Mechanical Ventilation in Emergency Medicine

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Second Edition

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Contents

Introduction

1

Mechanical ventilation is a procedure often performed in patients in the emergency department (ED) who present in respiratory distress. The indications of mechanical ventilation include airway protection, treatment of hypoxemic respiratory failure, treatment of hypercapnic respiratory failure, or treatment of a combined hypoxic and hypercapnic respiratory failure. On some occasions, patients are also intubated and placed on mechanical ventilation for emergency procedures in the ED, such as the traumatically injured and combative patient who needs prompt imaging. However, the initiation of mechanical ventilation requires a great degree of vigilance, as committing to this therapy can affect the patient's overall course.

Historically, mechanical ventilation was not taught as a core component of Emergency Medicine practice. Instead, principles of ventilation have been left to intensivists and respiratory therapists. However, with increasing boarding times in the ED and increased acuity of our patients, emergency physicians are frequently caring for mechanically ventilated patients for longer and longer periods of time. Additionally, the data supporting the importance of good ventilator management in all critically ill patients continues to increase. With the COVID-19 pandemic, the importance of proper ventilator management became all that more important in the ED and beyond.

Compared to many of the other procedures and assessments emergency physicians perform, basic mechanical ventilation management is relatively simple. While there are occasional patients who are very difficult to oxygenate and ventilate, requiring specialist assistance, the vast majority of patients can be cared for by applying straightforward, evidence-based principles. Ventilator management can seem intimidating due to varied and confusing terminology (with many clinicians using synonyms for the same modes or settings), slight variation among brands of ventilators, unfamiliarity, or ceding management to others. The objectives of this text are to:

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1. familiarize ED clinicians with common terms in mechanical ventilation,
2. review key principles of pulmonary physiology relevant to mechanical ventilation,
3. understand interpretation of blood gases as related to the management of the ventilated ED patient,
4. discuss the basic principles of selecting ventilator settings,
5. develop strategies for caring for the ventilated ED patients with ARDS, asthma, COPD, and traumatic brain injury,
6. assess and respond to emergencies during mechanical ventilation.

A few words about the style and function of these educational materials are in order. First, the authors assume that the readers are knowledgeable, experienced clinicians who happen to be new to mechanical ventilation. The explanations of ventilation are deliberately simplified in response to other manuscripts and texts, which may at times overcomplicate the subject. Second, the principles herein are deliberately repeated several times throughout the text, working on the educational principle that presenting the same information in different ways enhances understanding and recall. Third, the goal of these materials is to present key concepts. Readers should know that sophisticated modern ventilators may have backup modes or other safeguards that allow for automated switching of modes or other adaptations for patient safety. The details of this complex ventilation function are beyond the scope of this text. However, it is the authors' contention that a thorough understanding of core principles will allow any emergency clinician to provide evidence-based critical care to their ventilated patients, as well as communicate effectively with their colleagues in critical care and respiratory therapy. As with many aspects of medicine, there are multiple correct ways to present data about mechanical ventilation. In this text, we will use the same method repeatedly to facilitate recall.

For the sake of brevity, this text will not focus on the details of clinical management beyond mechanical ventilation, assuming that clinicians are otherwise familiar with the medical management of the conditions discussed.

Terminology and Definitions

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Ventilator Basics

Control (target) variables are the targets that are set, based on the mode of mechanical ventilation chosen. For example, there are *pressure-controlled* and *volume-controlled* modes of ventilation.

Conditional variables are the dependent variable in mechanical ventilation. For example, in volume-controlled modes of ventilation, the tidal volume is a set parameter, while the pressure is a conditional variable and can vary from breath to breath.

Trigger—the factor that initiates inspiration. A breath can be pressure trigger, flow triggered, or time triggered.

Cycle—the determination of the end of inspiration and the beginning of exhalation. For example, the mechanical ventilator can be volume, pressure, or time cycled.

Physiology Terms

Airway resistance refers to the resistive forces encountered during the mechanical respiratory cycle. The normal airway resistance is ≤5 cmH_2O.

Lung compliance refers to the elasticity of the lungs, or the ease with which they stretch and expand to accommodate a change in volume or pressure. Lungs with a low compliance, or high elastic recoil, tend to have difficulty with the inhalation process and are colloquially referred to as "stiff" lungs. An example of poor compliance would be a patient with a restrictive lung disease, such as pulmonary fibrosis. In contrast, highly compliant lungs, or ones with a low elastic recoil, tend to have more difficulty the exhalation process, as seen in obstructive lung diseases.

Derecruitment is the loss of gas exchange surface area due to atelectasis. Derecruitment is one of the most common causes of gradual hypoxemia in intubated patients and can be minimized by increasing PEEP.

Recruitment is the restoration of gas exchange surface area by applying pressure to reopen collapsed or atelectatic areas of lung.

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Predicted body weight (PBW), or *ideal body weight (IBW)*, is the weight that should be used in determining ventilator settings, never the actual body weight. Lung volumes are determined largely by sex and height, and therefore, these two factors are used in determining predicted body weight. The formula for men is: PBW (kg) = 50 + 2.3 (height (in) – 60) and for women is: PBW (kg) = 45.5 + 2.3 (height (in) – 60).

Phases of Mechanical Breathing

Initiation phase is the start of the mechanical breath, whether triggered by the patient or the machine. With a patient initiated breath, you will notice a slight negative deflection (negative pressure, or sucking). This phase of the respiratory cycle is highlighted in Fig. 2.1.

Inspiratory phase is the portion of mechanical breathing during which there is a flow of air into the patient's lungs to achieve a maximal pressure, the peak airway pressure (PIP or Ppeak), and a tidal volume (TV or VT). Note the red line in Fig. 2.2.

Plateau phase does not routinely occur in mechanically ventilated breaths, but may be checked as an important diagnostic maneuver to assess the plateau pressure (P_{plat}). With cessation of airflow, the plateau pressure and the tidal volume (TV or VT) are briefly held constant, as illustrated in Fig. 2.3.

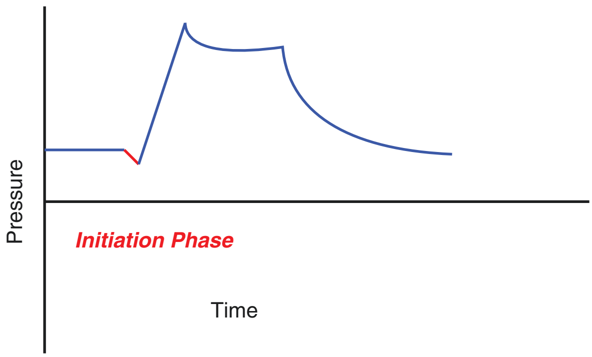

Fig. 2.1 Waveform illustrating initiation phase, or triggering

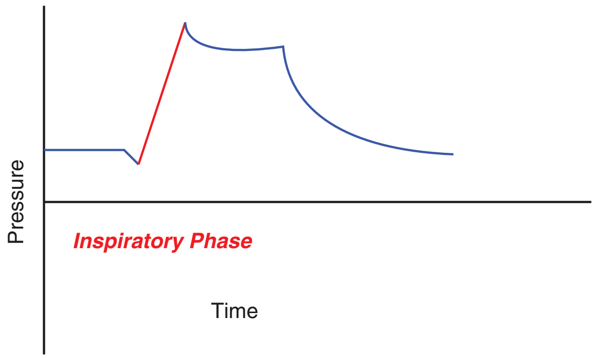

Fig. 2.2 Waveform illustrating inspiratory phase

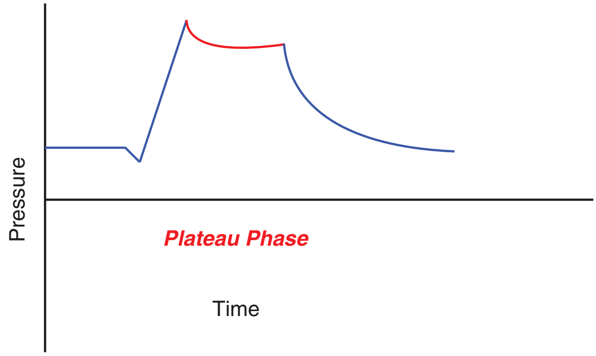

Fig. 2.3 Waveform illustrating plateau phase

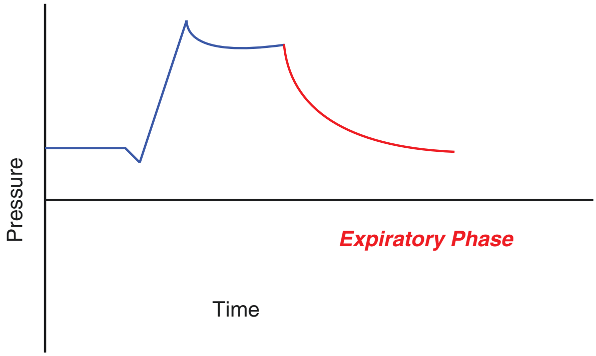

Fig. 2.4 Waveform illustrating expiratory phase

Exhalation is a passive process in mechanical breathing. The start of the exhalation process can be either volume cycled (when a maximum tidal volume is achieved), time cycled (after a set number of seconds), or flow cycled (after achieving a certain flow rate). The expiratory phase is show in Fig. 2.4.

Ventilator Settings

Peak inspiratory pressure (PIP or Ppeak), as shown in Fig. 2.5 (airway pressures), is the maximum pressure in the airways at the end of the inspiratory phase. This value is often displayed on the ventilator screen. Since this value is generated during a time of airflow, the PIP is determined by both airway resistance and compliance. By convention, all pressures in mechanical ventilation are reported in "cmH_2O." It is best to target a PIP ≤ 35 cmH_2O.

Plateau pressure (P_{plat}) is the pressure that remains in the alveoli during the plateau phase, during which there is a cessation of airflow, or with a breath-hold. To calculate this value, the clinician can push the "inspiratory hold" button on the ventilator. The plateau pressure is effectively the pressure at the alveoli with each mechanical breath and reflects the compliance in the airways. To prevent lung injury, the P_{plat} should be maintained at ≤30 cmH_2O. See Fig. 2.5.

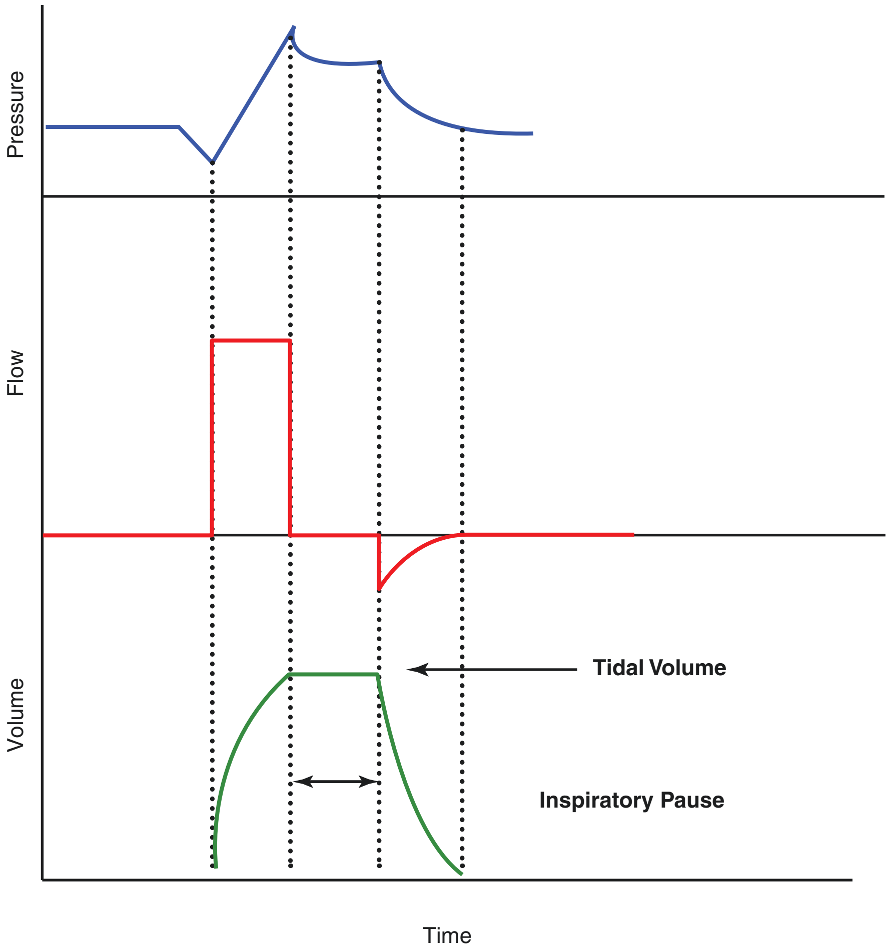

Fig. 2.5 Typical ventilator waveforms illustrating volume, flow, and pressure

Positive end-expiratory pressure (PEEP), as demonstrated in Fig. 2.5, is the positive pressure that remains at the end of exhalation. This additional applied positive pressure helps prevent atelectasis by preventing the end-expiratory alveolar collapse. PEEP is usually set at 5 cmH_2O or greater, as part of the initial ventilator settings. PEEP set by the clinician is also known as *extrinsic PEEP*, or *ePEEP*, to distinguish it from the pressure that can arise with air trapping. By convention, if not otherwise specified, “PEEP” refers to ePEEP.

Intrinsic PEEP (iPEEP), or auto-PEEP, is the pressure that remains in the lungs due to incomplete exhalation, as can occur in patients with obstructive lung diseases. This value can be measured by holding the “expiratory pause” or “expiratory hold” button on the mechanical ventilator.

Driving pressure (ΔP) is the term that describes the pressure changes that occur during inspiration and is equal to the difference between the plateau pressure and PEEP (P_{plat} – PEEP). For example, a patient with a P_{plat} of 30 cmH_2O and a PEEP of 10 cmH_2O would have a driving pressure of 20 cmH_2O. In other words, 20 cmH_2O would be the pressure that exerted to expand the lungs.

Inspiratory time (iTime) is the time allotted to deliver the set tidal volume (in volume control settings) or set pressure (in pressure control settings).

Expiratory time (eTime) is the time allotted to fully exhale the delivered mechanical breath.

I:E ratio, or the inspiratory to expiratory ratio, is usually expressed as 1:2, 1:3, etc. The I:E ratio can be set directly, or indirectly on the ventilator by changing the inspiratory time, the inspiratory flow rate, or the respiratory rate. By convention, decreasing the ratio means increasing the expiratory time. For example, 1:3 is a decrease from 1:2, just like 1/3 is less than 1/2.

Peak inspiratory flow is the rate at which the breath is delivered, expressed in L/min. A common rate is 60 L/min. Increasing and decreasing the inspiratory flow is a means of indirectly affecting the I:E ratio. A patient with a respiratory rate set at 20, who is not overbreathing, has 3 seconds for each complete cycle of breath. If you increase the inspiratory flow, the breath is given faster, and that leaves more time for exhalation. Thus, inspiratory flow indirectly changes the I:E ratio.

Tidal volume (TV or V_T) is the volume of gas delivered to the patient with each breath. The tidal volume is best expressed in both milliliters (ex: 450 mL) and milliliters/kilogram (ex: 6 mL/kg) of predicted body weight, much as one might describe a drug dosage in pediatrics. Clinicians can choose to set the ventilator in a volume control mode, where the tidal volume will be constant for each breath. In pressure control modes, the pressure is constant, but the tidal volume is an independent variable and will vary slightly with each breath. Regardless, every mode of ventilation delivers a tidal volume. Figure 2.5 illustrates the correlation between the tidal volume, the flow of air, and the pressure waveforms. This is similar to what may be seen on a ventilator screen. For a clinical example of similar waveforms from a patient's ventilator screen, please reference Fig. 6.1.

Respiratory rate (RR or f) is the mandatory number of breaths delivered by the ventilator per minute. However, it is important to be mindful that the patient can breathe over this set rate, and therefore one must report both your set RR, or mandatory breaths, and the patient's actual RR, or spontaneous breaths. Both of these values can be found on the ventilator screen. In addition, it is important to remember that the RR is a key factor in determining time for exhalation. For example, if a patient has a RR of 10 breaths per minute (bpm), he will have 6 seconds per breath ((60 seconds/min) / 10 bpm = 6 sec/breath). A RR of 20 bpm, only allows 3 seconds for the entire respiratory cycle.

Minute ventilation (VĖ, Vė, or MV) is the ventilation the patient receives in one minute, calculated as the tidal volume multiplied by the respiratory rate (TV × RR) and expressed in liters per minute (L/min). Most healthy adults have a baseline minute ventilation of 4–6 L/min, but critically ill patients, such as those attempting to compensate for a metabolic acidosis, may require a minute ventilation of 12–15 L/min, or even higher, to meet their demands.

Fraction of inspired oxygen (FiO_2) is a measure of the oxygen delivered by the ventilator during inspiration, expressed at a percentage. Room air contains 21% oxygen. A mechanical ventilator can deliver varying amounts of oxygen, up to 100%.

Ventilator Modes

Conventional Modes of Ventilation

Assist control (AC) is a commonly used mode of ventilation and one of the safest modes of ventilation in the emergency department. Patients receive the same breath, with the same parameters as set by the clinician, with every breath. They may take additional spontaneous breaths, or overbreathe, but every breath will deliver the same set parameters. Assist control can be volume-targeted (volume control, AC/VC) where the clinician sets a desired volume, or pressure-targeted (pressure control, AC/PC) where the clinician selects a desired pressure.

Synchronized intermittent mandatory ventilation (SIMV) is a type of intermittent mandatory ventilation, or IMV. The set parameters are similar to those in AC, and the settings can be volume controlled (SIMV-VC) or pressure controlled (SIMV-PC). Similar to AC, each mandatory breath in SIMV will deliver the identical set parameters. However, with additional spontaneous breaths, the patient will only receive pressure support or CPAP. For example, in SIMV-VC we can set a TV, and as long as the patient is not breathing spontaneously, each delivered mechanical breath will achieve this tidal volume. However, spontaneous breaths in this mode of ventilation will have more variable tidal volumes, based on patient effort and airway factors.

Pressure-regulated volume control (PRVC) is a type of assist control that combines the best attributes of volume control and pressure control. The clinician selects a desired tidal volume, and the ventilator gives that tidal volume with each breath, at the lowest possible pressure. If the pressure gets too high and reaches a predefined maximum level, the ventilator will stop the airflow and cycle into the exhalation phase to prevent excessive airway pressure and resulting lung injury. In this mode of ventilation, the pressure target is adjusted based on lung compliance, to help achieve the set tidal volume. It is important to be mindful of the peak airway pressure, as the set tidal volume may not be achieved if this limit is reached, thereby affecting the patient's minute ventilation.

Pressure support is a partial support mode of ventilation in which the patient receives a constant pressure (the PEEP) as well as a supplemental, "supporting" pressure when the ventilator breath is triggered. In this mode, the clinicians can set the PEEP and the additional desired pressure over the PEEP. However, the peak inspiratory airflow, the respiratory rate, and the tidal volume are all dependent variables and determined by the patient's effort. The patient triggers every breath, and when the patient stops exerting effort, the ventilator stops administering the driving pressure, or the desired pressure over PEEP. Therefore, patients placed on this mode of ventilation must be able to take spontaneous breaths. Most ventilators have predetermined backup modes of controlled ventilation should the patient have persistent apneic episodes.

Noninvasive positive pressure ventilation (NIPPV) refers to two noninvasive modes of ventilation, in which the patient's airway is not secured with an endotracheal tube. Rather, these modes of ventilation are delivered through a tight-fitting facemask or nasal prongs. There are several indications and clear contraindications to these modes of ventilation, as discussed in the text. Both CPAP and BPAP are noninvasive modes of ventilation.

Continuous positive airway pressure (CPAP) is a partial support mode of ventilation, in which the patient received a constant airway pressure throughout the respiratory cycle. The peak inspiratory airflow, respiratory rate, and tidal volume are all dependent variables and determined by the patient's effort. Therefore, the patient must be awake, minimally sedated, able to protect his or her airway, and able to take spontaneous breaths during this mode of ventilation.

Bilevel positive airway pressure (BPAP or BiPAP) is a partial support mode of ventilation, in which the patient receives two levels of airway pressure throughout the respiratory cycle. A high *inspiratory pressure (iPAP)* is similar to the peak airway pressure setting. The lower *expiratory pressure (ePAP)*, similar to PEEP, is clinically apparent at the end of expiration and helps maintain alveolar distention. The patient must be awake, minimally sedated, able to protect his or her airway, and able to take spontaneous breaths during this mode of ventilation.

Unconventional modes of ventilation: There are other modes of ventilation occasionally used in specific circumstances in ICUs, including airway pressure release ventilation (APRV), also referred to as bi-level or bi-vent, high-frequency oscillatory ventilation, proportional assist ventilation (PAV), and neurally adjusted ventilatory assist (NAVA), but these modes are not appropriate in the ED without expert consultation.

Suggested Reading

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3 Review of Physiology and Pathophysiology

Gas Exchange

The diagram in Fig. 3.1 represents a normal cluster of alveoli with a normal capillary, which facilitates the delivery of carbon dioxide (CO_2) and picking up oxygen (O_2) from the alveoli.

Figure 3.1 is highly simplified for conceptual emphasis. However, a slightly more detailed diagram illustrating the role of hemoglobin is important to understand the fundamental concepts of gas exchange (Fig. 3.2).

CO_2 dissolves quite readily, about 20 times faster than oxygen. The dissolved components of carbon dioxide (carbonic anhydrase, hydrogen, and bicarbonate) travel in the blood, and easily cross the capillary wall and into the alveolus. The components of CO_2 transport are indicated in Fig. 3.2 as green dots in the serum.

Because CO_2 crosses so readily into the alveolus from the serum, ventilation occurs readily.

Conversely, the path for oxygen is less simple (Fig. 3.3). Oxygen is transported largely bound to hemoglobin (Hgb) inside the red blood cells. The Hgb in this schematic demonstrate the four binding sites per Hgb molecule inside the red blood cells. Oxygen is represented by small blue dots. The concentration of oxygen is high in the alveoli, and it diffuses down the concentration gradient, into the capillary, into the RBC, and binds with Hgb.

While this binding allows for great efficiency in carrying oxygen, oxygen's solubility is much lower, leading to a slower transit time for oxygen to cross the capillary-alveolar interface.

A small amount of oxygen is carried dissolved in the plasma, but compared to the amount bound to hemoglobin, this amount is trivial. The oxygen-carrying capacity of the blood is described by the equation:

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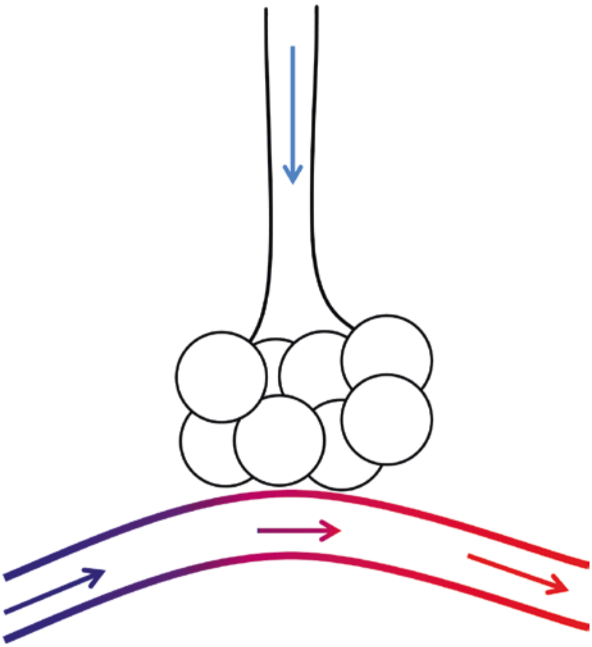


Fig. 3.1 Schematic of normal alveoli and capillary

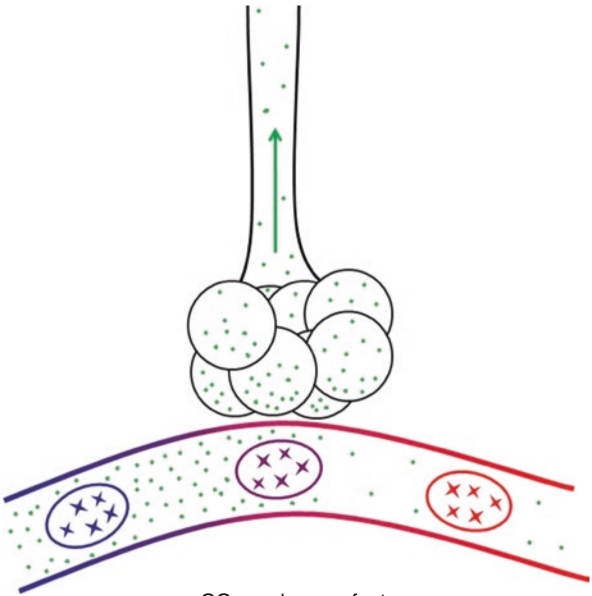
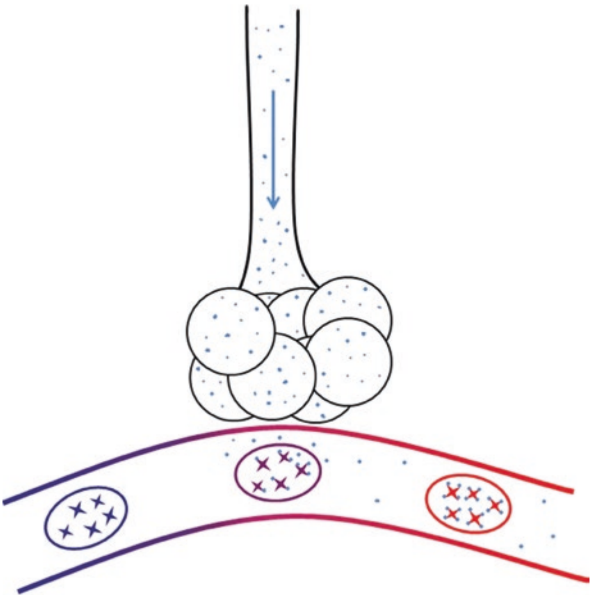


Fig. 3.2 Carbon dioxide uptake by the alveoli. Green dots = blood

Fig. 3.3 Oxygen uptake by capillary and hemoglobin. Small blue dots = oxygen



$$\text{Delivery of Oxygen} = \text{Cardiac Output} \times (\text{Hgb} \times 1.39 \times \text{Oxygen Saturation}) + (\text{PaO}_2 \times 0.003).$$

This equation intuitively makes sense, as the amount of oxygen that can be delivered depends on the amount of Hgb available to carry it, and the amount of oxygen dissolved in the bloodstream is a small fraction of the total.

Issues with Oxygenation

Hypoxemia

There are five broad physiologic causes of hypoxemia: shunting, V/Q mismatch, alveolar hypoventilation, decreased partial pressure of oxygen, and decreased diffusion. Understanding these mechanisms allows the clinician at the bedside to quickly develop a differential diagnosis for hypoxemia and target diagnostics to assess for the precise etiology. We will review each mechanism in detail.

V/Q mismatch is a broad term that indicates that the ventilation and perfusion of lung units are not optimally aligned. Ventilation is the delivery of oxygen and offloading of carbon dioxide at the capillary level. Perfusion is the flow of blood within the lung parenchyma. At the two extremes, lung units can have perfusion without ventilation, (shunt), or ventilation without perfusion, (dead space). With commonly encountered clinical insults, such as pneumonia or ARDS, patients will have components of both and exhibit a range in-between on a micro-level. It can be helpful to consider them each in more detail, however.

Shunt can also occur on a more macro-level. When an area of the lung is perfused, but not ventilated, such that the inspired oxygen cannot reach the alveoli for gas exchange, that results in an intra-pulmonary *shunt*. Examples of shunts are depicted in Figs. 3.4 and 3.5.

In this example, alveoli are lined with fluid, which blocks the diffusion of oxygen and carbon dioxide.

In this example, alveoli are compressed, decreasing the surface area available for diffusion.

There are several different causes of intra-pulmonary shunt, including atelectasis, pneumonia, pulmonary edema, acute respiratory distress syndrome (ARDS), hemothorax or pneumothorax, and hyperinflation or auto-PEEPing. All of these pathological processes prevent effective gas exchange at the alveoli. Intra-pulmonary shunts can also occur with normal lungs. As an example, in patients with cirrhosis, vasodilation can lead to large volumes of blood bypassing the alveoli without proximity to the capillary wall for diffusion, resulting in hypoxemia.

Shunt can also occur in the cardiac system, with patent foramen ovale (PFO) or other congenital or acquired connections between the right and left circulation. At times, the increased stress on the right heart and/or increased intrathoracic pressure from mechanical ventilation may cause a right to left shunt to develop through a previously clinically silent connection, such as a PFO, as seen in Fig. 3.6.

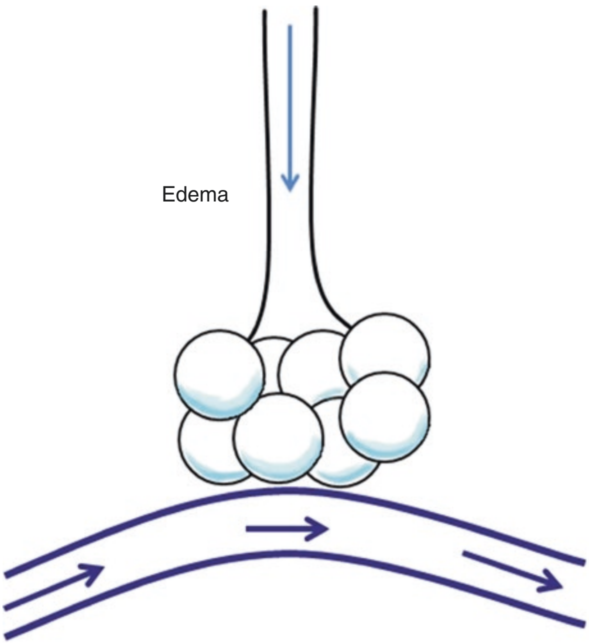


Fig. 3.4 Fluid-filled alveoli inhibiting gas exchange

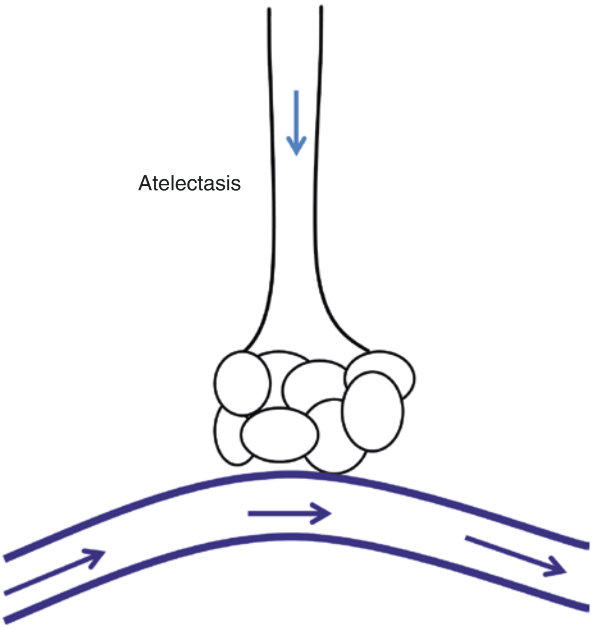


Fig. 3.5 Collapsed alveoli inhibiting gas exchange

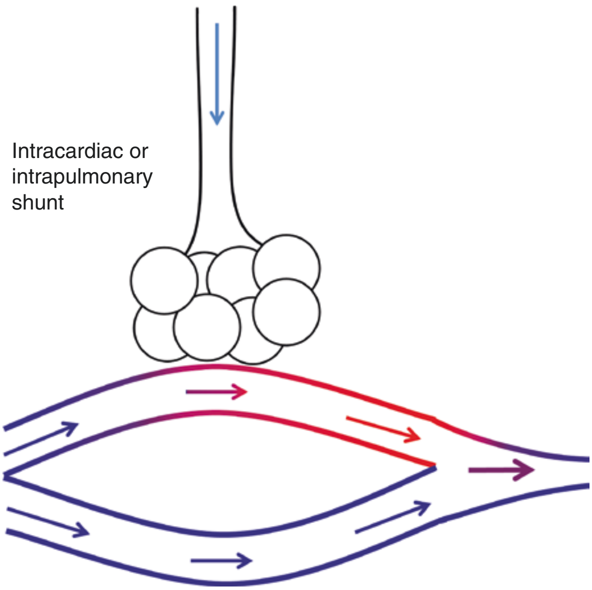


Fig. 3.6 Shunting can occur at the organ-level, with shunts in the heart or lungs. This diagram depicts oxygenated blood that flowed past alveoli, picking up oxygen, mixing with deoxygenated blood that bypassed alveoli

When an area has ventilation, but no perfusion, this is *dead space* (Fig. 3.7). In other words, the airways are functioning normally, but there is disease process in the vasculature. The best example would be a patient in cardiac arrest who is intubated and ventilated, but there is an interruption of chest compressions. Dead space can be

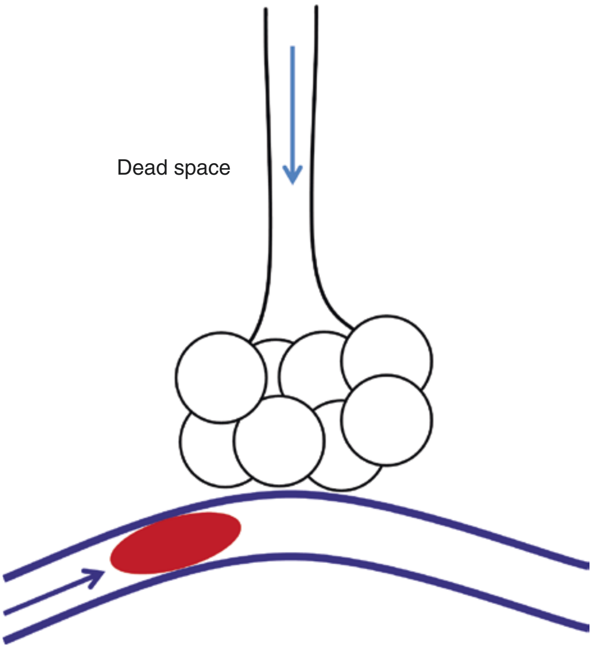


Fig. 3.7 Decreased perfusion inhibiting gas exchange

Table 3.1 Etiologies of hypoxemia from shunts or dead space

Shunts	Dead space
Atelectasis	Pulmonary embolus
Pneumonia	Low cardiac output
Pulmonary edema	Hyperinflation
ARDS	
Pneumothorax/hemothorax	
Hyperinflation	

anatomic and physiologic, such as oxygenation but lack of gas exchange that occurs in the upper airways, such as the trachea. There can also be pathological causes of dead space, such as this diagram of a pulmonary embolism blocking the circulation.

Other examples of dead space include low cardiac output and hyperinflation, as occurs in obstructive lung disease. In diseases such as chronic obstructive pulmonary disease (COPD), there can be a significant level of hyperinflation or auto-PEEP, which can lead to compressive collapse of the capillaries involved in gas exchange, thereby leading to impaired gas exchange. Dead space ventilation can lead to both hypoxia and hypercapnia, due to the lack of diffusion capability and CO_2 retention. Table 3.1 provides clinical examples of shunts as compared to dead space. To think simply, shunt is when gases are not traveling to the air space/bloodstream interface, and dead space is when the bloodstream is not adequately meeting the air spaces.

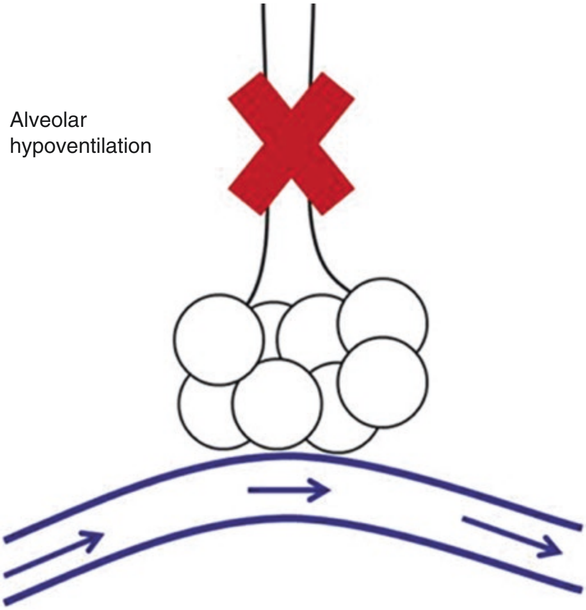


Fig. 3.8 Decreased airflow to alveoli inhibiting gas exchange

There are several other mechanisms of hypoxemia. The next most common mechanism is alveolar hypoventilation. If a patient is not breathing adequately to facilitate gas exchange, such as with an opioid overdose or splinting due to rib fractures hypoxemia can result (Fig. 3.8).

Occasionally, hypoxemia can result from a decreased partial pressure of oxygen. While this commonly occurs at altitude, it is less commonly seen in the ED (Fig. 3.9).

Patients may be hypoxemic due to decreased diffusion. Decreased diffusion can occur with increased interstitial thickness, as occurs in interstitial lung disease (Fig. 3.10), but probably even more commonly, diffusion is decreased due to a loss of surface area, as occurs with emphysema (Fig. 3.11).

Hypoxic Vasoconstriction

When an area of the lung is hypoxic, or there is impairment in the oxygen delivery, the lung tries to optimize ventilation and perfusion ratio (V/Q matching) by means of *hypoxic vasoconstriction*. In this schematic below, the cluster of alveoli is not receiving oxygen. Therefore, the arterioles leading to the alveoli constrict, diverting blood away from this under-ventilated area, in an effort to improve oxygenation (Fig. 3.12).

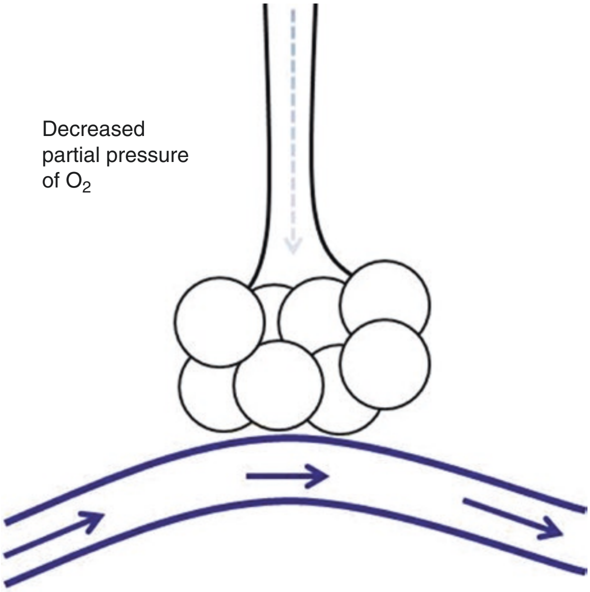


Fig. 3.9 Decreased partial pressure of oxygen inhibiting oxygenation

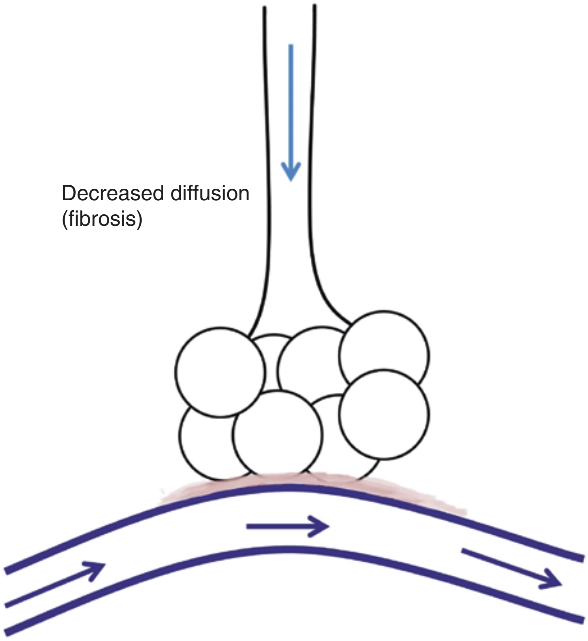


Fig. 3.10 Increased interstitial thickness inhibiting gas exchange

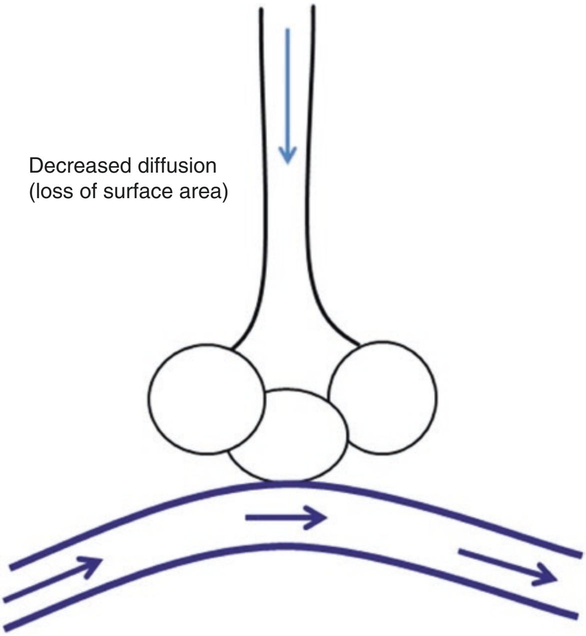


Fig. 3.11 Loss of surface area inhibiting gas exchange

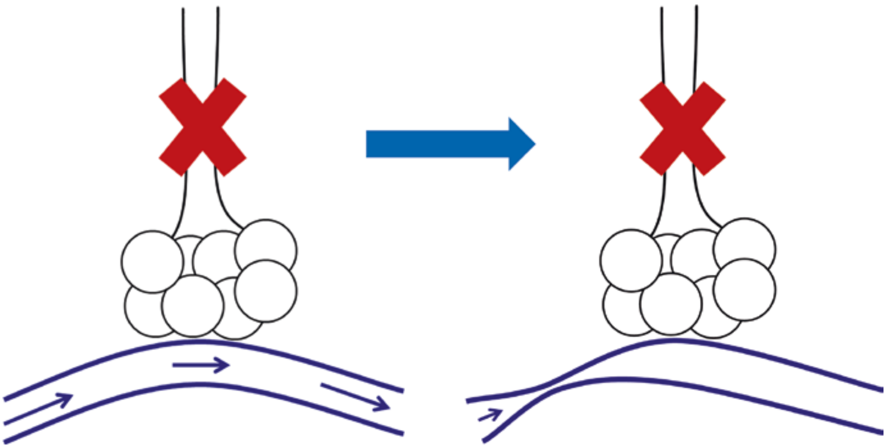


Fig. 3.12 Hypoxemic vasoconstriction leads to decreased perfusion of ineffective lung units

Atelectasis and Derecruitment

Maximizing V/Q matching, by preventing atelectasis, is a key principle in management of respiratory failure. Alveolar derecruitment, or atelectasis, leads to the creation of shunt. Such shunt is physiologic when lying supine to sleep. However, it is compounded by excessive lung weight (as with pulmonary edema), chest wall weight (as with morbid obesity), abdominal contents and distention (as with small

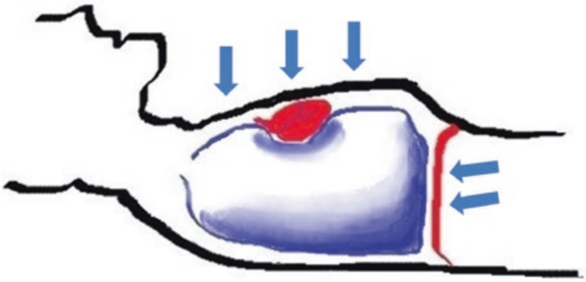


Fig. 3.13 Collapse of many lung units, or atelectasis on a large scale, is derecruitment

bowel obstruction), and even cardiac compression (as with pericardial effusion). The addition of sedation and paralysis to positive pressure ventilation can further augment this derecruitment. The diagram in Fig. 3.13 reflects the pressures leading to compression of the lungs when lying a patient supine – the weight of the heart, the weight of the chest wall, the weight of the abdominal contents, and the weight of the lungs themselves.

Issues with Ventilation

Many of the same issues that lead to problems with oxygenation can lead to problems with ventilation, clinically manifested as hypercapnia. Patients in respiratory failure may present with predominantly hypoxemia, predominantly hypercapnia, or both.

Some of the variability in hypoxemia and hypercapnia arises from the differential transport of oxygen and carbon dioxide as described above. Three of the major etiologies of hypoxemia, dead space, alveolar hypoventilation, and decreased diffusion, also lead to hypercapnia. While a patient may have disproportionate hypercapnia, a patient having a completely normal oxygenation with clinically important hypercapnia is unlikely to occur, as oxygen transport is more involved and therefore more susceptible to physiologic derangements.

The arterial alveolar gradient (A-a gradient) is useful to determine if the patient has a combined oxygenation-ventilation problem or simply an oxygenation problem. Although not necessary for many patients who present in the ED with a clear etiology of respiratory failure (e.g., a clearly evident pneumonia), checking an A-a gradient for patients with hypoxemia of uncertain etiology may help narrow the differential diagnosis.

The A-a gradient is the difference between the alveolar pressure of oxygen (PAO_2) and the pressure of the oxygen in the arterial blood (PaO_2). This measurement requires an ABG.

$$A - a\,\text{gradient} = PAO_2 - PaO_2$$

Table 3.2 Normal and increased A-a gradients

Normal A-a gradient	Increased A-a gradient
Low partial pressure O_2	V/Q mismatch
Alveolar hypoventilation	Cardiac or pulmonary shunt
	Decreased diffusion

The oxygen pressure in the airways physiologically exists at a higher pressure, which facilitates the flow of oxygen down the concentration gradient from the alveoli into the bloodstream.

The PAO_2 is calculated using the alveolar gas equation, or:

$$PAO_2 = PiO_2 - PaCO_2 / 0.8$$

Where the PiO_2 is the pressure of the inspired oxygen.

A normal A-a gradient is <15 mmHg for most patients (Table 3.2).

Compliance and Resistance

Two other important physiologic concepts to review are *compliance* and *resistance*.

Resistance is the impedance of flow in the tubing and airways, and therefore can only occur when there is airflow. According to Ohm's law:

$$\text{Resistance } (R) = \Delta\,\text{pressure} / \Delta\,\text{volume}$$

$$R = (\text{Peak inspiratory pressure} - \text{Plateau pressure}) / \text{Tidal volume}$$

$$R = (PIP - P_{plat}) / (TV)$$

Assuming a constant tidal volume, the resistance equation can be simplified to:

$$R \approx (PIP - P_{plat})$$

Normal airway resistance should be ≤5 cmH20. Resistance is a factor in ventilating all patients, but can become particularly important when ventilating patients with COPD or asthma. The resistance in a system increases with decreasing diameter. While common examples include a very small endotracheal tube (ETT) or bronchospasm leading to narrowing of the airways, recall that a "decrease in the diameter" can also occur at just one point, such as with kinking or biting of the ETT, or a mucous plug in a large airway.

Compliance refers to the distensibility of the system and is the inverse of elastance. In other words, it a measure of the lung's ability to stretch and expand. The more elastic a system, or higher the "recoil," the lower the compliance. A common analogy to understand the concepts of elastance is to analyze the recoil of springs. Imagine a very tightly wound and stiff spring. This spring is difficult to stretch and wants to stay in the coiled position. This spring would have a high elastance and low compliance. Envision a second, loosely coiled spring. Very little force is required to stretch out this spring, and therefore, it has low elastance but high compliance.

Although compliance commonly is used to describe the lung parenchyma, remember that compliance actually involves all components of the system. In other words, a patient with pulmonary edema may have low compliance due to an issue with the lung parenchyma, but another patient may have a similarly low compliance due to severe chest wall stiffness after a third-degree burn. Clinically, knowing the exact cause of decreased compliance in a given patient can be challenging. Physicians should not, therefore, always assume that it is always related to "stiff lungs."

The *peak inspiratory pressure (PIP)* represents pressures in the entire airway system, and it is a measure of both the resistance and compliance. The *plateau pressure (P_{plat})*, which is measured when there is an absence of airflow during the plateau phase of breathing, is the reflection of the pressure delivered to the alveoli and the compliance of the system.

In the schematic shown in Fig. 3.14, the top "lungs" are healthy. The lungs on the left have a resistance problem, or impairment in air flow. The lungs on the right have a compliance problem, or impairment in stretch and recoil. In this diagram, both figures could have elevated peak inspiratory pressures (PIP), due to the excess pressure generated in the system. However, only the right-hand figure would have an elevated plateau pressure (P_{plat}), since this process occurs when there is absence of air flow.

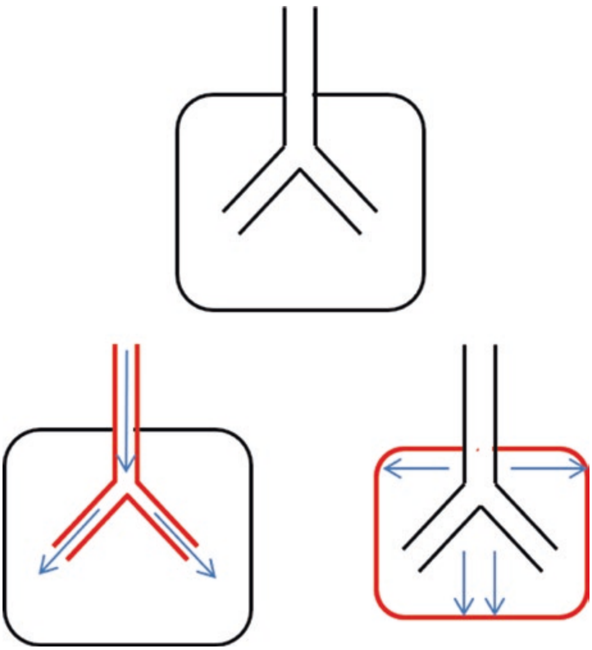


Fig. 3.14 Resistance to flow in airways versus decreased distensibility of the entire respiratory system

$$\text{Compliance } (\text{C}) = \Delta \text{ volume} / \Delta \text{ pressure}$$

$$\text{C} = \text{Tidal volume} / \text{Plateau pressure} - \text{Peak inspiratory pressure}$$

$$\text{C} = (\text{TV}) / (\text{P}_{\text{plat}} - \text{PEEP})$$

Therefore, when trouble shooting high pressure ventilator alarms, two values are needed. The peak airway pressure (PIP) should be displayed on the ventilator screen, while the plateau pressure (P_{plat}) is obtained by holding the "inspiratory hold" or "inspiratory pause" button on the ventilator. An elevated PIP and normal P_{plat} is indicative of increased airway resistance. An elevated PIP and elevated P_{plat} is indicative of an abnormal compliance. Determining whether the patient has a resistance problem or a compliance problem can assist in the differential diagnosis of respiratory failure in the ED, as outlined in Table 3.3.

Atelectasis, or collapse of alveoli and decruitment, is another key physiologic concept in mechanical ventilation. Atelectasis has multiple detrimental effects in ventilated patients. First, atelectasis decreases the surface area for gas exchange. Atelectasis also worsens compliance. Consider blowing up a small party balloon. To start to open the balloon, a large amount of pressure is required. Once the balloon starts to inflate, blowing it up further is easy, until it reaches the point of overdistention. So maintaining alveoli at a minimal "open" state allows for ease of opening pressure. Atelectasis leads to shunt and can cause impaired oxygenation.

Air trapping, also referred to as *breath-stacking*, can lead to the development of *auto-PEEP*, *or intrinsic PEEP (iPEEP)*. These pressures should be differentiated from applied PEEP, or extrinsic PEEP (ePEEP). ePEEP refers to the additional end-expiratory positive pressure set during mechanical ventilation to prevent alveolar collapse and recruitment. In contrast, auto-PEEP, or iPEEP, is a pathophysiological process that can occur when the ventilator initiates the next breath prior to complete exhalation. While this is most common in patients with prolonged expiratory phases,

Table 3.3 Characteristics of high resistance and abnormal compliance

High resistance High PIP, low/normal P_{plat}	Abnormal compliance High PIP, high P_{plat}
Kinked/obstructed ETT	Mainstem intubation
Mucus plugging	Atelectasis
Bronchospasm	Pulmonary edema
ETT too narrow (small)	ARDS
Coughing	Hemo−/pneumothorax
Obstructive lung disease	Pneumonia
	Bronchospasm (obstructive lung disease)
	Pulmonary fibrosis (restrictive lung disease)
	Obesity
	Abdominal compartment syndrome
	Circumferential burns of the chest
	Scoliosis
	Supine position

such as asthma or COPD, it can also occur in patients who have a fast respiratory rate or those who are being ventilated with large tidal volumes. The amount of auto-PEEP can be measured by pressing the "expiratory hold" or "expiratory pause" button on the ventilator. When this button is pressed, the ventilator will display the total PEEP. The auto-PEEP is the difference between the total PEEP and the set PEEP.

$$\text{Auto} - \text{PEEP}\left(\text{iPEEP}\right) = \text{Total PEEP} - \text{ePEEP}$$

The schematic in Fig. 3.15 represents the effects of air trapping. The red arrows represent increasing levels of iPEEP. Please note that this diagram is for illustration purposes only and does not represent the expected tracings on actual ventilator screens.

Air trapping, or auto-PEEP, can lead to significant adverse cardiopulmonary effects. The increased intrathoracic pressure from auto-PEEP can decrease venous return and lead to hemodynamic instability, even cardiac arrest in severe cases. The increased pressures may also result in a pneumothorax or pneumomediastinum. Additionally, air trapping can lead to ineffective ventilation due to collapse of the capillaries responsible for gas exchange, with worsening hypercarbia and hypoxemia. While this may seem like a paradox, as one may assume that increasing the minute ventilation, or moving more air, will improve ventilation, there is a limit to the beneficial effects. Once the lungs are overdistended, gas exchange is ineffective. In these circumstances, allowing the patient sufficient time to exhale can decrease CO_2 retention.

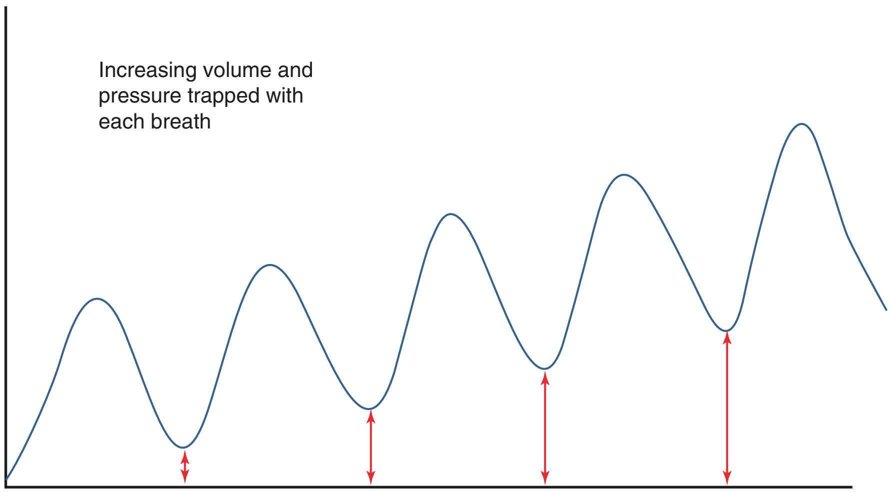


Fig. 3.15 Conceptual illustration of air trapping

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4 Blood Gas Analysis

Care of the patient with respiratory failure is multifaceted. Mechanical ventilation, whether noninvasive or invasive, will be optimized when you monitor patient progress closely. This principle is beneficial for treating patients with primary respiratory, cardiac, or neurologic injury alike. The arterial blood gas (ABG) is the optimal measurement tool to evaluate oxygenation and ventilation for any patient. In some cases, one ABG may be preferred in that it establishes the initial PaO_2 to FiO_2 ratio and allows a comparison of the $PaCO_2$ to end-tidal CO_2 ($ETCO_2$) measurements to provide ongoing monitoring. In other cases, where oxygenation does not need to be determined precisely, a venous blood gas (VBG) will be adequate. This chapter will review when each of these measurement tools is needed and how to read each one.

ABG

ABG is the most accurate and the most invasive measurement of oxygenation and ventilation. It is prudent to consider less invasive measurements, such as VBG or end-tidal CO_2, if adequate. The potential complications of an arterial puncture or indwelling arterial line include thrombotic or embolic vascular occlusion, bleeding, vasospasm, infection, and of course pain. However, once a patient is intubated, they have already undergone a very invasive procedure, so the additional procedure of an ABG may be minimal by comparison.

ABG results are often misinterpreted and thus become less useful. Following is a simple stepwise method to use for interpretation of ABG results and will optimize the use of respiratory adjuncts for oxygenation and ventilation. The first concept to embrace is that there are two body systems at play when interpreting an ABG—respiratory and metabolic. These two systems work to maintain pH; if there is a respiratory issue, the metabolic system will compensate and keep the pH in a normal range by regulating acid-base balance through renal excretion or retention. If there is an underlying metabolic issue, the respiratory system will likewise

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compensate to maintain pH by adjusting minute ventilation. In general, the respiratory system can change pH more rapidly than the metabolic system.

Different Types of Respiratory Failure

Type 1 (hypoxemic—problem with lung or lung vasculature) respiratory failure exists when oxygen levels are low, and $PaCO_2$ is normal or low. This occurs when gas exchange is not happening adequately at the interface between alveoli and capillaries, such as in congestive heart failure, pneumonia, or pulmonary embolus.

Type 2 (hypercapnic—problem with ventilation) respiratory failure exists when $PaCO_2$ is high, and PaO_2 is normal or low. This happens when ventilation is not adequate, such as in opiate overdose.

Types 1 and 2 can both be present, such as in the case of severe COPD. In this case, $PaCO_2$ would be high because ventilation is not adequate, but PaO_2 is also not contributing enough to compensate.

Stepwise Interpretation

1. Look at pH—does it represent acidosis or alkalosis?
2. Look at $PaCO_2$—is it trying to compensate for the pH or contributing to the pH?
   (a) If the pH is low and the $PaCO_2$ is low, then the respiratory system is trying to compensate for a metabolic acidosis.
   (b) If the pH is low and the $PaCO_2$ is high, then the respiratory system is contributing to the acidosis.
   (c) If the pH is high and the $PaCO_2$ is high, then the respiratory system is trying to compensate for a metabolic alkalosis.
   (d) If the pH is high and the $PaCO_2$ is low, the respiratory system is contributing to the alkalosis.
3. Look at the base component.
   (a) Is the HCO_3 high or low? If the pH and $PaCO_2$ suggested a metabolic problem, the HCO_3 should be high. If the problem is respiratory, then the HCO_3 will help to figure out if the metabolic system has had enough time to compensate.
   (b) Is there an excess of base (elevated BE)? This indicates that the metabolic system has been working on the problem and suggests a more chronic problem. If the BE is normal (−3 to +3), then the metabolic system hasn't kicked in.
4. Look at the oxygen—this is where we differentiate between types 1 and 2.
   (a) If the PaO_2 is low and $PaCO_2$ is normal, this is type 1.
   (b) If the $PaCO_2$ is high, this is at least a type 2 but can also have a component of type 1, that is, ventilation is clearly an issue, but is there also something wrong with the lungs or lung vasculature contributing? To figure this out, calculate the alveolar-arterial (A-a) gradient. If the A-a gradient is high, this will tell you that there is a type 1 component or problem with the lungs/lung vasculature contributing to the problem.

(c) Here is the formula for A-a gradient:

$$\mathbf{A - a\ gradient = PAO_2 - PaO_2}$$

(The oxygen pressure in the airways physiologically exists at a higher pressure, which facilitates the flow of oxygen down the concentration gradient from the alveoli into the bloodstream.)

The PAO_2 is calculated using the alveolar gas equation, or:

$$PAO_2 = PiO_2 - PaCO_2 / 0.8$$

where the PiO_2 is the pressure of the inspired oxygen.

A normal A-a gradient is <15 mmHg for most patients.

ABG Versus VBG

Having acknowledged the invasiveness and potential complications of the ABG, when can information from a VBG be an adequate substitute? Part of this answer depends on the underlying pathophysiology. For patients in shock or with a mixed acid-base disorder, a VBG is not reliable for clinically useful information. For other patients, there is a good correlation between ABG and VBG for pH, HCO_3, base excess, and lactate levels. PCO_2, PO_2, and potassium levels do not correlate well with VBG; thus, if your patient's situation requires monitoring of these levels, an ABG is indicated. In the case of PCO_2, a single VBG level may help determine hypercarbia as a cause of respiratory depression.

Suggested Reading

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5 Noninvasive Respiratory Support

The clinician should first assess whether the patient has an oxygenation problem or a ventilation problem. Many patients will have both simultaneously. Determining which problem is more acute will help determine the next appropriate steps in support. Please note that in patients with airway compromise, profoundly altered mental status, or severe shock, this modality is contraindicated, and airway protection with endotracheal intubation is indicated.

Oxygen Support

Many patients who present with hypoxemia can be adequately supported by supplemental oxygen. Patients should be given only the minimal concentration of oxygen needed to maintain a desired oxygen level, as hyperoxia, or too much oxygen, is increasingly appreciated as a risk factor for poor outcomes [1].

High Flow Nasal Cannula

High flow nasal cannula (HFNC) is an excellent means of supporting hypoxemic patients [2]. As illustrated in Fig. 5.1, a typical nasal cannula can provide up to 6 L/min of supplemental oxygen. Each additional L/min provides about 4% extra oxygen. For example, increasing from 2 L to 6 L will increase the FiO_2 from approximately 28% to 44%. HFNC, conversely, can provide about 45–60 L/min, depending upon the variations of the setup. Whereas the typical nasal cannula provides additional oxygen blended with the ambient air, the HFNC has a blender attached to the apparatus. This means that HFNC has two components, the L/min delivered and the percentage of oxygen delivered. Figure 5.1 illustrates the differences between flow and percent oxygen for these mechanisms.

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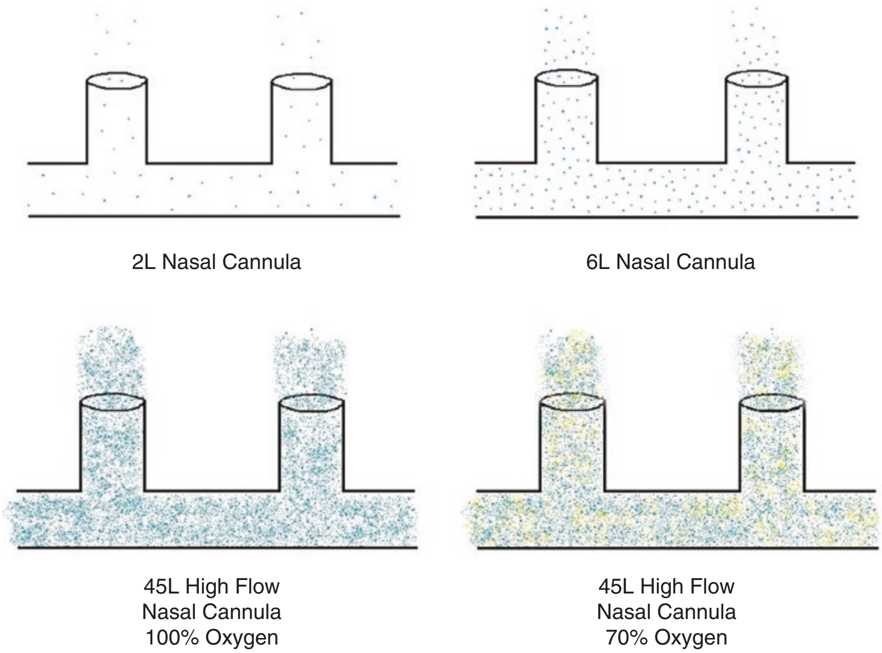

Fig. 5.1 In this illustration, the blue dots represent theoretical oxygen delivery. A small amount of oxygen is delivered and mixed with ambient air in the typical nasal cannula, in the top two figures. The bottom two depict HFNC, showing the increased flow as well as the ability to blend oxygen and air at desired concentrations

Table 5.1 Contraindications to HFNC

Contraindications to HFNC
Airway compromise
Facial trauma
Other indication for intubation
Altered mental status
Severe shock
Primarily hypercapnic respiratory failure

HFNC not only provides the option of delivering a high concentration of oxygen (90–100%) but also provides a level of positive pressure, given the higher flows, which is somewhat mitigated by the lack of a closed system. This positive pressure and associated CO_2 washout are helpful to some degree in hypercapnic respiratory failure, making HFNC an excellent initial option for respiratory support. Table 5.1 lists contraindications for HFNC.

Noninvasive Positive Pressure Ventilation

Noninvasive positive pressure ventilation (NIPPV) is one of the most important advances in the emergency and critical care of patients with respiratory failure. Numerous studies have demonstrated improved outcomes for patients with respiratory failure from COPD and congestive heart failure (CHF) using noninvasive ventilation [3–5].

As opposed to invasive ventilation after placement of an endotracheal tube (ETT), NIPPV is delivered via a tight-fitting face mask or nasal prongs. There are several indications for NIPPV, as it is a highly effective method to oxygenate and ventilate many patients. However, there are a few key contraindications. Patients must be awake and able to protect their airway, as this is not a definitive airway. If the patient is too obtunded to remove the mask, should they vomit or have any other threat to the airway, they should not be placed on NIPPV. Additionally, nausea and vomiting are contraindications, due to the risk of aspiration. Facial trauma, precluding the tight-fitting mask, is a contraindication, as is recent GI surgery (such as a partial gastrectomy) that would put recently placed suture lines at risk. These contraindications are outlined in Table 5.2.

There are two forms of NIPPV: continuous positive pressure ventilation and bi-level positive airway pressure.

Continuous positive pressure ventilation (CPAP) is a continuous positive pressure that is delivered throughout the respiratory cycle and, along with the FiO_2, assists with oxygenation by recruiting alveoli, preventing alveolar collapse, and decreasing the work of breathing. In function, CPAP is analogous to positive end-expiratory pressure (PEEP) for an intubated patient. The difference between CPAP and PEEP is one of nomenclature, as the PEEP is only measurable at the end of expiration.

In patients with CHF, CPAP can increase intrathoracic pressure to decrease venous return and therefore reduce lung congestion. In addition, this positive pressure can also decrease the afterload on the left ventricle, leading to increased stroke volume and cardiac output. CPAP is primarily used in the treatment algorithm of patients with hypoxemic respiratory failure, or those who need the additional positive pressure to assist with alveolar recruitment.

Table 5.2 Contraindications to noninvasive ventilation

Contraindications to noninvasive ventilation
Obtundation, inability to remove mask
GI pathology with vomiting or high risk for vomiting
Recent ENT or GI surgery
Airway compromise
Facial trauma
Other indication for intubation
Altered mental status
Severe shock
Severe hypoxemic respiratory failure

Bi-level positive airway pressure (BPAP or BiPAP) is another mode of NIPPV, which provides two different levels of pressure throughout the respiratory cycle. The higher pressure, or the inspiratory peak airway pressure (IPAP), is analogous to the PIP of invasive ventilation. A second lower pressure, the expiratory peak airway pressure (EPAP), is similar to the CPAP described above or PEEP applied in invasive mechanical ventilation. Providing these pressures, in addition to the FiO_2, assists in improving the patient's oxygenation. The difference between the IPAP and EPAP serves as the driving pressure and assists with ventilation. In contrast to CPAP, which is beneficial in hypoxemia, BPAP is useful in patients with hypoxemic and hypercapnic respiratory failure. Figure 5.2 illustrates a typical BPAP ventilator screen.

Where bi-level positive airway pressure differs from CPAP is that once a patient triggers a breath, the machine will provide additional, supportive pressure, or the inspiratory positive airway pressure (IPAP.) By assisting the patients with IPAP, BPAP is a great tool for patients with poor ventilation, such as COPD patients. The clinician can set both the IPAP and the EPAP with BPAP, based upon the patient's

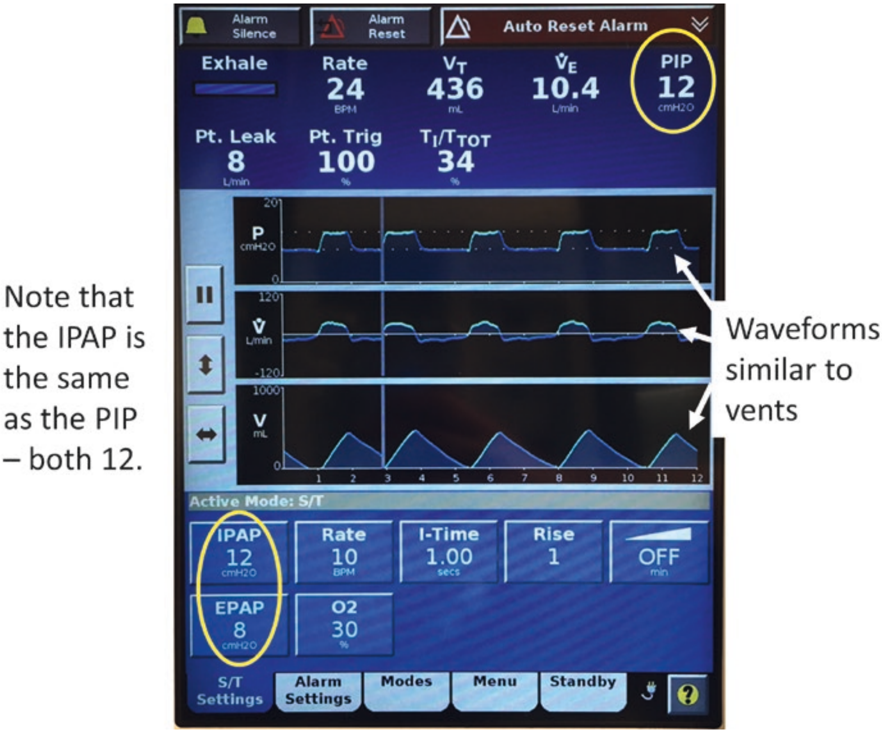

Fig. 5.2 Typical screen for BPAP, highlighting the IPAP, EPAP, and the peak inspiratory pressure, the PIP. By convention, with noninvasive ventilation, the IPAP and the PIP are the same. The waveforms are similar to those of invasive mechanical ventilation. Please refer to Figs. 2.5 and 6.1 for additional examples

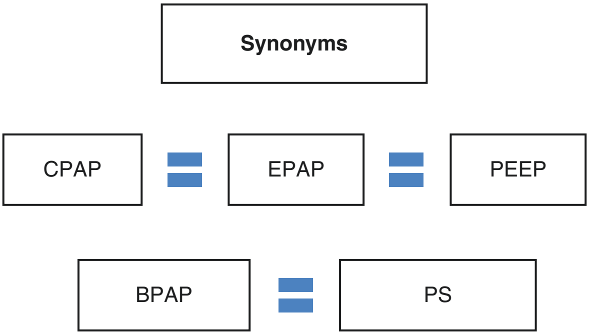

Fig. 5.3 Although several terms are used for the same principles, the concepts are simple. Continuous positive airway pressure (CPAP), expiratory positive airway pressure (EPAP), and positive end-expiratory pressure (PEEP) all refer to a baseline positive pressure, over which the patient breathes. Bi-level positive airway pressure (BPAP) and pressure support (PS) are both modes of ventilation in which a patient receives an additional pressure over the baseline pressure to support their ventilation. By convention, BPAP refers to this pressure being provided via a mask, and PS refers to this pressure being provided via an endotracheal tube

needs. In this way, BPAP is very similar to pressure support, discussed in detail in this chapter. Figure 5.3 demonstrates the multiple synonyms that represent the same concepts.

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6 Modes of Invasive Mechanical Ventilation

Modes of Invasive Ventilation

As already illustrated, the terminology used for mechanical ventilation can be confusing, as many clinicians use various terms for the same settings. The "*mode*" of ventilation simply refers to the way the ventilator is set to interact with the patient. A key distinguishing factor among modes is whether the patient can alter the breath they receive or whether the ventilator will administer the same breath each time, regardless of the patient's effort.

Assist control (AC) is one of the most commonly used modes of ventilation. AC can be set to target (control) either a pressure or a volume, as described in further detail below. In assist control, the clinician sets the independent variable (tidal volume or pressure), the respiratory rate, the positive end-expiratory pressure (PEEP), and the FiO_2. If the patient exerts no respiratory effort, she/he will receive the preset mandatory breaths. If the patient starts to breathe spontaneously, or "triggers" the ventilator, the ventilator will deliver that identical breath with the preset controlled parameter. This allows the patient to "overbreathe," but she/he cannot alter the other clinician-set properties of the breath. For example, if a patient is set to receive 400 mL per breath in AC volume-controlled ventilation, at a flow of 60 L/min, with a respiratory rate of 12 breaths per minute, this is what the patient will receive if they make no efforts to breathe. If the patient is then less sedated and starts to make respiratory efforts, he or she can increase the respiratory rate, and each breath will still have approximately 400 mL delivered at a flow of 60 mL/min with each additional spontaneous breath.

In Fig. 6.1, the flow curve is on the top line and the pressure curve on the bottom line. Note that every waveform is identical. Also note that there is no downward deflection at the initiation of each breath, indicating that these are machine-triggered breaths.

As we continue to review ventilator screenshots, it is important to start recognizing patterns, as the placement of the volume, flow, and pressure curves can vary based on clinician preference and do not reflect patient physiology.

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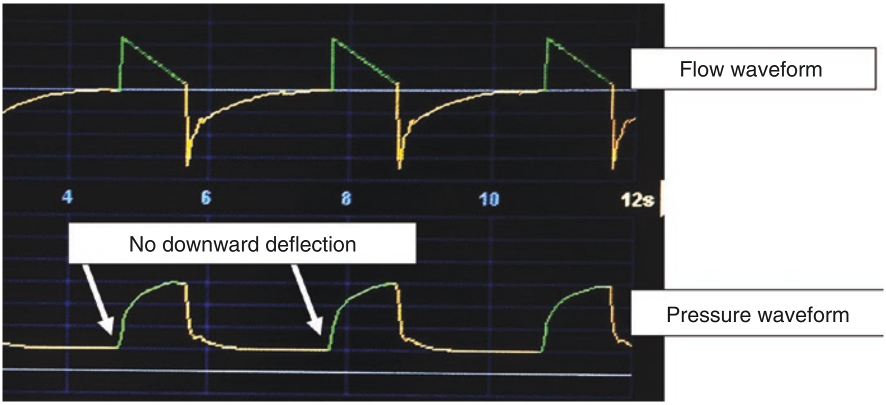

Fig. 6.1 Illustration of typical volume control waveforms for pressure and flow

Synchronized intermittent mandatory ventilation (SIMV) involves components of both AC and PS. A respiratory rate is set in SIMV, but it is usually a low rate, such as eight to ten breaths per minute. The patient will receive those "mandatory" breaths, and they will receive the set breath parameters, with a set volume or pressure, rate, and flow or inspiratory time, as determined by the clinician, just as in AC. However, in between those mandatory breaths, the patient can take additional spontaneous breaths with pressure support, allowing them to vary their breathing pattern. This mode was previously used as a weaning mode, but studies have shown that it offers no benefit over other modes.

Pressure support (PS or PSV) is a partially supported or spontaneous, pressure-controlled mode of ventilation for an intubated patient. In this mode, there is no set respiratory rate or tidal volume, and the patient must be awake enough to trigger each breath. The patient receives a set baseline pressure, the PEEP, and, with the triggering of the breath, an additional, supportive pressure above that baseline to help overcome the resistive airway forces and decrease the work of breathing. The clinician sets the PEEP and the supporting pressure.

The other significant difference is that in pressure support, the ventilator can sense when the patient stops exerting effort for the breath. Once the flow drops to a preset limit (usually 25%), the ventilator stops providing the additional pressure support for that breath. In this way, the patient has more control over the breathing pattern.

Figure 6.2 shows a ventilator screenshot of a patient breathing with PSV. Note the downward deflection at the initiation of each breath, indicating that the patient triggered the breath. Also note that in contrast to the last diagram of a patient breathing on AC ventilation, the patient on PSV generated flow waveforms has subtle variety in shape, size, and rhythm, because the patient determines each breath. However, the pressure waveforms on the top line are constant across these five breaths, because the ventilator is providing that maximum pressure, as dialed in by the clinician. Finally, note that in this figure, the pressure waveform is now on top

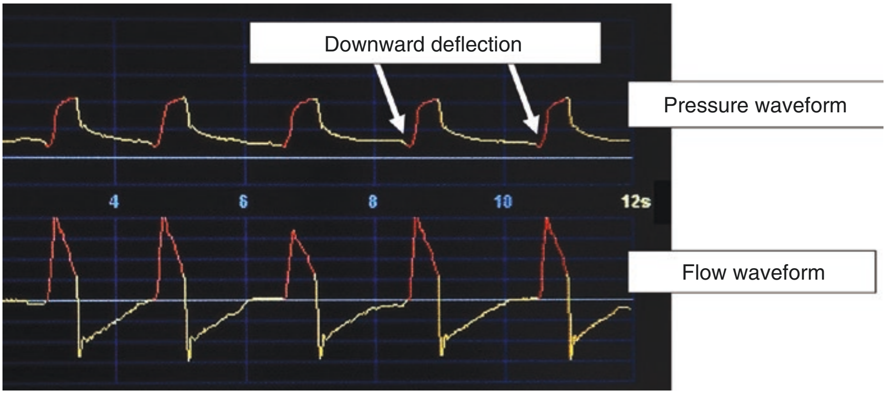

Fig. 6.2 Illustration of typical pressure support waveforms

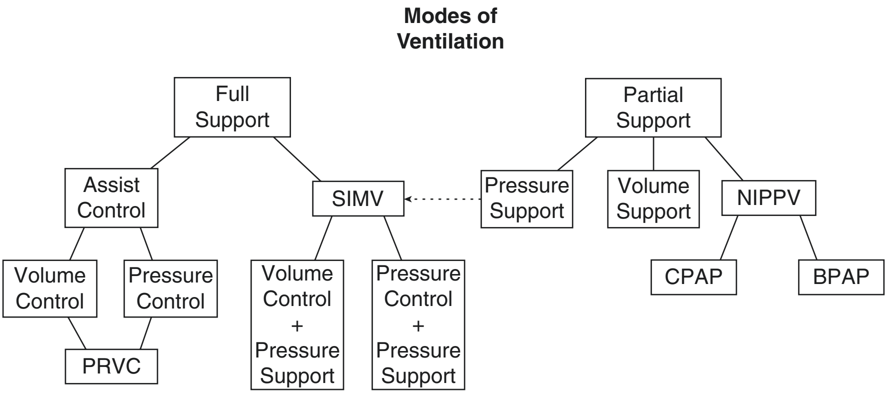

Fig. 6.3 Relationship among the commonly used modes of mechanical ventilation. Note that SIMV usually incorporates aspects of both assist-control ventilation and pressure support ventilation. Pressure regulated volume control is a volume-targeted mode that has a maximum pressure allowed to reach that volume

and the flow is on the bottom. Again, this is a matter of preference and reflects nothing about the patient's physiology.

Figure 6.3 demonstrates the relationship among the commonly used modes of ventilation, separating them as full support or partial support modes.

The modes used most commonly will vary from hospital to hospital. By and large, as long as the patient is getting the level of support appropriate for their condition (a critically ill patient with severe respiratory failure, requiring heavy sedation receiving full support, or a patient intubated for airway swelling only requiring partial support, as examples), the mode has not really been shown to make a significant difference in outcome [1].

Each mode, assist control, SIMV, or partial support modes can be set to be volume-targeted (such as volume control, or VC) or pressure-targeted (pressure

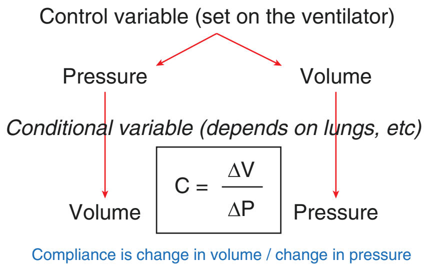

Fig. 6.4 Compliance is the relationship between the change in pressure and the change in volume. For any ventilator setting, the clinician can only set the pressure or the volume. The compliance of the respiratory system will determine the other value

control, PC). When the volume is set ("volume control" or "volume-targeted" ventilation), the patient's resistance and compliance will determine the pressures. When the pressure is set ("pressure control" or "pressure-targeted ventilation"), the resistance and compliance will determine the volume.

Understanding this relationship is important for clinicians to monitor the ventilated patient. This relationship is illustrated in Fig. 6.4.

Beyond the mode, clinicians should understand other basic ventilator settings and their relationships. The following examples illustrate the settings of the ventilator.

In volume control AC (AC/VC), the provider sets a predetermined tidal volume (e.g., 500 mL), flow rate (e.g., 60 mL/min), and respiratory rate (e.g., 12 breaths/min). In this mode of ventilation, the inspiratory to expiratory (I:E) ratio is indirectly determined by the RR and flow rate, as demonstrated below, since this mode of ventilation is not determined by a set time, otherwise known as "time cycled."

For the VC settings:

$$\text{TV} = 500\text{mL}$$

$$\text{Flow} = 60\,\text{L} / \text{min} = 1\,\text{L} / \text{sec}$$

$$\text{RR} = 20\,\text{breaths} / \text{min}$$

The resulting calculations demonstrate the I:E ratio:

$$\text{Total cycle time}\left(\text{TCT}\right) = \left(60\,\text{sec/ min}\right) / \left(20\,\text{breaths} / \text{min}\right) = 3\,\text{sec/ breath cycle}$$

$$\text{Inspiratory time}\left(\text{iTime}\right) = \left(500\text{mL}\right) / \left(1\,\text{L} / \text{sec}\right) = 0.5\,\text{sec}$$

$$\text{Expiratory Time}\left(\text{eTime}\right) = \text{TCT} - \text{iTime} = 3\,\text{sec} - 0.5\,\text{sec} = 2.5\,\text{sec}$$

$$\text{I} : \text{E ratio} = 1 : 5$$

In contrast, in pressure control AC (AC/PC), the ventilator is set to give the desired pressure for a set time. For example, the clinician can set the ventilator for peak pressure such as 15 cmH20 and the inspiratory time such as 1 second. Therefore, one can set the I:E ratio directly, since PC is time cycled or, in other words, gives the selected pressure for a set time.

For the PC settings:

$$\text{Set Pressure} = 15\,\text{cmH}_2\text{O}$$

$$\text{RR} = 20\,\text{breaths / min}$$

$$\text{Inspiratory time} = 0.5\,\text{sec}$$

The resulting calculation demonstrates the I:E ratio:

$$\text{Total cycle time}\left(\text{TCT}\right) = \left(60\,\text{sec/ min}\right) / \left(20\,\text{breaths / min}\right) = 3\,\text{sec/ breath cycle}$$

$$\text{Inspiratory time}\left(\text{iTime}\right) = 0.5\,\text{sec}$$

$$\text{Expiratory Time}\left(\text{eTime}\right) = \text{TCT} - \text{iTime} = 3\,\text{sec} - 0.5\,\text{sec} = 2.5\,\text{sec}$$

$$\text{I : E ratio} = 1 : 5$$

Pressure-regulated volume control, or PRVC, is another mode of mechanical ventilation that blends the best aspects of both volume and pressure targeted ventilation. It is an assist-control (AC) mode is largely volume targeted, in that the clinician selects a desired tidal volume. However, the ventilator strives to administer the tidal volume at the lowest possible pressure, based on the peak pressure limit set by the clinician. If the peak inspiratory pressure reaches the limit set by the clinician, the ventilator will then cycle to expiration phase to protect the lungs from barotrauma before the set tidal volume is achieved. The clinician will then be alerted to the high pressures, allowing for an intervention to assist in reaching the desired tidal volume.

Pressures on the Ventilator

Modern mechanical ventilators all deliver positive pressure ventilation, as opposed to the negative pressure ventilation used in normal physiologic breathing. This pressure, which allows for both oxygenation and ventilation, can be potentially detrimental to the patient in excess. Therefore, the goal is to use the minimum pressure required to oxygenate and ventilate adequately, while minimizing the risks of barotrauma and volutrauma.

The *peak inspiratory pressure (PIP)* represents pressures in the entire airway system and is a measure of both the resistance and compliance. The PIP is displayed on the vent screen with each breath.

The *plateau pressure* (P_{plat}), which is measured when there is an absence of airflow during the plateau phase of mechanical breathing, is the reflection of the pressure delivered to the alveoli and the compliance of the system. Therefore to prevent alveolar injury, the P_{plat} should be maintained ≤ 30 cmH_2O. The P_{plat} is not directly displayed on the ventilator, but can be calculated by pressing the inspiratory pause button, briefly allowing all pressures to equilibrate when there is an absence if airflow. The machine will then display this calculated value.

In Fig. 6.5, the pressure waveform is on the top, and the flow is on the bottom. The PIP is a little over 50 cmH_2O, looking at the scale to the left of the screen. The P_{plat} is 38 cmH_2O, as noted on the scale on the left in the first breath on this image, as well as calculated value noted on the upper right hand corner of the ventilator, after pressing the inspiratory pause button. This indicates that there is a compliance problem. The difference between the PIP and P_{plat} is greater than 5 cmH_2O, indicating there is also a resistance problem. This tracing was taken from the ventilator of a patient with end-stage COPD who developed pneumonia.

To return to our diagram of resistance and compliance in Fig. 6.6, we can imagine that the patient on the left side may have a very high PIP, given the resistance in

Fig. 6.5 Ventilator screen showing the relationship between the peak inspiratory pressure (PIP) and the plateau pressure (P_{plat}) The P_{plat} is only seen with an inspiratory hold maneuver

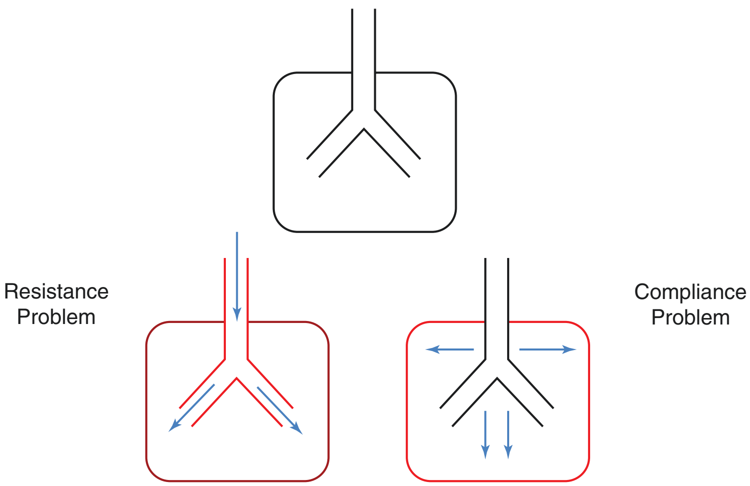

Fig. 6.6 Resistance versus compliance

the system. But, with healthy lungs and a normal compliance, the P_{plat} would be low or normal. Therefore, there may be a large gap between the PIP and P_{plat}, indicating an issue with airway resistance. In the lungs on the right, the PIP could still be elevated, since there is a lot of excess pressure being delivered to this system, and PIP includes measures of both resistance and compliance. In addition, the P_{plat} would also be elevated in this diagram because there Is excess pressure being delivered to the alveoli. However, if the difference between the elevated PIP and P_{plat} is <5 cmH_2O, this would indicate a compliance problem alone.

Another important pressure measure on the ventilator is that of the *autoPEEP* or *intrinsic PEEP (iPEEP)*. When air is trapped in the alveoli at the end of exhalation, it exerts a pressure above and beyond the set PEEP. This pressure can actually be quantified on the ventilator by pressing the expiratory pause button, allowing the ventilator to briefly equilibrate the pressure at the end of expiration.

In Fig. 6.7, the clinician has performed an expiratory hold maneuver, as noted in the first breath on this diagram. The expiratory hold calculates the total PEEP (PEEPTOT) in the system. Assuming the clinician set a PEEP of 5 cmH_2O, we can determine the intrinsic PEEP as follows:

$$\text{PEEPTOT} = \text{ePEEP} + \text{iPEEP}.$$

Therefore, as iPEEP, as noted in the top of this figure is approximately 4.6 cmH_2O. In other words, this patient is not completely exhaling at the end of each breath, leaving some additional pressure in the alveoli. This can also be noted in the flow tracings on the bottom of this figure, as the value at the end of each breath does not return to baseline. With such excess pressure at the end of exhalation, as can be seen in patients with COPD, the work of breathing can dramatically increase, leading to problems with ventilation.

Fig. 6.7 Ventilator screen demonstrating an expiratory hold maneuver. The total PEEP is 9.8, for an intrinsic PEEP, also known as autoPEEP, of 4.6

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Suggested Reading

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7 Understanding the Ventilator Screen

Ventilators at times may seem intimidating as there are numerous waveforms and values on the screen. Additionally, the data are presented slightly differently on the screens of each mechanical ventilator brand, potentially increasing confusion. However, using the terms we have just reviewed, close inspection of ventilator screens will show that most of the waves and data are actually simple to interpret, given a little familiarity. To increase clinicians' comfort with ventilator screens, we have deliberately selected screenshots from a few different types of machines and modes of ventilation. Additionally, we have changed the colors of the backgrounds to black and white to demonstrate that the presentation is less important than the data provided.

Key concepts for evaluating ventilator screens are as follows:

1. The values set by clinicians are found on the bottom of the screen. The patient's response is located at the top of the screen.
2. Data are provided in both numerical and graphical contexts on the screen.
3. Much like studying EKGs, interpreting flow lines simply comes with experience. Unlike EKGs, however, there are fewer variations to know! Ventilators provide three types of tracings: flow, pressure, and volume. Some mechanical ventilators show all three, while other brands allow the clinician to choose two tracings to display on the screen. Fortunately, all are labeled directly on the ventilator screens.

Figure 7.1 illustrates typical pressure, flow, and volume waveforms on a ventilator screen.

Please reference the theoretical illustration of Fig. 2.5, highlighting the relationship between the volume, flow, and pressure.

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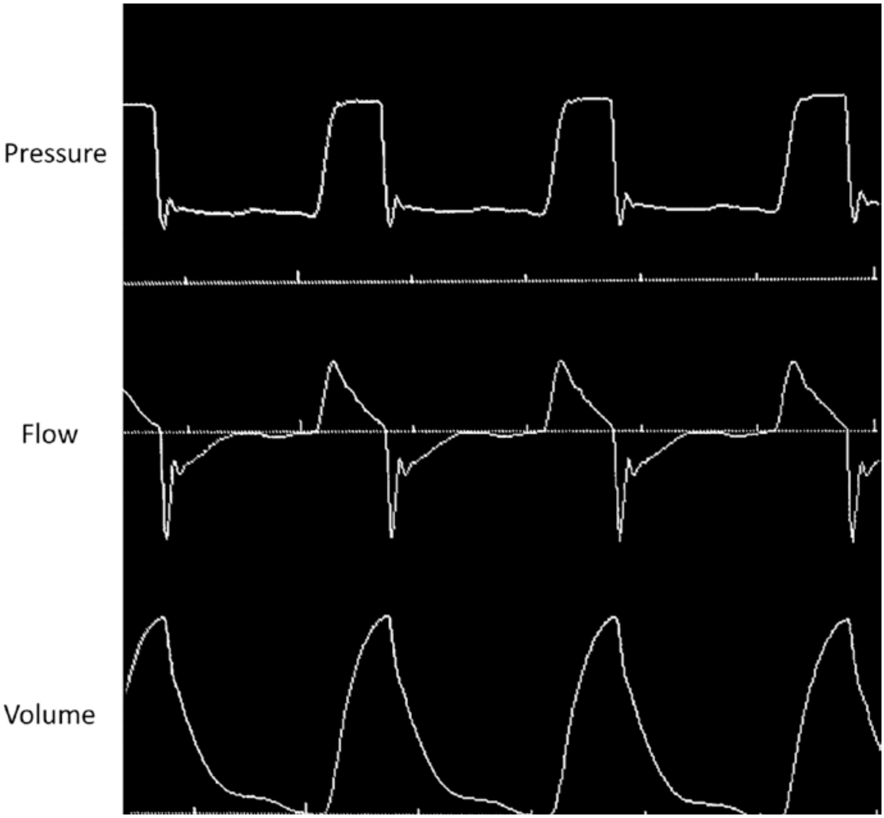

Fig. 7.1 Typical waveforms for pressure, flow, and volume are illustrated

Examine the image of the mechanical ventilator screen in Fig. 7.2 closely, and try to answer the following questions:

1. What is the PEEP?
2. What is the respiratory rate? Is the patient overbreathing the ventilator? How could you tell?
3. What is the set tidal volume? What is the tidal volume the patient is actually receiving?
4. What is the peak inspiratory pressure? What is the P_{plat}?
5. What is the I:E ratio? Is this set directly or indirectly on this particular patient?
6. What do the 40 cmH_2O, 70 l/min, and 400 ml signify to the left side of the screen?
7. What is the minute ventilation?

Answers for Fig. 7.2:

1. PEEP is 10 cmH_2O.
2. The set RR is 28. This patient is not overbreathing, as the rate up top is also 28.

Fig. 7.2 Example ventilator screen from an ICU patient

3. The set tidal volume (VT) is 380, but the patient is receiving 388 mL (on most recent inhaled tidal volume, VTi) and 386 mL as the most recent exhaled tidal volume (VTe). These small variations are to be expected from breath to breath.
4. The peak inspiratory pressure (PIP) is 25. The question about the P_{plat} is somewhat of a trick—but it is an important learning point. The P_{plat} cannot be determined from this screen. P_{plat} requires an inspiratory hold, and that maneuver is not being performed on this screen.
5. The I:E ratio is 1:2.5. We don't see any setting for the specific I:E ratio at the bottom of the screen, and the I:E is set indirectly. Please refer to Chap. 6 for a discussion of setting the I:E indirectly.
6. The top tracing is the pressure, and the 40 cmH_2O labeled at the left side of the screen signifies the top pressure. The flow waveform is in the middle, with the "70 L/min" above that waveform. The bottom waveform is volume. Note the "400 mL" on the top left above the waveform.
7. The minute ventilation (MVe) is 11.1.

Figure 7.3 is another ventilator screen from a different mechanical ventilator brand. Again, practice looking for certain values.

1. What is the set tidal volume?
2. What is the PEEP?

Fig. 7.3 Example ventilator screen. Note that although the design differs slightly from Fig. 7.2, the general formatting is consistent. The variables set by the clinician are at the bottom, and resultant values and graphical information are on the top of the screen

3. What is the set respiratory rate? (Hint: some ventilator brands use “frequency” as a synonym for “rate.” This can be denoted by an “f.”) Is the patient overbreathing?
4. What is the PIP? What is the P_{plat}?
5. What is inspiratory flow?
6. What is the tidal volume per kilogram of predicted body weight?

Answers for Fig. 7.3:

1. The set tidal volume (V_T) is 400 mL. The actual tidal volume (V_{TE}) is 418 mL. The “E” references exhaled. To ensure accuracy, the exhaled tidal volume (as opposed to the inhaled) is used for calculations. Unfortunately, different brands of ventilators use different abbreviations for the same concepts.
2. The PEEP is 14 cmH_2O.
3. The set rate (or frequency) is 24 breaths per minute, and the patient is not overbreathing.
4. The PIP is 48 (which is extremely high!). As with the last image, P_{plat} is not shown on this screen.

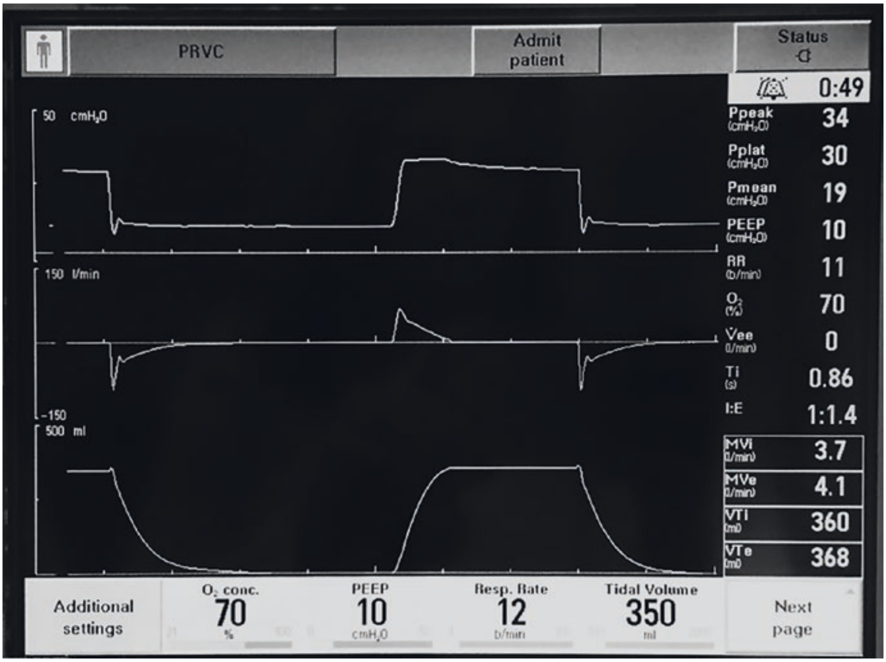

Fig. 7.4 Example ventilator screen, demonstrating an inspiratory hold

5. The inspiratory flow is 44 L/min. Note that this is shown on the bottom in the settings as well as at on the waveforms. See that the sharp peak of the flow waveform is at approximately 45 by the scale to the left.
6. 6.1 mL/kg. On the bottom left of the screen, the respiratory therapist has entered the patient's predicted body weight (69 kg) into the settings. The set volume of 400 is 5.8 mL/kg, and the actual tidal volume of 418 is 6.1. Recall that slight variations such as 400 to 418 are normal in breath to breath variation.

Figure 7.4 provides key information not seen in the prior examples.

1. What is the tidal volume? What is the PEEP?
2. What is the PIP? What is the P_{plat}?
3. What is the FiO_2? (Hint: this is also known as the concentration of oxygen.)

Answers for Fig. 7.4:

1. The set tidal volume is 350 mL, with an exhaled of 368 mL and an inhaled of 360. The exhaled is the volume we record when considering what the patient is actually receiving. The PEEP is 10.
2. The PIP (or peak inspiratory pressure or Ppeak) is 34 cmH_2O. The P_{plat} is finally shown on this screen, as this is captured during an inspiratory hold maneuver. The P_{plat} is 30 cmH_2O.
3. The fraction of inspired oxygen, or FiO_2, is 70%, or 0.7. It is indicated by the "O_2 conc" on the bottom of the screen.

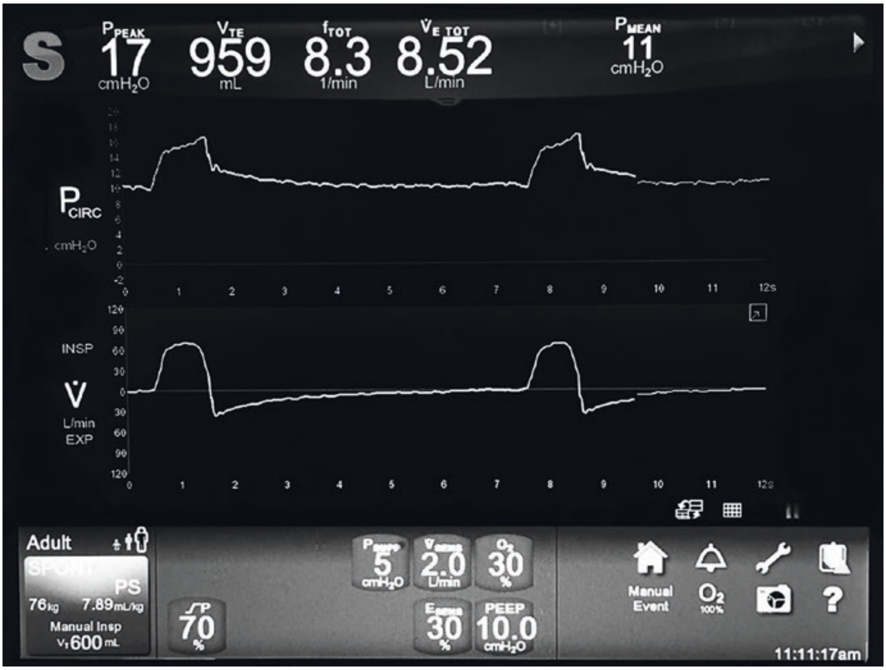

Fig. 7.5 Example ventilator screen, highlighting yet another style, yet the same basic data are all provided

As a final example of the variability, yet similarity, among ventilator interfaces, Fig. 7.5 is a screenshot from yet another style of ventilator.

1. What is the mode?
2. What tidal volume is the patient receiving? Bonus: What does this tell you about the patient's compliance?
3. What is the respiratory rate (or frequency)?
4. What is the PIP? Bonus: Is it possible to check a P_{plat}?
5. What is the minute ventilation?

Answers for Fig. 7.5:

1. Pressure support. There are several clues. The "S" in the upper L hand corner indicates "Support." Review Fig. 6.2. There is a "C" in that corner, indicating that the most recent breath delivered was a "Controlled" breath. Many ventilators will also show an "A" for "Assist" when a patient is in assist-control mode and triggers a breath.

   The other clues are that there is no set respiratory rate and the settings at the bottom of the screen feature pressures.

2. The patient is receiving 959 mL of tidal volume. This is a very high volume and may need to be intervened upon!
3. There is no set respiratory rate, but the patient is averaging 8.3 breaths per minute.
4. The PIP is 17. This should be, and is, very close to the set pressure support of 5 cmH_2O + the PEEP of 10 cmH_2O. It is not uncommon to have small variances in numbers between the set and delivered pressures and volumes.
5. The minute ventilation is 8.52. This is intuitive, as the patient is taking about a liter per breath, and is breathing just over eight times a minute, leading to 8.5 L/min of airflow.

Suggested Reading

Mosier JM, Hypes C, Joshi R, et al. Ventilator strategies and rescue therapies for management of acute respiratory failure in the emergency department. Ann Emerg Med. 2015;66:529–41.
Singer BD, Corbridge TC. Basic invasive mechanical ventilation. South Med J. 2009;102(12):1238–45.

Placing the Patient on the Ventilator

8

Anticipating Physiologic Changes

Critically ill patients are at high risk of deterioration with intubation and initiation of mechanical ventilation. Much of this text is devoted to reviewing the effects positive pressure ventilation (PPV) can have on pulmonary physiology. However, mechanical ventilation can also have extrapulmonary effects that warrant review. Specifically, PPV can lead to an increase in the intrathoracic pressure, which leads to decreased venous return and decreased preload. While we use this principal to care for those with congestive heart failure (CHF), in excess, this phenomenon can lead to a decrease in the cardiac output and hypotension, especially in the intravascularly depleted patient, those with shock physiology, or with air trapping. Additionally, PPV leads to decrease the left ventricular afterload. Again, using the patient with an acute CHF exacerbation as an example, this principle can lead to an increase in the stroke volume and cardiac output.

When intubating and placing the patient on the ventilator, the emergency medicine clinician should anticipate these effects. A volume depleted patient, such as a patient with a GI bleed or sepsis, may have hemodynamic collapse with initiation of positive pressure ventilation. Therefore, clinicians should consider treating such patients with hydration and possible vasopressor support in the peri-intubation period to avoid hemodynamic compromise and collapse.

When initiating mechanical ventilation in the ED, the practitioner must be conscientious to ensure adequate gas exchange to meet the metabolic demands of the patient. For example, a patient with severe metabolic acidosis with respiratory compensation might be very tachypneic due to their disease process. The clinician must be cognizant of the patient's pre-intubation tachypnea and increased minute volume and set the respiratory rate accordingly on the ventilator to help meet the patient's metabolic demands. Failure to do so can be detrimental for the patient and lead to worsened acidosis and rapid decompensation.

Along the same lines, the practitioner must be careful to set and then adjust the ventilator settings to prevent further decompensation or injury. For example,

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excessive volumes ventilator can lead to volutrauma and impaired gas exchange by causing collapse of the capillaries that participate in the ventilatory process. Excess intrathoracic pressure can lead to hemodynamic instability due to decreased preload, or barotrauma with overdistension of the alveoli or pneumothoraces.

Setting the Ventilator

The goal of reviewing the terms, physiology, and concepts behind mechanical ventilation is to be able to put the pieces together and improve our care of mechanically ventilated patients in the ED. Also, please remember that ventilator settings may require adjustment as the patient's disease evolves or resolves. Therefore, once the initial settings are placed, the clinician must reassess the patient and continuously make adjustments to meet the patient's metabolic demands, while trying to reduce or prevent harm.

To that end, let's practice selecting ventilator settings. Imagine that you just intubated a patient who presented to your ED after an overdose of an unknown medication, leading to apnea and a GCS of 3. How would you select ventilator settings for this patient?

Mode: To start, select a mode. Most patients in the ED, especially shortly after intubation, should be ventilated in assist control (AC). Assist control would be appropriate for our hypothetical patient, as she is making no respiratory efforts. The next decision involves selecting a volume-targeted or a pressure-targeted mode. In the clear majority of cases, this decision is one of personal preference and local customs. Numerous studies have found no differences for patients ventilated with one or the other. Most clinicians chose volume-targeted assist control, or volume control.

Tidal Volume (TV): The appropriate tidal volume is based upon the patient's height and biological sex, as these parameters determine predicted body weight and lung size. Take care to use predicted body weight or ideal body weight and not actual body weight which can greatly overestimate the appropriate tidal volume. In contrast to older practices, which used "high" tidal volumes of 10–12 mL/kg, current practice based on several trials suggests that patient should be ventilated with "lower" or more physiologic tidal volumes of 6–8 mL/kg.

Respiratory Rate (RR): A reasonable approach is to consider the desired minute ventilation and chose a respiratory rate to approximate this value. Assuming there are no acid-base derangements, targeting relatively normal minute ventilation is appropriate. If we selected a tidal volume of 400 based on her height, a respiratory rate of 15 breaths per minute will lead to a minute ventilation of 6 L/min (400 mL × 15 bpm).

Conversely, if there is an acid-base disturbance, such as can occur with the ingestion of a toxin like ethylene glycol or in sepsis, the patient will need larger minute ventilation to correct the acidosis. The tidal volume should again be set using low tidal volume ventilation of 6–8 mL/kg. As most patients with significant acidosis with be tachypneic to compensate for this acid/base disturbance, the initial ventilator respiratory rate should be set higher than a normal physiologic rate. For example, setting her rate at 24 breaths per minute will give a minute ventilation of 9.6 L/min (400 mL × 24 bpm). About 20–30 minutes after selecting the initial settings, clinicians should check an arterial blood gas (ABG) to assess acid/base status and oxygenation and make ventilator changes as needed to adjust the minute volume and oxygenation. Also, as the disease process improves, the respiratory rate may need to be adjusted to avoid overcorrection and alkalosis, which can be just as determinate.

Stated another way, the minute volume (TV × RR) affects the patient's ventilation. Starting with low tidal volume ventilation of 6–8 mL/kg, providers should be cognizant of the disease process, the preexisting respiratory rate, and the acid/base status when determining the initial ventilator respiratory rate. As the patient improves or deteriorates clinically, these minute volume should be adjusted to meet the patient's metabolic demands and avoid further injury.

PEEP: PEEP should always be set to a minimum of 5 cmH_2O, to reduce atelectasis and help overcome the increased work associated with breathing through an endotracheal tube. The conditions that will require a higher PEEP are those leading to worsening hypoxemia, wherein more atelectasis or derecruitment would be detrimental. Additionally, patients with large abdominal or chest walls may require a higher PEEP to prevent compression from abdominal contents. The concept behind the ideal PEEP is illustrated in Fig. 8.1. Every patient will have a relationship

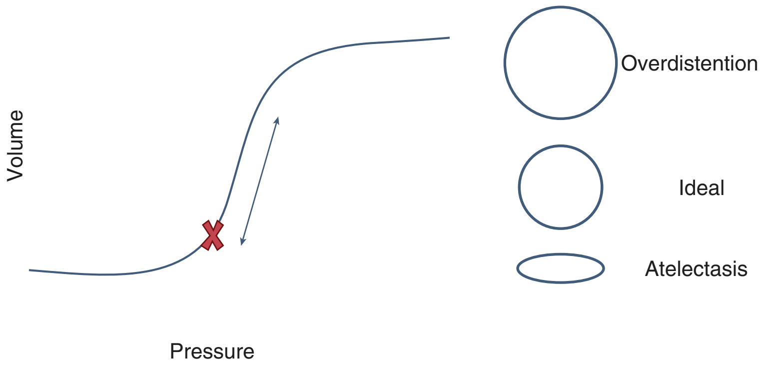

Fig. 8.1 Theoretical representation of ideal PEEP. The PEEP should be high enough to prevent atelectasis with exhalation, but low enough that inhalation does not result in overdistention. The red "x" in this diagram shows this ideal spot for the relationship between the volume and pressure for this hypothetical patient. The double-ended arrow represents the changes in inspiration and exhalation

between the change in pressure and the change in volume with each breath. The PEEP should be set above the threshold for atelectasis, but such that the breath will not lead to overdistention.

Using our hypothetical patient intubated for a GCS of 3, if she has a small to average habitus, a PEEP of 5 is likely appropriate to start. If she is heavier or has a larger abdomen or chest wall, she may be more prone to atelectasis. This would make a higher initial PEEP of 7–10 cmH2O reasonable. It is important to bear in mind that increasing the PEEP can lead to decreased preload in a dehydrated or hypovolemic patient. Therefore, a provider must consider the need for hydration or additional vasopressor support with an increased PEEP requirement to avoid hemodynamic compromise. This must be balanced, as stated above, with overdistention and impaired gas exchanged.

FiO_2: The initial FiO_2, or fraction of inspired oxygen, is the percentage of oxygen delivered by the ventilator. At room air we inspire 21% FiO_2, a number which can be altered by altitude. The initial FiO_2 should be determined again by the patient's disease process and need for intubation. More specifically, patient who are hypoxic pre-intubation may benefit from a higher FiO_2, which those who are saturating well before intubation may not require such high doses of oxygen.

Oxygenation on mechanical ventilation is determined by the PEEP and FiO_2 settings. As stated with ventilation above, the provider should obtain an ABG within 30 minutes of intubation to determine the PaO_2, or the partial pressure of oxygen, to wean these settings as needed. As with other parameters, the clinician should continually reevaluate the settings during the patient's disease course and make adjustments as needed to meet the needs of the patient while avoiding harm, as supra-oxygenation is as harmful and hypoxia.

Inspiratory Flow and I:E Ratio: The inspiratory flow and I:E ratios are commonly set at 60 L/min and 1:1.5 to 1:2, respectively. Common inspiratory times are 0.75–1 second. In certain circumstances, such as in airway obstruction with asthma, allowing more time for exhalation is beneficial. In these cases, one can increase the inspiratory flow or decrease the I:E ratio, to 1:3 or 1:4. Bear in mind that increasing the flow rate can lead to increased thoracic pressure and decreased preload.

Examine the ventilator screen shown in Fig. 8.2.

In this example, the patient's set respiratory rate is 26. Therefore, the total cycle time is calculated as follows:

$$\text{Total cycle time}(\text{TCT}) = (60\,\text{sec/ min}) / (26\,\text{breaths / min}) = 2.3\,\text{seconds}$$

Inspiratory time (iTime or TI) is 0.90 seconds, as noted on the right hand side of the screen.

Therefore, the expiratory time is 1.4 seconds. (2.3 seconds–0.9 seconds).

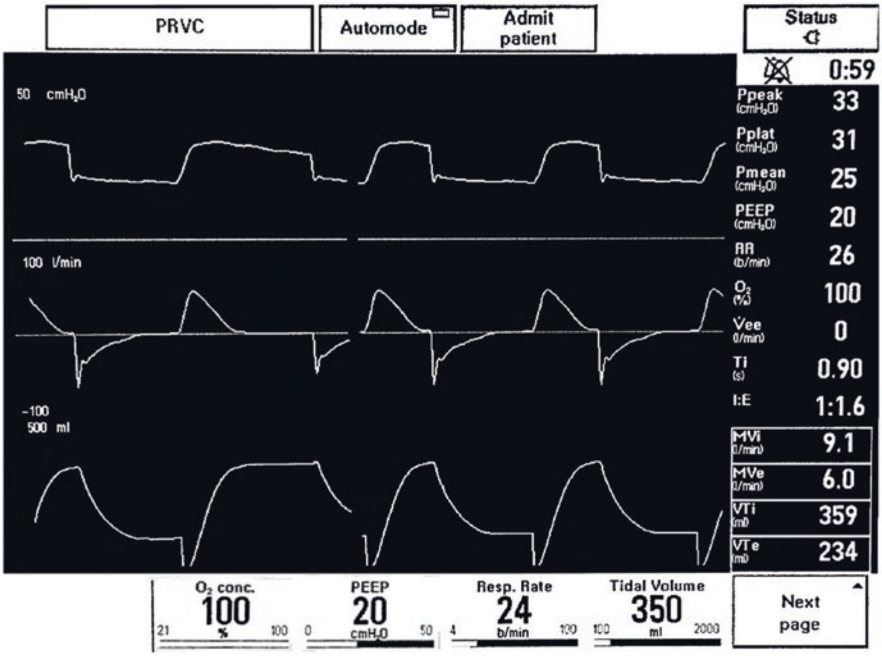

Fig. 8.2 Ventilator screen demonstrating the relationship between respiratory rate, inspiratory time, and I:E ratio

The ratio of inspiratory time to expiratory time (I:E ratio) is therefore 0.9: 1.4, or approximately 1: 1.6.

At the bedside, ventilators will provide this information, just as illustrated in Fig. 8.2. The clinician does not have to perform the calculations, but understanding the concepts is important for setting and adjusting the ventilator. To return to the example of our patient intubated for the overdose, we could consider what changes we would make if she had bronchospasm. In addition to treating with bronchodilators, we would give more time for exhalation, and it is important to understand that this would mean either decreasing the respiratory rate or decreasing the inspiratory time.

After Initial Settings

Mechanical ventilation is a dynamic intervention, and once a patient is intubated and mechanically ventilated, the clinician must continuously reassess the patient and determine the best settings to help meet the metabolic and oxygen demands while avoiding any additional injuries. All intubated patients should have an arterial blood gas (ABG) checked 20–30 minutes after intubation. While venous blood gases (VBG) are excellent in the ED and are useful for evaluating a patient’s pH and ventilation, a VBG cannot provide any data regarding oxygenation. Most patients are intubated and started on a FiO_2 of 100%, although this can be titrated down, to reduce the risks of oxygen toxicity, a condition increasingly appreciated in relation to numerous causes of critical illness.

To report these ventilator settings, such as when speaking to the intensivist, one would say, "The patient is on Assist Control/Volume Control, with a tidal volume 400mL, rate of 15 breaths per minute, PEEP 5 cmH_2O, and an FiO_2 of 100%. She is occasionally overbreathing to a rate of 18 breaths per minute. Her initial ABG after 30 minutes on these settings showed…".

Patients also remain at risk for hemodynamic perturbations after the initiation of ventilation or with changes in ventilation, due to changes in physiology along with the fluctuations in preload and afterload. Therefore, clinicians must continue to be mindful of the patient's intravascular status in ventilated patients and resuscitate these patients as needed.

Suggested Reading

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9 Specific Circumstances: Acute Respiratory Distress Syndrome (ARDS)

Acute respiratory distress syndrome (ARDS) is a condition of diffuse alveolar damage and inflammation, secondary to any number of possible processes. ARDS is defined by four criteria [1]:

1. The condition must be acute (<7 days).
2. The findings are not solely explained by cardiogenic pulmonary edema.
3. The chest X-ray must have bilateral opacities, as shown in Fig. 9.1.
4. While on at least 5 cmH_2O of positive pressure ventilation, the ratio of PaO_2 to FiO_2 (expressed as a decimal, such as 0.7) must be <300.
   (a) Mild ARDS is a PaO2/FiO2 ratio of 200–300.
   (b) Moderate ARDS is 100–199.
   (c) Severe ARDS is <100.

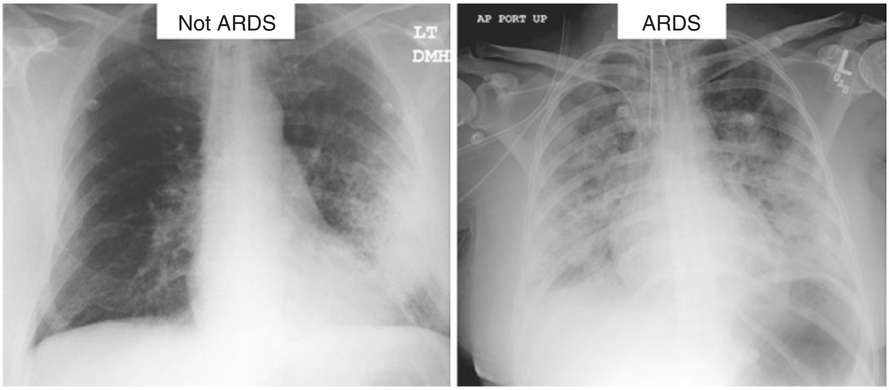

Fig. 9.1 Chest X-rays illustrating the difference between ARDS and pneumonia. Note that both patients can be severely hypoxemic, but ARDS has bilateral, diffuse infiltrates

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Although patients rarely present to the ED in fulminant ARDS, as it usually develops later in the critically illness, ARDS can be seen in the ED. Of all the interventions in critical care, few have been as reproducibly beneficial to patients as low tidal volume ventilation [2]. Positive pressure ventilation, especially with large tidal volumes or high pressures, has been shown to cause injury in both patients with ARDS as well as patients who do not yet have ARDS. Prevention of ARDS, or prevention of exacerbating ARDS with ventilator-induced lung injury, is a key benefit of active ventilator management in the ED.

Many of the maneuvers used in severe hypoxemia to improve oxygenation and ventilation can be deleterious in the long term, if not managed carefully. Increasing the mean airway pressure (MAP) is one of the major goals of positive pressure ventilation, and higher MAPs are often associated with improved oxygenation. The factors that increase MAP are those that either increase the pressure in the airways, such as tidal volume, PEEP, or, AutoPEEP, or the amount of time the positive pressure is delivered, such as the inspiratory time.

However, despite short-term improvement in oxygenation, high pressures in the alveoli are also associated with worse long-term outcomes. Therefore, the clinician must balance the risk of increasing the MAP with using good, evidence-based ventilator management, shown in Fig. 9.2.

Tidal volumes are best represented in both mLs and mLs/kg of predicted body weight. The predicted body weight is a surrogate for the patient's anticipated lung volume. Lung volumes depend upon a patient's height and biological sex. Actual body weight should never be used as a replacement for the predicted body weight.

The ventilator screen below shows an example of a low tidal volume strategy. The patient was set on 330 ml/kg, or 6.35 ml/kg of PBW, as indicated in the bottom left-hand corner for Fig. 9.3.

Once the initial tidal volume is selected, the pressures should be assessed. In ARDS, as well as other patients, maintaining a P_{plat} <30 cmH_2O is key to preventing ventilator-induced lung injury [2]. Note that the P_{plat} will be determined by the tidal

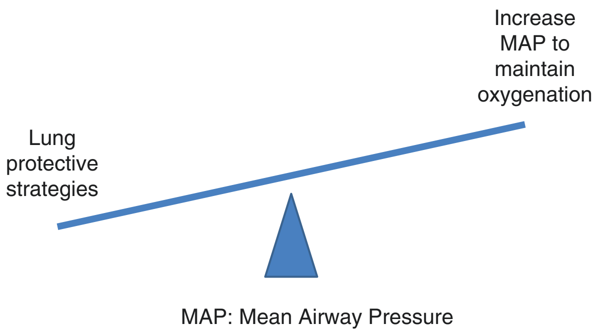


Fig. 9.2 Although the clinician should do what they must to stabilize a hypoxemic patient, the principles of evidence-based ventilator management should hold weight. For example, large tidal volumes may lead to a rapid improvement in hypoxemia, but their use trades short-term benefit for long-term harm

Fig. 9.3 A ventilator screen showing an inspiratory pause to calculate a plateau pressure (P_{plat}). In this case, the P_{plat} is 32, greater than the target of ≤30 cmH_2O. The patient is receiving over 6 mL/kg of TV, so decreasing the TV would be a good first step to correct this elevated P_{plat}

volume given and the compliance of the respiratory system. ARDS usually results in decreased compliance, resulting in stiff lungs.

Using an inspiratory hold, the P_{plat} should be confirmed to be less than 30 cmH_20. If P_{plat} is >30 cmH_20, a lower tidal volume should be selected, even down to 4 ml/kg of predicted body weight.

In Fig. 9.3, an inspiratory hold has been performed, providing a P_{plat} of 32. This tells us two things:

1. There is a minimal difference between the PIP (35 cmH_2O) and the P_{plat} (32 cmH_2O) indicating that the patient has only a compliance problem without a resistance problem.
2. The P_{plat} is too high. The tidal volume should be lowered to 5 ml/kg PBW and reassessed.

The image of a ventilator screen in Fig. 9.4 shows another example of performing an inspiratory pause to calculate a P_{plat}.

PEEP is the next setting to address. Clearly, oxygenation is a critical factor for these patients. PEEP increases the mean airway pressure (MAP) and thereby improves oxygenation. PEEP additionally can help prevent further derecruitment.

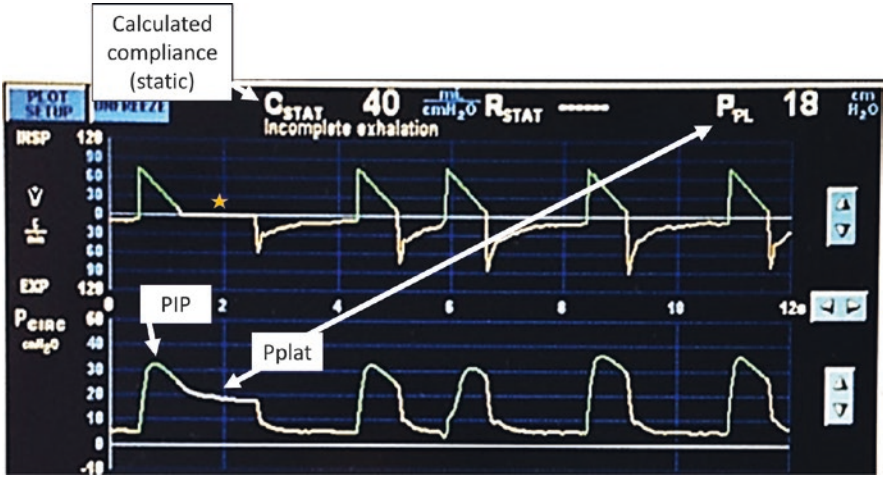


Fig. 9.4 This is another example of using an inspiratory hold to check a P_{plat}. The gold star shows where flow has ceased to allow pressures to equilibrate. The P_{plat} is 18 cmH_2O in this example. The ventilator automatically calculates a compliance of 40 mL/cmH_2O. A normal compliance is about 80–100 mL/cmH_2O, and expected for a ventilated patient is approximately 60 mL/cmH_2O, as all ventilated patients are less compliant that those breathing with normal respirations

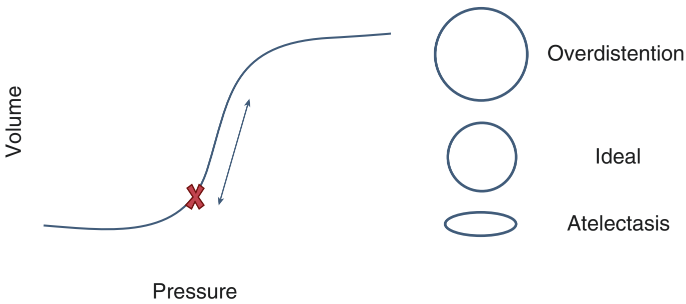


Fig. 9.5 The optimal PEEP is one that prevents both atelectasis as well as overdistention. In this theoretical graph of volume and pressure, the red X demonstrates the ideal PEEP setting. With each breath, the volume and pressure will increase, but the alveoli will not become overdistended. With exhalation, the volume and pressure will decrease, but the alveoli will not collapse

A physiologic goal in setting PEEP is to prevent atelectasis without extending into overdistention. A theoretical optimal PEEP would be at the red "X", and each breath would move up and down the pressure/volume slope as indicated by the light blue arrow, as shown in Fig. 9.5.

Many of these patients will need moderate to high PEEPs of 8–16 cmH_2O and, at times, even greater. The PEEP will contribute to the P_{plat}, and therefore, the P_{plat} should be checked with any PEEP change, just as with any TV change. The time when an increase in PEEP will not, or will only minimally, increase the P_{plat} is when the patient is derecruited, and increasing the PEEP helps recruit collapsed lung. In

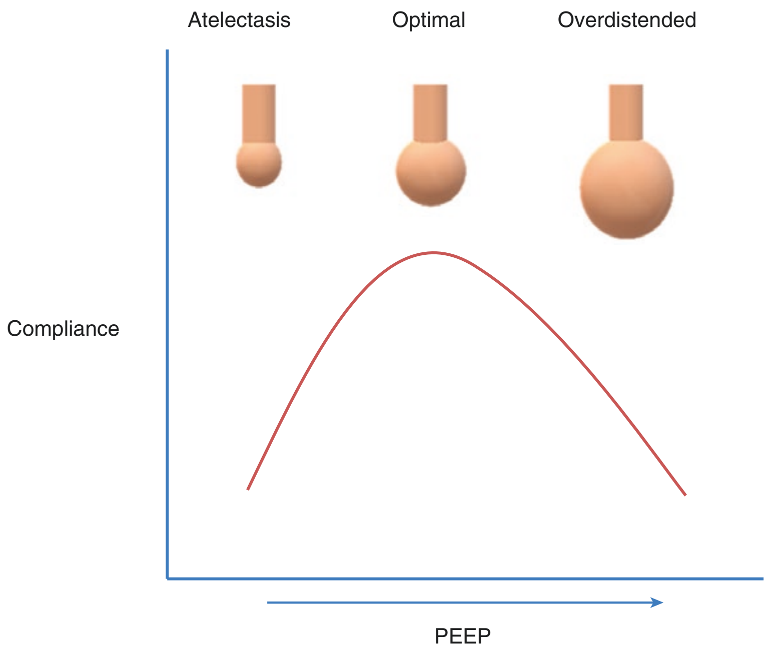


Fig. 9.6 By selecting the optimal PEEP, the clinician can also optimize compliance. The lungs are poorly compliant when atelectatic and are also poorly compliant when they are overdistended

this instance, the increase in PEEP can actually improve compliance and therefore not increase the P_{plat}. This is illustrated in Fig. 9.6.

This is the principle behind performing a recruitment maneuver and a "BestPEEP" trial to find a PEEP that optimizes compliance—preventing both atelectasis and overdistention. This is discussed in more detail below, under "*Recruitment Maneuvers*."

Driving pressure (ΔP) is the term that describes the pressure changes that occur during inspiration and is equal to the difference between the plateau pressure and PEEP (P_{plat} – PEEP). For example, a patient with a P_{plat} of 30 cmH_2O and a PEEP of 10 cmH_20 would have a driving pressure of 20 cmH_2O. In other words, 20 cmH_2O would be the pressure that exerted to expand the lungs. Studies have shown that a driving pressure of <15 cmH_2O is associated with better outcomes in patients with ARDS [3]. Driving pressure is illustrated in Fig. 9.7.

While most patients will be started on a FiO_2 of 100%, especially if hypoxemic, the FiO_2 should be decreased as tolerated after checking an ABG. An ABG in the ED provides important information for patients intubated with hypoxemia, as it allows the clinician to calculate the PaO_2 to FiO_2 (P/F) ratio, and thereby categorizes the severity of the patient's ARDS. While most patients with ARDS do not die of hypoxemia, per se, the P/F does correlate with outcomes. As such, this level can be used to guide other therapies, including proning, as well as gauge responses to interventions.

Fig. 9.7 This image from a ventilator shows the relationship among the PIP, P_{plat}, driving pressure, and the PEEP. Of these, only the PEEP is set on the ventilator. The PIP is a factor of the resistance and compliance and the P_{plat} the compliance. Both are impacted directly by the TV (the higher the TV, the higher the PIP and P_{plat}.) The driving pressure is also a factor of the compliance. It can be impacted directly by the TV and the PEEP. Optimizing PEEP can improve compliance, thereby lowering the P_{plat} and lowering the driving pressure

Once an ABG is checked, however, oxygen saturations are appropriate to follow in the ED. Oxygen toxicity is increasingly appreciated in numerous conditions, as decreasing the FiO_2 as much as is safely tolerated is appropriate [4–6]. A reasonable target is an SpO_2 of 92–96%.

Patients being ventilated with low tidal volumes will require a higher rate to maintain minute ventilation. Most patients with ARDS will require RR of 20 breaths per minute or greater. This is especially important to consider as many patients with ARDS will be hypermetabolic, with increased CO_2 production. This is illustrated in Fig. 9.8.

Initial Ventilator Settings in ARDS

Tidal volume	4–8 ml/kg PBW, starting with 6 ml/kg
Respiratory rate	Higher, often >20 breaths per minute
PEEP	≥8 cmH_2O, avoiding overdistention
FiO_2	Decrease as tolerated, $SpO_2 \geq 92\%$

Severe Hypoxemia

At times, patients may have refractory, severe hypoxemic respiratory failure. After checking all ventilator settings as described above, the clinician should employ additional evidence-based maneuvers.

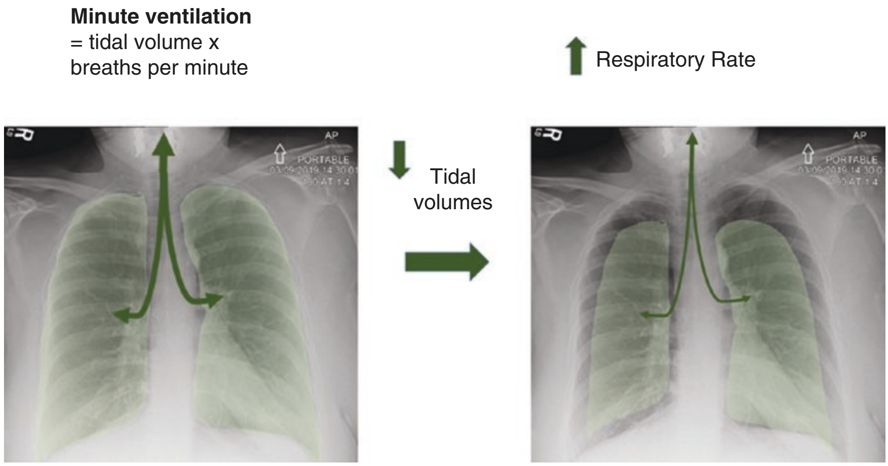


Fig. 9.8 Minute ventilation is determined by the volume of air per breath (TV) and the number of breaths per minute (respiratory rate). A decrease in the TV mandates an increase in the respiratory rate to maintain the minute ventilation

Neuromuscular Blockade

At times, a patient may be well sedated yet dyssynchronous with the ventilator. Ventilator dyssynchrony is associated with worse outcomes and should be avoided. In years past, neuromuscular blockade, specifically with cisatracurium, was used in patients with moderate to severe ARDS. A recent trial published in 2019, however, did not find improved mortality with routine neuromuscular blockade use in ARDS [7]. However, neuromuscular blockade was also not associated with increased harm. As such, it can be considered in patients who remain dyssynchronous with the ventilator despite appropriate sedation. This image of the ventilator in Fig. 9.9 shows how a patient looks when dyssynchronous. The waveforms should be smooth and regular. When they are jagged and irregular, the patient is fighting the ventilator. Additionally, note that while the patient is set on low tidal volume ventilation at 380 ml, the patient is actually taking in about 800 ml through significant respiratory efforts.

Recruitment Maneuvers

In well-sedated and possibly chemically relaxed patients, the first maneuver is to provide a recruitment maneuver. Recalling that decruitment is a common cause of hypoxemia, gently recruiting alveoli can improve oxygenation. The damage to the lungs is heterogeneous. Some areas are atelectatic, some are fluid-filled, some are already over distended, and some are even normal. The concept behind a recruitment maneuver is simple: the application of sustained pressure to open up collapsed alveoli [8]. However, there are two potential downsides (Fig. 9.10).

Fig. 9.9 This ventilator screen demonstrates ventilator dyssynchrony. The ventilator is set at low tidal volumes, but the patient is taking around 800 mLs per breath

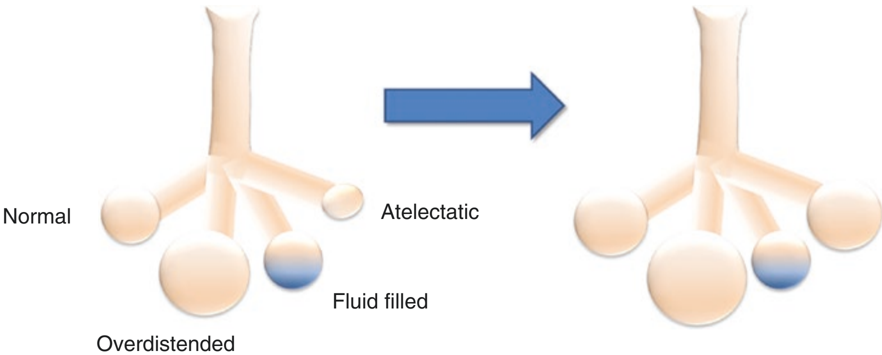


Fig. 9.10 ARDS is a heterogeneous condition. The "alveoli" here represent areas of lung, with various lung units being normal, fluid-filled, overdistended, or atelectatic. The recruitment maneuver may transiently over-distend the normal and already overdistended lung units, but the expectation is that recruiting the atelectatic areas will overall improve oxygenation after the maneuver is completed

However, note that the normal and overdistended areas may also become even more overdistended. This overdistention from the previously "good" parts of the lung can lead to decreased gas exchange during the recruitment, as shown in Fig. 9.11, causing desaturation. This effect should be temporary and improve after the maneuver.

The second effect is that the patient can become hemodynamically unstable, due to a significant increase in the intrathoracic pressure and resultant decrease in preload and increase in right ventricular afterload (Fig. 9.12). Again, this should be

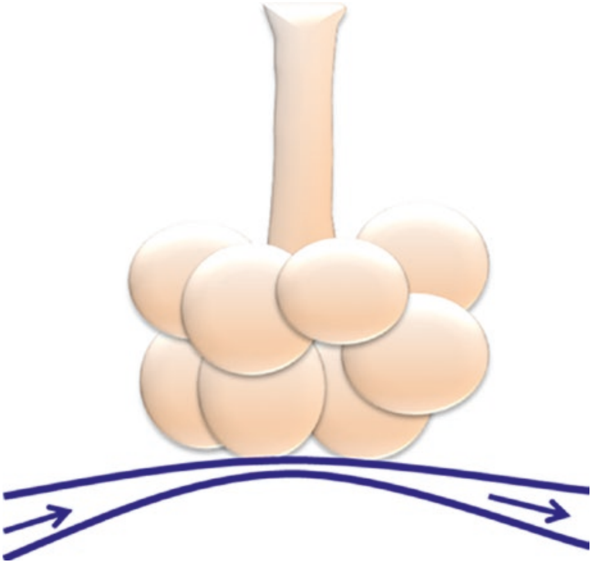


Fig. 9.11 A lung unit that becomes overdistended can surpass the pressure in the capillaries, transiently reducing blood flow and gas exchange to that portion of the lung. This is what is responsible for the temporary desaturation during a recruitment maneuver

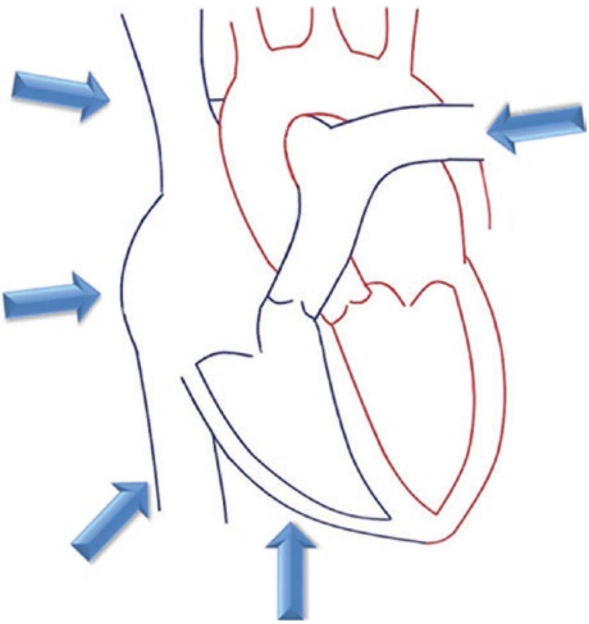


Fig. 9.12 The increased intrathoracic pressure can be deleterious to the right heart. The increase in pressure will decrease preload, which can lead to hemodynamic instability for patients who are volume responsive. Importantly, it can also lead to increased right ventricular afterload and RV failure. As such, recruitment maneuvers should be done with caution, if not avoided altogether, in patients who are hemodynamically unstable or volume depleted

temporary and resolved with a reduction in the pressure, but in unstable or preload dependent patients, this can precipitate hemodynamic collapse. Recruitment maneuvers should never be performed without a respiratory therapist, nurse, and physician present. All clinicians should be aware of the risks of transient hypoxemia and hypotension.

There are many methods of performing recruitment maneuvers. One of the methods least likely to cause hemodynamic perturbations is to serially increase PEEP in small increments [9]. A trial of large increases in PEEP (25 cmH_2O, 35 cmH_2O, then 45 cmH_2O with a final PIP of up to 60 cmH_2O) was stopped early for futility [10]. As such, we recommend a more gradual approach.

Once the patient is stabilized after intubation, the recruitment maneuver and the best PEEP can be determined, using a decremental compliance PEEP trial. This can be repeated every 24 hours for patients who continue to require high levels of ventilatory support.

The FiO_2 should be set at 1.0, and the patient appropriately sedated and relaxed if needed. The ventilator should be set to pressure control ventilation, with a PC of 15 cmH_2O, inspiratory time of 3 sec, rate of 10 breaths per minute. Then, increase PEEP 3 cmH_2O every five breaths until the applied PEEP is between 25 and 35 cmH_2O, and the maximum PIP is between 40 and 50 cmH_2O. Ventilate at this level for 1 min. If the patient desaturates or becomes hypotensive at any point, stop and return to the prior PEEP. This illustration, Fig. 9.13, indicates the stepwise PEEP approach.

From here, the best compliance decremental PEEP trial should be performed. The next step is to change to volume control ventilation (VCV) at 4–6 ml/kg PBW and set PEEP at 20–25 dependent on patient severity of lung injury. The respiratory rate should be set to a rate that does not result in autoPEEP, usually 20–30 breaths/

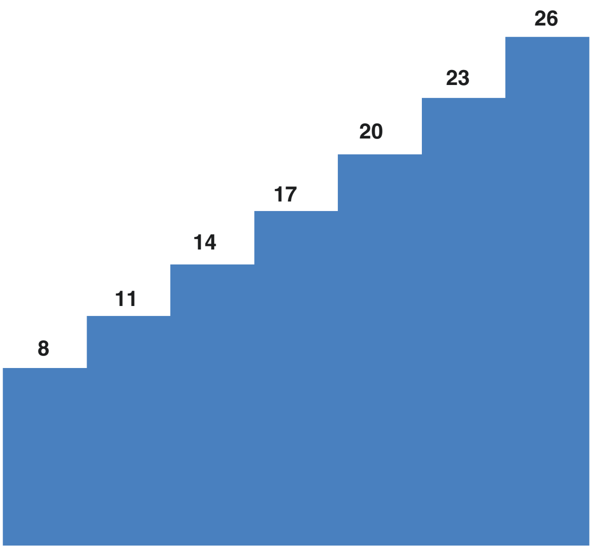


Fig. 9.13 Stepwise increase in PEEP for a gradual, gentle recruitment manuever

minute. Measure dynamic compliance, then decrease the PEEP by 2 cmH_2O, holding for 30 seconds at a time, and reassessing dynamic compliance each time. Initially the compliance will increase as PEEP is decreased, but with derecruitment, compliance will decrease. Once it is obvious that compliance is decreasing, the trial can be stopped. A clear pattern will indicate the PEEP with the best compliance. To set the ventilator, recruit the lung a second time, then set at the best PEEP +2 cmH_2O to optimize oxygenation as well. Figure 9.14 demonstrates the concept.

Below is an example from clinical practice in Fig. 9.15. The patient was placed on PEEP of 20, a tidal volume of 400, and a decremental best PEEP trial performed

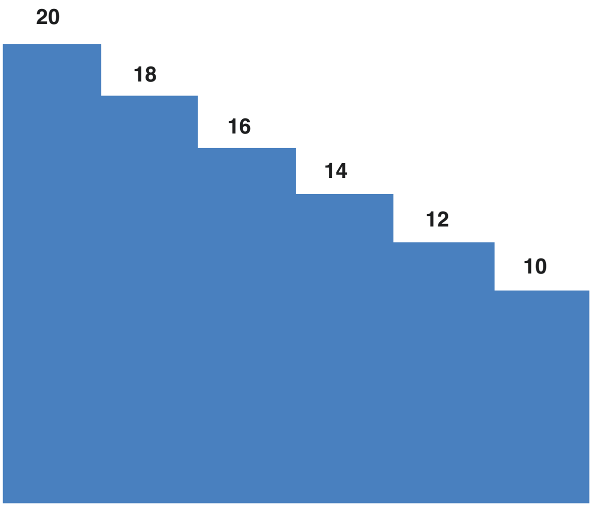
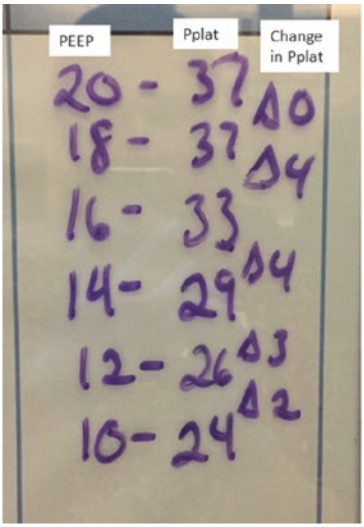


Fig. 9.14 Decremental decrease in PEEP illustrating a best PEEP trial



PEEP	Pplat	Driving Pressure	Compliance (TV 400mL)
20	37	17	23.5
18	37	19	21.1
16	33	17	23.5
14	29	15	26.7
12	26	14	28.6
10	24	14	28.6

Fig. 9.15 Data collected during a best PEEP trial, showing the change in driving pressure related to the change in compliance. In this example, a PEEP of 10 or 12 cmH_2O had the lowest driving pressure (14 cmH_2O). The PEEP was then set to 12 to optimize oxygenation

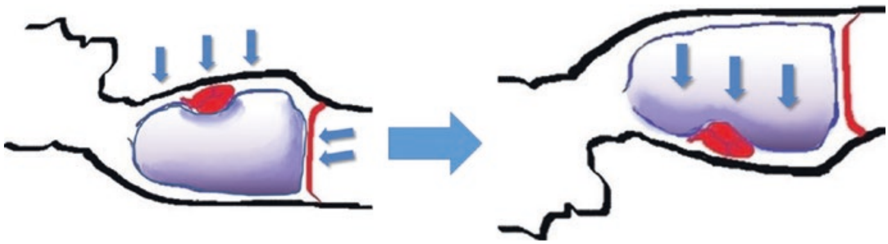


Fig. 9.16 The posterior aspect of the lungs holds a large surface area for gas exchange. Additionally, moving the heart anteriorly and off the lungs helps reduce atelectasis behind the heart. Coupled with mechanical changes in the chest wall, proning can significantly improve oxygenation

as described. The values were written on the patient's white board at the time of the testing, and a worksheet with the driving pressure and compliance were then filled in to determine the optimum PEEP. As both 10 and 12 had good compliance, 12 cmH_2O was selected in this example.

Proning

For patients with a PaO_2/FiO_2 ratio of less than 150, the next maneuver is proning the patient, or placing them in the proned position, to improve oxygenation to the posterior lungs (Fig. 9.16). Proning the patient improves V/Q matching and allows the patient to have gas exchange along the posterior aspects of the lungs. Proning has been shown to improve mortality in severe ARDS in a large multicenter study [9]. Additionally, patients with COVID-19 have been treated successfully with proning. However, this maneuver requires specialized expertise and a coordinated effort among providers to avoid dislodging the endotracheal tube and patient harm. If a patient has such severe hypoxemia that non-intensivists are considering proning, expert consultation should be sought.

Inhaled Pulmonary Vasodilators

Another consideration is the administration of inhaled pulmonary vasodilators, such as inhaled nitric oxide (not to be confused with nitrous oxide, the anesthetic agent) or prostacyclins, such as epoprostenol. Hypoxemic patients generally have heterogeneous lung pathology, with some damaged areas, not participating in oxygenation and ventilation, as well as some relatively unharmed areas that are doing the bulk of gas exchange. Inhaled pulmonary vasodilators will vasodilate the areas that are participating in gas exchange, effectively increasing blood flow to the good areas of the lung and allowing the ineffective areas to continue to have hypoxemic vasoconstriction. This principle is illustrated in Fig. 9.17.

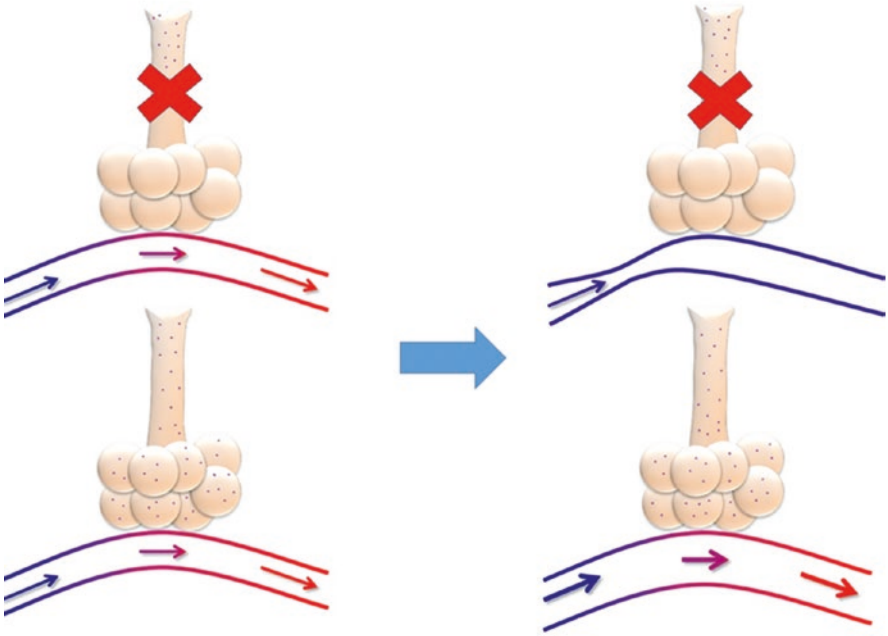


Fig. 9.17 Inhaled pulmonary vasodilators only reach the alveoli of lung units participating in gas exchange. They dilate the capillaries for these "good" lung units and thereby direct more blood flow to the areas participating in gas exchange

ECMO

Finally, patients with severe, refractory hypoxemia may be referred to an extracorporeal membrane oxygenation (ECMO) center for consideration of ECMO support.

The following ventilator screen in Fig. 9.18 illustrates settings for a patient with severe ARDS. The tidal volume is 400, which is appropriate for the patient's height and sex. The respiratory rate is 30, to maintain minute ventilation of approximately 12. A PEEP of 18 is required, and the patient is still on 100% FiO_2. Note that the PIP is 47 and the P_{plat} is 43. The clinicians can try to decrease the tidal volume; however, these values are so far from 30 cmH_2O, and it is unlikely that they will be able to achieve this goal.

The data for venovenous (VV) ECMO in severe ARDS not related to COVID-19 are mixed. The largest trial, the EOLIA trial [11], ECMO for severe ARDS was stopped early at 249/331 patients enrolled for predefined futility. There was no significant mortality benefit at day 60, but 28% of the conventional treatment group had crossover to ECMO rescue. This has led to a lot of controversy as to how the results of the trial should be interpreted. Although it is a negative trial, proponents of ECMO note that when patients from the control group received ECMO, it was

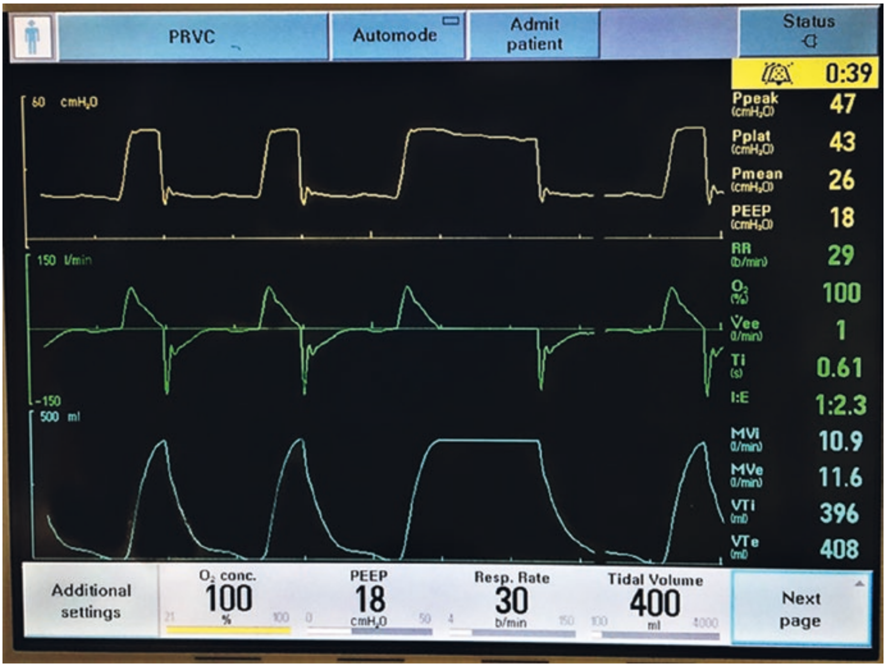


Fig. 9.18 In this example, the patient has severe, refractory ARDS. She is set on low TV ventilation, but her P_{plat} remains extremely elevated. Her driving pressure is extremely high at 25 cmH_2O (P_{plat} 43; PEEP 18), and her compliance is very poor (400 mL TV/25 cmH_2O = 16 mL/cmH_2O). Continuing to ventilate her on these settings will only cause further lung injury. Assuming no contraindications, this is a patient who should be considered for VV ECMO

started later when they were sicker, and seven crossover control patients even underwent VA ECMO for arrest. They also note that conventional treatment had a high rate of failure necessitating ECMO.

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10 Specific Circumstances: Asthma and COPD

In asthma, the patient has constriction of the bronchial smooth muscles in the airways, leading to reversible air trapping. This is indicated in the schematic of Fig. 10.1. Note that the bronchial muscles do not extend into the small airways.

Intubation of an asthmatic in the ED is a dreaded complication of this illness, as asthmatics can deteriorate rapidly on the ventilator without close monitoring and active management. The goal with a ventilated asthmatic is to prevent breath-stacking or autoPEEP and the hemodynamic instability that can result.

Before discussing the ventilator management of asthma, clinicians should note that intubation of an asthmatic should trigger even more active management with medications, rather than less. Intubated asthmatic patients should continue to receive aggressive treatment with bronchodilators, steroids, magnesium, as well as deep sedation and possibly even neuromuscular blockade in the initial hours after intubation, in an effort to relax the chest wall musculature and decrease the work of breathing. Please note that neuromuscular blockade only works on skeletal muscle and therefore will not bronchodilate smooth muscle in the airways. In addition, it is very critical to be aware of the patient's intravascular volume status, as the excess positive pressure can lead to hemodynamic collapse and cardiac arrest. Moreover, the excess pressure, including the auto-PEEP, can result in barotrauma, such as the development of a pneumothorax very quickly in this patient population.

Four ventilator maneuvers increase expiratory time, namely, decreasing the respiratory rate, decreasing the I:E ratio, decreasing the inspiratory time, or increasing the inspiratory flow. Of these, decreasing the respiratory rate is the most effective means to allow more time to exhale.

Figure 10.2 shows a schematic of 30 seconds with two patients, set with the same I:E ratio of 1:2. The first patient has a rate of 10 breaths per minute, allowing 6 seconds per breath cycle. The second patient has only 3 seconds per breath cycle, given the respiratory rate of 20. The blue represents inspiration and the red the time for exhalation. Note that even with the same I:E, the lower rate offers a substantially longer time to exhale.

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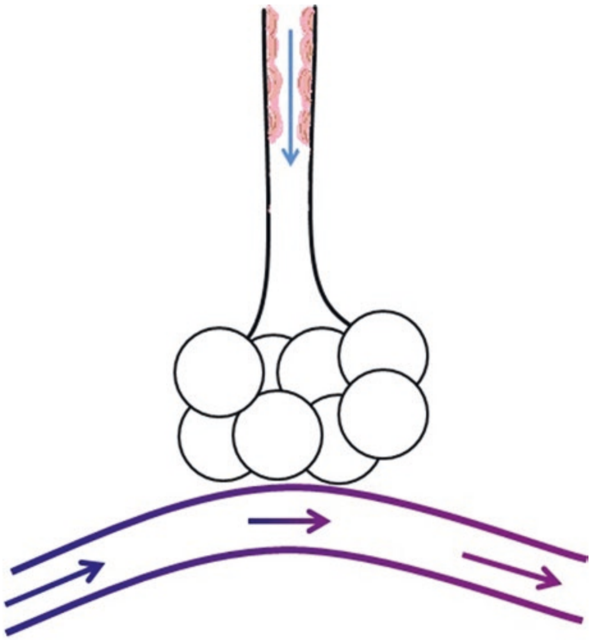


Fig. 10.1 In asthma, the patient has intermittent constriction of the smooth muscles of the bronchi, thereby limiting airflow

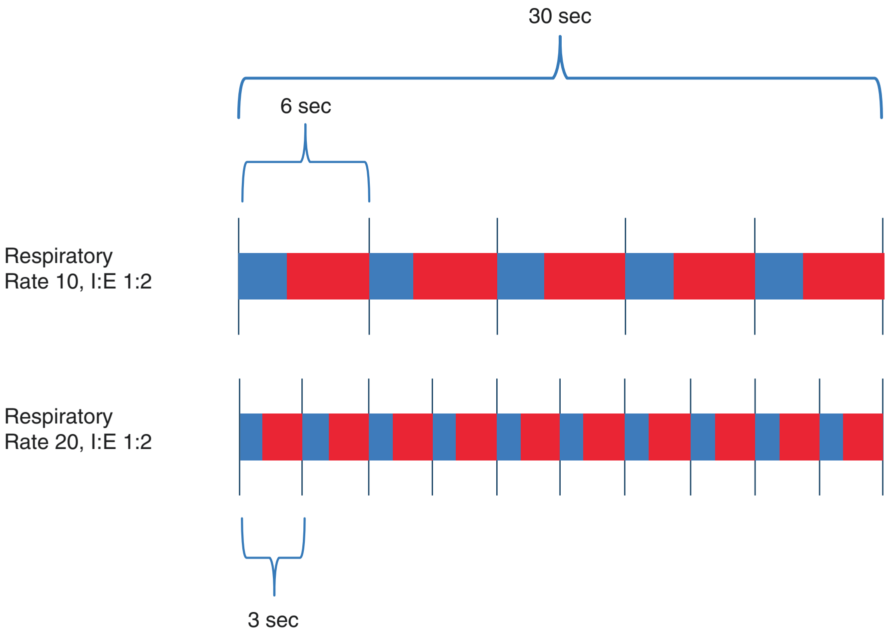


Fig. 10.2 In this figure, the red represents time to exhale. Not the dramatic effect of decreasing the respiratory rate from 20 to 10, even while all other parameters are held equal

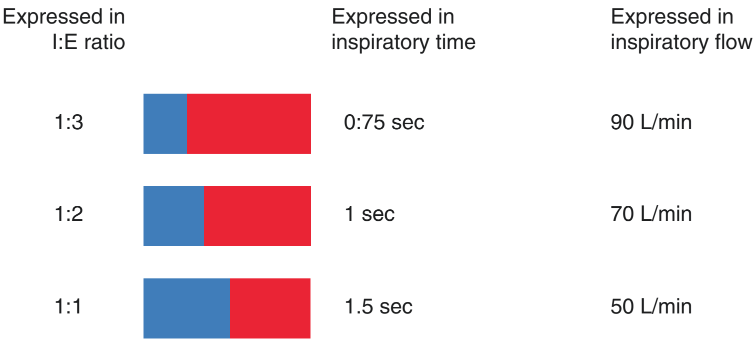


Fig. 10.3 This figure illustrates the relationship between the I:E ratio, the inspiratory time, and the inspiratory flow. Decreasing the I:E ratio, decreasing the inspiratory time, and increasing the inspiratory flow all provide more time for exhalation

In looking further at this diagram, one can imagine the effects of changing the I:E ratio, the inspiratory flow, or the I time. Fig. 10.3 shows a hypothetical example of the effects of these changes in a patient on volume control. In a given patient, the exact values will vary, but the purpose of the illustration is to show the relationship among the parameters of I:E, inspiratory time, and inspiratory flow.

In addition to a slow respiratory rate, a low I:E ratio, a short inspiratory time, and/or a fast inspiratory flow rate, asthmatics should also be ventilated with low tidal volumes. Considering that the larger the tidal volume, the more the patient needs to exhale, this is fairly intuitive.

In monitoring an intubated asthmatic, looking for signs of air trapping on the ventilator is key. In the ventilator tracing in Fig. 10.4, note that the flow tracing, the middle curve, does not return to the baseline before the next breath (red arrows). This represents that the patient is still exhaling when the next breath is given, which can be an early sign of air trapping. If you were caring for this patient, how would you address this air trapping?

In this patient, you could first decrease the respiratory rate or increase sedation if the patient is overbreathing. The I:E ratio is only 1:2, as noted on the right-hand side of the ventilator screen, so changing the I time to make a ratio of 1:3 or 1:4 is also appropriate. Also continued treatment with bronchodilators to decrease the bronchospasm associated with this disease will also mitigate the excess auto-PEEP (Fig. 10.5).

Recall that to quantify the pressure exerted by air trapping, one should check for autoPEEP by checking an expiratory hold button on the mechanical ventilator. In this tracing, what is the autoPEEP, or the intrinsic PEEP? What is the total PEEP?

The intrinsic PEEP is 11 cmH_2O, and the total PEEP is 12 cmH_2O. This indicates that the patient was only set on 1 cmH_2O of PEEP, which is an unusual setting, and was used in this circumstance for demonstration purposes only.

Thus, to set the ventilator for an asthmatic patient, select a low tidal volume of 6–8 mL/kg of predicted or ideal body weight. The respiratory rate should be low, less than 20 breaths per minute, and often around 10, to allow enough time for

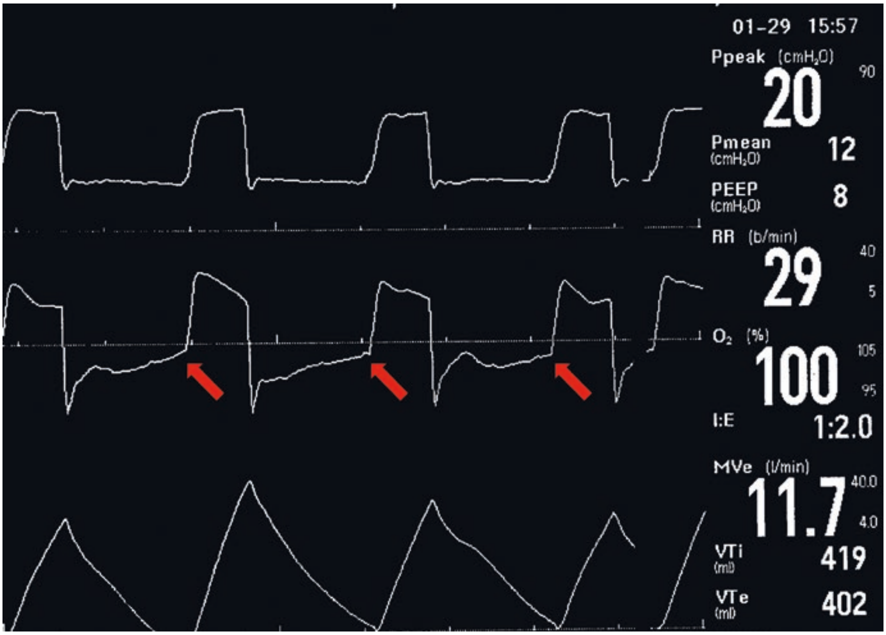
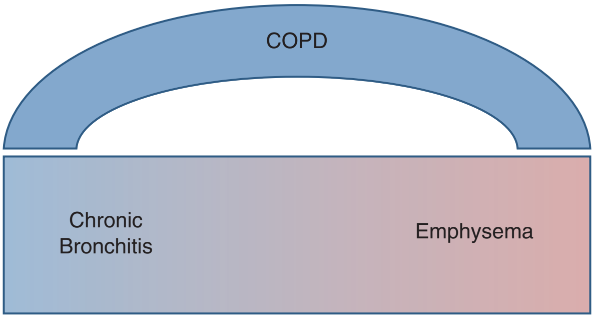


Fig. 10.4 In this image of a ventilator screen, the flow does not return to the baseline before the next breath is given, indicating that the patient is still exhaling when being forced to inhale. This creates air-trapping. Note that the quantity of air trapping cannot be determined by the flow waveform; this is qualitative data only

exhalation. The I:E ratio should be changed to 1:3 or less, which can be done by changing the respiratory rate or flow rate or directly altering this ratio on the ventilator. The PEEP should be set at 5 cmH_2O. The FiO_2 should be down-titrated as tolerated to maintain an adequate pulse oximetry (SpO_2) reading. These patients continue to receive sedation as needed and possibly neuromuscular blockage if required to cause chest wall muscle relaxation and prevent ventilator desynchrony. The patient should be treated with bronchodilators, such as continuous nebulizer treatments, magnesium to allow for smooth muscle relaxation, and steroids. Close monitoring for breath stacking and autoPEEP is required in this mechanically ventilated patient population. AutoPEEP should be monitored periodically or after any ventilator change with an expiratory hold, and changes should be made to the treatment algorithm and ventilator settings to combat the excessive PEEP. Arterial blood gases (ABGs) should be checked to ensure that the patient is being adequately ventilated and oxygenated.

Fig. 10.5 The amount of air trapping can be quantified by an expiratory hold. In this figure, the PEEP was turned to 1 cmH_2O (for illustration purposes only, not recommended for clinical practice). The total PEEP is 12 cmH_2O, and the autoPEEP, or intrinsic PEEP, is therefore 11 cmH_2O

Fig. 10.6 Both chronic bronchitis and emphysema fall under the umbrella term of COPD. Most patients with COPD will have aspects of both disease processes



COPD

There are two types of obstructive lung disease falling under the umbrella of COPD, namely, chronic bronchitis and emphysema (Fig. 10.6).

Chronic bronchitis can resemble the asthmatic schematic above, with the notable exception that muscle hypertrophy is not entirely reversible. Additionally, chronic bronchitis is associated with increased mucous production (Fig. 10.7).

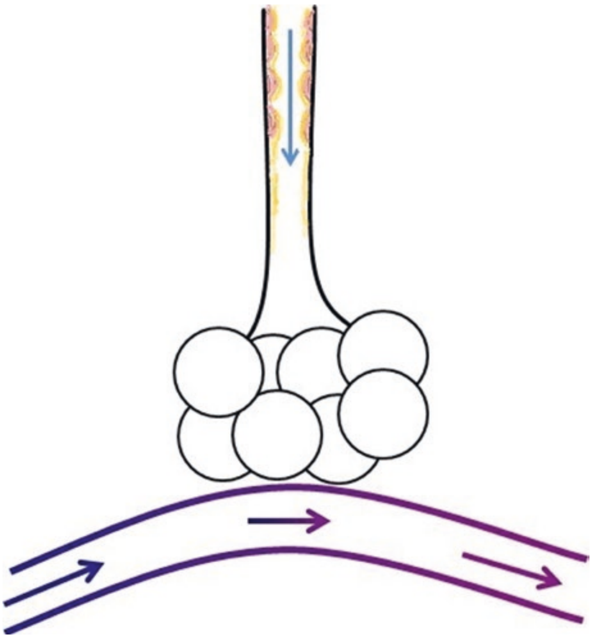


Fig. 10.7 In chronic bronchitis, the patient has narrowing of the airways with nonreversible hypertrophy and increased mucous production

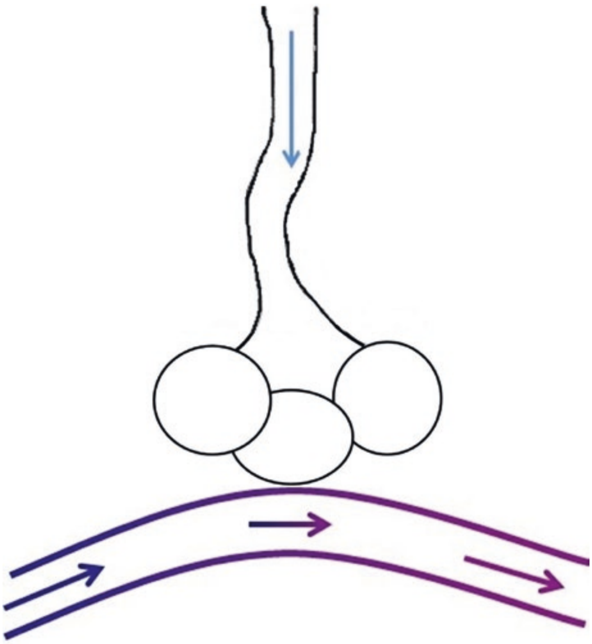


Fig. 10.8 In emphysema, there is parenchymal destruction, with loss of alveoli and collapsibility of the small airways

Emphysema is a disease of parenchymal destruction. Not only is there loss of alveoli, resulting in decreased surface area, or decreased diffusion area (leading to an increased diffuse capacity for carbon monoxide, or DLCO), but the small airways can become floppy due to the loss of other tissues holding them open (Fig. 10.8).

Understanding the pathophysiology of COPD is important for considering how to best ventilate these patients. It should be noted, however, that most patients with COPD have some mixing of elements of chronic bronchitis and emphysema. These conditions exist on a spectrum rather than a dichotomy.

Most patients with COPD are now managed with BPAP, with improved outcomes over intubation. However, on occasion, a patient with COPD is not a candidate for BPAP or fails to improve with a trial of BPAP, mandating intubation and invasive mechanical ventilation. Many of the principles that apply in mechanical ventilation for asthma also apply in COPD. Both are obstructive diseases, and in both processes, the patients require adequate time to exhale. Therefore, low tidal volumes, low respiratory rates, and low I:E ratios are appropriate. However, a key difference involves the role of PEEP.

Patients with COPD are at high risk of developing autoPEEP. Due to their obstructive disease, they require additional time to exhale. However, the mechanism of obstruction can differ between asthma and COPD, especially COPD with emphysematous changes as illustrated above. With the destruction of parenchyma, the small airways can collapse with exhalation, trapping air behind. In this circumstance, this trapped air leads to autoPEEP. Increasing the set PEEP, to match the autoPEEP, is not necessarily an intuitive solution. However, as illustrated by the diagram below, increasing the PEEP in this select patient population to prevent collapse of these small airways can allow the patient to exhale more fully (Fig. 10.9).

Reexamine the tracing of Fig. 10.5 from the asthma section, imaging that this patient has COPD (Fig. 10.10). If this patient has 11 cmH_2O of autoPEEP, or intrinsic PEEP, how would you adjust their extrinsic PEEP?

To match the autoPEEP, 11 cmH_2O would be an appropriate PEEP selection this in patient to help sent open the airways and allow for more complete exhalation. Remember however that this could lead to decreased preload and hemodynamic compromise in the hypovolemic patient, and an appropriate resuscitation may be required.

Finally, patients with COPD are often chronically hypoxemic. Physical exam findings of chronic hypoxemia can include nail clubbing. These patients may also have an elevated hemoglobin level on the CBC, indicating the patient's

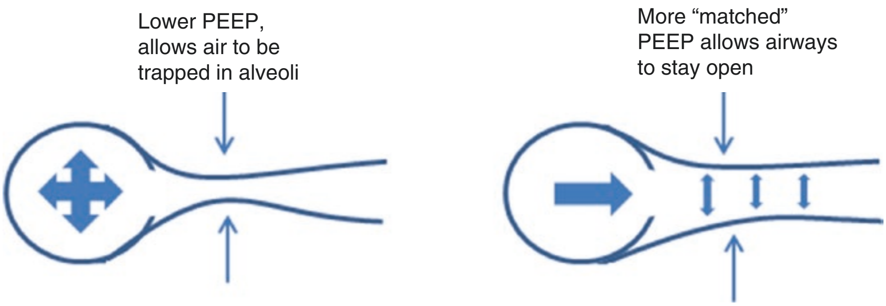


Fig. 10.9 This figure illustrates how increasing PEEP to match autoPEEP can help reduce air trapping. By maintaining the patency of the small airways, the patient is better able to exhale

Fig. 10.10 Illustration of an autoPEEP, or intrinsic PEEP, of 11

compensation for their chronic lung disease and thus avoiding central cyanosis. Because these patients are baseline hypoxemic, ventilation is often a relatively greater issue for them than hypoxemia, and therefore the oxygen saturation for a patient with COPD should be targeted a pulse oximetry (SpO_2) reading of ~88–92% in most circumstances. This is increasingly important as more data demonstrating the risks of hyperoxia continue to accumulate.

As with other mechanically ventilated patients, clinicians should obtain an ABG ~ 30 minutes after intubating a COPD patient to ensure adequate oxygenation and ventilation. In the appropriate clinical setting, such patients may be able to tolerate some hypercapnia, or permissive hypercapnia, to avoid preventing the drive to breath.

Suggested Reading

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11 Specific Circumstances: Neurologic Injury

Patients with neurologic injury requiring mechanical ventilation are an especially fragile patient population in the ED. Although EM clinicians cannot prevent the primary injury, as this has already occurred by the time the patient presents, preventing secondary injury is key. Patients with neurologic injury have specific needs for oxygenation, ventilation, and blood pressure management. In essence, the most important concept is to keep parameters normal—neither too high nor too low. These patients require very careful monitoring as the brain not only has no physiologic reserve but within the closed system is highly sensitive to small pressure changes that can be easily triggered by oxygen or carbon dioxide level changes.

Traumatic Brain Injury

Management of oxygenation and ventilation in patients with traumatic brain injury has been well studied. Numerous studies have shown that early proper management of patients with TBI can improve outcomes or, at least, reduce the risk of secondary injury [1–3]. Studies of prehospital intubation and ventilation have shown worse outcomes for patients with TBI, due in large part to the hyperventilation that can occur in a setting with less monitoring [3, 4].

TBI patients are at risk for increased intracranial pressure due to space occupying lesions and edema. Hyperventilation in the setting of TBI decreases CO_2 levels and subsequently results in vasoconstriction, as illustrated in Fig. 10.1, allowing for more space in the cranial vault for a traumatic lesion to expand without causing herniation. This maneuver is very effective and can give the patient time to get to definitive care, which may be placement of a ventriculostomy or even craniectomy—both of which allow for the elevated intracranial pressure to expand outward instead of causing herniation. However, another secondary effect of hypocapnia is decreased blood flow, which may put injured tissue at risk of ischemic injury, as illustrated in Fig. 11.1. The injured brain is most sensitive in the first 24 hours, so any maneuver that decreases perfusion can have more significant effects. In addition

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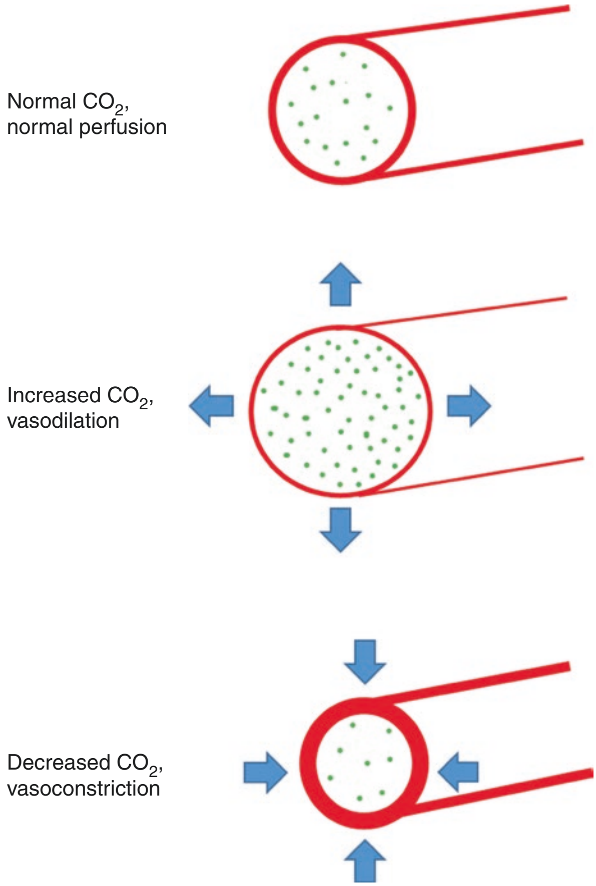

Fig. 11.1 This figure illustrates the effects of $PaCO_2$ on the cerebral vasculature. In the top image, the $PaCO_2$ is normal, around 40 mmHg. In the second, the concentration is much increased, as may be seen with hypoventilation. This leads to vasodilation and can increase intracranial pressure. The bottom illustration demonstrates a low $PaCO_2$, as would be seen with hyperventilation. This causes vasoconstriction and can lead to worsening ischemia in vulnerable areas of the brain

to this, the brain works to adjust the recognized alkalosis and as such brings the pH back to normal, resulting in vasodilation—this works against the patient with intracranial hypertension by taking up space and increasing the risk of herniation.

Although in past decades clinicians used to recommend hyperventilation of patients at risk for increased ICP, this is no longer recommended due to worsening outcomes with this hypoperfusion. Guidelines recommend targeting a normal $PaCO_2$ of 35–40.

Prior to intubation, ventilation with a bag-valve-mask should be minimized, to reduce the risk of unintentional hyperventilation. If BVM is used, it is advisable to be vigilant about avoiding forceful bagging technique and maintaining a low rate of breaths. Minute ventilation of 7–8 liters/min is appropriate to start, recognizing that the trauma patient is likely to be hypermetabolic.

All patients with neurologic injury are at risk for ARDS. In other patient populations, permissive hypercapnia is accepted to maintain low tidal volumes and allow for adequate time to exhale. However, permissive hypercapnia is not appropriate in neurologically injured patients, and the ventilator should be adjusted accordingly.

Capnography can be very useful in this population. As the capnography is started, checking an ABG is beneficial to correlate the partial pressure of carbon dioxide ($PaCO_2$) with the end-tidal CO_2 ($ETCO_2$). Some patients, especially those with chest trauma or underlying lung disease, may have substantial dead space, leading to a larger than anticipated discrepancy between the two. Normally, the difference should be about 5. Once the relationship is established, the $ETCO_2$ can be followed for trends, assuming no significant changes in the pulmonary status.

Hypoxemia in TBI is also associated with worsened outcomes and secondary injury, but clinicians may not realize that numerous studies have shown that patients are frequently hyperoxygenated in emergency scenarios following intubation. This hyperoxia is also deleterious, with a supposed mechanism of worsening repercussion injury and free radical production. Therefore, normoxia is the goal, and this is another reason an ABG should be checked 15–20 minutes after intubation. The FiO_2 should be lowered, targeting a PaO_2 75–100, for a corresponding O_2 saturation of 95–99%, depending upon the individual oxygen-hemoglobin dissociation curve.

TBI patients are also very susceptible to further injury from hypotension and hypertension. With intubation and initiation of ventilation, the clinicians should take care to manage hemodynamics aggressively. Volume-depleted patients should be resuscitated with fluids or blood, as indicated by the circumstance, and any patient with hypotension or at risk for hypotension should receive vasopressors to maintain cerebral perfusion pressure. Similarly, laryngeal stimulation may at times lead to hypertension, and if blood pressure becomes too high, consider sedation and opioids as first line agents. It is rare that the patient with TBI will require antihypertensive medication, as the brain is in most cases autoregulating to maintain cerebral perfusion.

Ischemic Stroke

Patients with ischemic stroke may require intubation and mechanical ventilation for a variety of reasons, including the need for airway protection, respiratory failure after aspiration, or need for invasive procedures. While the need for intubation in ischemic stroke portends a poor prognosis, it is imperative that the emergency clinician prevent secondary injury to the vulnerable area of the brain, the penumbra, to the greatest extent possible.

Just as with TBI, patients with ischemic stroke are at risk for hypocapnia-induced vasoconstriction, leading to secondary ischemia. This vasoconstriction can then worsen outcomes by decreasing perfusion to the penumbra. Similarly, hypercapnia should be avoided to reduce vasodilation and the consequent increase in intracranial pressure. Therefore, clinicians should target normal $PaCO_2$ parameters of 35–45 mmHg [5]. The risk of increased ICP in an acute ischemic stroke is lower than in TBI, allowing a more liberal $PaCO_2$ target. Low tidal volume ventilation should be initiated, with a goal of 6–8 mL/Kg of predicted body weight. The MV should be targeted to 5–6 L/min, as these patients are less likely to be hypermetabolic than those with TBI.

Both hypoxia and hyperoxia can be damaging to the patient with ischemic stroke. Current guidelines recommend maintaining oxygen saturation greater than 94%, but do not provide a recommended upper limit [6]. Because hyperoxia has been associated with increased mortality in ischemic stroke patients [7], the minimum FiO_2 required to maintain an O_2 saturation of 95% or above should be used. The value of ABGs in the neurologic injury population is not only to evaluate hypoxemia but also to evaluate for hyperoxia, which is not readily detected with pulse oximetry.

Hypotension must be assiduously avoided in the ischemic stroke patient. These patients are at risk of dehydration, and volume repletion before intubation, if possible, is advisable. Allowing for hypertension is advised until vessel recanalization is performed (if this is planned) in order to perfuse penumbral tissue at risk for progression to infarction. However, hypertension increases the risks of hemorrhage, especially in cases of intravenous thrombolysis, and the target should be less than 180/105.

Spontaneous Intracerebral Hemorrhage

The principles of management for patients with intracerebral hemorrhage are similar to patients with TBI and ischemic stroke. These patients are similarly susceptible to the ischemic effects of hypocapnia from hyperventilation [8]. Because the risk of increased intracranial pressure is higher in ICH than ischemic stroke, targeting a $PaCO_2$ of 35–40 mmHg is reasonable. Hyperoxia has been associated with increased mortality in this population as well. Patients with intracranial hemorrhage are at risk of developing ARDS and, as such, should also be ventilated with low tidal volumes of 6–8 mL/Kg predicted body weight. Close monitoring with ABGs and capnography is mandatory.

As with other patients with neurologic injury, the blood pressure can vary widely, and maintaining normal perfusion is key. The clinicians should be aware of the risks of hemodynamic lability with intubation and initiation of ventilation and be prepared to treat hypertension or hypotension rapidly, using push dose agents as needed. Blood pressure goals for intracerebral hemorrhage should be at least <180 mmHg, but in some cases, such as patients with no baseline hypertension, <140 mmHg may be appropriate [9].

Status Epilepticus

Patients in status epilepticus requiring intubation have a few unique challenges. If possible, only short-acting paralytics should be used with intubation to minimize obscuring the exam. Recall that these patients will be hypermetabolic, with a lactic acidosis, and the MV should be increased accordingly, likely starting at least at 8–10 L/min. Acid-base status should be followed closely to reduce the risk of secondary injury from additional metabolic insults. The core concepts are highlighted in Table 11.1.

Table 11.1 Goals for mechanical ventilation in neurologic injury

Traumatic brain injury	Ischemic stroke	Intracerebral hemorrhage	Status epilepticus
$PaCO_2$ 35–40 mmHg	$PaCO_2$ 35–45 mmHg	$PaCO_2$ 35–40 mmHg	$PaCO_2$ 35–45 mmHg
MV 7–8 L/min	MV 5–6 L/min	MV 6–7 L/min	MV 8–10 L/min
PaO_2 75–100 mmHg	PaO_2 75–100 mmHg	PaO_2 75–100 mmHg	PaO_2 75–100 mmHg
O_2 saturation 95–99%	O2 saturation 95–99%	O_2 saturation 95–99%	O_2 saturation 95–99%
SBP >100 or >110	SBP 140–180	SBP <140 to <180	Varies by etiology

$PaCO_2$ partial pressure of oxygen, *MV* minute ventilation, *PaO_2* partial pressure of oxygen, *SBP* systolic blood pressure

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Troubleshooting the Ventilated Patient 12

Patients in the Emergency Department (ED) are at high risk for deterioration after intubation. Understanding the common causes of ventilator pressure alarms and having a systematic approach to their evaluation and treatment is key to acting in these high-stress situations.

When the ventilator loses pressure, the *low-pressure alarm* will go off. The differential for the low-pressure alarm includes a break in the circuity, anywhere from the ventilator to the lungs. Causes can include the following:

- A disconnection of the ventilator tubing and endotracheal tube (ETT).
- Increased patient effort—air hunger, "sucking in" during breaths.
- ETT displacement or extubation—the ETT tip may be right at the level of the vocal cords, and therefore, the patient is not yet desaturating.
- The ETT cuff may have a leak, allowing the delivered air to escape.
- Although less likely, a hole may also be present somewhere in the circuit.

The *high-pressure alarm* similarly can arise from an issue anywhere from the patient to the ventilator.

- If the patient is fighting or "bucking" while on the mechanical ventilator, the peak airway pressure can rise.
- Elevation in autoPEEP.
- Any impedance to airflow, such as a mainstem intubation, bronchospasm, a pneumothorax, or a mucous/bloody plug in the ETT or airways can also lead to elevated pressures.
- A disruption in the ETT, such as kinking or biting of the tube may be responsible.

An alarm on the vent should NEVER be ignored, and these alarms should be thought of as a pre-code situation. Ventilated patients are among the highest risk for deterioration in the ED, and they must be attended to promptly for both high-pressure and low-pressure alarms. It is important in such situations to

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Fig. 12.1 DOPES mnemonic for assessing cause of alarm or deterioration for an intubated patient

Differential for ventilator alarms:

D - Dislodged tube (check with $ETCO_2$, direct visualization)

O - Obstructed tube (mucus plug, blood, kink)

P - Pneumothorax

E - Equipment failure (ventilator, tubing, ETC)

S - Stacked breathing (autopeep)

assemble your team to fully assess the patient, including the assistance of a Respiratory Therapist (RT) if available.

To quickly assess for issues, clinicians should recall the DOPES mnemonic, outlined in Fig. 12.1. Running quickly through this mnemonic will remind clinicians to consider common causes of deterioration while on the ventilator.

A separate, but related mnemonic, DOTTS, reminds clinicians how to respond to a ventilator alarm. Although the type of alarm (low pressure or high pressure) will influence the differential, the immediate actions are all the same. The first maneuver disconnecting the patient from the ventilator immediately takes much of the equipment out of the equation. The patient should then be connected to 100% oxygen via a bag and ventilation provided by bagging. Consider adding a PEEP valve to the bag valve mask (BVM) in patient who require ≥5 cmH_2O of PEEP to prevent decruitment. Bagging a patient with a BVM can be diagnostic, allowing the clinician to assess if there is excess resistance to bagging and about the compliance of the lungs with this maneuver.

The next maneuver is to pass a flexible suction catheter down the ETT after preoxygenation, checking patency of the tube and relieving any obstruction. Suctioning, in the case of a mucous plug, may also be therapeutic.

Once the above steps are performed, the clinical should be able to generate a differential diagnosis for the possible causes of the ventilator alarm, as shown in Fig. 12.2. As indicated, the clinician can tweak the ventilator to improve oxygenation and/or ventilator or if autoPEEP is thought to be an issue. A bedside US to assess can be performed for assess for lung sliding if a pneumothorax is suspected.

To review, a low-pressure alarm is due to a break in the ventilator circuitry, as outlined above.

To treat high-pressure alarms, the algorithmic approach should be taken to generate a differential diagnosis, while treating possible causes. Once the DOPES and DOTTS pathways have been evaluated, the clinical should review the airway pressures on the ventilator to generate additional information.

An elevation in the peak airway pressure (P_{peak}) with a normal or low plateau pressure (P_{plat}) is suggestive of increased resistance in the system. The issue is often

Approach to ventilator alarms:

D - Disconnect the patient from the ventilator, provide gentle chest pressure

O - Oxygen 100% by manual bagging; check compliance by squeezing the bag.

T - Tube position/function (pass a suction catheter)

T - Tweak the vent

S - Sonography (PTX; mainstem intubation; plugging)

Fig. 12.2 DOTTS mnemonic for steps in assessing and treating the deterioration of an intubated patient

Fig. 12.3 Causes of elevated Peak airway pressure (P_{peak}) and airway resistance

Increased Airway Resistance
Ventilator desynchrony, “bucking” the ventilator
Bronchospasm
Mainstem intubation
Pneumothorax
Kinked or obstructed ETT
Mucus plugging
ETT too small
Water in ventilator tubing

located from the ventilator to the end of the ETT. As noted in Fig. 12.3, the differential diagnosis can include mucus plugging, a kinked or small or obstructed ETT, water in the ventilator tubing, or bronchospasm.

Alternatively, an elevation in the P_{plat} would suggest issues with compliance, or a problem from the ETT to the diaphragms. The clinical should consider problems with the chest wall, pleura, pleural space, and lung parenchyma. Figure 12.4 reviews some of the causes of an elevated P_{plat}.

Finally, with an elevation in both the Plat and P_{peak}, with the difference between P_{peak} and Plat ≥5 cmH_2O suggests both resistance and compliance problems. Bronchospasm could cause such ventilator alarms, and this could be confirmed by pressing the expiratory pause button and reviewing the flow curves as review in prior chapters.

Let’s review a case to review and apply these concepts.

You are treating an 85-year-old woman with a history of COPD, heart failure, hypertension, and diabetes, who arrived at your ED by EMS after a witnessed

Lungs	Chest wall
Mainstem intubation	Obesity
Pneumonia	Multiple rib fractures, flail chest
Pulmonary edema	Circumferentrial burns
Adult respiratory distress syndrome (ARDS)	Abdominal compartment syndrome
Pulmonary fibrosis	Scoliosis
Asthma/COPD, bronchospasm	Supine position
Pulmonary embolus	
Pneumothorax, hemothorax	

Fig. 12.4 Causes of altered airway compliance and an evaluated Plat

syncopal episode requiring intubation in the field. As per your EMS colleagues, the patient complained of shortness of breath 3 days prior and was evaluated by a cardiologist with an increase in her diuretic dose. She collapsed onto the couch at home today.

Upon arrival to the ED, the patient is being bagged by EMS. You confirm that the ETT cuff is past the cords, and the patient is transitioned onto your stretcher. She has a strong pulse, with her initial vital signs: BP 101/58 mmHg, HR 85 bpm (on metoprolol), RR bagged, SpO_2 100%, and temperature 97.6 °F. She has breath sounds bilaterally, with end-expiratory wheezing in all lung fields. Her trachea is midline. You place the patient onto your hospital ventilator with the following settings:

$$\text{AC / VC, TV 350mL}\left(\text{6mL / kg}\right)\text{, RR 12, PEEP 5 cmH}_2\text{O, FiO}_2\text{ 100\%}$$

Immediately you note the high-pressure alarm, and note that the patient is only receiving a TV ~ 100 mL. Consider your next steps.

You auscultate again, this time hearing minimal to no air movement bilaterally. Following the DOPES mnemonic, you check the ETT placement again with direct laryngoscopy and confirm that the ETT cuff is beyond the cords. Concerned about right mainstem placement, you retract the ETT slightly, without any improvement in her breath sounds but with the ETT cuff remaining past the vocal cords. You suction her ETT without any return. You perform a bedside ultrasound with good lung sliding bilaterally, making a pneumothorax less likely. You disconnect her from the ventilator and resume bagging the patient. The RT tells you that it is very hard to bag the patient.

You then notice that the patient is becoming more hypotensive with systolic blood pressures in the 70s. Concerned about a pre-code situation, you ask the RT to stop bagging the patient and disconnect her from the oxygen source. You apply a gentle pressure to her chest wall and hear a very long sign of exhalation. You then reconnect the patient to the ventilator and notice that while her blood pressure and oxygen levels have improved, the flow curve does not return to baseline. Applying the expiratory pause button, her intrinsic PEEP is 10 cmH_2O. You decide to treat the patient with bronchodilators, magnesium, steroids, and increase her PEEP to 10 cmH_2O, being mindful of her preload as she previously became hypotensive with increased airway pressures. You also search for other causes of her shortness of breath and admit the patient to the ICU.

Suggested Reading

Archambault PM, St-Onge M. Invasive and noninvasive ventilation in the emergency department. Emerg Med Clin North Am. 2012;30(2):421–49, ix.

Wood S, Winters ME. Care of the intubated emergency department patient. J Emerg Med. 2011;40(4):419–27.

Case Studies in Mechanical Ventilation 13

Case 1

A 64-year-old woman presents to the ED with one day of feeling unwell, with myalgias, HA, nausea, and fevers. The patient is in respiratory distress. Her vitals are Temp: 36.6C (98F), HR 121, BP 89/45, SpO_2 82% on room air. Her chest radiograph is shown in Fig. 13.1.

1. What are the options for respiratory support for this patient? What are the risks and benefits of each?
2. You elect to intubate the patient. How would you set this patient's ventilator? What other information would you need to know?
3. What are your goals for ventilating this patient?
4. Does this patient have ARDS? How can you tell?

Fig. 13.1 CXR for Case 1

S. R. Wilcox et al., *Mechanical Ventilation in Emergency Medicine*,
https://doi.org/10.1007/978-3-030-87609-8_13

Fig. 13.2 Ventilator screen for Case 1

5. The ABG is 7.14/54/69 on 100%. How do you interpret this ABG?
6. Figure 13.2 shows the patient's ventilator screen.
   (6a) What is the TV?
   (6b) What is the PEEP?
   (6c) What is the PIP?
   (6d) What is the P_{plat}?
7. What changes would you make to this ventilator?

Case 2

A 56-year-old man with a long history of smoking about two packs per day, with suspected COPD, presents with wheezing, shortness of breath, and chest tightness. The EMS team gave him bronchodilators, but he arrives in the ED in extremis. His vitals are Temp: 36.6C (98F), HR 115, BP 160/82, and an SpO_2 87% while receiving nebulized albuterol with oxygen.

1. What are the options for respiratory support for this patient? What are the risks and benefits of each? What are absolute and relative contraindications to these types of support?

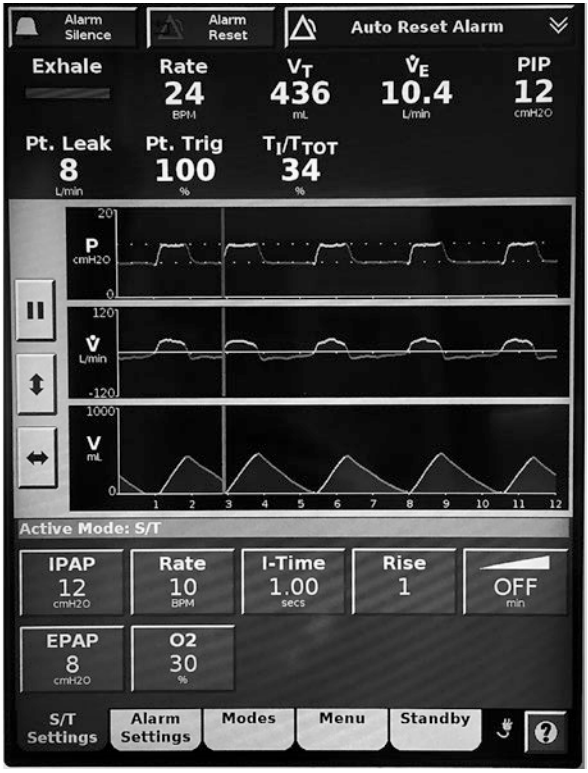

Fig. 13.3 Ventilator screen for Case 2

2. How would you select ventilation settings for this scenario? What are your goals of respiratory support?
3. How do you assess the adequacy of ventilation on noninvasive ventilation?
4. Figure 13.3 shows the patient's ventilator screen.
   (4a) What is the IPAP?
   (4b) What is the EPAP?
   (4c) What do each of those mean?
   (4d) What is the tidal volume?
   (4e) What is the minute ventilation?
5. The patient has an ABG of 7.25/75/68 on the BPAP. How would you interpret this ABG in this scenario?
6. The patient is awaiting transport to the ICU when the alarms in the room start to go off. The repeat vitals are BP 85/45, HR 130, RR 30, SpO_2 85%. How would you approach this emergency?

Case 3

A 24-year-old man with a long history of poorly controlled asthma presents to the ED with increasing shortness of breath after an upper respiratory infection. You use all the appropriate pharmacologic measures, but the patient is beginning to look fatigued and remains in distress.

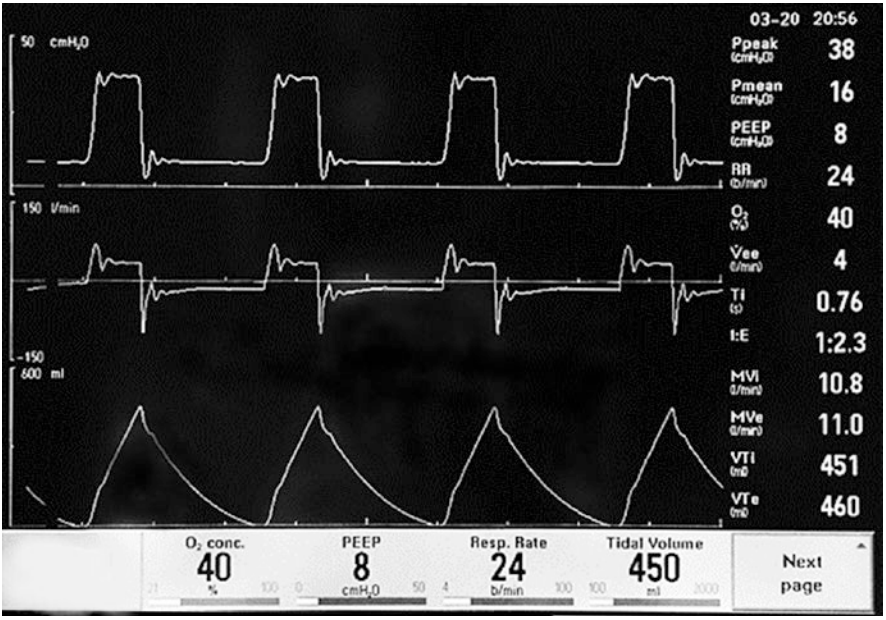

Fig. 13.4 Ventilator screen for Case 3

1. What are the options for respiratory support for this patient? What are the risks and benefits of each?
2. You select to intubate the patient. How would you set this patient's ventilator? What other information would you need to know?
3. Figure 13.4 shows the patient's ventilator screen.
   (3a) What is the TV?
   (3b) What is the PEEP?
   (3c) What is the PIP?
   (3d) What is the I:E ratio?
4. Do you think this patient has a compliance problem or a resistance problem? How could you tell?
5. Can you quantify air trapping?
6. What changes would you make to these ventilator settings?
7. While awaiting his ICU bed, the low-pressure alarm starts to go off on the ventilator. How do you address this?

Case 4

A 28-year-old man presents after a single-passenger MVC. He is obtunded on arrival with gurgling respirations and a GCS of 5. He is intubated promptly after arrival in the ED.

1. The respiratory therapist asks you if she should put him on the ventilator now or if she should keep bagging the patient, since he is probably going to CT scan soon. What do you say? Why?

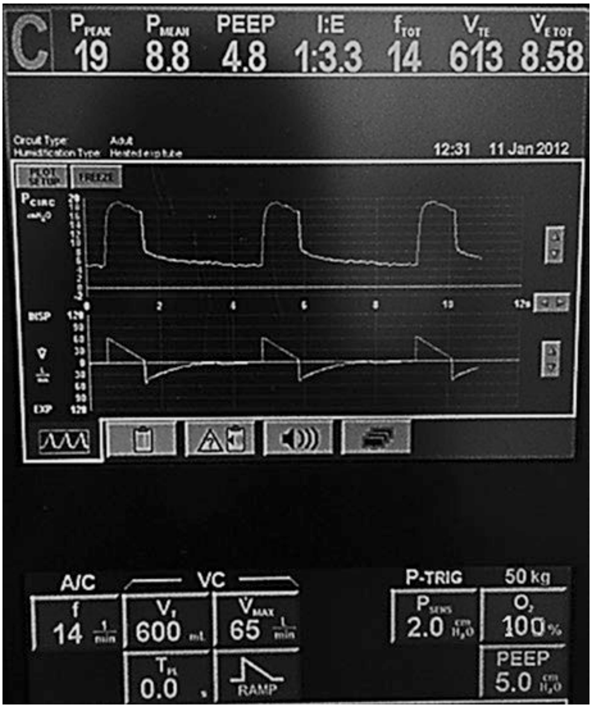

Fig. 13.5 Ventilator screen for Case 4

2. How would you set the patient's ventilator when you do put him on the vent? What additional data would you like to know?
3. Figure 13.5 shows the ventilator screen.
   (3a) What is the TV?
   (3b) What is minute ventilation?
   (3c) What is the PIP?
4. His ABG returns as 7.54/28/225.
   What changes would you make to these vent settings?
5. The patient's head CT shows a large subdural hematoma. Would that change your approach to your ventilator settings? Would you lower the PEEP?
6. If this patient developed a hemothorax, what changes would you expect to see on the ventilator?

Case 5

50ish year old man with an unknown past medical history presents with 2–3 days of progressive shortness of breath. His room air saturation in triage is in the mid-70s, and he is in too much distress to give much history. A nonrebreather is immediately placed, and his oxygen saturation increases to low-80s. He is started on BiPAP while the intubation equipment set up. His saturation remains in the mid-80s despite the BiPAP. He is intubated, and his SpO_2 remains in the mid-80s.

1. His chest radiograph is shown in Fig. 13.6. What is the physiologic reason his saturation remains persistently low?
2. What are the causes of this phenomenon? His chest radiograph is shown in Fig. 13.6. What is on your differential for this particular patient?
3. His ventilator screen is shown in Fig. 13.7.

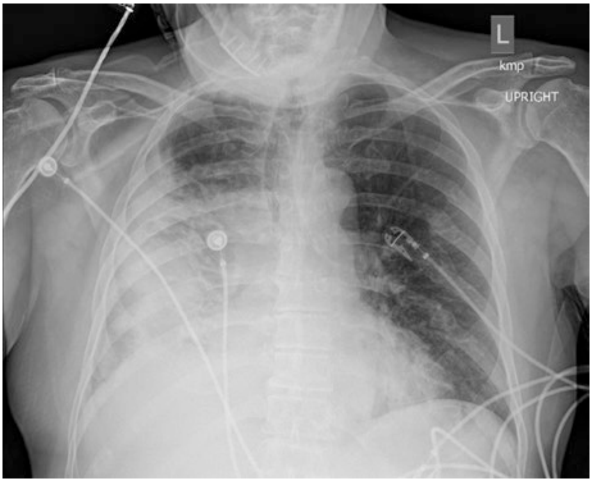

Fig. 13.6 CXR for Case 5

Fig. 13.7 Ventilator screen for Case 5

   (3a) What is his tidal volume?
   (3b) What is his respiratory rate? Is he overbreathing? How do you know?
   (3c) What is his minute ventilation?
   (3d) What maneuver is being performed in this picture? What does this tell us?
4. Does he have a problem with: compliance, resistance, both, or neither?
5. What procedures might you consider now?
6. What adjunctive therapies may provide some benefit? What benefits?

Case Study Answers

Case 1

1. There are three options for respiratory support for this patient.
   1. The patient could be trialed on high flow nasal cannula. The benefit of this is that it provides excellent noninvasive support for oxygenation. The downside is that if the patient develops shock, or any other form of instability, high flow nasal cannula will not be sufficient. Additionally, some studies have shown that the more severe the hypoxemia, the less likely the patient is to do well on high flow nasal cannula. As we see later in the case, the patient has severe hypoxemia, and as such, this is not an ideal option for her.
   2. Noninvasive positive pressure ventilation. This is an excellent way to oxygenate and ventilate a patient as well, especially for patients with underlying COPD or cardiogenic pulmonary edema. However, noninvasive ventilation has not been shown to improve outcomes in patients with de novo respiratory failure (i.e., without prior underlying cardiopulmonary disease.) Additionally, if the patient has any alterations mental status or develops shock, the patient will need to be intubated.
   3. Intubation and mechanical ventilation. While this method has the downside of being the most invasive, for a patient in septic shock, this is often the most reasonable option. Invasive mechanical ventilation allows for control of the volumes and pressures the patient receives.
2. This patient should be set on low tidal volume ventilation, with a goal of 6–8 mL/kg of predicted body weight. Her major issue is that she is hypoxemic, and therefore, she should have adequate PEEP support. The additional data point that is important is to know her height so that her predicted body weight can be calculated.
3. The goals for ventilating this patient are to maintain her on low tidal volume ventilation, keeping her plateau pressure less than 30 cmH_2O. The PEEP should be set to maintain adequate oxygenation while optimizing compliance, trying to minimize derecruitment that occurs with sedation after intubation. The FiO_2 should be decreased as soon as possible, targeting and oxygen saturation of 92–96%. This patient is appropriate for permissive hypercapnia to allow for low tidal volumes and pressures.

4. This patient meets the criteria for ARDS, as she has an acute process, she has bilateral infiltrates on her chest X-ray, and she has a PaO_2/FiO_2 ratio of less than 300 based on the ABG data. We do not have information about her cardiac function, but we have no reason to suspect that this presentation is fully explained by cardiogenic pulmonary edema. As such, this patient has severe ARDS, with a PaO_2/FiO_2 of 69.
5. In addition to the severe ARDS as indicated by her PaO_2/FiO_2 ratio of 69 (69/1.0), she also has a combined metabolic and respiratory acidosis given that she has a mild hypercapnia at 54 mmHg but has a substantial acidemia at 7.14.
6. (6a) The TV is set at 380 mL. Her last inspiration was 392 mL, and her last exhalation was 366 mL.
   (6b) 10
   (6c) 45
   (6d) 37
7. This is a challenging question because the patient is already presumably on a low tidal volume at 380. Her peak inspiratory pressure and plateau pressure are both very elevated. Increasing PEEP may help improve her compliance. If the patient has large areas of derecruited lung, performing a recruitment maneuver and increasing her PEEP may open previously atelectatic lung units and thereby improve compliance. Increasing her tidal volume is not an option as her pressures are already so high.

   One method to improve her oxygenation after optimizing her recruitment is proning. She should be proned if her PaO_2/FiO_2 remains less than 150. If her compliance and oxygenation cannot be improved with less invasive means such as recruitment and proning, she may be considered for ECMO cannulation, as she is already receiving maximal ventilatory support.

Case 2

1. For patient was COPD, the target oxygen levels are often 88–92%. Therefore, while this patient is certainly hypoxemic, his increased work of breathing is a greater concern that his relatively mild to moderate hypoxemia. High flow nasal cannula may help with management of mild hypercapnia, and improving oxygenation can sometimes decrease the work of breathing. However, with the patient's suspected COPD, noninvasive ventilatory support is likely a better option. Noninvasive ventilatory support has been shown to improve outcomes in patients with COPD. This patient could be intubated and mechanically ventilated; however, unless the patient has a contraindication, most patients with COPD should be trialed on bilevel positive pressure ventilation first. The absolute contraindications to high flow nasal cannula include airway compromise. The absolute contraindications to noninvasive ventilatory support are airway compromise, severely altered mental status, recent ENT/upper GI surgeries, small bowel obstruction, or other pathology that will put the patient at high risk for vomiting.

2. When initiating bilevel noninvasive support, one will often begin with relatively low settings of 10/5 cmH_2O. The patient's resultant tidal volume, respiratory rate, and overall comfort can then be reassessed. A blood gas should be checked approximately 15–30 minutes after initiation of support to ensure that the patient is trending toward improvement.
3. The noninvasive ventilator will provide a tidal volume and a minute ventilation just as with the patient on invasive mechanical ventilation. In addition to monitoring these values, monitoring the patient clinically; looking at the oxygen saturation, the respiratory rate, the work of breathing, and the accessory muscle use; and checking blood gases are important to ensure the adequacy of ventilation.
4. (4a) 12
   (4b) 8
   (4c) In BPAP, on noninvasive ventilation, the inhaled positive airway pressure (IPAP) is equivalent to the PIP in invasive ventilation. The expired positive airway pressure (EPAP) is equivalent to PEEP or CPAP. In this example, the EPAP of 8 is the baseline pressure. With every breath, the patient receives an additional 4cmH_2O of support, for a total of 12 cmH_2O.
   (4d) 10.4 L/min
5. The patient has a chronic respiratory acidosis with a superimposed acute respiratory acidosis. The patient also has some hypoxemia with a partial pressure of oxygen of 68 mmHg on 30% FiO_2.
6. The DOPES and DOTTS mnemonic devices are designed for patients who are intubated. However, similar concepts can apply to the patient on positive pressure ventilation. DOPES begins with displacement, which is not relevant in this scenario. However, obstruction, pneumothorax, equipment failure, and stacking provide a reasonable start for the building a differential diagnosis. Similarly, the patient does not necessarily have to be disconnected and bagged with 100% oxygen; however, taking the patient briefly off the noninvasive ventilation, talking to the patient, assessing the patency of the airway, considering obstruction, and listening for bilateral breath sounds for a possible pneumothorax are all reasonable steps to complete within the first 30 seconds of assessing the patient.

Case 3

1. This case with the patient presenting in respiratory failure with severe asthma. The patient has reactive airways disease leading to an obstructive process. As oxygenation is not his primary issue, high flow nasal cannula is unlikely to be the best option. It is reasonable to try the patient on noninvasive positive pressure ventilation, with an understanding that if the patient does not respond promptly, he will require intubation. The downsides of noninvasive ventilation with asthma are that the patient is at risk for air trapping and cannot be heavily sedated with noninvasive ventilation. Additionally, if the patient is starting to fatigue or developing altered mental status, noninvasive ventilation is not an appropriate means of supporting the patient. The patient can be intubated and mechanically

ventilated; however, this is also can be dangerous for a patient with asthma. The patient's high risk of air trapping with mechanical ventilation must be aggressively treated and monitored.

2. The key principles of managing this patient's ventilator involve maintaining a low respiratory rate, a low I:E ratio, and potentially a high flow rate. All of these are methods of lengthening the expiratory time. Of all the interventions, keeping a low respiratory rate is the most effective in giving the patient adequate time to exhale. Additionally, patients with asthma should be ventilated with low tidal volume ventilation to minimize the amount of gas that needs to be exhaled. Permissive hypercapnia, or allowing the patient to have a mild to moderate respiratory acidosis, is acceptable in a patient with asthma.
3. (3a) 450 mL
   (3b) 8 cmH_2O
   (3c) 38 cmH_2O
   (3d) 1:2.3
4. This patient likely has a resistance problem. The way to be certain is to check his pulmonary mechanics. By checking a plateau pressure, or checking an inspiratory hold, the clinician can determine the difference between his peak inspiratory pressure and his plateau pressure. If this difference is 5 cm of water or less, the patient has minimal issues with resistance. Conversely, if the patient has a high peak inspiratory pressure but a low plateau pressure, this indicates a significant issue with resistance. The inspiratory hold maneuver is not shown in Fig. 13.4; however, when it was checked, the patient had a plateau pressure of only 24. This indicates that the patient's issue was with resistance, not compliance.
5. Air-trapping can be readily quantified at the bedside by performing an expiratory hold maneuver. This will give the autoPEEP or the intrinsic PEEP (iPEEP). Although the expiratory hold maneuver is also not shown in Fig. 13.4, the autoPEEP in this scenario was 9 cmH_2O. This indicates that the patient has 9 cmH_2O of pressure trapped in his lungs due to inability to fully exhale. Looking closely at the patient's monitor, one can see that the patient is not fully exhaling as the waveform for flow never reaches the baseline before the next breath occurs. Recall that evaluating the waveform is a qualitative measure only and does not quantify the amount of air-trapping.
6. This patient has elevated peak inspiratory pressure. In addition to providing aggressive care with continuous bronchodilators, steroids, magnesium, and any other medically appropriate interventions, the patient's ventilator should be adjusted. His respiratory rate is 24, which is far too high for a patient with asthma. It would be reasonable to drop the respiratory rate to 12 breaths per minute and reassess. Paradoxically, allowing the patient time to exhale can improve the $PaCO_2$ by reducing air trapping and improving ventilation. Additionally, clinician could decrease the tidal volume to minimize the volume of gas the patient must exhale. Lastly, although some PEEP is always appropriate, the patient's PEEP could be decreased to 5 cmH_2O.
7. Although this patient is high risk for breath stacking, which would lead to a high-pressure alarm, the question stem indicates the patient has a low-pressure

alarm. The mnemonic DOPES addresses etiologies leading to both low- and high-pressure alarms. Displacement and equipment failure are the two most likely causes of low-pressure alarms. In this scenario, the patient had coughed vigorously, and he had partially self-extubated leading to the low-pressure alarm sounding.

Case 4

1. Although the patient is likely to travel to the CT scanner soon, a patient with neurologic injury should be placed on a mechanical ventilator as soon as possible to minimize the risk of unintentional secondary injury. Use of a mechanical ventilators, including portable or transport ventilators for travel to radiology or interfacility transport, is important to ensure a consistent ventilation by means of providing consistent tidal volumes and respiratory rate. This monitoring and consistency minimize risk of inadvertent hyper- or hypoventilation.
2. This is a trauma patient with an apparent neurologic injury. It is appropriate to place the patient on low tidal volume ventilation, with a tidal volume of 6–8 mL/kg of predicted body weight, and set the respiratory rate such that the patient has a starting minute ventilation of at least 7–8 L/m. Therefore, it is also important to know how tall the patient is such that his predicted body weight can be calculated. The patient should be given at least 5 of PEEP, and the FiO_2 should be weaned as rapidly as possible, targeting an oxygen saturation of 95–99%. An ABG should be checked within 15–30 minutes after intubation to ensure a $PaCO_2$ of 35–40 and a PaO_2 of 80–100.
3. (3a) The tidal volume is set for 600 mL, and the patient is receiving 613 mLs.
   (3b) 8.58 L/min
   (3c) 19 cmH_2O
4. This ABG indicates that the patient is being hyperventilated. His $PaCO_2$ is in the 20s, below the target of 35–40. Therefore, his respiratory rate or tidal volume should be decreased to decrease his overall minute ventilation. Additionally, the patient has hyperoxia with a PaO_2 of 225. This level is too high and could lead to secondary brain injury. The FiO_2 should be dropped substantially, likely to 60%, monitoring the SPO_2 to ensure that the patient does not become hypoxemic.
5. Even with a subdural hematoma, keeping the patient on a low amount of PEEP is appropriate. Patients with trauma and other neurologic injuries are at high risk for development of ARDS. PEEP is thought to help prevent ARDS to the extent that it prevents "atelectatrauma," or the injury to alveoli that occur with repeated opening and snapping shut. Five cmH_2O of PEEP is an appropriate minimum for all patients.
6. If the patient developed a hemothorax, the patient's compliance should go down. This would be manifested and an increase and in peak inspiratory pressure and the plateau pressure.

Case 5 Answers

1. This patient has a large area of shunt. This can be seen on the patient's chest X-ray with a near opacification of their right no field. This area of law is still receiving blood flow, but it is not participating meaningfully in ventilation. Therefore, the patient has a large VQ mismatch, leading to shunt. This shunt is so large, it is difficult to overcome. This is why the patient is receiving supplemental oxygen without a significant improvement in oxygenation.
2. Anything that can cause a loss of surface area on the alveolar surface (think: alveoli filling with fluid, blood, or purulent material) can cause a shunt. This can include edema or an infection such as pneumonia. Another cause is collapse of functioning alveoli, or atelectasis. This patient has a particular geographic distribution to this opacity. As such, top items on the differential would include a very severe pneumonia, a large pleural effusion causing collapse of the right lung and thereby causing atelectasis, or a collapse of the lung distal to an obstruction in the bronchus, such as a mucous plug, a foreign body, or a mass. At this point, with the given data, it is difficult to tell what in particular is causing this patient severe hypoxemia.
3. (3a) 420 mL
   (3b) 32 breaths per minute. He is not overbreathing, as the total breaths are 31. Note that a slight variation (32–31) is normal depending upon the moment of measurement.
   (3c) 13.1 L/min
   (3d) This is an inspiratory hold. This allows for the determination of the P_{plat}. The P_{plat} is 29 cmH_2O in this example.
4. This patient has a P_{plat} of 29, which is high, but not drastically so. However, the PIP is 39. This ten-point discrepancy indicates a mild to moderate resistance problem. This can help us with the differential diagnosis. A resistance problem, or impedance to airflow, indicates an issue in the airways, the endotracheal tube, or ventilator circuit.
5. It would be reasonable at this point to obtain more information. The resistance problem is present, but not immediately life-threatening. Trying bronchodilators and reassessing the resistance would be reasonable. A bedside ultrasound could evaluate for an effusion, and a CT scan could help delineate the pathology. In this particular case, the patient had a large tumor obstructing his right mainstem bronchus, and this was resulting in a post-obstructive pneumonia. Bronchodilators did not help; he underwent a bronchoscopy to evaluate the lesion. A plug of mucous was exacerbating the obstruction, and removal of that plug allowed better aeration of the right lung.
6. This patient could benefit from repositioning. In this case, one would place the patient with the good lung down. The goal is to maximize ventilation-perfusion matching, to optimize blood flow (assisted by gravity) to the good lung. Given the heterogeneity of some good lung units with other areas of severe shunt, inhaled bronchodilators could also be beneficial to improve V/Q matching and thereby improve oxygenation.

Suggested Reading

Archambault PM, St-Onge M. Invasive and noninvasive ventilation in the Emergency Department. Emerg Med Clin North Am. 2012;30(2):421–49, ix.

Mosier JM, Hypes C, Joshi R, et al. Ventilator strategies and rescue therapies for management of acute respiratory failure in the Emergency Department. Ann Emerg Med. 2015;66:529–41.

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Conclusions and Key Concepts

14

In summary, the management of mechanical ventilation is a vital procedure performed by emergency medicine clinicians to assist with oxygenation and ventilation, decrease the work of breathing, and help the patient meet their metabolic demands while critically ill. It is also important to recognize that mechanical ventilation can lead to several complications, which must be considered and minimized in all intubated patients. While no text can replace care from respiratory therapists and intensivists, having a shared vocabulary and understanding will allow for improved collaboration and care of these patients.

As a reminder, the goals of this text are to:

Familiarize ED Clinicians with Common Terms in Mechanical Ventilation

- Many terms are used interchangeably in mechanical ventilation, leading to confusion. Select appropriate terms, and use them consistently.
- Key concepts include: tidal volume, respiratory rate, minute ventilation, PEEP, resistance, compliance, peak inspiratory pressure, plateau pressure, autoPEEP, and derecruitment. Understanding the meaning of each term and how to apply it to a patient in the ED is crucial to understanding mechanical ventilation.
- Modes of ventilation are assist-control (including volume control and pressure control, as well as pressure-regulated volume control), pressure support, and synchronized intermittent mandatory ventilation.

S. R. Wilcox et al., *Mechanical Ventilation in Emergency Medicine*,
https://doi.org/10.1007/978-3-030-87609-8_14

Review the Fundamental Principles of Pulmonary Physiology Relevant to Mechanical Ventilation

- There are two types of V/Q mismatch: Shunt is perfusion without ventilation, and dead space is ventilation without perfusion. The body tries to optimize V/Q matching by hypoxemic vasoconstriction.
- Resistance involves flow, and compliance is the distensibility of the entire system. The peak inspiratory pressure includes factors of resistance and compliance; however, plateau pressure only involves compliance.

Discuss the Basic Principles of Selecting Ventilator Settings

- Ventilator screens provide much data, but in general on most ventilators, the settings selected by the clinician appear along the bottom, and the patient's response appears along the top.
- Tidal volume should be selected for 6–8 mL/kg of predicted body weight, based upon height and sex. The respiratory rate should be selected to target a reasonable minute ventilation.
- PEEP should be set at minimum of 5cmH2O and titrated higher as needed to correct for hypoxemia and derecruitment, with a goal of optimizing compliance. The ideal PEEP will minimize both atelectasis and overdistention in the majority of the lungs.
- Once the ventilator settings are selected, the patient must be continuously reassessed, settings such be titrated based on ABG results, and peak inspiratory pressures and plateau pressures monitored to reduce harm.

Develop Strategies for Caring for the Ventilated ED Patients with ARDS, Asthma, COPD, and Neurologic Injury

- ARDS: The most important concepts in the management of ARDS patients are low tidal volume ventilation while targeting a plateau pressure <30 cmH_2O. These patients might also require high levels of PEEP for improved oxygenation. Checking an ABG on these patients is critical to determine the PaO_2 to FiO_2 ratios and titrate their FiO_2 requirements. They may develop severe hypoxemia, even after intubation, requiring recruitment maneuvers to assist with oxygenation and neuromuscular blockade for ventilator synchrony. Additional techniques can be considered, including proning, use of inhaled pulmonary vasodilators, and even ECMO.
- Asthma: These patients are at high risk of breath stacking, leading to autoPEEP. They should be ventilated with similarly low tidal volumes, a low respiratory rate, and low I:E ratios. They must be monitored for air trapping, and autoPEEP checked with an expiratory hold.

- COPD: COPD patients often respond well to BPAP. If they require intubation, they should be treated very similarly to asthmatic patients. One difference is that patients with COPD may require higher levels of PEEP to match autoPEEP from collapsible distal airways.
- Neurologic Injury: These patients are at risk for secondary injury during and after intubation from hypoxemia as well as hypocapnia. Therefore, efforts should be made not to hyperventilate these patients, instead targeting normoxia and eucapnia.

Assess and Respond to Emergencies during Mechanical Ventilation

- The differential diagnosis for ventilator alarms is DOPES: displacement, obstruction, pneumothorax, equipment failure, and stacked breathing.
- The mnemonic for action is DOTTS: disconnect, oxygen (bagging), tube position, tweak the vent, and sonography.