FROM OXYGEN MASK TO EXPERT VENTILATOR

A E T C M

Mechanical Ventilation

A Complete Guide — Beginner to Expert

Oxygen delivery devices, high-flow nasal cannula, CPAP and BiPAP, ventilator basics for the final-year student, waveforms, troubleshooting, ARDS, weaning and rescue therapies — with graphs, tables and cases

Oxygen devices · HFNC · CPAP & BiPAP · Modes · Waveforms & loops · ABG & settings · Troubleshooting · Asynchrony · ARDS · Asthma & COPD · Weaning · Tracheostomy · Paediatrics · ECMO · 14 cases



By

Dr. Gireesh Kumar K.P.

For MBBS students, interns, residents, emergency physicians, intensivists, anaesthetists and critical care nurses

Mechanical Ventilation

From Oxygen Mask to Expert Ventilator Management

Oxygen therapy · High-flow nasal cannula · CPAP and BiPAP
Ventilator basics · Modes · Graphics · Troubleshooting · ARDS · Weaning · ECMO



Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM)

First Edition · October 2026

Disclaimer. This book is a learning and quick-reference guide for medical students and registered practitioners. Ventilator settings, drug doses and protocols given here follow international and Indian critical-care guidance and landmark trials current at the time of writing (October 2026), but every patient is different. Always adapt to the individual patient, follow the ventilator manufacturer's instructions and your institution's protocols, and seek senior help when in doubt. Figures are schematic illustrations, not recordings from patients. The author and publisher accept no liability for any consequence arising from the use of this information.

Preface

Every night, in every emergency department and intensive care unit in India, a young doctor stands in front of a ventilator for the first time — often alone, often at 3 a.m., with a patient whose life depends on the next decision. The screen is full of numbers, the alarms are ringing, and the textbooks seem to have been written for someone who already knows the answers. This book is written for that doctor.

It starts where the patient starts: with oxygen. The first part explains the physiology you need at the bedside and then every oxygen device — nasal cannula, face masks, reservoir masks and Venturi masks with their flows and FiO_2 — followed by high-flow nasal cannula and non-invasive ventilation with CPAP and BiPAP: when to use them, how to set them, what goes wrong and, most importantly, how to recognise failure before it is too late.

The second part introduces the ventilator from first principles, the way I would teach a final-year MBBS student on the first day of an ICU posting: what a breath is, what trigger, target and cycle mean, the equation of motion, the basic settings and the common modes. The third part takes you to the bedside — intubation, initial settings, monitoring, waveforms, blood gases, troubleshooting, asynchrony, sedation and the care bundles that prevent harm.

The fourth part builds up to expert practice: ARDS and lung-protective ventilation, obstructive lung disease, brain injury, metabolic acidosis, poisoning and neuromuscular disease, advanced modes and transpulmonary pressure, heart–lung interactions, weaning and extubation, tracheostomy, paediatrics, transport, complications and ECMO. The book ends with fourteen case scenarios and pocket reference cards.

Graphs and tables are used generously, because ventilation is best understood visually. Learn the curves, and the ventilator will start to talk to you. I hope this book gives you the confidence to ventilate safely — and the humility to keep learning from every patient.



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October 2026

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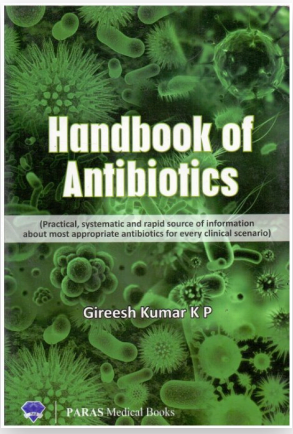
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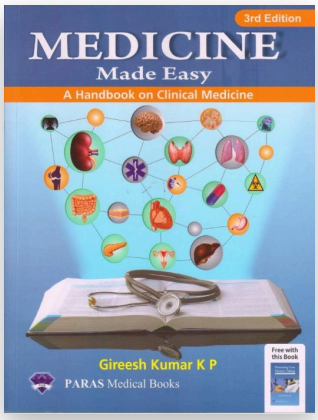
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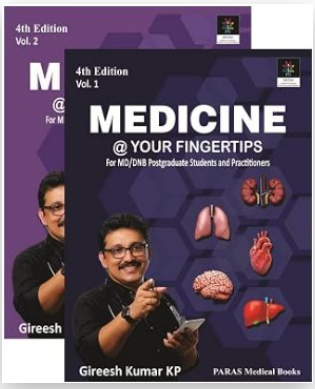
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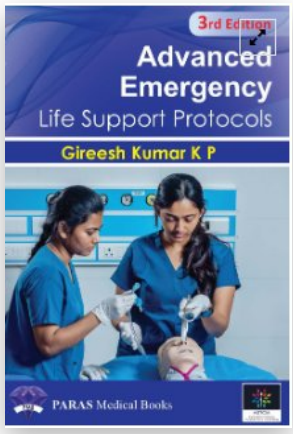
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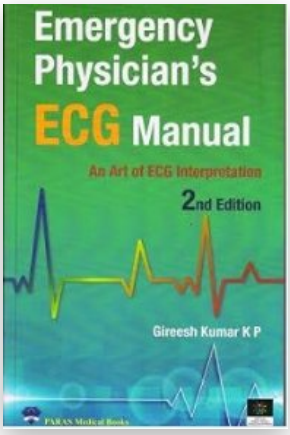
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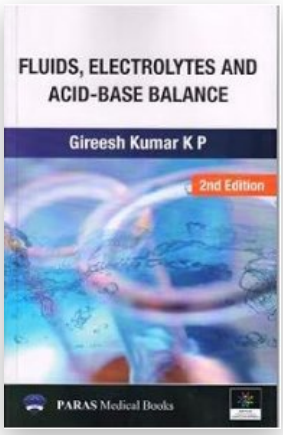
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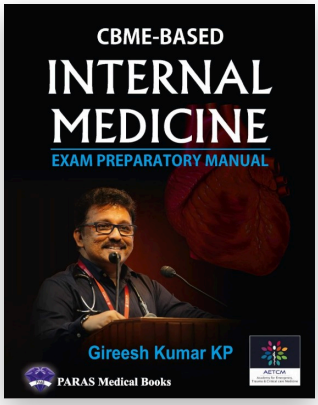
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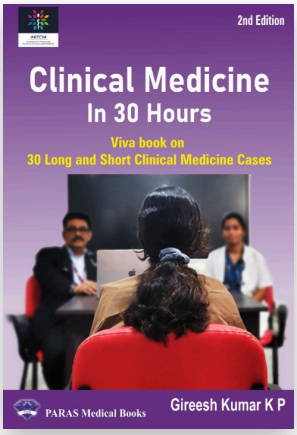
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CHAPTER 1

Respiratory Physiology for the Bedside

The few concepts that explain every oxygen device and every ventilator setting

Why we breathe — two separate jobs

The lungs do two different jobs. **Oxygenation** moves oxygen from air into blood; it depends mainly on the inspired oxygen fraction (FiO_2) and on how much lung is open (mean airway pressure, PEEP). **Ventilation** removes carbon dioxide; it depends on how much gas moves in and out each minute (minute ventilation = tidal volume × respiratory rate). Keep the two apart in your mind: on the ventilator, **FiO_2 and PEEP fix oxygen; rate and tidal volume fix CO_2.**

Problem	Measured by	Main determinants	Ventilator knobs
Hypoxaemia (oxygenation failure, Type 1)	PaO_2, SpO_2, PaO_2/FiO_2 (P/F) ratio	FiO_2, alveolar recruitment, V/Q matching, shunt, diffusion	FiO_2, PEEP, mean airway pressure, inspiratory time, prone position
Hypercapnia (ventilatory failure, Type 2)	$PaCO_2$, pH	Minute ventilation, dead space, CO_2 production	Respiratory rate, tidal volume (or pressure above PEEP), reduce dead space

The oxygen cascade

Oxygen falls step by step from air (PO_2 ≈ 160 mmHg at sea level) to the mitochondria (<5 mmHg). The alveolar gas equation tells you the alveolar PO_2 (PAO_2) and therefore the best PaO_2 you can expect.

Formula	Use	Normal / example
$PAO_2 = FiO_2 \times (Patm - PH_2O) - PaCO_2/0.8$	Alveolar oxygen	Room air: 0.21 × (760 − 47) − 40/0.8 ≈ 100 mmHg
A–a gradient = $PAO_2 - PaO_2$	Separates hypoventilation (normal gradient) from lung disease (raised gradient)	Normal ≈ (age/4) + 4, or <10–15 mmHg in the young
P/F ratio = PaO_2 / FiO_2	Severity of hypoxaemia; ARDS classification	Normal ≈ 400–500; <300 mild, <200 moderate, <100 severe ARDS
S/F ratio = SpO_2 / FiO_2	When no ABG (valid when SpO_2 ≤97%)	S/F 315 ≈ P/F 300; S/F 235 ≈ P/F 200
Oxygenation index (OI) = (FiO_2 × mean Paw × 100) / PaO_2	Severity in children/neonates and ARDS	>16 severe; >25–40 consider ECMO (neonates)
$CaO_2 = (1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)$	Oxygen content (ml/dl)	Hb 15, SaO_2 98% → ≈ 20 ml/dl
$DO_2 = CO \times CaO_2 \times 10$	Oxygen delivery (ml/min)	≈ 1000 ml/min (5 L/min × 20 ml/dl × 10)

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The oxyhaemoglobin dissociation curve

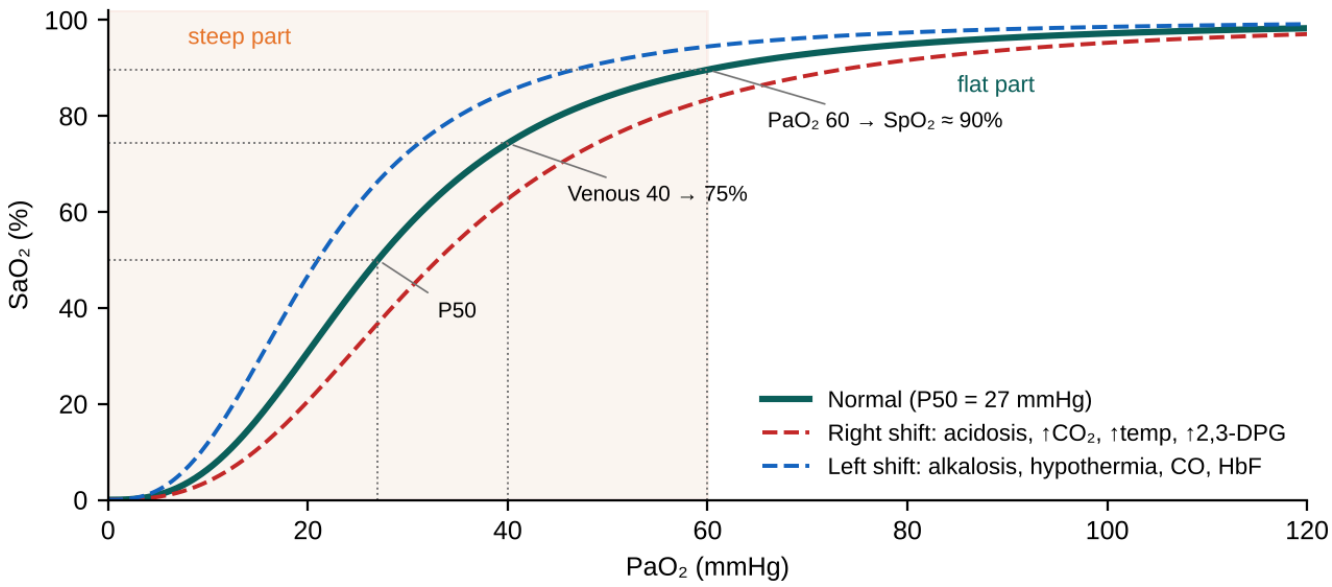


Figure 1.1. Oxyhaemoglobin dissociation curve. The flat upper part means SpO_2 above 94% gives little extra oxygen content; on the steep part, a small fall in PaO_2 causes a big fall in saturation.

PaO_2 (mmHg)	SaO_2 (approx.)	Clinical meaning
100	98%	Normal arterial blood on room air
80	95%	Lower limit of normal
60	90%	"Edge of the cliff" — below this saturation falls steeply
40	75%	Normal mixed venous blood
27	50%	P50 (normal) — shifts right with acidosis, fever, high CO_2, 2,3-DPG

KEY POINT **Right shift** (acidosis, hypercapnia, fever, raised 2,3-DPG) releases oxygen to tissues; **left shift** (alkalosis, hypothermia, carbon monoxide, fetal Hb) holds oxygen. Pulse oximeters over-read in dark skin, CO poisoning and methaemoglobinaemia (stuck near 85%).

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Five causes of hypoxaemia

Mechanism	A–a gradient	Response to 100% O_2	Examples
Low inspired O_2	Normal	Corrects	High altitude, faulty gas supply
Hypoventilation	Normal	Corrects	Opioid/sedative overdose, neuromuscular weakness, obesity hypoventilation
V/Q mismatch	Raised	Corrects well	COPD, asthma, pneumonia, pulmonary embolism
Shunt (perfusion without ventilation)	Raised	**Poor** correction (needs PEEP/recruitment)	ARDS, lobar collapse, pulmonary oedema, intracardiac shunt
Diffusion limitation	Raised	Corrects	Interstitial lung disease, exercise

Lung mechanics in three numbers

Every breath must overcome two loads: the **elastic** load of stretching the lungs and chest wall (compliance) and the **resistive** load of pushing gas through tubes (resistance). Their product, the time constant, tells you how fast the lung fills and empties.

Term	Formula	Normal (intubated adult)	Increased / decreased in
Static compliance (Cstat)	Tidal volume / (Plateau − PEEP)	50–100 ml/cmH_2O	↓ ARDS, pulmonary oedema, pneumonia, fibrosis, obesity, pneumothorax, abdominal distension; ↑ emphysema
Dynamic compliance (Cdyn)	Tidal volume / (Peak − PEEP)	40–80 ml/cmH_2O	Falls with both stiffness and resistance
Airway resistance (Raw)	(Peak − Plateau) / inspiratory flow (L/s)	5–10 cmH_2O/L/s (higher with ET tube)	↑ bronchospasm, secretions, kinked/narrow tube, COPD
Time constant (τ)	Resistance × compliance	≈ 0.2–0.5 s	Long in COPD/asthma (slow emptying → auto-PEEP); short in ARDS
Driving pressure (ΔP)	Plateau − PEEP (= VT/Crs)	Keep <15 cmH_2O	Best single predictor of mortality in ARDS

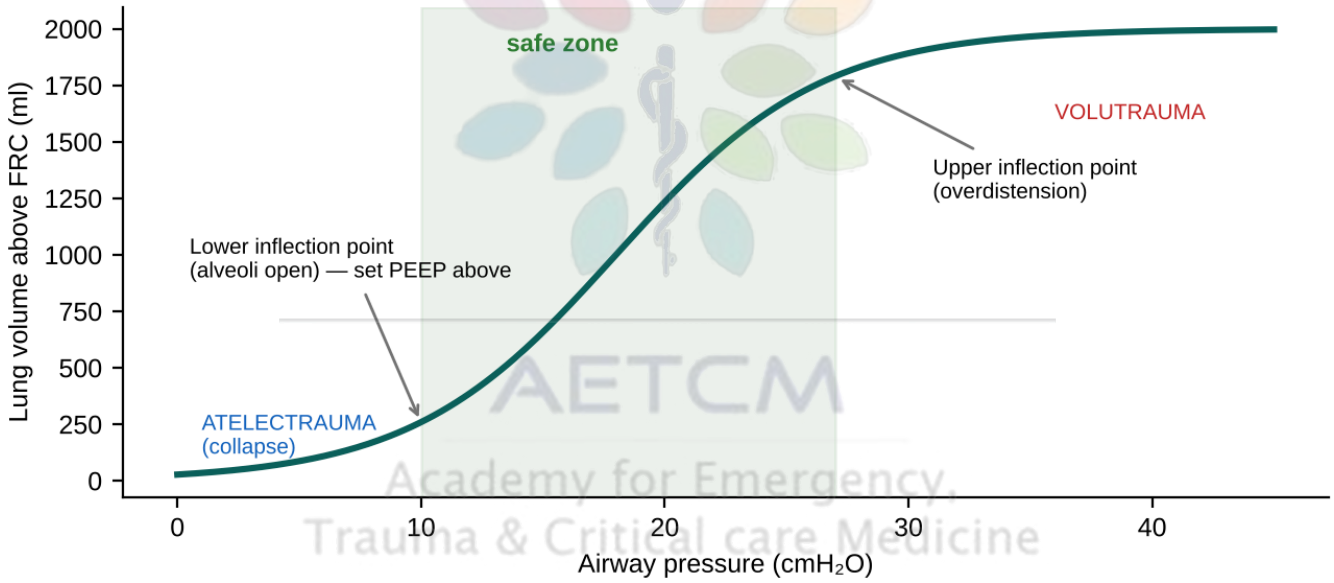


Figure 1.2. Pressure–volume curve of the respiratory system: lower and upper inflection points. Ventilate between them — above the lower (open the lung with PEEP) and below the upper (avoid overdistension).

KEY POINT It takes about **3 time constants** for 95% of a breath to empty. If τ = 0.8 s (COPD), expiration needs ≈ 2.4 s; a shorter expiratory time traps gas and creates auto-PEEP.

Dead space and CO_2

Concept	Detail
Anatomical dead space	≈ 2 ml/kg (≈ 150 ml); ET tube, HME and catheter mount add 50–100 ml of apparatus dead space
Alveolar dead space	Ventilated but not perfused alveoli: pulmonary embolism, low cardiac output, overdistension by high PEEP

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Concept	Detail
Bohr equation	VD/VT = ($PaCO_2$ − $PECO_2$)/$PaCO_2$; normal 0.3; >0.6 in severe ARDS predicts death
Alveolar ventilation	VA = (VT − VD) × RR; $PaCO_2 \propto VCO_2 / VA$
Rapid shallow breathing	Wastes ventilation on dead space: RR 40 × VT 200 ml (8 L/min) ventilates alveoli less than RR 15 × VT 500 ml (7.5 L/min)
$ETCO_2$	Normally 2–5 mmHg below $PaCO_2$; the gap widens when dead space rises

Respiratory failure — the bedside classification

Type	Definition	Common causes	First support
Type 1 (hypoxaemic)	PaO_2 <60 mmHg with normal or low $PaCO_2$	Pneumonia, ARDS, pulmonary oedema, PE, ILD	Oxygen → HFNC → CPAP/NIV → intubation
Type 2 (hypercapnic)	$PaCO_2$ >45–50 mmHg with pH <7.35	COPD, asthma, neuromuscular disease, obesity hypoventilation, sedative overdose	Controlled oxygen (88–92%) → BiPAP → intubation
Type 3 (perioperative)	Atelectasis after surgery/anaesthesia	Abdominal/thoracic surgery, pain, obesity	Analgesia, incentive spirometry, CPAP, early mobilisation
Type 4 (shock)	Respiratory muscles steal cardiac output in shock	Septic, cardiogenic shock	Early intubation to rest muscles and reduce O_2 demand

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CHAPTER 2

Oxygen Therapy and Delivery Devices

Nasal cannula to non-rebreather mask — flows, FiO_2, uses and pitfalls

Oxygen is a drug — prescribe a target

Patient group	Target SpO_2	Source
Most acutely ill adults	94–98% (BTS); 92–96% acceptable (WHO, ICU trials)	BTS 2017, WHO
Risk of hypercapnia (COPD, obesity hypoventilation, neuromuscular disease, kyphoscoliosis, bronchiectasis/CF)	**88–92%**	BTS/GOLD
Mechanically ventilated adults	92–96% (avoid both hypoxaemia and hyperoxia)	ICU trials (ICU-ROX, HOT-ICU)
Critical illness — cardiac arrest (ROSC), shock, major trauma, sepsis	Start 15 L/min reservoir mask, then titrate to 94–98%	BTS
Acute MI / stroke without hypoxaemia	**No oxygen** if SpO_2 ≥90–94%	AVOID, DETO2X trials
Pregnancy	≥95% (fetal oxygenation)	—
Preterm neonates	90–95%	Avoids retinopathy of prematurity
Carbon monoxide / cyanide poisoning, pneumothorax (absorption)	Highest possible FiO_2 regardless of SpO_2	—

KEY POINT Hyperoxia is harmful: absorption atelectasis, coronary and cerebral vasoconstriction, free-radical injury and, in COPD, CO_2 retention (loss of hypoxic vasoconstriction, Haldane effect). **Titrate down as soon as the target is reached.**

Classification of oxygen delivery devices

Category	Principle	Devices	FiO_2 delivered
Low-flow, variable performance	Device flow is less than the patient's inspiratory flow (30–60 L/min in distress); room air is entrained, so FiO_2 varies with breathing pattern	Nasal cannula, simple face mask, partial rebreather, non-rebreather mask	24–90% (unpredictable)
High-flow, fixed performance	Total flow exceeds patient demand; FiO_2 stays constant	Venturi mask, high-flow nasal cannula (HFNC), air-entrainment nebuliser	24–100% (precise)
Closed / sealed systems	Sealed interface with reservoir	Bag-valve-mask, anaesthesia circuit, NIV mask, ventilator	Up to 100%

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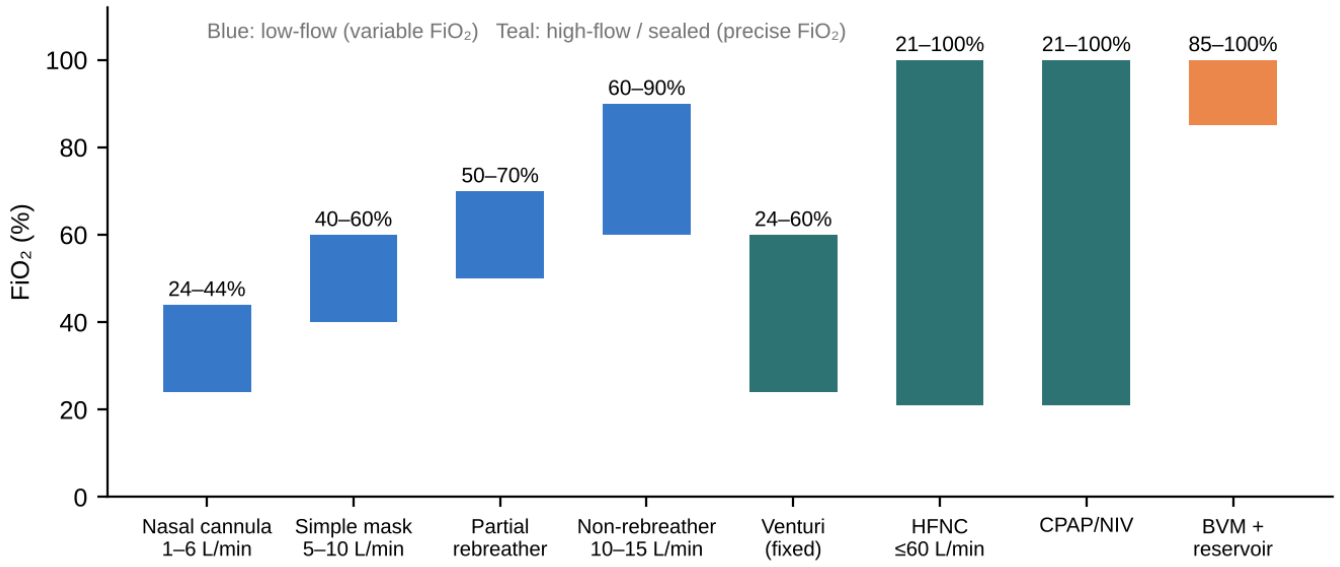


Figure 2.1. Approximate FiO_2 delivered by common oxygen devices at their usual flow rates.

Nasal cannula (nasal prongs)

Flow (L/min)	Approximate FiO_2
1	24%
2	28%
3	32%
4	36%
5	40%
6	44% (maximum; higher flows dry and irritate the mucosa)

- **Rule of thumb:** each 1 L/min adds about 4% to 21%.
- **Uses:** mild hypoxaemia, stable patients, long-term oxygen therapy, COPD (1–2 L/min), during meals.
- **Advantages:** comfortable, patient can eat and talk; works even with mouth breathing (oxygen pools in the nasopharynx).
- **Pitfalls:** dry nasal mucosa above 4 L/min (humidify), pressure sores over ears, blocked nose; FiO_2 falls as the respiratory rate rises.

Simple face mask (Hudson mask)

Flow (L/min)	Approximate FiO_2
5–6	40%
6–7	50%
7–10	60%

- **Minimum 5 L/min** — lower flows allow exhaled CO_2 to be rebreathed from the mask (≈ 100–200 ml dead space).
- **Uses:** moderate hypoxaemia in patients **not** at risk of hypercapnia; short-term use (post-operative, transport).
- **Pitfalls:** uncomfortable, must be removed to eat, unknown FiO_2, claustrophobia, aspiration risk with vomiting.

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Partial rebreather and non-rebreather (reservoir) masks

Feature	Partial rebreather	Non-rebreather mask (NRBM)
Valves	No one-way valves; first one-third of exhaled gas (dead-space gas, O_2-rich) enters the bag	One-way valve between bag and mask; one-way flaps on side ports
Flow	10–15 L/min (bag should not collapse)	**10–15 L/min**; fill the bag before applying; bag should stay at least one-third full on inspiration
FiO_2	50–70%	60–90% (true 100% is never achieved because of leaks)
Uses	Moderate–severe hypoxaemia	Severe hypoxaemia, trauma, shock, CO poisoning, cluster headache, pre-oxygenation when nothing better is available
Pitfalls	CO_2 rebreathing if flow too low	Suffocation risk if oxygen supply fails (side flap should be left open on one side); patient can vomit into the mask; not for long-term use

Venturi (air-entrainment) mask — fixed FiO_2

Oxygen passes through a narrow jet; the high-velocity stream entrains room air through side ports in a fixed ratio. Total flow (oxygen + entrained air) exceeds the patient's demand, so the FiO_2 is predictable — ideal for **COPD and other patients at risk of CO_2 retention**.

Colour of valve (common convention — check the label)	FiO_2	Oxygen flow (L/min)	Air:O_2 entrainment ratio	Total flow (L/min)
Blue	24%	2–4	25:1	50–100
White	28%	4–6	10:1	44–66
Yellow	35%	8–10	5:1	48–60
Red	40%	10–12	3:1	40–48
Green	60%	12–15	1:1	24–30

KEY POINT At 40% and 60% the total flow may fall below the demand of a breathless patient (inspiratory flow 50–60 L/min) — the FiO_2 then becomes variable. **Increase the oxygen flow by 50%** if the respiratory rate is >30/min; this does not change the FiO_2 but increases total flow. Colour codes vary between manufacturers — always read the FiO_2 printed on the valve.

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Other oxygen devices

Device	Description	FiO_2 / flow	Use
Tracheostomy mask / collar	Mask over the stoma with humidified (nebuliser) gas	28–60% via air-entrainment nebuliser, 10–15 L/min	Tracheostomised patients weaning off the ventilator
T-piece (Briggs adaptor)	T-shaped tube attached to the ET or tracheostomy tube, with reservoir limb	Set FiO_2 from blender/nebuliser, flow ≥2–3 × minute ventilation	Spontaneous breathing trial, weaning
Oxygen hood / head box	Plastic box over infant's head	FiO_2 up to 80–90%, flow 10–15 L/min (to wash out CO_2)	Neonates and infants
Oxygen tent	Large canopy	40–50% (variable)	Children (rarely used now)

Device	Description	FiO_2 / flow	Use
Bag-valve-mask (Ambu bag) with reservoir	Self-inflating bag + reservoir bag + O_2 15 L/min	≈ 90–100% (only with good seal or when squeezed; passive flow through bag is unreliable unless a non-rebreathing valve allows spontaneous breathing)	Resuscitation, pre-oxygenation with PEEP valve
Mapleson C / Jackson–Rees circuit	Flow-inflating bag	Up to 100% with fresh gas ≥2–3 × minute ventilation	Pre-oxygenation, transport, paediatric
Oxygen concentrator	Molecular sieve (zeolite) concentrates O_2 from air	90–95% O_2 at 1–10 L/min	Home and ward oxygen, rural hospitals
Nasal catheter / nasopharyngeal catheter	Catheter in nostril	Similar to cannula	Rare (older practice)

Oxygen supply in Indian hospitals

Source	Capacity / calculation
Duration of a cylinder	Duration (min) = (cylinder pressure in psi × cylinder factor) / flow (L/min); or = litres of O_2 remaining / flow
Cylinder factor	D 0.16; E 0.28; H/K 3.14 (L/psi)
Example: E cylinder at 2000 psi, flow 10 L/min	(2000 × 0.28) / 10 = 56 minutes (keep a safety margin; replace at 200–500 psi)
Indian "D-type" (jumbo) cylinder	≈ 7000 L of oxygen (46.7 L water capacity at ≈ 150 bar) — lasts ≈ 11–12 hours at 10 L/min
Indian "B-type" cylinder	≈ 1500 L (10 L water capacity) — ≈ 2.5 hours at 10 L/min
Liquid medical oxygen (LMO) / PSA plants	Piped supply at 4 bar (≈ 60 psi); 1 L liquid O_2 ≈ 860 L gas
Colour code (India)	Medical oxygen cylinder: **black body with white shoulder**; nitrous oxide: French blue — always read the label before connecting

Humidification

- Nasal cannula ≤4 L/min and short-term mask oxygen: humidification not needed.
- Flows >4 L/min for long periods, thick secretions, tracheostomy or ET tube: use humidification (bubble humidifier, heated humidifier or HME).
- Bubble humidifiers add little moisture at high flows and are an infection risk if not changed — use sterile water, change daily.

Oxygen toxicity and hazards

Hazard	Detail
Pulmonary oxygen toxicity	FiO_2 >0.6 for >24–48 h: tracheobronchitis, ARDS-like injury; aim to reduce FiO_2 below 0.6 using PEEP
Absorption atelectasis	Nitrogen washout behind closed airways at FiO_2 near 1.0
CO_2 narcosis	Uncontrolled oxygen in COPD raises $PaCO_2$ (V/Q worsening, Haldane effect, reduced drive)
Retinopathy of prematurity	Preterm infants — target 90–95%

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Hazard	Detail
Fire	Oxygen-enriched environment: no smoking, no oil-based products, care with defibrillation and diathermy
Drug interactions	Bleomycin, amiodarone, paraquat poisoning — use the lowest FiO_2 possible

Weaning off oxygen

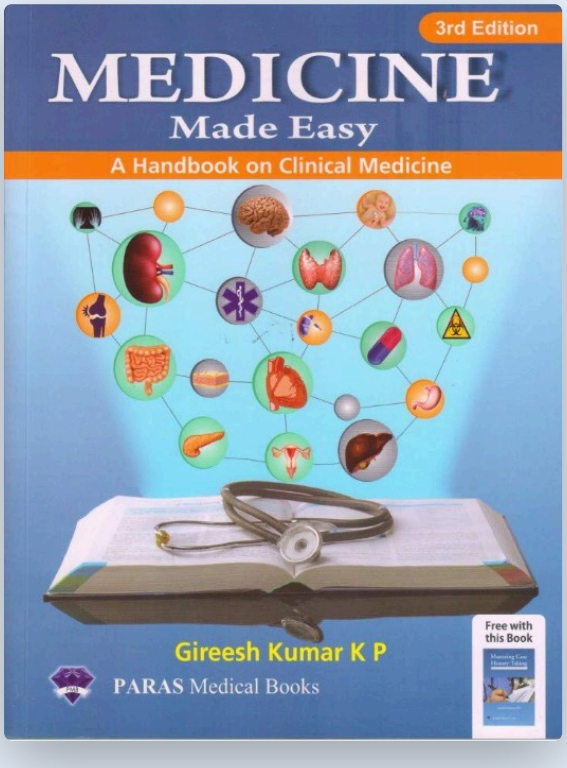
- Reduce FiO_2/flow stepwise when SpO_2 is stable at the upper end of target for 4–8 hours.
- Switch mask to nasal cannula at 2–4 L/min, then 1 L/min, then stop; recheck SpO_2 after 5 minutes and at intervals.
- Discontinue when SpO_2 remains in target on room air at rest and on mobilising (6-minute walk if needed).

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CHAPTER 3

High-Flow Nasal Cannula (HFNC)

Heated, humidified high-flow oxygen — physiology, settings, ROX index and failure

What HFNC is

HFNC delivers heated (37 °C), fully humidified gas through wide-bore nasal prongs at flows of **up to 60 L/min in adults** (2 L/kg/min in children) with a precisely blended FiO_2 of 0.21–1.0. Because the flow matches or exceeds the patient's own inspiratory flow, very little room air is entrained and the set FiO_2 is the delivered FiO_2.

How HFNC helps — five mechanisms

Mechanism	Effect
Fixed, high FiO_2	Flow meets peak inspiratory demand, so FiO_2 is reliable (unlike NRBM)
Dead-space washout	Flushes CO_2 from the nasopharynx; each breath starts with fresh gas → lower $PaCO_2$ and work of breathing
Low-level PEEP	≈ 1 cmH_2O per 10 L/min with mouth closed (≈ 3–5 cmH_2O at 50–60 L/min); improves end-expiratory lung volume
Heated humidification	Preserves mucociliary clearance, loosens secretions, reduces bronchoconstriction from cold dry gas; improves comfort
Reduced work of breathing	Lower inspiratory resistance and metabolic cost of warming/humidifying gas

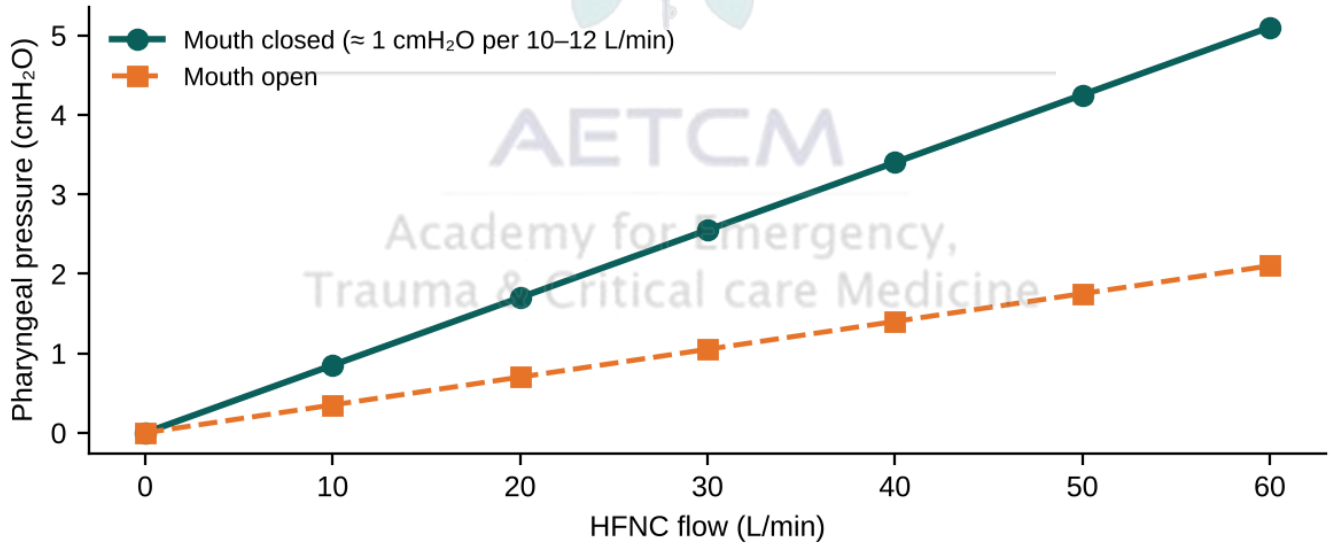


Figure 3.1. Approximate nasopharyngeal pressure generated by HFNC at different flows (mouth closed vs open).

Indications

Strong evidence	Reasonable / selected use
Acute hypoxaemic respiratory failure (pneumonia, COVID-19) — reduces intubation compared with conventional oxygen in meta-analyses; lower mortality in FLORALI (P/F ≤200 subgroup)	Mild hypercapnic COPD exacerbation (pH 7.25–7.35) if NIV is not tolerated, or between NIV sessions

Strong evidence	Reasonable / selected use
Post-extubation support in low-risk patients (instead of conventional O_2) and alternating with NIV in high-risk patients	Pre-oxygenation and apnoeic oxygenation during intubation (60 L/min left on during laryngoscopy)
Bronchiolitis and paediatric respiratory distress	Do-not-intubate and palliative patients (comfort, dyspnoea relief)
Immunocompromised hypoxaemic patients	Bronchoscopy, procedural sedation in hypoxaemic patients; acute heart failure (mild)

Contraindications and cautions

- Need for immediate intubation (arrest, GCS ≤8, inability to protect the airway, massive haemoptysis).
- Severe hypercapnic acidosis (pH <7.25) — use NIV.
- Facial trauma, base-of-skull fracture, nasal obstruction, epistaxis, recent nasal surgery.
- Undrained pneumothorax (relative).

Setting up HFNC

Step	Detail
1. Choose the prong size	Prongs should occlude ≤50% of each nostril (leaves room to exhale)
2. Water chamber	Sterile water; set temperature 37 °C (34 °C if the patient finds it too warm)
3. Initial flow	Hypoxaemic adult: **50–60 L/min**; COPD/comfort: 30–40 L/min; children 1–2 L/kg/min (max 2 L/kg/min up to 12 kg, then ≈ 0.5 L/kg for each additional kg)
4. Initial FiO_2	0.6–1.0, then titrate to SpO_2 92–96% (88–92% in hypercapnic risk)
5. Reassess at 1 hour	Respiratory rate, SpO_2, work of breathing, ROX index, ABG if hypercapnia

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The ROX index — when HFNC is failing

ROX = (SpO_2 / FiO_2) / respiratory rate. It was validated in pneumonia with acute hypoxaemic respiratory failure (Roca 2019) and is easy to calculate at the bedside.

Time on HFNC	ROX predicting success	ROX predicting failure (intubation)
2 hours	≥4.88	<2.85
6 hours	≥4.88	<3.47
12 hours	≥4.88	<3.85
Example	SpO_2 92%, FiO_2 0.6, RR 30 → (92/0.6)/30 = 5.1 → likely success	SpO_2 88%, FiO_2 1.0, RR 36 → (88/1.0)/36 = 2.4 → prepare to intubate

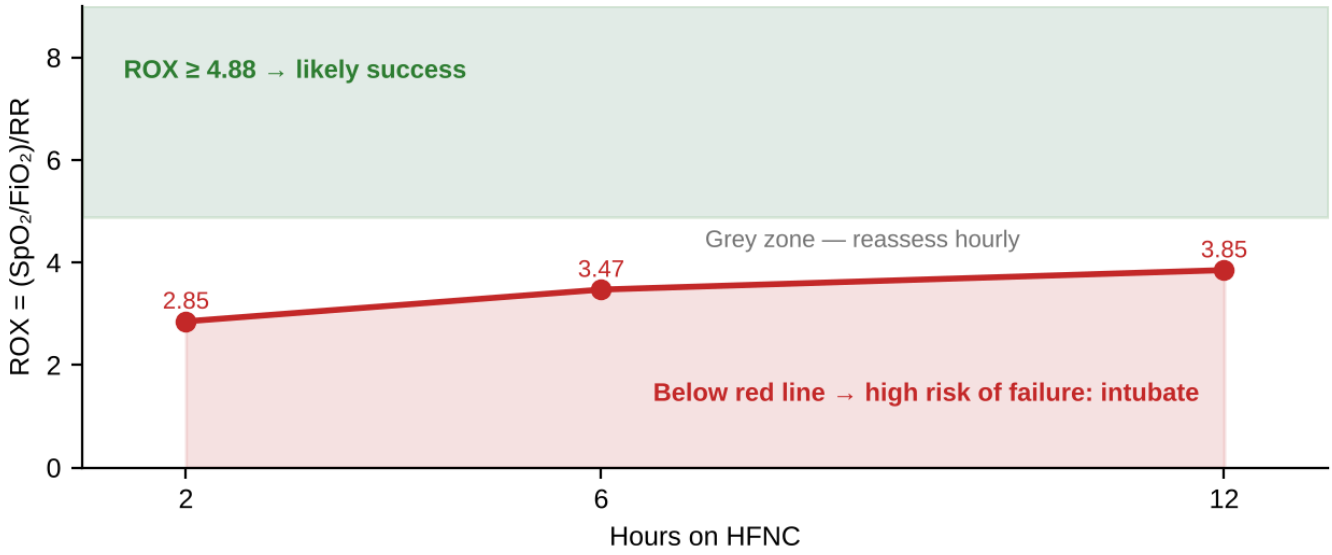


Figure 3.2. ROX index thresholds over the first 12 hours of HFNC.

KEY POINT **Do not delay intubation.** In HIGH-FLOW observational data, intubation after >48 hours of failing HFNC was associated with higher mortality. Persistent RR >30–35/min, rising FiO_2 need (>0.7–0.8 at 60 L/min), paradoxical breathing, falling ROX, rising $PaCO_2$, altered sensorium or haemodynamic instability are signs of failure.

Weaning HFNC

Step	Action
1	Reduce **FiO_2** first, in steps of 0.05–0.1, to ≤0.4 while keeping SpO_2 in target
2	Then reduce **flow** by 5–10 L/min every few hours to 20–30 L/min
3	When FiO_2 ≤0.4 and flow ≤20–30 L/min with RR <25, switch to conventional nasal cannula/mask
Children	Many units stop directly from 1 L/kg/min in bronchiolitis when stable

Problems with HFNC and solutions

Problem	Solution
Condensation ("rain-out") in tubing	Use heated-wire circuits; keep tubing below the patient's head; check temperature
Patient too warm / discomfort	Reduce temperature to 34 °C or reduce flow
Nasal sores, dryness	Correct prong size, padding, check humidifier is working
Noise	Reassure; lower flow if acceptable
Mouth breathing	Reduces PEEP effect but FiO_2 is still reliable — encourage mouth closure; chin strap is not needed
Rising oxygen consumption	High-flow at 60 L/min uses ≈ 36–60 L O_2/min at high FiO_2 — plan oxygen supply (especially during surges)
Delayed intubation	Use ROX/clinical criteria; decide early

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HFNC versus other devices

Feature	Non-rebreather mask	HFNC	CPAP/NIV
FiO_2	Variable 0.6–0.9	Precise up to 1.0	Precise up to 1.0
PEEP	None	3–5 cmH_2O (flow-dependent)	Set 5–15 cmH_2O
Pressure support	None	None	Yes (BiPAP)
Comfort, eating, talking	Poor	Excellent	Poor
CO_2 clearance	Rebreathing possible	Dead-space washout	Best (with pressure support)
Best for	Short-term rescue	Hypoxaemic failure, post-extubation	Hypercapnic COPD, cardiogenic oedema, OHS

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CHAPTER 4

Non-Invasive Ventilation — Principles

CPAP and BiPAP, interfaces, indications and contraindications

What is NIV?

Non-invasive ventilation (NIV) delivers positive pressure through a sealed mask instead of an endotracheal tube. It supports breathing while keeping the patient awake, able to cough, talk and swallow, and avoids the complications of intubation (VAP, sedation, airway trauma). Two forms are used: **CPAP** (one continuous pressure) and **BiPAP / bilevel NIV** (a higher inspiratory pressure, IPAP, and a lower expiratory pressure, EPAP).

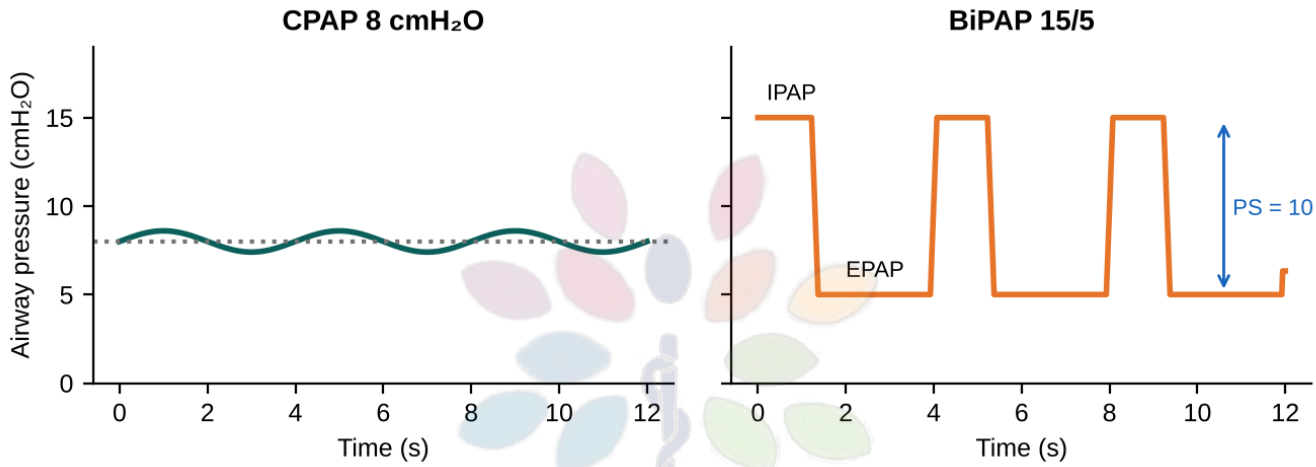


Figure 4.1. Airway pressure in CPAP (one level) and BiPAP (IPAP above EPAP; the difference is pressure support).

CPAP versus BiPAP

Feature	CPAP	BiPAP (bilevel)
Pressure	Single continuous pressure (e.g. 5–10 cmH_2O) throughout the cycle	IPAP (e.g. 10–20) during inspiration, EPAP (e.g. 4–8) during expiration
Main effect	Recruits alveoli, raises functional residual capacity, improves oxygenation; reduces preload and afterload of the left ventricle	EPAP = PEEP effect; **IPAP − EPAP = pressure support** → bigger tidal volume, CO_2 removal, rests respiratory muscles
Fixes	Oxygenation (Type 1 failure), cardiogenic pulmonary oedema, obstructive sleep apnoea	Ventilation (Type 2 failure): COPD, obesity hypoventilation, neuromuscular disease, chest wall disease
Pressure support for CO_2	No	Yes
Typical start	5–8 cmH_2O, increase to 10–12	IPAP 10–12 / EPAP 4–5, increase IPAP by 2–5 every 10–30 min to 18–25

How positive pressure helps

Effect	Mechanism
Better oxygenation	Recruitment of collapsed alveoli, increased FRC, reduced shunt, redistribution of lung water

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Effect	Mechanism
Reduced work of breathing	Pressure support unloads respiratory muscles; EPAP offsets intrinsic PEEP in COPD so the patient triggers easily
CO_2 clearance	Larger tidal volume and minute ventilation
Cardiac	Raised intrathoracic pressure reduces venous return (preload) and LV transmural pressure (afterload) → helpful in LV failure, harmful in hypovolaemia/RV failure
Upper airway splinting	Keeps pharynx open in obstructive sleep apnoea and obesity

Indications — strength of evidence (ERS/ATS 2017 guideline)

Condition	Recommendation	Mode
COPD exacerbation with hypercapnic acidosis (pH ≤7.35, $PaCO_2$ >45)	**Strong — first-line**; reduces intubation and mortality	BiPAP
Cardiogenic pulmonary oedema	**Strong**	CPAP or BiPAP (equally effective)
Immunocompromised patients with acute respiratory failure	Conditional (HFNC is an alternative)	CPAP/BiPAP
Post-operative respiratory failure (abdominal, thoracic surgery)	Conditional	CPAP/BiPAP
Prevention of post-extubation failure in high-risk patients	Conditional	BiPAP (alternate with HFNC)
Palliative / do-not-intubate patients for dyspnoea	Conditional	BiPAP
Chest trauma (flail chest, contusion) with hypoxaemia	Conditional	CPAP/BiPAP (with good analgesia; drain pneumothorax first)
Obesity hypoventilation syndrome, neuromuscular disease, kyphoscoliosis (acute-on-chronic)	Recommended in practice	BiPAP (with backup rate)
Pre-oxygenation before intubation in hypoxaemic patients	Recommended (PREOXI trial 2024)	BiPAP/CPAP
Asthma, de novo hypoxaemic failure (pneumonia, ARDS), pandemic viral illness	No recommendation / use only in experienced units with early review	BiPAP / helmet CPAP
Weaning: early extubation to NIV in hypercapnic patients failing SBT	Conditional	BiPAP

Contraindications

Absolute	Relative
Respiratory or cardiac arrest; need for immediate intubation	Agitation or poor cooperation (consider cautious sedation, e.g. dexmedetomidine)
Inability to protect the airway, GCS ≤8 (except hypercapnic coma in COPD under close watch)	Copious secretions, weak cough
Facial trauma, burns, deformity preventing mask fit	Haemodynamic instability (shock, uncontrolled arrhythmia, acute coronary syndrome with instability)
Upper airway obstruction (fixed)	Recent upper GI or oesophageal surgery, upper GI bleeding

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Absolute	Relative
Vomiting, bowel obstruction	Undrained pneumothorax
Untreated pneumothorax	Severe hypoxaemia (P/F <100–150) — high failure rate

Interfaces

Interface	Advantages	Disadvantages	Best for
Oronasal (full-face) mask	Most widely used; handles mouth breathing	Claustrophobia, nasal bridge sores, aspiration risk, dead space	First choice in acute respiratory failure
Nasal mask / nasal pillows	Comfortable, can talk and eat, less dead space	Mouth leak; needs closed mouth (chin strap)	Chronic/home NIV, OSA, stable patients
Total face mask	Seals around the face perimeter, no nasal bridge pressure	Larger dead space, claustrophobia	Nasal bridge ulcers, long sessions
Helmet	Excellent tolerance, no facial pressure, good for CPAP; allows long continuous use	Noisy, CO_2 rebreathing if flow low (needs ≥60 L/min), poor for pressure support synchrony, arm pressure sores	Hypoxaemic failure (CPAP 10–12 cmH_2O), COVID-19
Mouthpiece	Daytime support, intermittent	Needs cooperation	Neuromuscular disease

Choosing the machine

Machine type	Features	Comment
Dedicated NIV (bilevel) ventilator	Single-limb circuit with intentional leak (exhalation port); excellent leak compensation	Vented mask or exhalation port must be present — never block it (CO_2 rebreathing)
ICU ventilator with NIV mode	Double-limb circuit, non-vented mask; accurate FiO_2 and monitoring	Leak compensation less efficient in older models
Home BiPAP/CPAP machines	Portable, limited FiO_2 (O_2 bled into circuit)	FiO_2 unknown; adequate for stable chronic use
Flow-driven CPAP (Boussignac, Venturi CPAP, helmet CPAP with flow generator)	Simple, no ventilator needed	Useful in wards and transport; needs high O_2 flow

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NIV terminology

Term	Meaning
IPAP	Inspiratory positive airway pressure (total pressure during inspiration)
EPAP	Expiratory positive airway pressure (= PEEP/CPAP)
Pressure support (PS)	IPAP − EPAP (on ICU ventilators PS is set **above** PEEP — avoid confusion)
Rise time	Speed at which pressure reaches IPAP; slower (longer) for COPD comfort, faster for air hunger
Trigger sensitivity	Flow change that starts a breath; auto-trigger with leaks if too sensitive
Cycle (expiratory trigger)	% of peak flow at which the machine switches to EPAP (default 25%; raise to 40–50% in COPD so expiration starts earlier)
Backup rate	Timed breaths delivered if the patient does not breathe (S/T mode)
Ti min / Ti max	Limits on inspiratory time (COPD: Ti max ≈ 1.0–1.2 s)

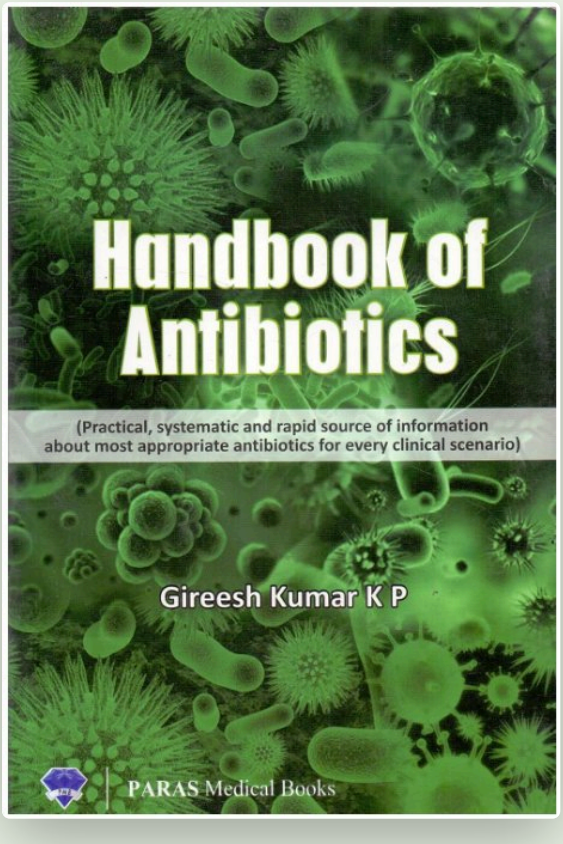
Term	Meaning
S, S/T, T, AVAPS, iVAPS	Spontaneous; spontaneous-timed; timed; average volume-assured pressure support (targets a tidal volume)

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CHAPTER 5

Non-Invasive Ventilation in Practice

Setting up, titrating, monitoring, troubleshooting and detecting failure

Step-by-step: starting BiPAP in a COPD exacerbation

Step	Action
1. Decide and explain	Confirm indication (pH ≤7.35, $PaCO_2$ >45 despite medical therapy); explain to the patient; set a ceiling of care (is intubation appropriate if NIV fails?)
2. Position	Sit up at 30–45° or more
3. Choose mask	Measure with the sizing gauge; oronasal mask first; protect the nasal bridge (hydrocolloid dressing)
4. Initial settings	**IPAP 10–12, EPAP 4–5 cmH_2O**, S/T mode, backup rate 12–15/min, Ti 0.8–1.2 s, rise time slow, cycle 30–40%
5. Hold, then strap	Hold the mask to the face for the first minutes; then fasten straps — two fingers should pass under the strap
6. Titrate IPAP	Increase IPAP by 2–5 cmH_2O every 10–30 minutes to **IPAP 18–25** (aim exhaled VT 6–8 ml/kg IBW, RR <25, comfort)
7. Titrate EPAP	Increase EPAP (6–8) if ineffective triggering (intrinsic PEEP), obstructive apnoeas, or hypoxaemia
8. Oxygen	Entrain O_2 into the mask/circuit; titrate to SpO_2 88–92%
9. Reassess	Clinical review at 30–60 min; **ABG at 1 hour** and 4 hours; continue as long as possible in the first 24 hours (≥20 h/day initially)
10. Document	Settings, leak, VT, RR, SpO_2, ABG, decision plan if NIV fails

Initial settings for common conditions

Condition	Mode	Starting pressures	Titrate to
COPD exacerbation	BiPAP S/T	IPAP 10–12 / EPAP 4–5	IPAP 18–25, EPAP 5–8; VT 6–8 ml/kg; pH normalising
Cardiogenic pulmonary oedema	CPAP or BiPAP	CPAP 8–10; or IPAP 10–15 / EPAP 5–8	CPAP 10–12; SpO_2 ≥94%, RR <25; give nitrates, diuretics
Obesity hypoventilation	BiPAP S/T (or AVAPS)	IPAP 14–16 / EPAP 6–8	EPAP 8–12 (upper airway), IPAP 20–30; target VT 8 ml/kg IBW
Neuromuscular disease (myasthenia, motor neuron disease, muscular dystrophy), kyphoscoliosis	BiPAP S/T with backup rate	IPAP 10–12 / EPAP 4–5, backup rate 14–16	Normal $PaCO_2$, comfort; avoid NIV in bulbar weakness
Hypoxaemic failure (pneumonia, early ARDS)	CPAP (helmet) or BiPAP	CPAP 8–10; or PS 6–8, EPAP 8–10	Keep VT ≤8–9.5 ml/kg (large VT predicts failure — HACOR, Carteaux)
Pre-oxygenation for intubation	BiPAP/CPAP, FiO_2 1.0	PS 5–10, PEEP 5–10	3–5 minutes before RSI
Post-extubation (high risk)	BiPAP alternating with HFNC	IPAP 10–12 / EPAP 5–6	24–48 h

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What to monitor

Parameter	Target / alarm
Respiratory rate	Should fall within 1–2 hours; >30–35/min after 1–2 h is a warning sign
Exhaled tidal volume	6–8 ml/kg IBW (COPD); >9.5 ml/kg in hypoxaemic failure predicts failure
Leak	Unintentional leak <20–30 L/min (vented masks have intentional leak in addition)
SpO_2	88–92% (hypercapnic) or 92–96%
ABG	pH and $PaCO_2$ improvement at 1–2 h and 4–6 h
Sensorium	GCS, flapping tremor, drowsiness (rising CO_2)
Haemodynamics	BP, heart rate (hypotension from reduced preload)
Synchrony	Patient's effort matches machine cycling (watch chest, waveforms)
Comfort and skin	Dyspnoea score, mask pressure areas, eye irritation

Predictors of NIV failure — the HACOR score (hypoxaemic patients)

Variable	Category	Points
Heart rate	≤120	0
Heart rate	≥121	1
pH	≥7.35	0
pH	7.30–7.34	2
pH	7.25–7.29	3
pH	<7.25	4
GCS	15	0
GCS	13–14	2
GCS	11–12	5
GCS	≤10	10
PaO_2/FiO_2	≥201	0
PaO_2/FiO_2	176–200	2
PaO_2/FiO_2	151–175	3
PaO_2/FiO_2	126–150	4
PaO_2/FiO_2	101–125	5
PaO_2/FiO_2	≤100	6
Respiratory rate	≤30	0
Respiratory rate	31–35	1
Respiratory rate	36–40	2
Respiratory rate	41–45	3
Respiratory rate	≥46	4

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KEY POINT **HACOR >5 after 1 hour of NIV predicts >80% failure** — intubate early rather than late. In hypoxaemic patients, also watch tidal volume (>9.5 ml/kg) and very negative oesophageal swings (patient self-inflicted lung injury, P-SILI).

Signs of NIV failure in hypercapnic patients

Time	Failure signs → consider intubation
1–2 hours	pH not improving or falling (especially pH <7.25), $PaCO_2$ rising, RR not falling, worsening drowsiness or agitation
Any time	Respiratory/cardiac arrest, haemodynamic instability, GCS falling, copious secretions, vomiting/aspiration, inability to tolerate mask, worsening hypoxaemia
Late failure (after 48 h)	Often due to comorbidity, hyperglycaemia, sleep disturbance — high mortality; review goals of care

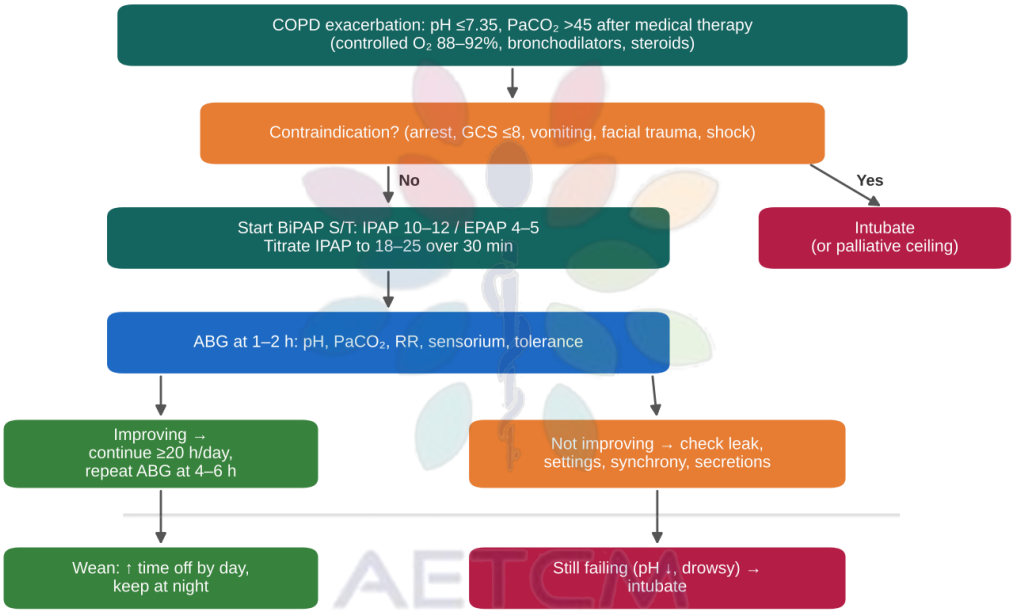


Figure 5.1. NIV decision pathway in acute hypercapnic respiratory failure.

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Troubleshooting NIV

Problem	Cause	Solution
Large leak	Wrong mask size, loose straps, mouth leak with nasal mask, nasogastric tube	Refit/resize, chin strap, change to oronasal or total-face mask, NG tube seal pad; reduce IPAP if very high
Rising $PaCO_2$	Inadequate IPAP, leak, rebreathing (blocked exhalation port), hyperoxia, patient-ventilator asynchrony	Increase IPAP, fix leak, check exhalation port, lower FiO_2 to SpO_2 88–92%, check trigger
Persistent hypoxaemia	Low EPAP, secretions, pneumothorax, progression	Increase EPAP and FiO_2; physiotherapy; CXR; consider intubation
Ineffective triggering (patient breathes but no breath delivered)	Intrinsic PEEP, leak, insensitive trigger	Increase EPAP to offset PEEPi, reduce leak, increase trigger sensitivity

Problem	Cause	Solution
Auto-triggering	Leak, cardiac oscillations, over-sensitive trigger	Reduce leak, decrease trigger sensitivity
Delayed cycling (patient fights expiration)	Leak prolongs inspiration	Raise cycle threshold (40–50%), set Ti max 1.0–1.2 s
Gastric distension, aerophagia	High IPAP (>20–25)	Keep IPAP ≤20–25; NG tube if severe
Nasal bridge ulcer	Mask too tight	Hydrocolloid dressing, alternate interfaces, loosen straps
Dry mouth, eye irritation	Leak, no humidification	Heated humidifier, refit mask, eye lubricants
Agitation, claustrophobia	Fear, hypoxia, hypercapnia, delirium	Reassurance, coaching, low-dose dexmedetomidine or haloperidol (avoid benzodiazepines/opioid excess); rule out hypercapnia
Hypotension	Reduced venous return	Fluids if responsive, reduce pressures

Weaning from NIV

Approach	Detail
When	Clinical improvement, pH ≥7.35, RR <25, underlying cause treated (usually after 24–72 h)
How	Increase time off NIV during the day first (e.g. 2–4 h off, then longer), keep NIV at night; or reduce IPAP progressively
Stop	When off NIV during the day with stable ABG; consider home NIV if persistent hypercapnia ($PaCO_2$ >53 mmHg 2–4 weeks after discharge — HOT-HMV)

NIV location and staffing

- Deliver acute NIV in an ICU, HDU or dedicated respiratory ward with nurses trained in NIV (1:2 nursing in the first hours).
- A clear plan for failure (intubation or palliative ceiling) must be documented **before** starting.

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CHAPTER 6

The Ventilator from First Principles

For the final-year student meeting a ventilator for the first time

What a ventilator does — and does not do

A mechanical ventilator is a machine that **pushes gas into the lungs** and then **lets it come out**. It does not "breathe for the lungs" in any magical way: it only generates pressure and flow. When you breathe normally, your diaphragm makes the pressure inside the chest negative and air is sucked in. A ventilator does the opposite: it makes the pressure at the mouth (or tube) positive and air is pushed in. Expiration is almost always passive — the elastic lung recoils and gas flows out.

The ventilator buys time. It keeps oxygen in and carbon dioxide out, rests tired muscles and protects the airway, while we treat the cause — pneumonia, sepsis, overdose, heart failure. It cures nothing by itself, and every breath it gives can injure the lung if we are careless.

Feature	Normal breathing	Positive-pressure ventilation
Pressure in the chest during inspiration	Negative (−2 to −8 cmH_2O pleural pressure)	Positive (airway pressure 15–30 cmH_2O)
Venous return	Increased during inspiration	Decreased → may cause hypotension
Distribution of gas	To dependent (lower) lung, which is better perfused	To non-dependent (upper) lung — V/Q mismatch
Airway protection	Glottis, cough	Cuffed tube; cough suppressed
Humidification	Nose and upper airway	Must be added (HME or heated humidifier)

Indications for intubation and ventilation — think "A-B-C-D"

Category	Examples
A — Airway	Unable to protect airway (GCS ≤8, bulbar palsy), obstruction (angio-oedema, burns, epiglottitis), massive haemoptysis
B — Breathing (oxygenation)	Hypoxaemia despite HFNC/NIV (SpO_2 <90% on FiO_2 >0.6, P/F <100–150)
B — Breathing (ventilation)	Rising $PaCO_2$ with acidosis despite NIV, exhaustion, apnoea, RR >35–40 with fatigue, paradoxical breathing
C — Circulation	Shock (reduces work of breathing and oxygen consumption), post-cardiac arrest
D — Disability / Drugs / Decline expected	Status epilepticus, need for deep sedation or procedure, anticipated deterioration (burns, transfer), severe agitation preventing care

KEY POINT The decision to intubate is **clinical** — do not wait for an ABG in a patient who is obviously tiring.

The parts of a ventilator system

Component	Function
Gas supply	Oxygen and medical air (pipeline at ≈ 4 bar / 50 psi) blended to the set FiO_2
Control panel / screen	Settings, monitored values, waveforms, alarms

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Component	Function
Inspiratory valve	Delivers flow according to the mode
Inspiratory limb of circuit	Carries gas to the patient (through humidifier)
Y-piece	Joins inspiratory and expiratory limbs at the patient; flow sensor often here
HME filter or heated humidifier	Warms and humidifies gas; HME also filters bacteria/viruses
Catheter mount and ET/tracheostomy tube	Interface with the patient
Expiratory limb and valve	Returns exhaled gas; the expiratory valve holds PEEP
Sensors	Pressure, flow, O_2; calculate volume and mechanics
Backup battery	Runs 30 min–several hours during power failure

The "breath" — four phases and three variables

Every mechanical breath can be described by answering three questions: **what starts it (trigger)**, **what controls it during inspiration (target or limit)** and **what ends it (cycle)**. After the breath, the baseline pressure (PEEP) is held during expiration.

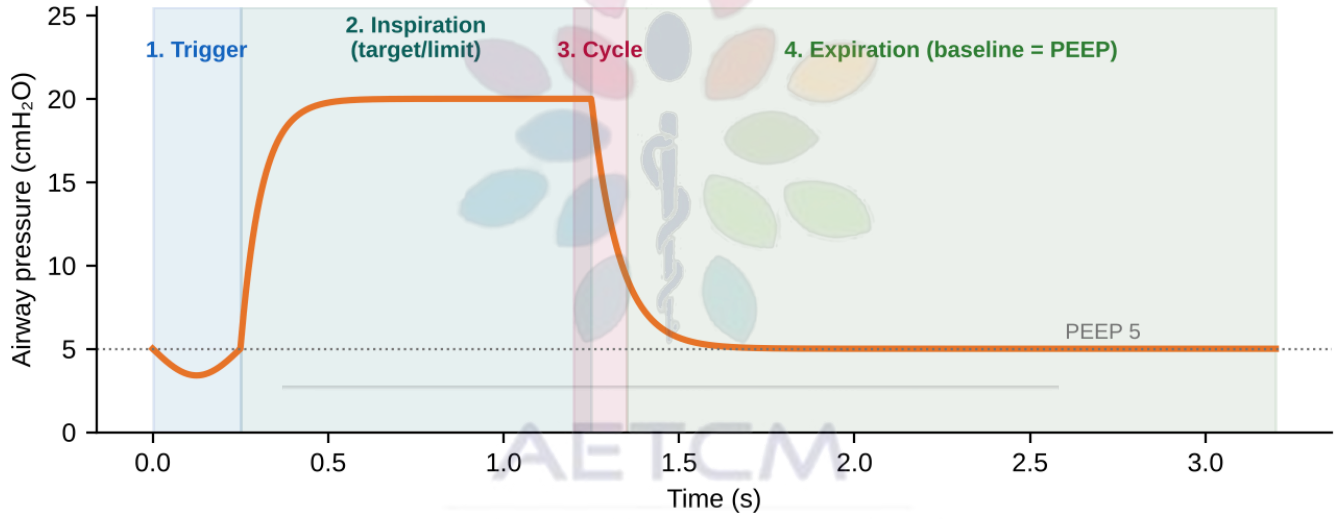


Figure 6.1. The four phases of a mechanical breath: trigger → inspiration (target) → cycle → expiration (baseline PEEP).

Phase variable	Question	Options
Trigger	What starts inspiration?	**Time** (machine-triggered, set rate); **pressure** (patient effort drops airway pressure by 1–2 cmH_2O); **flow** (patient effort diverts 1–3 L/min of the bias flow) — flow trigger is more sensitive and preferred
Target / limit	What is controlled during inspiration?	**Volume / flow** (volume control: set VT and flow — pressure varies); **pressure** (pressure control/support: set pressure — volume varies)
Cycle	What ends inspiration?	**Volume** or **time** (volume control); **time** (pressure control: set Ti); **flow** (pressure support: inspiration ends when flow falls to 25% of peak)
Baseline	What happens in expiration?	PEEP — pressure held above atmospheric

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Breath types

Breath type	Triggered by	Cycled by	Example
Mandatory, machine-triggered	Time (machine)	Machine (time/volume)	Controlled breath in a paralysed patient
Mandatory, patient-triggered (assisted)	Patient	Machine	Patient triggers an A/C breath — still gets the full set VT/pressure
Spontaneous, supported	Patient	Patient (flow)	Pressure support breath
Spontaneous, unsupported	Patient	Patient	CPAP without pressure support

The equation of motion — the only equation you need

The pressure the ventilator must generate (plus any pressure from the patient's muscles) is spent on two things: pushing gas through the airways (resistance × flow) and stretching the lungs (volume ÷ compliance), on top of PEEP.

Equation	Meaning
Pvent + Pmus = (Flow × Resistance) + (Volume / Compliance) + PEEP	Total pressure = resistive pressure + elastic pressure + baseline
Peak pressure (PIP)	Resistive + elastic + PEEP → rises with **both** airway resistance and stiff lungs
Plateau pressure (Pplat)	Elastic + PEEP (no flow during an inspiratory hold) → rises **only** with stiff lungs/chest wall
Peak − plateau	Resistive pressure → large gap means **airway** problem (bronchospasm, secretions, kinked/biting tube)

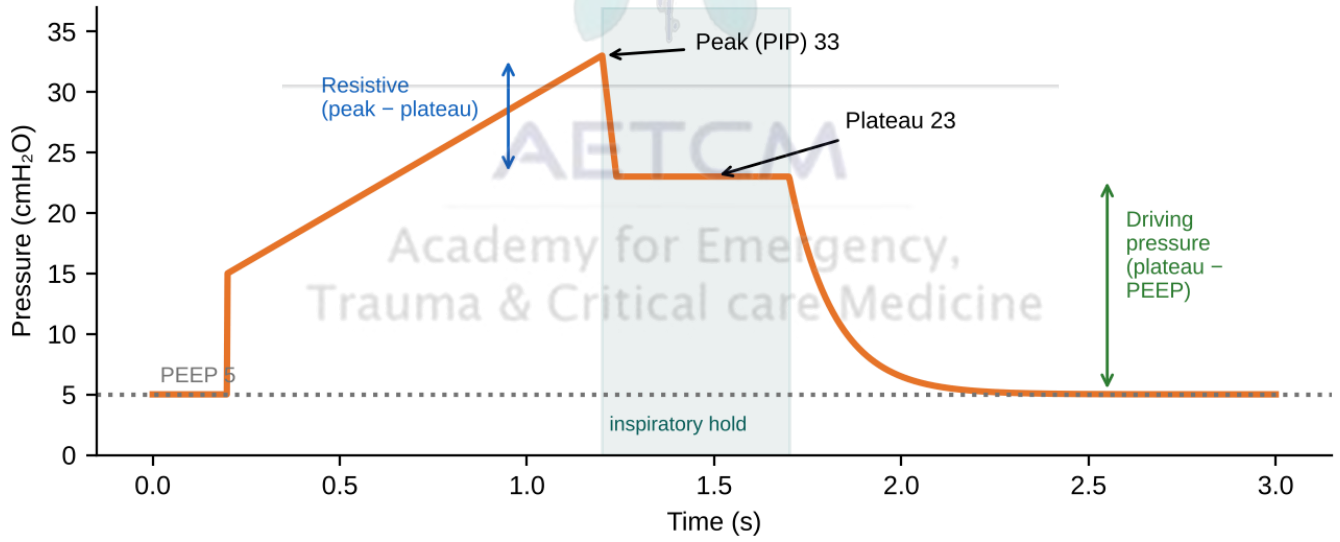


Figure 6.2. Airway pressure with an inspiratory hold: peak pressure, the drop to plateau (resistance), plateau (compliance) and PEEP.

The basic settings — what every knob means

Setting	Meaning	Usual adult start
Mode	How breaths are delivered (Chapter 7)	Volume or pressure assist-control
Tidal volume (VT)	Volume per breath	**6–8 ml/kg predicted (ideal) body weight**; 6 ml/kg in ARDS

Setting	Meaning	Usual adult start
Respiratory rate (RR, f)	Set (backup) breaths per minute	14–20/min (higher in metabolic acidosis, lower in asthma)
Inspiratory pressure (Pinsp / PC above PEEP)	Pressure applied in pressure modes	10–20 cmH_2O, titrated to VT
PEEP	End-expiratory pressure	5 cmH_2O (higher in hypoxaemia)
FiO_2	Inspired oxygen fraction	1.0 initially, then reduce to SpO_2 92–96%
Inspiratory flow / Ti / I:E	Speed and duration of inspiration	Flow 40–60 L/min; Ti 0.8–1.2 s; I:E 1:2 (1:3–1:5 in asthma/COPD)
Trigger sensitivity	Effort needed to start a breath	Flow 2–3 L/min or pressure −1 to −2 cmH_2O
Pressure support (PS)	Help for spontaneous breaths	5–15 cmH_2O
Flow pattern	Square (constant) or decelerating ramp	Decelerating gives lower peak pressure and better distribution
Rise time / slope	Speed of pressure rise in pressure modes	Moderate; faster in air hunger
Alarms	High/low pressure, volume, minute ventilation, RR, apnoea	Peak pressure alarm ≈ 10 cmH_2O above usual peak

Predicted (ideal) body weight — always use it, never actual weight

Men	Women
PBW (kg) = 50 + 0.91 × (height in cm − 152.4)	PBW (kg) = 45.5 + 0.91 × (height in cm − 152.4)

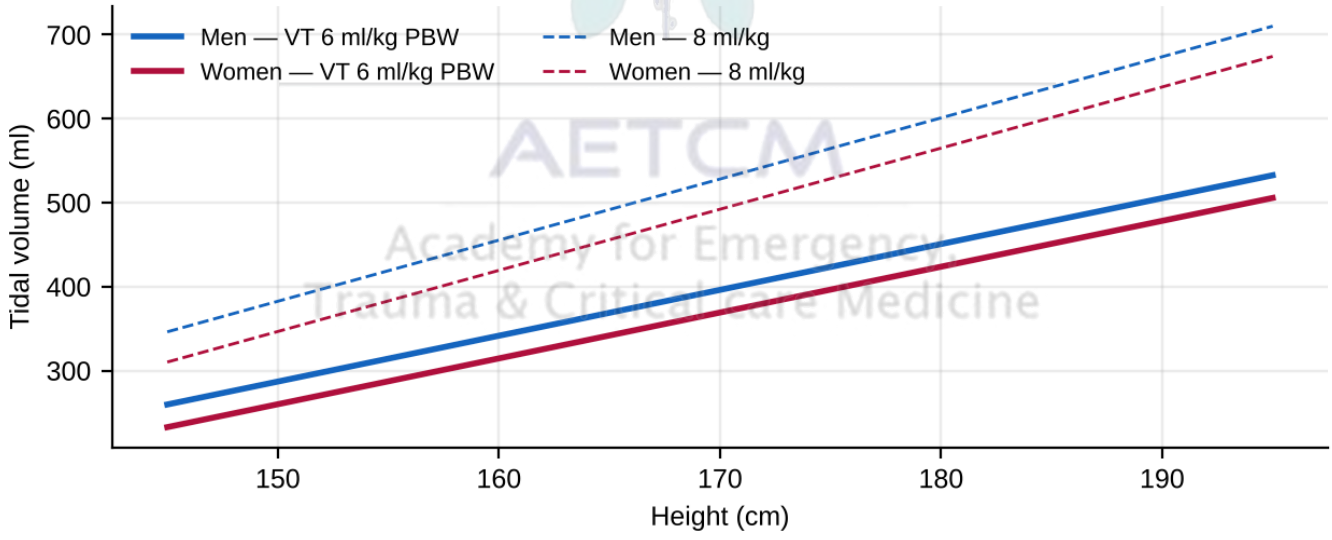


Figure 6.3. Predicted body weight and 6 ml/kg tidal volume by height (men and women).

Height (cm)	PBW male (kg)	VT 6 ml/kg (male)	PBW female (kg)	VT 6 ml/kg (female)
150	47.8	287	43.3	260
155	52.4	314	47.9	287
160	56.9	341	52.4	314
165	61.5	369	57.0	342

Height (cm)	PBW male (kg)	VT 6 ml/kg (male)	PBW female (kg)	VT 6 ml/kg (female)
170	66.0	396	61.5	369
175	70.6	423	66.1	396
180	75.1	451	70.6	424
185	79.7	478	75.2	451

KEY POINT Lungs do not grow with obesity — a 120-kg man who is 165 cm tall has lungs sized for 61 kg. Using actual weight would give a dangerous tidal volume of 720–960 ml. Measure height (or use ulna length / knee height) in every ventilated patient.

Glossary of pressures on the screen

Displayed value	Meaning	Usual safe limit
Ppeak (PIP)	Highest pressure during inspiration	<35–40 cmH_2O
Pplat	Pressure during inspiratory hold — reflects alveolar pressure	**≤30 cmH_2O** (≤27–28 in ARDS)
PEEP (set)	Baseline pressure	5–15 (up to 20+) cmH_2O
Total PEEP / intrinsic PEEP	Pressure measured with an expiratory hold	PEEPi = total PEEP − set PEEP (should be ≈ 0)
Pmean	Average pressure across the cycle — main determinant of oxygenation	10–20 cmH_2O
Driving pressure	Pplat − PEEP	**<15 cmH_2O**
VTe	Exhaled tidal volume (what really entered the lung)	6–8 ml/kg PBW
MVe	Exhaled minute volume	6–10 L/min (higher with acidosis, sepsis)
P0.1	Airway occlusion pressure in first 100 ms — respiratory drive	1.5–3.5 cmH_2O (>4 high drive; <1 low)

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CHAPTER 7

Modes of Ventilation

Volume control, pressure control, SIMV, pressure support, CPAP and dual modes — explained simply

The naming problem

Every manufacturer invents its own names (PRVC, VC+, Autoflow, APV, BiLevel, BIPAP, DuoPAP…). Do not memorise names — ask the three questions of Chapter 6: **What is the control variable (volume or pressure)? What is the breath sequence (every breath mandatory, some mandatory, or all spontaneous)? What triggers and cycles the breath?** Every mode then falls into one of a few families.

Breath sequence	Meaning	Common names
CMV (continuous mandatory ventilation) = Assist-control (A/C)	**Every** breath is mandatory — machine-triggered at the set rate or patient-triggered, but always the full set volume/pressure	VC-AC, PC-AC, CMV, A/C, IPPV
IMV (intermittent mandatory ventilation)	Set number of mandatory breaths; between them the patient breathes spontaneously (with or without pressure support)	SIMV-VC, SIMV-PC (+PS)
CSV (continuous spontaneous ventilation)	**All** breaths are patient-triggered and patient-cycled	PSV, CPAP, PAV, NAVA, CPAP+PS

Volume-controlled ventilation (VCV)

Feature	Detail
Set	Tidal volume, rate, inspiratory flow (or Ti), flow pattern, PEEP, FiO_2, trigger
Guaranteed	**Tidal volume** (and minute ventilation)
Variable	Airway pressure — rises if compliance falls or resistance rises
Advantages	Fixed VT and minute ventilation; easy to measure plateau, compliance, resistance; familiar
Disadvantages	Barotrauma risk if lungs stiffen (watch Pplat); fixed flow may not meet patient demand → flow starvation and asynchrony
Best for	Initial ventilation, paralysed patients, measuring mechanics, controlling $PaCO_2$ (TBI, metabolic acidosis)

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Pressure-controlled ventilation (PCV)

Feature	Detail
Set	Inspiratory pressure (above PEEP), inspiratory time, rate, rise time, PEEP, FiO_2
Guaranteed	**Peak airway pressure**
Variable	Tidal volume — falls if compliance falls, resistance rises or patient stops effort; rises suddenly when lungs improve
Advantages	Limits airway pressure; decelerating flow matches demand and improves gas distribution; better comfort
Disadvantages	VT not guaranteed (hypoventilation or volutrauma) — set VT alarms carefully
Best for	ARDS, bronchopleural fistula/leaks, paediatrics, patients with high flow demand

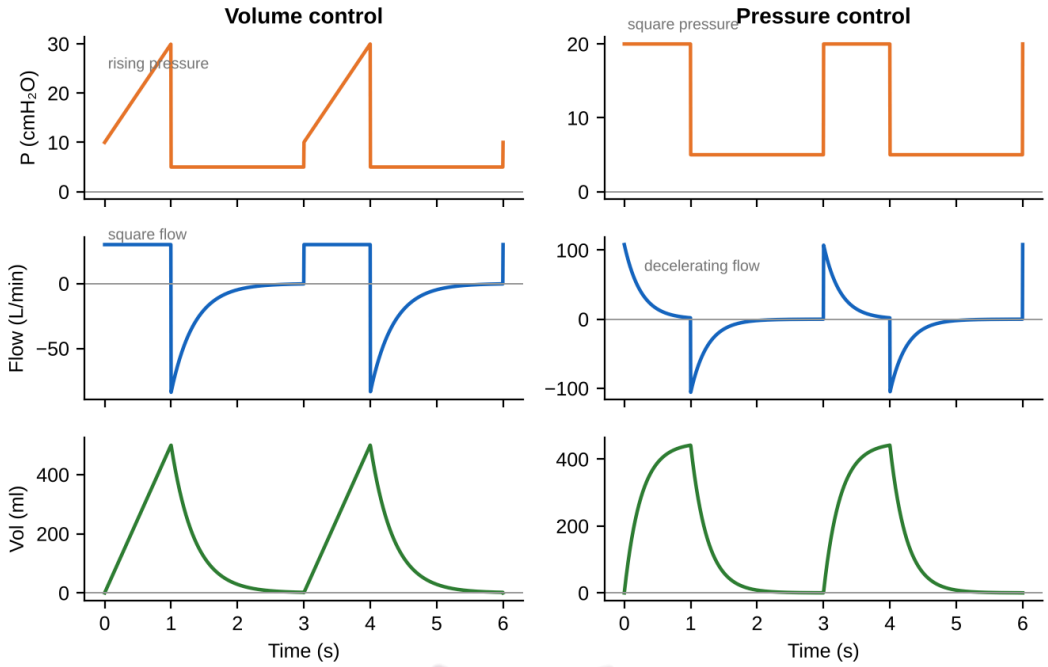


Figure 7.1. Waveforms in volume control (square flow, rising pressure) and pressure control (square pressure, decelerating flow).

VCV versus PCV at a glance

Parameter	Volume control	Pressure control
Pressure waveform	Rising to a peak (shark-fin with square flow)	Square (constant)
Flow waveform	Square (constant) or set ramp	Decelerating (exponential), determined by patient mechanics
VT if compliance falls	Same (pressure rises)	Falls
Pressure if compliance falls	Rises	Same
Inspiratory time determined by	Flow and VT	Set Ti
Alarm to watch	High pressure	Low exhaled VT / minute volume

Dual-control (adaptive pressure) modes — PRVC / VC+ / Autoflow

The operator sets a **target tidal volume**; the ventilator delivers pressure-controlled breaths and adjusts the pressure breath-by-breath (usually by 1–3 cmH_2O) to achieve that volume, using the lowest pressure needed. It combines the decelerating flow of PC with the volume guarantee of VC.

- **Caution:** if the patient makes a strong effort, the machine sees a big volume and **reduces** support — worsening fatigue. Leaks also confuse the algorithm.

Synchronised intermittent mandatory ventilation (SIMV)

A set number of mandatory breaths (volume or pressure) is synchronised with the patient's effort; extra breaths are spontaneous, usually with pressure support. It was popular for weaning but studies show it **prolongs** weaning compared with pressure support or daily spontaneous breathing trials. Use is now limited.

Pressure support ventilation (PSV)

Feature	Detail
Trigger	Patient (flow/pressure) — **no backup rate** (set apnoea ventilation alarm)
Target	Pressure above PEEP (e.g. PS 10 cmH_2O)
Cycle	Flow — inspiration ends when flow falls to a % of peak (expiratory trigger sensitivity, ETS, default 25%)
Patient controls	Rate, inspiratory time, tidal volume (depends on effort, PS and mechanics)
Uses	Weaning, comfortable spontaneous breathing, SBT (PS 5–8 + PEEP 5)
Adjust	Target RR <25–30 and VT 6–8 ml/kg; ETS 40–60% in COPD (earlier cycling), 5–15% in restrictive lung for longer inspiration
Risks	Apnoea in sedated patients; over-assistance → low drive, ineffective efforts, central apnoeas at night

CPAP on the ventilator

- Constant PEEP with no added pressure support; all breaths spontaneous. Used for spontaneous breathing trials (CPAP 5 or "PEEP 5 / PS 0"), and for patients with good drive and mechanics.

Advanced modes (detailed in Chapter 19)

Mode	Principle	Main use
APRV (airway pressure release ventilation)	Long high pressure (P-high, T-high 4–6 s) with brief releases (T-low 0.4–0.8 s); spontaneous breathing throughout	Recruitment in ARDS (rescue)
BiLevel / BIPAP / DuoPAP	Two pressure levels with free spontaneous breathing at both	Pressure control allowing patient breathing
PAV+ (proportional assist ventilation)	Support proportional to patient effort, based on measured resistance and compliance	Synchrony, weaning
NAVA (neurally adjusted ventilatory assist)	Triggered and cycled by diaphragmatic electrical activity (Edi catheter)	Severe asynchrony, neonates
ASV / INTELLiVENT-ASV	Closed-loop: targets minute ventilation and optimal rate/VT by Otis equation	Automated ventilation and weaning
Volume support (VS)	Pressure support that adjusts to reach a target VT	Weaning
Automatic tube compensation (ATC)	Adds pressure to overcome ET tube resistance	Weaning, SBT
HFOV (high-frequency oscillation)	Very small VT at 3–15 Hz around a high mean pressure	Neonates; adult use declined after OSCILLATE trial

Which mode should I choose?

Situation	Suggested mode	Why
Just intubated, sedated, unstable	VC-AC (or PC-AC)	Full support, guaranteed ventilation
ARDS	VC-AC with VT 6 ml/kg (or PC-AC keeping VT ≤6–8 and Pplat ≤30)	Lung protection; easy plateau measurement
Severe asthma / COPD	VC-AC with low rate, high flow, long expiratory time	Control of minute ventilation and auto-PEEP

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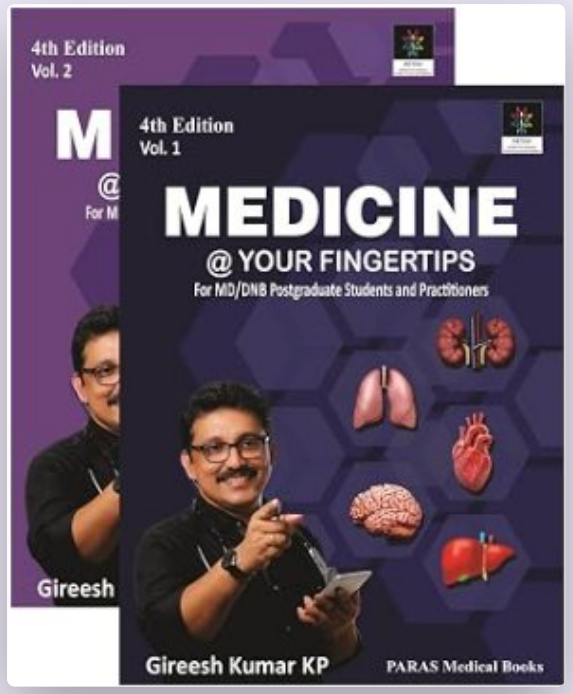
Situation	Suggested mode	Why
Recovering, awake, good drive	PSV	Comfort, exercise of respiratory muscles, weaning
Spontaneous breathing trial	PS 5–8 + PEEP 5, or CPAP 5, or T-piece	Test of readiness
Severe asynchrony	Switch VC ↔ PC, adjust flow/Ti/ETS; consider PAV+/NAVA	Match patient demand
Large air leak / bronchopleural fistula	PC-AC, lowest PEEP and Ti	Minimises leak

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CHAPTER 8

Intubation and Initial Ventilator Settings

From the decision to intubate to the first hour on the ventilator

Preparation — STOP-MAID checklist

Letter	Item
S — Suction	Yankauer suction working, under the pillow
T — Tools	Laryngoscope (direct and video), blades, bougie, stylet, ET tubes (7.0–7.5 women, 7.5–8.0 men), 10 ml syringe, tie, supraglottic device, cricothyroidotomy kit
O — Oxygen	Pre-oxygenation source (NRBM 15 L/min + nasal cannula 15 L/min, or BiPAP/HFNC), BVM with PEEP valve
P — Positioning	Ear-to-sternal notch, head-up 20–30° (ramped in obesity)
M — Monitors	SpO_2, ECG, BP (cycle every 2–3 min), **waveform capnography**
A — Assistant	Roles allocated; drugs drawn and labelled; "plan A, B, C" verbalised
I — IV access	Two lines; fluids and push-dose vasopressor ready (phenylephrine 100 µg or adrenaline 10–20 µg)
D — Drugs	Induction agent, neuromuscular blocker, post-intubation sedation

Rapid sequence intubation — drugs

Drug	Dose	Notes
Ketamine	1–2 mg/kg IV (0.5–1 mg/kg in shock)	Haemodynamically stable; bronchodilator — preferred in asthma, shock
Etomidate	0.2–0.3 mg/kg IV	Cardiovascularly stable; single dose adrenal suppression
Propofol	1–2 mg/kg IV (0.5 mg/kg in elderly/shock)	Hypotension — avoid in shock
Midazolam	0.1–0.3 mg/kg	Slow onset, hypotension; mainly for sedation
Fentanyl	1–3 µg/kg	Blunts pressor response (raised ICP, aortic dissection)
Succinylcholine	1.5 mg/kg IV	Onset 45–60 s, lasts 8–10 min; avoid in hyperkalaemia, burns/crush >24 h, neuromuscular disease, malignant hyperthermia
Rocuronium	1.0–1.2 mg/kg IV	Onset 60 s, lasts 45–60 min; reversed by sugammadex 16 mg/kg
Vecuronium	0.1 mg/kg	Slower onset; maintenance paralysis

Confirmation of tube placement

- **Waveform capnography** for ≥5–6 breaths — the gold standard ("no trace = wrong place", even in cardiac arrest a low trace persists with CPR).
- Bilateral chest rise and air entry, no epigastric gurgling, misting.
- Chest X-ray: tip 3–5 cm above the carina (at the level of the clavicular heads, T2–T4); usual depth at the teeth ≈ 21 cm (women) and 23 cm (men).
- Cuff pressure 20–30 cmH_2O (manometer).

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The first settings — a universal starting point

Setting	Start with	Then
Mode	Volume assist-control (VC-AC)	Change once stable if needed
Tidal volume	**6–8 ml/kg PBW** (6 ml/kg if ARDS risk)	Check Pplat ≤30
Rate	16–20/min	Adjust to pH/$PaCO_2$; match pre-intubation minute ventilation in metabolic acidosis
PEEP	5 cmH_2O	Higher (8–15) in hypoxaemia, obesity
FiO_2	1.0	Wean to SpO_2 92–96% within 30–60 min
Inspiratory flow	50–60 L/min, decelerating	60–100 L/min in COPD/asthma to lengthen expiration
Trigger	Flow 2 L/min	Avoid auto-triggering
Alarms	High pressure 35–40; low VT 75% of set; apnoea 20 s	—

Initial settings by clinical situation

Situation	VT (ml/kg PBW)	Rate	PEEP	Key point
Normal lungs (overdose, neuro, surgery)	6–8	12–16	5	Avoid hyperoxia; plan early extubation
ARDS	6 (4–8)	20–35	Per PEEP/FiO_2 table	Pplat ≤30, driving pressure ≤15, permissive hypercapnia (pH ≥7.20)
Asthma / COPD	6–8	**8–12**	5 (or ≤80% PEEPi in COPD)	I:E 1:4–1:5; accept $PaCO_2$ high; watch auto-PEEP
Severe metabolic acidosis (DKA, salicylate, sepsis)	8	**25–35** (match pre-intubation MV)	5	Do not let $PaCO_2$ rise after intubation — can cause arrest
Raised ICP / TBI	6–8	To $PaCO_2$ 35–40 mmHg	5–8	Brief hyperventilation ($PaCO_2$ 30–35) only for herniation
Cardiogenic shock / LV failure	6–8	16–20	5–10	PEEP reduces LV afterload; beware hypotension
RV failure / massive PE	6	16–20	5 (low)	Avoid high Pplat and hypercapnia; intubation is dangerous — support BP first
Obesity	6–8 (PBW)	16–20	**10–15**	Reverse Trendelenburg, recruitment
Pregnancy (3rd trimester)	6–8	To $PaCO_2$ 30–32 mmHg (normal for pregnancy)	5–10	Left lateral tilt; normal pregnancy $PaCO_2$ is low
Neuromuscular disease	8	12–16	5	Normal lungs; comfortable volumes, early tracheostomy if prolonged
Bronchopleural fistula	Lowest possible	Lowest	Lowest	PC mode, short Ti, permissive hypercapnia

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The first 30 minutes after intubation — checklist

Check	Action
Tube	Capnography trace, depth at teeth, secured, CXR ordered
Haemodynamics	Post-intubation hypotension is common (vasodilation, loss of sympathetic drive, PPV) — fluids, vasopressor
Sedation and analgesia	Start **analgesia-first** sedation before the NMB wears off (fentanyl + propofol/midazolam/ketamine) — awareness under paralysis is a never-event
Ventilator mechanics	Peak, plateau, driving pressure, PEEPi, exhaled VT
ABG	20–30 minutes after settings; adjust rate/VT for pH and $PaCO_2$, FiO_2/PEEP for oxygen
Orogastric tube	Decompress stomach; confirm position
Head-up 30–45°, eye care, mouth care	VAP and pressure-injury prevention
Document	Settings, tube size and depth, grade of view, complications

KEY POINT **Post-intubation hypotension** (SBP <90 within 30 min) occurs in up to 1 in 4 critically ill patients. Pre-load fluid in shock, use ketamine or reduced doses, have vasopressor running or drawn up, and avoid excessive bagging (raised intrathoracic pressure and auto-PEEP).

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CHAPTER 9

Monitoring the Ventilated Patient

Pressures, mechanics, alarms and the daily ventilator round

Measuring plateau pressure and auto-PEEP

Manoeuvre	How	What it shows
Inspiratory hold (pause)	Press "inspiratory hold" for 0.5–2 s at end-inspiration in a **passive** patient on volume control (square flow)	Plateau pressure (alveolar pressure) → compliance and driving pressure
Expiratory hold	Press "expiratory hold" for 2–4 s at end-expiration	Total PEEP; intrinsic PEEP = total − set PEEP
Valid only if	Patient relaxed, no leak, no active breathing (sedate briefly if necessary)	Patient effort makes readings unreliable

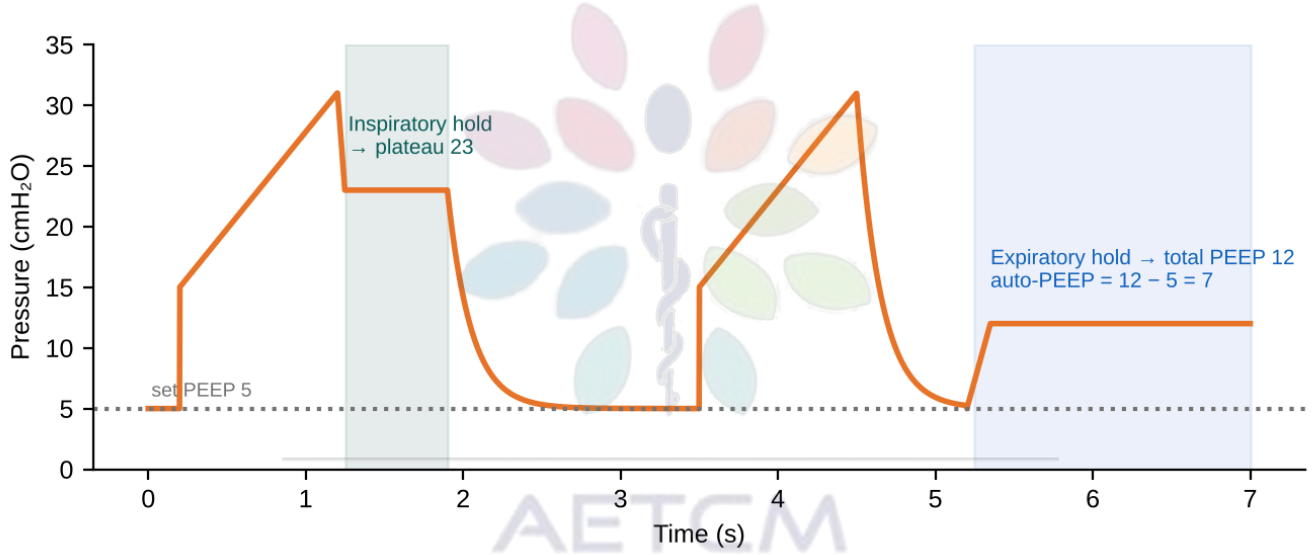


Figure 9.1. Inspiratory and expiratory hold manoeuvres on the pressure–time curve. The expiratory hold reveals hidden auto-PEEP.

Worked example — calculate the mechanics

Given	Calculation	Result
VT 450 ml, flow 60 L/min (1 L/s), PIP 34, Pplat 24, PEEP 8	Cstat = 450 / (24 − 8)	**28 ml/cmH₂O** (low — stiff lung)
Same	Raw = (34 − 24) / 1	**10 cmH₂O/L/s** (normal for ET tube)
Same	Driving pressure = 24 − 8	**16 cmH₂O** (slightly high — reduce VT to 6 ml/kg)
Same	Cdyn = 450 / (34 − 8)	17 ml/cmH₂O
Time constant	τ = R × C = 10 × 0.028	0.28 s (3τ = 0.84 s to empty)

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High peak pressure — is it the airway or the lung?

Pattern	Peak	Plateau	Gap (peak − plateau)	Causes
Airway (resistance) problem	↑	Normal	**Large (>10)**	Bronchospasm, secretions, biting/kinked tube, small tube, mucus plug, high flow rate
Lung/chest wall (compliance) problem	↑	↑	Normal (<10)	Pneumothorax, ARDS progression, pulmonary oedema, atelectasis/collapse, right main-bronchus intubation, abdominal distension, auto-PEEP, chest wall rigidity, patient fighting
Low peak	↓	↓	—	Leak (cuff, circuit, disconnection), patient pulling breath (high effort in VC), extubation

The essential monitoring parameters

Parameter	Target / normal	Act when
SpO_2	92–96% (88–92% COPD)	<88% or >97% on FiO_2 >0.4 (reduce FiO_2)
$ETCO_2$	35–45 mmHg (close to $PaCO_2$)	Sudden fall: disconnection, extubation, cardiac arrest, massive PE; gradual rise: hypoventilation, fever
Exhaled VT	6–8 ml/kg PBW	Difference between set and exhaled >10% = leak
Plateau pressure	≤30 cmH_2O	Reduce VT
Driving pressure	≤15 cmH_2O	Reduce VT, optimise PEEP
Auto-PEEP	≈ 0	>5 cmH_2O: reduce rate/VT, lengthen expiratory time, bronchodilate
Mechanical power	<17 J/min (proposed)	Reduce rate, VT, pressures
Respiratory rate (total vs set)	Total close to set in sedated patient	Total ≫ set: pain, anxiety, acidosis, high drive, auto-triggering
P0.1	1.5–3.5 cmH_2O	>3.5–4: excessive drive (consider more support/sedation); <1: over-assistance
Pocc (occlusion pressure)	Pmus ≈ 0.75 × Pocc	Pmus >10–15 indicates injurious effort (P-SILI risk)
Cuff pressure	20–30 cmH_2O	Check every 8 hours

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Mechanical power — the energy delivered to the lung

Mechanical power combines all the components of a breath (VT, rate, pressures, flow) into the energy transferred to the lung each minute. A simplified formula for volume control is: **MP (J/min) = 0.098 × RR × VT (L) × (Ppeak − ½ × driving pressure)**. Values above about 17 J/min have been associated with higher mortality. It reminds us that **respiratory rate matters**, not only tidal volume.

Ventilator alarms — what to do

Alarm	Common causes	First action
High pressure	Coughing, biting, secretions, bronchospasm, kinked tube, pneumothorax, endobronchial tube, asynchrony	Look at patient → suction → check tube → peak vs plateau → treat cause
Low pressure / disconnection	Circuit disconnection, cuff leak, extubation, chest-drain leak	Check connections from patient to machine; listen for leak; check tube depth; bag if needed

Alarm	Common causes	First action
Low exhaled VT / MV	Leak, reduced effort (PS mode), stiff lungs in PC mode, obstruction	Check leak and patient; increase support
High MV / high RR	Pain, anxiety, fever, acidosis, hypoxaemia, auto-triggering	Assess cause; treat pain/fever; adjust trigger
Apnoea	Over-sedation, neurological deterioration, PS mode without backup	Switch to backup/controlled mode; assess sedation
High PEEP / auto-PEEP	Gas trapping, expiratory limb obstruction, wet HME/filter	Change filter, lengthen expiration
Low FiO_2 / gas supply	Oxygen pipeline failure, O_2 sensor fault	Switch to cylinder, check sensor calibration
Power / battery	Mains failure	Connect to UPS/mains; have BVM ready

KEY POINT **If in doubt, disconnect the patient from the ventilator and hand-ventilate with a bag and 100% oxygen** — this separates a machine problem from a patient problem.

The daily ventilator round — FAST HUGS BID + ventilator check

Item	Question
Indication	Why is the patient still ventilated? Is the cause improving?
Settings	Mode, VT/PBW, rate, PEEP, FiO_2 — still appropriate?
Mechanics	Pplat, driving pressure, compliance, resistance, auto-PEEP — better or worse?
Gas exchange	P/F ratio, $PaCO_2$, pH trend
Sedation	RASS target (−1 to 0 in most); daily sedation interruption done?
Spontaneous breathing trial	Screened for readiness? SBT done?
Imaging	CXR (tube position, new infiltrates, pneumothorax), lung ultrasound
Bundle	Head-up 30–45°, oral care, cuff pressure, subglottic suction, DVT and stress-ulcer prophylaxis, glucose, nutrition
Lines and tubes	Days in place, still needed?
Goals	Family meeting, goals of care, tracheostomy timing

Lung ultrasound at the bedside

Pattern	Meaning
A-lines with lung sliding	Normal aerated lung (or asthma/COPD/PE)
Absent sliding, no B-lines, lung point	Pneumothorax (lung point is diagnostic)
Multiple B-lines (≥3 per space), bilateral	Interstitial syndrome — pulmonary oedema, ARDS
Consolidation with dynamic air bronchograms	Pneumonia
Static air bronchograms, absent sliding on one side	Atelectasis / endobronchial intubation
Anechoic space above diaphragm	Pleural effusion
Diaphragm excursion <1 cm or thickening fraction <20–30%	Diaphragm dysfunction — weaning failure risk

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CHAPTER 10

Ventilator Graphics — Waveforms and Loops

Reading the screen: pressure, flow and volume curves, P–V and F–V loops

Three scalars on every screen

Modern ventilators show three curves against time: **pressure–time**, **flow–time** and **volume–time**. Read them from top to bottom, breath by breath. Each tells a part of the story: pressure shows how hard the machine pushes, flow shows the direction and speed of gas (above the line = into the patient, below = out), and volume shows how much went in and whether it all came back.

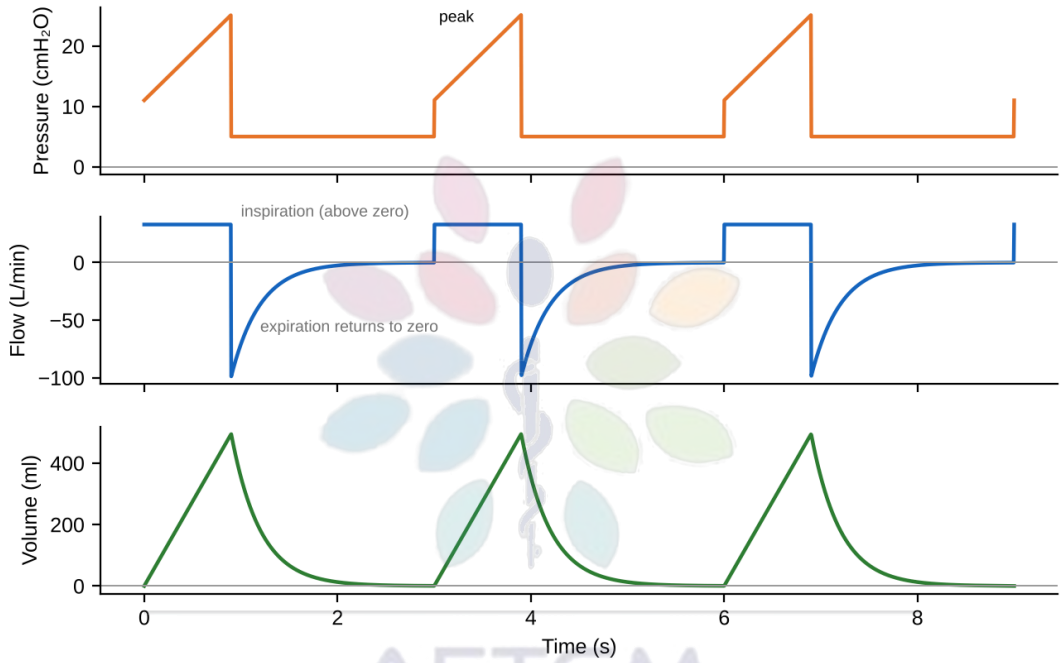


Figure 10.1. Normal scalars in volume control: pressure rises to a peak, square inspiratory flow, linear volume rise and complete exhalation.

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Pressure–time curve

Feature	Interpretation
Baseline above zero	PEEP
Small negative dip before inspiration	Patient trigger (pressure trigger); absent in machine-triggered breaths
Shape in VC (constant flow)	Linear rise to a peak (resistive step + elastic ramp)
Concave upward ("scooped") in VC	Patient pulling harder than the set flow — **flow starvation**
Square shape	Pressure control/support
Overshoot at start of PC breath	Rise time too fast
Plateau with inspiratory hold	Alveolar pressure (Pplat)
Stress index (shape in constant-flow VC)	Linear (SI ≈ 1): good; concave down (SI <1): recruitment possible; concave up (SI >1): overdistension

Flow–time curve — the most useful curve

Feature	Interpretation
Square inspiratory flow	Volume control with constant flow
Decelerating inspiratory flow	Pressure control/support, or set ramp in VC
Inspiratory flow reaching zero before cycling (PC)	All set pressure has equilibrated — Ti adequate or long
Inspiratory flow not reaching zero at end of inspiration (PC)	Ti too short or high resistance — more time would add volume
Expiratory flow returning to zero before next breath	Complete exhalation — no auto-PEEP
Expiratory flow not returning to zero before next breath	**Air trapping / auto-PEEP** — lengthen expiratory time
Saw-tooth oscillations on expiratory limb	Secretions or water in the circuit — suction/drain
Scooped, slowly decaying expiratory flow with low peak	Airway obstruction (bronchospasm)
Small positive deflections in expiration without a breath	Ineffective triggering (missed efforts)

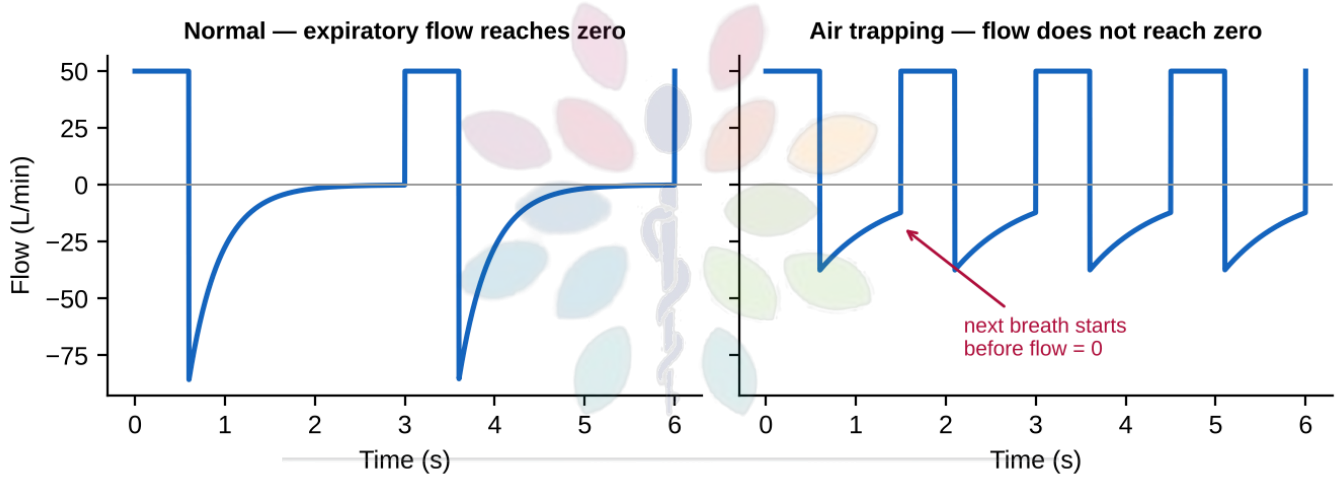


Figure 10.2. Air trapping: expiratory flow does not return to baseline before the next breath starts (auto-PEEP), compared with normal.

Volume–time curve

Feature	Interpretation
Rise during inspiration, fall to zero in expiration	Normal
Expiratory limb does not return to zero (drops vertically at the next breath)	**Leak** (cuff, circuit, bronchopleural fistula) or air trapping
Exhaled < inhaled volume by >10–15%	Significant leak
Larger volume than set	Active patient effort in PC/PS, or calibration error

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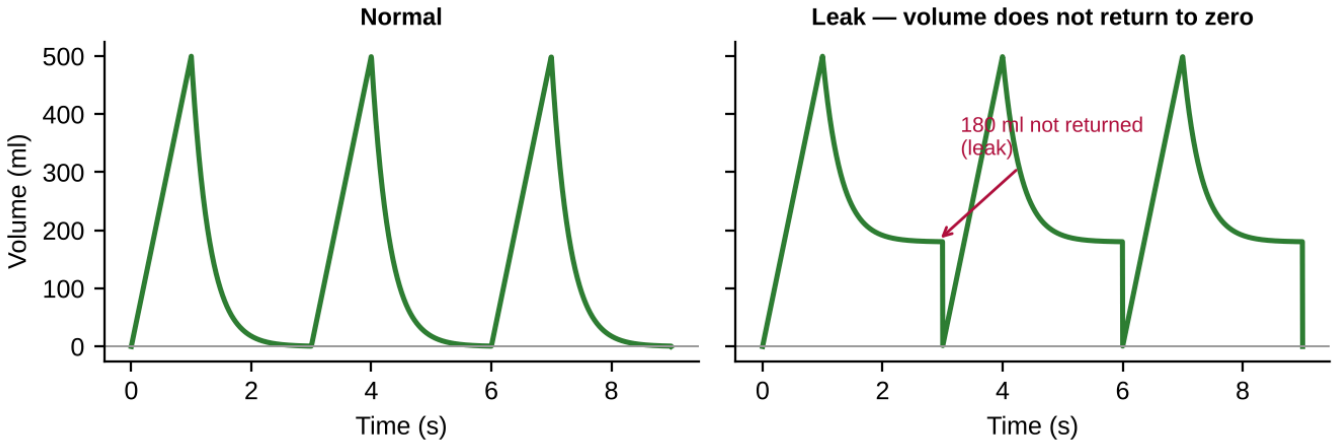


Figure 10.3. Leak on the volume–time curve: the exhaled volume never returns to baseline and the trace is reset at the next breath.

Pressure–volume (P–V) loop

Feature	Interpretation
Loop direction	Mandatory breaths: counter-clockwise; patient-triggered breaths start with a small clockwise "tail" on the left (trigger work)
Slope of the loop	Compliance — flatter slope = stiffer lung (ARDS, fibrosis); steeper slope = compliant lung
Lower inflection point (LIP)	Opening of collapsed alveoli — set PEEP a little above it (historical approach)
Upper inflection point / "beaking"	Overdistension at end of inspiration ("duck-bill") — reduce VT or pressure
Wide loop (hysteresis)	High airway resistance
Large trigger tail	Insensitive trigger or high effort

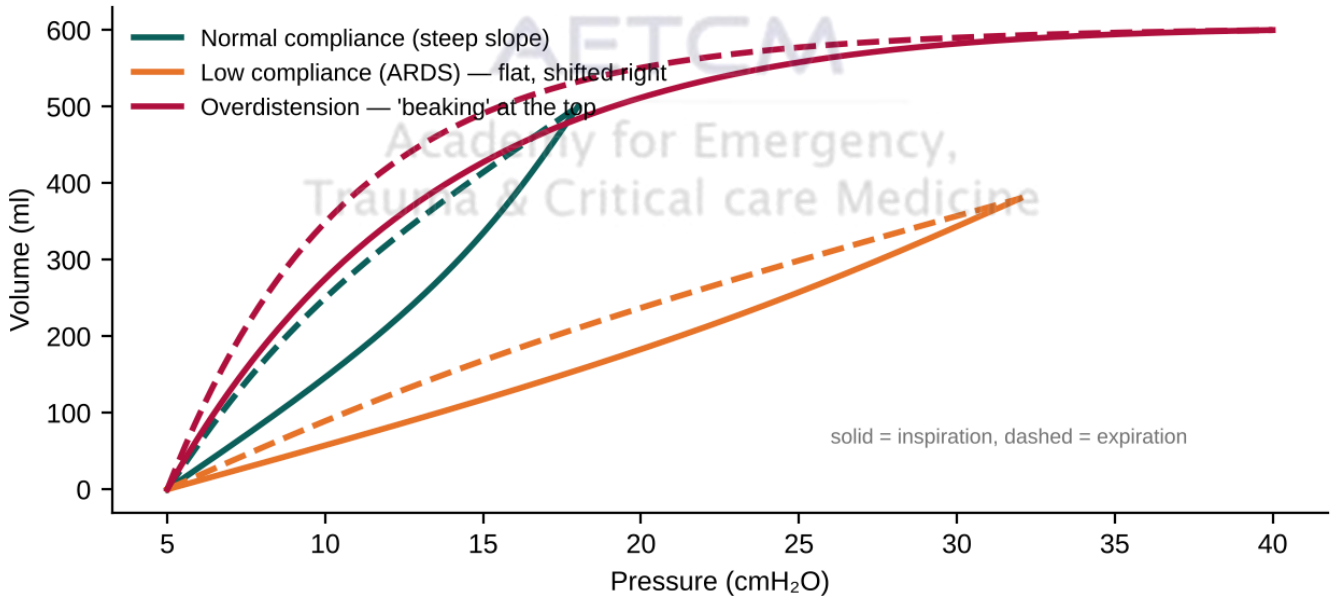


Figure 10.4. Pressure–volume loops: normal, low compliance (rightward, flat), and overdistension "beaking" at the top.

Flow–volume (F–V) loop

Feature	Interpretation
Scooped (concave) expiratory limb	Airway obstruction (asthma, COPD) — improves after bronchodilator
Expiratory limb not reaching zero flow	Auto-PEEP
Saw-tooth pattern	Secretions — suction
Loop does not close	Leak

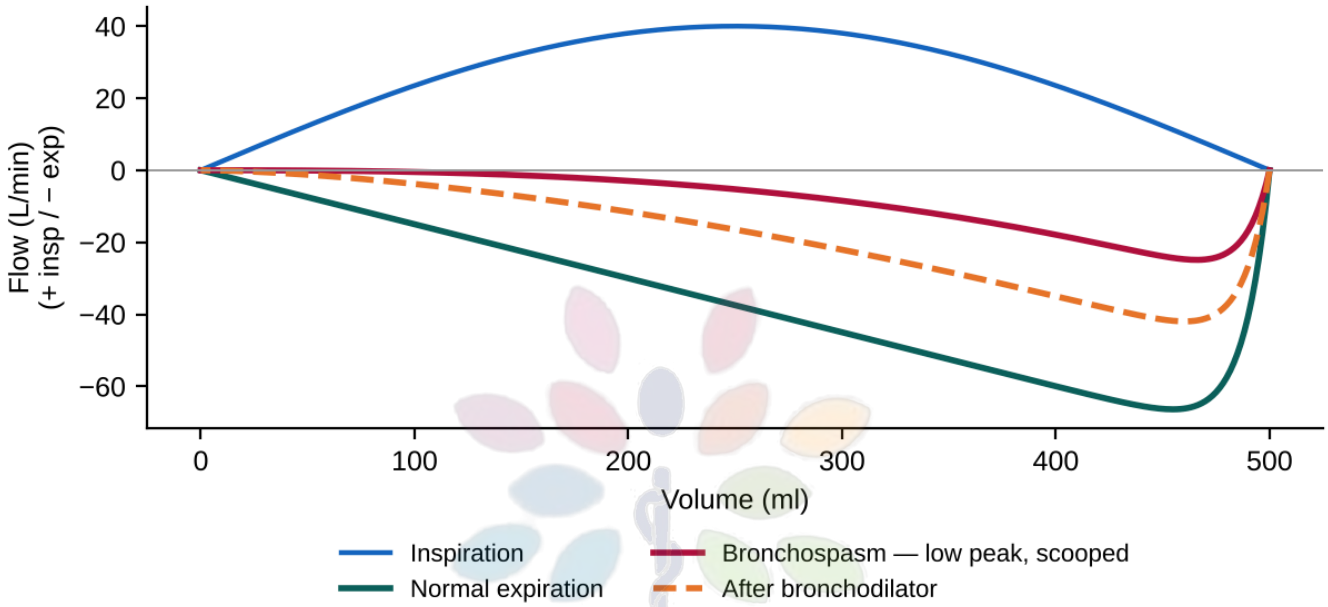


Figure 10.5. Flow–volume loops: normal versus bronchospasm (scooped expiratory limb that improves after bronchodilator).

Reading waveforms — a stepwise approach

Step	Ask
1. Mode	What is controlled — volume (square flow) or pressure (square pressure)?
2. Trigger	Machine or patient? Are there efforts that do not trigger?
3. Inspiration	Does flow meet demand (pressure curve shape)? Does PC flow reach zero?
4. Cycling	Does the patient keep inhaling after the machine cycles (double trigger, delayed cycling)?
5. Expiration	Does flow reach zero (auto-PEEP)? Does volume return to zero (leak)?
6. Mechanics	Peak, plateau, driving pressure, loop slope
7. Compare with the patient	Look at the chest, abdomen, neck muscles at the same time

Common waveform patterns — summary table

Problem	Pressure–time	Flow–time	Volume–time	Fix
Auto-PEEP	Raised total PEEP on expiratory hold	Expiratory flow not reaching zero	—	↓ rate, ↓ VT, ↑ inspiratory flow, bronchodilator

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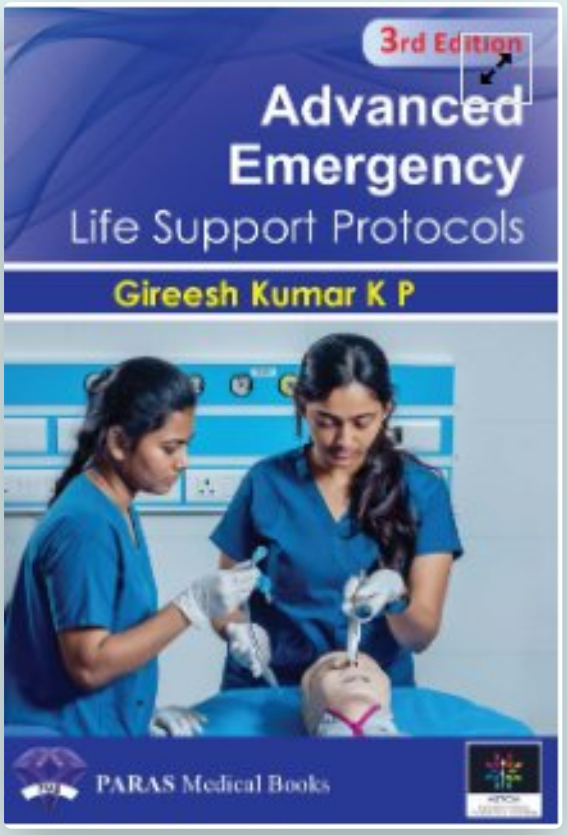
Problem	Pressure–time	Flow–time	Volume–time	Fix
Leak	Fails to maintain pressure	Expiratory flow smaller than inspiratory	Does not return to zero	Check cuff, circuit, chest drain
Secretions	Fluctuations	Saw-tooth	—	Suction
Bronchospasm	High peak, normal plateau	Low peak expiratory flow, prolonged expiration	—	Bronchodilators, ↑ expiratory time
Flow starvation (VC)	Concave "scooped" inspiratory pressure	Fixed square flow	—	↑ flow, switch to PC/PRVC, treat drive
Ineffective efforts	Small dips without a breath	Small positive bumps in expiration	—	Reduce auto-PEEP, reduce over-assistance, adjust trigger
Double triggering	Two stacked breaths	Two inspirations without full expiration	Stacked (≈ double VT)	↑ Ti or VT, ↓ ETS, consider sedation
Overdistension	Concave-up pressure (SI >1)	—	—	↓ VT/PEEP

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CHAPTER 11

Blood Gases and Adjusting the Ventilator

A simple system for oxygenation and ventilation changes

Five-step ABG interpretation

Step	Question	Normal / rule
1	Is the patient acidaemic or alkalaemic?	pH 7.35–7.45
2	Is the primary problem respiratory or metabolic?	$PaCO_2$ 35–45 mmHg; HCO_3^- 22–26 mmol/l
3	Is compensation appropriate?	Metabolic acidosis: expected $PaCO_2 = 1.5 \times HCO_3^- + 8$ (± 2) (Winter's); acute respiratory acidosis: HCO_3^- ↑ 1 per 10 $PaCO_2$; chronic: ↑ 3.5 per 10
4	Anion gap (if metabolic acidosis)	$AG = Na^+ - (Cl^- + HCO_3^-) = 8–12$ (add 2.5 for each 1 g/dl fall in albumin below 4)
5	Oxygenation	PaO_2, P/F ratio, A–a gradient

Fix oxygenation — FiO_2 and mean airway pressure

Lever	How it helps	Typical step
FiO_2	Raises alveolar O_2 — quickest fix; little effect on shunt	0.1–0.2; aim FiO_2 ≤0.6
PEEP	Recruits alveoli, reduces shunt, raises mean airway pressure	2–3 cmH_2O at a time; check BP and Pplat
Inspiratory time / I:E	Raises mean airway pressure	Ti 1.0 → 1.2 s; I:E 1:2 → 1:1.5 (watch auto-PEEP)
Recruitment manoeuvre	Opens collapsed lung	Selected patients (Chapter 16)
Prone position	Homogenises ventilation, improves V/Q	P/F <150 in ARDS — 16+ h/day
Treat the cause	Suction, physiotherapy, drain effusion/pneumothorax, diuresis, antibiotics	—

Fix ventilation — minute ventilation

Lever	How it helps	Typical step
Respiratory rate	Raises minute ventilation, lowers $PaCO_2$	2–4 breaths/min (watch expiratory time and auto-PEEP)
Tidal volume (or Pinsp in PC)	Raises alveolar ventilation more efficiently	50 ml (stay ≤8 ml/kg, Pplat ≤30)
Reduce dead space	Remove extra tubing, HME → heated humidifier	50–100 ml less dead space
Reduce CO_2 production	Treat fever, avoid overfeeding with carbohydrate, control shivering	—
Permissive hypercapnia	Accept $PaCO_2$ 50–80, pH ≥7.20 for lung protection (not in raised ICP, severe PH, sickle crisis)	—

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Formulas to predict the new setting

Goal	Formula	Example
New rate to reach target $PaCO_2$	New RR = current RR × current $PaCO_2$ / desired $PaCO_2$	RR 14, $PaCO_2$ 60 → target 40: 14 × 60/40 = **21/min**
New VT to reach target $PaCO_2$	New VT = current VT × current $PaCO_2$ / desired $PaCO_2$	VT 400, $PaCO_2$ 50 → 40: 500 ml (only if Pplat allows)
New FiO_2 for target PaO_2 (rough)	New FiO_2 = desired PaO_2 × current FiO_2 / current PaO_2	PaO_2 200 on 0.8 → target 80: 0.32 (then confirm by SpO_2)
Expected PaO_2 on a given FiO_2 (normal lung)	PaO_2 ≈ FiO_2 (%) × 5	FiO_2 40% → PaO_2 ≈ 200

Quick adjustment chart

ABG finding	Change
PaO_2 low, $PaCO_2$ normal	↑ FiO_2; ↑ PEEP; check tube, secretions, CXR
PaO_2 high (SpO_2 >97%)	↓ FiO_2 first (to ≤0.4–0.5), then ↓ PEEP slowly
$PaCO_2$ high, pH low (respiratory acidosis)	↑ RR or ↑ VT (if Pplat permits); check for auto-PEEP, dead space, sedation in PS mode
$PaCO_2$ low, pH high (respiratory alkalosis)	↓ RR (if mandatory), ↓ VT; treat pain/anxiety if patient-triggered; consider PS mode
Metabolic acidosis	Keep $PaCO_2$ at compensated level (Winter's); ↑ RR; treat the cause
Metabolic alkalosis	Do not hyperventilate; correct chloride/potassium; acetazolamide if weaning delayed
Hypoxaemia + hypercapnia in ARDS	Prioritise lung protection; ↑ RR (up to 35), permissive hypercapnia, prone

SpO_2/FiO_2 titration protocol (nurse-driven)

SpO_2	Action
>96%	↓ FiO_2 by 0.05–0.1 every 15–30 min to minimum 0.3
92–96%	No change
88–91%	↑ FiO_2 by 0.1; inform doctor if FiO_2 >0.6
<88%	↑ FiO_2 to 1.0, check DOPES, call doctor

$ETCO_2$ and $PaCO_2$

- Normally $PaCO_2$ exceeds $ETCO_2$ by 2–5 mmHg; the gap widens with increased dead space (PE, low cardiac output, high PEEP, COPD).
- A sudden fall of $ETCO_2$ suggests disconnection, cardiac arrest, massive PE, or a falling cardiac output; a rise with return of circulation suggests ROSC.
- Use $ETCO_2$ trends, but confirm with ABG after any major change.

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Venous blood gas and other shortcuts

Variable	Venous vs arterial
pH	Venous ≈ 0.03–0.04 lower
PCO_2	Venous ≈ 4–6 mmHg higher (unreliable in shock); a normal VBG PCO_2 excludes arterial hypercapnia
HCO_3^-	Similar (venous ≈ 1–2 higher)
PO_2	Not useful — use SpO_2
$ScvO_2$ / SvO_2	Oxygen extraction; $ScvO_2$ <70% suggests inadequate DO_2

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CHAPTER 12

Troubleshooting the Ventilated Patient

Sudden desaturation, high pressures, hypotension and the "fighting the ventilator" patient

Sudden deterioration — DOPES

Letter	Cause	Check	Fix
D — Displacement	Tube out, in oesophagus, in right main bronchus, cuff above cords	Capnography, tube depth, air entry, laryngoscopy	Reposition / re-intubate
O — Obstruction	Secretions, mucus plug, blood clot, kinked tube, patient biting	Pass suction catheter; high peak with normal plateau	Suction, saline lavage, bite block, change tube
P — Pneumothorax	Barotrauma, central line, trauma	High Pplat, unilateral absent air entry, hypotension, ultrasound (no sliding)	Needle/finger thoracostomy then chest drain
E — Equipment	Ventilator failure, disconnection, oxygen supply failure, wet filter	Disconnect and bag with 100% O_2	Replace equipment, call technician
S — Stacked breaths	Auto-PEEP / dynamic hyperinflation (asthma, COPD)	Expiratory flow not reaching zero; hypotension	Disconnect for 30–60 s, press on chest; then ↓ rate, ↑ expiratory time

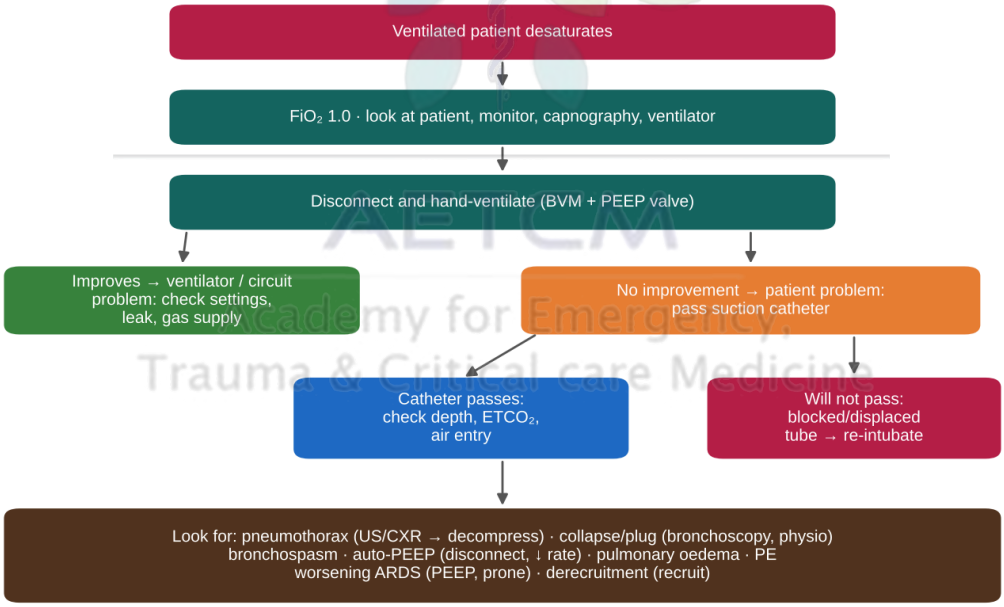


Figure 12.1. Algorithm for sudden desaturation in a ventilated patient.

Sudden desaturation — step by step

Step	Action
1	FiO_2 1.0; look at the patient, monitor, ventilator screen and capnography

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Step	Action
2	Disconnect from ventilator and hand-ventilate with BVM + PEEP valve — if the patient improves, the problem is the ventilator/circuit
3	Pass a suction catheter — confirms tube patency and clears secretions
4	Check tube depth and capnography; examine chest (unequal air entry → endobronchial tube or pneumothorax)
5	Bedside ultrasound / CXR: pneumothorax, collapse, effusion, pulmonary oedema
6	Consider other causes: PE, worsening ARDS, cardiac failure, bronchospasm, aspiration, positional change (lateral position with bad lung down), procedures (suction-related derecruitment)
7	Recruit (if derecruitment after suction/disconnection) and restore PEEP

High peak pressure alarm — algorithm

Finding	Likely cause	Action
Peak ↑, plateau normal	Airway resistance: secretions, bronchospasm, tube kink, biting	Suction, bronchodilator, bite block, check tube
Peak ↑, plateau ↑	Compliance: pneumothorax, endobronchial intubation, collapse, oedema, abdominal compartment, asynchrony/coughing	Examine, ultrasound, CXR, treat cause; sedate if fighting
Peak ↑ only during patient effort	Patient coughing or fighting	Assess pain, agitation, delirium, hypoxia; adjust settings; sedation

Hypotension after intubation or on the ventilator

Cause	Clue	Action
Reduced venous return (positive pressure)	Hypovolaemia, high PEEP	Fluid bolus, reduce PEEP/VT
Sedative drugs	Recently induced/increased sedation	Reduce dose, vasopressor
Auto-PEEP	Asthma/COPD, high rate, aggressive bagging	Disconnect briefly, reduce rate
Tension pneumothorax	High Pplat, unilateral signs	Decompress immediately
Underlying shock / RV failure	Sepsis, PE, MI	Echo; vasopressors, inotropes
Arrhythmia, tamponade	ECG, echo	Specific treatment

The patient "fighting the ventilator"

Step	Consider
1. Rule out life threats	DOPES, hypoxaemia, pneumothorax
2. Patient factors	Pain, anxiety, delirium, full bladder, fever, metabolic acidosis, high respiratory drive, sepsis, withdrawal
3. Ventilator factors	Inadequate flow (flow starvation), Ti mismatch, trigger problems, auto-PEEP, inappropriate mode, too low/high VT
4. Adjust ventilator	Increase flow or switch to PC/PS; match Ti; adjust trigger, ETS; treat auto-PEEP
5. Only then sedate	Analgesia first (fentanyl); then propofol/dexmedetomidine; NMB only if severe ARDS or unsafe

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Cuff leak and tube problems

Problem	Sign	Action
Cuff leak	Audible leak, low exhaled VT, gurgling, low pressure alarm	Check cuff pressure; re-inflate; check pilot balloon; if cuff burst — exchange tube over a bougie/airway exchange catheter
Tube too shallow (cuff at cords)	Leak despite cuff inflation; tube depth reduced	Deflate cuff, advance 1–2 cm, reinflate; CXR
Endobronchial intubation	Unilateral air entry, high pressure, desaturation	Withdraw to 21/23 cm (adjust to CXR)
Blocked tube	Unable to pass suction catheter, very high pressure	Suction, lavage; if not cleared — re-intubate
Accidental extubation	Vocalising, no $ETCO_2$, large leak	BVM, re-intubate if needed (some may tolerate NIV/HFNC)

Power or gas failure

- Keep a self-inflating bag with mask and PEEP valve at every ventilated bed.
- Check UPS/backup battery daily; know the oxygen cylinder changeover procedure.
- Ventilator failure: bag the patient with 100% O_2 and call for a replacement — never troubleshoot the machine while the patient is unventilated.

Hypercapnia that does not improve

Cause	Action
Low minute ventilation (PS mode, sedation)	Switch to controlled mode or increase support
Dead space (HME, long catheter mount, PE, high PEEP)	Remove extra tubing, use heated humidifier; review PEEP
Auto-PEEP limiting VT	Treat obstruction, lengthen expiration
High CO_2 production	Fever, overfeeding, sepsis, shivering, bicarbonate infusion
Leak	Fix leak

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CHAPTER 13

Patient–Ventilator Asynchrony

Recognising and fixing the mismatch between the patient's brain and the machine

Why asynchrony matters

Asynchrony occurs when the timing or size of the ventilator breath does not match what the patient wants. It is present in up to a quarter of breaths in many patients. An **asynchrony index above 10%** (asynchronous events ÷ total breaths × 100) is associated with longer ventilation, more sedation, diaphragm injury and higher mortality. Most asynchrony is visible on the waveforms if you look for it.

Classification

Phase	Type	Mechanism
Trigger	Ineffective effort (missed trigger)	Patient effort fails to trigger — auto-PEEP, weak effort, over-assistance, insensitive trigger
Trigger	Auto-triggering	Machine delivers breaths without patient effort — leak, cardiac oscillations, water in circuit, over-sensitive trigger
Trigger	Double triggering	One effort triggers two breaths — patient's neural Ti longer than machine Ti, or high demand
Trigger	Reverse triggering	Machine breath triggers diaphragm contraction (entrainment) in deeply sedated patients — may cause breath stacking
Flow	Flow starvation (insufficient flow)	Set flow in VC lower than patient demand → concave pressure curve
Flow	Excessive flow / fast rise	Pressure overshoot, early cycling, discomfort
Cycle	Premature cycling	Machine stops inspiration before patient finishes → double trigger, discomfort
Cycle	Delayed cycling	Machine continues inspiration after the patient has started to exhale → expiratory muscle activation, pressure spike at end-inspiration; common in COPD with PS

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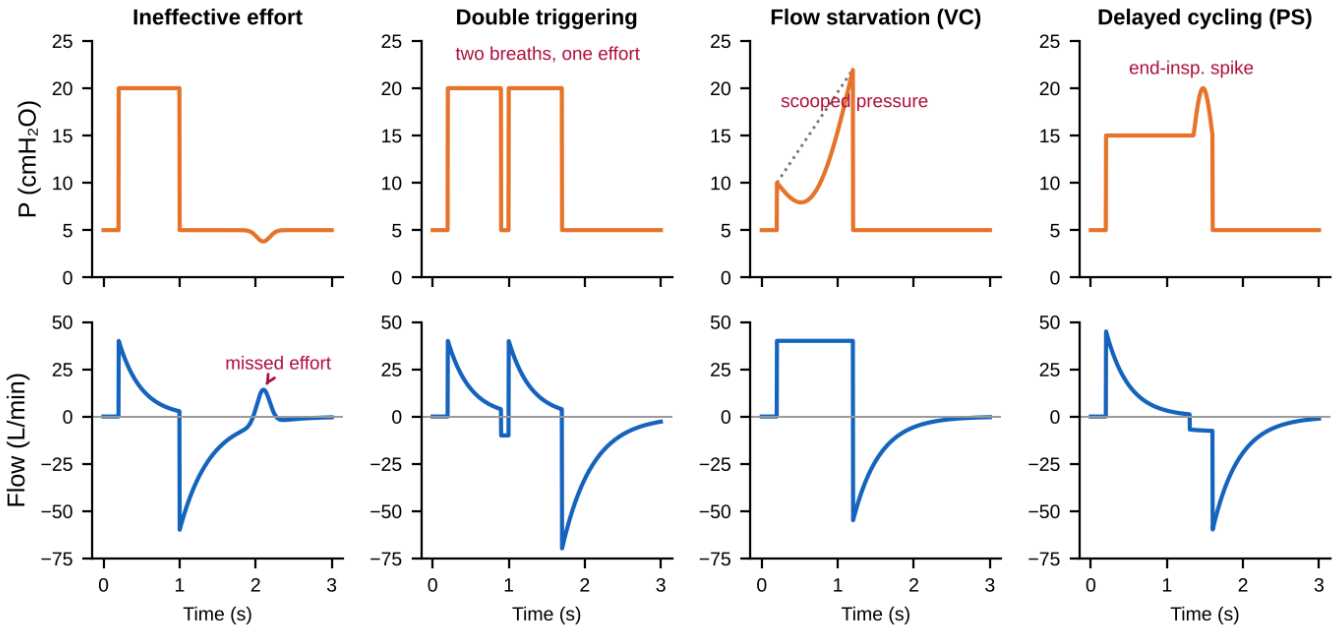


Figure 13.1. Common asynchronies on the pressure and flow curves: ineffective effort, double triggering, flow starvation and delayed cycling.

Recognition and treatment

Asynchrony	Waveform clue	Treatment
Ineffective efforts	Small dip in pressure and positive bump in expiratory flow without a delivered breath	↓ auto-PEEP (↓ VT/rate, ↑ expiratory time, bronchodilator); apply external PEEP ≈ 70–80% of PEEPi in COPD; ↓ PS if over-assisted; ↑ trigger sensitivity; ↓ sedation
Auto-triggering	Regular breaths at a high rate without a negative dip; rate matches heart rate	Fix leak, drain circuit water, ↓ trigger sensitivity
Double triggering	Two consecutive inspirations with little/no expiration; volume stacking ≈ 2 × VT	↑ Ti or VT (within limits), switch VC → PC, ↓ ETS in PS; if ARDS with high drive — ↑ sedation or NMB
Reverse triggering	Machine-triggered breath followed by patient effort at a fixed ratio (1:1, 1:2)	↓ sedation, change rate, switch to PS when possible
Flow starvation	Concave (scooped) pressure curve during VC inspiration	↑ peak flow (60–90 L/min), decelerating ramp, switch to PC/PRVC, reduce drive (fever, acidosis, pain)
Premature cycling	Flow trace shows a second small inspiration immediately after cycling	↑ Ti (PC), ↓ ETS (PS) e.g. 25% → 15%
Delayed cycling	Pressure spike at end of inspiration; abrupt fall in flow	↑ ETS (PS) to 40–60%, ↓ Ti (PC), ↓ PS

KEY POINT Before increasing sedation, ask: **is the ventilator doing what the patient wants?** Most asynchrony is better fixed by adjusting the ventilator than by deeper sedation.

High respiratory drive and P-SILI

Patients with acute lung injury and a strong drive can generate very large negative pleural pressures. Their own efforts cause local overstretch and pendelluft (gas moving from non-dependent to dependent lung) and increase transvascular

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pressure — **patient self-inflicted lung injury (P-SILI)**. Signs: P0.1 >3.5–4 cmH_2O, Pocc <−15 to −20 cmH_2O, large VT (>8–10 ml/kg) despite low support, large oesophageal pressure swings (ΔPes >10–15).

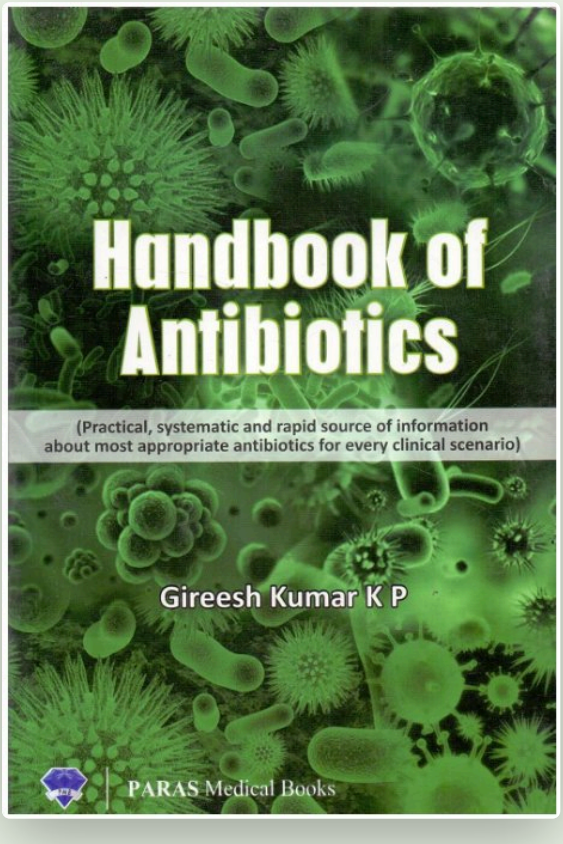
Strategy	Detail
Treat drivers	Fever, pain, acidosis, anxiety, hypoxaemia
Adjust support	Avoid both under-assistance (exhaustion) and over-assistance
Sedation	Propofol, opioids reduce drive; dexmedetomidine improves comfort
Deep sedation ± NMB	For short periods (24–48 h) in severe ARDS with injurious effort
Monitor	P0.1, Pocc, oesophageal pressure (Chapter 19)

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CHAPTER 14

Sedation, Analgesia, Delirium and Paralysis

eCASH, the ABCDEF bundle and drug doses

Principles — eCASH

Letter	Principle
e — early	Start comfort measures early after intubation
C — Comfort	Patient should be calm, comfortable, communicative
A — Analgesia first	Treat pain before giving sedatives (analgosedation)
S — minimal Sedation	Light sedation (RASS 0 to −1) unless a specific indication for deep sedation
H — Human care	Family presence, communication, sleep promotion, early mobilisation

Richmond Agitation–Sedation Scale (RASS)

Score	Term	Description
+4	Combative	Violent, danger to staff
+3	Very agitated	Pulls or removes tubes, aggressive
+2	Agitated	Frequent non-purposeful movement, fights ventilator
+1	Restless	Anxious but movements not aggressive
0	Alert and calm	—
−1	Drowsy	Sustained awakening (eye contact) to voice >10 s
−2	Light sedation	Briefly awakens to voice <10 s
−3	Moderate sedation	Movement or eye opening to voice but no eye contact
−4	Deep sedation	No response to voice; movement to physical stimulation
−5	Unarousable	No response to voice or physical stimulation

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Pain assessment — CPOT (Critical-Care Pain Observation Tool)

Domain	0	1	2
Facial expression	Relaxed	Tense (frowning)	Grimacing, eyelids tightly closed
Body movements	Absence of movement / normal	Protection (slow, touching pain site)	Restlessness, pulling tube, striking
Muscle tension (passive flexion/extension of arm)	Relaxed	Tense, rigid	Very tense
Compliance with ventilator (intubated)	Tolerating	Coughing but tolerating	Fighting ventilator

KEY POINT CPOT >2 (out of 8) indicates significant pain. In a non-communicating patient, an increase in heart rate or BP alone is not a reliable pain sign.

Sedative and analgesic drugs

Drug	Bolus	Infusion	Advantages	Cautions
Fentanyl	25–100 µg	25–200 µg/h (0.5–3 µg/kg/h)	Rapid, haemodynamically stable	Accumulates in fat after days; chest wall rigidity with large boluses
Morphine	2–5 mg	2–10 mg/h	Cheap	Histamine release, renal accumulation
Remifentanil	—	0.05–0.25 µg/kg/min	Ultra-short acting; not affected by organ failure	Expensive, hyperalgesia on stopping
Propofol	0.5–1 mg/kg (cautious)	5–50 µg/kg/min (0.3–3 mg/kg/h)	Rapid on/off — preferred for light sedation	Hypotension; PRIS with >4 mg/kg/h for >48 h (acidosis, rhabdomyolysis, arrhythmia); triglycerides; lipid calories (1.1 kcal/ml)
Midazolam	1–2 mg	1–10 mg/h (0.02–0.1 mg/kg/h)	Seizures, alcohol withdrawal, haemodynamic stability	Accumulates (renal/hepatic failure, obesity); delirium; prolonged ventilation — avoid routine use
Dexmedetomidine	No bolus (bradycardia)	0.2–1.4 µg/kg/h	Arousable sedation, less delirium; no respiratory depression — use during NIV and weaning	Bradycardia, hypotension; not for deep sedation
Ketamine	0.5–1 mg/kg	0.1–1 mg/kg/h (analgesic dose 0.1–0.3)	Bronchodilator, opioid-sparing, haemodynamically stable	Emergence phenomena, secretions, tachycardia
Haloperidol	2.5–5 mg IV	—	Agitated delirium (short-term)	QT prolongation, EPS
Quetiapine	25–50 mg 12-hourly enteral	—	Delirium with agitation, sleep	Sedation, QT

Daily sedation interruption (spontaneous awakening trial, SAT)

Step	Detail
Safety screen	No active seizures, alcohol withdrawal, escalating sedation for agitation, NMB, raised ICP, myocardial ischaemia
Stop sedation	Continue analgesia for pain
Pass	Opens eyes to voice or tolerates ≥4 h without agitation, anxiety, RR >35, SpO_2 <88%, arrhythmia
Fail	Restart sedation at **half** the previous dose and titrate
Pair with SBT	"Wake up and breathe" (ABC trial) — reduces ventilator days and mortality

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The ABCDEF bundle (ICU Liberation)

Element	Content
A	Assess, prevent and manage pain
B	Both spontaneous awakening and spontaneous breathing trials
C	Choice of analgesia and sedation (light, non-benzodiazepine)
D	Delirium — assess (CAM-ICU), prevent and manage
E	Early mobility and exercise
F	Family engagement and empowerment

Delirium — CAM-ICU

Feature	Assessment
1. Acute change or fluctuating mental status	Compared with baseline, or fluctuating RASS/GCS in last 24 h
2. Inattention	"Squeeze my hand when I say A" — SAVEAHAART; >2 errors = positive
3. Altered level of consciousness	RASS other than 0
4. Disorganised thinking	Yes/no questions ("Will a stone float on water?") and command
Delirium present	Features 1 + 2 + (3 or 4)

- Prevention: sleep hygiene (lights off, earplugs), glasses and hearing aids, reorientation, early mobility, avoid benzodiazepines and anticholinergics, treat pain, correct metabolic causes.

Neuromuscular blockade (NMB)

Indication	Drug and dose	Monitoring
Severe ARDS (P/F <150) in the first 48 h with dyssynchrony/injurious effort	Cisatracurium 0.15–0.2 mg/kg bolus, then 1–3 µg/kg/min (organ-independent elimination)	Train-of-four (aim 1–2 twitches), BIS if available; deep sedation **must** precede and accompany NMB
Raised ICP, status asthmaticus with dangerous dyssynchrony, hypothermia (shivering), transport	Vecuronium 0.1 mg/kg bolus, 0.8–1.2 µg/kg/min; atracurium 0.5 mg/kg, 5–10 µg/kg/min; rocuronium 0.6 mg/kg, 5–10 µg/kg/min	Eye care, DVT prophylaxis, pressure areas

KEY POINT Routine NMB is not beneficial in moderate–severe ARDS with light sedation (ROSE trial, 2019). Prolonged NMB, especially with steroids, causes ICU-acquired weakness. Stop as soon as possible.

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CHAPTER 15

Care of the Ventilated Patient

Humidification, suctioning, cuff care, VAP prevention, positioning and nutrition

Humidification

Device	How it works	Advantages	Disadvantages / contraindications
Heat and moisture exchanger (HME / HMEF)	Captures exhaled heat and moisture and returns it on inspiration; filter version blocks microbes	Cheap, simple, filters, no condensation	Adds dead space (50–100 ml) and resistance; avoid in thick/bloody secretions, large leaks (BPF), hypothermia, minute ventilation >10 L/min, small VT (ARDS — dead space), during nebulisation
Heated humidifier (active)	Heated water chamber + heated-wire circuit; delivers 37 °C, 44 mg/L	Best for long-term ventilation, thick secretions, ARDS, children	Condensation, cost, burns/over-heating if misused

KEY POINT Change HMEs every 24–48 h or when soiled; never use HME and heated humidifier together. During aerosol therapy, remove the HME and place the nebuliser on the inspiratory limb (aerosol would otherwise be trapped in the HME).

Endotracheal suction

Point	Recommendation
Indication	Only when needed — visible/audible secretions, saw-tooth flow waveform, rising peak pressure, desaturation (not routinely by the clock)
Catheter size	≤50% of the internal diameter of the tube (adults ≤70%); French size ≈ (ETT ID in mm × 3) / 2 — e.g. 8.0 ETT → 12 Fr
Depth	Shallow (to the tip of the tube + 1 cm) preferred over deep suction
Pressure	−80 to −150 mmHg (adults); −80 to −100 (children)
Duration	≤10–15 seconds per pass
Pre-oxygenation	FiO_2 1.0 (or +0.2) for 30–60 s before and after
Closed suction system	Preferred in high PEEP/FiO_2, ARDS, infectious disease (prevents derecruitment and aerosol)
Saline instillation	Not routine
Complications	Hypoxaemia, derecruitment, bradycardia (vagal), bronchospasm, mucosal trauma, raised ICP

Endotracheal tube and cuff care

- Cuff pressure **20–30 cmH_2O** measured with a manometer every 8 hours (and after repositioning) — below 20 risks micro-aspiration; above 30 causes mucosal ischaemia and tracheal stenosis.
- Record tube size and depth at teeth/lips every shift; secure without pressure on lips and corners of the mouth; move tube side daily.
- Subglottic secretion drainage tubes (SSD) reduce VAP when ventilation >48–72 h is expected.

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Ventilator-associated pneumonia (VAP) — prevention bundle

Measure	Detail
Head of bed elevation	30–45° unless contraindicated
Daily sedation interruption and SBT	Reduces ventilator days (most powerful measure)
Oral care	Toothbrushing 2–3 times a day; chlorhexidine 0.12% is no longer routinely recommended (possible mortality signal) — use per local policy
Subglottic secretion drainage	For expected ventilation >48–72 h
Cuff pressure 20–30 cmH_2O	Prevents micro-aspiration
Avoid unnecessary circuit changes	Change only if visibly soiled or malfunctioning
Early mobilisation and enteral nutrition	—
Stress ulcer prophylaxis only if indicated	PPIs may increase pneumonia risk
Hand hygiene, closed suction, avoid re-intubation	—

VAP diagnosis (clinical)

Criterion	Detail
Timing	Pneumonia developing >48 h after intubation
Clinical	New or progressive infiltrate + 2 of: fever >38 °C or <36 °C, leukocytosis/leukopenia, purulent secretions, worsening oxygenation
Microbiology	Endotracheal aspirate (semi-quantitative) or BAL (≥10^4 CFU/ml) before antibiotics
CPIS (Clinical Pulmonary Infection Score)	>6 suggests VAP (temperature, WBC, secretions, P/F, CXR, culture)
Common organisms in India	Acinetobacter baumannii, Klebsiella pneumoniae (often carbapenem-resistant), Pseudomonas aeruginosa, MRSA — guide therapy by local antibiogram

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Positioning

Position	Indication
Head-up 30–45°	All ventilated patients (VAP, aspiration, ICP)
Prone position	Moderate–severe ARDS (P/F <150) — 16–18 h/day (Chapter 16)
Lateral, "good lung down"	Unilateral lung disease (better V/Q); "bad lung down" in massive haemoptysis or lung abscess to protect the good lung
Reverse Trendelenburg	Obesity, abdominal distension — improves FRC
Regular turning (2-hourly)	Pressure injury prevention

Nutrition and other care

Element	Recommendation
Enteral nutrition	Start within 24–48 h if haemodynamically stable; trophic then build to 20–25 kcal/kg/day and protein 1.2–2.0 g/kg/day
Glucose	Target 140–180 mg/dl
Stress ulcer prophylaxis	If coagulopathy, shock, ventilation >48 h with other risk factors (pantoprazole 40 mg daily)
VTE prophylaxis	Enoxaparin 40 mg SC daily (or UFH) unless bleeding risk; mechanical devices
Eye care	Lubricants, taping/moisture chambers in sedated patients (exposure keratopathy)
Early mobility	Passive → active-assisted → sitting → standing; even on the ventilator when stable
Communication	Writing boards, picture charts, speaking valves (tracheostomy)
Bowel care	Laxatives with opioids; monitor gastric residuals only if intolerance

Nebulised and inhaled drugs on the ventilator

- Use a vibrating-mesh or jet nebuliser on the **inspiratory limb**, 15–30 cm from the Y-piece; remove the HME (use filter on expiratory limb).
- Metered-dose inhaler with spacer chamber in the inspiratory limb: 4–8 puffs of salbutamol, actuated at the start of inspiration.
- Turn off bias flow/heated humidifier only if recommended by the manufacturer; watch for expiratory filter blockage.

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CHAPTER 16

Acute Respiratory Distress Syndrome (ARDS)

Definition, lung-protective ventilation, PEEP, prone position and rescue therapies

Definition — Berlin (2012) and the 2023 global update

Criterion	Berlin definition	Global definition 2023 (additions)
Timing	Within 1 week of a known insult or new/worsening respiratory symptoms	Same
Imaging	Bilateral opacities on CXR/CT not fully explained by effusion, collapse or nodules	Also lung ultrasound (by trained operator)
Origin	Not fully explained by cardiac failure or fluid overload	Same
Oxygenation	PEEP ≥5 cmH_2O: mild P/F 201–300; moderate 101–200; severe ≤100	Includes non-intubated patients on HFNC ≥30 L/min or NIV/CPAP ≥5 (P/F ≤300 or S/F ≤315 with SpO_2 ≤97%); resource-limited settings: no PEEP/flow requirement

KEY POINT Common causes in India: pneumonia (bacterial, influenza, COVID-19), sepsis, scrub typhus, leptospirosis, malaria (falciparum/vivax), dengue, aspiration, pancreatitis, trauma, transfusion (TRALI), organophosphate and paraquat poisoning.

The lung-protective ventilation protocol (ARDSNet)

Step	Action
1. Calculate PBW	Use height (Chapter 6)
2. Mode	Volume assist-control
3. Initial VT	8 ml/kg PBW, reduce by 1 ml/kg every ≤2 h to **6 ml/kg**
4. Rate	To approximate baseline minute ventilation (not >35/min)
5. Plateau pressure	Check every 4 h and after each change in PEEP/VT: if >30 cmH_2O → reduce VT to 5 or 4 ml/kg; if <25 and VT <6 → raise VT by 1 ml/kg
6. Driving pressure	Aim ≤15 cmH_2O
7. Oxygenation goal	PaO_2 55–80 mmHg or SpO_2 88–95%
8. pH goal	7.30–7.45; if 7.15–7.30 increase RR to 35; if <7.15 increase RR to 35 and consider raising VT by 1 ml/kg (Pplat may exceed 30); bicarbonate if needed
9. I:E	1:1 to 1:3

ARDSNet PEEP/FiO_2 tables

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0
Lower PEEP (cmH_2O)	5	5	8	8	10	10	10	12	14	14	14	16	18	18–24

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FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5	0.5	0.5–0.8	0.8	0.9	1.0	1.0
Higher PEEP (cmH_2O)	5	8	10	12	14	14	16	16	18	20	22	22	22	24

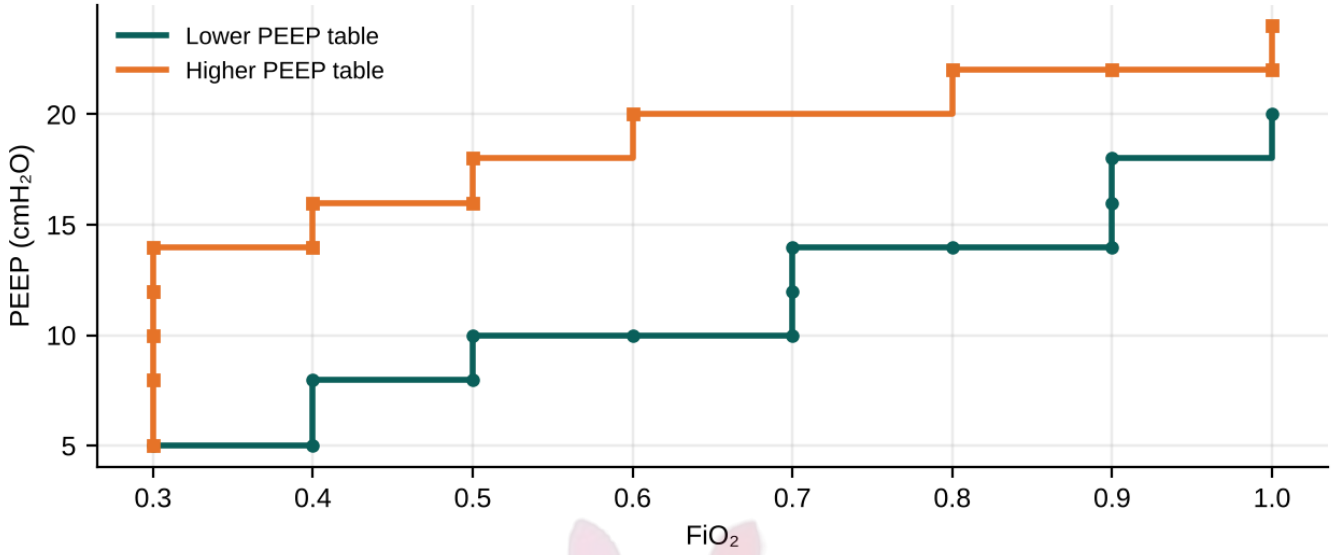


Figure 16.1. ARDSNet lower and higher PEEP/FiO_2 combinations.

KEY POINT Use the **higher PEEP table** in moderate–severe ARDS (P/F <200) if the lung is recruitable (driving pressure and compliance improve, Pplat stays ≤30, BP stable); the lower table in mild ARDS or non-recruitable lungs.

Other ways to set PEEP

Method	Principle	Comment
PEEP/FiO_2 table	Pragmatic, protocolised	Simple — first choice in most units
Best compliance (decremental PEEP trial)	After recruitment, reduce PEEP 2 cmH_2O every few minutes; choose PEEP with best compliance + 2	Needs stable, sedated patient
Driving pressure	Choose PEEP giving lowest driving pressure at the same VT	Simple bedside method
Recruitment-to-inflation ratio (R/I)	One-breath derecruitment from PEEP 15 to 5	R/I >0.5 → recruitable (higher PEEP)
Oesophageal pressure (transpulmonary pressure)	Set PEEP so end-expiratory PL is 0 to +2 cmH_2O	Useful in obesity, abdominal hypertension
Electrical impedance tomography (EIT)	Balance overdistension and collapse	Where available
Stress index	Linear pressure–time curve in VC	Concave-up = overdistension

Prone position ventilation

Item	Detail
Indication	Moderate–severe ARDS with **P/F <150 on FiO_2 ≥0.6 and PEEP ≥5** after 12–24 h of optimisation (PROSEVA)

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Item	Detail
Duration	**≥16 consecutive hours per day**; continue daily until P/F ≥150 with PEEP ≤10, FiO_2 ≤0.6, 4 h after supine
Benefit	Mortality 32.8% → 16% at 28 days (PROSEVA, 2013); more homogeneous ventilation, better V/Q, drainage of secretions, relief of heart compression
Contraindications	Unstable spine, open abdomen/chest, raised ICP, massive haemoptysis, unstable fractures (pelvis, face), pregnancy (relative), recent sternotomy, severe haemodynamic instability
Team	4–5 people; airway doctor at the head controls the turn; secure tube, lines and drains
Complications	Pressure ulcers (face, knees, chest), ET tube displacement/obstruction, facial oedema, ocular injury, brachial plexus injury, vomiting
Awake proning (non-intubated)	COVID-19 hypoxaemia on HFNC — reduced intubation if ≥8 h/day; less evidence in other conditions

Recruitment manoeuvres

- Sustained inflation (e.g. 30–40 cmH_2O for 30–40 s) or stepwise PEEP increases can open collapsed lung.
- The ART trial (2017) showed **higher mortality** with aggressive stepwise recruitment and titrated PEEP — routine aggressive recruitment is not recommended.
- Use brief, moderate recruitment only after derecruitment events (disconnection, suction) in recruitable patients, with haemodynamic monitoring.

Adjuncts in ARDS

Therapy	Recommendation
Conservative fluid strategy	Once shock resolved — neutral/negative balance (FACTT); improves ventilator-free days
Neuromuscular blockade	Short course (≤48 h) in severe ARDS with dyssynchrony despite deep sedation; not routine (ROSE)
Corticosteroids	Dexamethasone 6 mg daily × 10 days in COVID-19 ARDS; consider methylprednisolone 1 mg/kg/day or dexamethasone 20 mg × 5 days then 10 mg × 5 days (DEXA-ARDS) in early moderate–severe non-COVID ARDS (ESICM/SCCM 2024 conditional); hydrocortisone in severe community-acquired pneumonia
Inhaled nitric oxide / epoprostenol	Improves oxygenation temporarily; no mortality benefit — rescue only
High-frequency oscillation	Not recommended in adults (OSCILLATE)
ECMO (VV)	Severe ARDS despite optimal care (EOLIA criteria, Chapter 26)
Permissive hypercapnia	pH ≥7.20 accepted; avoid in raised ICP, severe PH, RV failure

Refractory hypoxaemia ladder

Step	Action
1	Confirm optimal basics: VT 6 ml/kg, PEEP per table, sedation, synchrony, suction, exclude pneumothorax/effusion/collapse
2	Higher PEEP (if recruitable), conservative fluids
3	Prone position ≥16 h

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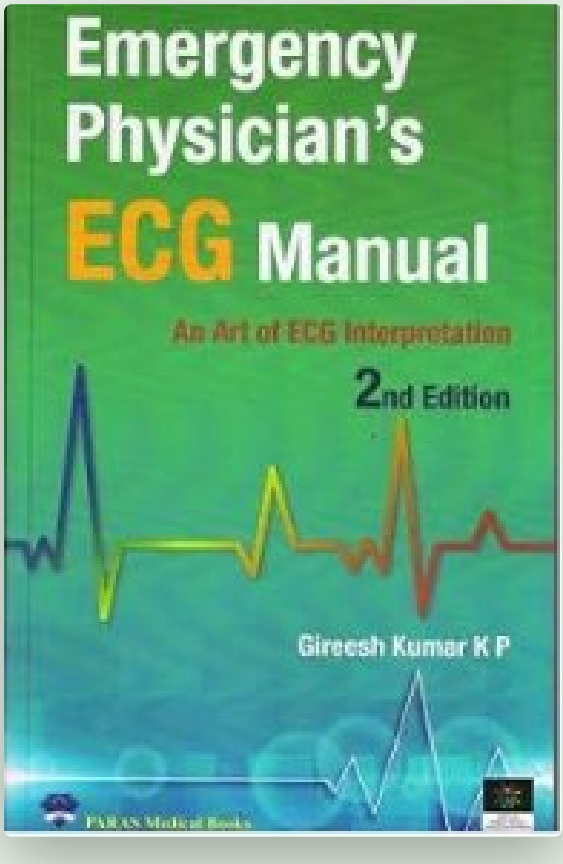
Step	Action
4	Neuromuscular blockade (short)
5	Inhaled pulmonary vasodilator (rescue)
6	Refer for VV-ECMO (P/F <50 for >3 h, or <80 for >6 h, or pH <7.25 with $PaCO_2$ ≥60 for >6 h despite RR 35 and Pplat ≤32)

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CHAPTER 17

Obstructive Lung Disease — Asthma and COPD

Dynamic hyperinflation, auto-PEEP and how to ventilate safely

The problem: dynamic hyperinflation

In severe asthma and COPD, narrowed airways empty slowly (long time constant). If the next breath starts before the lung has emptied, gas is trapped and end-expiratory lung volume rises breath by breath — **dynamic hyperinflation**. The trapped gas creates a positive alveolar pressure at end-expiration — **intrinsic or auto-PEEP**. It causes hypotension (reduced venous return), barotrauma, increased work of triggering and, in the extreme, pulseless electrical activity.

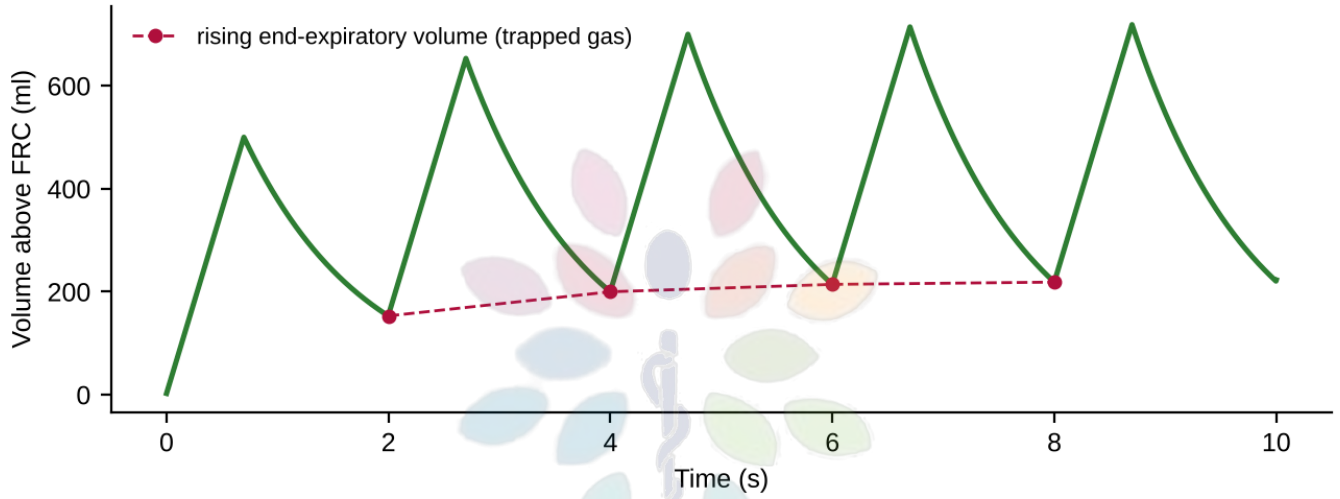


Figure 17.1. Breath stacking: each breath starts before full exhalation, so end-expiratory volume rises (dynamic hyperinflation).

Determinants of air trapping

Factor	Effect	What to do
Minute ventilation (rate × VT)	Main determinant — more gas in, more trapped	**Low rate (8–12/min)** and moderate VT (6–8 ml/kg)
Expiratory time	Shorter expiration traps gas	Long expiratory time: I:E 1:4–1:5
Inspiratory flow	Higher flow shortens inspiration → longer expiration	Peak flow 60–100 L/min, square wave
Airway resistance	Bronchospasm, secretions	Bronchodilators, steroids, suction
Tube size	Small tube adds resistance	Use ≥8.0 mm ETT in adults

Initial settings in severe asthma

Setting	Value
Mode	VC-AC
VT	6–8 ml/kg PBW
Rate	**8–10/min**

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Setting	Value
Inspiratory flow	80–100 L/min (square)
I:E	1:4 or longer (expiratory time ≥4 s)
PEEP	0–5 cmH_2O (set low; in asthma external PEEP does not help and may worsen hyperinflation)
FiO_2	To SpO_2 92–96%
Targets	Pplat <30 (ideally <25), auto-PEEP <10, accept $PaCO_2$ 60–90 with pH ≥7.15–7.20 (**permissive hypercapnia**)

Initial settings in COPD

Setting	Value
Mode	VC-AC initially; PSV once stable
VT	6–8 ml/kg PBW
Rate	10–14/min
Inspiratory flow	60–80 L/min
I:E	1:3–1:4
PEEP	Set external PEEP ≈ 70–80% of measured PEEPi (typically 5–8) — reduces triggering effort without worsening hyperinflation (the "waterfall" effect)
$PaCO_2$ target	Patient's **baseline** $PaCO_2$ (chronically high; HCO_3^- ≈ 30–35) — aim for normal pH, not normal $PaCO_2$ (over-ventilation causes post-hypercapnic alkalosis and weaning failure)
PS mode	ETS 40–60% (early cycling), short rise time

Measuring hyperinflation

Measurement	How	Danger level
Auto-PEEP	Expiratory hold in a passive patient	>10 cmH_2O
Plateau pressure	Inspiratory hold	>25–30 cmH_2O
Flow–time curve	Expiratory flow does not reach zero	Any persistent trapping
VEI (volume at end-inspiration)	Apnoea for 40–60 s, collect total exhaled volume	>20 ml/kg (high barotrauma risk)

Crisis: hypotension or arrest in a ventilated asthmatic

Step	Action
1	**Disconnect the ventilator** for 30–60 seconds and allow full exhalation (gentle manual chest compression)
2	Exclude tension pneumothorax (often bilateral) — ultrasound, decompress if suspected
3	Fluid bolus; vasopressor if needed
4	Reconnect with lower rate (6–8/min) and longer expiratory time

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Medical treatment while ventilated

Drug	Dose
Salbutamol	MDI 4–8 puffs via spacer every 20 min initially, or nebulised 2.5–5 mg (continuous 10–15 mg/h in severe asthma)
Ipratropium	0.5 mg nebulised every 6 h (first 24–48 h)
Corticosteroids	Hydrocortisone 100 mg IV 6-hourly or methylprednisolone 40–60 mg 6–12-hourly (asthma); prednisolone 40 mg/day × 5 days or IV equivalent (COPD)
Magnesium sulphate	2 g IV over 20 min (asthma)
Ketamine	1–2 mg/kg then 0.5–2 mg/kg/h — bronchodilator sedative
Adrenaline	0.3–0.5 mg IM, or infusion 0.05–0.5 µg/kg/min (refractory asthma)
Aminophylline	5 mg/kg loading (if not on theophylline) then 0.5 mg/kg/h — narrow therapeutic window; limited role
Inhalational anaesthetics (sevoflurane, isoflurane)	Refractory status asthmaticus (anaesthesia support)
Heliox	Helium–oxygen 70:30 or 80:20 reduces turbulent flow — where available
ECMO / $ECCO_2R$	Refractory hypercapnia with acidosis
Antibiotics	COPD exacerbation with purulent sputum or on ventilator (amoxicillin–clavulanate, doxycycline, macrolide, or per local resistance)

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CHAPTER 18

Ventilation in Special Situations

Brain injury, heart failure, acidosis, poisoning, neuromuscular disease, pregnancy, obesity, trauma and more

Traumatic brain injury and raised ICP

Target	Value	Comment
PaO_2	80–120 mmHg (SpO_2 ≥94%)	Avoid hypoxaemia (doubles mortality) and extreme hyperoxia
$PaCO_2$	**35–40 mmHg** (normocapnia)	Hyperventilation ($PaCO_2$ 30–35) only as a short bridge for signs of herniation — causes cerebral vasoconstriction and ischaemia
PEEP	5–10 cmH_2O usually safe	Effect on ICP minimal if MAP maintained; avoid very high PEEP in poor compliance brain
VT	6–8 ml/kg	Lung protection still applies; avoid permissive hypercapnia
Position	Head-up 30°, head midline, ETT tie not compressing jugular veins	—
Sedation	Propofol/fentanyl; avoid coughing during suction (lignocaine, bolus sedation)	—
Monitoring	$ETCO_2$ continuously, ABG	—

Cardiogenic pulmonary oedema and cardiogenic shock

- Positive pressure reduces LV preload and afterload — CPAP/NIV first in pulmonary oedema; when intubated, PEEP 8–10 helps.
- Intubation may precipitate collapse (loss of sympathetic tone, induction drugs) — use ketamine/etomidate in reduced doses, have vasopressor/inotrope running.
- Avoid hypoxaemia and high FiO_2 once stable; plan early weaning (weaning-induced pulmonary oedema is common — Chapter 21).

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Right ventricular failure, massive PE and pulmonary hypertension

Principle	Action
Avoid intubation if possible	Induction, PPV and hypotension can cause arrest — HFNC/NIV, thrombolysis first
If intubated	Low Pplat (<27), low-moderate PEEP, VT 6 ml/kg; avoid hypercapnia, acidosis and hypoxaemia (raise PVR)
Haemodynamic support	Noradrenaline to maintain RV perfusion; dobutamine/milrinone; inhaled NO or prostacyclin
Avoid fluid overload	RV dilation reduces LV filling (interdependence)

Severe metabolic acidosis (DKA, sepsis, salicylate, methanol, lactic acidosis)

KEY POINT The patient's own minute ventilation may be 20–30 L/min (Kussmaul). After intubation, a "normal" ventilator rate of 14/min lets $PaCO_2$ rise sharply → pH falls → cardiac arrest. **Avoid intubation when possible. If intubation is unavoidable, match the pre-intubation minute ventilation (RR 25–35, VT 8 ml/kg) and check ABG within 15–30 minutes.** In salicylate poisoning, plan haemodialysis.

Organophosphate poisoning (common in India)

Issue	Management
Respiratory failure	Bronchorrhoea, bronchospasm, muscle weakness, central depression, intermediate syndrome (24–96 h)
Atropine	Double dose every 5 min (start 1.8–3 mg) until atropinised (clear chest, HR >80, SBP >80, dry axillae), then infusion 10–20% of loading dose per hour
Pralidoxime	30 mg/kg bolus then 8 mg/kg/h (use per local protocol; evidence limited)
Ventilation	Frequent suction; often prolonged; watch for intermediate syndrome (neck flexion weakness, cranial nerve palsies) — intubate early
Avoid succinylcholine	Prolonged paralysis (cholinesterase inhibition) — use rocuronium

Neuromuscular disease — Guillain–Barré syndrome and myasthenia gravis

Intubate if (20/30/40 rule)	Value
Vital capacity	<20 ml/kg
Maximum inspiratory pressure (NIF)	Less negative than −30 cmH_2O
Maximum expiratory pressure	<40 cmH_2O
Other	Bulbar weakness with aspiration, rapid progression, autonomic instability, single-breath count <15

- Ventilate with normal-lung settings (VT 6–8 ml/kg, PEEP 5); avoid NIV in GBS with bulbar weakness; myasthenic crisis may respond to BiPAP if no hypercapnia/bulbar failure.
- Plan early tracheostomy if prolonged ventilation is expected (GBS often weeks).
- Snake bite (neurotoxic krait/cobra envenomation): ventilate until anti-snake venom and neostigmine (cobra) reverse paralysis — often 24–72 h; excellent outcome if ventilated in time.

Obesity

Problem	Solution
Reduced FRC, rapid desaturation, atelectasis	Ramped position, pre-oxygenate with NIV/HFNC, PEEP 10–15 (or set by oesophageal pressure)
VT by actual weight → volutrauma	VT by **PBW**
High Pplat due to chest wall weight	Pplat up to 30–35 may be acceptable if transpulmonary pressure is low — measure oesophageal pressure where possible
Positioning	Reverse Trendelenburg 30°
Extubation	Extubate to NIV/HFNC, sitting upright

Pregnancy

Change	Implication
Respiratory alkalosis is normal	Target $PaCO_2$ 30–32 mmHg; $PaCO_2$ 40 is relative hypercapnia (fetal acidosis)
Reduced FRC, increased O_2 demand	Rapid desaturation — pre-oxygenate well; difficult airway (oedema) — smaller tube (6.0–7.0)

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Change	Implication
Aortocaval compression	Left lateral tilt 15–30° after 20 weeks
Oxygenation target	SpO_2 ≥95%, PaO_2 ≥70 mmHg
Prone position	Possible with padding; consider delivery for maternal benefit in late pregnancy

Chest trauma and bronchopleural fistula

Situation	Approach
Flail chest / pulmonary contusion	NIV/CPAP with epidural or regional analgesia; intubate for failure; consider surgical rib fixation
Pneumothorax on ventilator	Always chest drain (positive pressure turns simple into tension)
Bronchopleural fistula	Lowest effective VT, PEEP, Ti and rate; PC mode; limit suction on drain; independent lung ventilation or endobronchial valves in severe leaks
Massive haemoptysis	Bleeding side down, single-lumen tube into the healthy main bronchus or double-lumen tube, bronchial artery embolisation

Burns and inhalation injury

- Early intubation for stridor, hoarseness, deep facial/neck burns, soot in the airway (oedema worsens over 24–48 h); use large tube (≥8.0).
- 100% O_2 for carbon monoxide; hydroxocobalamin for suspected cyanide.
- Nebulised heparin and N-acetylcysteine used in some centres for inhalation injury; lung-protective ventilation; frequent bronchoscopic toilet.

Post-cardiac arrest

- SpO_2 92–98%, $PaCO_2$ 35–45 mmHg (normocapnia), VT 6–8 ml/kg, avoid hyperoxia and hyperventilation; temperature control; early coronary angiography when indicated.

Children and neonates — see Chapter 23

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CHAPTER 19

Advanced Modes and Advanced Monitoring

APRV, PAV+, NAVA, ASV, oesophageal pressure, stress index, P0.1 and EIT

Airway pressure release ventilation (APRV)

APRV is a time-cycled, pressure-limited mode that holds a high pressure (P-high) for a long time (T-high) and briefly releases it to a low pressure (P-low) for a short time (T-low). The patient breathes spontaneously throughout. It maximises mean airway pressure and recruitment; CO_2 is removed during the releases and by spontaneous breathing.

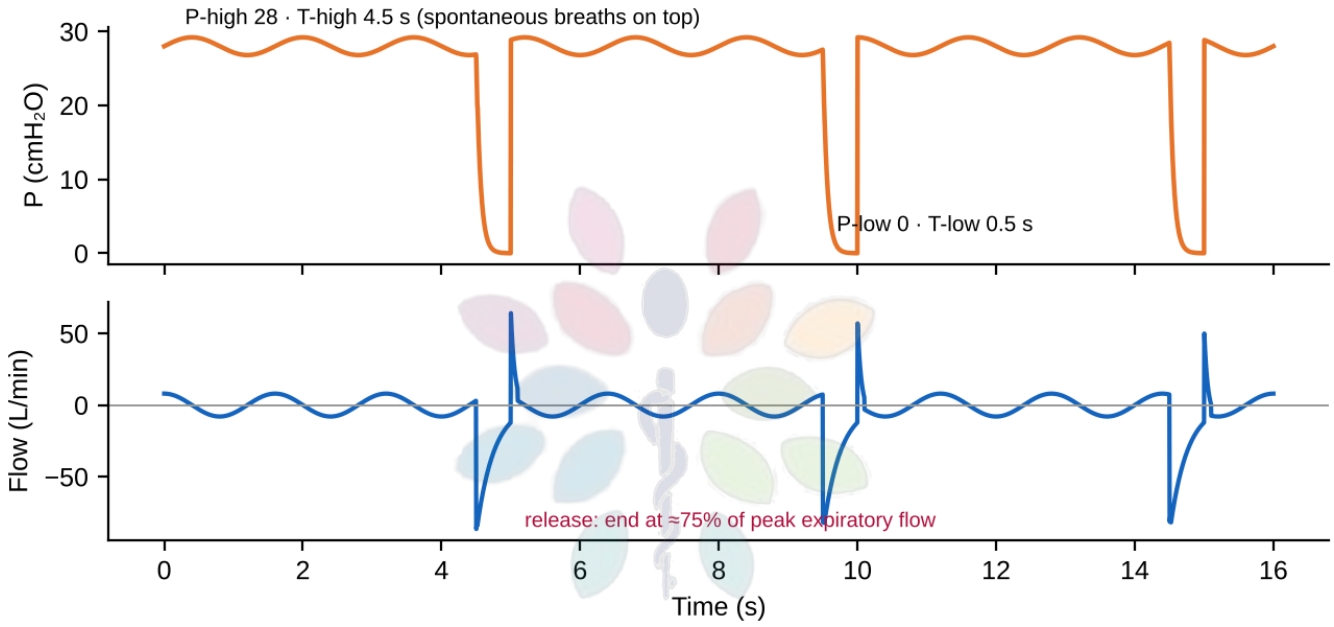


Figure 19.1. APRV pressure and flow: long P-high with brief releases; T-low is set so expiratory flow is terminated at about 75% of peak expiratory flow.

Setting	Typical starting value	Adjust
P-high	Plateau pressure on conventional ventilation (20–30 cmH_2O, ≤30)	↑ for oxygenation; ↓ in weaning ("drop and stretch")
P-low	0 cmH_2O (intrinsic PEEP maintained by short T-low)	—
T-high	4–6 s	↑ for oxygenation/weaning; ↓ to increase release frequency for CO_2
T-low	0.4–0.8 s (adults) — set so expiratory flow falls to 50–75% of peak (termination of PEFR)	Shorter if derecruitment; longer to remove CO_2
FiO_2	To SpO_2 target	—
Weaning	Reduce P-high by 2 cmH_2O and lengthen T-high progressively to CPAP	—

- **Advantages:** recruitment, preserved spontaneous breathing, less sedation, better haemodynamics in some patients.
- **Disadvantages:** large tidal volumes during release/spontaneous breaths (volutrauma), not suitable for obstructive disease, complex; no proven mortality benefit — use in experienced units as rescue.

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Proportional assist ventilation (PAV+)

- The ventilator measures resistance and elastance and gives pressure in proportion to the patient's effort ("gain" 5–95%). More effort → more support. Improves synchrony, allows natural variability; target Pmus 5–10 cmH_2O. Not for apnoeic patients or large leaks.

Neurally adjusted ventilatory assist (NAVA)

- A special nasogastric catheter detects the diaphragmatic electrical activity (Edi). The breath is triggered, sized and cycled by the Edi signal (NAVA level × Edi). Excellent synchrony; useful in neonates and in patients with severe asynchrony or leaks. Edi also monitors drive and over-/under-assistance.

Adaptive support ventilation (ASV) and closed loops

- The clinician sets %minute volume (100% = 0.1 L/kg/min in adults); the ventilator chooses the rate and VT pattern of least work (Otis equation) and adapts mandatory/spontaneous breaths. INTELLiVENT-ASV adds closed-loop control of PEEP and FiO_2 by SpO_2 and of ventilation by $ETCO_2$. Reduces manual adjustments and supports protocolised weaning.

Transpulmonary pressure and oesophageal manometry

Airway pressure acts on both the lung and the chest wall. The pressure that actually distends the lung is the **transpulmonary pressure (PL = Palv − Ppl)**. Oesophageal pressure (Pes), measured with a balloon catheter in the lower third of the oesophagus, estimates pleural pressure.

Measurement	Formula	Target
End-expiratory PL	PEEP total − Pes(end-exp)	0 to +2 cmH_2O (prevents collapse) — guides PEEP in obesity, abdominal hypertension
End-inspiratory PL (elastance-derived)	Pplat × (EL / ERS)	<20–25 cmH_2O (prevents overdistension)
Driving transpulmonary pressure	ΔPL	<10–12 cmH_2O
Inspiratory oesophageal swing (ΔPes)	Pes change during effort	5–10 cmH_2O normal effort; >15 = injurious effort (P-SILI)
Validation	Occlusion test: ΔPes/ΔPaw ≈ 0.8–1.2	—

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Measures of respiratory drive and effort (no catheter needed)

Measure	How	Interpretation
P0.1	Automated on many ventilators: pressure drop in the first 100 ms of an occluded effort	<1 low drive/over-assistance; 1.5–3.5 normal; >3.5–4 high drive (risk of injurious effort, weaning failure)
Pocc (ΔPocc)	Pressure fall during an end-expiratory occlusion with an effort	Pmus ≈ −0.75 × ΔPocc; Pmus >10–15 cmH_2O or predicted ΔPL,dyn >20 indicates excessive effort
Pmi (pressure muscle index)	Plateau − peak in PS with inspiratory hold	>6 cmH_2O suggests high effort
Diaphragm ultrasound	Thickening fraction	<15% weak/over-assisted; 15–40% normal effort; >40% high effort

Stress index (SI)

- In constant-flow volume control in a passive patient, the shape of the pressure–time curve during inspiration is fitted to $P = a \cdot t^b + c$. b = 1 (linear) ideal; b <1 (downward concavity) = tidal recruitment (consider more PEEP); b >1 (upward concavity) = overdistension (reduce VT/PEEP).

Electrical impedance tomography (EIT)

- A belt of 16–32 electrodes around the chest shows real-time regional ventilation. Used to titrate PEEP (balance collapse and overdistension), detect pneumothorax and assess recruitment and pendelluft. Available in tertiary centres.

Volumetric capnography and dead space

- Plotting CO_2 against exhaled volume gives phase I–III; the slope and area separate anatomical and alveolar dead space. Increasing dead space fraction (VD/VT >0.6) predicts mortality in ARDS and indicates overdistension when PEEP is raised.

High-frequency ventilation

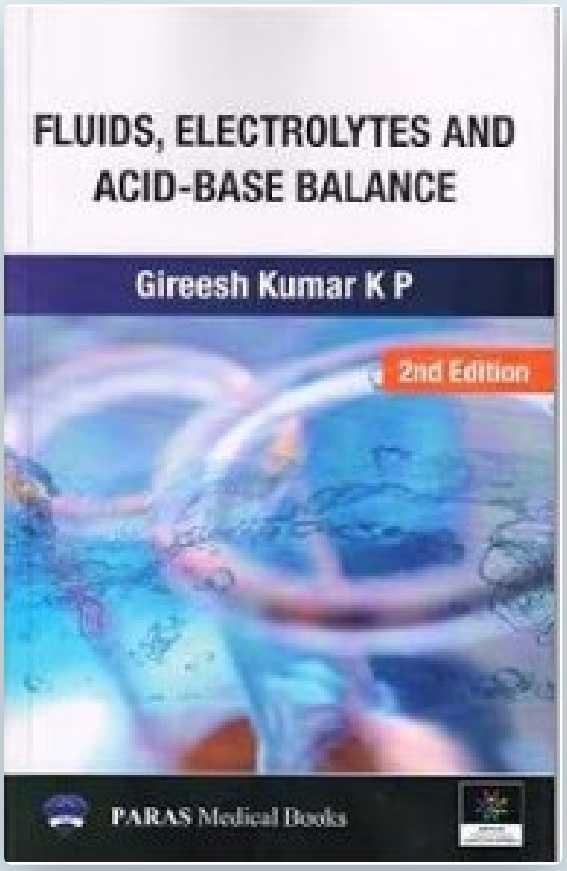
Type	Principle	Use
HFOV	Oscillation 3–15 Hz with active expiration around a constant mean pressure	Neonatal RDS, air-leak syndromes; adults not recommended (OSCILLATE, OSCAR)
HFJV (jet)	High-velocity jets at 100–600/min	Airway surgery, bronchopleural fistula, neonates
HFPPV / high-frequency percussive (VDR)	Percussive breaths superimposed on pressure control	Burns/inhalation injury in some centres

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CHAPTER 20

Heart–Lung Interactions

How positive pressure changes the circulation — and how to use it to assess fluid responsiveness

Effects of positive-pressure ventilation on the heart

Structure	Effect of positive pressure / PEEP	Clinical consequence
Venous return (RV preload)	↓ — raised right atrial pressure reduces the gradient for venous return	Hypotension in hypovolaemia; worse with high PEEP and auto-PEEP
RV afterload	↑ when lung volume rises above FRC (compresses alveolar vessels) or with hypoxic/hypercapnic vasoconstriction	Acute cor pulmonale in ARDS (20–25%) — limit driving pressure and $PaCO_2$, prone
LV preload	↓ (series effect of reduced RV output; septal shift)	—
LV afterload	↓ — positive intrathoracic pressure reduces LV transmural pressure	Benefit in LV failure/pulmonary oedema; weaning increases LV afterload → weaning-induced pulmonary oedema
Oxygen consumption	↓ when respiratory muscles are rested (up to 20–30% of total VO_2 in distress)	Benefit in shock

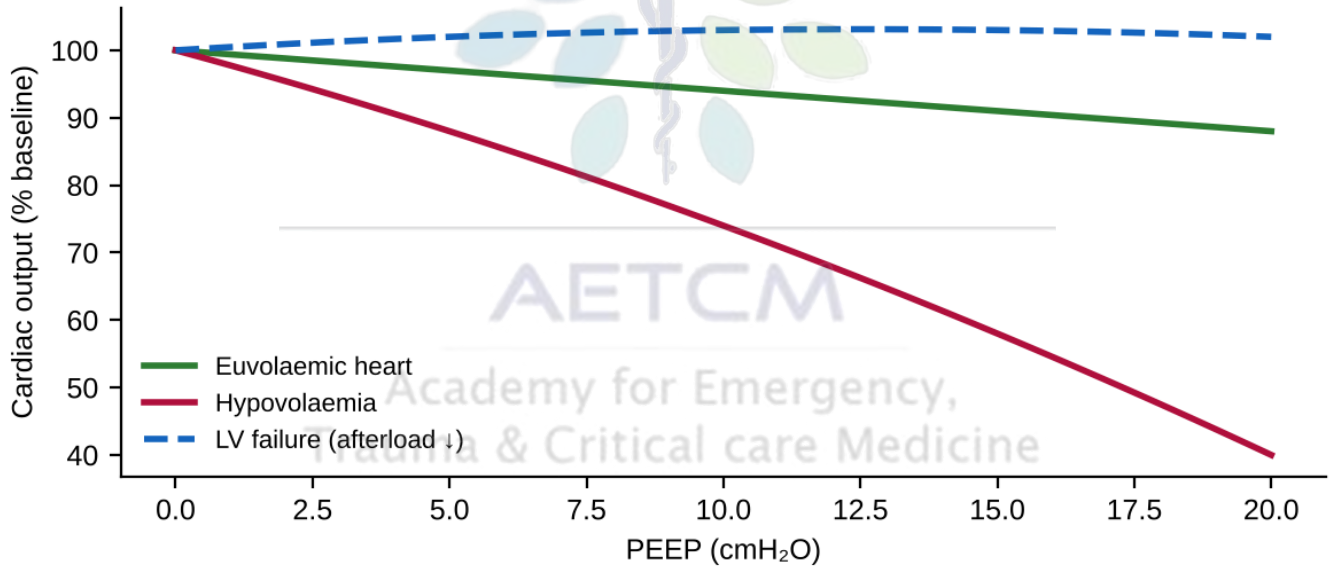


Figure 20.1. Effect of increasing PEEP on venous return/cardiac output in hypovolaemia versus euvolaemia (schematic).

Dynamic indices of fluid responsiveness

During a mechanical breath, stroke volume swings: inspiration transiently increases LV output and then, after the pulmonary transit time, reduces it. The bigger the swing, the more the heart is on the steep part of the Frank–Starling curve — that is, **fluid responsive**.

Index	Formula	Threshold for fluid responsiveness
Pulse pressure variation (PPV)	(PPmax − PPmin) / mean PP × 100	**>12–13%** (grey zone 9–13%)

Index	Formula	Threshold for fluid responsiveness
Stroke volume variation (SVV)	(SVmax − SVmin) / mean SV × 100	>10–12%
IVC distensibility (ventilated)	(Dmax − Dmin)/Dmin × 100	>18%
SVC collapsibility (TOE)	—	>36%
Passive leg raise	↑ cardiac output/VTI by >10% (real-time measure)	Works in spontaneous breathing and arrhythmia
End-expiratory occlusion test	Hold expiration 15 s; ↑ CO/pulse contour by >5%	Works with low VT
Tidal volume challenge	Increase VT from 6 to 8 ml/kg for 1 min; ↑ PPV by ≥3.5%	Validates PPV at low VT

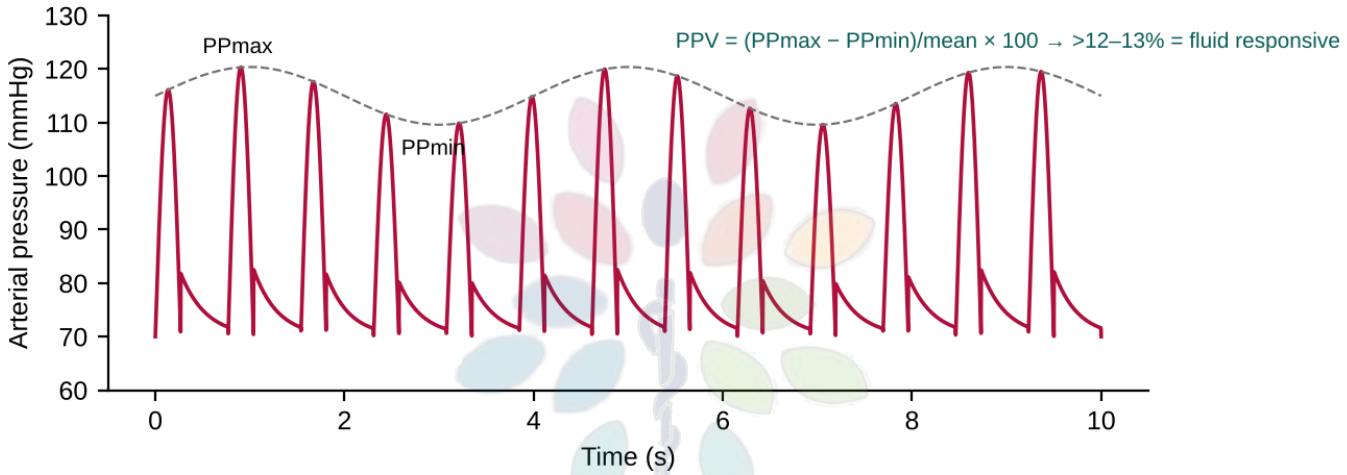


Figure 20.2. Pulse pressure variation on the arterial line during mechanical breaths: large swings indicate fluid responsiveness.

When PPV/SVV are unreliable

Limitation	Reason
Spontaneous breathing efforts	Irregular intrathoracic pressure
Arrhythmias (AF, frequent ectopics)	Beat-to-beat variation unrelated to breathing
Low tidal volume (<8 ml/kg) or low compliance (Crs <30)	Small pleural pressure change — false negative (use TV challenge)
Open chest	No pleural pressure change
Raised intra-abdominal pressure, RV failure	False positive
Very high RR (HR/RR <3.6)	Timing problem

Weaning-induced pulmonary oedema (WIPO)

- Switching from positive pressure to spontaneous breathing increases venous return and LV afterload and raises oxygen demand. In patients with LV dysfunction, valvular disease or fluid overload this precipitates pulmonary oedema during the SBT.
- Suspect when the SBT fails with rising BP, tachycardia, desaturation and B-lines; confirm with rise in BNP/plasma protein, E/e′ on echo, B-lines on lung ultrasound.

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- Treat with diuretics, nitrates, control of hypertension and ischaemia; extubate to NIV.

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CHAPTER 21

Weaning and Liberation from the Ventilator

Readiness screening, spontaneous breathing trials, extubation and post-extubation support

Think about weaning from the day of intubation

About 40% of the time on a ventilator is spent weaning. Delay causes VAP, muscle weakness and delirium; premature extubation causes re-intubation, which carries a high mortality. The key is a **daily, protocolised screen** followed by a **spontaneous breathing trial (SBT)** in every patient who passes.

Weaning category (WIND / ICC)	Definition	Outcome
Simple	Extubated after the first SBT	≈ 60–70% of patients; mortality ≈ 5–10%
Difficult	Needs up to 3 SBTs or up to 7 days after the first SBT	Mortality higher
Prolonged	>3 SBT failures or >7 days of weaning after the first SBT	Mortality ≈ 30%; tracheostomy, weaning units

Daily readiness screen

Domain	Criteria
Cause	Underlying cause improving or resolved
Oxygenation	SpO_2 ≥90% on **FiO_2 ≤0.4–0.5 and PEEP ≤5–8**; P/F ≥150–200
Haemodynamics	Stable, no or low-dose vasopressor (e.g. noradrenaline <0.1 µg/kg/min), no active ischaemia
Neurology	Arousable (RASS −1 to +1), follows commands (desirable), adequate cough
Respiratory	RR <35, spontaneous effort present, manageable secretions
Metabolic	pH ≥7.30, correctable electrolytes (K, Mg, PO_4), Hb ≥7–8 g/dl, temperature <38.5 °C

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Weaning predictors

Index	How	Threshold suggesting success
Rapid shallow breathing index (RSBI, f/VT)	RR ÷ VT (in litres) during 1 min of spontaneous breathing (CPAP 0–5, no PS)	**<105** breaths/min/L (lower is better; <80 good)
Maximum inspiratory pressure (NIF)	Occlude airway for 20 s	More negative than −20 to −30 cmH_2O
Vital capacity	Maximal inspiration then exhalation	>10–15 ml/kg
Minute ventilation	—	<10 L/min
P0.1	—	<4 cmH_2O
Diaphragm thickening fraction / excursion	Ultrasound	>30% / >1–1.5 cm
Cough peak flow	Spirometer at ETT	>60 L/min
Cuff leak test	Leak volume after cuff deflation (VC mode)	>110 ml or >12–24% of VT

KEY POINT Predictors only support clinical judgement — the **SBT itself** is the best test.

Spontaneous breathing trial

Item	Detail
Method	**Pressure support 5–8 cmH_2O + PEEP 5** (more likely to succeed, Subirà 2019), or T-piece, or CPAP 5 / ATC
Duration	30 minutes (most patients) to 120 minutes (high-risk, previous failure)
FiO_2	Same as before the trial (or ≤0.5)
Pair with	Sedation interruption earlier the same morning
Monitor	RR, SpO_2, HR, BP, mental status, work of breathing; RSBI at 2–5 minutes

SBT failure criteria

Objective	Subjective
RR >35/min for >5 min	Agitation, anxiety, diaphoresis
SpO_2 <90% (or <88% COPD)	Decreased consciousness
HR >140 or change >20%	Accessory muscle use, paradoxical breathing
SBP >180 or <90 mmHg	Dyspnoea
Arrhythmia; pH <7.32 or ↑ $PaCO_2$ >10 mmHg (if measured)	—

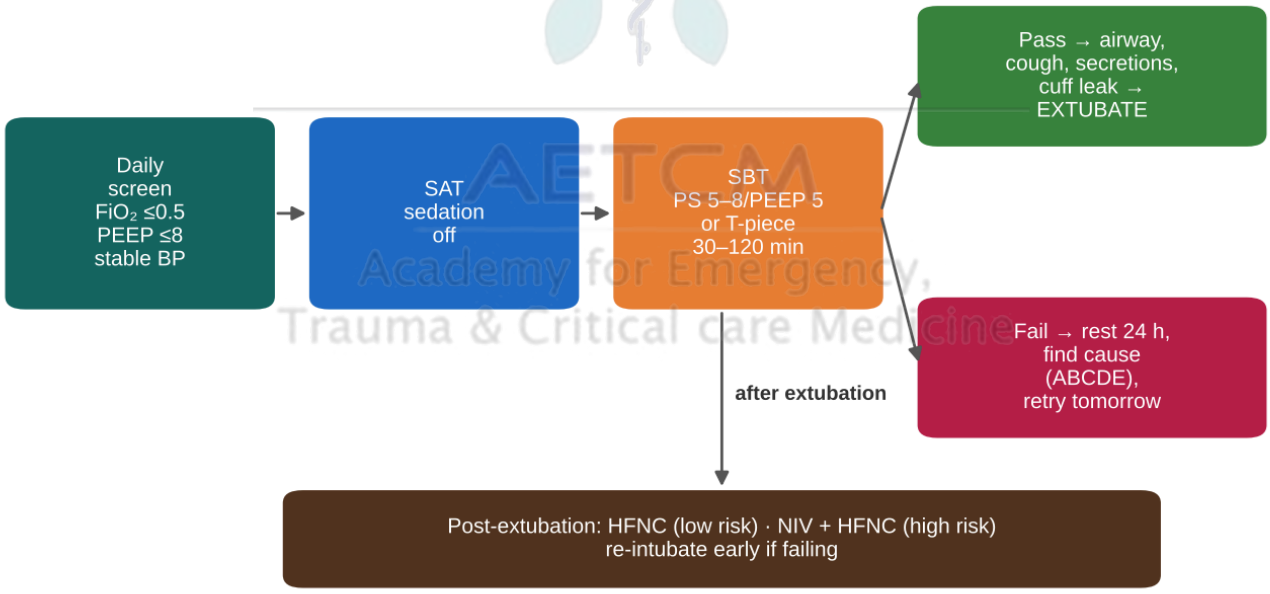


Figure 21.1. Weaning flow: daily screen → SAT → SBT → extubation readiness → extubation (with post-extubation support in high risk).

After a failed SBT

Cause (think "ABCDE")	Check / treat
Airway / lung	Secretions, bronchospasm, unresolved pneumonia, effusion, auto-PEEP

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Cause (think "ABCDE")	Check / treat
Brain	Delirium, sedation, anxiety, neurological weakness
Cardiac	Weaning-induced pulmonary oedema, ischaemia, arrhythmia — diuretics, nitrates
Diaphragm and muscles	ICU-acquired weakness, critical illness polyneuropathy, phrenic palsy, electrolyte deficiency (K, PO_4, Mg), hypothyroidism, steroids
Endocrine / metabolic / nutrition	Metabolic alkalosis, over-feeding, anaemia

- Return to a **rested, comfortable mode** (e.g. PS adjusted for RR <25) for 24 hours, correct the cause and repeat the SBT next day. Avoid daily "gradual reduction" schemes such as SIMV weaning.

Ready to extubate? Beyond the SBT

Question	Assessment
Can the patient protect the airway?	GCS ≥8–10, follows commands, cough strength (white card test, cough peak flow), gag/swallow
Secretions manageable?	Suction frequency ≤2-hourly, not copious
Upper airway patent?	Cuff leak test in high-risk (prolonged intubation, traumatic/difficult intubation, women, large tube): if absent → methylprednisolone 20 mg IV 4-hourly × 4 doses (or 40 mg) starting 12–24 h before extubation
Difficult airway plan?	Experienced operator present; airway exchange catheter in known difficult airway

Extubation procedure

Step	Action
1	Explain; sit up 45–90°; fasting for 4–6 h (feeds stopped)
2	Pre-oxygenate (FiO_2 1.0 for a few minutes); suction ETT and oropharynx/subglottic port
3	Deflate cuff; remove tube at peak inspiration (or with positive pressure) to expel secretions
4	Apply oxygen (or HFNC/NIV per risk); encourage cough
5	Monitor closely for 1–2 hours; ABG if concern; watch for stridor

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Post-extubation support

Risk group	Support
Low risk	Conventional oxygen or HFNC (HFNC reduces re-intubation vs conventional O_2 — Hernández 2016)
High risk: age >65, heart failure, COPD/hypercapnia, BMI >30, weak cough, prolonged ventilation, APACHE II >12, failed SBTs, airway problems	**Prophylactic NIV alternating with HFNC** immediately after extubation (HIGH-WEAN 2019)
Hypercapnic during SBT	NIV immediately after extubation
Post-extubation stridor	Nebulised adrenaline 1 mg (1:1000) in 5 ml, IV dexamethasone; heliox; re-intubate if failing

KEY POINT **Do not use NIV to rescue established post-extubation respiratory failure** in unselected patients — it delays re-intubation and increases mortality (Esteban 2004). Re-intubate early. Extubation failure = re-intubation within 48–72 hours (≈ 10–20%).

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CHAPTER 22

Tracheostomy and Prolonged Ventilation

Timing, techniques, tube types, care, decannulation and home ventilation

When and why

Point	Evidence / practice
Indications	Prolonged ventilation (>10–14 days expected), failed weaning, inability to protect the airway (neurological, bulbar palsy), copious secretions, upper airway obstruction, head and neck surgery
Timing	Early (≤7–10 days) vs late: no mortality difference (TracMan 2013) but less sedation and earlier mobilisation; individualise — earlier in severe brain injury, high cervical spinal injury, GBS
Benefits	Comfort, less sedation, easier mouth care, lower dead space and resistance, speech, faster weaning, transfer out of ICU
Techniques	Percutaneous dilatational tracheostomy (PDT) at bedside with bronchoscopic/ultrasound guidance; surgical tracheostomy (difficult anatomy, coagulopathy, obesity, unstable neck)

Tracheostomy tubes

Type	Feature	Use
Cuffed	Seal for ventilation and aspiration protection	Ventilated and early patients
Uncuffed	No seal	Long-term, weaned patients with good swallow
Fenestrated	Hole on the outer curve allows airflow to the larynx when inner cannula removed	Speech, weaning — use non-fenestrated inner cannula for suction/ventilation
Double-lumen (inner cannula)	Inner cannula can be removed and cleaned	Reduces blockage — standard on wards
Adjustable flange / extra-long	Obesity, deep neck	—
Subglottic suction port	Drains secretions above cuff	Reduces VAP

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Daily tracheostomy care

- Humidification (HME "Swedish nose" or heated); suction as needed; inner cannula cleaned 8-hourly.
- Cuff pressure 20–30 cmH_2O; stoma care and dressing daily; tie tension (one finger).
- **Bedside emergency kit:** spare tubes (same size and one size smaller), tracheal dilator, suction, obturator, syringe, BVM, oxygen; bed-head sign showing tube type, size and whether the upper airway is patent.

Tracheostomy emergencies (NTSP algorithm, simplified)

Step	Action
1	Call for help; apply oxygen to **both face and tracheostomy**
2	Remove speaking valve/cap and inner cannula
3	Pass a suction catheter — if it passes, the tube is patent: ventilate/suction

Step	Action
4	If it does not pass, deflate the cuff — the patient may breathe around the tube
5	Still not improving: **remove the tracheostomy tube**
6	Oxygenate from above (face mask/BVM with stoma covered) or via the stoma (paediatric mask/LMA on stoma)
7	Re-insert (if stoma >7 days old) or intubate orally (if upper airway patent); laryngectomy patients can only be ventilated through the stoma

Decannulation

Step	Criteria / action
Ready	Off ventilator ≥24–48 h, effective cough, manageable secretions (suction <2–4-hourly), safe swallow, patent upper airway
Cuff deflation trials	Deflate cuff for increasing periods; speaking valve (Passy-Muir) with cuff deflated
Downsize / cap	Cap the tube (cuff deflated) for 24–48 h; observe SpO_2, work of breathing
Remove	Occlusive dressing over stoma; usually closes in 5–7 days

Chronic critical illness and long-term ventilation

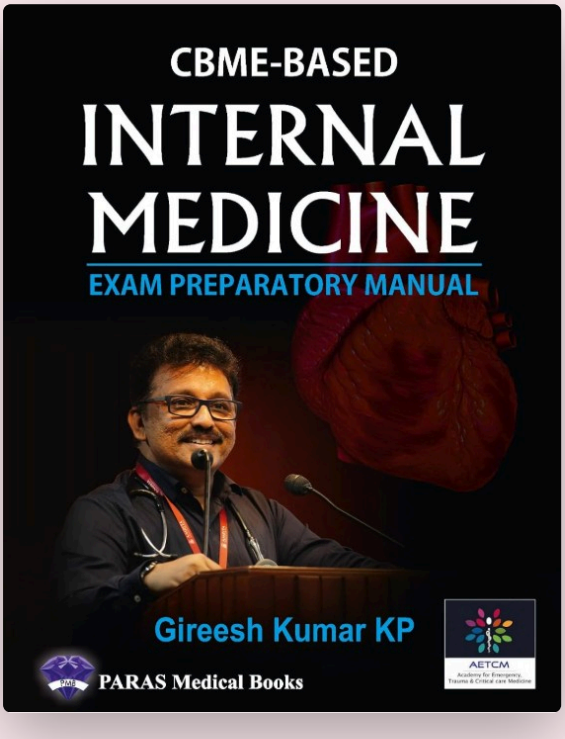
Issue	Approach
Weaning units	Multidisciplinary weaning; progressive daytime off-ventilator periods (tracheostomy mask trials)
Home ventilation	Neuromuscular disease, high spinal injury, OHS, COPD with persistent hypercapnia — portable ventilators, caregiver training, backup power
Rehabilitation	Physiotherapy, inspiratory muscle training, swallow therapy, nutrition
Palliative care	Goals-of-care discussions; compassionate extubation when appropriate

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CHAPTER 23

Paediatric and Neonatal Ventilation — Essentials

Differences from adults, settings, CPAP in neonates and common conditions

How children differ

Feature	Implication
Small airways, large tongue, high anterior larynx, narrowest point historically at cricoid	Higher resistance; cuffed tubes now preferred (cuff pressure ≤20 cmH_2O)
Compliant chest wall, horizontal ribs, diaphragm-dependent	Rapid fatigue; recession; FRC easily lost → need PEEP
High metabolic rate, O_2 consumption 6–8 ml/kg/min	Rapid desaturation during apnoea
Small tidal volumes	Circuit compliance and dead space matter — measure VT at the airway (proximal sensor)
Normal RR	Neonate 40–60; infant 30–40; child 20–30; adolescent 12–20

Tube size and depth

Age	Cuffed ETT ID (mm)	Depth at lips (cm)
Preterm <1 kg	2.5 (uncuffed)	6–7
Term neonate	3.0–3.5 (uncuffed) or 3.0 cuffed	9–10 (weight in kg + 6)
1 year	3.5 cuffed	11–12
>2 years	(Age/4) + 3.5 cuffed	(Age/2) + 12, or 3 × ETT size

Initial settings — children (PALS / PALICC-2)

Setting	Value
Mode	Pressure control (or PRVC) common; volume control acceptable
VT	5–8 ml/kg PBW (≤6 in PARDS with poor compliance)
Plateau / Pinsp	Pplat ≤28 cmH_2O (≤32 with stiff chest wall); driving pressure ≤15
PEEP	5 cmH_2O, higher in PARDS (PEEP/FiO_2 table)
Rate	Infant 25–30; child 20–25; adolescent 15–20
Ti	0.5–0.6 s (infant), 0.7–0.8 s (child), 1 s (adolescent)
SpO_2 target	92–97% (PARDS with PEEP ≥10: 88–92% acceptable)

Neonatal respiratory support

Support	Detail
Bubble CPAP	Simple, low-cost, widely used in Indian NICUs; nasal prongs/mask; CPAP 5–8 cmH_2O; flow 5–8 L/min; FiO_2 titrated to SpO_2 90–95%

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Support	Detail
Indications for CPAP	Respiratory distress syndrome (RDS) of prematurity, transient tachypnoea of the newborn, apnoea of prematurity, post-extubation
Surfactant	RDS in preterm on CPAP with FiO_2 >0.30 (or >0.40) — poractant 200 mg/kg initial (or beractant 100 mg/kg) via ETT or LISA/MIST technique
Caffeine citrate	20 mg/kg loading, then 5–10 mg/kg/day for apnoea of prematurity and to aid extubation
Conventional ventilation (neonate)	PIP 15–20, PEEP 5–6, Ti 0.3–0.4 s, rate 40–60, VT 4–6 ml/kg (volume-targeted preferred)
HFOV	MAP 2 above conventional, frequency 10–15 Hz, amplitude to chest wiggle — rescue for severe RDS, air leaks, PPHN
Inhaled nitric oxide	Persistent pulmonary hypertension of the newborn (term/near-term)
Oxygen target	Preterm 90–95% (avoid ROP); term 92–97%

Common paediatric conditions

Condition	Ventilatory approach
Bronchiolitis	HFNC 1–2 L/kg/min or CPAP; intubate for apnoea/exhaustion; long expiratory time if obstructive
Pneumonia / PARDS	Lung-protective, PEEP/FiO_2 tables, prone if severe
Status asthmaticus	Same principles as adults — low rate, long expiration, ketamine
Croup, upper airway obstruction	Dexamethasone 0.15–0.6 mg/kg, nebulised adrenaline; intubate with a tube 0.5 mm smaller
Scrub typhus / dengue with ARDS (India)	Lung-protective ventilation; careful fluid management in dengue capillary leak

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CHAPTER 24

Emergency Department, Transport and Resource-Limited Ventilation

Ventilating in the ED, during transfer and when resources are scarce

Ventilation in the emergency department

- Patients boarded in the ED on a ventilator are often ignored after intubation — the first hours matter. Apply the same standards: PBW-based VT, Pplat check, ABG within 30 min, sedation and analgesia, head-up, OG tube.
- ED-initiated lung-protective ventilation reduces ARDS and mortality (LOV-ED, 2017).
- Hand over: indication, tube size/depth, grade of view, settings, ABG, sedation, problems.

Transport ventilators and transfers

Item	Recommendation
Before leaving	Stabilise (airway secured, lines, chest drains, vasopressors on pumps), confirm tube position (CXR, $ETCO_2$), sedation adequate
Equipment	Transport ventilator with PEEP and alarms, waveform capnography, monitor, suction, BVM with PEEP valve, airway kit, drugs, spare batteries
Oxygen calculation	Required O_2 (L) = (MV × FiO_2 + ventilator driving gas consumption if pneumatic) × journey time × 2 (safety)
Example	MV 8 L/min, FiO_2 1.0, journey 60 min → 480 L; carry ≥960 L (one Indian B-type cylinder ≈ 1500 L)
During transfer	Continuous $ETCO_2$ and SpO_2; secure the tube before every move; **never clamp** a bubbling chest drain
Altitude/air transfer	Gas expansion — drain pneumothoraces, replace air in ETT cuff with saline or monitor cuff pressure

Bag-valve ventilation — doing it well

Point	Detail
Rate	10/min (one breath every 6 s) in adults
Volume	Just enough to see chest rise (≈ 400–500 ml; one-third to half of a 1.6 L bag)
PEEP valve	Add PEEP 5–10 cmH_2O in hypoxaemia
Avoid	Fast, forceful breaths — gastric insufflation, auto-PEEP, hypotension, raised ICP

Ventilation when resources are scarce

Challenge	Practical solution
Shortage of ventilators	Prioritise by triage protocol; use NIV/HFNC/awake proning early; bubble CPAP in children
No ABG	SpO_2/FiO_2 ratio, $ETCO_2$ trends, venous gas, clinical signs
No ICU-grade monitoring	Measure Pplat with inspiratory hold, use PBW tables, nurse-driven SpO_2 titration

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Challenge	Practical solution
Oxygen shortage	Titrate to lowest acceptable SpO_2, oxygen concentrators, minimise leaks, use reservoir systems efficiently, HFNC uses most oxygen
Power failure	UPS, manual bag rosters
Snake-bite and OP poisoning ventilation in district hospitals	Simple volume ventilators, or manual bag ventilation by trained staff working in rotation, have saved many lives when no ICU ventilator is available

Splitting ventilators?

KEY POINT Sharing one ventilator between two patients was attempted during COVID-19; it is **not recommended** except as an absolute last resort, because tidal volume, PEEP and alarms cannot be individualised and cross-infection risk is high.

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CHAPTER 25

Complications of Mechanical Ventilation

Ventilator-induced lung injury, barotrauma, infection, haemodynamics, muscles and the mind

Ventilator-induced lung injury (VILI)

Mechanism	Cause	Prevention
Volutrauma	Overdistension by large tidal volumes	VT 6 ml/kg PBW, Pplat ≤30, driving pressure ≤15
Barotrauma	High transpulmonary pressure → alveolar rupture: pneumothorax, pneumomediastinum, subcutaneous emphysema	Limit pressures, avoid auto-PEEP
Atelectrauma	Repeated opening and closing of alveoli (shear)	Adequate PEEP
Biotrauma	Release of inflammatory mediators → multiple organ failure	All of the above
Ergotrauma / mechanical power	Energy load from rate, VT, pressures, flow	Limit rate and pressures, not only VT
Oxygen toxicity	FiO_2 >0.6 for prolonged periods	Use PEEP to reduce FiO_2; target SpO_2 92–96%
P-SILI	Vigorous spontaneous efforts in injured lungs	Control drive (Chapter 13)

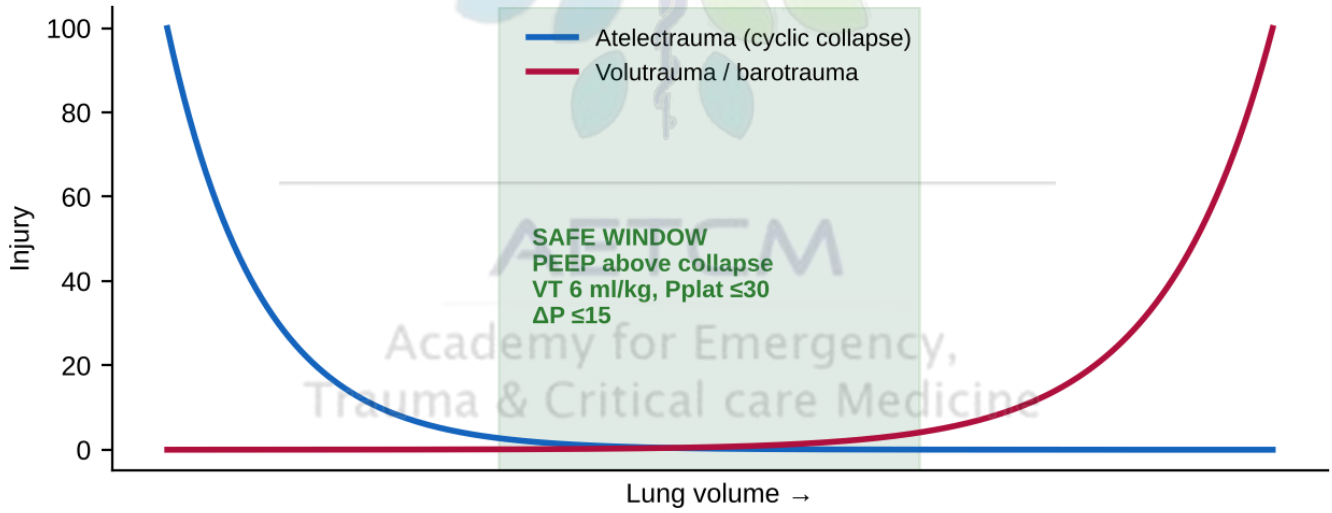


Figure 25.1. The "safe window" of ventilation: between collapse (atelectrauma) at low volumes and overdistension (volutrauma) at high volumes.

Pneumothorax on the ventilator

- Sudden rise in peak and plateau pressures, desaturation, hypotension, unilateral reduced air entry, tracheal deviation (late), subcutaneous emphysema.
- Ultrasound: absent lung sliding, absent B-lines, lung point.
- Treatment: immediate decompression (finger thoracostomy at 4th–5th intercostal space mid-axillary line, or needle at 2nd ICS MCL / 4–5th ICS AAL) followed by chest drain. **All pneumothoraces on positive pressure need a drain.**

Infectious complications

Complication	Detail
Ventilator-associated pneumonia (VAP)	5–15 per 1000 ventilator days; higher in India; MDR Gram-negatives (Chapter 15)
Ventilator-associated tracheobronchitis	Purulent secretions without new infiltrate
Sinusitis	Nasal tubes — use oral routes
Ventilator-associated events (VAE)	Surveillance definition: worsening oxygenation (↑ PEEP ≥3 or FiO_2 ≥0.2 for ≥2 days) after stability

Airway and tube complications

Complication	Prevention
Accidental extubation	Secure tube, adequate sedation, restraints only when essential, two-person turning
Tube blockage	Humidification, suction
Laryngeal injury, post-extubation stridor	Appropriate tube size, cuff pressure ≤30, avoid prolonged intubation
Tracheal stenosis, tracheomalacia, tracheo-oesophageal fistula	Cuff pressure monitoring
Pressure injuries of lips, tongue, nose	Reposition tube daily

Haemodynamic, renal and other organ effects

Organ	Effect
Cardiovascular	Reduced venous return and cardiac output; acute cor pulmonale
Kidney	Reduced renal perfusion, increased ADH and renin → fluid retention
Liver and gut	Reduced splanchnic flow; ileus; stress ulcers
Brain	Raised ICP with high PEEP/hypercapnia; delirium
Eyes	Exposure keratopathy, chemosis (prone)

Muscles and long-term outcomes

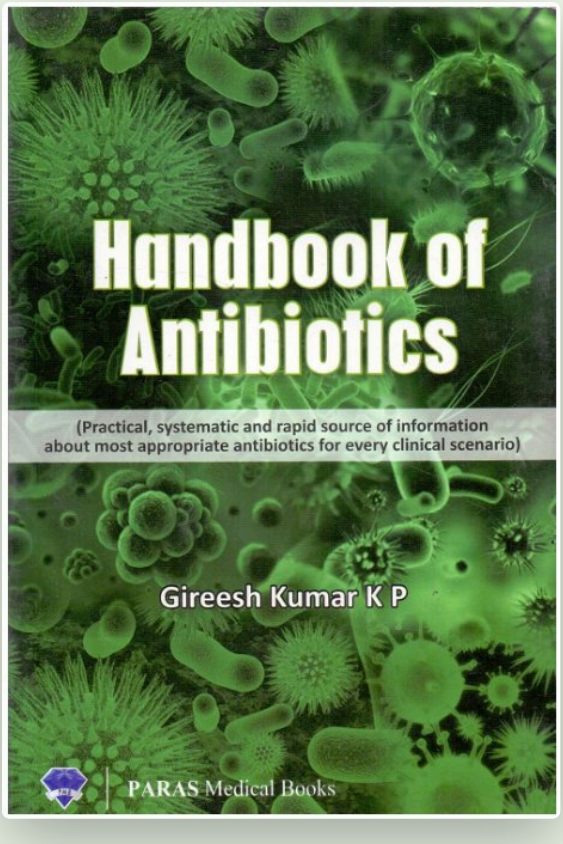
Problem	Detail
Ventilator-induced diaphragm dysfunction	Diaphragm atrophy within 18–24 h of controlled ventilation; avoid over-assistance, allow spontaneous breathing early
ICU-acquired weakness	Critical illness polyneuropathy and myopathy (sepsis, steroids, NMB, hyperglycaemia) — MRC sum score <48; early mobilisation
Post-intensive care syndrome (PICS)	Physical weakness, cognitive impairment, anxiety, depression, PTSD in survivors and families — ICU diaries, follow-up clinics
Dysphagia after extubation	Swallow assessment before oral feeding

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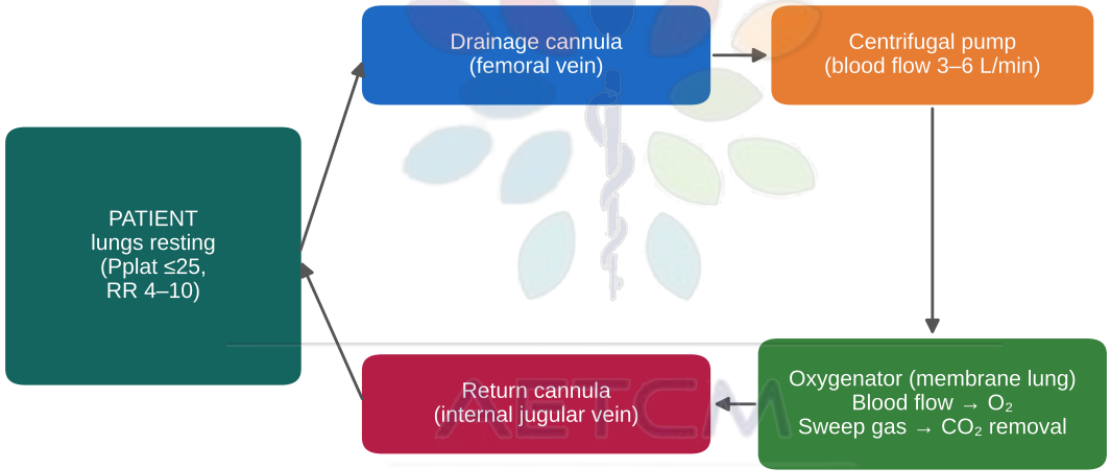
CHAPTER 26

ECMO and Extracorporeal Support

When the ventilator is not enough — VV-ECMO, VA-ECMO and $ECCO_2R$

Types of extracorporeal support

Type	Circuit	Supports	Main indications
Veno-venous (VV) ECMO	Blood drained from a large vein (femoral/IJ), oxygenated, returned to a vein	Lungs only (gas exchange)	Severe ARDS, refractory hypercapnia, bridge to lung transplant
Veno-arterial (VA) ECMO	Drained from vein, returned to artery (femoral/central)	Heart and lungs	Cardiogenic shock, refractory cardiac arrest (ECPR), massive PE, myocarditis, post-cardiotomy
$ECCO_2R$ (low-flow)	Small cannula, 0.4–1.5 L/min blood flow	CO_2 removal only	Hypercapnic COPD/asthma, ultra-protective ventilation (not proven — REST trial)



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Figure 26.1. VV-ECMO circuit: drainage cannula → pump → oxygenator (membrane lung, sweep gas) → return cannula.

VV-ECMO indications (EOLIA / ELSO)

Criterion	Despite optimal ventilation (FiO_2 ≥0.8, VT 6 ml/kg, PEEP ≥10, prone, NMB)
Severe hypoxaemia	P/F <50 mmHg for >3 h
Persistent hypoxaemia	P/F <80 mmHg for >6 h
Severe hypercapnic acidosis	pH <7.25 with $PaCO_2$ ≥60 mmHg for >6 h (RR increased to 35, Pplat ≤32)
Murray (lung injury) score	≥3 or uncompensated hypercapnia with pH <7.20

KEY POINT Refer early to an ECMO centre — outcomes are best when ECMO is started within 7 days of mechanical ventilation. Relative contraindications: ventilation >7–10 days with high pressures/FiO_2, irreversible disease without transplant option, severe brain injury, contraindication to anticoagulation, advanced age/frailty, malignancy with poor prognosis.

Ventilation on VV-ECMO — "lung rest"

Setting	Typical value
Mode	Pressure control
Plateau pressure	≤25 cmH_2O
Driving pressure	≤15 (often ≤10–12)
PEEP	10–15 cmH_2O
Rate	4–10/min
FiO_2	0.3–0.5 (oxygenation from ECMO)
Oxygenation on ECMO	Determined by blood flow (aim 60–80 ml/kg/min); SpO_2 85–92% acceptable
CO_2 removal on ECMO	Determined by sweep gas flow (start 1:1 with blood flow); lower $PaCO_2$ slowly (<20 mmHg in first 24 h — avoid cerebral injury)
Prone on ECMO	Feasible and may help (PRONECMO, mixed evidence)

Weaning ECMO

- Improve native lung (compliance, CXR), reduce sweep gas to zero ("off sweep trial") while on acceptable ventilator settings (FiO_2 ≤0.6, Pplat ≤30, RR ≤28); if gas exchange stable for 6–24 h, decannulate.

Complications

Complication	Detail
Bleeding	Cannulation sites, intracranial, GI — anticoagulation with heparin (anti-Xa/aPTT targets)
Thrombosis	Oxygenator, circuit, limb ischaemia (VA: distal perfusion cannula)
Haemolysis, thrombocytopenia, HIT	Monitor plasma free Hb, platelets
Infection	Cannula-related
VA-specific	LV distension (no ejection), differential hypoxaemia (Harlequin/North–South syndrome) — needs venting or VAV configuration

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ECMO in India

- Available in tertiary centres in major cities; transport ECMO services are growing. Early discussion with the regional ECMO centre (before the patient becomes too unstable for transfer) is essential.

CHAPTER 27

Case-Based Learning — 14 Ventilator Scenarios

From the first night on call to the expert ventilator round

Case 1 — COPD exacerbation on the ward

A 68-year-old man with COPD has RR 32, SpO_2 84% on 6 L/min mask. ABG: pH 7.26, $PaCO_2$ 72, PaO_2 50, HCO_3^- 31.

Question	Answer
Problem	Acute-on-chronic hypercapnic respiratory failure, worsened by uncontrolled oxygen
Oxygen	Change to Venturi 28% (white) — target SpO_2 88–92%
Support	BiPAP S/T, IPAP 12 → titrate to 18–20, EPAP 5, backup 14, FiO_2 to target
Recheck	ABG at 1 h: pH 7.31, $PaCO_2$ 62 — improving; continue; recheck at 4 h
Failure plan	If pH falls, drowsiness or intolerance → intubate (unless ceiling of care agreed)

Case 2 — Pneumonia on HFNC

A 45-year-old woman with pneumonia is on HFNC 60 L/min, FiO_2 0.7. SpO_2 91%, RR 34.

Question	Answer
ROX index	(91/0.7)/34 = 3.8
Interpretation at 6 h	Between 3.47 and 4.88 — indeterminate; reassess in 1–2 h; ROX <3.47 at 6 h → intubate
Other warning signs	Thoracoabdominal asynchrony, rising FiO_2 needs, altered sensorium
Prepare	Pre-oxygenate with HFNC/BiPAP; ketamine induction; lung-protective settings after intubation

Case 3 — Calculate the settings

A 172-cm man with ARDS is intubated. Set the initial ventilation.

Step	Calculation
PBW	50 + 0.91 × (172 − 152.4) = 67.8 kg
VT 6 ml/kg	≈ 400 ml
Rate	20–24/min (target MV ≈ 8–9 L/min)
PEEP/FiO_2	Start FiO_2 1.0, PEEP 10 → use the table as FiO_2 falls
Check	Pplat 28 → driving pressure 18 → too high: reduce VT to 5 ml/kg (340 ml), raise RR to 28

Case 4 — High pressure alarm

Ventilated patient: PIP rose from 25 to 45 cmH_2O; Pplat 22 (previously 20). SpO_2 89%.

Question	Answer
Gap	Peak − plateau = 23 → airway resistance problem
Causes	Secretions, mucus plug, bronchospasm, biting, kinked tube

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Question	Answer
Action	Suction (thick plug removed) → PIP 27; consider bronchodilator, humidification check

Case 5 — High peak and plateau

PIP 40, Pplat 37, sudden hypotension and absent breath sounds on the left.

Question	Answer
Diagnosis	Tension pneumothorax (compliance problem)
Action	Immediate decompression, chest drain; reduce VT/PEEP; check for ongoing air leak

Case 6 — Severe asthma, hypotension after intubation

A 22-year-old with severe asthma is intubated; ventilator set VT 500, RR 20. BP falls to 70/40.

Question	Answer
Cause	Dynamic hyperinflation (auto-PEEP) — expiratory flow does not return to zero; RR too high
Immediate action	Disconnect for 30–60 s, gentle chest compression; fluids; exclude pneumothorax
New settings	RR 10, VT 6–7 ml/kg, flow 80–100 L/min, I:E ≥1:4, PEEP 0–5; accept $PaCO_2$ 70 with pH 7.20

Case 7 — DKA patient becomes drowsy

A patient with DKA (pH 6.95, $PaCO_2$ 15, HCO_3^- 3) is intubated for drowsiness and set on RR 14, VT 450.

Question	Answer
Danger	Pre-intubation minute ventilation was ≈ 25 L/min; now 6.3 L/min → $PaCO_2$ will rise → pH may fall below 6.8 → arrest
Action	RR 28–30, VT 8 ml/kg; ABG at 15–30 min; target $PaCO_2$ near Winter's prediction (1.5 × 3 + 8 ≈ 12–14); continue insulin/fluids

Case 8 — Fighting the ventilator in VC mode

The pressure–time curve shows a scooped (concave) inspiratory pressure; RR 32; patient distressed. Set flow 40 L/min.

Question	Answer
Diagnosis	Flow starvation
Action	Increase flow to 60–80 L/min or switch to PC/PRVC; treat pain, fever, acidosis

Case 9 — Double triggering in ARDS

On VT 6 ml/kg, waveform shows two breaths delivered back-to-back with total volume ≈ 12 ml/kg.

Question	Answer
Problem	Double triggering — patient's neural inspiration longer than machine Ti; stacked volume defeats lung protection
Action	Increase Ti slightly, consider PC mode, increase sedation; short-term NMB in severe ARDS

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Case 10 — Severe ARDS refractory hypoxaemia

P/F 70 on FiO_2 1.0 and PEEP 14, VT 6 ml/kg, Pplat 30, pH 7.22.

Question	Answer
Next steps	Prone position ≥16 h; NMB 48 h; conservative fluids; check recruitability (R/I); consider higher PEEP table
If no improvement in 6 h	Contact ECMO centre (P/F <80 for >6 h meets EOLIA criteria)

Case 11 — The SBT

Day 6 of pneumonia; FiO_2 0.35, PEEP 5, RASS 0, noradrenaline stopped. SBT on PS 5/PEEP 5: after 30 min RR 24, VT 400 ml.

Question	Answer
RSBI	24 / 0.4 = 60 → favourable
Further checks	Cough strength, secretions, mental status, cuff leak
Plan	Extubate; post-extubation HFNC (or NIV + HFNC if high risk)

Case 12 — SBT failure with hypertension

During SBT: BP rises from 130/70 to 190/100, SpO_2 86%, new B-lines on ultrasound. History of ischaemic heart disease.

Question	Answer
Diagnosis	Weaning-induced pulmonary oedema
Plan	Return to support; diuretic, nitrates, control BP; repeat SBT next day; extubate to NIV

Case 13 — Organophosphate poisoning

A farmer with OP poisoning has copious secretions, pinpoint pupils and SpO_2 82%.

Question	Answer
Priorities	Atropine boluses to dry secretions; intubate (rocuronium, **not** succinylcholine); frequent suction; PEEP 5–8
Watch for	Intermediate syndrome on day 2–4 — neck flexor weakness, respiratory muscle weakness during weaning

Case 14 — Tracheostomy emergency

A patient with a 3-day-old percutaneous tracheostomy on the ward desaturates; no breath sounds, suction catheter will not pass.

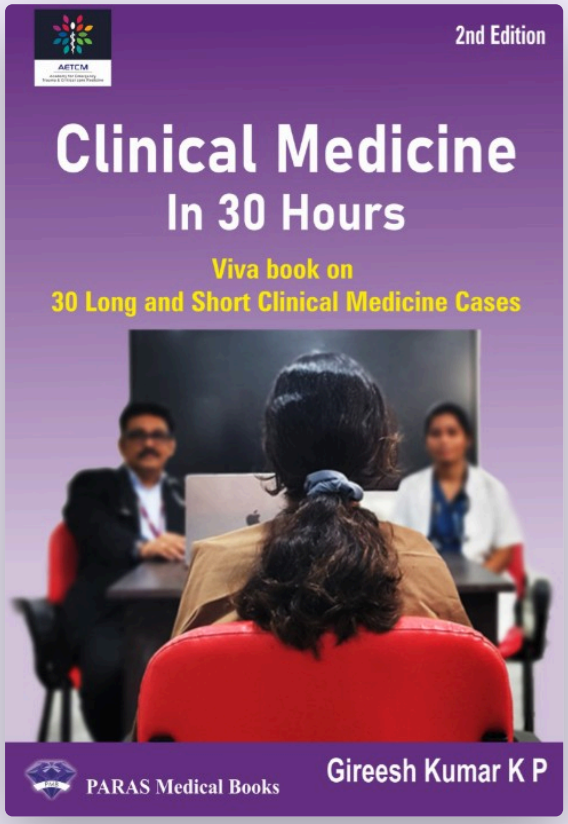
Question	Answer
Action	Call for help, O_2 to face and stoma, remove inner cannula, deflate cuff; if no improvement remove the tube
Then	Oxygenate from above (stoma covered) — stoma is fresh (<7 days), so **oral intubation** is preferred over blind re-insertion

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APPENDIX A

Formulas and Normal Values

Everything you need to calculate at the bedside

Respiratory formulas

Parameter	Formula	Normal / target
Predicted body weight (male)	50 + 0.91 × (height cm − 152.4)	—
Predicted body weight (female)	45.5 + 0.91 × (height cm − 152.4)	—
Minute ventilation	VT × RR	5–8 L/min (≈ 100 ml/kg/min)
Alveolar ventilation	(VT − VD) × RR	≈ 4–5 L/min
Alveolar gas equation	$PAO_2 = FiO_2$ (Patm − 47) − $PaCO_2/0.8$	≈ 100 mmHg on room air
A–a gradient	$PAO_2 - PaO_2$	(Age/4) + 4
P/F ratio	PaO_2 / FiO_2	>400
S/F ratio	SpO_2 / FiO_2	315 ≈ P/F 300
Oxygenation index	(FiO_2 × Pmean × 100) / PaO_2	<5 normal; >16 severe
Oxygen saturation index	(FiO_2 × Pmean × 100) / SpO_2	Paediatric ARDS (≥7.5 moderate, ≥12.3 severe)
Static compliance	VT / (Pplat − PEEPtotal)	50–100 ml/cmH_2O
Dynamic compliance	VT / (PIP − PEEP)	40–80 ml/cmH_2O
Airway resistance	(PIP − Pplat) / flow (L/s)	5–10 cmH_2O/L/s (intubated)
Driving pressure	Pplat − PEEP	≤15 cmH_2O
Time constant	R × C	0.2–0.5 s
Mechanical power (VC)	0.098 × RR × VT(L) × (PIP − ½ΔP)	<17 J/min
Dead space fraction (Bohr–Enghoff)	($PaCO_2$ − $PECO_2$) / $PaCO_2$	0.2–0.3
RSBI	RR / VT (L)	<105
ROX index	(SpO_2/FiO_2) / RR	≥4.88
Inspiratory time (VC, square flow)	VT (L) / flow (L/s)	e.g. 0.5 L / 1 L/s = 0.5 s
I:E ratio	Ti : (60/RR − Ti)	1:2
Oxygen content	(1.34 × Hb × SaO_2) + 0.003 × PaO_2	18–20 ml/dl
Oxygen delivery	CO × CaO_2 × 10	≈ 1000 ml/min
Winter's formula	Expected $PaCO_2$ = 1.5 × HCO_3^- + 8 ± 2	—
Anion gap	Na − (Cl + HCO_3)	8–12 mmol/l
Cylinder duration	Pressure (psi) × factor / flow	E factor 0.28

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Normal arterial blood gas (sea level, room air)

Variable	Normal range
pH	7.35–7.45
$PaCO_2$	35–45 mmHg (4.7–6.0 kPa)
PaO_2	80–100 mmHg (10.6–13.3 kPa)
HCO_3^-	22–26 mmol/l
Base excess	−2 to +2 mmol/l
SaO_2	95–100%
Lactate	<2 mmol/l

Unit conversions

From	To
1 kPa	7.5 mmHg
1 cmH_2O	0.74 mmHg (1 mmHg = 1.36 cmH_2O)
1 bar	100 kPa ≈ 14.5 psi ≈ 1020 cmH_2O
1 atmosphere	760 mmHg = 101.3 kPa

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APPENDIX B

Quick-Reference Cards

Pocket summaries for the bedside

Card 1 — Oxygen device selector

Need	Device	Flow	FiO_2
Mild hypoxaemia	Nasal cannula	1–6 L/min	24–44%
Moderate hypoxaemia	Simple face mask	5–10 L/min	40–60%
Severe hypoxaemia / emergency	Non-rebreather mask	15 L/min	60–90%
COPD / CO_2 retainer	Venturi mask	Per valve	24–60% fixed
Hypoxaemic failure	HFNC	40–60 L/min	21–100%
Hypercapnic acidosis	BiPAP	IPAP 10–25 / EPAP 4–8	As needed
Pulmonary oedema	CPAP	8–12 cmH_2O	As needed

Card 2 — First ventilator settings (adult)

Setting	Value
Mode	VC-AC
VT	6–8 ml/kg PBW (6 in ARDS)
RR	16–20 (8–12 asthma/COPD; 25–35 metabolic acidosis)
PEEP	5 (higher in hypoxaemia/obesity)
FiO_2	1.0 → titrate to SpO_2 92–96%
Flow	50–60 L/min (80–100 in obstruction)
Checks	Pplat ≤30, ΔP ≤15, auto-PEEP ≈ 0, ABG at 30 min

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Card 3 — Fix the gas

Problem	First moves
Low PaO_2	↑ FiO_2, ↑ PEEP, suction, recruit, prone, treat cause
High $PaCO_2$	↑ RR, ↑ VT (if Pplat allows), ↓ dead space, check auto-PEEP
Low $PaCO_2$	↓ RR/VT, treat pain/anxiety
High SpO_2	↓ FiO_2 first

Card 4 — Emergencies

Emergency	Action
Sudden desaturation	100% O_2 → disconnect & bag → suction → DOPES
High peak, normal plateau	Airway: suction, bronchodilator, check tube

Emergency	Action
High peak and plateau	Lung: pneumothorax, endobronchial tube, oedema, auto-PEEP
Hypotension	Auto-PEEP? (disconnect) Pneumothorax? Hypovolaemia? Sedation?
Accidental extubation	BVM → re-intubate or NIV/HFNC
Tracheostomy blocked	O_2 to face and stoma → inner cannula → suction → cuff down → remove tube

Card 5 — Liberation checklist

Step	Criteria
Screen	FiO_2 ≤0.5, PEEP ≤8, stable BP, awake, cough
SAT	Sedation off — calm and awake
SBT	PS 5–8/PEEP 5 or T-piece, 30–120 min
Pass	RR <35, SpO_2 ≥90%, HR/BP stable, no distress, RSBI <105
Extubate	Airway protection, secretions, cuff leak
Support	HFNC (low risk); NIV + HFNC (high risk)

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APPENDIX C

Abbreviations and Key Trials

Terms used in this book and the evidence behind the practice

Abbreviations

Abbreviation	Meaning	Abbreviation	Meaning
A/C (AC)	Assist-control	NAVA	Neurally adjusted ventilatory assist
APRV	Airway pressure release ventilation	NIF	Negative inspiratory force
ARDS	Acute respiratory distress syndrome	NIV	Non-invasive ventilation
ASV	Adaptive support ventilation	NMB	Neuromuscular blockade
BiPAP	Bilevel positive airway pressure	NRBM	Non-rebreather mask
CPAP	Continuous positive airway pressure	P/F	PaO_2/FiO_2 ratio
Crs / Cstat	Respiratory system / static compliance	PAV	Proportional assist ventilation
ECCO$_2$R	Extracorporeal CO_2 removal	PBW	Predicted body weight
ECMO	Extracorporeal membrane oxygenation	PC / PCV	Pressure control (ventilation)
EIT	Electrical impedance tomography	PEEP / PEEPi	Positive end-expiratory pressure / intrinsic PEEP
EPAP / IPAP	Expiratory / inspiratory positive airway pressure	PIP	Peak inspiratory pressure
$ETCO_2$	End-tidal carbon dioxide	Pplat	Plateau pressure
ETS	Expiratory trigger sensitivity (cycle %)	PRVC	Pressure-regulated volume control
FiO_2	Fraction of inspired oxygen	PS / PSV	Pressure support (ventilation)
FRC	Functional residual capacity	P-SILI	Patient self-inflicted lung injury
HFNC	High-flow nasal cannula	RASS	Richmond Agitation–Sedation Scale
HME	Heat and moisture exchanger	RSBI	Rapid shallow breathing index
I:E	Inspiratory to expiratory time ratio	SAT / SBT	Spontaneous awakening / breathing trial
IBW	Ideal body weight	SIMV	Synchronised intermittent mandatory ventilation
MV	Minute ventilation	VAP	Ventilator-associated pneumonia
ΔP	Driving pressure	VC / VCV	Volume control (ventilation)
Pes / PL	Oesophageal / transpulmonary pressure	VILI	Ventilator-induced lung injury
Ti / Te	Inspiratory / expiratory time	VT	Tidal volume

Landmark trials

Trial (year)	Finding
ARDSNet ARMA (2000)	VT 6 vs 12 ml/kg PBW: mortality 31% vs 40%

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Trial (year)	Finding
ALVEOLI (2004)	Higher vs lower PEEP table: no difference overall
Amato driving pressure (2015)	Driving pressure best predicts survival in ARDS
PROSEVA (2013)	Prone ≥16 h/day in P/F <150: mortality halved
ACURASYS (2010) / ROSE (2019)	Early cisatracurium helped / no benefit with lighter sedation
ART (2017)	Aggressive recruitment + titrated PEEP increased mortality
OSCILLATE / OSCAR (2013)	HFOV no benefit / harm in adults
EOLIA (2018)	VV-ECMO in very severe ARDS: 35% vs 46% mortality (secondary analyses favourable)
FLORALI (2015)	HFNC reduced intubation in P/F ≤200 and mortality vs O_2/NIV
Roca ROX (2019)	ROX ≥4.88 predicts HFNC success
HIGH-WEAN (2019)	NIV + HFNC after extubation reduces re-intubation in high-risk patients
Girard ABC (2008)	Paired SAT + SBT reduces ventilator days and mortality
Subirà (2019)	30-min PS-8 SBT more successful than 2-h T-piece, no excess re-intubation
TracMan (2013)	Early tracheostomy did not reduce mortality
PREOXI (2024)	NIV pre-oxygenation reduced hypoxaemia during intubation vs face mask
DEXA-ARDS (2020)	Dexamethasone reduced mortality and ventilator days in moderate–severe ARDS
RECOVERY (2020)	Dexamethasone reduced mortality in ventilated COVID-19

KEY POINT Ventilation practice continues to evolve. Follow your institution's protocols and the latest guidelines (ATS/ERS, ESICM, SCCM, ISCCM).

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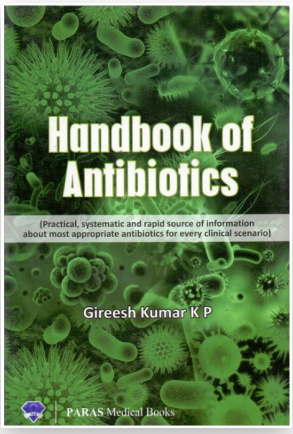
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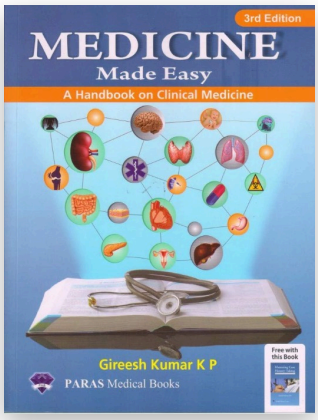
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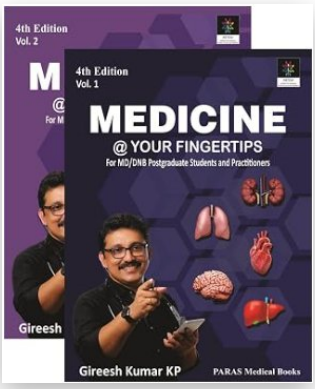
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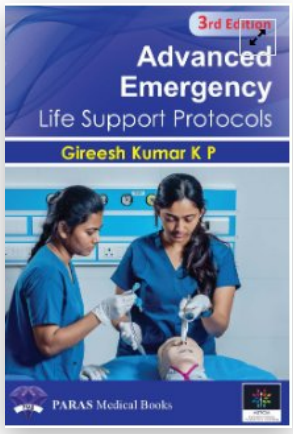
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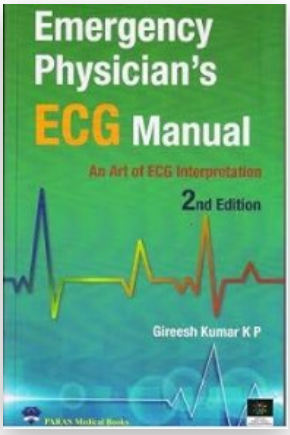
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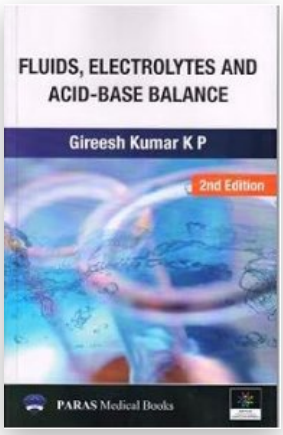
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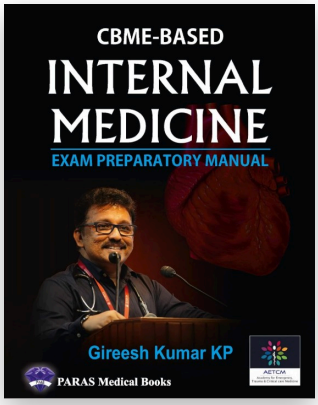
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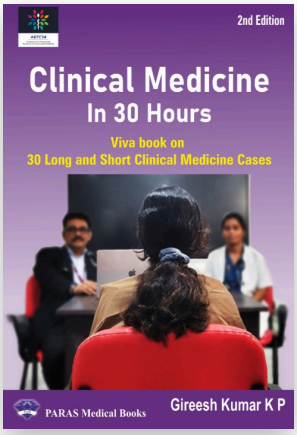
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