PATIENT CARE AND MANAGEMENT IN CRITICALLY ILL

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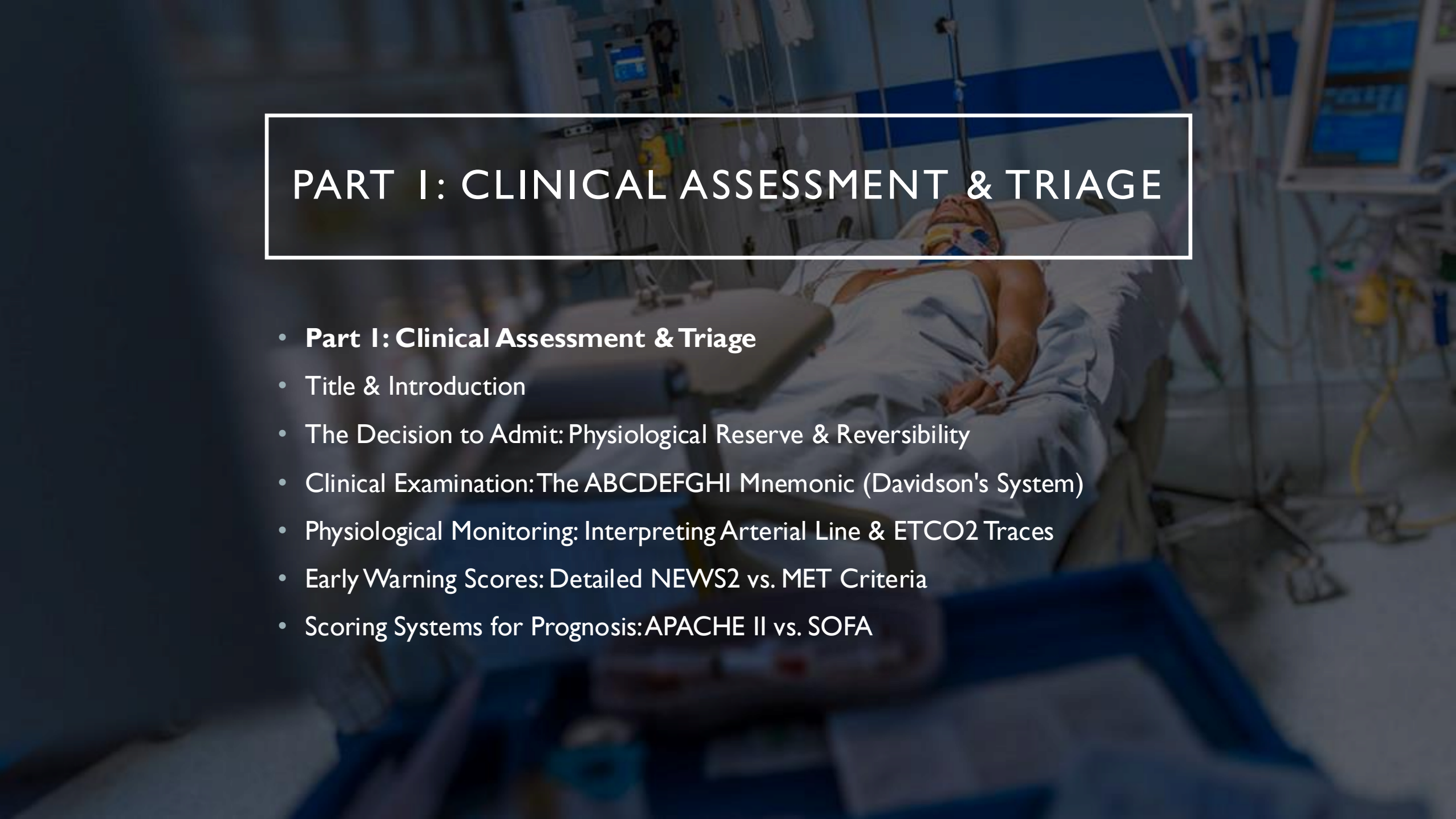
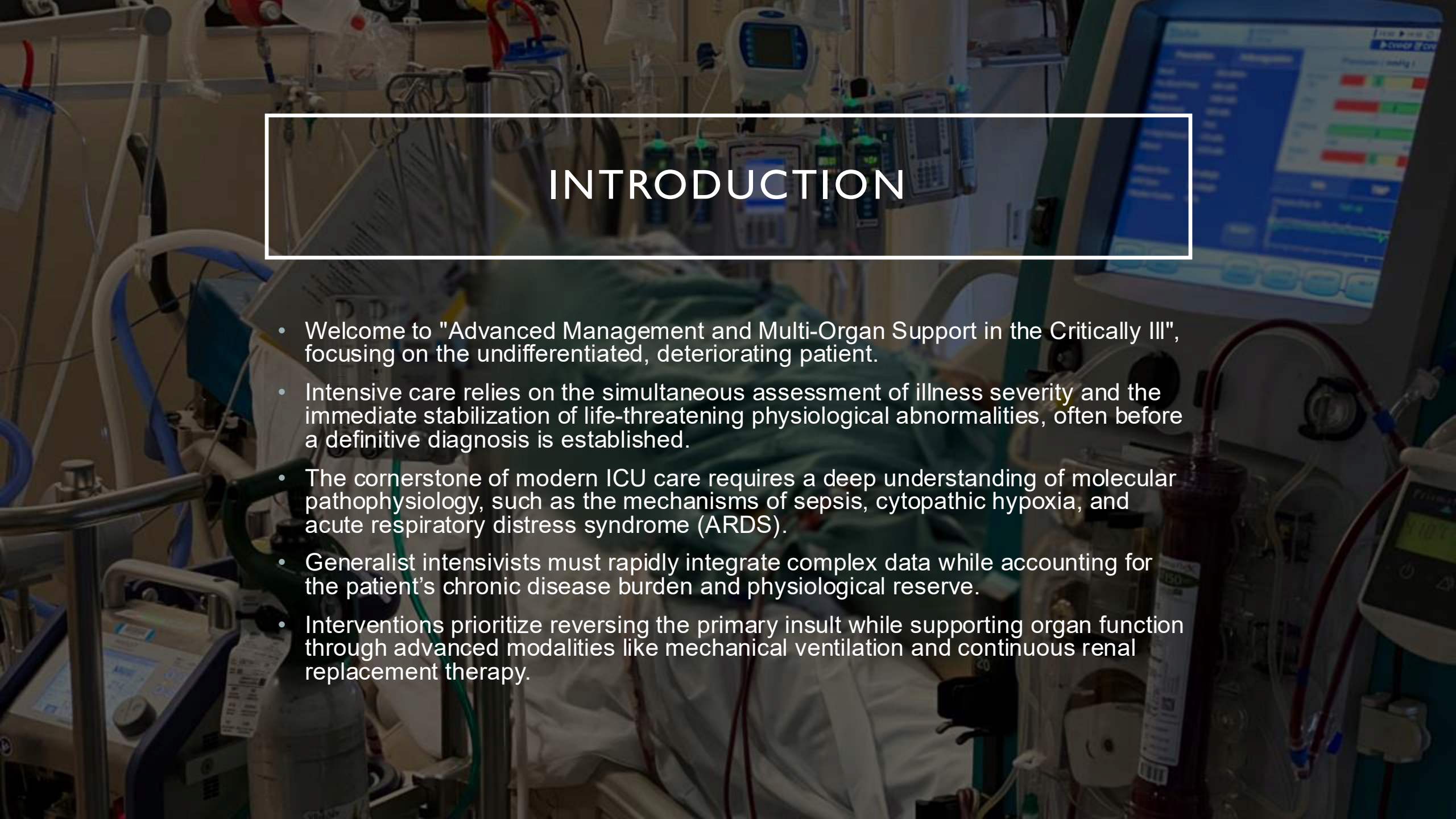
21ST BATCH MBBS 2022 ADMISSION

GOVERNMENT MEDICAL COLLEGE ERNAKULAM



PART 1: CLINICAL ASSESSMENT & TRIAGE

- **Part 1: Clinical Assessment & Triage**
- Title & Introduction
- The Decision to Admit: Physiological Reserve & Reversibility
- Clinical Examination: The ABCDEFGHI Mnemonic (Davidson's System)
- Physiological Monitoring: Interpreting Arterial Line & ETCO2 Traces
- Early Warning Scores: Detailed NEWS2 vs. MET Criteria
- Scoring Systems for Prognosis: APACHE II vs. SOFA



INTRODUCTION

- Welcome to "Advanced Management and Multi-Organ Support in the Critically Ill", focusing on the undifferentiated, deteriorating patient.
- Intensive care relies on the simultaneous assessment of illness severity and the immediate stabilization of life-threatening physiological abnormalities, often before a definitive diagnosis is established.
- The cornerstone of modern ICU care requires a deep understanding of molecular pathophysiology, such as the mechanisms of sepsis, cytopathic hypoxia, and acute respiratory distress syndrome (ARDS).
- Generalist intensivists must rapidly integrate complex data while accounting for the patient’s chronic disease burden and physiological reserve.
- Interventions prioritize reversing the primary insult while supporting organ function through advanced modalities like mechanical ventilation and continuous renal replacement therapy.

THE DECISION TO ADMIT: PHYSIOLOGICAL RESERVE & REVERSIBILITY

- Admission decisions require rigorous evaluation of illness severity, physiological reserve, and the reversibility of the underlying disease process.
- Anticipatory Care Planning (ACP) is essential at admission to determine the appropriate 'ceiling of care' and resuscitation status, respecting the patient's personal beliefs.
- Level 2 care (High-Dependency Unit) supports single failing organ systems (e.g., non-invasive ventilation or moderate vasopressors).
- Level 3 care (ICU) is reserved for multi-organ support or patients requiring endotracheal intubation and invasive mechanical ventilation.
- Decisions must weigh the magnitude of intensive interventions against the realistic hope of returning the patient to a quality of life they find acceptable

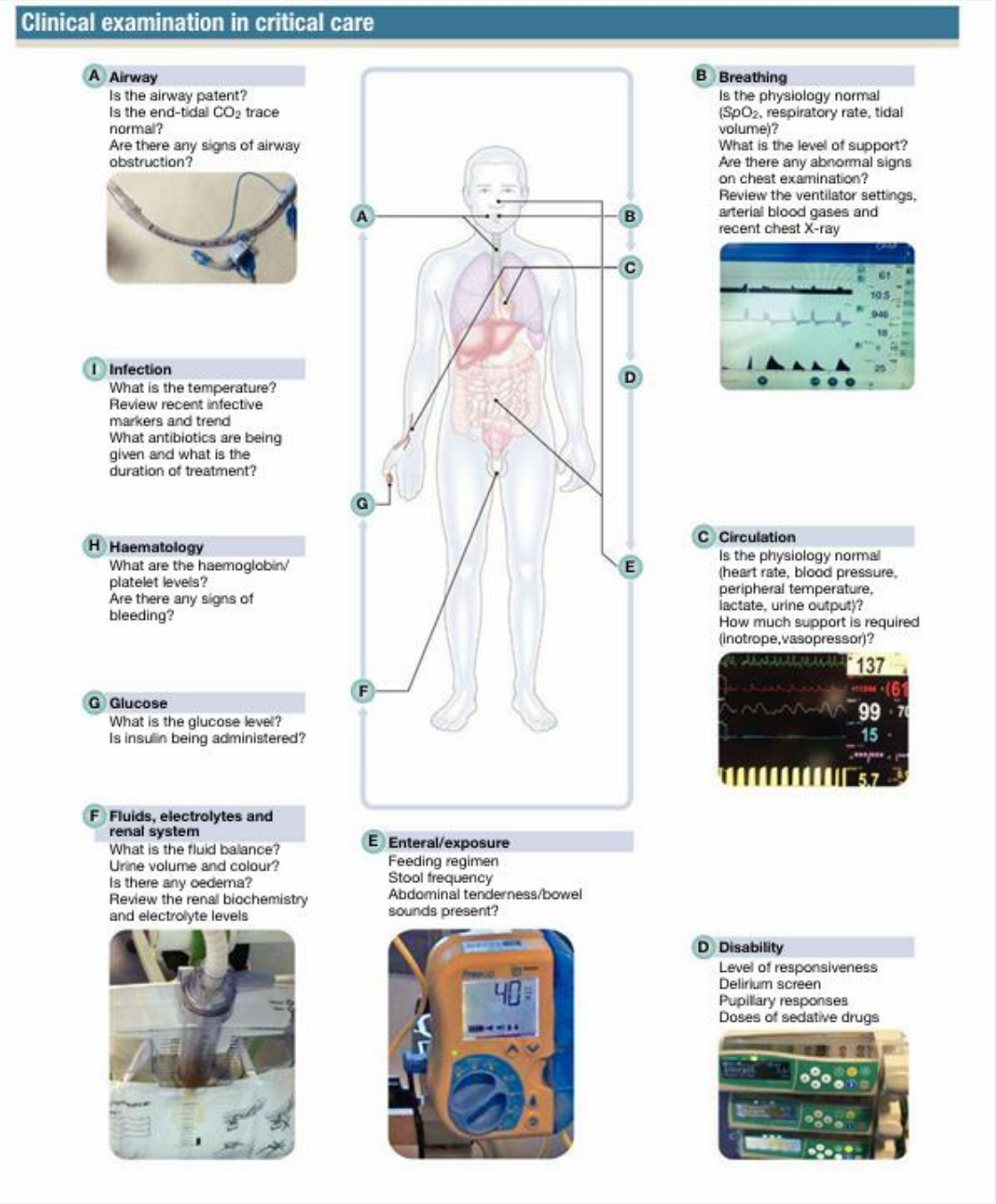
9.23 Levels of care and the corresponding admission criteria for intensive care unit (ICU) and high-dependency unit (HDU)

Level of care	Criteria	Appropriate location
3	Patients requiring/likely to require endotracheal intubation and invasive mechanical ventilatory support Patients requiring support of two or more organ systems (e.g. inotropes and haemofiltration) Patients with chronic impairment of one or more organ systems (e.g. COPD or severe ischaemic heart disease) who require support for acute reversible failure of another organ	ICU
2	Patients requiring detailed observation or monitoring that cannot be provided at ward level: Direct arterial BP monitoring CVP monitoring Fluid balance Neurological observations, regular GCS recording Patients requiring support for a single failing organ system, excluding invasive ventilatory support (IPPV): Non-invasive respiratory support (p. 207) Moderate inotropic or vasopressor support Renal replacement therapy in an otherwise stable patient Step-down from intensive care requiring additional monitoring or single organ support	HDU
1	Patients in whom frequent but intermittent observations and medical review are sufficient	General ward setting

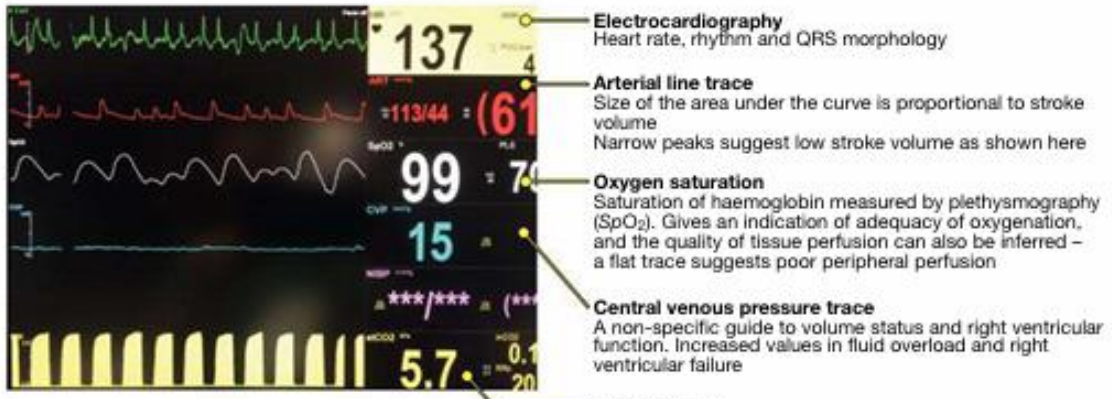
(BP = blood pressure; COPD = chronic obstructive pulmonary disease; CVP =central venous pressure; GCS = Glasgow Coma Scale; IPPV = intermittent positive pressure ventilation)

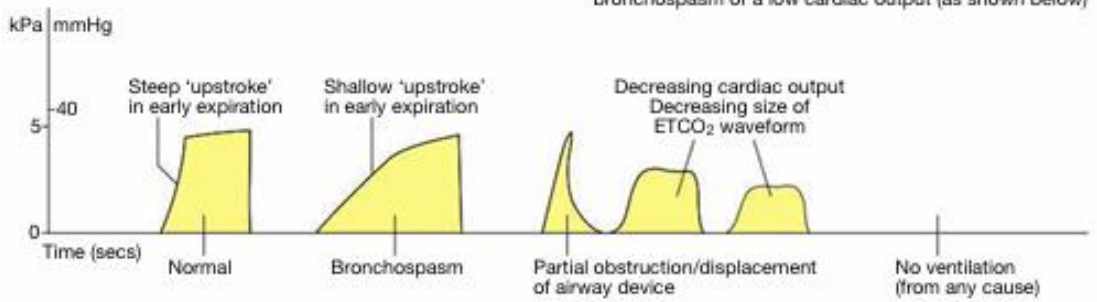
CLINICAL EXAMINATION: THE ABCDEFGHI

- **A**irway: Assess patency and look for normal end-tidal CO_2 ($ETCO_2$)traces.
- **B**reathing: Evaluate SPO_2, respiratory rate, tidal volume, arterial blood gases, and ventilator settings.
- **C**irculation: Assess heart rate, mean arterial pressure (MAP), lactate clearance, urine output, and vasopressor requirements.
- **D**isability: Evaluate Glasgow Coma Scale (GCS), pupillary responses, sedation dosing, and perform daily delirium screening.
- **E**xposure/Enteral: Monitor feeding regimens, bowel sounds, and uncover the patient to look for hidden pathology.
- **F**luids/Renal: Strictly monitor fluid balance, urine output, edema, and renal electrolytes.
- **G**lucose, **H**aematology, & **I**nfection: Maintain glucose targets, assess hemoglobin/platelets, and conduct daily infection surveillance tracking antibiotic duration and temperature.



Monitoring





Bedside physiological data commonly monitored in an intensive care unit setting.

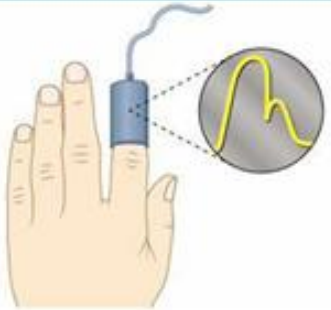
i Pulse oximetry (SpO_2)

Basic principles

- Uses the different red and infrared absorption profiles of oxyhaemoglobin and deoxyhaemoglobin to estimate arterial oxyhaemoglobin saturation (SaO_2)
- Only pulsatile absorption is measured
- A poor trace correlates with poor perfusion

Sources of error

- Carboxyhaemoglobin – absorption profile is the same as oxyhaemoglobin: falsely elevated SpO_2
- Methaemoglobinaemia – SpO_2 will tend towards 85%
- Ambient light/poor application of probe/severe tricuspid regurgitation (pulsatile venous flow): falsely depressed SpO_2
- Reduced accuracy below 80% saturation
- Hyperbilirubinaemia does not affect SpO_2



PHYSIOLOGICAL MONITORING: INTERPRETING ARTERIAL LINE & ETCO2 TRACES

- The area under the arterial line curve is directly proportional to stroke volume; narrow peaks indicate poor stroke volume and potential hypovolaemia.
- Pulse oximetry SPO_2 utilizes the different red and infrared absorption profiles of oxyhaemoglobin and deoxyhaemoglobin.
- SpO_2 has reduced accuracy below 80% and can be falsely elevated by carboxyhaemoglobin, as it shares an absorption profile with oxyhaemoglobin.
- $ETCO_2$ is typically lower than arterial $PaCO_2$ by a variable amount.
- A shallow upstroke in early expiration on an $ETCO_2$ capnograph signifies bronchospasm, while a decreasing wave size indicates a falling cardiac output.
- Central Venous Pressure (CVP) tracing is a non-specific guide to right ventricular function and volume status, elevating in right heart failure and fluid overload

EARLY WARNING SCORES: DETAILED NEWS2 VS. MET CRITERIA

- Rapid response systems aim to identify physiological deterioration early to prevent in-hospital cardiac arrest and allow prompt intervention.
- Medical Emergency Teams (MET) traditionally trigger based on a single parameter (e.g., severe hypotension), offering high sensitivity but low specificity.
- The National Early Warning Score (NEWS2) allocates a score (0-3) to variables including respiratory rate, SpO_2 temperature, BP, heart rate, and neurological response.
- Composite scores like NEWS2 combine high sensitivity with improved specificity, mapping out the cumulative physiological derangement.
- A critical limitation of composite scores is missing a significant single-parameter deterioration (e.g., a drop of 2 GCS points might not breach the composite escalation threshold).

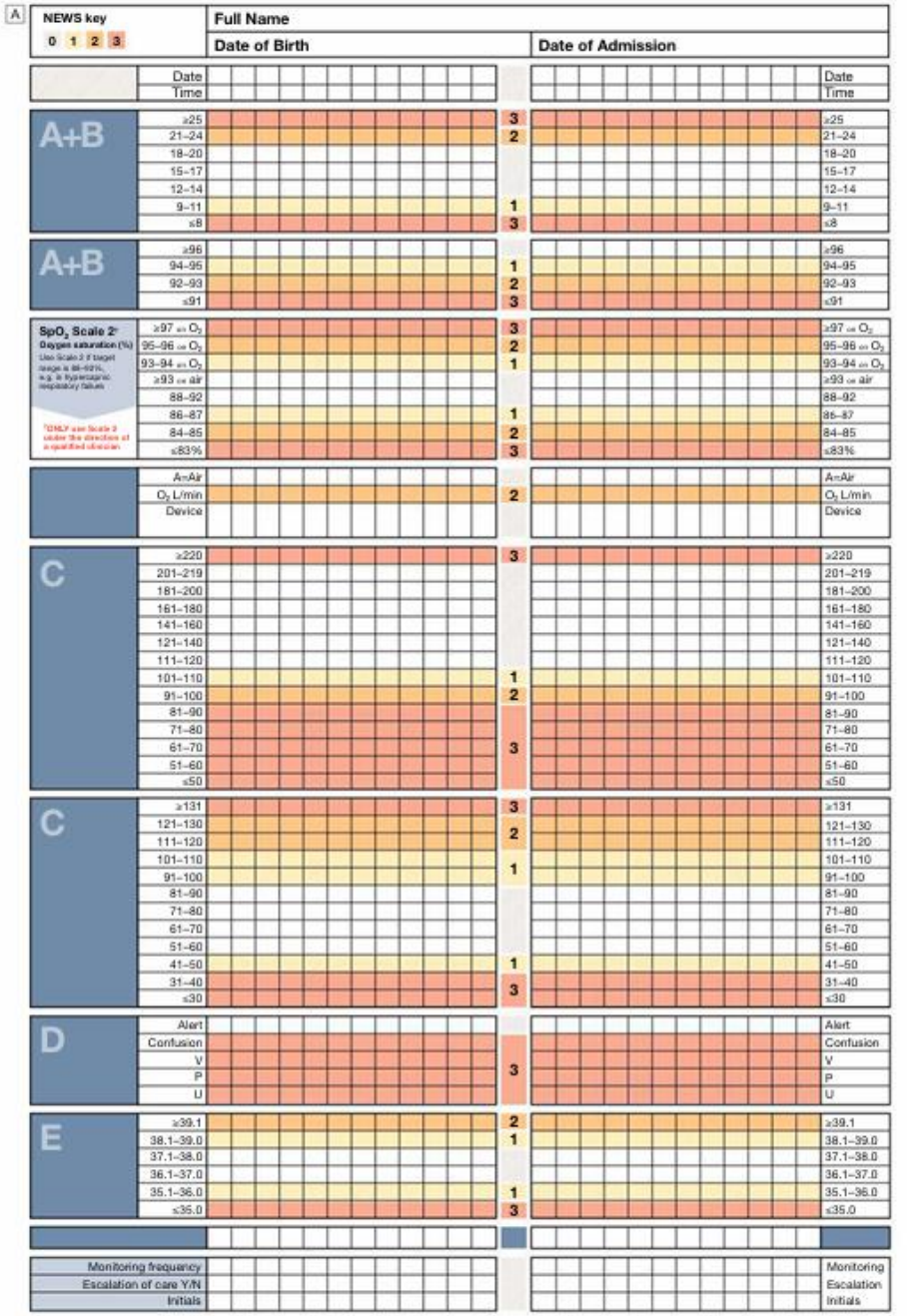


Fig. 9.8 **Identifying and responding to physiological deterioration.** A An example of an early warning score chart. (NEWS = National Early Warning Score; V/P/U = Verbal/Pain/Unresponsive). B (opposite) Responses to physiological deterioration. (ICU = intensive care unit; NEWS = National Early Warning Score) *(A and B) From Royal College of Physicians. National Early Warning Score (NEWS) 2: Standardising the assessment of acute-illness severity in the NHS. Updated report of a working party. London: RCP; December 2017.*

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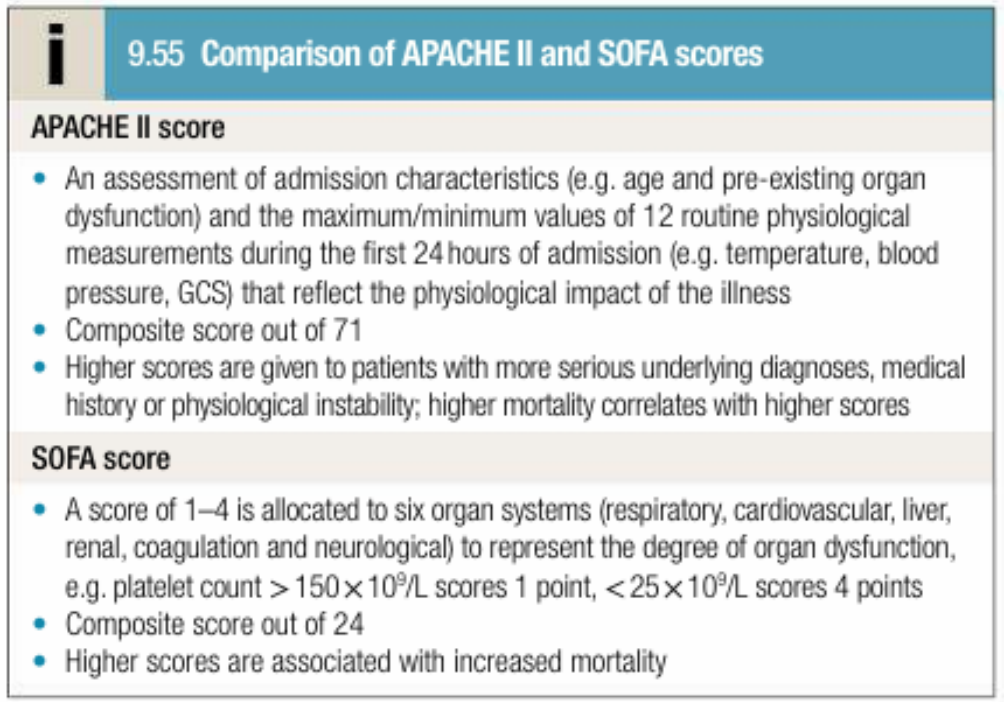
B

NEW score		Frequency of monitoring
0		Minimum 12 hourly • Continue routine NEWS monitoring
Total 1–4	Minimum 4–6 hourly	• Inform registered nurse, who must assess the patient • Registered nurse decides whether increased frequency of monitoring and/or escalation of care is required
3 in single parameter	Minimum 1 hourly	• Registered nurse to inform medical team caring for the patient, who will review and decide whether escalation of care is necessary
Total 5 or more Urgent response threshold	Minimum 1 hourly	• Registered nurse to immediately inform the medical team caring for the patient • Registered nurse to request urgent assessment by a clinician or team with core competencies in the care of acutely ill patients • Provide clinical care in an environment with monitoring facilities
Total 7 or more Emergency response threshold	Continuous monitoring of vital signs	• Registered nurse to immediately inform the medical team caring for the patient – this should be at least at specialist registrar level • Emergency assessment by a team with critical care competencies, including practitioner(s) with advanced airway management skills • Consider transfer to a level 2 or 3 clinical care facility, i.e. higher-dependency unit or ICU • Clinical care in an environment with monitoring facilities

SCORING SYSTEMS FOR PROGNOSIS: APACHE II VS. SOFA

- Critical care scoring systems categorize patient cohorts to measure the severity of illness and predict mortality.
- APACHE II (Acute Physiology Assessment and Chronic Health Evaluation) calculates a score out of 71 based on age, chronic health, and the maximum/minimum values of 12 physiological variables in the first 24 hours.
- SOFA (Sequential Organ Failure Assessment) allocates 1-4 points across six organ systems: respiratory, cardiovascular, liver, renal, coagulation, and neurological.
- The SOFA score totals 24 points; higher scores strongly correlate with increased mortality.
- An acute increase in the SOFA score of >2 points is the foundational criterion for diagnosing sepsis-induced organ dysfunction in the Sepsis-3 definitions.



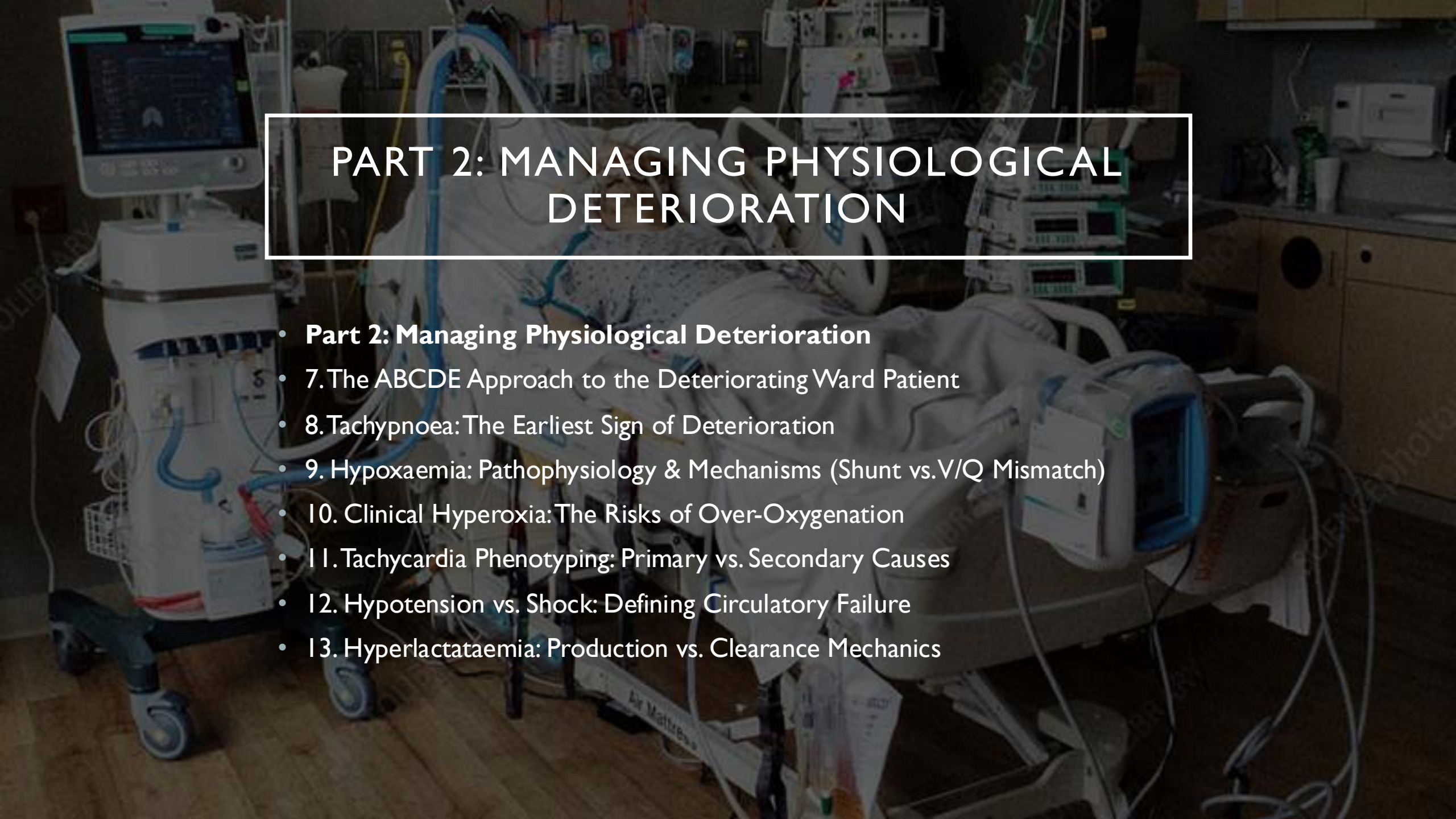
9.55 Comparison of APACHE II and SOFA scores

APACHE II score

- An assessment of admission characteristics (e.g. age and pre-existing organ dysfunction) and the maximum/minimum values of 12 routine physiological measurements during the first 24 hours of admission (e.g. temperature, blood pressure, GCS) that reflect the physiological impact of the illness
- Composite score out of 71
- Higher scores are given to patients with more serious underlying diagnoses, medical history or physiological instability; higher mortality correlates with higher scores

SOFA score

- A score of 1–4 is allocated to six organ systems (respiratory, cardiovascular, liver, renal, coagulation and neurological) to represent the degree of organ dysfunction, e.g. platelet count $>150\times10^9$/L scores 1 point, $<25\times10^9$/L scores 4 points
- Composite score out of 24
- Higher scores are associated with increased mortality



PART 2: MANAGING PHYSIOLOGICAL DETERIORATION

- **Part 2: Managing Physiological Deterioration**
- 7. The ABCDE Approach to the Deteriorating Ward Patient
- 8. Tachypnoea: The Earliest Sign of Deterioration
- 9. Hypoxaemia: Pathophysiology & Mechanisms (Shunt vs. V/Q Mismatch)
- 10. Clinical Hyperoxia: The Risks of Over-Oxygenation
- 11. Tachycardia Phenotyping: Primary vs. Secondary Causes
- 12. Hypotension vs. Shock: Defining Circulatory Failure
- 13. Hyperlactataemia: Production vs. Clearance Mechanics

THE ABCDE APPROACH TO THE DETERIORATING WARD PATIENT

- **C**ontrol: Immediately address massive hemorrhage or lethal arrhythmias (e.g., VT) before airway assessment.
- **A**irway & **B**reathing: Confirm patency, assess work of breathing, check oxygen saturations, and perform early arterial blood gas (ABG) sampling.
- **C**irculation: Perform a focused exam tracking HR, rhythm, blood pressure, JVP, and peripheral perfusion. Peripheral pulse loss indicates severe shock.
- **D**isability: Formally score the Glasgow Coma Scale (GCS) and mandatorily check capillary blood glucose to exclude severe hyper- or hypoglycemia.
- **E**xposure & Evidence: Expose the patient to check for hidden pathology (e.g., rashes, abdominal distension, leg swelling) and gather collateral evidence from charts and family.

i 9.29 Glasgow Coma Scale (GCS)*

Eye opening (E)	
• Spontaneous	4
• To speech	3
• To pain	2
• Nil	1
Best motor response (M)	
• Obeys commands	6
• Localises to painful stimulus	5
• Flexion to painful stimulus or withdraws hand from pain	4
• Abnormal flexion (internal rotation of shoulder, flexion of wrist)	3
• Extensor response (external rotation of shoulder, extension of wrist)	2
• Nil	1
Verbal response (V)	
• Orientated	5
• Confused conversation	4
• Inappropriate words	3
• Incomprehensible sounds	2
• Nil	1
Coma score = E + M + V	
Always present GCS as breakdown, not a sum score (unless 3 or 15)	
• Minimum sum	3
• Maximum sum	15

*Record the best score observed. When the patient is intubated, there can be no verbal response. The suffix 'T' should replace the verbal component of the score, and the remainder of the score is therefore a maximum of 10.

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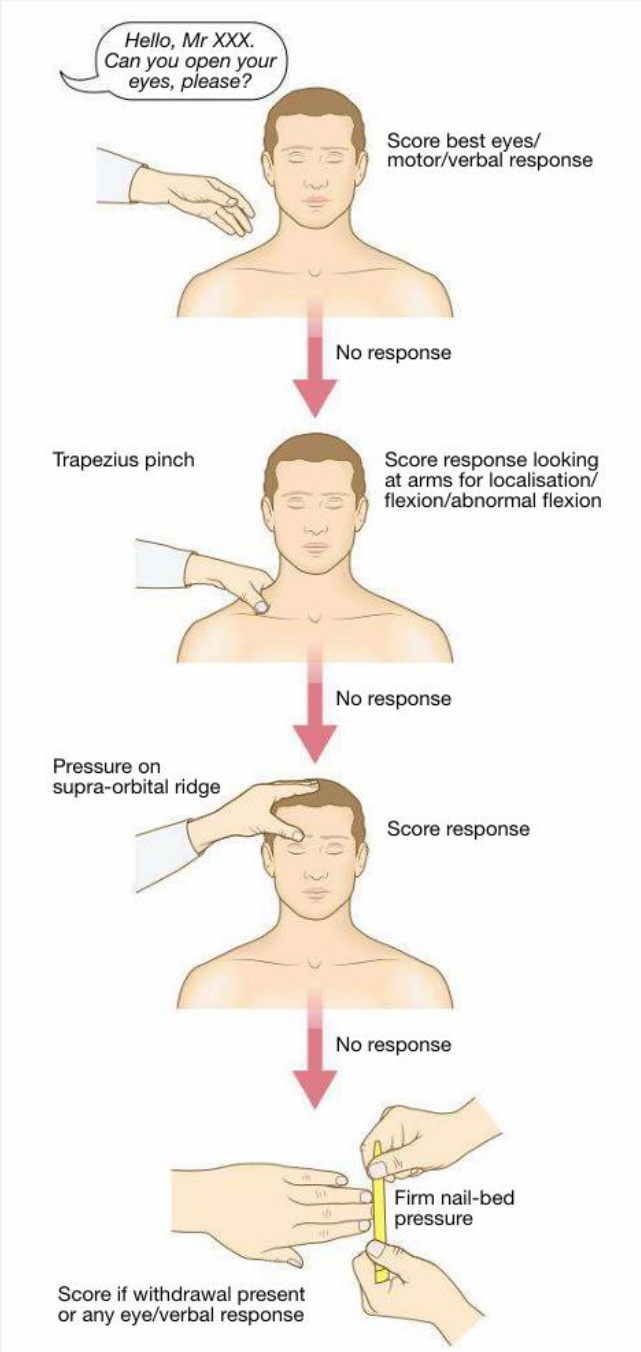

Fig. 9.5 Assessment of the Glasgow Coma Scale (GCS) score in an obtunded patient. Avoid using a sternal rub, as it causes bruising.

TACHYPNOEA: THE EARLIEST SIGN OF DETERIORATION

- Tachypnoea is universally the earliest and most sensitive sign of clinical deterioration in the ward patient.
- Primary tachypnoea originates from intrinsic cardiopulmonary pathology, such as pulmonary edema, pneumonia, or pulmonary embolism.
- Secondary tachypnoea is compensatory hyperventilation driven by metabolic acidosis, commonly seen in sepsis, ketoacidosis, hemorrhage, or visceral ischemia.
- ABG analysis distinguishes the cause: a base deficit > 2 mEq/L (Base Excess < 2 mEq/L) confirms a metabolic driver.
- A lactate > 4 mmol/L (36 mg/dL) combined with tachypnoea mandates immediate escalation to a higher level of care.

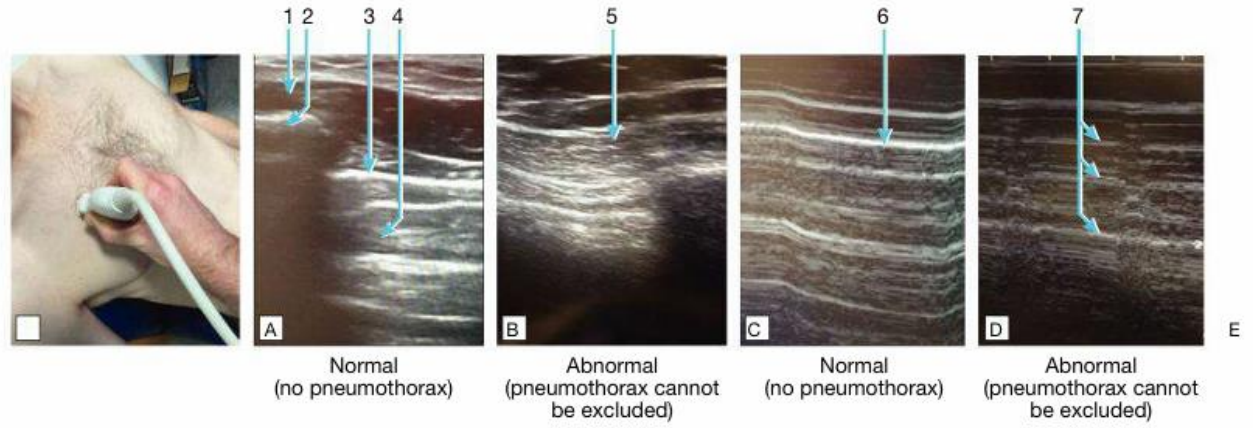

Fig. 9.9 Using ultrasound to rule out an anterior pneumothorax. A Probe position and orientation. B and C Two-dimensional (2D) ultrasound images. D and E M-mode ultrasound images. Key: (1) Intercostal muscle. (2) Rib. (3) Normal bright pleural line –'shimmering appearance' of sliding pleura. (4) Lung. (5) Absent 'shimmering' in pneumothorax and lung not visible. (6) Normal – 'sea shore' sign excludes pneumothorax at that location. The 'sea shore' is represented by the bright granular line with lung (sea) deeper to the bright line. (7) Absent granular pleural line and a repeating linear pattern or 'barcode' sign suggest the presence of pneumothorax.

HYPOXAEMIA: PATHOPHYSIOLOGY & MECHANISMS (SHUNT VS. V/Q MISMATCH)

- Hypoxia is inadequate tissue oxygenation, whereas hypoxaemia specifically denotes a low PaO_2.
- The Bohr effect: Elevated capillary PCO_2 and acidosis shift the oxyhaemoglobin dissociation curve to the right, enhancing O_2 offloading at the tissue level.
- Due to the sigmoidal shape of the curve, SaO_2 plummets dangerously when PaO_2 falls below 8 kPa (60 mmHg).
- **Shunt:** Alveoli are perfused but completely unventilated (e.g., pneumonia, alveolar hemorrhage). Deoxygenated blood bypasses gas exchange, causing Type I respiratory failure.
- **V/Q Mismatch:** Alveoli are under-ventilated relative to their perfusion (e.g., COPD, asthma), leading to an alveolar PO_2 drop and PCO_2 rise (Type II respiratory failure).

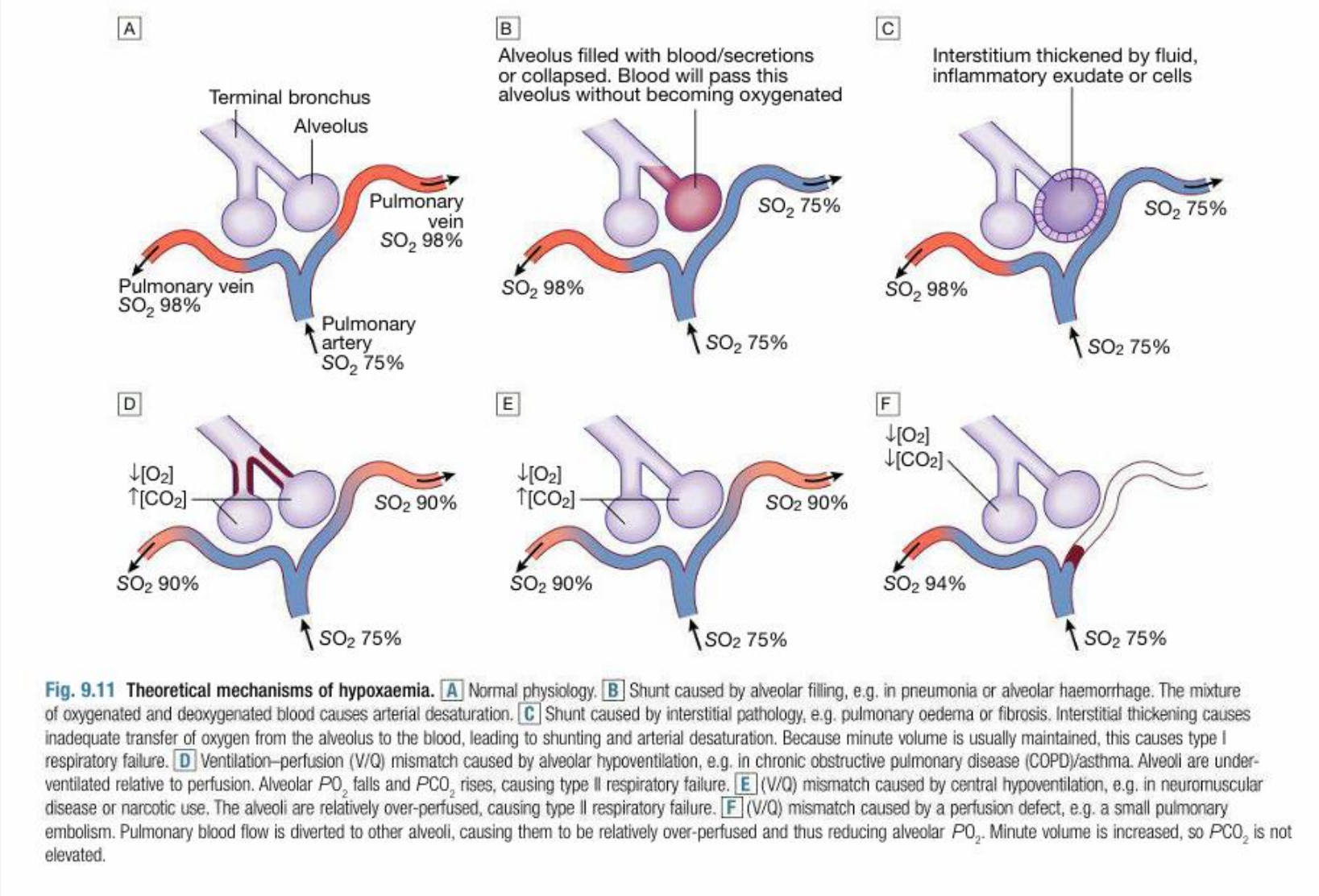

Fig. 9.11 Theoretical mechanisms of hypoxaemia. A Normal physiology. B Shunt caused by alveolar filling, e.g. in pneumonia or alveolar haemorrhage. The mixture of oxygenated and deoxygenated blood causes arterial desaturation. C Shunt caused by interstitial pathology, e.g. pulmonary oedema or fibrosis. Interstitial thickening causes inadequate transfer of oxygen from the alveolus to the blood, leading to shunting and arterial desaturation. Because minute volume is usually maintained, this causes type I respiratory failure. D Ventilation–perfusion (V/Q) mismatch caused by alveolar hypoventilation, e.g. in chronic obstructive pulmonary disease (COPD)/asthma. Alveoli are under-ventilated relative to perfusion. Alveolar PO_2 falls and PCO_2 rises, causing type II respiratory failure. E (V/Q) mismatch caused by central hypoventilation, e.g. in neuromuscular disease or narcotic use. The alveoli are relatively over-perfused, causing type II respiratory failure. F (V/Q) mismatch caused by a perfusion defect, e.g. a small pulmonary embolism. Pulmonary blood flow is diverted to other alveoli, causing them to be relatively over-perfused and thus reducing alveolar PO_2. Minute volume is increased, so PCO_2 is not elevated.

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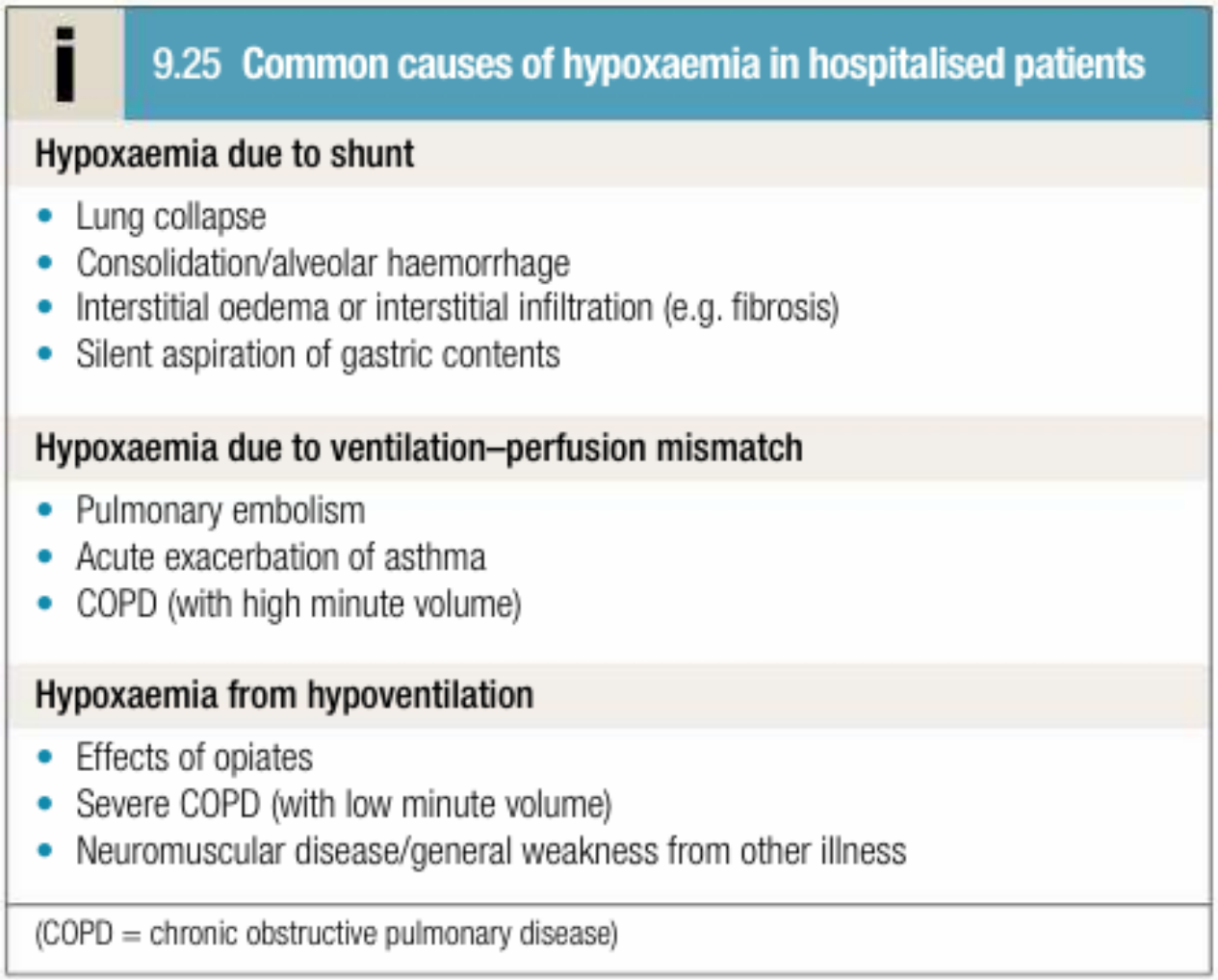


9.25 Common causes of hypoxaemia in hospitalised patients

Hypoxaemia due to shunt

- Lung collapse
- Consolidation/alveolar haemorrhage
- Interstitial oedema or interstitial infiltration (e.g. fibrosis)
- Silent aspiration of gastric contents

Hypoxaemia due to ventilation–perfusion mismatch

- Pulmonary embolism
- Acute exacerbation of asthma
- COPD (with high minute volume)

Hypoxaemia from hypoventilation

- Effects of opiates
- Severe COPD (with low minute volume)
- Neuromuscular disease/general weakness from other illness

(COPD = chronic obstructive pulmonary disease)

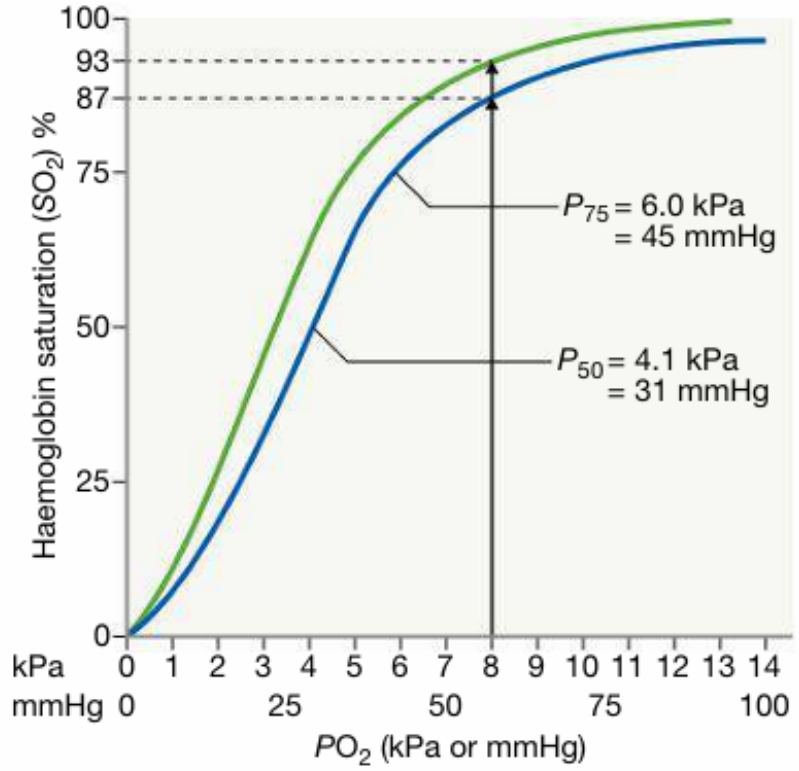


Fig. 9.10 The haemoglobin–oxygen dissociation curve and the effect of CO_2 on oxygen saturations. In pulmonary embolism, compensatory hyperventilation often occurs. $PaCO_2$ decreases, shifting the oxyhaemoglobin saturation curve to the left (green line). Therefore, despite a low PaO_2 (8 kPa/60 mmHg), the oxygen saturation reading is 93%.

9.24 Prescribing oxygen in critical illness

- Oxygen should be prescribed to achieve a target saturation of 94%–98% for most critically unwell patients.
- 88%–92% is a more appropriate target range for those at risk of hypercapnic respiratory failure.
- In patients with acute myocardial infarction or stroke, do not start oxygen unless SpO_2 is below 92%, as hyperoxia may be harmful.

CLINICAL HYPEROXIA: THE RISKS OF OVER-OXYGENATION

- Supplemental oxygen is a drug and must be carefully titrated; the universal target SpO_2 for most critically ill patients is 94-98%.
- Patients at risk of hypercapnic respiratory failure (e.g., severe COPD) should be targeted to an SpO_2 of 88-92%.
- Hyperoxia causes direct free radical-induced tissue damage, which is significantly harmful in acute stroke and myocardial infarction.
- Excessive oxygen impairs ventilation-perfusion matching by overriding protective hypoxic pulmonary vasoconstriction in under-ventilated lung units.
- High oxygen tensions decrease the efficiency of carbon dioxide buffering by oxyhaemoglobin and blunt the hypoxic respiratory drive.

TACHYCARDIA PHENOTYPING: PRIMARY VS. SECONDARY CAUSES

- A heart rate > 110 \beats/min in an adult is abnormal and must never be attributed merely to 'anxiety' without investigation.
- A rate > 160 beats/min indicates severe compromise and should trigger urgent critical care escalation.
- Secondary causes are vastly more common than intrinsic cardiac dysrhythmias and include hypovolaemia, severe sepsis, occult hemorrhage, and hyper-metabolic states.
- Primary dysrhythmias (e.g., AF with rapid ventricular response) often precipitate secondary to another insult like infection.
- If haemodynamic compromise occurs during rapid AF, chemical cardioversion with IV amiodarone is preferred, as it is efficacious and well-tolerated in critical illness.

i **9.28 Hypotension in relation to cardiac output: clinical signs and possible causes**

	High cardiac output	Low cardiac output
Signs	Warm hands Pulsatile head movement High-volume/strong pulse Low venous pressure	Cold/clammy peripheries Peripheral cyanosis Raised venous pressure (except in haemorrhage)
Causes	Sepsis Allergy Drug overdose (e.g. antihypertensive) Acidosis (e.g. diabetic ketoacidosis) Thyrotoxicosis Beri-beri	Bleeding Aortic stenosis and failed compensation Dysrhythmia Obstructive (pulmonary embolism/ tamponade/dynamic hyperinflation as in severe asthma) Chronic heart failure

9.45 Categories of shock

Category	Description
Hypovolaemic	Can be haemorrhagic or non-haemorrhagic in conditions such as hyperglycaemic hyperosmolar state (p. 723) and burns
Cardiogenic	See page 202
Obstructive	Obstruction to blood flow around the circulation, e.g. major pulmonary embolism, cardiac tamponade, tension pneumothorax
Septic	See page 198
Anaphylactic	Inappropriate vasodilatation triggered by an allergen (e.g. bee sting), often associated with endothelial disruption and capillary leak (p. 183)
Neurogenic	Caused by major brain or spinal injury, which disrupts brainstem and neurogenic vasomotor control. High cervical cord trauma may result in disruption of the sympathetic outflow tracts, leading to inappropriate bradycardia and hypotension. Guillain–Barré syndrome (p. 1192) can involve the autonomic nervous system, resulting in periods of severe hypo- or hypertension
Others	e.g. Drug-related such as calcium channel blocker overdose; Addisonian crisis

9.26 Calculation of mean arterial pressure (MAP)

$$\text{MAP} = \text{Diastolic blood pressure} + \frac{(\text{systolic} - \text{diastolic})}{3}$$

At normal heart rates, the heart, on average, spends two-thirds of the cycle in diastole. The MAP reflects this by weighting the value towards the diastolic blood pressure.

HYPOTENSION VS. SHOCK: DEFINING CIRCULATORY FAILURE

- Mean Arterial Pressure (MAP) represents the driving perfusion pressure:MAP = Diastolic BP + (Systolic -Diastolic) / 3.
- A target MAP 65 mmHg maintains adequate renal and end-organ perfusion in the majority of patients.
- Shock is specifically defined as inadequate oxygen delivery (DO_2) failing to meet tissue metabolic demands, resulting in cellular dysoxia.
- Hypotension Shock. Patients can be severely shocked with a normal blood pressure (compensated shock), heavily reliant on profound vasoconstriction.
- Shock is categorized into: Hypovolaemic, Cardiogenic, Obstructive, Septic (Distributive), and Anaphylactic.

HYPERLACTATAEMIA: PRODUCTION VS. CLEARANCE MECHANICS

- A serum lactate > 2 mmol/L (18 mg/dL) denotes hyperlactataemia and is a key criterion for septic shock.
- A lactate level > 8 mmol/L (72 mg/dL) denotes critical severity and is associated with exceptionally high mortality.
- Type A Lactic Acidosis (Production): Driven by anaerobic metabolism due to severe tissue hypoxia, ischemia (e.g., ischemic gut), or profound shock.
- Type B Lactic Acidosis: Driven by aerobic mechanisms, including accelerated glycolysis via exogenous epinephrine/beta-2 adrenoceptor stimulation, or impaired hepatic clearance.
- Clearance of lactate should be actively measured to track the adequacy of acute fluid and vasopressor resuscitation.

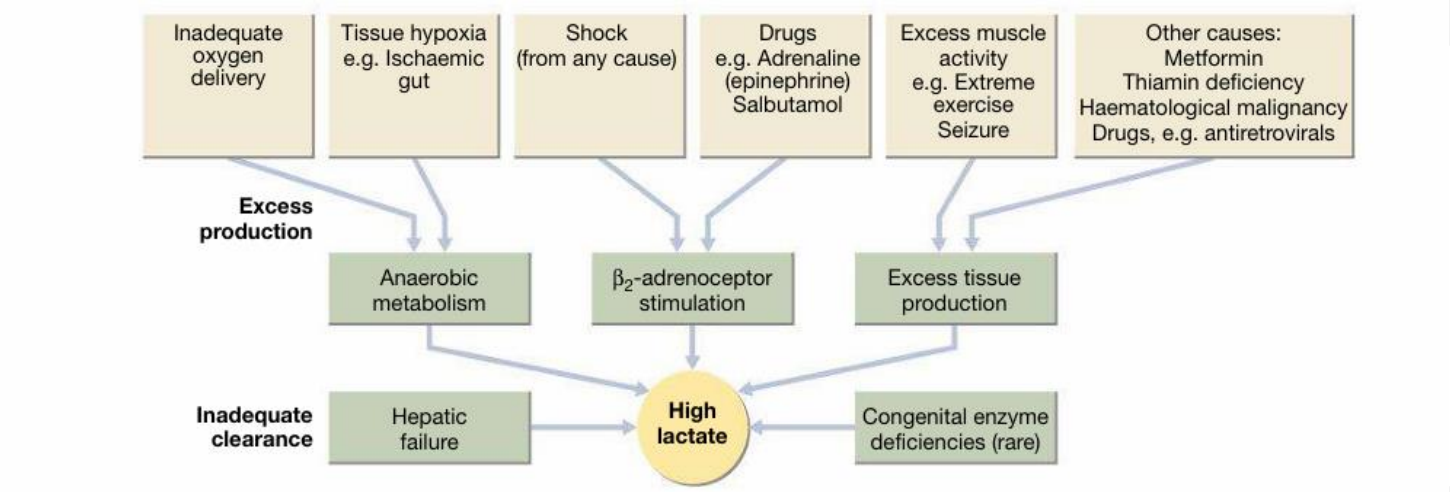


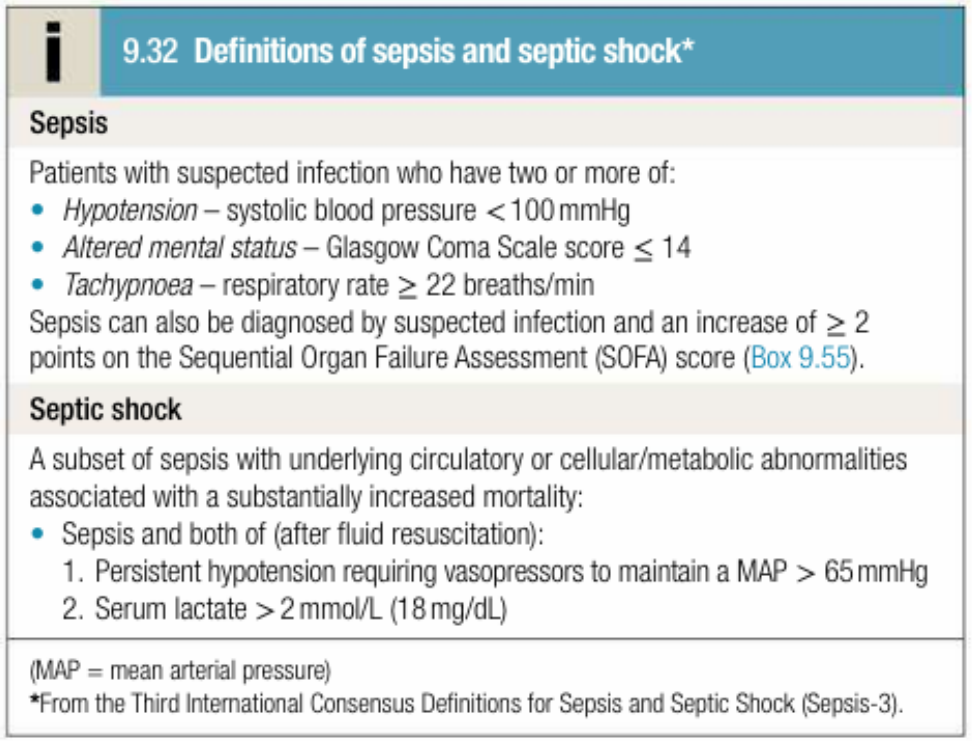
Fig. 9.14 Physiology of hyperlactataemia.

PART 3: SEPSIS & MULTI-ORGAN FAILURE

- **Part 3: Sepsis & Multi-Organ Failure**
- 14. Definitions: Sepsis-3 Criteria & Septic Shock
- 15. Pathophysiology of Sepsis: Macrovascular, Microvascular, & Cellular
- 16. The 'Sepsis Six' & The 1-Hour Management Bundle
- 17. Sepsis Mimics: When it isn't Infection
- 18. ARDS: Pathogenesis & The Berlin Definition

SEPSIS-3 CRITERIA & SEPTIC SHOCK

- **Sepsis:** Life-threatening organ dysfunction caused by a dysregulated host inflammatory response to infection.
- Organ dysfunction is clinically identified by an acute increase in the Sequential Organ Failure Assessment (SOFA) score of >2 points.
- **Septic Shock:** A subset of sepsis involving profound circulatory, cellular, and metabolic abnormalities leading to substantially increased mortality.
- Clinical criteria for Septic Shock include: persistent hypotension requiring vasopressors to maintain a MAP 65 mmHg.
- Additionally, patients must have a serum lactate > 2 mmol/L despite adequate intravenous fluid resuscitation.



9.32 Definitions of sepsis and septic shock*

Sepsis

Patients with suspected infection who have two or more of:

- *Hypotension* – systolic blood pressure < 100 mmHg
- *Altered mental status* – Glasgow Coma Scale score ≤ 14
- *Tachypnoea* – respiratory rate ≥ 22 breaths/min

Sepsis can also be diagnosed by suspected infection and an increase of ≥ 2 points on the Sequential Organ Failure Assessment (SOFA) score (Box 9.55).

Septic shock

A subset of sepsis with underlying circulatory or cellular/metabolic abnormalities associated with a substantially increased mortality:

- Sepsis and both of (after fluid resuscitation):
  1. Persistent hypotension requiring vasopressors to maintain a MAP > 65 mmHg
  2. Serum lactate > 2 mmol/L (18 mg/dL)

(MAP = mean arterial pressure)

*From the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3).

PATHOPHYSIOLOGY OF SEPSIS: MACROVASCULAR, MICROVASCULAR, & CELLULAR

- **Macrovascular:** Severe vasodilatation, hypovolaemia (due to capillary leak), and septic cardiomyopathy reduce effective oxygen delivery despite a paradoxically high cardiac output.
- **Microvascular:** Endothelial damage causes widespread microvascular thrombosis and Disseminated Intravascular Coagulation (DIC), creating functional arteriovenous shunts that bypass tissue beds.
- **Cellular:** Mitochondrial injury results in "cytopathic hypoxia" where cells cannot metabolize available oxygen, and severe injury triggers caspase-mediated apoptosis.
- This extraction failure means central venous oxygen saturations $ScvO_2$ may be paradoxically high (>80%) despite cellular starvation.
- Following the hyper-inflammatory phase, patients enter a period of profound immunosuppression, increasing susceptibility to secondary nosocomial infections.

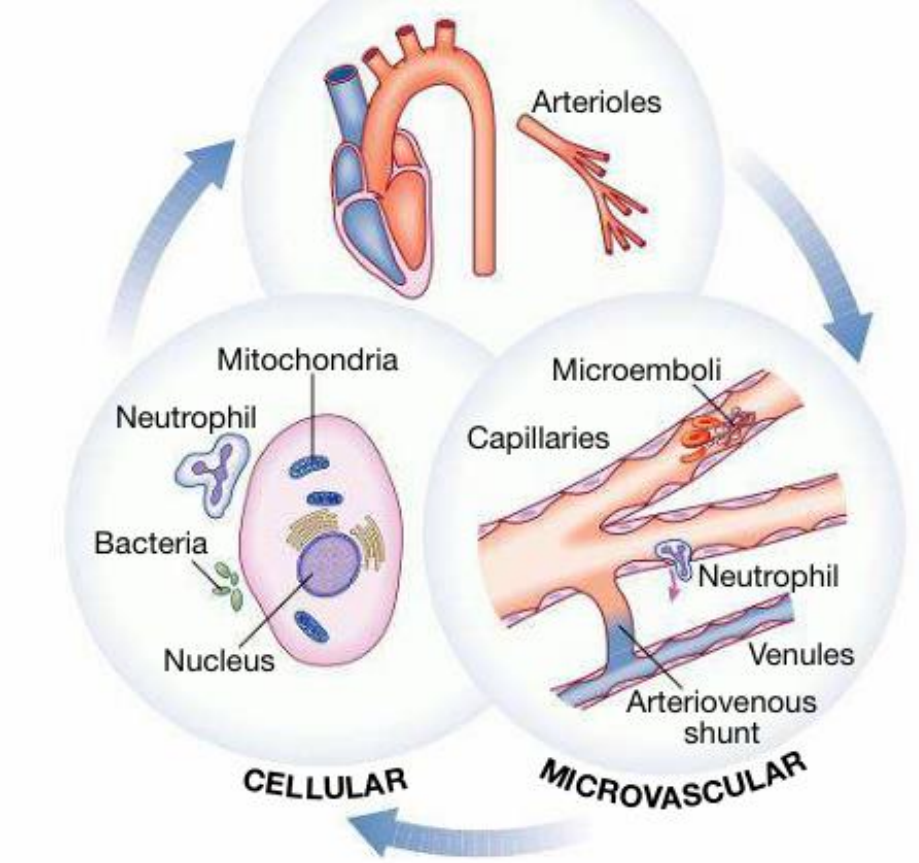
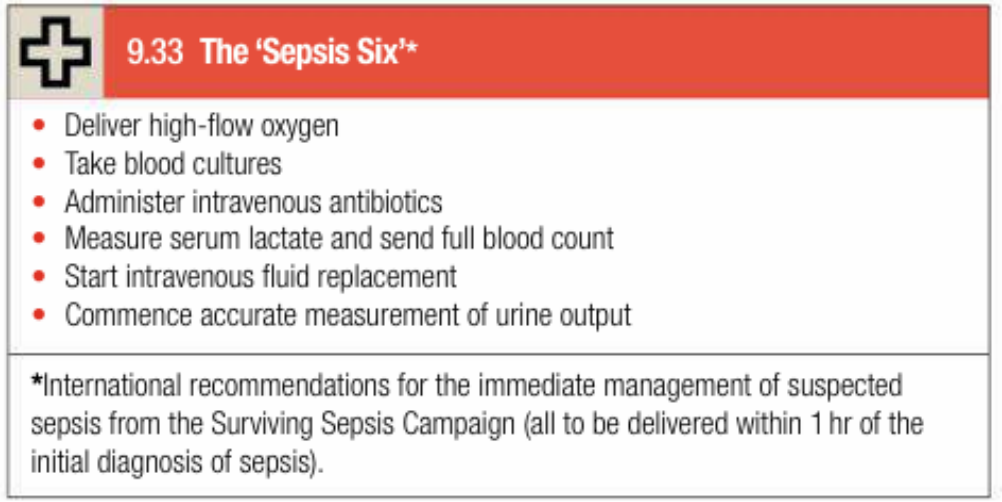


Fig. 9.13 Pathophysiology of organ damage in sepsis. *Macrovascular.* Severe hypovolaemia, vasodilatation or septic cardiomyopathy can reduce oxygen delivery, causing tissue hypoxia. Paradoxically, most patients with sepsis have an increased cardiac output and oxygen delivery. *Microvascular.* Tissue injury can occur from hypoxia secondary to microvascular injury and thrombosis. Damaged epithelium permits neutrophils, proteins and fluid to leak out. *Shunting.* Organs fail in sepsis despite supranormal blood flow. It is likely that arteriovenous shunt pathways exist within vascular beds; these shunts open up in septic shock. *Cellular.* Cells are damaged by a number of mechanisms in sepsis: (1) direct injury by microorganisms; (2) injury from toxins produced by immune cells, e.g. oxygen free radicals; (3) mitochondrial injury causing cytopathic hypoxia – cells are unable to metabolise oxygen; (4) apoptosis – if the cell injury is sufficient, caspase enzymes are activated within the nucleus and programmed cell death occurs; (5) hypoxia from micro- and macrovascular pathology.

THE 'SEPSIS SIX' & THE 1-HOUR MANAGEMENT BUNDLE

- Deliver high-flow oxygen targeting appropriate SpO_2 parameters (94-98%).
- Take blood cultures immediately before antimicrobial administration.
- Administer broad-spectrum intravenous antibiotics ideally within 1 hour of recognition. Every hour of delay increases mortality by 5-10%.
- Measure serum lactate and send a full blood count to assess severity and cellular dysoxia.
- Start rapid IV fluid replacement: administer at least 30 mL/kgof balanced crystalloids within the first 3 hours for hypoperfusion.
- Commence accurate, continuous measurement of hourly urine output to track organ perfusion.



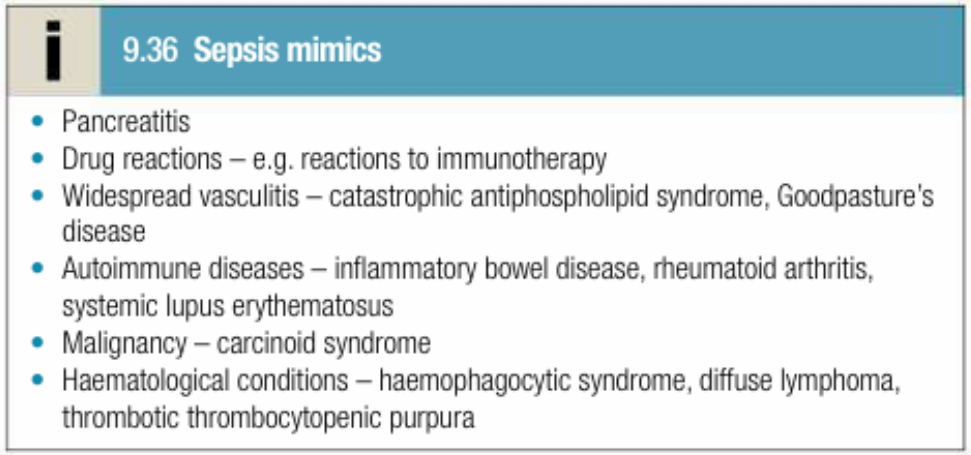
9.33 The 'Sepsis Six'*

- Deliver high-flow oxygen
- Take blood cultures
- Administer intravenous antibiotics
- Measure serum lactate and send full blood count
- Start intravenous fluid replacement
- Commence accurate measurement of urine output

*International recommendations for the immediate management of suspected sepsis from the Surviving Sepsis Campaign (all to be delivered within 1 hr of the initial diagnosis of sepsis).

SEPSIS MIMICS: WHEN IT ISN'T INFECTION

- Up to 20% of patients treated for severe systemic inflammation do not have an underlying infectious aetiology.
- A massive systemic inflammatory response is triggered by the release of danger-associated molecular patterns ('alarmins') from injured tissues.
- Inflammatory mimics: Severe acute pancreatitis, major burns, polytrauma, and severe drug reactions.
- Autoimmune and Vasculitic mimics: Catastrophic antiphospholipid syndrome, Goodpasture’s disease, systemic lupus erythematosus.
- Malignancy and Haematological mimics: Carcinoid syndrome, haemophagocytic lymphohistiocytosis (HLH), thrombotic thrombocytopenic purpura (TTP).

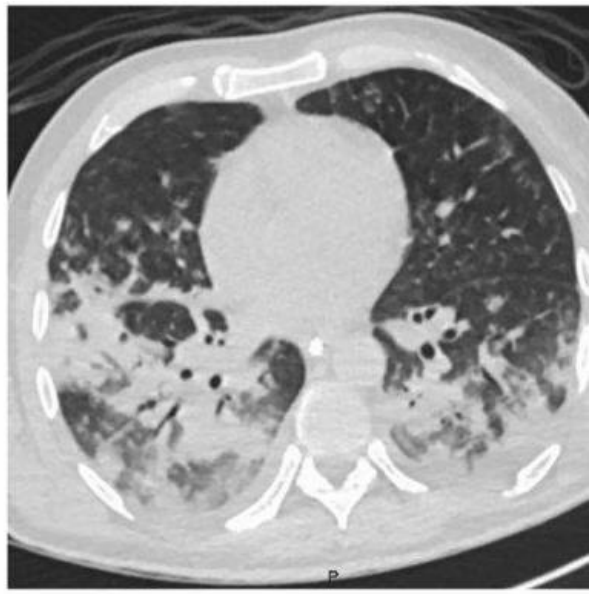


9.36 Sepsis mimics

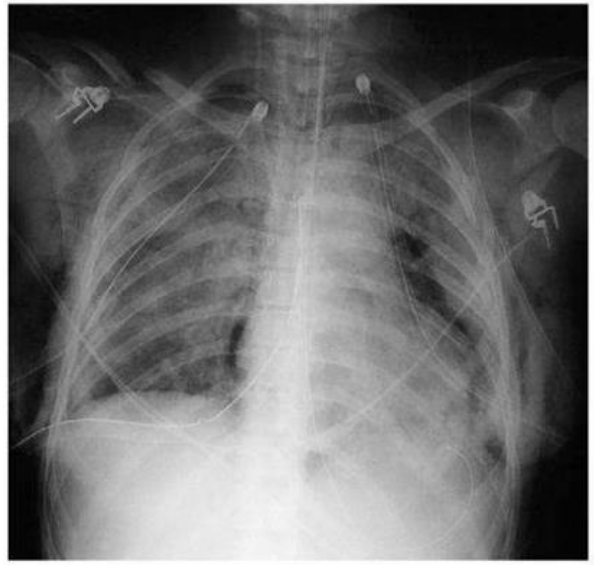
- Pancreatitis
- Drug reactions – e.g. reactions to immunotherapy
- Widespread vasculitis – catastrophic antiphospholipid syndrome, Goodpasture's disease
- Autoimmune diseases – inflammatory bowel disease, rheumatoid arthritis, systemic lupus erythematosus
- Malignancy – carcinoid syndrome
- Haematological conditions – haemophagocytic syndrome, diffuse lymphoma, thrombotic thrombocytopenic purpura

ARDS: PATHOGENESIS & THE BERLIN DEFINITION

- Acute Respiratory Distress Syndrome (ARDS) is a diffuse neutrophilic alveolitis causing increased capillary permeability, protein-rich exudate, and hyaline membrane formation.
- The condition fundamentally causes massive intrapulmonary shunting, profound hypoxaemia, and severe loss of lung compliance (stiff lungs).
- **Berlin Definition:** Acute onset (< 1 week) of bilateral chest X-ray opacities not fully explained by cardiac failure or fluid overload.
- Objective assessment (e.g., echocardiography) is required to exclude hydrostatic pulmonary edema if no clear risk factor for ARDS is present.
- Severity is stratified by the PaO_2/FiO_2 ratio (assessed on PEEP >5 cmH_2O: Mild (200-300 mmHg), Moderate (100-200 mmHg), Severe (<100 mmHg).



g. 9.16 **CT scan of the thorax in a patient with severe ARDS.** Note th
thology is mainly in the dorsal (dependent) parts of the lung.



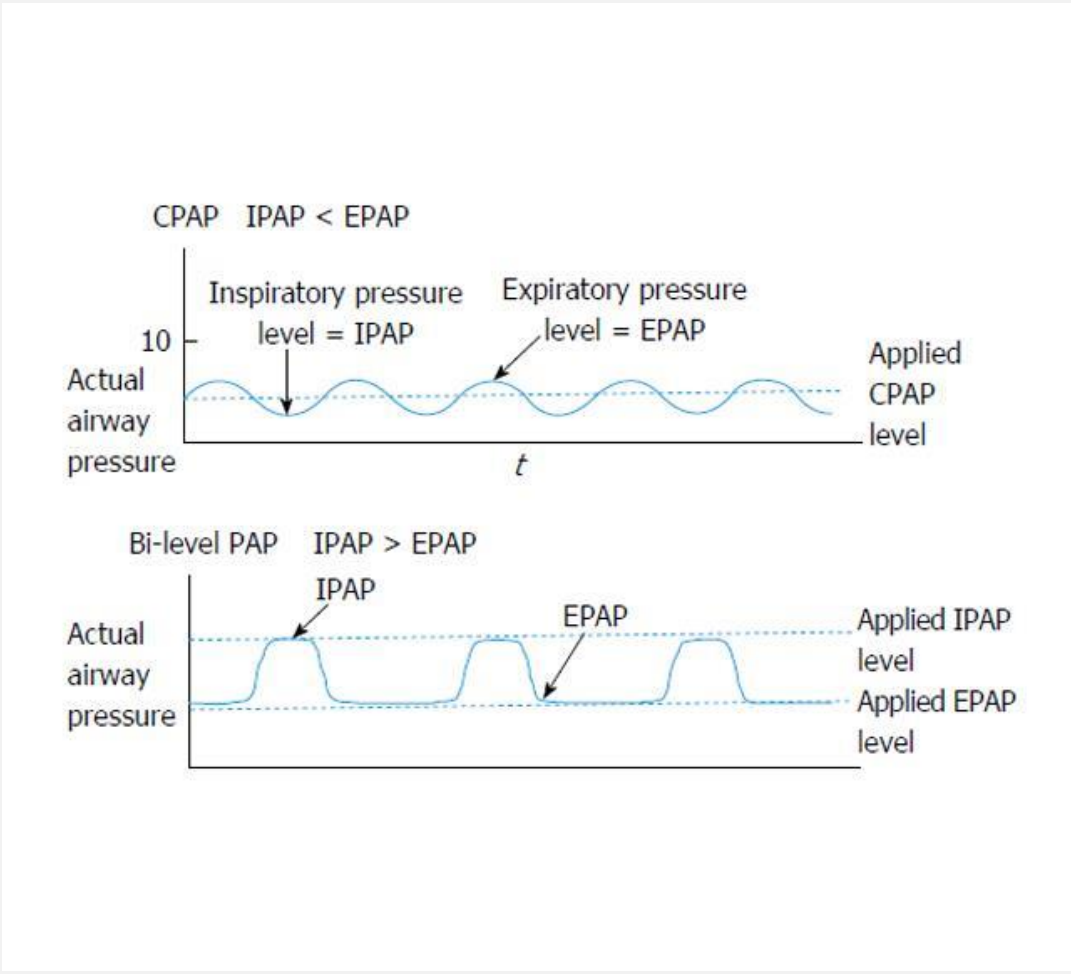
9.15 **Chest X-ray in acute respiratory distress syndrome (ARDS).** Note
eral lung infiltrates, pneumomediastinum, pneumothoraces with bilateral chest
s, surgical emphysema and fractures of the ribs, right clavicle and left scapula.

PART 4: STABILIZATION & ORGAN SUPPORT

- **Part 4: Stabilization & Organ Support**
- 19. Non-Invasive Respiratory Support: HFNC, CPAP, & BiPAP
- 20. Mechanical Ventilation: Lung Protective Strategies (6 \text{ mL/kg} PBW)
- 21. Ventilator-Induced Lung Injury (VILI): Mechanisms of Baro/Volutrauma
- 22. Refractory Hypoxemia: Prone Positioning & VV-ECMO Mechanics
- 23. Cardiovascular Support: Noradrenaline vs. Adrenaline vs. Vasopressin
- 24. Invasive Haemodynamic Monitoring: Interpreting PAC/Swan-Ganz Data
- 25. Renal Support: Continuous (CRRT) vs. Intermittent Dialysis in ICU
- 26. Neurological Support: Managing ICP & Status Epilepticus

NON-INVASIVE RESPIRATORY SUPPORT: HFNC, CPAP, & BIPAP

- **High-Flow Nasal Cannulae (HFNC):** Delivers fully humidified high-flow oxygen, providing variable PEEP and enhanced comfort; superior to NIV for isolated Type I respiratory failure (e.g., pneumonia).
- **CPAP (Continuous Positive Airway Pressure):** Maintains 5-10 cm H_2O continuously to recruit collapsed alveoli and enhance alveolar fluid clearance; highly effective for cardiogenic pulmonary edema and atelectasis.
- **BiPAP (Bi-level NIV):** Delivers distinct inspiratory ($15-25 H_2O and expiratory (4-10 H_2O) pressures to offload the diaphragm and augment tidal volume.
- BiPAP is the first-line therapy for Type II (hypercapnic) respiratory failure secondary to acute exacerbations of COPD.
- Non-invasive support requires a cooperative, spontaneously breathing patient with an intact airway reflex.



MECHANICAL VENTILATION: LUNG PROTECTIVE STRATEGIES (6 ML/KG PBW)

- Mechanical ventilation must prioritize a **Lung-Protective Strategy** to avoid exacerbating lung injury in ARDS.
- Target a tidal volume (TV) of 6 mL/kg based on Predicted Body Weight (PBW), not actual body weight, to avoid overdistension.
- Maintain Plateau Pressure (P_{plat}) < 30 H_2O (measured via an inspiratory hold) to limit stress on the alveoli.
- Maintain Driving Pressure (P_{plat} - PEEP) < 14 H_2O which reflects the strain on the remaining 'functional' recruitable lung volume.
- Utilize higher PEEP in moderate-severe ARDS to maintain functional residual capacity and prevent derecruitment.
- Employ Permissive Hypercapnia (tolerating moderate $PaCO_2$ elevations) to facilitate low tidal volumes, providing Ph >7.20.

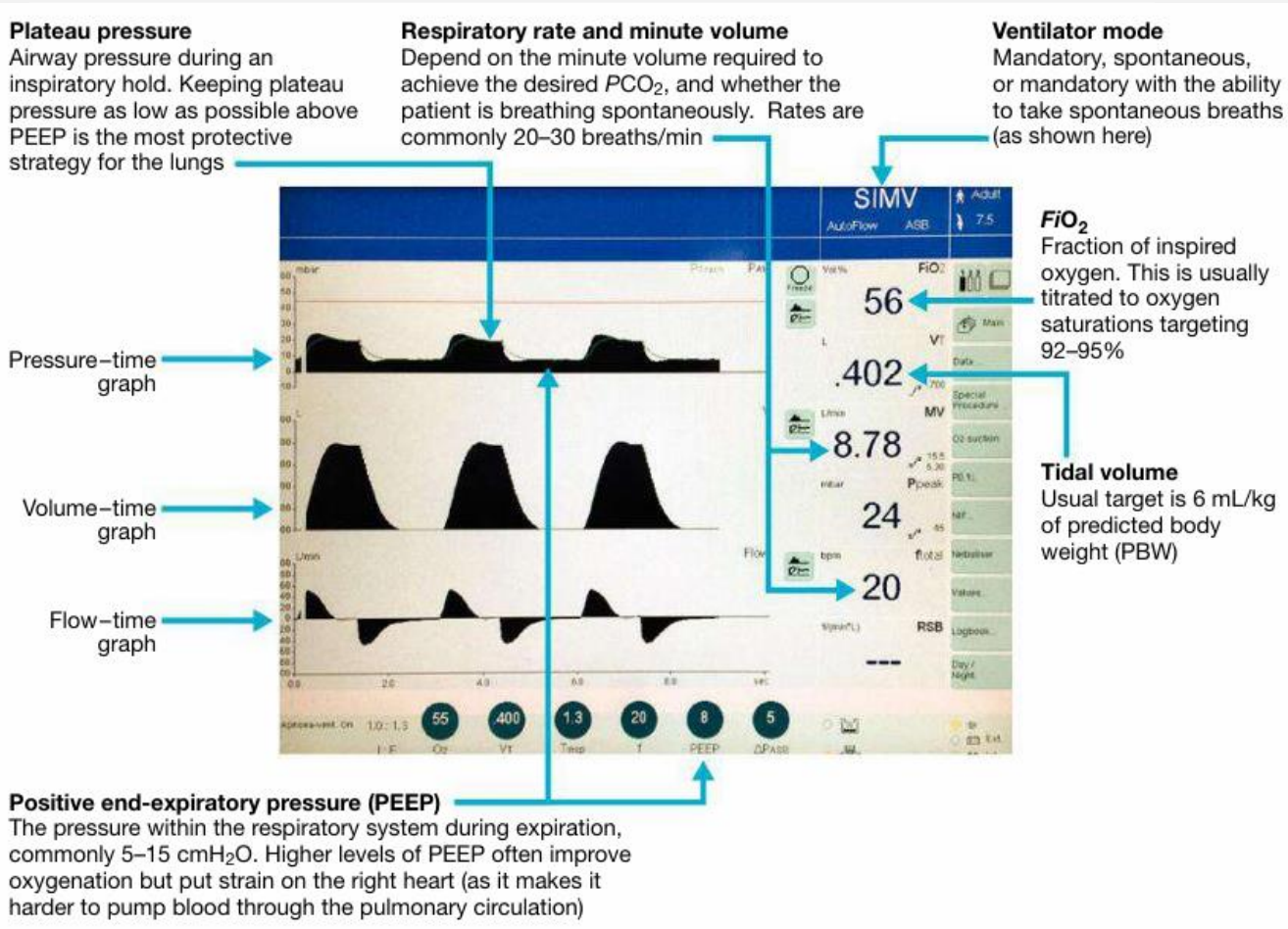
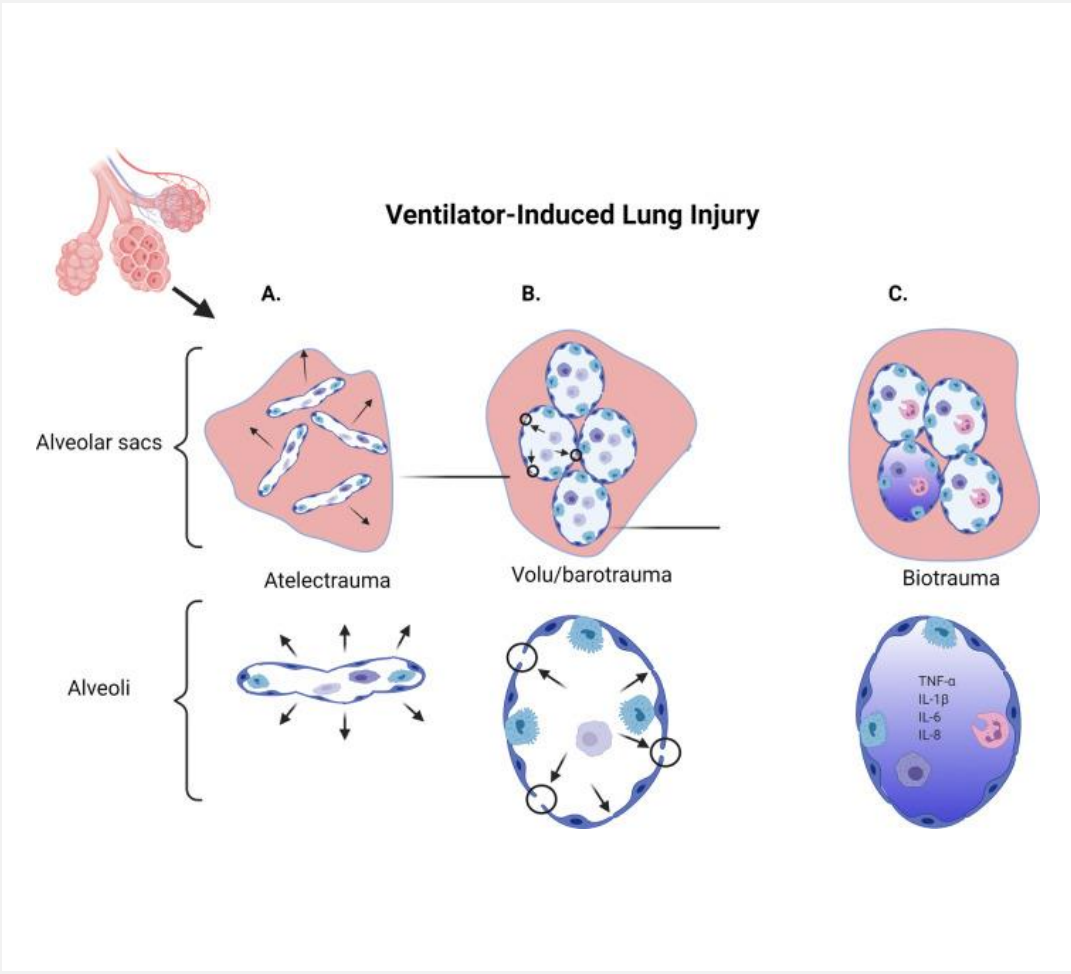


Fig. 9.23 Settings to be considered when commencing mechanical ventilation.

VENTILATOR-INDUCED LUNG INJURY (VILI): MECHANISMS OF BARO/VOLUTRAUMA

- Positive pressure ventilation induces mechanical strain, resulting in Ventilator-Induced Lung Injury (VILI).
- **Volutrauma:** Direct alveolar overdistension caused by excessively large tidal volumes relative to functional lung size.
- **Barotrauma:** Gross structural rupture from high peak/plateau airway pressures, leading to pneumothorax or pneumomediastinum.
- **Atelectrauma:** Shearing stress injury caused by the cyclical opening and collapsing of atelectatic alveoli during tidal breathing.
- **Biotrauma:** Physical stretch triggers profound local and systemic release of inflammatory cytokines, worsening multi-organ failure.



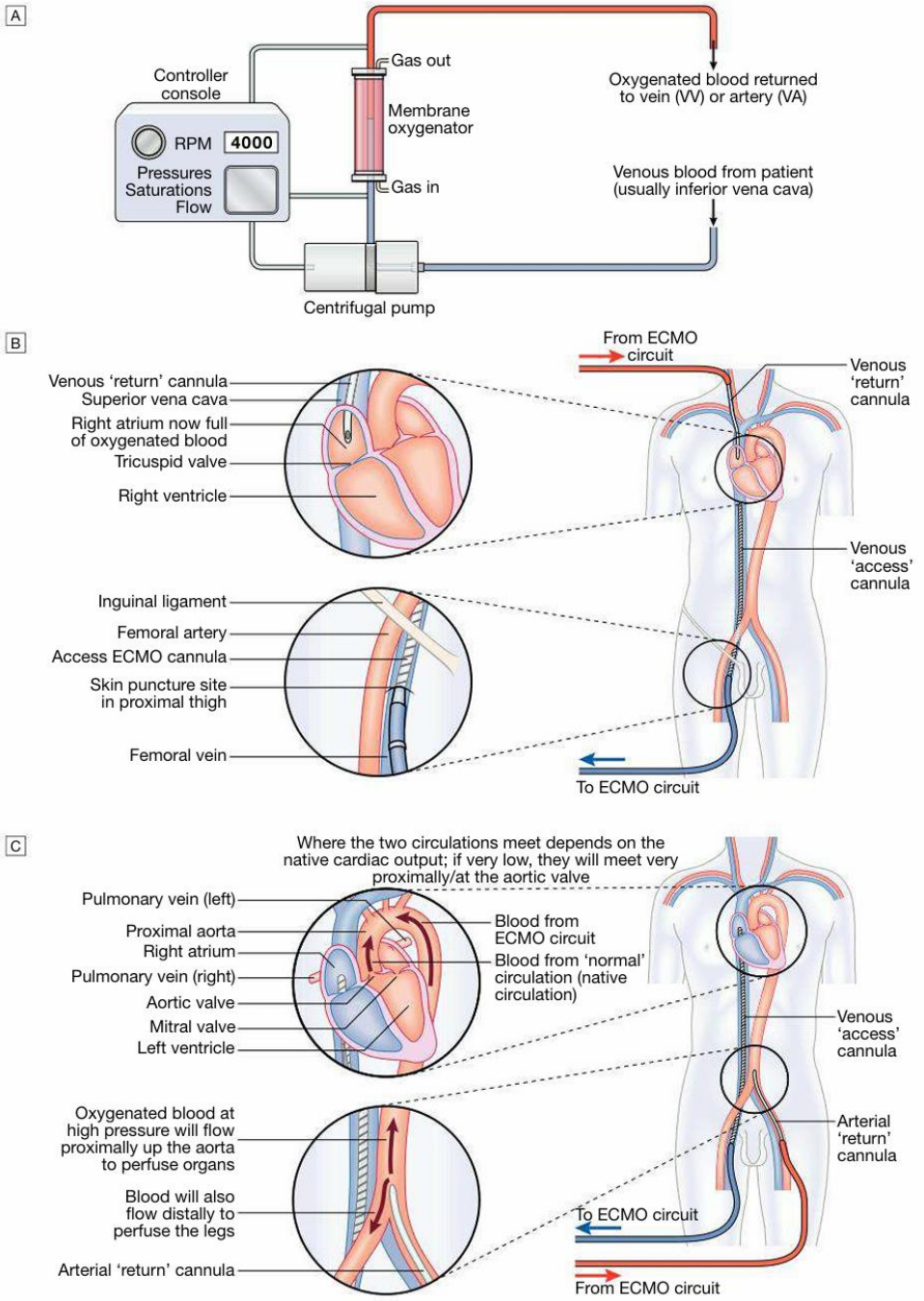


Fig. 9.24 **Principles of extracorporeal membrane oxygenation (ECMO).** A Basic ECMO circuit: venous–arterial (VA) and venous–venous (VV). B Example of a VV ECMO circuit. C Example of a VA ECMO circuit.

REFRACTORY HYPOXEMIA: PRONE POSITIONING & VV-ECMO MECHANICS

- **Prone Positioning:** Indicated for severe ARDS. Placing patients prone alters fluid distribution, recruits posterior alveoli, and dramatically improves V/Q matching.
- Prone ventilation must be sustained for >12 hours/day to reduce mortality and mitigate VILI in dependent lung zones.
- **Extracorporeal Membrane Oxygenation (VV-ECMO):** Used when optimal conventional ventilation (P_{plat} <30) fails to maintain oxygenation or manage profound respiratory acidosis.
- A centrifugal pump extracts venous blood (e.g., from the IVC), drives it through a membrane oxygenator (O_2in, CO_2out), and returns it to the right atrium.
- VV-ECMO provides isolated respiratory support but relies entirely on the patient's native cardiac output to perfuse organs.

CARDIOVASCULAR SUPPORT: NORADRENALINE VS. ADRENALINE VS. VASOPRESSIN

- **Noradrenaline (Norepinephrine):** The universal first-line vasopressor for septic shock. Potent alpha$_1$ vasoconstrictor with modest beta$_1$ inotropy; highly effective at restoring MAP.
- **Vasopressin:** An ADH analogue added (typically when noradrenaline reaches 0.25-0.5 \mu g/kg/min) to combat profound vasoplegia and spare catecholamine doses.
- **Adrenaline (Epinephrine):** A potent beta$_1$ and alpha$_1$ agonist used as a second/third-line agent or in cardiogenic shock. It significantly augments cardiac output but generates aerobic hyperlactataemia.
- **Dobutamine:** A profound beta$_1$ inotrope (with some beta$_2$ vasodilatation) utilized specifically for low cardiac output states, such as septic cardiomyopathy.

9.46 Actions of commonly used vasoactive agents

	Action		
Drug	Vasoconstrictor	Inotrope	Chronotrope
Adrenaline (epinephrine)	++	+++	++
Noradrenaline (norepinephrine)	++++	+	+
Dobutamine	–	++++	+++
Vasopressin	+++++	No action	– (reflex bradycardia)

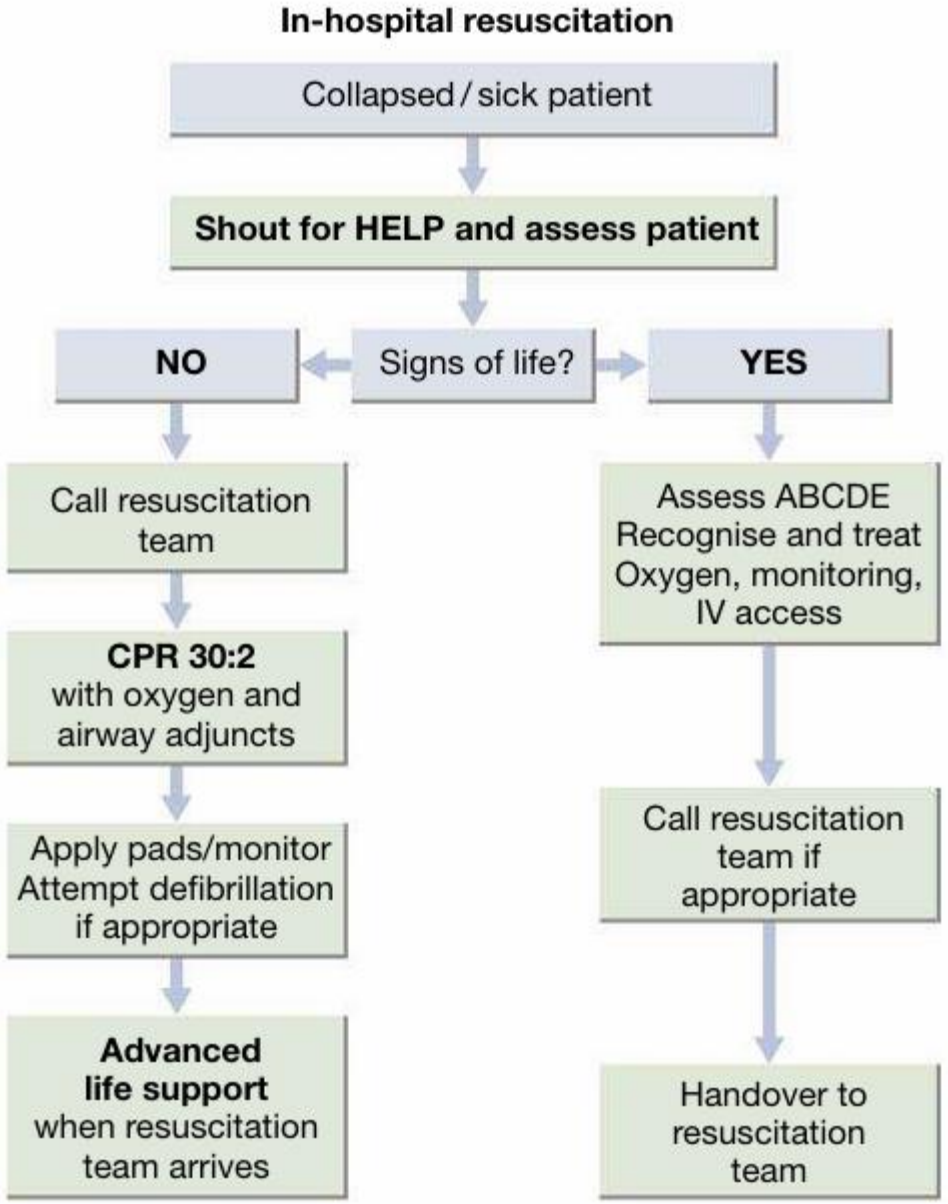


Fig. 9.21 Algorithm for adult basic life support. For further information, see www.resus.org.uk. (CPR = cardiopulmonary resuscitation) *From Resuscitation Council (UK) guidelines:* https://www.resus.org.uk/resuscitation-guidelines/in-hospital-resuscitation/.

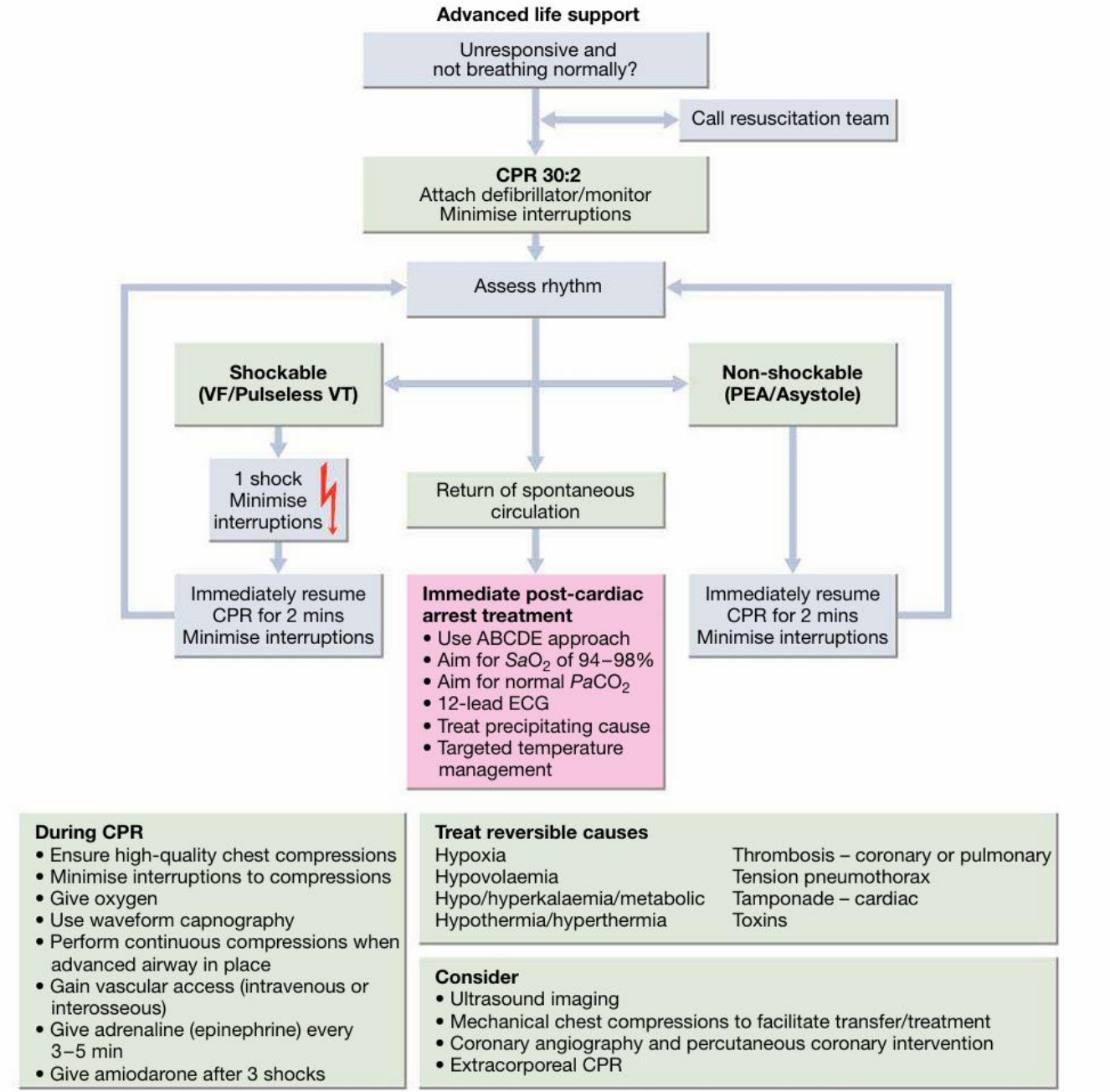


Fig. 9.22 Algorithm for adult advanced life support. For further information, see www.resus.org.uk. (CPR = cardiopulmonary resuscitation: PEA = pulseless electrical activity; VF = ventricular fibrillation; VT = ventricular tachycardia) *From Resuscitation Council (UK) guidelines:* https://www.resus.org.uk/resuscitation-guidelines/adult-advanced-life-support/.

INVASIVE HAEMODYNAMIC MONITORING: INTERPRETING PAC/SWAN-GANZ DATA

- Pulmonary Artery Catheters (PAC / Swan-Ganz) provide invasive measurement of right-sided heart pressures, pulmonary artery pressures, and cardiac output.
- **Pulmonary Artery Capillary Wedge Pressure (PACWP):** Acquired by briefly inflating a balloon in the PA. Normal is 2-10 mmHg; it indirectly reflects Left Atrial filling pressure.
- An elevated PACWP combined with low cardiac index (< 2.2 \ L/min/m^2) defines severe left ventricular failure or cardiogenic shock.
- The **Transpulmonary Gradient** ($PA_{diastolic}$ - PACWP) normally equals 1-5 mmHg; a higher gradient indicates intrinsic pulmonary arterial pathology.
- Mixed venous oxygen saturation (SvO_2), drawn from the distal port, measures whole-body oxygen extraction and delivery balance (normal 70%).

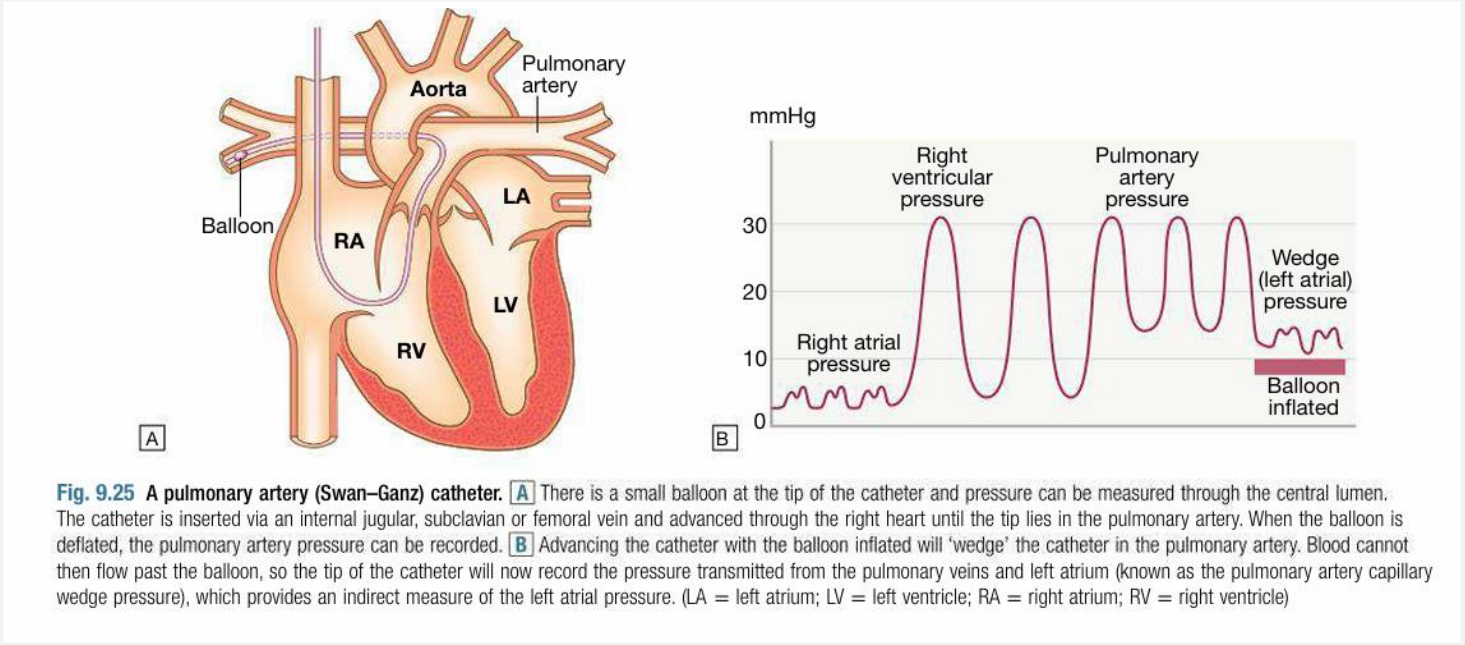
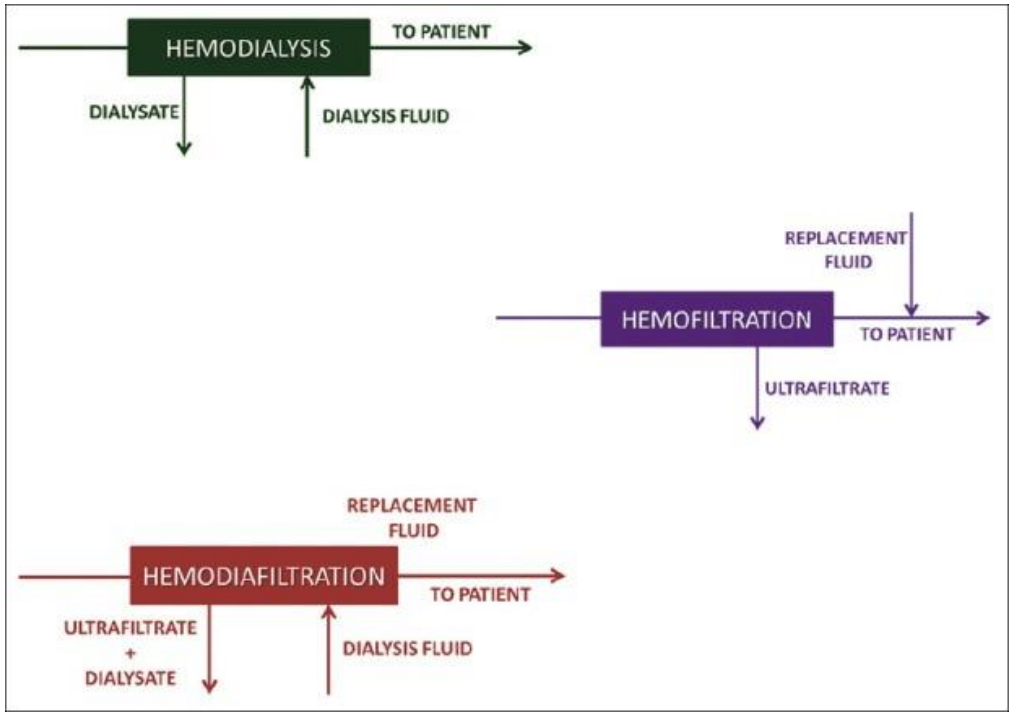


Fig. 9.25 A pulmonary artery (Swan–Ganz) catheter. A There is a small balloon at the tip of the catheter and pressure can be measured through the central lumen. The catheter is inserted via an internal jugular, subclavian or femoral vein and advanced through the right heart until the tip lies in the pulmonary artery. When the balloon is deflated, the pulmonary artery pressure can be recorded. B Advancing the catheter with the balloon inflated will 'wedge' the catheter in the pulmonary artery. Blood cannot then flow past the balloon, so the tip of the catheter will now record the pressure transmitted from the pulmonary veins and left atrium (known as the pulmonary artery capillary wedge pressure), which provides an indirect measure of the left atrial pressure. (LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle)

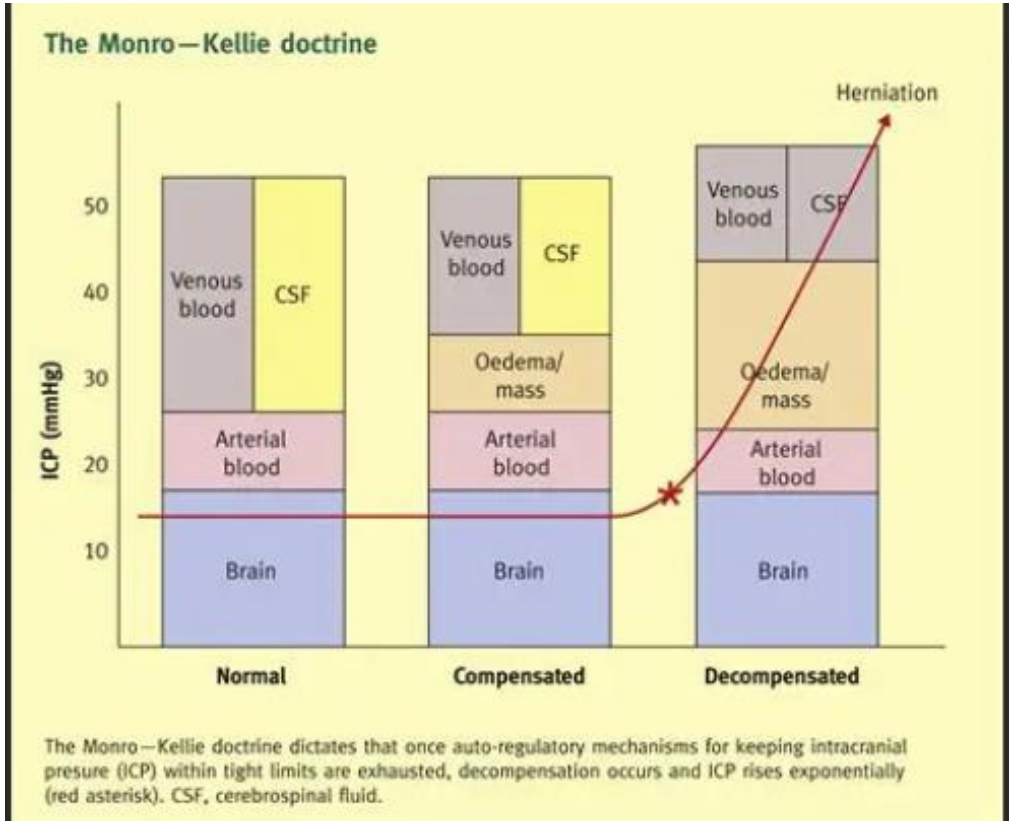
RENAL SUPPORT: CONTINUOUS (CRRT) VS. INTERMITTENT DIALYSIS IN ICU

- **Continuous Renal Replacement Therapy (CRRT):** The modality of choice for critically ill patients with haemodynamic instability, providing gentle, continuous fluid and solute removal.
- **Intermittent Haemodialysis (IHD):** Utilizes high dialysate flows (300-400 mL/min) over 3-4 hours, which can cause severe hypotension, cerebral edema shifts, and repeated ischemic insults to the recovering kidney.
- Solute clearance occurs via **Diffusion** (concentration gradient across a membrane, e.g., CVVHD) or **Convection** ('solvent drag' driven by hydrostatic pressure, e.g., CVVHF).
- CRRT requires continuous anticoagulation to prevent filter clotting; Regional Citrate Anticoagulation is preferred as it minimizes systemic bleeding risks.
- CRRT targets specific urea clearance goals, typically an effluent flow rate of 20-25 mL/kg/hour.

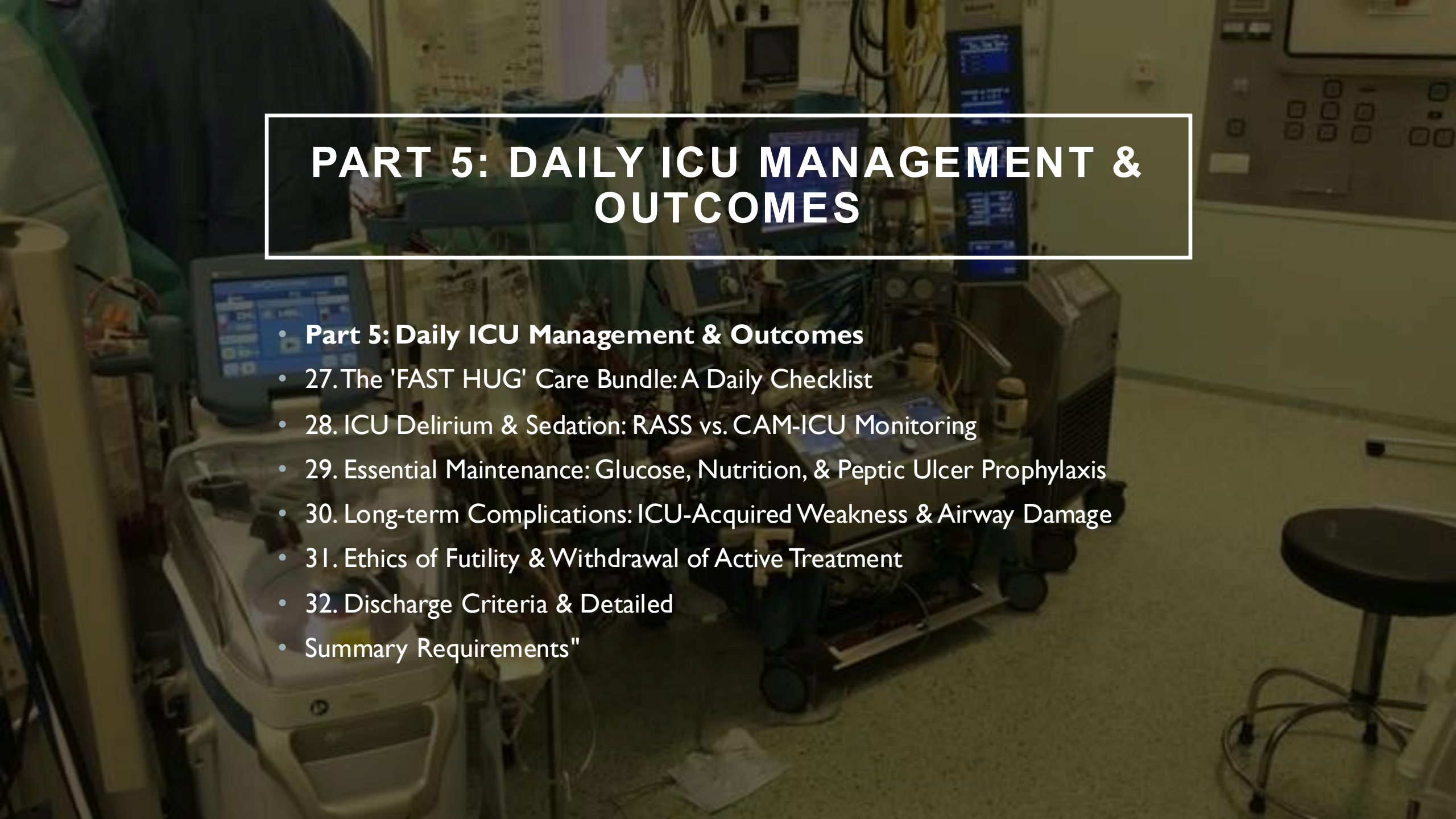


NEUROLOGICAL SUPPORT: MANAGING ICP & STATUS EPILEPTICUS

- The primary goal in acute brain injury is to optimize Cerebral Perfusion Pressure (CPP): CPP = MAP - ICP. The absolute target is CPP > 60 mmHg.
- Normal ICP is < 15 mmHg; sustained ICP > 20-30 mmHg limits cerebral perfusion and risks fatal tentorial herniation.
- Managing raised ICP: Ensure strict normocapnia, elevate the head of the bed 30-45° to facilitate venous drainage, and administer osmotic agents like mannitol or hypertonic saline.
- **Status Epilepticus** vastly increases cerebral oxygen demand and ICP. It must be rapidly terminated.
- Status protocol: First-line benzodiazepines, followed by phenytoin or levetiracetam. Refractory cases require deep suppression with sodium thiopental or ketamine infusions.



The Monro—Kellie doctrine dictates that once auto-regulatory mechanisms for keeping intracranial presure (ICP) within tight limits are exhausted, decompensation occurs and ICP rises exponentially (red asterisk). CSF, cerebrospinal fluid.



PART 5: DAILY ICU MANAGEMENT & OUTCOMES

- **Part 5: Daily ICU Management & Outcomes**
- 27. The 'FAST HUG' Care Bundle: A Daily Checklist
- 28. ICU Delirium & Sedation: RASS vs. CAM-ICU Monitoring
- 29. Essential Maintenance: Glucose, Nutrition, & Peptic Ulcer Prophylaxis
- 30. Long-term Complications: ICU-Acquired Weakness & Airway Damage
- 31. Ethics of Futility & Withdrawal of Active Treatment
- 32. Discharge Criteria & Detailed
- Summary Requirements"

THE 'FAST HUG' CARE BUNDLE: A DAILY CHECKLIST

Daily checklists like the 'FAST HUG' ensure essential evidence-based interventions are not missed during complex multi-organ support.

F - Feeding/Fluids: Initiate early enteral nutrition (<72 hours) to maintain gut barrier integrity.

A - Analgesia: Adopt an "analgesia first" strategy to control pain before increasing sedatives (evaluated via CPOT).

S - Sedation: Minimize continuous infusions; target a lucid, comfortable state (RASS 0 to -1).

T - Thromboprophylaxis: Administer Low-Molecular-Weight Heparin (LMWH) universally unless specifically contraindicated.

H - Head of bed elevation: Maintain >30 degree elevation to prevent micro-aspiration and Ventilator-Associated Pneumonia (VAP).

U - Ulcer Prophylaxis: Use PPIs/H2 blockers to prevent stress ulcers in mechanically ventilated or coagulopathic patients.

G - Glucose Control: Target moderate ranges (144-180 mg/dL or 8-10 mmol/L).

9.49 Richmond Agitation–Sedation Scale (RASS)

Score	Term	Description
+4	Combative	Overtly combative, violent or immediate danger to staff
+3	Very agitated	Pulls on/removes tubes or catheters, or aggressive to staff
+2	Agitated	Frequent non-purposeful movement or patient–ventilator dyssynchrony
+1	Restless	Anxious or apprehensive but no aggressive or vigorous movements
0	Alert and calm	
−1	Drowsy	Not fully alert but sustained awakening (> 10 secs) with eye opening/contact to voice
−2	Light sedation	Brief awakening (< 10 secs) with eye contact to voice
−3	Moderate sedation	Movement but no eye contact to voice
−4	Deep sedation	Movement to physical stimulation but no response to voice
−5	Unrousable	No response to voice or physical stimulation

ICU DELIRIUM & SEDATION: RASS VS. CAM-ICU MONITORING

- **RASS (Richmond Agitation-Sedation Scale):** Ranges objectively from +4 (Combative) to -5 (Unrousable). The target for most ventilated patients is 0 (Alert and calm) to -1 (Drowsy, maintains eye contact >10s).
- Routine deep sedation (RASS -4 to -5) is associated with prolonged ventilation, VAP, and severe ICU delirium.
- **CAM-ICU (Confusion Assessment Method):** A validated tool to screen for delirium. It requires the patient to be responsive to verbal stimuli (RASS >-3).
- CAM-ICU diagnosis requires: 1. Acute onset or fluctuating course AND 2. Inattention.
- PLUS EITHER: 3. Altered level of consciousness (RASS other than 0) OR 4. Disorganized thinking.

ESSENTIAL MAINTENANCE: GLUCOSE, NUTRITION, & PEPTIC ULCER PROPHYLAXIS

GLUCOSE CONTROL: STRESS HYPERGLYCAEMIA EXACERBATES CRITICAL ILLNESS. INITIATE IV INSULIN IF BLOOD GLUCOSE IS >180 MG/DL (10 MMOL/L).

TARGET BLOOD GLUCOSE BETWEEN 144-180 MG/DL (8-10 MMOL/L) TO STRIKE A BALANCE BETWEEN TOXICITY AND FATAL IATROGENIC HYPOGLYCEMIA.

NUTRITION: HYPERCATABOLISM LEADS TO RAPID MUSCLE WASTING. TARGET 1.1 - 2.5 G/KG/DAY OF PROTEIN DURING CONTINUOUS RENAL SUPPORT.

ENTERAL FEEDING IS VASTLY PREFERRED OVER PARENTERAL NUTRITION TO PREVENT MUCOSAL ATROPHY AND REDUCE INFECTIOUS COMPLICATIONS.

STRESS ULCER PROPHYLAXIS (SUP): PPIS REDUCE CLINICALLY SIGNIFICANT GI BLEEDING, BUT AGGRESSIVELY INCREASE RISKS FOR VAP AND *C. DIFFICILE*.

LONG-TERM COMPLICATIONS: ICU-ACQUIRED WEAKNESS & AIRWAY DAMAGE

- **Critical Illness Polyneuropathy:** Symmetrical, distal axonal nerve loss resulting in profound weakness but preserved sensory function; characterized by reduced action potential amplitudes.
- **Critical Illness Myopathy:** Intracellular loss of myosin filaments and muscle necrosis, frequently exhibiting a normal creatine kinase (CK) level.
- Risk factors for both include prolonged mechanical ventilation, use of neuromuscular blockers (NMBAs), high-dose corticosteroids, and poor glycemic control.
- **Airway Complications:** Prolonged endotracheal intubation or tracheostomy risks ischemic damage to tracheal cartilage, leading to late tracheal stenosis or tracheoesophageal fistulae.
- Post-Intensive Care Syndrome (PICS) encompasses long-term physical, cognitive, and emotional impairments requiring structured rehabilitation.

9.51 UK criteria for the diagnosis of brain death

Preconditions for considering a diagnosis of brain death

- The patient is deeply comatose:
  a. There must be no suspicion that coma is due to depressant drugs, such as narcotics, hypnotics, tranquillisers
  b. Hypothermia has been excluded – rectal temperature must exceed 35°C
  c. There is no profound abnormality of serum electrolytes, acid–base balance or blood glucose concentrations, and any metabolic or endocrine cause of coma has been excluded
- The patient is maintained on a ventilator because spontaneous respiration has been inadequate or has ceased. Drugs, including neuromuscular blocking agents, must have been excluded as a cause of the respiratory failure
- The diagnosis of the disorder leading to brain death has been firmly established. There must be no doubt that the patient is suffering from irremediable structural brain damage

Tests for confirming brain death

- All brainstem reflexes are absent:
  a. The pupils are fixed and unreactive to light
  b. The corneal reflexes are absent
  c. The vestibulo-ocular reflexes are absent – there is no eye movement following the injection of 20 mL of ice-cold water into each external auditory meatus in turn
  d. There are no motor responses to adequate stimulation within the cranial nerve distribution
  e. There is no gag reflex and no reflex response to a suction catheter in the trachea
- No respiratory movement occurs when the patient is disconnected from the ventilator for long enough to allow the carbon dioxide tension to rise above the threshold for stimulating respiration ($PaCO_2$ must reach 6.7 kPa/50 mmHg)

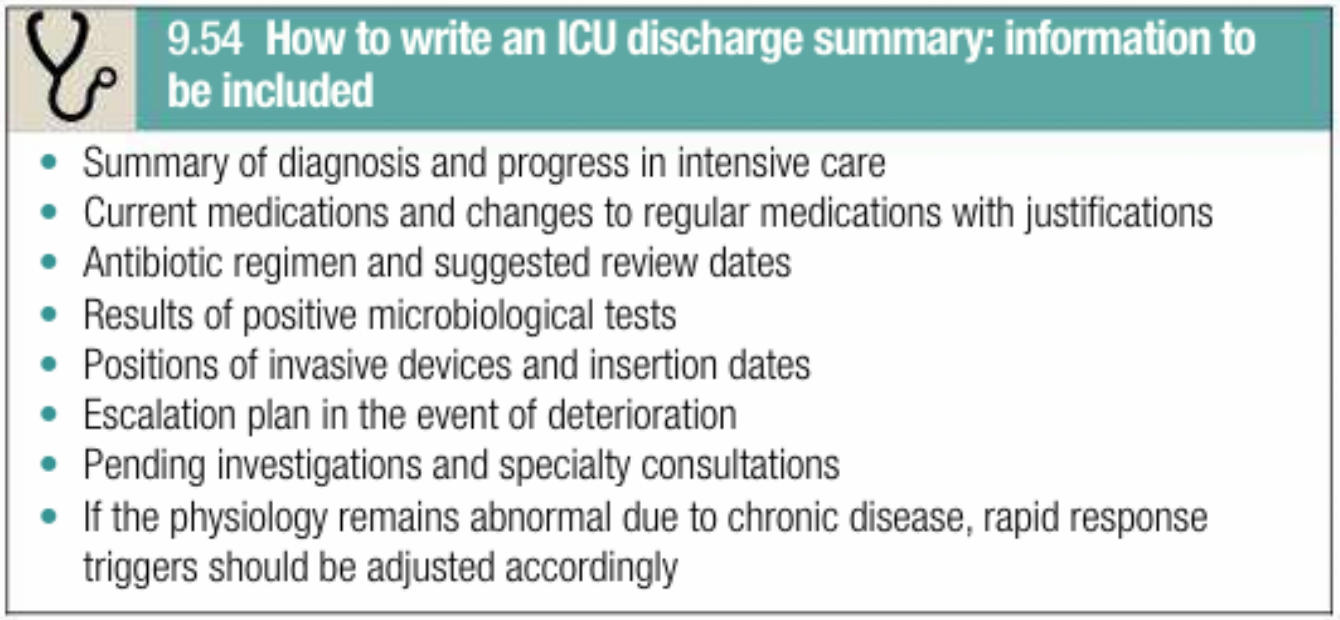
The diagnosis of brain death should be made by two doctors of a specified status and experience. The tests are usually repeated after a short interval to allow blood gases to normalise before brain death is finally confirmed.

ETHICS OF FUTILITY & WITHDRAWAL OF ACTIVE TREATMENT

- **Futility:** Deemed when the likelihood of recovery to a quality of life acceptable to the patient has passed, often seen in refractory multi-organ failure or severe neurological injury.
- **Palliative Care:** Integrating palliative principles early addresses severe physical and emotional suffering in coordination with maximal life support.
- **Brain Death:** Legally and medically defined as the irreversible loss of cortical and brainstem function.
- Diagnosis requires the strict absence of brainstem reflexes (pupillary, corneal, vestibulo-ocular, gag) and a positive apnoea test confirming no respiratory drive at $PaCO_2$ > 6.7 kPa (50 mmHg).
- **Withdrawal:** Intensity of care remains high, but focus shifts strictly to palliation. Discontinue vasopressors and ventilators, but aggressively maintain sedation and analgesia to ensure comfort.

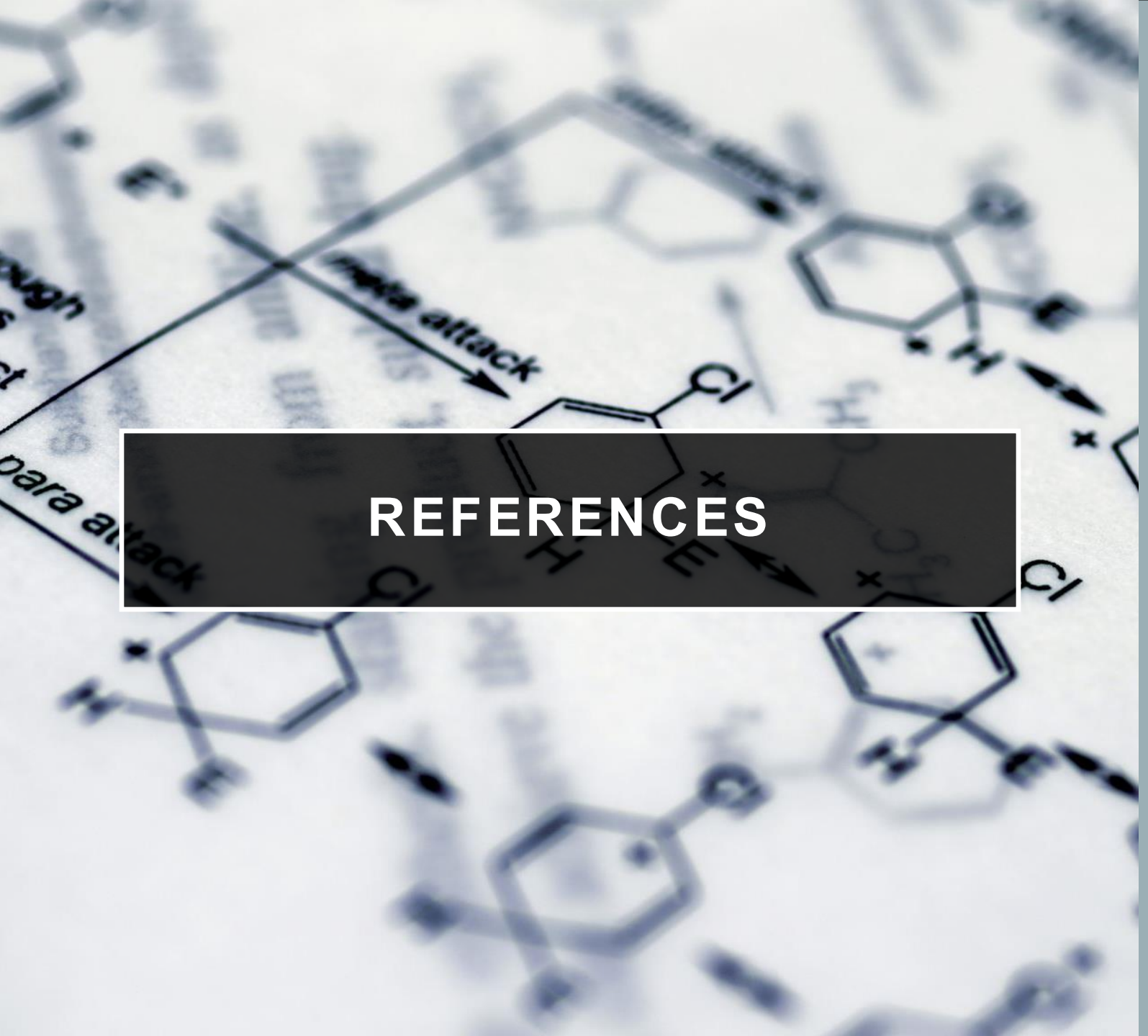
DISCHARGE CRITERIA & DETAILED SUMMARY REQUIREMENTS

- Patients are ready for discharge when the primary physiological insult has resolved, and they possess sufficient physiological reserve for ward-level care.
- Discharge timing matters: stepping patients down outside normal working hours increases the risk of both mortality and ICU readmission.
- Transfer requires structured handover of critically important information to ensure continuity and safety.
- A rigorous discharge summary must include the diagnosis, current medications, specific antibiotic stop dates, dates of invasive device insertions, and a clear escalation plan.
- Discharge planning should involve shared decision-making with the family and coordinate specific post-critical illness physical and cognitive follow-up.



9.54 How to write an ICU discharge summary: information to be included

- Summary of diagnosis and progress in intensive care
- Current medications and changes to regular medications with justifications
- Antibiotic regimen and suggested review dates
- Results of positive microbiological tests
- Positions of invasive devices and insertion dates
- Escalation plan in the event of deterioration
- Pending investigations and specialty consultations
- If the physiology remains abnormal due to chronic disease, rapid response triggers should be adjusted accordingly



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