

Invasive Mechanical Ventilation

Complete clinical topic overview. For the full 10-level interactive course, visit Master MV.

Covers core ventilator modes, respiratory mechanics (Cstat, Raw, driving pressure), waveform dyssynchrony analysis, VILI prevention, and liberation protocols.

Last reviewed: 2026-09-07

Updated through the 2026 Saudi Guideline for Mechanical Ventilation in Adults, the AARC 2024 spontaneous-breathing-trial guideline, and contemporary ventilation/liberation trials including FAST 2024, UK-ROX 2025, PROMIZING 2025, and ACTIVE 2026, while retaining ARDSNet lung-protective principles.

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GUIDELINE

Educational content only. Use this handout for structured review; verify patient-specific decisions against local protocols and current guidelines.

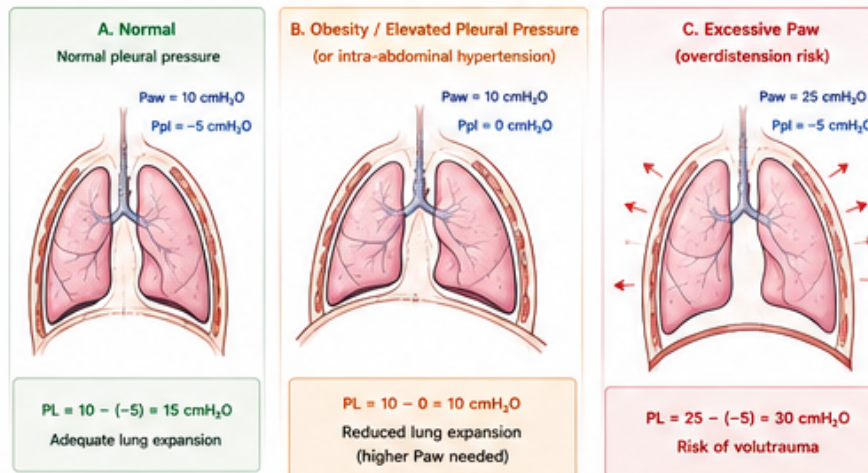
Overview

Invasive mechanical ventilation delivers positive-pressure gas to support pulmonary gas exchange and unload exhausted respiratory muscles while balancing the risks of ventilator-induced lung injury and hemodynamic collapse.

4. TRANSPULMONARY PRESSURE (PL)

The distending pressure across the lung = Airway pressure (Paw) – Pleural pressure (Ppl)

$$PL = Paw - Ppl$$



Key Points

- PL reflects the true distending pressure of the lung, not the airway pressure alone.
- Driving pressure (ΔP) = Paw – PEEP $\approx$ change in PL and correlates with lung strain and outcomes.
- Aim for adequate PL to open alveoli but avoid excessive PL to prevent lung injury.

transpulmonary pressure

Medical visual available in the online topic: ppv-cardiovascular-effects

Invasive mechanical ventilation is a life-sustaining supportive intervention designed to maintain alveolar ventilation, optimize arterial oxygenation, reduce the metabolic work of breathing, and allow therapeutic stabilization in acute respiratory failure, shock, or severe neurologic impairment.

Because positive-pressure ventilation reverses the normal negative-pressure physiology of spontaneous breathing, it introduces serious iatrogenic hazards: ventilator-induced lung injury (VILI: barotrauma, volutrauma, atelectrauma, biotrauma), severe reduction in right ventricular venous return (preload collapse), and patient-ventilator dyssynchrony. Critical care management requires meticulous parameter selection, continuous monitoring of respiratory mechanics (Cstat, Raw, Driving Pressure, Mechanical Power), and daily protocolized screening for liberation.

Differential Diagnosis of Sudden Ventilator Deterioration (DOPE Mnemonic)

Systematic bedside evaluation of acute high peak pressures and sudden hypoxemia during mechanical ventilation.

- D - Dislodged / Malpositioned Endotracheal Tube: Right mainstem intubation (high peak pressure, asymmetric breath sounds) or accidental extubation.
- O - Obstruction: Mucus plugging, tube kinking, biting on endotracheal tube, or herniated cuff.
- P - Pneumothorax: Sudden rise in peak and plateau pressures, unilateral absent breath sounds, subcutaneous emphysema, hemodynamic collapse.
- E - Equipment Failure: Ventilator malfunction, oxygen supply failure, circuit disconnect, or exhalation valve blockage.

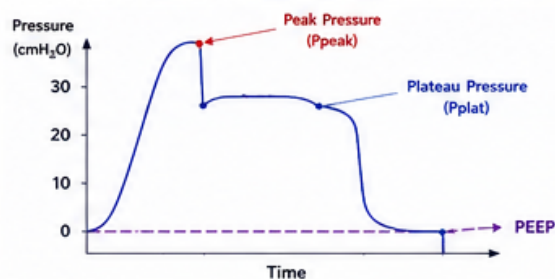
Diagnostic Evaluation: Respiratory Mechanics & Patient-Ventilator Asynchrony

Waveform analysis, static compliance calculation, driving pressure measurement, and asynchrony pattern recognition.

3. PEAK vs PLATEAU PRESSURE MECHANICS

Understanding what your ventilator shows

PRESSURE WAVEFORM (VOLUME CONTROL)

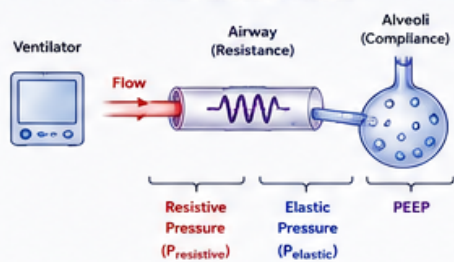


KEY DIFFERENCE

Peak Pressure is measured during flow.

Plateau Pressure is measured during a pause at zero flow (no resistance component).

AIRWAY & ALVEOLAR MODEL



PRESSURE EQUATIONS

$$P_{\text{peak}} = P_{\text{resistive}} + P_{\text{elastic}} + \text{PEEP}$$

$$= (\text{Flow} \times \text{Resistance}) + (\text{Volume} / \text{Compliance}) + \text{PEEP}$$

$$P_{\text{plat}} = P_{\text{elastic}} + \text{PEEP}$$

(measured during inspiratory hold at zero flow)

KEY POINTS

- P_{peak} includes both resistive and elastic components.
- P_{plat} reflects elastic load only (better indicator of lung stress).
- $P_{\text{peak}} - P_{\text{plat}} = \text{resistive pressure (airway resistance)}$.

ventilator pressures driving

1. DRIVING PRESSURE

The pressure that actually "drives" volume into the lungs

DEFINITION

Driving Pressure (ΔP) = Plateau Pressure (P_{plat}) - PEEP

$$\Delta P = P_{plat} - PEEP$$



WHY IT MATTERS

- ΔP correlates with lung stress/strain
- Higher ΔP → more ventilator-induced lung injury (VILI)
- Target $\Delta P \leq 15$ cmH₂O (when possible)

EXAMPLE

$P_{plat} = 28$ cmH₂O $PEEP = 8$ cmH₂O

$\Delta P = 28 - 8 = 20$ cmH₂O (elevated)

KEY POINTS

- Driving pressure reflects the distending pressure across the respiratory system.
- It is a better predictor of outcome than VT or PEEP alone.
- Aim for the lowest ΔP that achieves adequate ventilation.

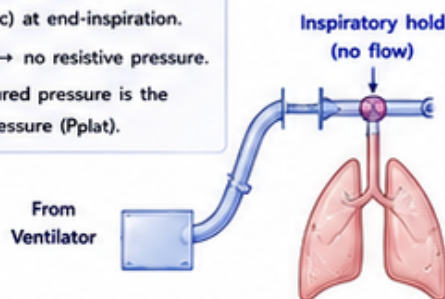
driving pressure mechanics

3. PLATEAU PRESSURE

Reflects the pressure in the alveoli (elastic pressure) at zero flow

HOW IT IS MEASURED

1. Perform an inspiratory hold (0.5 – 1 sec) at end-inspiration.
2. Flow = 0 → no resistive pressure.
3. The measured pressure is the Plateau Pressure (Pplat).



WHAT IT REPRESENTS

Pplat = Alveolar pressure
(Elastic pressure of the respiratory system)

It reflects the distending pressure on the alveoli and chest wall.

Ppeak vs Pplat

$P_{peak} = P_{plat} + (\text{Resistive pressure})$

Resistive pressure depends on flow and airway resistance.

TYPICAL VALUES

- Pplat < 30 cmH₂O (protective target)
- Pplat ≤ 28–30 cmH₂O in ARDS
- Higher Pplat → ↑ risk of VILI

KEY POINT: Plateau pressure removes the effect of airway resistance and reflects true lung/chest wall distending pressure.

plateau pressure mechanics

1. AIRWAY RESISTANCE (Raw)

$$R_{aw} = \Delta P / \text{Flow}$$

POISEUILLE'S LAW (Laminar Flow)

$$R_{aw} \propto \frac{\eta \cdot L}{r^4}$$

- Resistance is inversely proportional to the 4th power of radius
- Small decrease in radius → large increase in resistance

EFFECT OF AIRWAY RADIUS ON RESISTANCE (All other factors constant)

Normal radius (r)	50% ↓ radius (r/2)	70% ↓ radius (0.3r)	90% ↓ radius (0.1r)
			
Raw = 1x	Raw = 16x	Raw = 123x	Raw = 10,000x

CLINICAL SITUATIONS

Normal Airway

- Normal radius
- Minimal resistance
- Low peak-plateau gradient



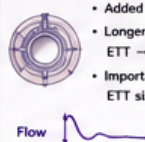
Bronchospasm / Secretions

- ↓ Radius
- ↑ Resistance
- ↑ Peak pressure
- ↑ Peak-plateau gradient

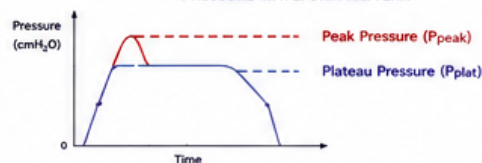


Endotracheal Tube (ETT)

- Added tube resistance
- Longer & smaller ETT → ↑ resistance
- Important in small ETT sizes



PRESSURE WAVEFORM PATTERN



- High Airway Resistance
- ↑ Peak Pressure
 - Plateau Pressure may be normal
 - ↑ (P_{peak} - P_{plat}) gradient

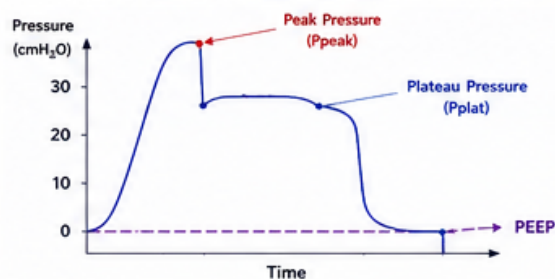
KEY POINT: Airway resistance is the major determinant of peak pressure and the peak-plateau gradient.

airway resistance poiseuille

3. PEAK vs PLATEAU PRESSURE MECHANICS

Understanding what your ventilator shows

PRESSURE WAVEFORM (VOLUME CONTROL)

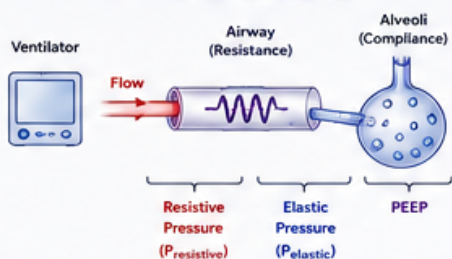


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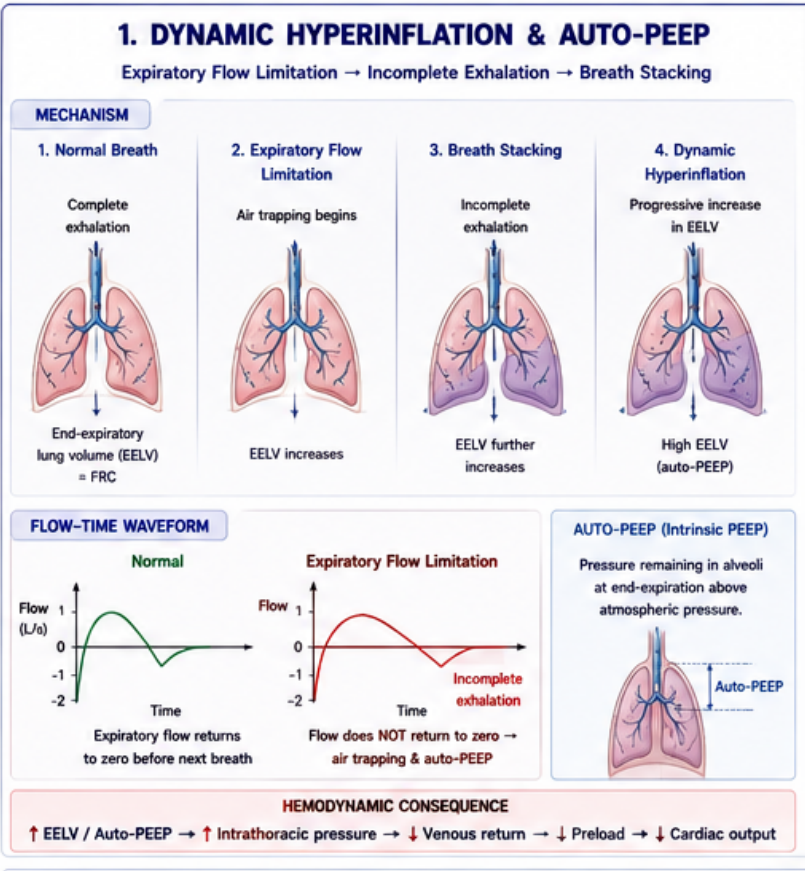
$$P_{\text{plat}} = P_{\text{elastic}} + \text{PEEP}$$

(measured during inspiratory hold at zero flow)

KEY POINTS

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peak vs plateau pressure mechanics



auto peep hyperinflation

Static Respiratory System Compliance (Cstat)	Formula: $C_{stat} = V_T / (P_{plat} - PEEP)$. Normal is 50-80 mL/cmH2O; in severe ARDS, Cstat drops < 20-30 mL/cmH2O indicating severe lung stiffness.
Driving Pressure (Delta P)	Formula: $\Delta P = P_{plat} - PEEP$. In observational studies (Amato et al., NEJM 2015), lower driving pressure is associated with improved survival; values ≤ 14 -15 cmH2O are commonly discussed in literature as a prognostic reference range rather than a universal hard cutoff.
Airway Resistance (Raw)	Formula: $R_{aw} = (P_{peak} - P_{plat}) / \text{Flow}$. Normal ≤ 10 -15 cmH2O/L/s. Elevated ($P_{peak} - P_{plat} > 10$ -15 cmH2O) indicates bronchospasm, mucus plugging, or small endotracheal tube.
Asynchrony Recognition	Inspect flow-time and pressure-time curves for: Trigger delay, Ineffective triggering (diaphragmatic effort without breath delivery), Double triggering (breath stacking), Flow starvation (concave pressure curve), and Premature/Delayed cycling.

Classification of Ventilator Modes & Control Variables

Phase variables governing mandatory and assisted breaths: trigger, limit, and cycle mechanisms across Volume Control, Pressure Control, and Pressure Support.

A ventilator breath is defined by four phase variables: (1) Trigger (what starts inspiration: time, pressure, or flow); (2) Target/Limit (what parameter is capped during inspiration: volume, flow, or pressure); (3) Cycle (what ends inspiration: volume, time, or flow); and (4) Baseline (what end-expiratory pressure is maintained: PEEP).

Volume Control (VC-CMV / AC-VC)	Volume-targeted, flow-limited, time- or volume-cycled. A fixed tidal volume and flow rate/waveform are delivered with every breath. Provides a set tidal volume per mandatory breath and therefore more predictable minute ventilation when delivery is accurate; actual ventilation can still be altered by leaks, patient interaction, dead space, or circuit/device factors. Airway pressures vary with compliance and resistance.
Pressure Control (PC-CMV / AC-PC)	Pressure-targeted, pressure-limited, time-cycled. A constant inspiratory pressure is applied for a set inspiratory time (Ti) with a decelerating flow waveform. Sets/limits inspiratory pressure at the selected pressure target; delivered tidal volume varies with compliance, resistance, and patient effort.
Pressure Support Ventilation (PSV)	Patient-triggered, pressure-limited, flow-cycled. The patient initiates every breath, and the ventilator augments inspiratory effort by delivering a set pressure above PEEP until inspiratory flow decays to a set percentage of peak flow (cycle-off threshold, typically 25%). Requires intact respiratory drive.
Dual-Control / Volume-Targeted Pressure Modes (PRVC / VC+)	Pressure-regulated volume control delivering decelerating flow while automatically adjusting inspiratory pressure breath-by-breath to achieve a target tidal volume at the lowest possible airway pressure.

Respiratory Mechanics: Compliance, Resistance, Driving Pressure, & Mechanical Power

Mathematical equations of motion, plateau pressure, static compliance, airway resistance, driving pressure (Amato et al.), and mechanical power.

The Equation of Motion for the respiratory system states: $P_{aw} = P_{elastic} + P_{resistive} + PEEP = (V_t / C_{stat}) + (R_{aw} \times \text{Flow}) + PEEP$. During an end-inspiratory pause (flow = 0), resistive pressure drops to zero, and airway pressure equilibrates to the Static Plateau Pressure (Pplat), which reflects alveolar distending pressure.

Peak Airway Pressure (Ppeak)	Total pressure required to overcome both airway resistance and lung/chest wall elastic recoil. $P_{peak} = P_{plat} + (R_{aw} \times \text{Flow})$.
Plateau Pressure (Pplat)	Measured via a 0.5-second end-inspiratory pause in a relaxed/sedated patient without spontaneous effort. Reflects true alveolar end-inspiratory pressure. Target $P_{plat} \leq 30$ cmH ₂ O to prevent volutrauma/barotrauma.

Static Compliance (Cstat)	$C_{stat} = V_t / (P_{plat} - PEEP_{total})$. Normal in intubated lungs is 50-80 mL/cmH ₂ O. In severe ARDS or pulmonary edema, Cstat plummets to < 20-30 mL/cmH ₂ O.
Airway Resistance (Raw)	$R_{aw} = (P_{peak} - P_{plat}) / \text{Flow (in L/sec)}$. Normal intubated Raw is 5-10 cmH ₂ O/L/s. In severe bronchospasm or small ETTs, Raw exceeds 20-40 cmH ₂ O/L/s.
Driving Pressure (DeltaP)	$\Delta P = P_{plat} - PEEP_{total} = V_t / C_{stat}$. Landmark study by Amato et al. (NEJM 2015) identified driving pressure as strongly associated with hospital survival in ARDS, reflecting tidal volume relative to respiratory system compliance. Lower driving pressure is generally desirable within an overall lung-protective strategy.
Mechanical Power (MP)	$MP \text{ (J/min)} = 0.098 \times RR \times V_t \times [P_{peak} - (\Delta P / 2)]$ (Gattinoni et al.). Represents the total mechanical energy transferred from the ventilator to the lung parenchyma per minute. Higher mechanical power (> 17-20 J/min) has been observationally associated with worse outcomes, though no single value serves as a universal bedside safe/unsafe threshold.

GUIDELINE

Interactive calculator available on the ICU Knowledge Map: driving-pressure

GUIDELINE

Interactive calculator available on the ICU Knowledge Map: static-compliance

Patient-Ventilator Interaction: Waveform Analysis & Dyssynchrony

Recognition and remediation of flow starvation, ineffective triggering, double triggering, auto-triggering, and reverse triggering.

Patient-ventilator dyssynchrony occurs in > 25% of mechanically ventilated patients and is independently associated with prolonged mechanical ventilation, increased sedation requirements, ICU delirium, and higher mortality:

1. Flow Starvation / Inadequate Flow	Waveform: Concave/scalloped pressure-time curve during inspiration in volume control. Cause: Patient inspiratory flow demand exceeds fixed set flow. Fix: Increase peak flow rate (e.g., to 70-90 L/min), switch to a decelerating flow profile, or change to Pressure Control / Pressure Support.
2. Ineffective Triggering / Missed Efforts	Waveform: Negative deflection on pressure-time curve with a blip in expiratory flow during expiration that fails to trigger a machine breath. Cause: Auto-PEEP imposing an inspiratory threshold load, or trigger sensitivity set too low. Fix: Treat dynamic hyperinflation first and, when expiratory flow limitation is present, cautiously titrate external PEEP below measured intrinsic PEEP while reassessing triggering, total PEEP, expiratory flow, lung volume, and hemodynamics; also optimize trigger sensitivity.

3. Double Triggering / Breath Stacking	Waveform: Two consecutive mandatory breaths delivered with virtually no expiratory pause, delivering double the set tidal volume. Cause: Patient neural inspiratory time exceeds ventilator inspiratory time (Ti set too short, or Vt set too low). Fix: Increase tidal volume, lengthen Ti, or adjust pressure support cycle-off criteria.
4. Auto-Triggering	Waveform: Rapid mandatory breath delivery without preceding negative patient effort. Cause: Overly sensitive trigger threshold, circuit condensation/water sloshing, cardiogenic oscillations from hyperdynamic heart. Fix: Switch from flow to pressure trigger, drain circuit water, optimize sensitivity.
5. Reverse Triggering	Waveform: Patient diaphragmatic contraction rhythmically triggered by the preceding mandatory machine breath. Occurs in deeply sedated patients. Can cause dynamic breath stacking. Fix: Adjust sedation depth or change respiratory rate.

Ventilator-Induced Lung Injury (VILI) Mechanisms

Barotrauma, volutrauma, atelectrauma, biotrauma, and ergotrauma pathways.

- Barotrauma: High transalveolar pressure causing macroscopic alveolar rupture, leading to pulmonary interstitial emphysema, pneumothorax, pneumomediastinum, and subcutaneous emphysema.
- Volutrauma: Regional alveolar overdistension from excessive end-inspiratory lung volume (strain), leading to alveolar-capillary membrane disruption, increased vascular permeability, and non-cardiogenic pulmonary edema.
- Atelectrauma: Shear stress injury caused by repetitive cyclic opening and closing of unstable, surfactant-depleted alveoli during tidal breathing. Prevented by adequate positive end-expiratory pressure (PEEP).
- Biotrauma: Mechanical deformation of epithelial and endothelial cells triggering intracellular signal transduction cascades that release pro-inflammatory cytokines (IL-6, IL-8, TNF-alpha) into the systemic circulation, driving multiorgan dysfunction syndrome (MODS).
- Ergotrauma: Lung injury caused by excessive cumulative kinetic energy and mechanical power delivered per unit time into heterogeneous lung tissue.

Liberation from Mechanical Ventilation: The ABCDEF Bundle & SBT Protocol

Spontaneous Awakening Trial (SAT), Spontaneous Breathing Trial (SBT), Rapid Shallow Breathing Index (RSBI), and Cuff Leak Test.

Liberation from mechanical ventilation should be evaluated daily via protocolized screening to avoid prolonged intubation:

1. Daily Readiness Screening	Screen at least daily once the cause of respiratory failure is improving. Readiness is a clinical judgment based on adequate spontaneous effort, acceptable oxygenation/ventilation on modest support, and cardiovascular stability; no single PaO ₂ /FiO ₂ , PEEP, pH, vasopressor dose, mental-status score, cough, or gag threshold is universally required before attempting an SBT.
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2. Spontaneous Awakening Trial (SAT)	Use a structured sedation-minimization/spontaneous-awakening approach when safe. Pause or reduce sedatives that impede wakefulness, but continue and titrate analgesia to pain and do not abruptly stop medications when withdrawal, seizures, intracranial physiology, paralysis, severe agitation, or another safety issue makes interruption inappropriate.
3. Spontaneous Breathing Trial (SBT)	AARC 2024 suggests that SBTs may be conducted with or without low-level pressure support and recommends a standardized daily assessment process; FiO ₂ should not be increased merely to make an SBT look successful. Use a reproducible local SBT method and interpret the whole clinical response rather than declaring one technique universally superior.
4. Rapid Shallow Breathing Index (RSBI)	RSBI = respiratory rate / tidal volume (L), but AARC 2024 suggests that calculating RSBI is not required to determine readiness for an SBT. If measured, use it as one supportive physiologic signal rather than a binary extubation cutoff; SBT tolerance, airway protection, secretion burden and the cause of prior failure matter more than one threshold.
5. Cuff Leak Test for Post-Extubation Stridor	Do not use a cuff-leak test routinely in every patient. In an otherwise extubation-ready patient at increased risk of post-extubation stridor, an absent/reduced leak increases risk but has imperfect specificity and should not become an automatic prolonged-intubation decision. ATS/CHEST guidance supports systemic corticosteroid pretreatment when a high-risk patient fails the test, started at least several hours before extubation according to the local protocol; a repeat cuff-leak test is not mandatory after steroid treatment.
6. Planned Post-Extubation NIV / HFNC	For selected patients at high risk of extubation failure, planned post-extubation support reduces reintubation risk. Evidence is strongest for prophylactic NIV (often alternated with HFNC) in appropriately selected high-risk patients, particularly those with chronic cardiac/respiratory disease or hypercapnia; HFNC is also useful in many populations. Choice should reflect the risk phenotype rather than applying NIV to every older or obese patient, and a mortality benefit is not universal.

Common ICU Pitfalls

Critical mistakes in ventilator setup, monitoring, and liberation.

- Calculating tidal volume based on actual body weight instead of Predicted Body Weight (PBW): Lung size is determined by height and biological sex, not adipose tissue; ventilating an obese patient based on actual weight delivers severely injurious tidal volumes (> 12-15 mL/kg PBW).
- Treating patient-ventilator dyssynchrony reflexively with deeper sedation or paralytics without analyzing the pressure/flow waveforms: Most dyssynchrony is easily solved by increasing peak inspiratory flow, matching auto-PEEP, or adjusting trigger sensitivity.
- Confusing Peak Pressure with Plateau Pressure: An elevated P_{peak} with a normal P_{plat} (< 28 cmH₂O) indicates an airway resistance problem (ETT biting, bronchospasm, secretions, kinked tube), NOT alveolar stiffness.
- Failing to recognize auto-PEEP as the cause of difficult weaning or post-intubation shock: Always perform an end-expiratory hold maneuver to measure intrinsic PEEP.
- Failing to plan post-extubation support in a genuinely high-risk patient: consider prophylactic NIV and/or HFNC according to the patient's risk phenotype, gas exchange and the evidence for the

relevant subgroup.

Controversies & Areas of Uncertainty

Optimal SBT technique (PSV vs T-piece) and driving pressure titration.

SBT Technique - Pressure Support (PSV) versus T-Piece: While the 2017 ATS/CHEST guidelines recommend PSV (5-8 cmH₂O) to overcome the resistive work of the endotracheal tube, critics argue that PSV may underestimate post-extubation work of breathing, leading to premature extubation in marginal patients. T-piece trials provide a truer reflection of unassisted breathing mechanics.

Driving Pressure-Guided vs PEEP/FiO₂ Table-Guided Ventilation: While traditional ARDS management utilizes standard PEEP/FiO₂ titration tables, randomized trials are evaluating whether individualizing PEEP to achieve the lowest Driving Pressure ($\Delta P = P_{\text{plat}} - \text{PEEP}$) or maximum static compliance improves clinical survival compared to empiric protocolized tables.

First-Hour Ventilator Setup: Set, Measure, Reassess

Choose the initial mode and settings from physiology, then immediately verify delivered ventilation, mechanics and hemodynamic tolerance.

1. Confirm airway position and patency, select a mode that reliably achieves the immediate gas-exchange goal, and calculate tidal volume from predicted rather than actual body weight when volume targeting is used.
2. Set oxygen, PEEP, respiratory rate, inspiratory flow/time and alarms to the phenotype: ARDS, obstructive disease, severe metabolic acidosis, RV failure and brain injury require different priorities.
3. Within minutes, inspect exhaled tidal volume, minute ventilation, pressure/flow waveforms, SpO₂/ABG when needed, blood pressure and signs of dynamic hyperinflation or excessive transpulmonary stress.
4. Treat the patient rather than the displayed mode name: the same nominal mode can deliver very different pressure, flow, effort and mechanical power depending on patient demand and ventilator implementation.

Bedside Patient-Ventilator Assessment

A repeatable safety examination integrating airway, settings, waveforms, mechanics, gas exchange, effort and circulation.

- Airway/circuit: tube depth, cuff, humidification, secretions, obstruction, water, kinks and ventilator/circuit integrity.
- Ventilator: verify what is actually delivered-Vt, minute ventilation, FiO₂, PEEP, inspiratory time/flow, trigger and cycling-not merely what was ordered.
- Mechanics: compare peak and plateau pressure when a valid inspiratory hold is feasible, estimate compliance and resistance, and measure intrinsic PEEP with an expiratory hold only when patient effort does not invalidate the maneuver.
- Interaction: identify ineffective efforts, double triggering, flow starvation, premature/delayed cycling and reverse triggering before reflexively deepening sedation.
- Whole patient: integrate gas exchange, work of breathing, diaphragm/respiratory drive, RV/LV hemodynamics and comfort; driving pressure and mechanical power are risk markers, not universal stand-alone targets for every phenotype.

From a Passed SBT to Safe Extubation

Passing an SBT demonstrates ventilatory capacity but does not by itself prove airway or post-extubation safety.

1. After SBT success, separately assess airway patency risk, secretion burden, cough/airway-protection capability, mental status trajectory and the probability of rapid recurrent pulmonary edema or hypercapnia.
2. Plan post-extubation respiratory support before removing the tube. Prophylactic NIV has the strongest evidence in selected high-risk patients, while HFNC is useful across several hypoxemic and post-extubation phenotypes; choose by physiology and local evidence rather than age alone.
3. A cuff-leak test is selective for patients at increased risk of laryngeal edema, not a universal extubation screen.
4. If clinically important post-extubation respiratory failure develops, use NIV/HFNC only when the phenotype and trajectory are appropriate; do not allow noninvasive support to delay reintubation in a deteriorating patient.

FOUNDATION SOURCES

1. Irwin & Rippe's Intensive Care Medicine, 9th Edition, 2023 - Chapters on Principles of Mechanical Ventilation, Patient-Ventilator Interaction, and Liberation from Mechanical Ventilation
2. Tobin's Principles and Practice of Mechanical Ventilation, 3rd Edition, 2013

LINKED EVIDENCE

1. American Thoracic Society / American College of Chest Physicians ATS/ACCP (2017). Liberation from Mechanical Ventilation in Critically Ill Adults. Status: current.
2. American Thoracic Society / European Society of Intensive Care Medicine / Society of Critical Care Medicine ATS/ESICM/SCCM (2017). Mechanical Ventilation in Adult Patients with ARDS. Status: historical.
3. Saudi national multidisciplinary mechanical ventilation task force Saudi MV 2026 (2026). Saudi Guideline for Mechanical Ventilation in Adults. Status: current.
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4. Yang KL, Tobin MJ. A prospective study of indexes predicting the outcome of trials of weaning from mechanical ventilation. N Engl J Med. 1991;324(21):1445-1450.