39 Mechanical Ventilation

Neil R. MacIntyre

Positive pressure mechanical ventilatory support provides pressure and flow to the airways to effect oxygen (O_2) and carbon dioxide (CO_2) transport between the environment and the pulmonary capillary bed. The overall clinical goal of mechanical ventilation is to maintain appropriate levels of O_2 and CO_2 content in the arterial blood while unloading the ventilatory muscles. An equally important goal is to provide this support without harming the lungs. Positive pressure mechanical ventilation can be applied through either an artificial airway or a tight-fitting mask (noninvasive ventilation, discussed in detail in Chapter 62).

DESIGN FEATURES OF MODERN MECHANICAL VENTILATORS

Most modern ventilators use high-pressure gas sources to drive gas flow.[1] Tidal breaths are generated by this gas flow and can be classified regarding what initiates the breath (trigger variable), what controls gas delivery during the breath (target or limit variable), and what terminates the breath (cycle variable).[2] In general, breaths can be initiated (triggered) by patient effort (assisted breaths) or by the machine timer (controlled breaths). Target or limit variables are either a set flow or a set inspiratory pressure. With flow targeting, the ventilator adjusts the pressure to maintain a clinician-determined flow pattern. In contrast, for pressure targeting, the ventilator adjusts flow to maintain a clinician-determined inspiratory pressure. Cycle variables are a set volume, flow, or a set inspiratory time. Breaths can also be cycled if the pressure limits are exceeded. Using this approach, breath delivery algorithms from modern mechanical ventilators can be broken into five basic breaths based upon trigger, target, and cycle criteria: (1) volume control (VC); (2) volume assist (VA); (3) pressure control (PC); (4) pressure assist (PA); and (5) pressure support (PS)[2] (Fig. 39-1).

The availability and delivery logic of the different breath types define the mode of mechanical ventilatory support.[2] The mode controller is an electronic, pneumatic, or microprocessor-based system designed to provide the proper combination of breaths according to set algorithms and feedback data (conditional variables). The five most common modes are volume assist-control (VACV), pressure assist-control (PACV), volume synchronized intermittent mandatory ventilation (V-SIMV), pressure intermittent mandatory ventilation (P-SIMV), and stand-alone pressure support ventilation (PSV).

Novel ventilator designs incorporate advanced monitoring and feedback functions into these controllers to allow continuous adjustments in mode algorithms as the patient's condition changes.[3] The most common of these new feedback designs is the addition of a volume target feedback feature to PACV or PSV, termed the *pressure-regulated volume control* (PRVC) and *volume support* (VS), respectively. This feature adjusts the inspiratory pressure level to achieve the volume target. Feedback mechanisms of pressure-targeted breaths can also incorporate inputs (e.g., end tidal CO_2, minute ventilation, or respiratory rate) in addition to tidal volume to adjust the inspiratory pressure (e.g., the proprietary "SmartCare" system).[4]

ADVERSE EFFECTS OF POSITIVE PRESSURE MECHANICAL VENTILATION

Ventilator-Induced Lung Injury

The lung can be injured when it is stretched excessively by positive pressure ventilation. The most well-recognized injury is an alveolar rupture, presenting as extraalveolar air in the mediastinum (pneumomediastinum), pericardium (pneumopericardium), subcutaneous tissue (subcutaneous emphysema), pleura (pneumothorax), and vasculature (air emboli).[5] The risk of extraalveolar air increases as a function of the magnitude and duration of alveolar overdistention. Thus, interactions of the respiratory system mechanics and mechanical ventilation strategies (e.g., high regional tidal volume and PEEP; both applied and intrinsic) that produce regions of excessive alveolar stretch (i.e., transpulmonary distending pressures more than 40 cm H_2O) for prolonged periods, create alveolar units that are at risk for rupture.

Parenchymal lung injury not associated with extraalveolar air and can also be produced by mechanical ventilation strategies that stretch the lungs beyond the normal maximum capacity (i.e., "volutrauma" associated with transpulmonary distending pressures >30 cm H_2O).[6-10] Pathologically, this manifests as diffuse alveolar damage and is associated with cytokine release[11] and bacterial translocation.[12]

In addition to being caused by simple overstretching of the lung, ventilator-induced lung injury (VILI) may have other determinants. Among these may be excessive tidal stretch (i.e., repetitive cycling of the lungs with tidal volumes larger than the normal 4 to 8-mL/kg ideal body weight)[13] and a shear stress phenomenon that occurs when injured alveoli are repetitively opened and collapsed during the ventilatory cycle.[9,14-16] Moreover, VILI may be worsened by increasing the frequency of excessive lung tidal stretching and from acceleration forces associated with rapid initial gas flow into the lung.[17]

VILI occurs clinically when low-resistance/high-compliance units receive a disproportionately high regional tidal volume in the setting of high alveolar distending pressures (see Fig. 39-2). Concerns regarding overdistention injury is the rationale for using "lung-protective" ventilator strategies that accept less than normal values for pH and O_2 partial pressure in exchange for lower (and safer) distending pressures and volumes.

Cardiac Effects

In addition to affecting ventilation and ventilation distribution, intrathoracic pressure changes resulting from positive-pressure ventilation can affect cardiovascular function.[18,19] In general, as the mean intrathoracic pressure is increased, the right ventricular filling is decreased. This is the rationale for using volume repletion to maintain cardiac output in the setting of high intrathoracic pressure. In addition, high intrathoracic pressures can increase the right ventricular afterload, further compromising cardiac output. Conversely, elevations in intrathoracic pressure can improve left ventricular function because of an

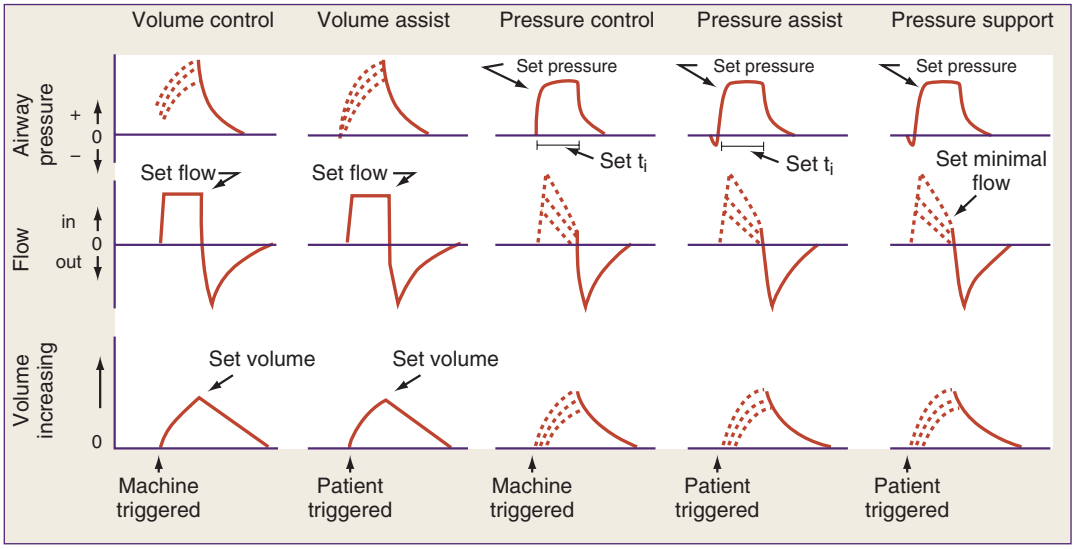

FIGURE 39-1 ■ Airway pressure, flow, and volume tracings over time depicting the five basic breaths are available on most modern mechanical ventilators. Breaths are classified by their trigger, target or limit, and cycle variables. (Adapted from MacIntyre NR. Mechanical ventilatory support. In: Dantzker D, MacIntyre NR, Bakow E, editors. Comprehensive Respiratory Care. Philadelphia: Saunders; 1995.)

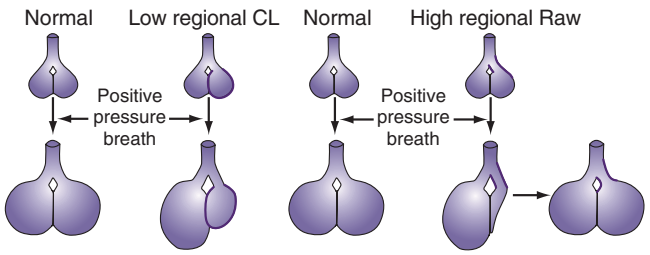

FIGURE 39-2 ■ Schematic effects of the ventilation distribution in two-unit lung models with homogeneous mechanical properties, abnormal compliance distribution, and abnormal resistance distribution. Note that in situations involving inhomogeneous lung mechanics, positive-pressure breaths are preferentially distributed to "healthier" regions of the lung and can produce regional overdistention even when a normal-sized global tidal volume is delivered. C_L, lung compliance; Raw, airway resistance. (Adapted from MacIntyre NR. Mechanical ventilatory support. In: Dantzker D, MacIntyre NR, Bakow E, editors. Comprehensive Respiratory Care. Philadelphia: Saunders; 1995.)

effective reduction in the afterload.[19] Indeed, the sudden release of intrathoracic pressure (e.g., during a ventilator disconnect or spontaneous breathing trial) can sometimes precipitate flash pulmonary edema because of the acute increase in afterload coupled with increased venous return.[20]

Intrathoracic pressures can also influence the distribution of perfusion. The relationship between alveolar and perfusion pressures in the three-zone lung model can help to explain this.[21] Specifically, the supine human lung is generally in a zone 3 (vascular distention) state. However, as the intraalveolar pressures rises, the zone 2 and zone 1 regions can appear, creating high $\dot{V}/\dot{Q}$ units. Indeed, increases in dead space (i.e., zone 1 of the lung) can be a consequence of ventilatory strategies using high ventilatory pressures or in the setting of high PEEP (either intrinsic or applied).

Positive pressure mechanical ventilation can affect other aspects of cardiovascular function. Specifically, dyspnea, anxiety, and discomfort from inadequate ventilatory support can lead to stress-related catechol release, with subsequent increases in myocardial O_2 demands and risk of dysrhythmias. In addition, coronary blood vessel O_2 delivery can be compromised by inadequate gas exchange from lung injury coupled with low mixed venous O_2 partial pressure due to high O_2 consumption demands by the ventilatory muscles.

Patient-Ventilator Dyssynchrony

Patients can interact with all three phases of an assisted breath: (1) trigger, (2) flow delivery, and (3) cycle.[22] Triggering dyssynchronies that manifest as unrecognized or delayed responses to the patient effort can be attributed to insensitive or unresponsive triggering mechanisms or intrinsic PEEP (PEEPi), causing an imposed triggering load on the respiratory muscles.[23] Excessive triggering ("double triggering") may be derived from circuit motion artifacts, premature breath cycling, or the recently described "reverse" triggering observed during controlled breaths.[22] Flow dyssynchrony occurs when the ventilator's flow delivery algorithm is not matched by the patient effort and is more likely to occur during fixed flow breaths (i.e., flow targeted). Cycle dyssynchrony occurs when the breath cycling criteria are either inappropriately short or long on the duration of effort. Patients dyssynchronous with any of these phases will have unnecessary loads placed on their respiratory muscles, thereby increasing the risk of muscle fatigue. Moreover, dyssynchronous interactions produce discomfort and a sense of dyspnea.

There is no doubt that many dyssynchronies are subtle and of little clinical relevance, and significant dyssynchronies that produce severe patient discomfort are frequently cited indications for the administration of sedatives in many ICUs.[22,23] Therefore, this may impact the ventilator duration as high sedation usage is linked to longer ventilator use.

Managing dyssynchronies can be a significant clinical challenge. Setting trigger sensitivity to be as sensitive as possible without auto-triggering is crucial. Judiciously, PEEPe in the setting of a triggering load from PEEPi can be helpful. Careful adjustments of flow magnitude, timing, and patterns (especially the use of pressure-targeted, variable-flow breaths) may help optimize flow and cycle synchrony. Finally, the newer interactive modes found for the proportional assist ventilation (PAV) and neurally adjusted ventilatory assist (NAVA) may offer help to optimize synchrony in the future.[4]

Oxygen Toxicity

Oxygen concentrations approaching 100% are known to cause oxidant injury to the airways and lung parenchyma.[24] The majority of the data supporting this concept, however, are derived from animal studies, and animals and humans often have different O_2 tolerances. It is unclear what the "safe" O_2 concentration or duration of exposure is in sick humans. Most consensus groups have argued that Fio_2 values less than 0.4 are safe for prolonged periods and that Fio_2 values greater than 0.8 should be avoided if possible.

Ventilator-Related Infections

Mechanically ventilated patients are at an increased risk of pulmonary infections for several reasons:[25,26] (1) The natural protective mechanism of glottic closure is compromised by an endotracheal tube. This permits the continuous seepage of oropharyngeal material into the airways. (2) The endotracheal tube itself impairs the cough reflex and serves as a potential portal for pathogens to enter the lungs. This is particularly important if the circuit is contaminated. (3) Airway and parenchymal injury from both the underlying disease and management complications make the lung prone to infections. (4) The intensive care unit (ICU) environment itself, with its heavy antibiotic use and the presence of very sick patients in close proximity, poses a risk for a variety of nosocomial infections, often from antibiotic-resistant organisms.

Preventing ventilator-associated tracheobronchitis and pneumonia is critical because the length of stay and mortality are heavily influenced by their development.[25,26] Handwashing and carefully chosen antibiotic regimens for other infections can have important benefits. Management strategies that avoid breaking the integrity of the circuit (e.g., circuit changes only when visibly contaminated) also appear to be helpful. Finally, the continuous drainage of subglottic secretions may be a simple way of reducing lung contamination with the oropharyngeal material.

APPLYING POSITIVE PRESSURE MECHANICAL VENTILATION

Tradeoffs

To provide adequate support while minimizing VILI, mechanical ventilation goals must involve tradeoffs. Specifically, the need for potentially injurious pressures, volumes, and supplemental O_2 must be weighed against the benefits of gas exchange support. To this end, a revision of the gas exchange goals has occurred over the past decade, including pH goals as low as 7.15 to 7.20, and O_2 partial pressure goals as low as 55 mm Hg, are now considered acceptable if the lung can be protected from VILI.[27] Ventilator settings are thus selected to provide at least this level of gas exchange support while simultaneously meeting two mechanical goals: (1) the provision of enough applied or "extrinsic" PEEP (PEEPe) to enlist the recruitable alveoli and (2) the avoidance of a PEEP–tidal volume combination that unnecessarily overdistends the lung regions at end inspiration. These goals embody the concept of a "lung-protective" mechanical ventilatory strategy,[28] and these principles guide the current recommendations for the specific management of parenchymal and obstructive lung disease.

Managing Parenchymal Lung Injury

Parenchymal lung injury describes disease processes that involve the air spaces and interstitium of the lung. In general, parenchymal injury produces stiff lungs and reduced lung volumes. In addition, the residual functional capacity is reduced, and the compliance curve is shifted to the right.[29] It is important to realize, however, that in all but the most diffuse diseases (e.g., diffuse cardiogenic edema), there are often marked regional differences in the degree of inflammation present and, thus, the degree of mechanical abnormalities that exist.[29-32] This heterogeneity can have a significant impact on the effects of a particular mechanical ventilation strategy. This is because delivered gases will preferentially go to the regions with the highest compliance and lowest resistance (i.e., the more normal regions) rather than to sicker regions with low compliance (see Fig. 39-2). A "normal-sized" tidal volume may thus be distributed preferentially to the healthier regions, resulting in a much higher regional tidal volume and the potential for regional overdistention injury.[30]

Frequency–tidal volume settings for supporting a patient with parenchymal lung injury must focus on limiting end-inspiratory stretching. The importance of this limitation on improving the outcome has been suggested by several clinical trials.[33,34] However, it was most convincingly demonstrated by the NIH ARDS Network trial that showed a 10% absolute reduction in mortality with a ventilator strategy using a pressure <30 cm H_2O and tidal volumes calculated on an ideal body weight of 6 mL/kg compared with 12 mL/kg.[35] As a result, the initial tidal volume settings should begin at a 6-mL/kg ideal body weight. Moreover, strong consideration should be given to further reducing this setting if plateau pressures, adjusted for any effects of excessive chest wall stiffness, exceed 30 cm H_2O. Increases in the tidal volume settings might be considered if there is marked patient discomfort or suboptimal gas exchange, provided that the subsequent plateau pressure does not exceed 30 cm H_2O. Respiratory rate settings are then adjusted to control the pH. Unlike in obstructive diseases (see later), the potential for air trapping in parenchymal lung injury is low if the breathing frequency is less than 35 breaths per minute and may not develop, even at frequencies exceeding 50 breaths per minute.

Although scaling tidal volume to ideal body weight is logical, recent studies have suggested that this may be overly simplistic in situations with extensive heterogeneous injury and little functioning lung ("baby lung"). Under these conditions, even a 6-mL/kg tidal volume may overdistend the remaining functional lung. To address this concern, strategies that scale the tidal volume to a measured functional residual capacity (i.e., targeting "lung strain")[36] or to system compliance (i.e., limiting the tidal volume/compliance as expressed by the "driving pressure" or plateau pressure minus PEEP to <15 cm H_2O)[37] have been proposed. These approaches await outcome studies.

The choice of pressure-targeted or volume-targeted breaths often depends more on clinician familiarity with the two modes than on important clinical differences between them. In general, pressure-targeted breaths are preferable when an absolute pressure limit is desired in the circuit or when the patient effort is very active with variable flow demands. In contrast, volume-targeted breaths are preferable when it is critical to maintain a certain level of minute ventilation. The volume feedback features on pressure-targeted breaths may help combine the advantages of variable flow and a tidal volume target.

Setting the inspiratory time and the inspiratory-expiratory (I:E) ratio in parenchymal injury involves several considerations. The normal I:E ratio is roughly 1:2 to 1:4, and such ratios produce the most comfort and are the usual initial ventilator settings. Assessment of the flow graphic should also be performed to ensure that an adequate expiratory time is present to avoid PEEPi and air trapping. I:E prolongation beyond the physiologic range of 1:1 (inverse ratio ventilation or IRV) can be used as an alternative to increasing PEEPe to improve $\dot{V}/\dot{Q}$ matching in severe respiratory failure.[38] A variation on IRV is airway pressure release ventilation (also known as *biphasic* or *bilevel ventilation*).[39] Airway pressure release ventilation (APRV) incorporates the ability to breathe spontaneously during the long inflation period of a pressure-controlled breath; a feature that may enhance alveolar recruitment and comfort. It must be emphasized, however, that although IRV/APRV strategies have a physiologic appeal, positive outcome studies supporting their use do not exist.[39]

There are both mechanical and gas exchange approaches to setting the PEEP-Fio_2 combination to support oxygenation. Mechanical approaches often use either a static pressure-volume plot to set the PEEP–tidal volume combination between the upper and lower inflection points[40] or stepwise increases in PEEPe to determine the PEEPe level that yields the greatest compliance.[41] A simpler mechanical approach that may reduce inflammatory cytokines involves analyzing

the airway pressure waveform during a set tidal volume with a constant flow breath (i.e., the "stress index").[42] If the pressure waveform demonstrates a steady increase, this implies that no recruitment-derecruitment or overdistention is occurring during the breath. In contrast, if the pressure waveform is concave upward, it suggests that overdistention is occurring; if the pressure waveform is concave downward, it implies derecruitment occurred during the previous exhalation. With any of these approaches, a recruitment maneuver could be used to recruit the maximal number of recruitable alveoli before setting the PEEPe. Fio_2 adjustments are then set as low as clinically acceptable.

Since these mechanical approaches are time-consuming and technically challenging, gas exchange criteria are often used to guide the PEEPe and Fio_2 settings. These generally involve algorithms designed to provide adequate values for the arterial partial pressure of O_2 while limiting PAO plateau pressures and minimizing Fio_2 (Table 39-1).[43] Note that constructing a PEEP-Fio_2 algorithm is usually an empiric exercise in balancing arterial O_2 saturation with Fio_2 and depends on the clinician's perception of the relative "toxicities" of high thoracic pressures, high Fio_2, and low arterial O_2 saturation. It is important to note that recent meta-analyses of three large trials comparing conservative versus aggressive PEEP-Fio_2 tables (mean PEEPe of 7-9 cm H_2O versus mean PEEPe of 14-16 cm H_2O) suggested a benefit to the more aggressive strategies in patients with more severe lung injury (i.e., Pao_2/Fio_2 < 200) and a benefit to the more conservative strategies in less severe lung injury.[43]

Managing Obstructive Airway Disease

Respiratory failure from airflow obstruction is a direct consequence of increases in airway resistance. Airway narrowing and increased resistance leads to two important mechanical changes: (1) the increased pressures required for airflow may overload the ventilatory muscles, producing a "ventilatory pump failure," with spontaneous minute ventilation inadequate for gas exchange, and (2) the narrowed airways create regions in the lungs that cannot properly empty and return to their normal resting volume, and PEEPi is produced.[44] These regions of overinflation create dead space and place the inspiratory muscles at a substantial mechanical disadvantage, which further worsens muscle function. Overinflated regions may also compress healthier regions of the lung, impairing $\dot{V}/\dot{Q}$ matching. Regions of air trapping and PEEPi also function as a threshold load to trigger mechanical breaths.[22,45,46]

Several gas exchange abnormalities can accompany worsening airflow obstruction. First, although there may be transient hyperventilation due to dyspnea in patients with asthma, worsening respiratory failure in those with obstructive lung disease is characterized by a falling minute ventilation as the respiratory muscles become fatigued in the face of airflow obstruction.[47] The result of this clinical situation is termed *hypercapnic respiratory failure*. Second, as noted earlier, regional lung compression and regional hypoventilation produce a $\dot{V}/\dot{Q}$ mismatch that results in progressive hypoxemia. However, alveolar inflammation and flooding are not characteristic features of respiratory failure due to pure airflow obstruction. Thus, shunts are less of an issue than in parenchymal lung injury. Third, overdistended regions of the lungs, coupled with underlying emphysematous changes in some patients, result in capillary loss and increase the dead space. This wasted ventilation further compromises the ability of the inspiratory muscles to supply adequate ventilation for alveolar gas exchange. Emphysematous regions have also reduced the recoil properties that can worsen air trapping. Finally, hypoxemic pulmonary vasoconstriction, coupled with chronic pulmonary vascular changes in some airway diseases, overloads the right ventricle, further decreasing the blood flow to the lung and making dead space worse.[47]

Setting the frequency–tidal volume pattern in obstructive lung disease involves many considerations that are similar to those in parenchymal lung injury. Specifically, tidal volumes should be sufficiently low (e.g., 6-mL/kg ideal body weight) to ensure that the plateau pressure is <30 cm H_2O. However, in obstructive disease, clinicians should be aware that high *peak* airway pressures, even in the presence of acceptable values for plateau pressure, may transiently subject regions of the lung to overdistention injury due to a pendelluft effect (see Fig. 39-2). Similar to parenchymal lung injury, tidal volume reductions should be considered to meet the plateau pressure goals. Tidal volume increases can be considered for comfort or gas exchange, provided that the plateau pressure values do not exceed 30 cm H_2O. The set rate is used to control the pH. Unlike parenchymal disease, however, the elevated airway resistance and often low recoil pressures of emphysema greatly increase the potential for PEEPi and air trapping, which limits the range of breath rates available.

The inspiratory-expiratory ratio in obstructive lung disease is set as low as possible to minimize the development of air trapping. For the same reason, approaches using IRV strategies are almost always contraindicated.

Since alveolar recruitment is less of an issue in obstructive lung disease than in parenchymal lung injury, the PEEP-Fio_2 steps in Table 39-1 should likely be shifted to a conservative strategy that emphasizes Fio_2 for oxygenation support. However, a specific role for applying PEEP in an obstructed patient occurs when PEEPi serves as an inspiratory threshold load on the patient's attempt to trigger a breath. Under these conditions, judicious application of PEEPe (up to 75% to 85% of PEEPie) can "balance" the expiratory pressure throughout the ventilator circuitry to reduce this triggering load and facilitate the triggering process.[45,46] In general, PEEPe below the PEEPi level has little effect on the total PEEP. However, in rare circumstances, small amounts of PEEPe have been shown to reduce PEEPi and air trapping, presumably from splinting open collapsing small airways.[48]

Managing Normal Lungs (Neuromuscular Injury and the Perioperative Setting)

The risk of VILI is thought to be lower in a mechanically ventilated patient with normal lungs because the lung mechanics are often near

TABLE 39-1 NIH ARDS Network PEEP-Fio_2 Tables

CONSERVATIVE PEEP APPROACH

Fio_2	30	40	40	50	50	60	70	70	70	80	90	90	90	1.0	1.0	1.0	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18	20	22	24

LIBERAL PEEP APPROACH

Fio_2	30	30	40	40	50	50	60	60	70	80	80	90	1.0	1.0
PEEP	12	14	14	16	16	18	18	20	20	20	22	22	22	24

Targets: Po_2 55-80, Spo_2 88%-95%. Move up one step if below target, down one step, if above target. Fio_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure (cm H_2O).
Data from the National Heart, Lung, and Blood Institute, National Institutes of Health.

normal, making regional overdistention less likely. More "generous" tidal volumes (e.g., up to 10 mL/kg IBW) are thus often used to improve comfort, maintain recruitment, and prevent atelectasis. Challenging these generous tidal volume practices, however, are a series of recent trials in the perioperative setting demonstrating that limiting plateau pressures and tidal volumes reduces postoperative respiratory complications.[49-51]

Regardless of the tidal volume settings, maximal distending pressures should be monitored and kept as low as possible while still being compatible with the other goals noted earlier. Certainly, plateau pressure should always be kept well below 30 cm H_2O. Low levels of PEEPe are often beneficial in preventing derecruitment (atelectasis) in these patients, who are often supine and incapable of secretion clearance or spontaneous sigh breaths.

Importantly, in the setting of severe CNS injury, concerns regarding the adverse effects of high intrathoracic pressure and high levels of P_{CO_2} may require adjustments to the basic lung-protective strategy (e.g., lower PEEPe settings and higher minute ventilation settings).

Recovering Respiratory Failure: The Ventilator Withdrawal Process

Once the cause of respiratory failure stabilizes and begins to reverse, attention turns to the ventilator withdrawal process. Numerous evidence-based guidelines have focused on the pivotal role of spontaneous breathing trials (SBTs) in determining the need for continued mechanical ventilatory support.[52,53] In general, once a patient has stabilized, has an adequate gas exchange, has low PEEP/F_{IO_2} needs, and is off vasopressors, daily SBTs should be initiated. Importantly, SBTs should be linked to a sedation minimization protocol for maximal success.

In patients comfortably tolerating an SBT for up to 2 h, an assessment should be made to determine if the artificial airway can be removed. In patients failing the SBT, comfortable forms of interactive ventilatory support should be provided until the next attempt at an SBT. This is usually the next day but may be more frequent in patients with rapidly recovering lung function (e.g., post anesthesia or drug overdose). Although the pressure-support mode is often used for this purpose, the pressure-assist control can also fill this role. When using pressure-assist control, the control rate is set quite low (or even to zero), and the inspiratory pressure is titrated to achieve comfort. Similar to pressure support, this approach is patient triggered and pressure targeted but is time cycled as opposed to flow cycling for pressure support.

More detailed discussions on the ventilator discontinuation process can be found in Chapter 63.

CONCLUSION

Positive pressure mechanical ventilatory support is a critical component in the management of patients with respiratory failure. However, it is important to note that this technology is supportive, not therapeutic, and it cannot cure lung injury. Indeed, the best we can hope for is to buy time by supporting gas exchange without harming the lungs.

Positive pressure mechanical ventilation is designed to provide substantial levels of respiratory support. The major goals are to unload the ventilatory muscles and optimize ventilation-perfusion matching to ensure adequate gas exchange. Important complications include ventilator-induced lung injury, cardiac compromise, oxygen toxicity, and patient discomfort. Applying ventilatory support often requires tradeoffs as clinicians attempt to balance gas exchange needs with the risk of these complications. Future innovations cannot focus simply on physiologic endpoints. Rather, innovations should demonstrate benefits in clinically relevant factors, such as mortality, ventilator-free days, barotrauma, and costs. Only then can we properly assess the often bewildering array of new approaches to this vital life-support technology.

KEY POINTS

1. Ventilator breath delivery is characterized by the trigger, target, and cycle variables.
2. The interaction of a positive-pressure breath and respiratory system mechanics is summarized by the equation of motion: Airway pressure = (Flow × Resistance) + (Volume/System Compliance) + PEEP
3. The goal of positive pressure mechanical ventilation is to provide adequate gas exchange while protecting the lung from overdistention and recruitment-derecruitment injury.
4. Positive pressure mechanical ventilation in obstructive lung disease poses the additional risk of producing overdistention from air trapping.

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