

04 - 313 Mechanical Ventilatory Support

313 Mechanical Ventilatory Support

patients who survive regain nearly normal lung function. Patients usually recover maximal lung function within 6 months. One year after endotracheal extubation, more than one-third of ARDS survivors have normal spirometry values and diffusion capacity. Most of the remaining patients have only mild abnormalities in pulmonary function. Unlike mortality risk, recovery of lung function is strongly associated with the extent of lung injury in early ARDS. Low static respiratory compliance, high levels of required PEEP, longer durations of mechanical ventilation, and high lung injury scores are all associated with less recovery of pulmonary function. Of note, when physical function is assessed 5 years after ARDS, exercise limitation and decreased physical quality of life are often documented despite normal or nearly normal pulmonary function. When caring for ARDS survivors, it is important to be aware of the potential for a substantial burden of psychological problems in patients and family caregivers, including significant rates of depression and posttraumatic stress disorder. Investigations into sequelae of COVID ARDS have provided additional insight into longterm ICU outcomes. Please see Chap. 205 for information regarding COVID prognosis and recovery.

PART 8 Critical Care Medicine ■ ■ FURTHER READING ARDS Definition Task Force: Acute respiratory distress syndrome: The Berlin definition. *JAMA* 307:2526, 2012. ARDS Network: Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 342:1301, 2000. Bellani G et al: Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. *JAMA* 315:788, 2016. Dequin P-F et al: Hydrocortisone in severe community-acquired pneumonia. *N Engl J Med* 388:1931, 2023. Gorman EA et al: Acute respiratory distress syndrome in adults: Diagnosis, outcomes, long-term sequelae, and management. *Lancet* 400:1157, 2022. Matthay MA et al: A new global definition of acute respiratory distress syndrome. *Am J Respir Crit Care Med* 209:37, 2024. Munshi L et al: Noninvasive respiratory support for adults with acute respiratory failure. *N Engl J Med* 387:1688, 2022. The National Heart, Lung, and Blood Institute Petal Clinical Trials Network: Early neuromuscular blockade in the acute respiratory distress syndrome. *N Engl J Med* 380:1996, 2019. ■ ■ WEBSITES ARDS Foundation: www.ardsusa.org ARDS Support Center for patient-oriented education: www.ards.org National Heart, Lung, and Blood Institute ARDS Clinical Trials information: www.ardsnet.org and www.petalnet.org Scott Schissel

Support Mechanical ventilation refers to devices that deliver positive pressure gas, of varying oxygen content, to patients with acute or chronic respiratory failure. Hypoxemic respiratory failure refractory to supplemental oxygen and requiring mechanical ventilation is most often due to ventilation-perfusion mismatch or shunt caused by processes such as pneumonia, pulmonary edema, alveolar hemorrhage, acute respiratory distress syndrome (ARDS), and sequelae of trauma or surgery. Hypercapnic respiratory failure is most frequently caused by severe exacerbations of obstructive lung disease, including asthma and

chronic obstructive pulmonary disease (COPD); loss of central respiratory drive from acute neurologic events, such as stroke, intracranial hemorrhage, or drug overdose; and respiratory muscle weakness from diseases such as Guillain-Barré syndrome. Mechanical ventilation may also be necessary if patients have an artificial airway placed (an endotracheal tube) due to poor airway protection, such as in coma or in the context of a large upper gastrointestinal hemorrhage and vomiting, or due to processes leading to large airway obstruction, such as laryngeal edema. Finally, since mechanical ventilation can lower the work of breathing compared to spontaneous ventilation, it is a useful adjunct therapy for shock and multiorgan system failure. **PRINCIPLES OF MECHANICAL VENTILATION** Although contemporary mechanical ventilators use positive pressure to inflate the lungs, a patient's response to pressure applied across the lung (transpulmonary pressure) depends on the elastic properties of their lungs and chest wall; the amount of pressure needed to inflate a lung is the same, therefore, whether applied positively via mechanical ventilation or negatively using the diaphragm and chest wall muscles. In ARDS, for example, lungs are "stiff" or poorly compliant and often require much more pressure to achieve a physiologic tidal volume (Fig. 313-1), which, over time, may lead to respiratory muscle fatigue. If a patient with ARDS is on mechanical ventilation and makes no spontaneous respiratory effort, using sedation and neuromuscular blockade, the amount of positive pressure needed to inflate the lung is equal to the negative inflation pressure required if the patient were spontaneously breathing; however, the work of breathing is removed on a ventilator, allowing for sustainable ventilation. Mechanical ventilation can be lifesaving by restoring adequate oxygenation and correcting hypercapnia. Optimal application of positive pressure ventilation, however, requires avoiding underinflation, which can cause cycles of alveolar recruitment then collapse and, at the other extreme, alveolar overinflation (Fig. 313-2); collectively, these processes can cause ventilator-induced lung injury by barotrauma and volume trauma. Optimal tidal volume ventilation occurs along the lung pressure-volume curve where respiratory system compliance is greatest, or where the smallest change in applied pressure leads to the greatest increase in lung volume (Fig. 313-2, shaded box). To prevent too low lung volumes at end-exhalation, where alveolar collapse occurs, the ventilator can be set to maintain a specified positive pressure at end-exhalation, or positive end-expiratory pressure (PEEP) (Fig. 313-2B). Lower tidal volume ventilation (goal 6 mL/kg of ideal body weight)

5 L Normal

Volume (liters and % total lung capacity)

2.5 L

ARDS

0.5 L

-30 -20 -10

Pressure (cmH₂O) FIGURE 313-1 Hypothetical pressure-volume curves of patients with normal lung function (normal) and acute respiratory distress syndrome (ARDS). A tidal volume breath of 0.5 L in the normal lung requires 8 cmH₂O of pressure (open box), but in ARDS requires 28 cmH₂O (shaded box).

5 L

Volume (liters and % total lung capacity)

B. Optimal PEEP: 20 cmH₂O D. Alveolar overdistension 2.5 L

ARDS

C. Protective ventilation A. Alveolar collapse

0.5 L

-30 -20 -10

Pressure (cmH₂O) FIGURE 313-2 Hypothetical pressure-volume curve of a patient with acute respiratory distress syndrome (ARDS), demonstrating optimal positive end-expiratory pressure (PEEP) and protective ventilation. A tidal volume breath of 0.5 L initiated at a PEEP of 20 cmH₂O (B), after the area of greatest alveolar collapse (A). End inhalation occurs within the most compliant portion of the pressure-volume curve (C) and at a pressure <30 cmH₂O, before the area where lung overdistension occurs (D), minimizing lung injury. can help prevent the end-inhalation, or “plateau,” pressure (measured just after flow stops at end-inhalation) from exceeding 30 cmH₂O; this approach minimizes barotrauma and volume trauma-induced lung injury, especially in ARDS patients (Fig. 313-2C). MECHANICAL VENTILATION MODES Mechanical ventilation entails controlling or monitoring the same basic variables involved in spontaneous, negative-pressure breathing, including respiratory rate, tidal volume (VT), inspiratory flow rate and time, and the fraction of inspired oxygen (Fio₂). In addition, the PEEP is a variable specific to positive-pressure ventilation and set by the clinician. The mechanical ventilation mode determines how much control the clinician and ventilator have over these variables versus the patient; for example, assist control (AC) mode allows for essentially complete operator control of all variables, whereas pressure support TABLE 313-1 Key Features of Commonly Used Mechanical Ventilation Modes VARIABLES SET BY CLINICIAN (INDEPENDENT) MONITORED VARIABLES (DEPENDENT) ADVANTAGES DISADVANTAGES MODE Assist control-volume control VT Respiratory rate PEEP Fio₂ Inspiratory flow rate Peak inspiratory airway pressure End-inhalation (plateau) pressure VE Assist control-pressure control Inspiratory driving pressure Respiratory rate PEEP Fio₂ Tidal volume VE Pressure-regulated volume control VT Respiratory rate PEEP Fio₂ Peak inspiratory airway pressure End-inhalation (plateau) pressure VE Pressure support Inspiratory pressure PEEP Fio₂ VT Respiratory rate VE Abbreviations: Fio₂, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; VE, minute ventilation; VT, tidal volume.

(PS) permits the patient to control important variables, such as respiratory rate, VT, and flow rates (Table 313-1).

■ ■ ASSIST CONTROL VENTILATION AC allows the clinician to control nearly all ventilator variables and is widely used when patients cannot safely participate in their own ventilatory efforts, such as when deeply sedated or unstable from acute respiratory failure or other critical illness. Most AC ventilation is in volume control mode where the operator sets a specific VT and respiratory rate, thereby assuring a minimum minute ventilation (VE). In addition to the set rate, patients can get additional, fully supported breaths at the set VT by making an inspiratory effort, which is sensed by the ventilator and triggers the breath. The inspiratory flow rate is set by the operator; thus, a dyspneic patient may meet resistance on inhalation if their desired flow rate is higher than the set rate, possibly leading to patient distress and increased work of breathing. In AC volume mode, the operator also sets the PEEP and Fio₂. Importantly, since VT is an independent variable in volume control (i.e., set by the clinician), the end-inhalation (or plateau) pressure is a dependent variable not controlled by the clinician but rather determined by the compliance of the lung. Inspiratory pressures must be monitored, therefore, to minimize barotrauma. CHAPTER 313 Mechanical Ventilatory Support Although AC is often volume controlled, it can be used in a pressure control mode, also referred to as pressure control ventilation (PCV). The key difference between volume control and PCV is that an inspiratory (or “driving”) pressure is set instead of a tidal volume in PCV; thus, every time the ventilator delivers a breath, it raises the airway pressure to the set amount above PEEP until inspiratory flow decreases below a set threshold, therefore ending inhalation. The resulting tidal volume will, therefore, vary depending on the compliance of the lung. In a sedated and paralyzed patient (making no respiratory effort), the pressure required to generate a specific tidal volume (x) using PCV should, in the same patient, be equal to the plateau pressure in volume control mode where tidal volume is set at x. Importantly, since lung compliance can change dynamically, tidal volume may also change with PCV; tidal volume and minute ventilation, therefore, must be monitored since there is no assurance of delivered ventilation volumes as with volume control. PCV is often used to limit peak airway and lung distending (plateau) pressures in situations where high pressure can cause harm, such as in ARDS or after thoracic surgery with fresh suture lines in the airways or lung parenchyma. Importantly, however, inspiratory flow rate and volume are dependent variables in PCV, unlike in volume control ventilation, and not set by the clinician. Spontaneously breathing patients on PCV can generate a relative negative pressure in the Guarantee minimum VT and VE Control VT, limiting volume trauma Barotrauma from high plateau pressure Patient-ventilator dyssynchrony, increased work of breathing Limit barotrauma (if patient respiratory efforts minimal) Inspiratory flow can vary with patient effort (improved comfort/ synchrony) Vt and VE not mandated; must monitor closely Patient’s respiratory effort can lead to large VT and volume trauma Patient effort can vary inspiratory flow, increasing comfort, and ventilator synchrony Guarantee minimum VT and VE Variable patient effort can lead to VT larger than set VT; monitor to prevent volume trauma Patient effort preserved and controls VT, inspiratory flow, and respiratory rate, allowing for ventilator synchrony Apnea and hypoventilation possible; must monitor respiratory rate, VT, and VE closely

5 L

Volume (liters and % total lung capacity)

2.5 L

B

A

0.5 L

PART 8 Critical Care Medicine

-30 -20 -10

Pressure (cmH₂O) FIGURE 313-3 Hypothetical pressure-volume curve of a patient on pressure control ventilation, paralyzed (A) and breathing spontaneously (B). (A) Paralyzed patient (light shaded box): positive end-expiratory pressure (PEEP), 10 cmH₂O; inspiratory (driving) pressure, 15 cmH₂O; end-inhalation (plateau) pressure, 25 cmH₂O; tidal volume (VT), 300 mL. (B) Breathing patient (dark shaded box): PEEP, 10 cmH₂O; inspiratory (driving) pressure, 15 cmH₂O; patient effort (negative “pulling” pressure), 10 cmH₂O; end-inhalation (plateau) pressure displayed on ventilator, 25 cmH₂O; net end-inhalation (transalveolar) pressure, 35 cmH₂O; VT, 700 mL. ventilator circuit, transiently decreasing the positive pressure below the set point; the ventilator responds by increasing gas flow until it restores the set pressure, resulting in higher inspiratory flow rates, a higher tidal volume for that breath, and importantly, increased pressure across the alveoli, equal to the absolute (negative) pressure generated by the patient plus the positive pressure set by the clinician (Fig. 313-3). Since mechanical ventilators do not routinely measure or graphically display the negative pressure generated by the patient, clinicians can be unaware of this additional transalveolar pressure and potential harm by volume and barotrauma; importantly, therefore, clinicians should monitor for increases in tidal volume on PCV. ■ ■PRESSURE-REGULATED VOLUME

CONTROL VENTILATION Advances in ventilator technology, such as flow and pressure sensors and microprocessors, allow for additional modes of mechanical ventilation that meld the benefits of volume and pressure control ventilation. Pressure-regulated volume control (PRVC) ventilation is a fully supported mode of ventilation where the clinician sets a target tidal volume, as in volume control ventilation, but it allows a patient to make spontaneous respiratory efforts and vary inspiratory flow rates, as in PCV, enhancing patient comfort and ventilator synchrony. PRVC senses patient inspiratory efforts and delivers the least amount of positive pressure to achieve the targeted tidal volume; since patient efforts can vary and ventilator adaptation is not instantaneous, tidal volumes can vary from breath to breath on PRVC. In disease states where tidal volume needs tight control to prevent volume trauma, such as in ARDS, PRVC must be used cautiously if the patient can make significant respiratory effort. ■ ■PRESSURE SUPPORT VENTILATION Pressure support ventilation (PSV) and PCV are very similar except there is no mandated ventilation or set mechanical respiratory rate on PSV, and ventilator support is entirely patient triggered and controlled. The clinician sets the Fio₂, PEEP, and maximum inspiratory pressure. When patients make a negative-pressure inspiratory effort on PSV, the ventilator senses this pressure change and increases positive pressure to the set inspiratory pressure level, maintaining it until flow decreases below a set threshold (often ~20% of peak inspiratory flow); at this point, inhalation ends and

pressure drops back to the set PEEP. The

tidal volume on PSV is monitored but not assured, is determined by lung compliance, and depends on the patient's sustaining an inspiratory effort. Tidal volume, minute ventilation, and respiratory rate, therefore, must be closely monitored on PSV to detect hypopnea/apnea and hypoventilation. PSV is often used when patients are less sedated and able to participate in respiratory work, such as when transitioning off mechanical ventilation or on a ventilator only for airway support. ■

■ **NONINVASIVE POSITIVE PRESSURE VENTILATION** Noninvasive ventilation (NIV) is historically referred to as positive pressure ventilation and is delivered via a nasal or full-face mask at a continuous pressure (continuous positive airway pressure [CPAP]) or at different inspiratory and expiratory pressures (bi-level positive airway pressure [BiPAP]). Most current noninvasive ventilators, however, can function in full support modes, including volume control ventilation. NIV is particularly beneficial for acute respiratory failure where the underlying cause responds quickly to treatment, minimizing the need for prolonged mechanical ventilatory support. For moderate acute hypercapnia, blood pH between 7.25 and 7.35, due to exacerbations of chronic obstructive pulmonary disease (COPD), NIV, for example, reduces the need for endotracheal intubation and shortens hospital length of stay; more severe acute respiratory acidosis from COPD exacerbations (blood pH <7.2) generally requires mechanical ventilation with an endotracheal tube. NIV can also be an important adjunct treatment for respiratory failure from acute cardiogenic pulmonary edema, where interventions, such as diuresis and vasodilator therapy, can rapidly improve gas exchange and respiratory mechanics. NIV, particularly with volume support modes, is effective in managing chronic respiratory failure from restrictive lung diseases, such as severe scoliosis and respiratory muscle weakness, and in COPD complicated by chronic hypercapnia, where nocturnal NIV reduces COPD-related hospital admissions. Despite the technical innovations in NIV and expanding clinical applications, several important contraindications to using mechanical ventilation without a secure airway, such as an endotracheal tube or tracheostomy tube, include delirium, difficulty managing respiratory secretions, and hemodynamic instability (Table 313-2).

STRATEGIES TO OPTIMIZE GAS EXCHANGE ON MECHANICAL VENTILATION ■ **ARTERIAL OXYGENATION** The optimal partial pressure of arterial oxygen (Pao₂) and oxygen saturation measured by pulse oximetry (Spo₂) during mechanical ventilation remain uncertain. Although tissue hyperoxia can cause oxidative injury with some clinical studies of mechanically ventilated patients suggesting worse clinical outcomes with higher Fio₂ and when Pao₂ frequently reaches supraphysiologic levels, randomized studies comparing conservative oxygen delivery to a more liberal oxygen strategy have not demonstrated a clear advantage to conservative oxygen delivery. In ARDS, targeting a lower Pao₂ of 55–70 mmHg (or Spo₂ of 88–92%) versus a higher, but more physiologic, Pao₂ of 90–105 mmHg (or Spo₂ >96%) did not lower mortality, with adverse events being more frequent in the lower Pao₂ group, including mesenteric ischemia. Pao₂ and Spo₂ targets, therefore, should be individualized to patients considering circumstances where even mild hyperoxia may be harmful, such as in recovery from ischemic brain injury, and, conversely, where lower Pao₂ levels (<55–70 mmHg) may be less optimal, such as in patients with ARDS and evidence of bowel dysfunction. **TABLE 313-2 Common Contraindications to Noninvasive Ventilation**

Inability to protect the airway, such as severe encephalopathy
High risk for aspiration, such as vomiting or severe upper gastrointestinal bleeding
Difficulty clearing respiratory secretions
Facial trauma or surgery
Upper airway obstruction or compromise
Significant hemodynamic instability

of the approach, there is no evidence that a supraphysiologic Pao₂ (>100 mmHg) has clinical benefit; thus, sustained hyperoxia should be avoided. Arterial hypoxemia refractory to standard mechanical ventilation techniques is common in severe acute lung disease, especially ARDS. In general, if the Fio₂ requirement is >0.6 or the Pao₂:Fio₂ ratio is <150 mmHg, additional interventions should be considered to improve arterial oxygenation. The application of adequate PEEP to prevent alveolar collapse during exhalation improves oxygenation by decreasing ventilation/perfusion (V./Q.) mismatch and shunt in areas of atelectatic lung. PEEP should ideally be set at the lower inflection point of the most compliant region of the lung pressure-volume curve (Fig. 313-2B). Although optimal PEEP may improve arterial oxygenation, achieving best PEEP has not been shown to improve clinical outcomes definitively and may have deleterious effects, including barotrauma with pneumothorax and hypotension from decreasing venous return to the right ventricle. Patients with refractory hypoxemia are often dyspneic on mechanical ventilation and make significant respiratory efforts dyssynchronous with the ventilator despite deep sedation, leading to poor ventilation and preventing optimal V./Q. matching. In this context, neuromuscular blockade can be very effective at restoring effective mechanical ventilation and optimizing gas exchange. Although a necessary intervention at times, neuromuscular blockade does not improve overall outcomes in ARDS, can contribute to critical illness myopathy, and requires adequately deep sedation to prevent conscious paralysis; thus, it should be used only when necessary to treat refractory hypoxemia. In ARDS, diseased lung is predominantly dependent, and placing the patient in a prone position for extended periods can significantly improve arterial oxygenation. The role of prone positioning in other disease states is unknown and can be associated with adverse events unless performed by a trained team, such as dislodging endotracheal tubes and central venous catheters. Delivery of pulmonary vasodilator medications through the airway can improve perfusion to ventilated alveolar units, therefore improving V./Q. matching and arterial oxygenation. Inhaled prostacyclins, such as epoprostenol, and nitric oxide are commonly used to treat refractory hypoxemia and can increase, on average, the Pao₂:Fio₂ ratio by 20–30 mmHg. Hypoxemia refractory to these multiple interventions may require consideration of transitioning to extracorporeal membrane oxygenation (ECMO; see below).

■ ■HYPERCAPNIA Except for rare circumstances of excess CO₂ production (VCO₂), which can occur in the setting of fever, sepsis, overfeeding, and thyrotoxicosis, most hypercapnia is due to inadequate alveolar ventilation (VA) from an increase in the fraction of dead space (VD) [the volume of each breath not participating in CO₂ exchange] relative to the total minute ventilation (VE), expressed as $VA = VE (1 - VD/VT)$. Normal physiologic dead space is approximately 150 mL (~2 mL/kg), making the VD/VT for a 500-mL tidal volume breath 0.3. In acute respiratory failure due to ARDS, for example, VD may increase due to poorly perfused but ventilated portions of lung while ventilation strategies lead to low VT; thus, a modest increase in VD to 200 mL and a low VT of 300 mL will result in a VD/VT of 0.66, a situation where hypercapnia may easily develop. Hypercapnia in the context of low tidal volume (6 mL/kg) ventilation for ARDS often causes acute respiratory acidosis that can be managed with higher respiratory rates, up to 30 breaths/min. Respiratory acidosis is often tolerated down to a pH of 7.2, so-called “permissive hypercapnia,” but progressive acidosis may require intravenous alkalinizing therapy (e.g., sodium bicarbonate or tromethamine) or accepting an increase in VT. In severe exacerbations of obstructive lung disease, COPD, and status asthmaticus, hypercapnia and acute respiratory acidosis are common despite mechanical ventilation, with average Paco₂ values of 65 mmHg and blood pH of 7.20 after initial endotracheal intubation. Poor alveolar ventilation is primarily due to dead space created by alveolar capillary compression in areas of alveolar overdistension and lung hyperinflation. Increasing minute ventilation by increasing the respiratory

rate or tidal volume will, therefore, often paradoxically worsen hypercapnia by increasing gas trapping and VD/VT . The optimal ventilator strategy for severe obstructive lung disease

TABLE 313-3 Adverse Effects of Hypercapnia
Pulmonary arterial vasoconstriction (possible worsening of right heart failure)
Rightward shift of the oxyhemoglobin curve
Cerebral vasodilation
Increased intracranial pressure
Sympathetic-adrenal stimulation
Reduced cardiac contractility (especially in the presence of β -adrenergic blocking therapy)
aSome effects decrease if cellular pH is corrected.

physiology entails using lower respiratory rates, usually 9–12 breaths/min, and moderate tidal volumes (7–9 mL/kg) to maintain a minute ventilation of ~ 10 L/min; higher minute ventilation usually worsens hyperinflation and can cause barotrauma. To prevent dyspneic patients from driving hyperventilation, deeper sedation and occasionally neuromuscular blockade are necessary until severe bronchial obstruction responds to medical therapy. Although permissive hypercapnia can minimize barotrauma and volume trauma during mechanical ventilation, hypercapnia has adverse effects including increased intracranial pressure, pulmonary artery vasoconstriction, and even depressed cardiac contractility (Table 313-3). The benefits and risks of a hypercapnia ventilatory strategy must, therefore, account for the individual patient's comorbid medical conditions, for example, acute neurologic injury and risk of critical increases in intracranial pressure.

CHAPTER 313 Mechanical Ventilatory Support
COMPLICATIONS OF MECHANICAL VENTILATION

■ **AIRWAY** Endotracheal intubation and mechanical ventilation can lead to several pulmonary and extrathoracic complications, especially when patients remain on mechanical ventilation for >7 days. Upper airway complications from endotracheal tube placement include vocal cord trauma (edema, avulsion, paralysis), tracheal stricture due to granulation tissue, and tracheomalacia. Vocal cord injury can lead to postextubation stridor (PES) and need for replacement of an endotracheal tube. PES risk factors include prolonged (>7 days) or traumatic intubation, large endotracheal tube size, previous episode of PES, and head/neck surgery or trauma. Patients with PES risk factors should have the balloon cuff deflated on their endotracheal tube and assessed for air passing across the balloon (so-called “cuff leak test”). Patients with no cuff leak have an approximate 30% risk of PES and may need further assessment for causes of PES, with endotracheal tube removal delayed until the underlying process is treated.

■ **ADVERSE CARDIOPULMONARY EFFECTS OF POSITIVE-PRESSURE VENTILATION** High positive intrathoracic pressure, such as sustained inspiratory plateau pressures >30 cmH₂O or high PEEP, can cause several manifestations of lung barotrauma, including worsening of acute lung injury, pneumomediastinum, pneumothorax, and even pneumoperitoneum. Although positive-pressure ventilation can improve left-sided heart failure by decreasing left ventricular preload and afterload, right ventricular failure and pulmonary arterial hypertension can worsen due to inadequate right ventricular preload and an increase in right

ventricular afterload and pulmonary vascular resistance; these effects on the right ventricular and pulmonary circulation should be considered when choosing a ventilatory strategy in patients with severe right-sided heart disease. In addition, blunted central venous return can cause upper and lower extremity edema, especially in the setting of aggressive IV fluid resuscitation and vascular leak related to the underlying critical illness.

■ **VENTILATOR-ASSOCIATED PNEUMONIA** Several factors during mechanical ventilation, such as violation of natural airway defenses, sedation with depressed cough, and micro-aspiration, all increase the risk of bacterial entry into the lower respiratory tract and development of pneumonia. Ventilator-associated pneumonia (VAP)

occurs in up to 15% of mechanically ventilated patients and causes death in nearly 50% of patients. VAP is a lower respiratory tract infection that occurs ≥ 48 h after initiating mechanical ventilation and requires the following: (1) new pulmonary opacities on chest x-ray, (2) a clinical change consistent with pneumonia (fever, increased sputum, leukocytosis, or increase in ventilator support, such as increased Fio₂ or PEEP), and (3) positive microbial culture obtained from the lower respiratory tract via deep endotracheal suctioning or bronchoscopy specimen (bronchoalveolar lavage or protected endobronchial brushing). Most VAP pathogens are typical hospital-acquired bacteria including *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and several other enteric gram-negative rods. In cases of suspected VAP, early empiric antibiotic therapy generally requires an intravenous β -lactam with broad gram-negative rod activity, such as piperacillin-tazobactam, cefepime, or ceftazidime. Empiric therapy for methicillin-resistant *S. aureus* (MRSA) with vancomycin or linezolid or for multidrug-resistant enteric gram-negative rods with a carbapenem should depend on local intensive care unit (ICU) infection control data or individual patient risk for these resistant bacteria. If possible, based on respiratory cultures, empiric antibiotic regimens should be narrowed and total treatment duration should be 7 days. Given the significant morbidity and mortality for VAP, prevention strategies are paramount and should be part of standardized care or “bundles.” VAP prevention interventions supported by clinical trial evidence include head-of-bed elevation to at least 30–45° (70% VAP reduction compared to supine position), specialized endotracheal tube use with a suction port above the cuff to minimize aspirated secretions (50% VAP reduction), minimization of ventilator circuit tubing changes (prevents bacterial entry), and hand hygiene before handling the ventilatory circuit. Practices with uncertain value in reducing VAP but still reasonable include limiting deep tracheal suctioning, daily sedation interruption, and routine mouth and dental care.

PART 8 Critical Care Medicine ■ ■ OTHER The systemic physiologic stress associated with mechanical ventilation and necessary adjunctive therapies, such as sedation and neuromuscular blockade, can cause significant extrathoracic complications. The more common disorders include gastrointestinal stress ulcers and bleeding, deep venous thrombosis and pulmonary embolism, sleep disruption and delirium, and critical illness-associated myopathy that sometimes leads to prolonged mechanical ventilation. To minimize the risk of these adverse events, ICUs should institute care bundles including daily interruption of sedatives and assessment for extubation and prophylaxis for deep venous thrombosis. Daily assessment Ready to extubate? Continue mechanical ventilation No Spontaneous breathing trial Passed? Failure/ reintubation Extubation Recurrent respiratory failure or high risk*? No Yes High-flow O₂ or NIV Stable/improved respiratory status? SUCCESS (off mechanical ventilation) Yes **FIGURE 313-4 Algorithm for discontinuing mechanical ventilation.** APACHE-II, Acute Physiology and Chronic Health Enquiry II; BMI, body mass index; COPD, chronic obstructive pulmonary disease; PEEP, positive end-expiratory pressure; NIV, noninvasive ventilation.

LIBERATION FROM MECHANICAL VENTILATION Discontinuing mechanical ventilation and transitioning a patient back to spontaneous breathing is often referred to as ventilator “weaning,” implying dependency on positive-pressure ventilation once started. Although patients on prolonged mechanical ventilation can develop respiratory muscle weakness, this occurs in a minority of patients. Approaching removal of ventilator support as a “wean” extends unnecessary mechanical ventilation time up to 40%. Liberating a patient from mechanical ventilation, therefore, should be more active by frequently assessing a patient’s readiness for spontaneous breathing, determined largely by resolution of the underlying process causing respiratory failure (Fig. 313-4). Important

criteria indicating a patient may be ready for extubation include the following: underlying disease process has improved, patient is awake and largely off sedative medications, $\text{Fio}_2 \leq 0.5$, $\text{PEEP} < 8$ cmH₂O, and $\text{Sao}_2 > 88\%$, stable hemodynamics, and manageable respiratory secretions with adequate cough. These criteria should be assessed daily, and if achieved, patients should have a spontaneous breathing trial (SBT), which is a maneuver wherein positive pressure is set to a minimum to compensate for endotracheal tube resistance (usually 5–7 cmH₂O) and the patient breathes spontaneously from 30 to 120 min. A patient “passes” the SBT if they appear comfortable overall (no marked anxiety or diaphoresis) and have a respiratory rate < 35 , $\text{Sao}_2 > 90\%$, systolic blood pressure between 90 and 180 mmHg, and heart rate change of $< 20\%$. Patients passing an SBT have a $> 70\%$ chance of successful extubation. Incorporating extubation “readiness” screening followed by SBT into a care protocol leads to 25% fewer ventilator days and a 10% decrease in ICU length of stay compared to traditional ventilator weaning. Although many physiologic variables correlate with successful liberation from mechanical ventilation, such as minute ventilation, negative inspiratory force generation, and the respiratory rate-to-tidal volume ratio (Tobin index), overrelying on these measures versus the outcome of an SBT leads to unnecessary delays in extubation. Risk factors for failing extubation even after a successful SBT include age > 65 , congestive heart failure, COPD, Acute Physiology and Chronic Health Enquiry (APACHE-II) score > 12 , body mass index (BMI) > 30 , significant secretions, more than two medical comorbidities, and > 7 days on mechanical ventilation. Patients with these risk factors transitioned immediately after extubation to noninvasive respiratory support using either high-flow oxygen or positive-pressure NIV have significantly lower rates of reintubation and need to resume mechanical ventilation. Although NIV • Underlying process improved • Awake, minimal sedation • $\text{FIO}_2, < 0.5$, $\text{PEEP} < 8$ cmH₂O • $\text{SaO}_2 > 88\%$ • Stable hemodynamics • Minimal secretions/good cough Yes • High-risk for respiratory failure • Age > 65 • Congestive heart failure • COPD • APACHE-II score > 12 • BMI > 30 • Significant secretions • > 2 medical comorbidities • > 7 days on mechanical ventilation Yes No

Revision #1

Created 2026-01-06 16:34:14 UTC by Omar Ayman

Updated 2026-01-06 16:34:14 UTC by Omar Ayman