Mechanical Ventilation in Emergency Medicine

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Second Edition

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ISBN 978-3-030-87608-1 ISBN 978-3-030-87609-8 (eBook)
https://doi.org/10.1007/978-3-030-87609-8

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

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:

S. R. Wilcox et al., *Mechanical Ventilation in Emergency Medicine*,
https://doi.org/10.1007/978-3-030-87609-8_1

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

2

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.

S. R. Wilcox et al., *Mechanical Ventilation in Emergency Medicine*,
https://doi.org/10.1007/978-3-030-87609-8_2

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.

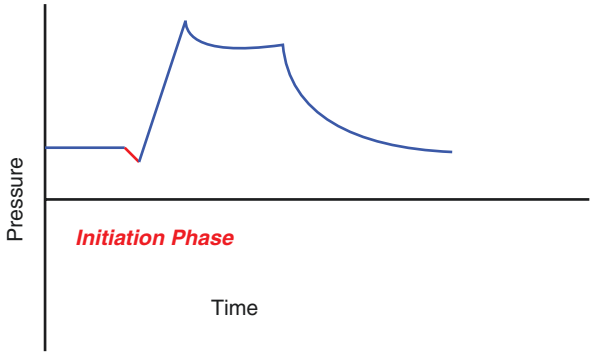

Fig. 2.1 Waveform illustrating initiation phase, or triggering

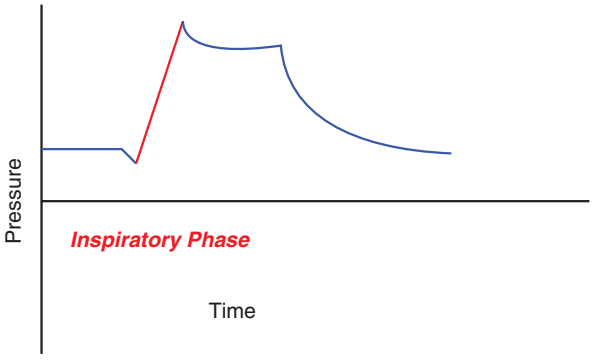

Fig. 2.2 Waveform illustrating inspiratory phase

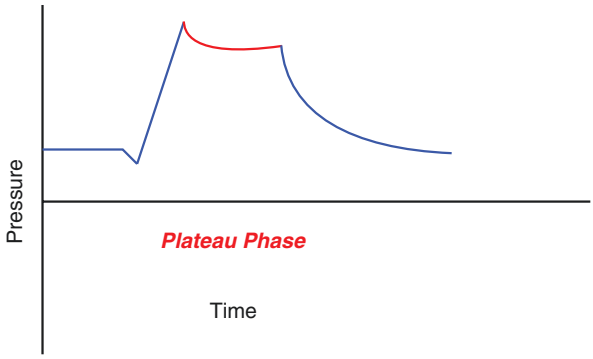

Fig. 2.3 Waveform illustrating plateau phase

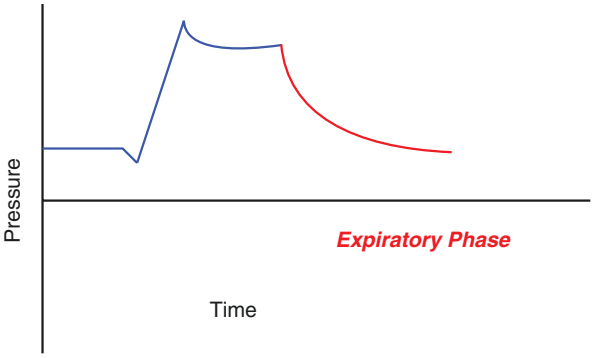

Fig. 2.4 Waveform illustrating expiratory phase

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 $\leq$ 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 $\leq$30 cmH_2O. See Fig. 2.5.

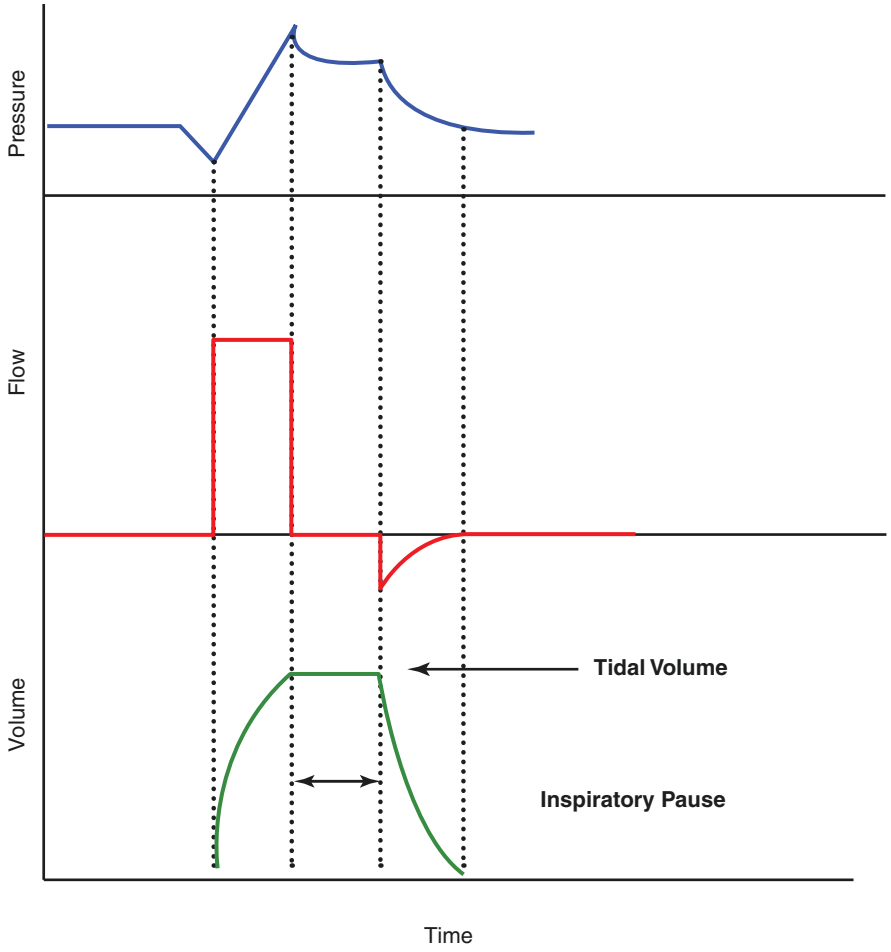

Fig. 2.5 Typical ventilator waveforms illustrating volume, flow, and pressure

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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Review of Physiology and Pathophysiology

3

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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https://doi.org/10.1007/978-3-030-87609-8_3

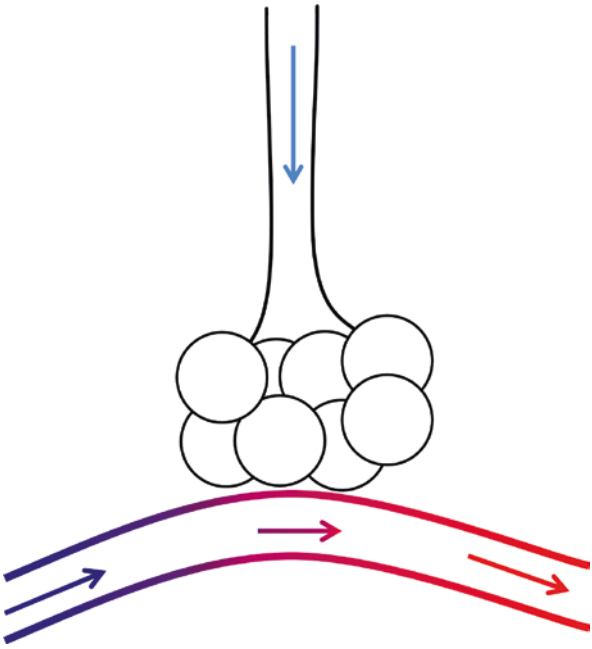

Fig. 3.1 Schematic of normal alveoli and capillary

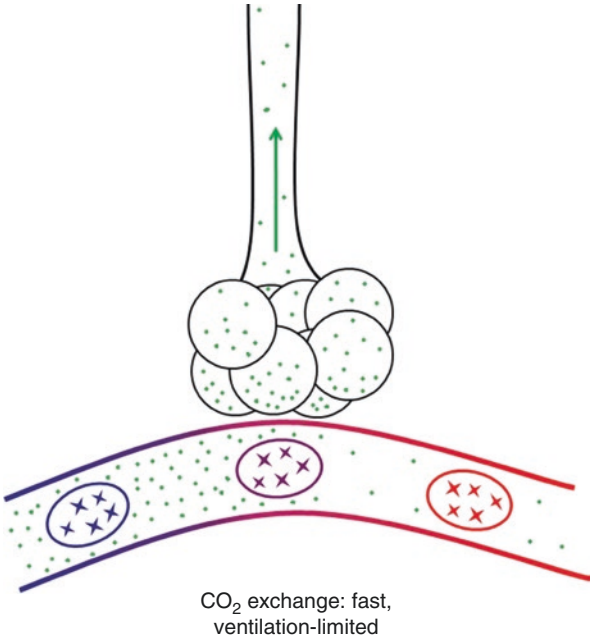

Fig. 3.2 Carbon dioxide uptake by the alveoli. Green dots = blood

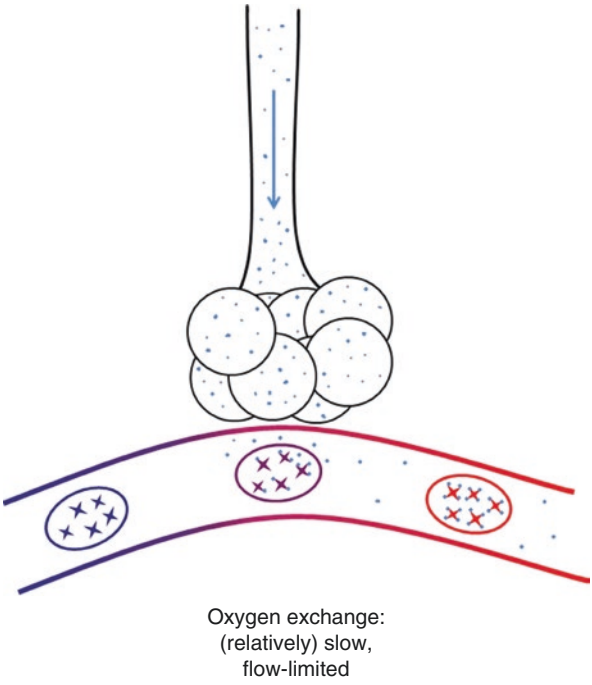

Fig. 3.3 Oxygen uptake by capillary and hemoglobin. Small blue dots = oxygen

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

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.

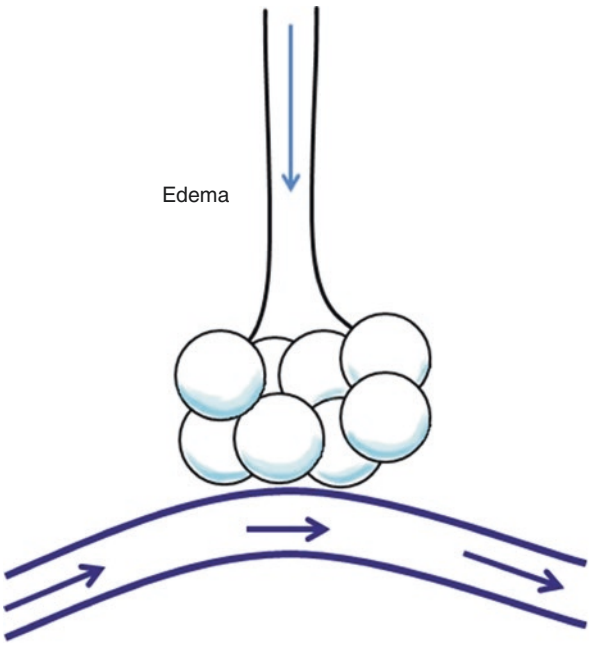

Fig. 3.4 Fluid-filled alveoli inhibiting gas exchange

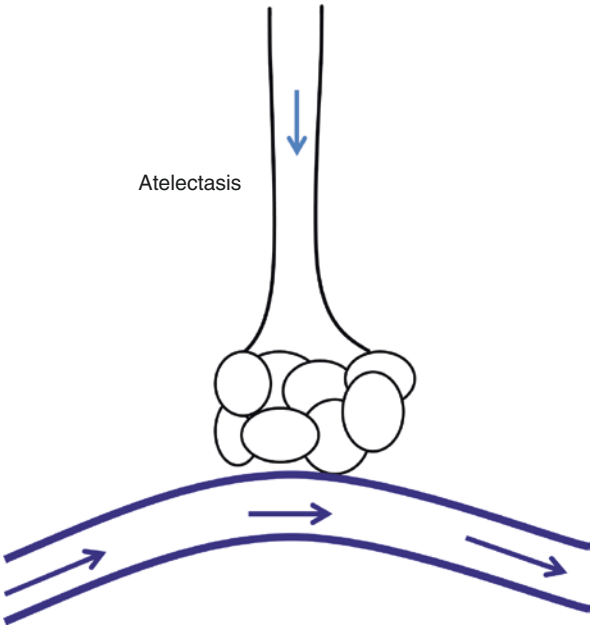

Fig. 3.5 Collapsed alveoli inhibiting gas exchange

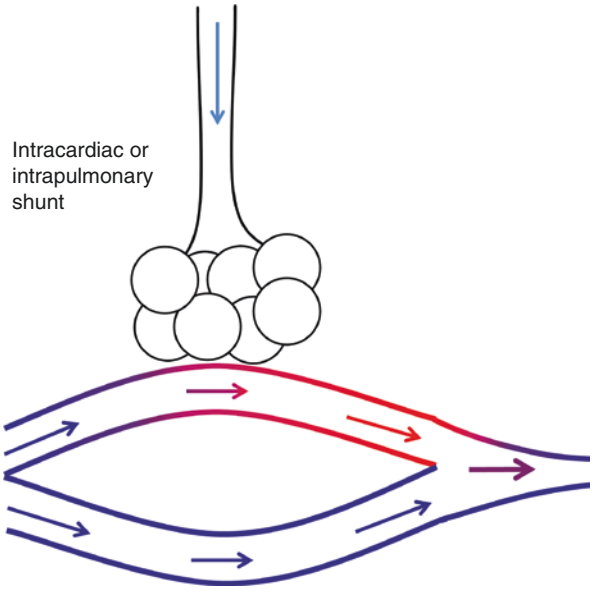

Fig. 3.6 Shunting can occur at the organ-level, with shunts in the heart or lungs. This diagram depicts oxygenated blood that flowed past alveoli, picking up oxygen, mixing with deoxygenated blood that bypassed alveoli

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

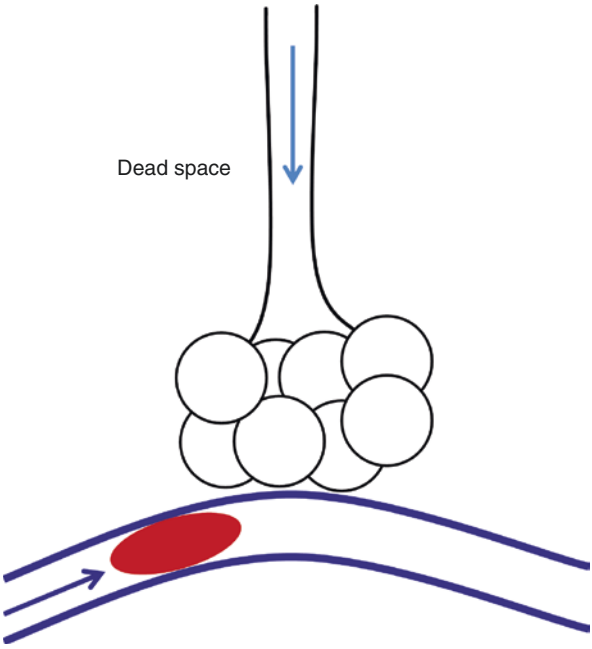

Fig. 3.7 Decreased perfusion inhibiting gas exchange

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.

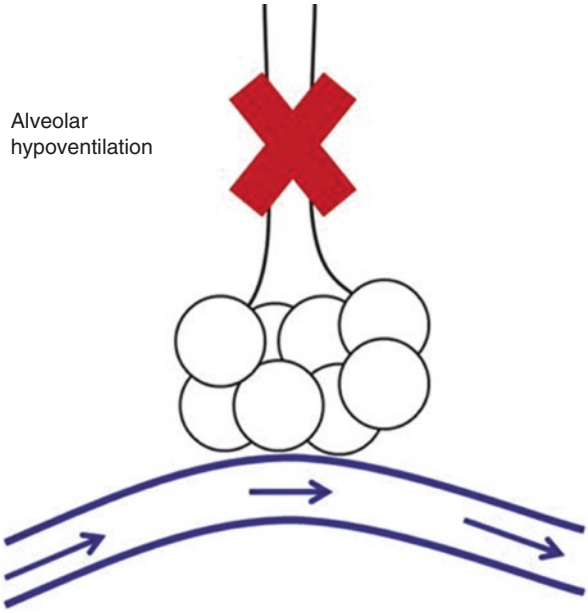

Fig. 3.8 Decreased airflow to alveoli inhibiting gas exchange

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).

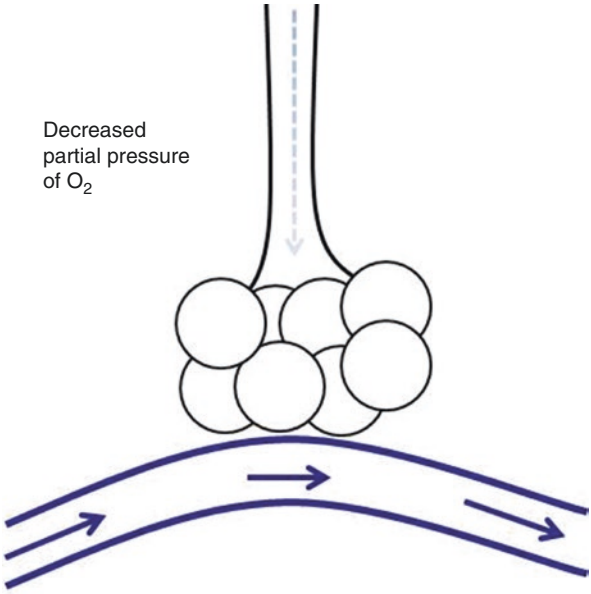

Fig. 3.9 Decreased partial pressure of oxygen inhibiting oxygenation

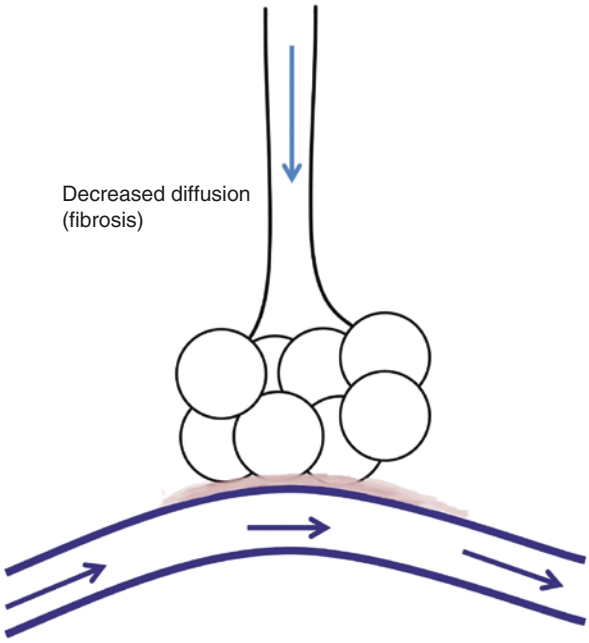

Fig. 3.10 Increased interstitial thickness inhibiting gas exchange

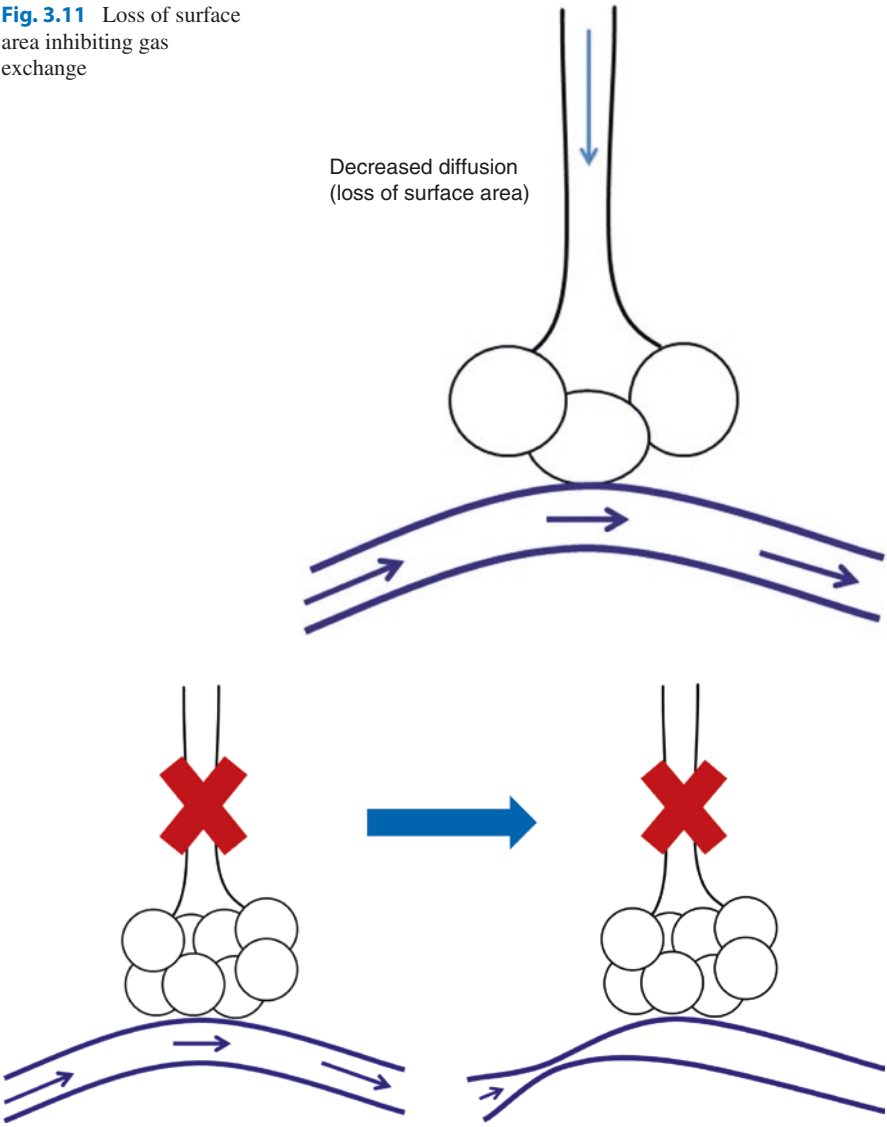

Fig. 3.11 Loss of surface area inhibiting gas exchange

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