{plat} < 30$, $\Delta P < 15$ cm H_2O .
- Biotrauma explains multiorgan failure — low V_T lowers IL-6/IL-8.
- Mechanical power (J/min) = $0.098 \times RR \times V_T \times [P_{peak} - \frac{1}{2}(P_{plat} - PEEP)]$.
- Pneumothorax on ventilation = tension until proved otherwise.

Textbook: Irwin and Rippe 9e, ch. 166 "Invasive Mechanical Ventilation and Extracorporeal Life Support", §Ventilator-Induced Lung Injury and Diaphragmatic Dysfunction (Definition, Pathophysiology, Prevention), §Ventilator Settings according to Disease Type; ch. 29, §Driving Pressure, §Mechanical Power, §Esophageal Pressure Monitoring; ch. 163, §Pathophysiology, §Low Tidal Volumes, Fig. 163.2; ch. 172 "Acute Exacerbation of Asthma", §Complications of Mechanical Ventilation.

Guidelines: ESICM, Grasselli G, Calfee CS, Camporota L, et al., "ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies", Intensive Care Med 2023;49:727–759, Rec 5.1.

doi:10.1007/s00134-023-07050-7. American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official ATS Clinical Practice Guideline", Am J Respir Crit Care Med 2024;209(1):24–36. doi:10.1164/rccm.202311-2011ST.

Paper II • April 2023 • Q6 • 10 marks • Long

Define the various modes of mechanical ventilation. How are these classified? [2+8]

Also asked as: Paper I • December 2022 • Q4(b) "Basic types of mechanical breath. [5]"

1. Definitions

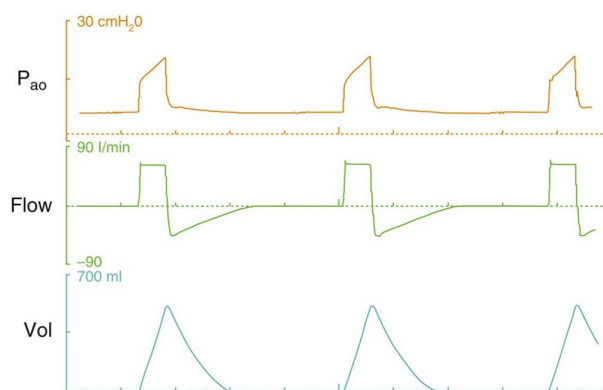
- **Invasive mechanical ventilation:** controlled or assisted breaths delivered through an endotracheal tube, tracheostomy, laryngeal mask or (rarely) oesophageal obturator airway.
- Ventilators apply supra-atmospheric pressure to drive bulk gas flow into the alveoli; expiration is usually passive.
- **Mode:** a ventilator pattern created by **which breath types are delivered and whether spontaneous breaths are allowed**.
- **Breath:** characterised by its **trigger** (how it starts), **target** (what governs gas flow) and **cycle** (how it ends).
- Controlled breath = machine-triggered (time); assisted breath = patient-triggered (effort).

2. Basic types of mechanical breath

Table 85. Defining characteristics of breath types (Irwin and Rippe 9e, Table 166.1)

Breath type	Trigger	Target	Cycle
Volume control (VC)	Time	Flow rate	Volume
Volume assist (VA)	Effort	Flow rate	Volume
Pressure control (PC)	Time	Pressure	Time
Pressure assist (PA)	Effort	Pressure	Time
Pressure support (PS)	Effort	Pressure	Flow rate
Spontaneous breath (SB)	Patient	Patient	Patient

- **Trigger:** time (60/frequency), pressure (fall at airway opening, typically 1–2 cm H₂O), flow (difference in bias flow, 2–3 L/min) or neural (diaphragm EMG). Flow trigger with an air leak (bronchopleural fistula) can cause autotriggering.
- **Target:** pressure-targeted (Pao constant → volume and flow depend on C_{rs}, R and Pmus) vs flow-targeted (flow constant → Pao depends on mechanics); linked by the **equation of motion**.
- **Cycle:** time (PC/PA, T_I 0.5–1.0 s), flow (PS — ~30% of peak flow; raise to ≥50% in airflow obstruction), volume (flow-targeted breaths) or neural (falling EAdi).
- Every mode has a **maximum pressure limit** (often 10 cm H₂O above peak) — alarms and vents the remaining breath.
- **Dual-control** breaths target pressure and flow in one breath (volume-assured PS) — no proven advantage.



Irwin and Rippe 9e, Figure 166.2 (ch. 166): volume assist-control — constant (square) flow, linear volume rise; pressure is the dependent variable set by respiratory mechanics.

3. Classification of modes

Table 86. Common modes are composed of breath types (Irwin and Rippe 9e, Table 166.2)

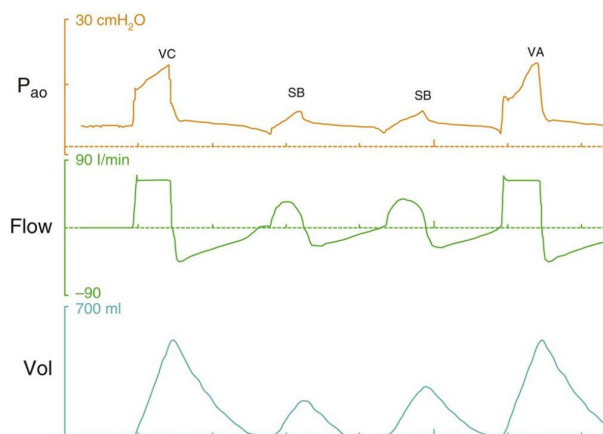
Mode	VC	VA	PC	PA	PS	SB
Volume assist-control (VACV)			—	—	—	—
Volume SIMV (V-SIMV)			—	—		
Pressure assist-control (PACV)	—	—			—	—
Pressure SIMV (P-SIMV)	—	—				
Pressure support (PSV)	—	—	—	—		—

Table 87. Practical classification of ventilator modes (built from Irwin and Rippe 9e, ch. 166)

Group	Modes	Who controls V_T /rate
Flow (volume)-targeted, control/assist	VACV, V-SIMV	Clinician sets V_T , flow, rate
Pressure-targeted, control/assist	PACV, P-SIMV	Clinician sets PI, T_I , rate; V_T varies
Spontaneous (support) modes	PSV ($\pm$ CPAP)	Patient sets rate and V_T
Adaptive / volume-targeted pressure	PRVC, VSV, VAPS	Ventilator adjusts PI to a target V_T
Inverse-ratio / open-lung	APRV (BIPAP)	Long T_{high} , brief release
High-frequency	HFOV	Constant mean Paw, tiny oscillations
Proportional	PAV, NAVA	Assist proportional to effort/EAdi

4. Volume-targeted modes

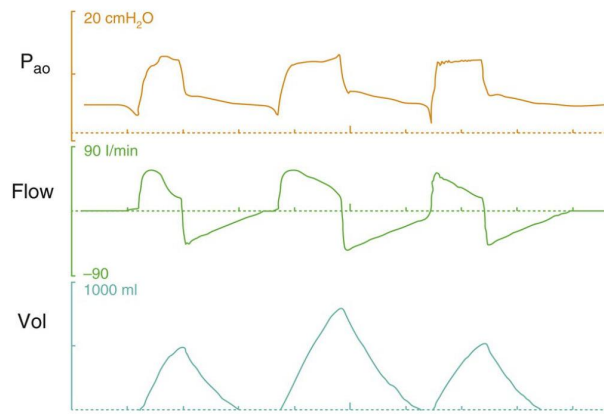
- **VACV:** time- or effort-triggered, flow-targeted, volume-cycled; clinician sets V_T , flow and rate; I:E follows. Extra breaths get the same V_T ; high drive $\rightarrow$ **double triggering** (common in lung-protective ARDS ventilation).
- **V-SIMV:** VC, VA, PS and SB; efforts inside the synchronisation window get a VA breath, earlier efforts a PS/spontaneous breath. Traditionally used for weaning, but **prolongs liberation**.
- Strength of flow-targeted modes: **mechanics are readily apparent** (constant flow) and V_T is easily limited despite changing effort.



Irwin and Rippe 9e, Figure 166.3 (ch. 166): V-SIMV in an actively breathing patient — controlled breath (VC), two unsynchronised spontaneous breaths (SB), then a synchronised assisted breath (VA) with lower pressure from patient effort.

5. Pressure-targeted modes

- **PACV:** PC + PA; set rate, PI and T_I ; pressure rises quickly to PI (rise time usually 0, 0.1–0.2 s may aid synchrony). Passive, unobstructed: $V_T = C_{rs} \times (PI - PEEP)$; obstruction/short T_I gives smaller V_T ; effort makes V_T larger.
- **PSV:** effort-triggered, pressure-targeted, flow-cycled — cannot be used in passive patients; rate and minute ventilation depend on drive; high PI can amplify periodic breathing and apnoeas.
- **P-SIMV:** PC, PA, PS, SB; passive patient = pressure control; SIMV "seems to impede weaning".
- Strength: variable flow (comfort) and built-in protection against runaway dynamic hyperinflation in severe obstruction.



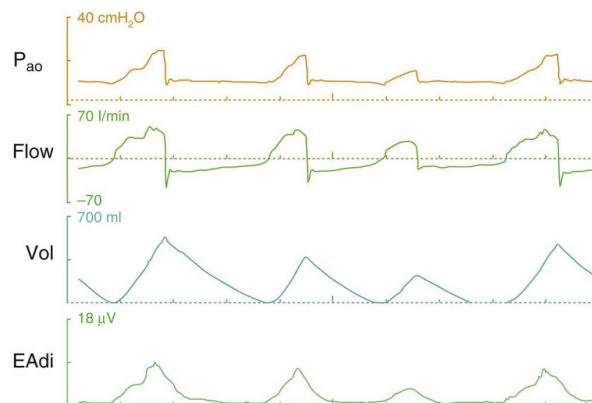
Irwin and Rippe 9e, Figure 166.5 (ch. 166): pressure support — constant airway pressure ($P_I + PEEP$), decelerating flow; tidal volume and flow vary breath to breath with patient effort.

6. Adaptive (dual-control) modes

- **PRVC**: pressure-targeted PC/PA breaths; clinician sets initial P_I , T_I , rate and V_T goal; ventilator adjusts P_I breath by breath. May increase work of breathing vs VACV; no patient-centred benefit.
- **VSV**: same principle with PS breaths — T_I and rate not set by clinician.
- **VAPS**: breath starts as PS and switches to a VC breath within the breath if target V_T will not be reached.

7. Special modes

- **APRV**: PC + PS + SB with very long T_I (T_{high}) and brief release (T_{low}) creating auto-PEEP; "biphasic positive airway pressure", "upside-down SIMV" — see APRV answer.
- **HFOV**: constant mean P_{aw} with small oscillatory volumes at high rate; OSCAR (795) no benefit, OSCILLATE (548) higher mortality 47% vs 35% → not for routine adult use.
- **PAV**: PS-type breath with P_I varying with instantaneous effort; ventilator measures R and E by occlusions every 8–15 breaths; assist 40–80%.
- **NAVA**: diaphragm electrical activity (EAdi) via nasogastric electrode triggers, sizes and cycles the breath (cycles at 40–70% of peak EAdi); backup to PACV/PSV if EAdi lost; more ventilator-free days in two trials.



Irwin and Rippe 9e, Figure 166.9 (ch. 166): NAVA — pressure-targeted breaths delivered in proportion to the electrical activity of the diaphragm (EAdi, lower trace).

8. Choosing a mode and initial settings

- Mode choice is mostly **physician preference** and probably does not change outcomes — principles of VILI, VIDD and dyssynchrony matter more.
- Apply a ventilator bundle to all: head-of-bed elevation, daily sedative interruption, SBT readiness testing.

Table 88. Initial settings by phenotype (Irwin and Rippe 9e, ch. 166, §Ventilator Settings according to Disease Type) [I&R]

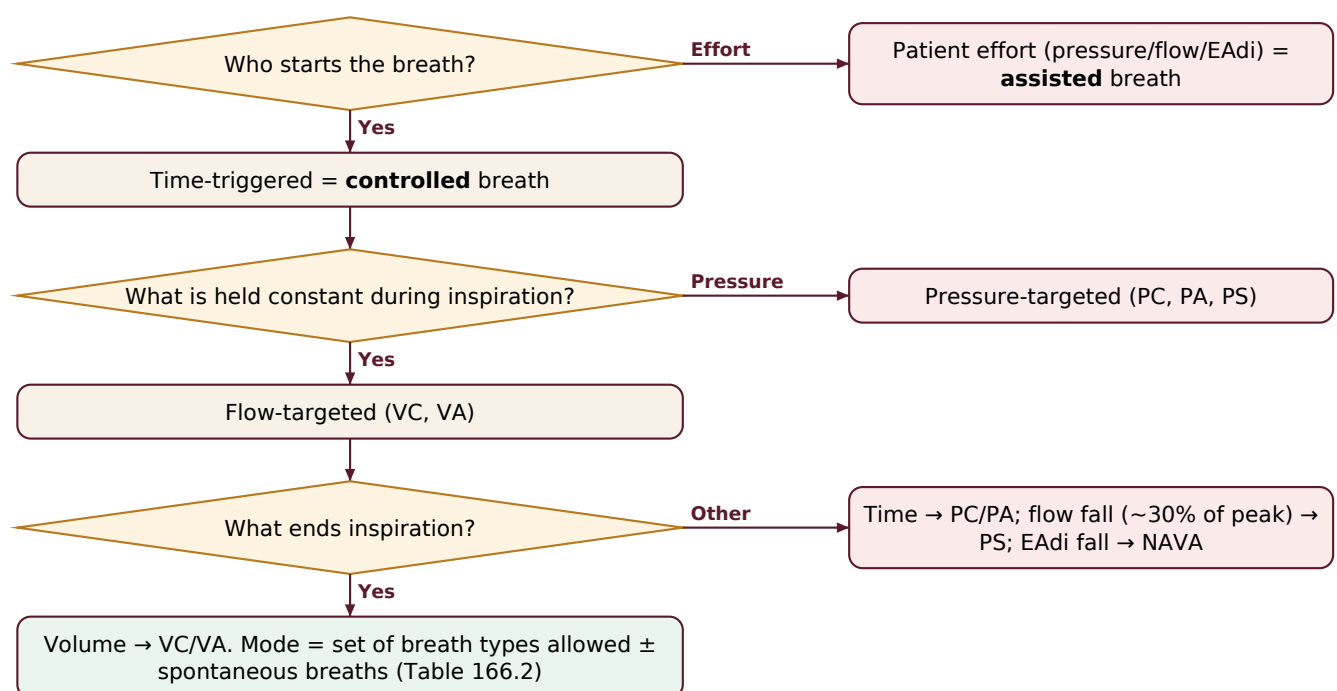
Phenotype	Mode	V_T / rate	PEEP / FiO_2	Key goal
Normal mechanics (overdose, brain injury, post-op)	VACV, PACV or PSV	6–10 mL/kg PBW; flow 50–60 L/min	5 cm H_2O ; 0.3–0.4	Near-normal gases; allow triggering
ARDS	VACV or PRVC	6 mL/kg PBW; RR 26–35	Controversial; $\uparrow FiO_2$	Limit $V_T/\Delta P$ (≤ 14); permissive hypercapnia
Severe airflow obstruction	Pressure- or flow-targeted	Minute ventilation < 10 L/min	5 (some ≥ 10) cm H_2O	Avoid hyperinflation
Chest-wall restriction	Any	Limited V_T	Minimum PEEP; prefer $\uparrow FiO_2$	Avoid haemodynamic compromise

MANAGEMENT BASIS

Irwin and Rippe 9e, ch. 166 (no guideline recommends one mode over another); ARDS tidal volume from ESICM 2023 Rec 5.1 / ATS 2024.

- ARDS: V_T 4–8 mL/kg PBW with $P_{plat} < 30$ cm H_2O whatever the mode. [ATS 2024; strong, moderate]
- HFOV not for routine use in moderate–severe ARDS. [ATS 2024; strong, high]
- Troubleshoot by waveforms: flow dyssynchrony (concave pressure trace) → raise flow to ≥ 60 L/min or switch to pressure-targeted; double triggering → brief 0.2 s end-inspiratory pause; delayed cycling in PS → raise cycle threshold. [I&R]

Flowchart. Classifying any breath (and hence the mode)



Drawn from Irwin and Rippe 9e, ch. 166, Tables 166.1–166.2 and §Defining Characteristics of Breath Types.

PEARLS — what wins marks

- Mode = combination of breath types ± spontaneous breaths; breath = trigger · target · cycle.
- Five breath types: VC (time/flow/volume), VA (effort/flow/volume), PC (time/pressure/time), PA (effort/pressure/time), PS (effort/pressure/flow).
- VACV: set V_T ; pressure varies. PACV: set PI ; $V_T = C_{rs} \times (PI - PEEP)$ if passive.
- PSV needs a triggering patient; flow-cycled at $\sim 30\%$ of peak (higher in COPD).
- SIMV prolongs weaning; PRVC no patient-centred benefit; HFOV harmful (OSCILLATE).
- PAV and NAVA = proportional modes; NAVA uses EAdi.
- Mode matters less than V_T , ΔP , synchrony and diaphragm protection.

Textbook: Irwin and Rippe 9e, ch. 166, Key Points; §Introduction; §Modes of Mechanical Ventilation (Physiology of PPV; Defining Characteristics of Breath Types — Trigger, Target, Cycle; Tables 166.1–166.2; VACV, V-SIMV, PACV, PSV, P-SIMV, PRVC/VSV, APRV, HFOV, VAPS, PAV, NAVA; Choosing a Ventilatory Mode); §Ventilator Settings according to Disease Type; §Patient Ventilator Dyssynchrony; Figs. 166.2, 166.3, 166.5, 166.9. Ch. 29, §Ventilator Triggering Variable.

Guidelines: ESICM, Grasselli G, Calfee CS, Camporota L, et al., "ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies", *Intensive Care Med* 2023;49:727–759, Rec 5.1. doi:10.1007/s00134-023-07050-7. American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official ATS Clinical Practice Guideline", *Am J Respir Crit Care Med* 2024;209(1):24–36. doi:10.1164/rccm.202311-2011ST.

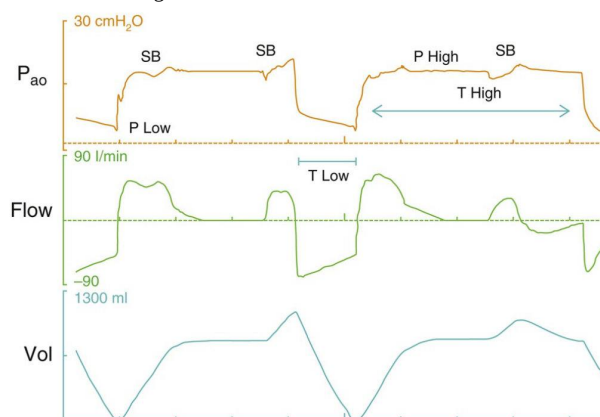
Paper III • December 2022 • Q10(b) • 5 marks • Intermediate

Role of APRV mode in ARDS. [5]

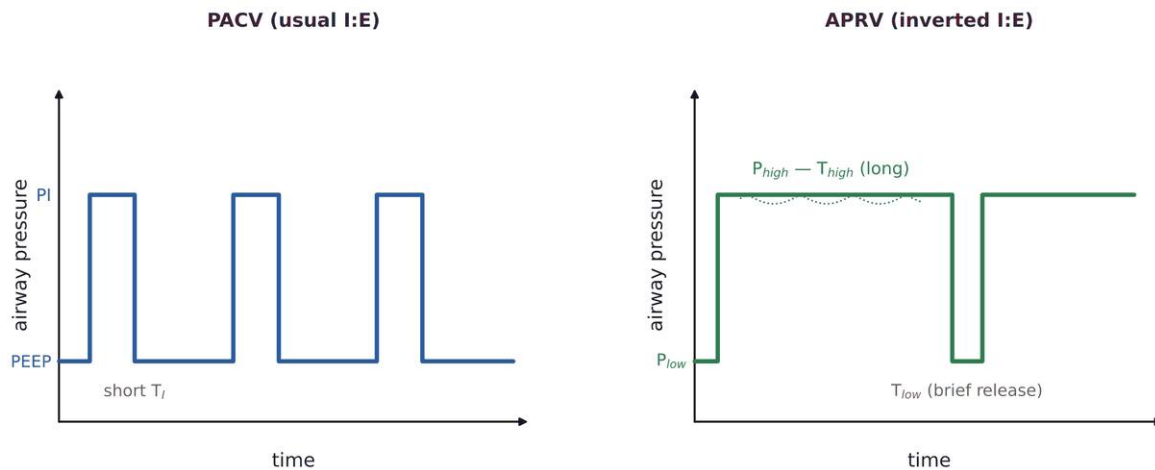
Also asked as: Paper I • May 2024 • Q9(c) "What is the current status of APRV mode in current practice in ICU? [3]"

Principle

- **APRV**: sustained high airway pressure with unrestricted spontaneous breathing, interrupted by **brief pressure releases** that provide ventilation while limiting derecruitment.
- Breath types: PC + PS + SB → time- and effort-triggered, pressure-targeted, time- and flow-cycled; defined by a **very long T_I** .
- Also called **biphasic positive airway pressure** (Europe) or "upside-down SIMV".
- Without spontaneous effort APRV equals PACV with inverted I:E — a form of **inverse-ratio ventilation**; created as a more comfortable way to deliver it.
- Spontaneous breaths are allowed in both T_{high} and T_{low} (as PS or SB).



Irwin and Rippe 9e, Figure 166.8 (ch. 166): APRV in an actively breathing patient — P_{high} (PI), P_{low} (PEEP), long T_{high} , brief T_{low} during which expiratory flow does not reach zero (auto-PEEP); spontaneous breaths (SB) at both levels.

Airway pressure vs time: pressure assist-control (PACV) vs APRV (schematic, not to scale)

Without spontaneous effort APRV equals PACV with inverted I:E (inverse-ratio ventilation). Dotted = spontaneous breaths allowed at both pressure levels.

Schematic (drawn for these notes) — Airway pressure against time for pressure assist-control (short inspiratory time) and APRV (long P_{high} with a brief T_{low} release; spontaneous breaths allowed at both pressure levels). Facts per Irwin and Rippe 9e, Ch 166 §Airway Pressure Release Ventilation. Schematic, not to scale.

Table 89. APRV settings (Irwin and Rippe 9e, ch. 166)

Setting	Meaning	How it is commonly chosen
P_{high}	Inspiratory pressure	Below upper inflection point of PV curve, or near P_{plat} measured on VACV
P_{low}	PEEP	Zero, by oxygenation, just above lower inflection point, or raised until V_T falls to 6 mL/kg PBW
T_{low}	Expiratory time (release)	Kept brief to create auto-PEEP — by expiratory flow curve or time constant (no agreement)
T_{high}	Inspiratory time	Follows from T_{low} and frequency
Frequency	Releases per minute	Generally about 10/min

Proposed physiological advantages

- Prolonged P_{high} maximises **recruitment**; brief releases avoid alveolar closure and re-expansion.
- Spontaneous breathing is thought to generate regionally variable transpulmonary pressures that favour recruitment of **dependent lung**.
- Recruitment and gas exchange often improve at **lower maximal airway pressures** than conventional ventilation; often used in severe ARDS to improve oxygenation.
- Possible lower sedation needs — not confirmed in all studies.

Disadvantages

- Tidal volume often **exceeds lung-protective values**, even with low- V_T protocols; spontaneous breaths above P_{high} add distension and large transpulmonary swings.
- Derecruitment during T_{low} could cause **atelectrauma**.
- Spontaneous breathing in **early severe ARDS** may worsen outcome (effort-dependent injury).
- No agreed method for setting T_{low} .

Evidence

Table 90. APRV trials (Irwin and Rippe 9e, ch. 163, 166)

Trial	Comparison	Result
Early RCT	APRV vs SIMV	Better physiology; stopped early for futility (no mortality/VFD gain)
2010 trauma RCT	APRV vs low- V_T	Equal safety; non-significant trend to more ventilator days and ICU stay
Single-centre RCT, 138 medical ARDS	Early APRV (<48 h) vs low- V_T	More VFDs, more successful extubation, fewer tracheostomies; NS trend to lower mortality
Summary (ch. 166)	—	"Clinical trials have shown no patient-centered advantages to this mode"

Role in ARDS and current status in ICU practice

MANAGEMENT BASIS

Irwin and Rippe 9e, ch. 163 and 166. The ESICM 2023 recommendations read in this build and the ATS 2024 guideline (as read) make no recommendation on APRV; ATS 2024 and ESICM 2023 low-tidal-volume recommendations apply to any mode.

- Insufficient evidence to support **routine use of APRV** for ARDS; not recommended over conventional low- V_T ventilation. [I&R]
- Choice may need to be tailored to the patient's injury and lung mechanics; most experience is in surgical/trauma ARDS. [I&R]
- A multicentre RCT of early APRV vs low- V_T (NCT04221737) was ongoing at the time of writing of the textbook. [I&R]
- Whatever the mode: V_T 4–8 mL/kg PBW and P_{plat} <30 cm H₂O [ATS 2024; strong, moderate] — so check exhaled V_T during APRV releases and spontaneous breaths.
- Current status: a **rescue/alternative mode for oxygenation** in some centres, not a standard of care; no survival benefit proven. [I&R]

PEARLS — what wins marks

- APRV = long P_{high}/T_{high} with brief release (T_{low}) + spontaneous breathing throughout.
- Settings: $P_{high} \approx P_{plat}$ or below UIP; P_{low} 0 or above LIP; T_{low} brief to keep auto-PEEP; ~10 releases/min.
- Passive patient on APRV = inverse-ratio PACV.
- Downsides: large V_T , atelectrauma during release, harmful effort in early severe ARDS.
- Status: improves oxygenation, no patient-centred benefit → not routine (I&R).

Textbook: Irwin and Rippe 9e, ch. 166, §Airway Pressure Release Ventilation, Fig. 166.8; §Positive End-expiratory Pressure (Preventing lung injury); ch. 163, §Management — Airway Pressure Release Ventilation.

Guidelines: American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official ATS Clinical Practice Guideline", *Am J Respir Crit Care Med* 2024;209(1):24–36. doi:10.1164/rccm.202311-2011ST. ESICM, Grasselli G, Calfee CS, Camporota L, et al., "ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies", *Intensive Care Med* 2023;49:727–759, Rec 5.1. doi:10.1007/s00134-023-07050-7.

Paper I • May 2024 • Q9(b) • 3 marks • Short

Closed loop ventilation. [3]

SOURCE NOTE

Irwin and Rippe 9e does not use the term "closed-loop ventilation" (searched "closed-loop", "closed loop", "SmartCare", "IntelliVent", "INTELLiVENT", "automated weaning", "knowledge-based"; hits only for insulin, hypothermia devices and bowel obstruction). It describes the individual feedback-controlled modes (PRVC, VSV, VAPS, adaptive support ventilation, PAV, NAVA), from which this answer is built. No society guideline on closed-loop ventilation was searched for (web budget).

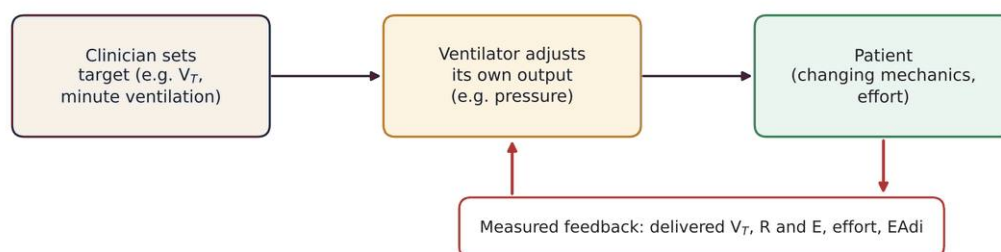
Concept

- Modern ventilators were developed to "respond more readily to changing conditions, and **adjust settings without clinician intervention**" — i.e. the ventilator measures a variable and adjusts its own output (feedback loop).
- Rapid microprocessors allow support to change **within a single breath**.
- Feedback signals used: delivered tidal volume, minute ventilation, respiratory mechanics (R, E), patient effort, diaphragm electrical activity (EAdi).
- Aims: maintain a target despite changing mechanics or effort, improve synchrony, protect lung and diaphragm, automate weaning.

Table 91. Feedback-controlled ("closed-loop") modes in Irwin and Rippe 9e

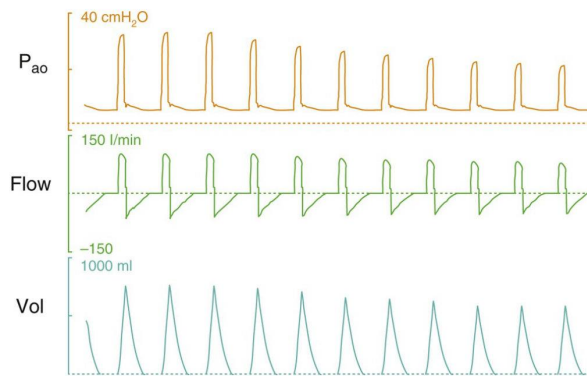
Mode	Measured input	What the ventilator adjusts	Evidence (I&R)
PRVC	Delivered V_T	Inspiratory pressure, breath by breath, to reach V_T goal	May ↑ work of breathing vs VACV; no patient-centred benefit
Volume support (VSV)	Delivered V_T	PS level (patient sets rate, T_I)	—
VAPS	Projected V_T within breath	Switches PS breath to VC breath	No apparent advantage
Adaptive support ventilation	Patient respiratory activity	Delivers preset minute ventilation; designed to wean automatically	Not adequately supported for routine weaning
PAV	R and E (occlusion every 8–15 breaths), effort	Pressure throughout inspiration (40–80% assist)	Better synchrony; may improve liberation vs PSV
NAVA	EAdi (oesophageal electrode)	Timing and size of assist; cycles at 40–70% of peak EAdi	More VFDs in difficult-to-wean and mild-ARDS trials

Closed-loop (feedback-controlled) ventilation



Examples: PRVC / VSV, VAPS, ASV, PAV, NAVA

Schematic (drawn for these notes) — Closed-loop (feedback-controlled) ventilation: the clinician sets a target, the ventilator adjusts its own output from measured feedback (delivered V_T , resistance and elastance, effort, EAdi). Facts per Irwin and Rippe 9e, Ch 166 §Closed-Loop Modes.



Irwin and Rippe 9e, Figure 166.7 (ch. 166): PRVC — after the target V_T is changed from 800 to 500 mL the ventilator lowers inspiratory pressure step by step to deliver the new target (time axis compressed).

Advantages and limitations

- Advantages: maintains V_T or minute ventilation as mechanics change; proportional modes protect against over- and under-assistance and improve synchrony. [I&R]
- Limitations: profusion of modes "often driven by marketing" and untested theories; PRVC may increase work of breathing; NAVA needs correct electrode position, intact drive and diaphragm anatomy. [I&R]
- Mode choice probably does not change outcome — lung-protective principles matter more. [I&R]

PEARLS — what wins marks

- Closed-loop = ventilator adjusts its own output from a measured feedback signal.
- Examples: PRVC/VSV (V_T), VAPS, ASV (minute ventilation), PAV (effort, R, E), NAVA (EAdi).
- ASV delivers a preset minute ventilation and aims at automatic weaning — evidence inadequate for routine use.
- NAVA cycles off at 40–70% of peak EAdi; falls back to PACV/PSV if signal lost.

Textbook: Irwin and Rippe 9e, ch. 166, §Physiology of Positive Pressure Ventilation, §Target (dual-control breaths), §Pressure-Regulated Volume Control and Volume Support, §Volume Assured Pressure Support, §Proportional Modes (PAV, NAVA), §Choosing a Ventilatory Mode, Fig. 166.7; ch. 168, §Predicting Successful Discontinuation (adaptive support ventilation).

Paper I • June 2022 • Q4(a) • 5 marks • Intermediate

Effect of changes in intra-thoracic pressure during mechanical ventilation on: Right ventricular function. [5]

Also asked as: Paper I • June 2022 • Q4(b) "Effect of changes in intra-thoracic pressure during mechanical ventilation on: Left ventricular function. [5]"

Principle

- The heart sits inside the thorax, surrounded by **pleural pressure (Ppl)**; ventilation alters cardiac loading **directly** (cyclic Ppl changes) and **indirectly** (work of breathing, gas exchange, pulmonary vascular resistance).
- In the passive patient both **PEEP and tidal inspiration raise Ppl**; the effect is amplified when chest-wall compliance is low.
- Spontaneous effort and positive pressure also alter all measured intravascular pressures — read them at **end-expiration**.

Table 92. Effects of raised intrathoracic pressure during mechanical ventilation (Irwin and Rippe 9e)

Determinant	Right ventricle	Left ventricle
Preload	↓ venous return to RV (ch. 166, 168, 172)	Positive pressure described as preload-reducing (ch. 167, 168); ch. 166 states raised Ppl increases LV preload
Afterload	↑ — mainly via increased lung volume; PEEP ↑PVR and PA pressure (ch. 174)	↓ — reduced LV transmural pressure (ch. 166, 167)
Output	Falls with excessive V_T /PEEP, mostly by impeded RV ejection	Rises in afterload-dependent failing LV; mitral regurgitation lessens
Interdependence	RV dilatation shifts septum → ↓LV filling	Secondary fall in LV output
Withdrawal of support	RV unloaded	LV may decompensate → pulmonary oedema, weaning failure

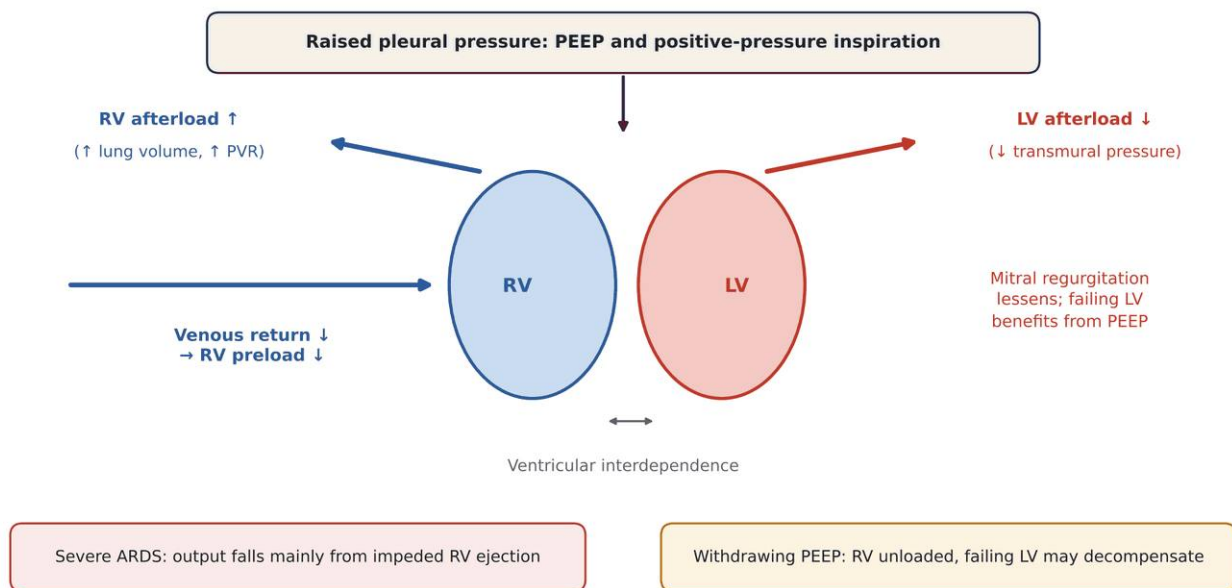
(a) Right ventricular function

- **Preload:** higher intrathoracic pressure decreases venous return; hypotension after intubation in severe asthma is from impeded venous return — fluids and less hyperinflation. [I&R ch. 172]
- **Afterload:** mechanical ventilation raises RV afterload, mediated mainly through increased lung volume; PEEP up-titration in ARDS raised mPAP 25 → 28 mm Hg and PVR 310 → 385 dyn·s·cm⁻⁵. [I&R ch. 174]
- In severe ARDS, excessive V_T or PEEP lowers cardiac output usually by **impeding RV ejection** rather than by dropping preload; acute cor pulmonale is less common with lung-protective V_T . [I&R ch. 166]
- The thin-walled RV tolerates volume but its stroke volume falls in proportion to afterload; RV pressure overload → **ventricular interdependence** and possible right-to-left shunt through the foramen ovale. [I&R ch. 174]
- **Hypercapnia** (permissive) raises PA pressure (± PVR, ↓RVEF). [I&R ch. 174]
- Favourable effects: recruiting PEEP lowers PVR; better oxygenation relieves hypoxic vasoconstriction; **prone position** reduces RV afterload and can boost output. [I&R ch. 166, 174]
- In PAH with RV failure: low V_T , low PEEP, avoid permissive hypercapnia; high peri-intubation collapse risk. [I&R ch. 174]

(b) Left ventricular function

- Raised Ppl reduces LV **transmural pressure** → ↓afterload; also lessens mitral regurgitation. [I&R ch. 166]
- In severe LV dysfunction PEEP treats pulmonary oedema, ↓mitral regurgitant fraction and ↑cardiac output. [I&R ch. 166]
- CPAP/NIV in cardiogenic pulmonary oedema: ↑FRC, ↓transmyocardial pressure, preload and afterload reduction; CPAP 10–12.5 cm H₂O improves dyspnoea, oxygenation and lowers intubation. [I&R ch. 167]
- **Withdrawal** of PEEP or ventilation can provoke decompensation or weaning failure — circulation cannot meet the demands of spontaneous breathing; hence extubation to NIV helps cardiac patients. [I&R ch. 166, 168]
- Increased work of breathing during weaning may "steal" oxygen from the heart and precipitate ischaemia; consider CPAP for marginal cardiac function. [I&R ch. 168]
- Indirect benefit: ventilation and PEEP lower the work of breathing and improve gas exchange. [I&R ch. 166]

Effects of raised intrathoracic pressure on the two ventricles

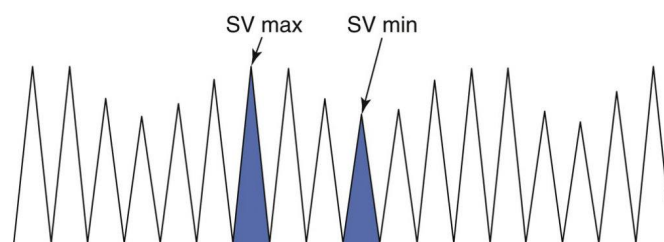


Schematic (drawn for these notes) — Effects of raised intrathoracic pressure (PEEP and positive-pressure inspiration) on the two ventricles: RV preload falls and RV afterload rises; LV afterload falls and mitral regurgitation lessens; ventricular interdependence. Facts per Irwin and Rippe 9e, Ch 166 §Heart-Lung Interaction; Ch 168; Ch 174.

MANAGEMENT BASIS

Irwin and Rippe 9e, ch. 166, 167, 168, 174 and 28 — no guideline addresses heart–lung interactions as such.

- Measure vascular pressures at **end-expiration**; do not reduce PEEP just to record "better" right atrial or wedge pressures. [I&R]
- Use respiratory variation to predict fluid responsiveness: **PPV 12.5%** and **SVV 11.6%** had moderate sensitivity/specificity in a meta-analysis; more accurate than CVP or PAOP. [I&R ch. 28]
- Do not disconnect PEEP to measure PAOP: venous return rises acutely and abrupt PEEP removal causes hypoxia that may not reverse quickly. [I&R ch. 19]
- In ARDS with acute cor pulmonale or shock, V-A rather than V-V ECMO is preferred. [I&R ch. 166]



Irwin and Rippe 9e, Figure 28.2 (ch. 28): stroke volume variation with respiration (SV max – SV min), the basis of dynamic preload indices during positive-pressure ventilation.

PEARLS — what wins marks

- Positive pressure: RV — ↓preload, ↑afterload; LV — ↓afterload (↓transmural pressure).
- Severe ARDS: output falls mainly from impeded RV ejection; prone position unloads the RV.
- PEEP helps the failing LV; withdrawing it can cause weaning-induced pulmonary oedema.
- Measure pressures at end-expiration; PPV >12.5% / SVV >11.6% suggest preload responsiveness.
- PAH/RV failure: low V_T , low PEEP, avoid hypercapnia.

Textbook: Irwin and Rippe 9e, ch. 166, §Positive End-expiratory Pressure (LV dysfunction), §Heart-Lung Interaction; ch. 174, §Physiology of the Pulmonary Circulation and RV, §Treatment — Mechanical Ventilation; ch. 167, §Cardiogenic Pulmonary Edema; ch. 168, §Cardiovascular Disease, §Managing Discontinuation Failure; ch. 172, §Complications of Mechanical Ventilation; ch. 28 "Minimally Invasive Hemodynamic Monitoring", §Cardiac Output — Clinical Utility, Fig. 28.2; ch. 19 "Pulmonary Artery Catheters", §Cardiac Output.

Paper III • May 2024 • Q10(b) • 5 marks • Intermediate

Non cardiac indications of ECMO with supporting evidence. [5]

Also asked as: Paper III • January 2025 • Q2(a) “What are the different modalities of ECMO (Extra-Corporeal Membrane Oxygenation) used in critically ill patients? [4]”

Definition and circuit

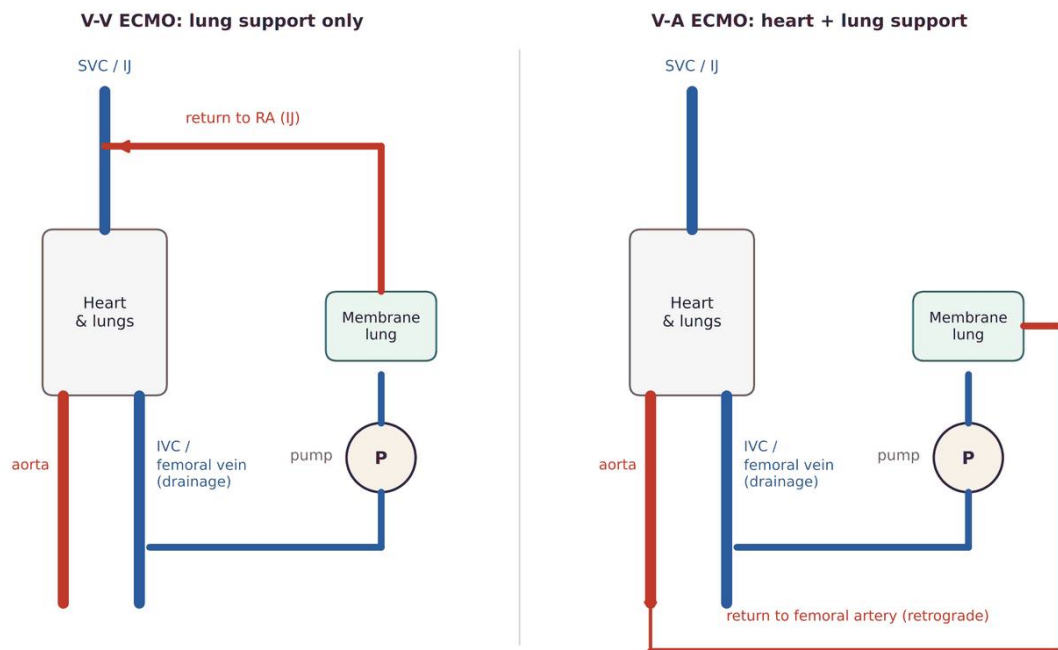
- **Extracorporeal life support (ECLS):** extracorporeal circuit + membrane lung + (usually) blood pump, supporting severe respiratory and/or cardiac failure as a bridge to recovery or to definitive therapy (e.g. transplantation).
- **Membrane lung** (hollow-fibre polymethylpentene): **blood flow** determines oxygenation; **sweep gas flow** determines CO₂ removal.
- Cannulae (percutaneous Seldinger, open, or semi-open) largely determine achievable flow; **centrifugal pumps** (less haemolysis, hand-crank backup) control flow by speed.

Modalities of ECLS

Table 93. ECLS modalities (Irwin and Rippe 9e, ch. 166)

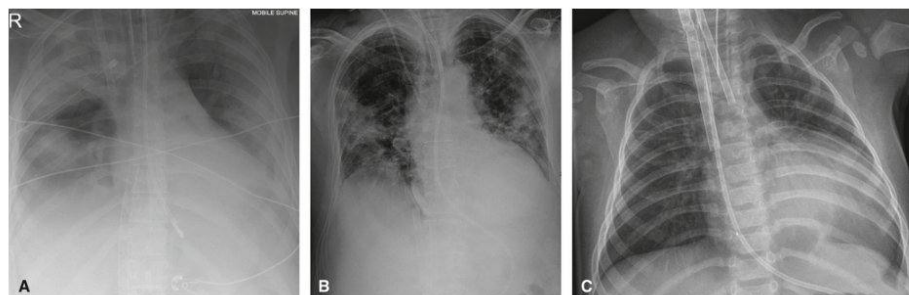
Modality	Access	Support	Key features
V-V ECMO	Two-site: drainage 23–29 Fr (femoral → IVC), return 21–23 Fr (IJ or other femoral → RA); or single-site dual-lumen IJ (~31F)	Lung only	Commonest for respiratory failure; recirculation; must capture ~70% of cardiac output; SaO ₂ mid-80s–low 90s acceptable
V-A ECMO	Drainage 23–29 Fr femoral vein; return 17–19 Fr femoral artery (retrograde aortic flow)	Heart + lung	Non-pulsatile flow; ↑LV afterload; differential oxygenation (coronaries, right arm, head may get desaturated blood)
V-V-A ECMO	Venous drainage, return to vein and artery	Heart + lung	Hybrid for combined cardiorespiratory failure
V-V ECCO₂R	Dual-lumen venous, smaller cannula	CO ₂ removal	~1 L/min removes all metabolic CO ₂ ; ultraprotective V _T 3–4 mL/kg
A-V pumpless ECCO₂R	Femoral artery drainage, contralateral femoral vein return	CO ₂ removal	Driven by patient BP; removes ~150 mL/min (~75% of production); no oxygenation

ECMO circuit configurations (schematic)



Schematic (drawn for these notes) — ECMO circuit configurations: V-V ECMO (drainage from the IVC or femoral vein, return to the right atrium via the internal jugular vein; lung support only) and V-A ECMO (drainage from the femoral vein, return to the femoral artery in retrograde direction; heart and lung support). Facts per Irwin and Rippe 9e, Ch 166 (ECLS modalities) and Ch 179 §ECMO Cannulae. Schematic, not to scale.

- V-V ECMO does not directly support the circulation but oxygenated pulmonary arterial blood may unload the RV by relieving hypoxic vasoconstriction; ARDS with acute cor pulmonale or shock → **V-A**. [I&R]
- V-A ECMO uses: cardiac failure, interval support for organ retrieval, extracorporeal CPR; flows kept somewhat lower than V-V to limit afterload. [I&R]
- Lung rest on ECMO: $V_T \leq 6$ mL/kg PBW or $\Delta P < 14$, PEEP 10–15, FiO_2 0.3–0.4, RR 6–10 (ultra-low $V_T \leq 4$ mL/kg possible); prone and awake/ambulatory ECMO feasible. [I&R]



Irwin and Rippe 9e, Figure 179.14 (ch. 179): ECMO cannula position on chest radiographs — A, "femorojugular" V-V ECMO (drainage cannula in the femoral vein ending in the IVC, return cannula via the internal jugular vein into the right atrium); B, "femorofemoral" V-V ECMO (drainage in the mid-IVC, return via the contralateral femoral vein into the right atrium); C, V-A ECMO (drainage cannula via the internal jugular vein with tip in the SVC, return cannula in the common carotid artery).

Non-cardiac indications — with evidence

Table 94. Respiratory ECMO trials and data (Irwin and Rippe 9e, ch. 166, 163)

Study	Design	Result
CESAR (2009, 180 adults)	Referral to ECMO centre vs conventional care	Death/severe disability at 6 months 37% vs 53% (RR 0.69); only 76% received ECMO; better protective ventilation at centre
EOLIA (2018, 249)	Early VV-ECMO vs conventional (crossover allowed) in very severe ARDS	60-day mortality 35% vs 46% (RR 0.76, P = .09); 28% crossed over (57% mortality); more bleeding
Meta-analysis CESAR + EOLIA	—	90-day mortality 36% vs 48% (RR 0.75)
Meta-analysis incl. observational	—	60-day mortality 34% vs 47% (RR 0.73)
ECCO ₂ R pragmatic RCT	Ultraprotective + ECCO ₂ R vs standard	90-day mortality 41.5% vs 39.5%; fewer VFDs; serious bleeding 31% vs 9%
Awake ECMO bridge to lung transplant	Retrospective, historical control	6-month post-transplant survival 80% vs 50%

- Other non-cardiac indications in Irwin and Rippe:
 - Severe air-leak syndromes; CO₂ retention despite Pplat >30; need for intubation on a lung-transplant list (ELSO criteria, Table 166.3).
 - Immediate respiratory collapse — **massive PE, airway obstruction** (Table 166.3); massive PE in pregnancy (case reports).
 - Primary graft dysfunction after lung transplant (early ECMO may be better).
 - COVID-19 ARDS — observational outcomes similar to non-COVID ARDS; H1N1 ARDS in pregnancy.
 - **Poisoning**: refractory cardiogenic shock, respiratory failure or arrest (shorter runs); **aluminium/zinc phosphide** — VA-ECMO cut mortality from 84.4% to 40% in one centre; hydrocarbon pneumonitis; status asthmaticus (pregnancy) — experienced centres.
 - Organ donation — interval support for organ retrieval (V-A).

MANAGEMENT BASIS

ATS ARDS guideline (2024); Surviving Sepsis Campaign 2026; ELSO VV-ECMO guideline (2021). No ISCCM ECMO guideline or position statement was found (search 8 Oct 2026); ESICM 2023 Domain 9 could not be read.

- VV-ECMO in **selected severe ARDS** — P:F <80 mm Hg or pH <7.25 with PaCO₂ ≥60 mm Hg, early (<7 days); provide in high-volume centres, consider transfer. [ATS 2024; conditional, low]
- VV-ECMO when conventional ventilation fails, in experienced centres (sepsis-associated ARDS). [SSC 2026; conditional, low]
- ELSO indications: P:F <80 after optimal management (incl. a prone trial); pH <7.25 despite optimal ventilation; bridge to lung transplant or PGD; severe asthma, eosinophilic pneumonia, pulmonary haemorrhage, thoracic trauma, inhalational injury, large bronchopleural fistula. [ELSO 2021]
- ELSO: only absolute contraindication — anticipated non-recovery without a decannulation plan; relative — CNS haemorrhage, bleeding/anticoagulation contraindication, immunosuppression, older age, ventilation >7 days with Pplat >30 and FiO₂ >90%. [ELSO 2021]
- Centres: at least 6 cases/year, ideally 15–20; higher volume = better outcome; earlier initiation better. [I&R, citing ELSO]
- Complications: infection, bleeding, thrombosis, distal limb ischaemia — more with V-A. [I&R]

PEARLS — what wins marks

- Membrane lung: blood flow → O₂; sweep gas → CO₂.
- V-V = lung support (ARDS); V-A = heart + lung; V-V-A = hybrid; ECCO₂R (V-V or A-V pumpless) = CO₂ only.
- V-A pitfalls: ↑LV afterload, differential (north-south) desaturation.
- EOLIA 35% vs 46% (NS); CESAR death/disability 37% vs 53%; pooled 90-day 36% vs 48%.
- ATS 2024: VV-ECMO in selected severe ARDS (P:F <80 or pH <7.25 + PaCO₂ ≥60, <7 days) — conditional, low.
- Non-cardiac uses: ARDS, hypercapnia, air leak, bridge to lung transplant, PGD, massive PE, airway obstruction, poisoning (phosphide).

Textbook: Irwin and Rippe 9e, ch. 166, Key Points; §Extracorporeal Life Support (Introduction, Key Circuit Components, Systems/Modes/Physiology, Supportive Therapies, Clinical Evidence and Indications, Table 166.3, Other Indications, Adverse Effects); ch. 163, §Extracorporeal Membrane Oxygenation; ch. 179 "Chest Radiographic Examination", §Evaluation of Tubes and Catheters — ECMO Cannulae, Fig. 179.14; ch. 96, §Cardiovascular Therapy; ch. 119 "Pesticide-Herbicide Poisoning", §Phosphides; ch. 112, §Halogenated Hydrocarbons; ch. 164 "Acute Respiratory Failure in Pregnancy", §Mechanical Ventilation, §Thromboembolism, §Pneumomediastinum.

Guidelines: American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official ATS Clinical Practice Guideline", Am J Respir Crit Care Med 2024;209(1):24–36. doi:10.1164/rccm.202311-2011ST. Extracorporeal Life Support Organization, Tonna JE, Abrams D, Brodie D, et al., "Management of Adult Patients Supported with Venovenous Extracorporeal Membrane Oxygenation (VV ECMO): Guideline from the ELSO", ASAIO J 2021;601–610 — indications, contraindications, configurations, rest settings, transfer. doi:10.1097/MAT.0000000000001432 (elso.org PDF). Surviving Sepsis Campaign 2026 (SCCM/ESICM), Crit Care Med 2026, statement C11. doi:10.1097/CCM.0000000000007075.

Paper III • December 2022 • Q8(a) • 5 marks • Intermediate

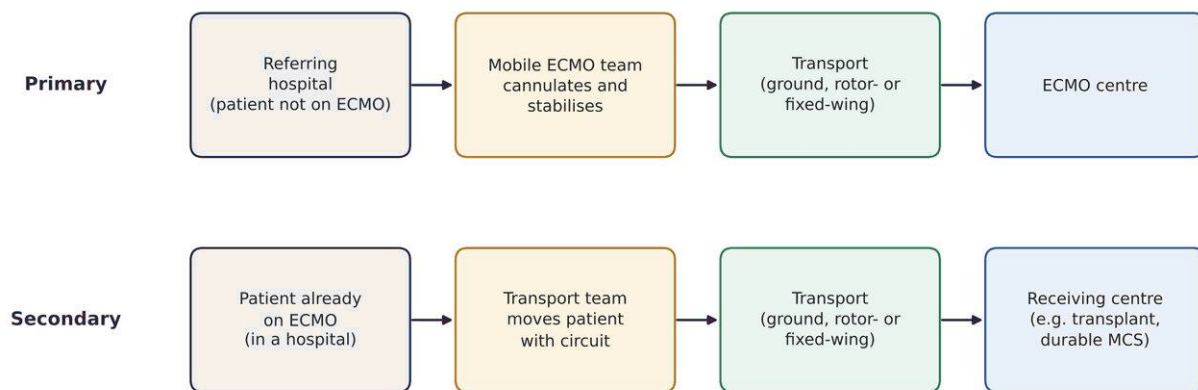
AIR transport of ARDS patient to ECMO centres (Inter hospital). [5]

Why transfer ARDS patients to an ECMO centre

- CESAR: referral to an ECMO centre improved 6-month survival without disability; centres also delivered more lung-protective ventilation (93% vs 70%) — supports transfer to large ECMO centres. [I&R]
- ELSO: centres should do ≥6 cases/year, ideally 15–20; higher volume, earlier initiation → better outcomes. [I&R]
- "Transfer to ECMO centers should be considered when feasible"; "ECMO should be provided in high-volume, dedicated centers"; organise programmes regionally. [ATS 2024]
- "If ECMO is to be a consideration, and transfer would be necessary, it should be done early" (RESP and Murray scores guide the decision). [ELSO 2021]
- Modern modular circuits facilitate inter-hospital transfer. [I&R]

Types of ECMO transport [ELSO 2022]

- **Primary:** a mobile ECMO team initiates ECMO at the referring hospital, then transfers the patient.
- **Secondary:** patient already on ECMO is moved to another centre (e.g. transplant, durable MCS, family request).
- Some patients may be transported **without** ECMO; mode chosen case by case.

Types of ECMO transport (ELSO 2022)

Some patients may be transported without ECMO; the mode is chosen case by case.

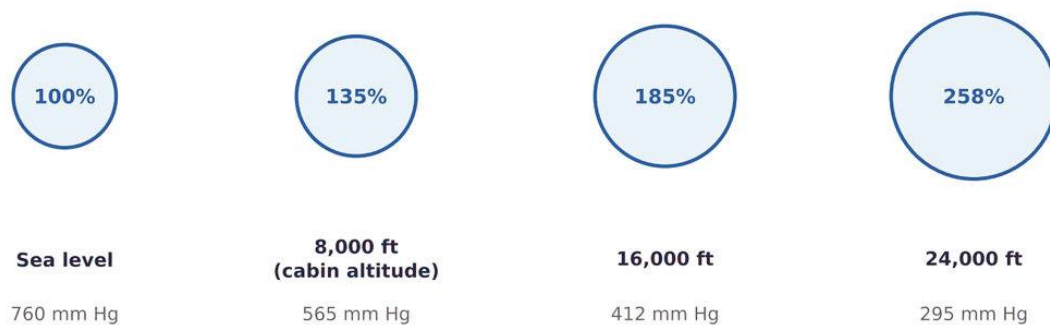
Schematic (drawn for these notes) — Types of ECMO transport: primary (a mobile ECMO team cannulates and stabilises the patient at the referring hospital) and secondary (a patient already on ECMO is moved with the circuit); vehicle is ground, rotor-wing or fixed-wing. Facts per ELSO Guideline for Transport and Retrieval of Patients with ECMO Support (ASAIO J 2022).

Table 95. Choice of transport vehicle (ELSO 2022)

Vehicle	Advantages	Limitations
Ground ambulance	Most common and available; larger units allow 360° access	Long distances, time
Rotor-wing (helicopter)	Speed over intermediate distances	Small cabin, limited crew; weather-dependent; liquid O ₂ flow varies with temperature; vibration and noise — alarms hard to hear
Fixed-wing	Long distances; high altitude → shorter, smoother flight, less fuel	Multiple patient transfers (ambulance–aircraft); equipment must be aviation-approved; barometric effects

Physiology of altitude relevant to the ARDS patient

- **Boyle law:** gas volume varies inversely with absolute pressure — at 8000 ft (565 mm Hg, 0.743 ATA) a gas bubble expands to 135%. [I&R, Table 177.4]
- Commercial aircraft keep cabin pressure equivalent to **8000 ft** while flying above 40,000 ft; loss of cabin pressure is a risk. [I&R ch. 177]
- O₂ fraction stays 20.95% up to ~70,000 ft, so inspired PO₂ falls with barometric pressure (Dalton law). [I&R ch. 177]
- ELSO flags the "effect of barometric pressure on endotracheal tube cuff" and, at low barometric pressure, "extra care must be taken to avoid hyper-oxygenation of the circuit". [ELSO 2022]

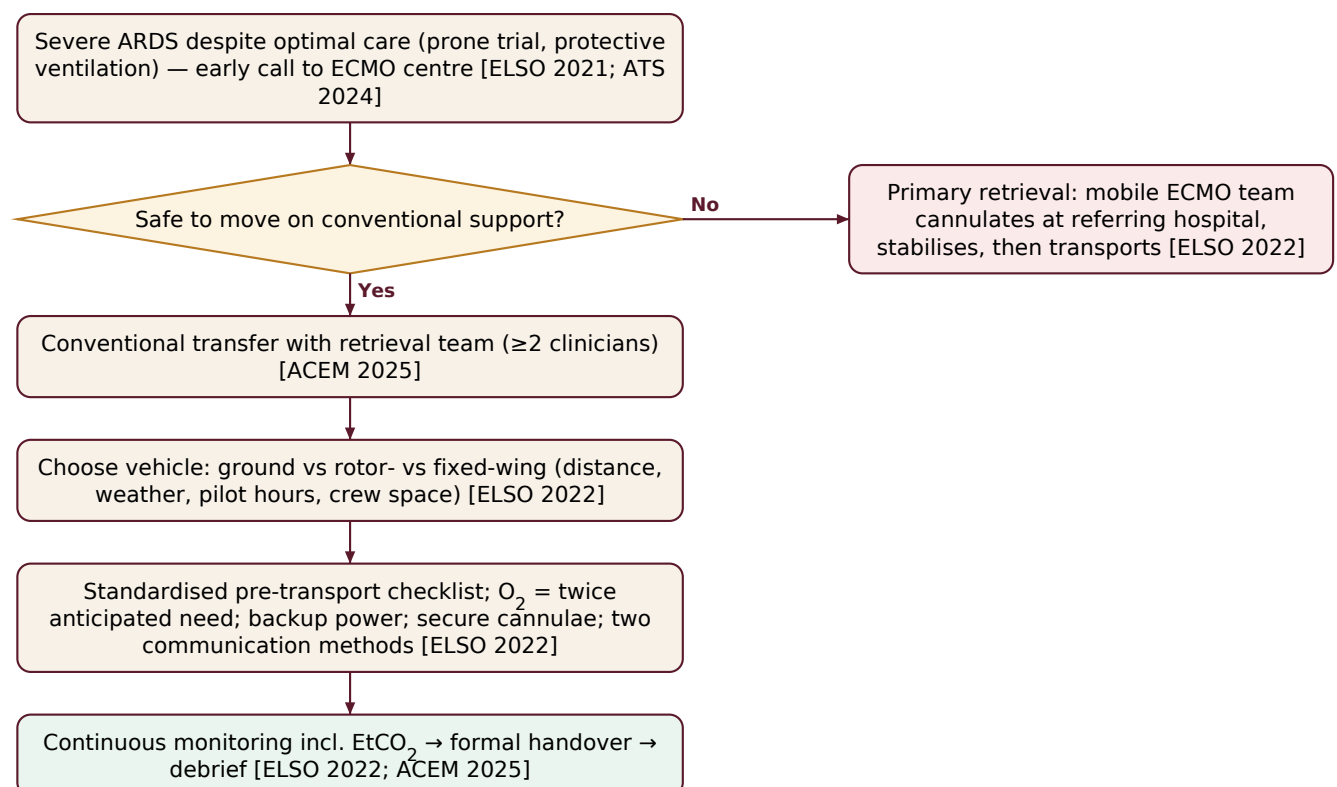
Boyle law at altitude: a gas bubble expands as pressure falls (not to scale)

Schematic (drawn for these notes) — Boyle law at altitude: a gas bubble expands to 135%, 185% and 258% of its sea-level volume at 8,000, 16,000 and 24,000 ft (barometric pressure 565, 412 and 295 mm Hg). Facts per Irwin and Rippe 9e, Ch 177 §Pathophysiology, Table 177.4. Not to scale.

MANAGEMENT BASIS

ELSO transport and retrieval guideline (ASAIO J 2022; expert opinion, ungraded); ACEM/ANZCA/CICM Joint Guideline for the Transport of Critically Ill Patients (2025; ungraded); ATS ARDS guideline 2024 and ELSO VV-ECMO guideline 2021 for referral.

Flowchart. Inter-hospital air transfer of a severe ARDS patient to an ECMO centre



Based on ELSO Guideline for Transport and Retrieval (2022) and ACEM/ANZCA/CICM Joint Guideline (2025).

Team

- ECMO transport team "should be composed of no less than: team lead; cannulating provider" plus ECMO specialist(s) (circuit set-up, priming, management to arrival) and a paramedic or equivalent. [ELSO 2022]
- "Retrieval teams should comprise a minimum of two clinicians, with the requisite scope of clinical practice." [ACEM 2025]
- Specialised multidisciplinary team; formal training in transport medicine and mobile ECMO, with simulation (power failure, cannula dislodgement, air entrainment). [ELSO 2022]

- Mobile ECMO "can be undertaken by regional or high-volume centers". [ELSO 2022]

Equipment and aircraft [ELSO 2022 unless stated]

- All equipment approved for aircraft use (Civil Aviation Authority/FAA); shielding against electromagnetic interference adds weight.
- **Oxygen**: "availability of twice the anticipated medical gas requirements"; consider O₂ concentrators.
- **Power**: sufficient power plus backup for all devices; uninterruptible power source for sensitive equipment.
- **Backup**: console, motor and/or **hand crank**; spare circuit components and priming fluid, within reach.
- Portable ventilator capable of invasive and non-invasive support; multi-function monitor; consumables in excess of estimated need. [ACEM 2025]

Preparation, monitoring and handover

- Standardised **pre-transport checklist** before leaving the sending facility; gradual (not sudden) changes in support; minimise non-essential drugs; cannulae well secured. [ELSO 2022]
- Monitor vital signs "including end-tidal CO₂ and invasive monitoring when able"; anticipate clinical changes en route; point-of-care tests. [ELSO 2022]
- "Continuous waveform capnography for all ventilated and/or sedated patients"; audible and visual alarms. [ACEM 2025]
- Two communication methods; in-flight link with pilot and crew. [ELSO 2022]
- "Formal endorsement and handoff to receiving team should occur"; debrief every case; quality metrics (cardiac arrest, air embolism, bleeding). [ELSO 2022]
- Level of care in transport should at least equal that at the referring point. [ACEM 2025]

Hazards in flight

- Circuit: pump stoppage, **air entrainment**, power failure, cannula dislodgement; oxygenator hyper-oxygenation at low pressure. [ELSO 2022]
- Patient: repeated bed–stretcher–aircraft transfers (fixed-wing), ETT cuff changes with barometric pressure; quality metrics track cardiac arrest, air embolism and bleeding. [ELSO 2022]
- Noise and vibration mask alarms (rotor-wing). [ELSO 2022]

PEARLS — what wins marks

- Transfer severe ARDS early to high-volume ECMO centres (CESAR; ATS 2024; ELSO 2021).
- Primary (mobile ECMO, cannulate at referring site) vs secondary (already on ECMO) transport.
- Rotor-wing: small cabin, weather-limited, noisy; fixed-wing: long distance, multiple transfers.
- Boyle law: gas expands to 135% at 8000 ft cabin altitude — watch ETT cuff and gas spaces.
- Carry twice the anticipated O₂, backup power and a hand crank; checklist before departure.
- Minimum team: team lead + cannulating provider + ECMO specialist (ELSO); ≥2 clinicians (ACEM).

Textbook: Irwin and Rippe 9e, ch. 163, §Extracorporeal Membrane Oxygenation (CESAR); ch. 166, §ECLS Introduction, §Other Indications (centre volume); ch. 177 "Gas Embolism Syndromes", §Decompression Sickness — Flying, Pathophysiology, Table 177.4.

Guidelines: Extracorporeal Life Support Organization, Labib A, August E, Agerstrand C, et al., "ELSO Guideline for Transport and Retrieval of Adult and Pediatric Patients with ECMO Support", ASAIO J 2022 (expert opinion; elso.org PDF). doi:10.1097/MAT.0000000000001653. Australasian College for Emergency Medicine / ANZCA / CICM, "Joint Guideline for the Transport of Critically Ill Patients" (ACEM G03, ANZCA PG52, CICM IC-10), August 2025 (ungraded) — team, equipment, monitoring, communication, handover. acem.org.au. American Thoracic Society, Qadir N, Sahetya S, Munshi L, et al., "An Update on Management of Adult Patients with Acute Respiratory Distress Syndrome: An Official ATS Clinical Practice Guideline", Am J Respir Crit Care Med 2024;209(1):24–36. doi:10.1164/rccm.202311-2011ST. Extracorporeal Life Support Organization, Tonna JE, Abrams D, Brodie D, et al., "Management of Adult Patients Supported with Venovenous Extracorporeal Membrane Oxygenation (VV ECMO): Guideline from the ELSO", ASAIO J 2021;601–610 — indications, contraindications, configurations, rest settings, transfer. doi:10.1097/MAT.0000000000001432 (elso.org PDF).

Paper I • April 2023 • Q8 • 10 marks • Long

Discuss indications, principles and physiology of gas exchange during high-flow nasal oxygen therapy. [2+2+6]

Also asked as: Paper I • January 2025 • Q3(a) "Describe the role of HFNC (high-flow nasal cannula) in critical care units, along with utility of "ROX" Index (Respiratory rate Oxygenation Index). [6+2]" • Paper I • December 2022 • Q6(a) "Physiological advantages of HFNC. [5]" • FNB • June 2019 • Q2(b) "High frequency nasal cannula. [5+5]"

1. Definition — what HFNC is

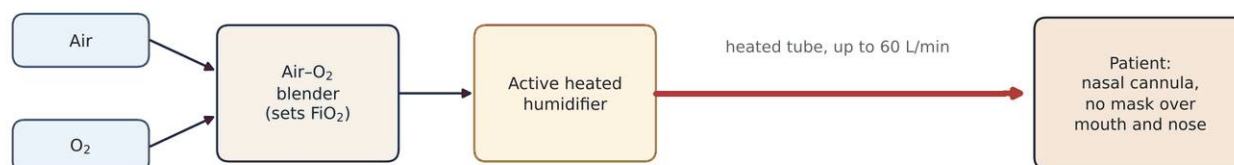
- **High-flow nasal cannula (HFNC / HFNT / HFNO):** delivery of **heated and humidified oxygen up to 60 L per minute** through nasal cannulae. [I&R ch. 169]
- Gas is typically an **oxygen–air mixture with adjustable FiO_2** ; flow rates "up to 60 L/min or higher". [ATS 2026]
- A form of **noninvasive respiratory support (NRS)**, alongside CPAP and NIV (facemask or helmet). [ATS 2026]
- Patients needing high O_2 flows (**>10 L/min**) by ordinary devices develop upper-airway dryness — dedicated devices humidify gas at high flow and **improve comfort**. [I&R ch. 169]
- Grew enormously during COVID-19: in the United States HFNT was the NRS used **most often**, mask NIV next, helmet a distant third. [I&R ch. 167]

2. Principles of the device

Table 96. Components and their purpose (Irwin and Rippe 9e ch. 169; ATS 2026)

Component	What it does	Why it matters
Air–oxygen blender	Sets FiO_2 independent of flow	Known, adjustable FiO_2 (ATS 2026)
High flow (up to 60 L/min)	Flow approaches/matches the patient's inspiratory flow	More consistent FiO_2 than conventional O_2 ; dead-space washout (ESICM 2023; I&R ch. 173)
Active heated humidifier	Warms and saturates gas with water vapour	Prevents mucosal drying; improves ciliary function and secretion clearance; comfort (I&R ch. 169; ATS 2026)
Nasal cannula interface	No mask over mouth and nose	Better tolerated than NIV (20 vs 6 h of use in first 24 h, post-cardiac-surgery RCT — I&R ch. 167)

High-flow nasal cannula: components and physiological effects



Physiological effects



Schematic (drawn for these notes) — High-flow nasal cannula set-up (air and O_2 through a blender and an active heated humidifier to a nasal cannula) and its physiological effects: consistent FiO_2 , PEEP of about 3–5 cm H_2O , dead-space washout, lower respiratory rate and work of breathing, and humidification. Facts per Irwin and Rippe 9e, Ch 169 §Humidity Therapy and §Oxygen Delivery Devices (HFNC); Ch 173 §Management — Oxygen; ESICM 2023; ATS 2026.

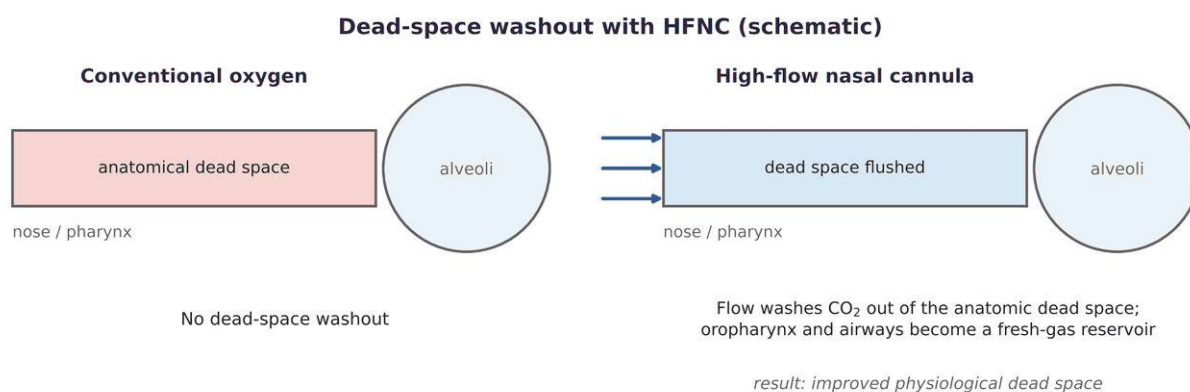
3. Physiology of gas exchange during HFNC

(a) Oxygenation

- At higher flows HFNO delivers a **more consistent FiO₂** than conventional oxygen therapy (COT). [ESICM 2023]
- The higher flow **matches the patient's inspiratory flow more effectively**. [I&R ch. 173]
- Generates **PEEP up to 3–5 cm H₂O**, depending on flow rate and breathing pattern. [ESICM 2023]
- This "small amount of PEEP" can **recruit lung units**. [I&R ch. 173]
- Net effect: **improved oxygenation** vs facemask oxygen in hypoxaemic respiratory failure. [I&R ch. 169]

(b) CO₂ clearance — dead-space washout

- Flow **washes CO₂ out of the anatomic dead space**, turning the oropharynx and conducting airways into a (fresh-gas) **reservoir**. [I&R ch. 173]
- Result: an **improvement of physiologic dead space**. [I&R ch. 169]
- "Reduces anatomical dead space and generates low levels of positive end-expiratory pressure." [ATS 2026]



Schematic (drawn for these notes) — Dead-space washout with HFNC: high flow washes CO₂ out of the anatomic dead space so the oropharynx and airways act as a fresh-gas reservoir, improving physiological dead space. Facts per Irwin and Rippe 9e, Ch 173 §Management — Oxygen and Ch 169. Not to scale.

(c) Breathing pattern and work of breathing

- **Lower respiratory rate** and **work of breathing per minute**. [ATS 2026]
- In COPD the greatest benefit is probably the **reduced respiratory rate** → **less air trapping**; the small PEEP may also **offset intrinsic PEEP**. [I&R ch. 173]
- Improved work of breathing vs facemask oxygen. [I&R ch. 169]

(d) Humidification and the airway

- Heated humidified gas **improves ciliary function and secretion clearance**. [ATS 2026]
- Humidification is imperative when upper-airway warming/humidifying structures are bypassed; adequate heat and humidity are needed for normal **mucociliary transport**. [I&R ch. 169]

Table 97. Physiological advantages of HFNC over conventional oxygen and NIV (sources as tagged)

Feature	Conventional O ₂ (mask/cannula)	HFNC	NIV (facemask)
FiO ₂ delivered	Cannula and simple mask deliver overlapping FiO ₂ ranges (I&R)	More consistent at high flow (ESICM)	Set on ventilator
Positive pressure	None	Low PEEP, up to 3–5 cm H ₂ O (ESICM)	Set EPAP + pressure support
Dead-space washout	No	Yes — anatomic dead space washed out (I&R ch. 173)	—
Humidification	Often none; dryness at >10 L/min (I&R)	Heated, humidified (I&R; ATS)	Depends on circuit
Tolerance	—	Better; adverse effects led to withdrawal in 0% vs 42.9% with NIV in one RCT (I&R ch. 167)	Mask discomfort, interface problems
Oxygenating power	Lower than HFNC (I&R ch. 169)	Better than facemask O ₂ (I&R ch. 169)	Better oxygenator than HFNC (post-cardiac surgery RCT, I&R ch. 167)

4. Indications and evidence (Irwin and Rippe)

- **De novo hypoxaemic failure / ARDS (FLORALI):** HFNC vs NIV vs standard O₂ (PaO₂/FiO₂ <300) — intubation not different overall but **significantly lower with HFNC when PaO₂/FiO₂ <200**; ICU and 90-day mortality lower with HFNC than NIV. [I&R ch. 167]
- Same trial (ch. 163): **hazard ratio for death 2.50 with NIV and 2.01 with standard cannula** vs HFNC; greater comfort with HFNC. [I&R ch. 163]
- Meta-analysis of 25 RCTs (3804 patients): HFNC reduced intubation but **not mortality**. [I&R ch. 167]
- **Immunocompromised ARF:** HFNC vs NIV — **44% reduction in intubation** and two-thirds reduction in 90-day mortality. [I&R ch. 167]
- **COVID-19:** Recovery–Respiratory Support trial — mask CPAP lowered intubation (36% vs 44%) but **HFNC did not** (44% vs 45%); a Colombian RCT (PaO₂/FiO₂ <200) found intubation 30% with HFNC vs 51% with standard O₂. [I&R ch. 167]
- **Post-extubation:** HFNC reduces post-extubation respiratory failure vs conventional O₂ (mortality and stay unchanged); in high-risk patients HFNC ≈ NIPPV for reintubation (Hernández: 22.8% vs 19.1%). [I&R ch. 167, 168]
- **Post-cardiac surgery** (Stephan, >800 patients): HFNC not inferior to NIV — treatment failure 21% vs 22%, reintubation 14% vs 14%. [I&R ch. 167]
- **DNI / palliation:** HFNC improves dyspnoea, lowers RR, improves O₂; better tolerated than NIV and can be used during NIV breaks. [I&R ch. 167]
- **Preoxygenation:** airway bundle includes NIPPV combined with HFNC, if feasible. [I&R ch. 8]
- **Bronchoscopy** in high-risk immunocompromised patients: NIV or HFNC used during the procedure. [I&R ch. 10]
- **Hypercapnic failure:** evidence limited mainly to case reports and a small exercise study. [I&R ch. 173]
- When a Venturi mask (FiO₂ 0.50) fails, HFNC is an acceptable alternative to a nonbreathing mask. [I&R ch. 169]

5. Management — recommendations

MANAGEMENT BASIS

ATS Clinical Practice Guideline on noninvasive respiratory support (Am J Respir Crit Care Med 2026); ESICM ARDS guideline (Intensive Care Med 2023) Rec 3.1; SSC 2026 (oxygen/HFNC statements); ERS/ESICM/ESCMID/ALAT severe CAP guideline (2023). ISCCM NIV/HFNC guideline (2020) could not be read in this build (register).

Table 98. Key recommendations on HFNC

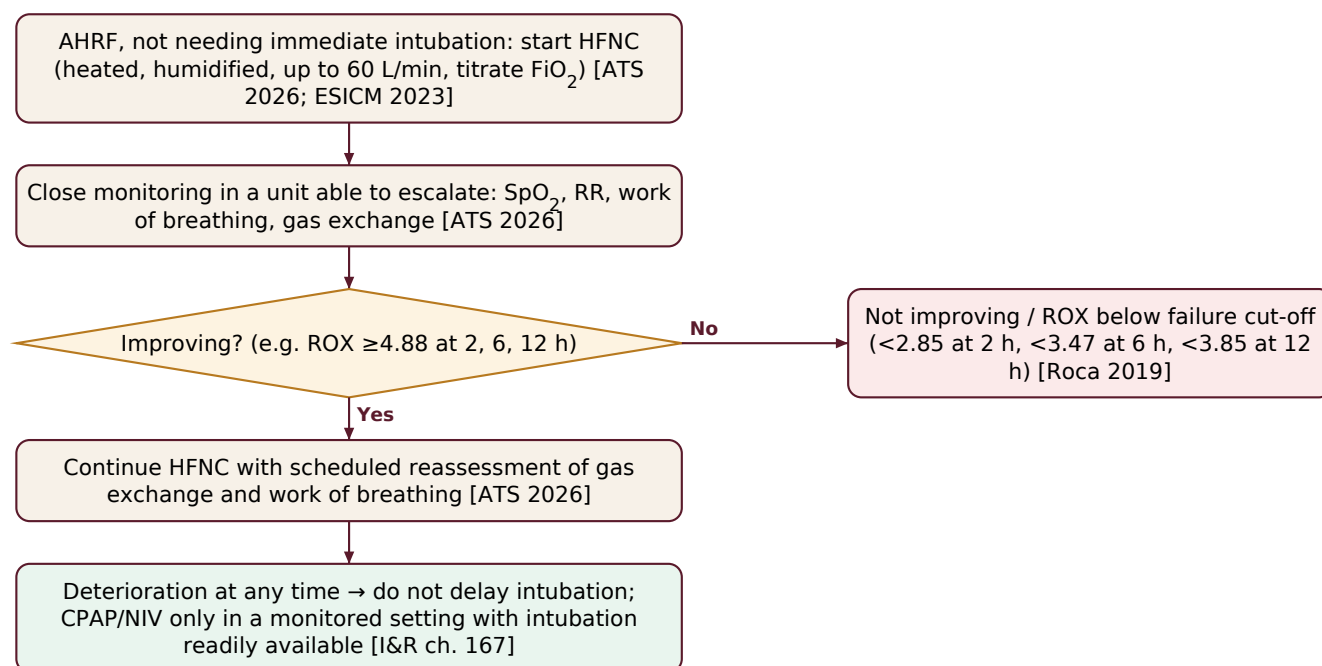
Situation	Recommendation	Strength · Body
De novo acute hypoxaemic respiratory failure	HFNC over standard oxygen therapy	Strong, moderate certainty · ATS 2026
AHRF (not cardiogenic oedema), to reduce intubation	HFNO over conventional O ₂	Strong; moderate level of evidence · ESICM 2023 Rec 3.1
AHRF — mortality	No recommendation for or against HFNO to reduce mortality	High level of evidence of no effect · ESICM 2023
HFNC vs NIV in AHRF	No recommendation for or against HFNC or CPAP compared with NIV	ATS 2026
Hypercapnic failure with mild acidaemia (pH >7.25)	HFNC as an alternative to NIV, if monitoring and escalation to NIV are promptly available	Conditional, low certainty · ATS 2026
Preoxygenation for intubation	HFNC or NIV via facemask over standard O ₂	Strong, moderate certainty · ATS 2026
Post-extubation (general)	HFNC or NIV via facemask over standard O ₂	Conditional, moderate certainty · ATS 2026
Post-extubation, high risk	NIV via facemask over standard O ₂ or HFNC	Conditional, moderate certainty · ATS 2026
Sepsis-related hypoxaemic failure	HFNC over conventional O ₂ ; HFNC as initial therapy over NIPPV	Conditional; very low / low · SSC 2026
Severe CAP with AHRF not needing immediate intubation	HFNO instead of standard oxygen	Conditional, very low · ERS/ESICM 2023

6. Monitoring, failure and the ROX index

- "Patients initiated on HFNC should be **closely observed for nonimproving or worsening gas exchange.**" [ATS 2026]
- Those requiring high flows or FiO₂ should ideally be managed in a unit capable of close supervision; continuous monitoring is essential to detect early failure. [ATS 2026]
- Institutions should establish **protocols defining criteria for intubation**, escalation pathways and staff training. [ATS 2026]
- Patients who failed HFNC had **higher mortality** than those on conventional O₂ — unclear whether from delayed intubation or more severe disease. [ESICM 2023]
- How long an HFNO trial should last, and whether the **ROX index** should be used to signal failure, is not clear. [ESICM 2023]
- **ROX index** = (SpO₂/FiO₂) ÷ respiratory rate. [Roca 2019]
- ROX ≥4.88 at 2, 6 or 12 h of HFNC → lower risk of intubation; ROX <2.85 (2 h), <3.47 (6 h), <3.85 (12 h) → predicts HFNC failure. [Roca 2019]
- If oxygenation persists poor, switching to CPAP or NIV before intubation may be considered — they deliver higher airway pressure more reliably (COVID-19 suggestion). [I&R ch. 167]
- Never let NRS **delay a needed intubation**; watch for patient self-inflicted lung injury from large tidal volumes. [I&R ch. 167]

SOURCE NOTE — ROX index

The ROX index is not described in Irwin and Rippe 9e (full-text search for "ROX", "respiratory rate-oxygenation"). Formula and cut-offs are from the validation study published in the ATS journal (Roca O et al., Am J Respir Crit Care Med 2019, abstract read) and are registered as a non-guideline source; see answer "ROX index and HACOR score" for detail.

Flowchart. HFNC in de novo acute hypoxaemic respiratory failure

Drawn from ATS 2026 implementation statements, ESICM 2023 Rec 3.1, ROX cut-offs of Roca et al 2019 and Irwin and Rippe 9e ch. 167.

7. Limitations and cautions

- Who benefits most and **when to escalate** remain undetermined. [I&R ch. 169]
- HFNC does not reliably deliver the higher airway pressures of CPAP/NIV — NIV is the better oxygenator. [I&R ch. 167]
- Early in COVID-19, concern about **aerosol dispersion** led some guidelines to discourage NRS; these concerns have **not panned out**. [I&R ch. 167]
- Hypercapnic failure: use only with mild acidaemia and NIV promptly available. [ATS 2026; conditional, low]

PEARLS — what wins marks

- HFNC = heated, humidified O₂–air mix, set FiO₂, flow up to 60 L/min.
- Mechanisms: consistent FiO₂ · dead-space washout · PEEP 3–5 cm H₂O · ↓RR and work of breathing · humidification/mucociliary clearance.
- FLORALI: intubation lower with HFNT only when PaO₂/FiO₂ <200; mortality lower vs NIV.
- ATS 2026: HFNC over standard O₂ in de novo AHRF — **strong, moderate certainty**; ESICM 2023 Rec 3.1 the same for intubation, no effect on mortality.
- Hypercapnic: HFNC only as alternative to NIV when pH >7.25 (conditional, low).
- ROX = (SpO₂/FiO₂)/RR; ≥4.88 reassuring; failure cut-offs 2.85/3.47/3.85 at 2/6/12 h.
- Trap: HFNC failure with delayed intubation is linked to higher mortality — set an escalation plan on day 1.

Textbook: Irwin and Rippe 9e, ch. 169 "Respiratory Adjunctive Therapies", §Humidity Therapy; §Oxygen Delivery Devices (item 5, HFNC); §Choice of Oxygen Delivery Device. Ch. 173 "Critical Care of Acute Exacerbations of COPD", §Management — Oxygen. Ch. 167 "Mechanical Ventilation—Part II: Noninvasive Mechanical Ventilation", §Key Points; §Immunodeficient Patients; §Hypoxemic Respiratory Failure; §Hypoxemic Respiratory Failure with COVID-19; §Extubation Failure; §Postoperative Respiratory Failure; §Patients with a DNI Status; §Summary. Ch. 163 "ARDS and Pulmonary Edema", §Noninvasive and Partial Support Ventilation. Ch. 168, §High-Flow Nasal Cannula Oxygen. Ch. 8, §Education and Intubation Management. Ch. 10, §Pulmonary Infiltrates in Immunocompromised Patients.

Guidelines: American Thoracic Society, Goel NN et al., "Noninvasive respiratory support for adult patients with acute respiratory failure", *Am J Respir Crit Care Med* 2026;212(9):2135–2158, Summary of Recommendations, PICO 1–2 background and implementation. doi:10.1093/ajrccm/aamag302. ESICM, Grasselli G et al., ARDS guidelines, *Intensive Care Med* 2023;49:727–759, Rec 3.1 and Domain 3 text. doi:10.1007/s00134-023-07050-7. Surviving Sepsis Campaign 2026, Prescott H et al., *Crit Care Med* 2026, oxygenation/HFNC statements (SCCM page). doi:10.1097/CCM.00000000000007075. ERS/ESICM/ESCMID/ALAT, Martin-Loeches I et al., severe CAP guidelines, *Eur Respir J* 2023;61:2200735, Question 2. doi:10.1183/13993003.00735-2022. Roca O et al., "An index combining respiratory rate and oxygenation to predict outcome of nasal high-flow therapy", *Am J Respir Crit Care Med* 2019;199(11):1368–1376 (abstract). doi:10.1164/rccm.201803-0589OC.



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