Waveform analysis during mechanical ventilation

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Prescribing mechanical ventilation to address acute or chronic respiratory failure is an essential element of critical care medicine. Ideally, the manner in which gas is delivered to a patient's pulmonary system should be adjusted based on how the delivered gas interfaces with the patient's pulmonary compliance and resistance. As none of these elements are readily discernable using conventional physical examination techniques, the clinician is aided by utilizing the panoply of waveforms that are displayed by the ventilator. This section explores the commonly available waveforms on most modern ventilators, their use in adjusting ventilator prescription, as well as their limitations. It is appropriate to review some basic aspects of pulmonary mechanics before exploring waveforms that reflect those dynamics.

Basic pulmonary mechanics

The respiratory system performs 2 main functions: carbon dioxide removal (ventilation) and oxygen uptake (oxygenation). It transports gas into the lung alveoli, where the oxygen and carbon dioxide diffuse in opposite directions across the alveolar-capillary membrane.[1] Although these 2 functions are often considered separately during discussions of mechanical ventilation, they are physiologically interrelated.

The basic gas-exchanging unit of the lung is the terminal respiratory (or lung) unit. It is defined as the portion of the lung that is distal to a terminal nonrespiratory bronchiole.[2,3] It contains further generations of branching respiratory bronchioles that in turn segment into alveolar ducts and finally into the terminal alveoli. It also houses a dedicated vascular bed that facilitates gas exchange. A pulmonary arteriolar branch enters the terminal respiratory unit along with the terminal bronchiole and anastomoses with the pulmonary venules through a dense capillary network along the alveolar wall. Direct communication occurs between the respiratory bronchioles and the surrounding alveoli through the canals of Lambert. Alveoli are also directly connected to each other through the pores of Kohn, thus allowing for collateral ventilation in the presence of small peripheral airway occlusion; this process is known as alveolar interdependence.[3,4] Alveolar opening and closing is facilitated by alveolar geometry and the presence of surfactant.

Alveoli are polyhedrons that have angles at their junctional surfaces. Thus, alveoli are able to entirely collapse by closing those angles. Surfactant, a detergentlike product produced by type II pneumocytes, reduces surface tension and allows coapted alveolar walls to separate more easily under the influence of gas entry as well as through alveolar interdependence. As alveoli accept gas at different rates, and may therefore have different volumes, a more open alveolus can help open a less open one by tension across their common wall as well as gas passage across the pores of Kohn.

The bulk volume of gas that is moved in and out of the respiratory tree with each breath is termed tidal volume (VT) and represents the sum of dead space ventilation and alveolar ventilation. Dead space ventilation, which normally represents approximately 20%-30% of the resting VT, is in turn composed of anatomical dead space (air conducting but non–gas-exchanging airways) and physiological dead space (nonperfused alveoli). The physiological dead space increases with lung injury and pulmonary disease. Minute ventilation (VE) is the volume

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of air transported in and out of the lung per minute and is the product of VT and the respiratory rate (RR).

Gas movement into alveoli is not a uniform process. The lung is composed of heterogeneous "compartments" or "units" that admit gas at different rates depending on their regional time constants (RTC) that are determined by each unit's unique resistance and compliance (see later in the article). In pulmonary disease such as acute lung injury-acute respiratory distress syndrome (ARDS), diseased or collapsed alveoli have longer RTC and thus the more normal alveoli with shorter RTC would preferentially admit gas.[5] This can cause alveolar overdistension, barotrauma, ventilator-induced lung injury, and further deterioration in pulmonary function especially when large volumes are delivered over a short time frame.[6]

Gas movement requires a pressure gradient between the oropharynx and the alveolus. In spontaneous (negative pressure) respiration, the expansion of the chest wall causes a negative transpulmonary pressure gradient that leads to an increase in alveoli volume and thus a decrease in alveolar pressure. Mechanical ventilation, however, establishes a positive pressure gradient between the proximal airway and the alveolus, thus "forcing" gas into the lungs. The relationship between the change in pressure and the corresponding change in volume is a function of the respiratory system compliance:

$$\text{Compliance} \approx \Delta V/\Delta P \ (\text{most commonly expressed as mL/cm } H_2O).$$

Compliance of the whole system is a function of the compliance of the lung as well as the compliance of the chest wall. In disease states, compliance can dramatically decrease because of several factors including but not limited to pulmonary or chest wall edema, and atelectasis. Another variable of interest is pulmonary resistance (R), an element that is inversely related to the square of the total cross-sectional area of gas-conducting airways. Pulmonary resistance determines the flow rate (Q) for a given pressure gradient and can be increased by bronchospasm, atelectasis, secretions, or airway edema such that:

$$\Delta P = \text{RQ}.$$

Measurements of volume and pressure changes at the alveolar level are unobtainable in clinical practice. As a surrogate, modern ventilators measure inhaled and exhaled volumes as well as the generated pressures in the ventilator circuit or proximal airway or both. Peak airway pressure corresponds to the maximal proximal airway pressure generated during inspiration. Mean airway pressure, a variable closely related to oxygenation, can also be computed and in most circumstances closely reflects mean alveolar pressure.[7] Using an end-inspiratory pause to allow pressure equilibration without dynamic gas flow, a plateau pressure can be measured. Given the absence of flow, plateau pressures are more representative of the pulmonary compliance (after adjusting for external or auto-positive end-expiratory pressure [PEEP]) than other airway pressure measurements. The difference between peak and plateau pressures reflects the pulmonary resistance; a normal peak to plateau gradient is < 4 cm H_2O pressure.

Given the complexity of the pulmonary system, it is appropriate to assess how the patient's lung is responding to the ventilator prescription. Waveform assessment is an excellent accompaniment to physical examination, arterial blood gas analysis, as well as carbon dioxide capnometry and capnography in achieving that goal.

Waveforms

Pressure-time trace

This tracing displays the airway pressure as a function of time. As such, it is initially driven by the selected waveform for gas delivery and secondarily by the selected maximal inspiratory flow rate; only the waveform will influence the conformation of the pressure-time trace because the gas flow rate solely affects the amplitude and duration of the trace. In adults, the 2 primary

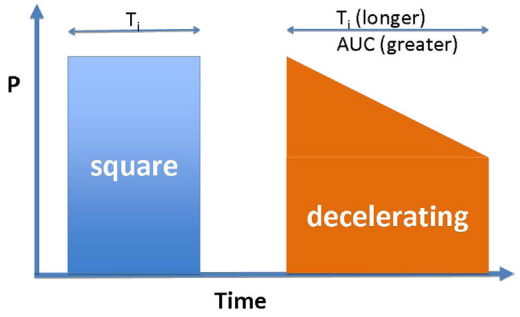

Fig. 1. Square vs decelerating waveforms. (Color version of figure is available online.)

waveforms that are utilized are the square and decelerating patterns (Fig 1). One can readily discern several substantial differences between these 2 tracings. For an equivalent VT in volume-cycled ventilation (VCV), it is clear that the square waveform results in a higher peak airway pressure, a shorter inspiratory time (Ti), and a smaller area under the curve (AUC); the AUC corresponds to the mean airway pressure (Pawmean) and therefore positively correlates with oxygenation. The converse is similarly true: the decelerating waveform describes a lower airway pressure, a longer Ti, and a larger AUC and a higher Pawmean.

Clinical management implications

Therefore, patients with unacceptably high airway pressures may be changed from a square to a decelerating pattern as part of a management strategy. Alternatively, the maximal inspiratory flow rate may be reduced to determine if delivering the gas at a slower rate will result in acceptable dynamics without changing the waveform. One should note that using the ARDSnet ventilation strategy, the ability to achieve normal pCO_2 is dependent on a high RR, a high flow rate, and a square waveform to create a short Ti and allow enough time for ventilation (CO_2 clearance)[8] (REF 8-ARDS). Thus, changing to a decelerating waveform with a lower flow rate may result in inadvertent CO_2 retention and may create undesirable respiratory acidosis. When acceptable, such a strategy may be termed permissive hypercapnia, but may require buffering with an alkalinizing buffer, such as sodium bicarbonate or tromethamine.[9] When adjusting aspects of a ventilator prescription that may affect Ti and therefore expiratory time (Te), one should routinely assess whether there is any limitation of expiratory flow that might create undesirable auto-PEEP. The flow-time trace is the displayed waveform that one should interrogate to do so.

Flow-time trace

In a manner analogous to the pressure-time trace, the flow-time trace is also conformationally driven by the selected waveform and amplitude and duration driven by the maximal inspiratory flow rate. Unlike the pressure-time trace, the flow-time trace has a negative (below baseline) component reflecting exhalation (Fig 2). Similar differences between the waveforms may be noted with regard to airway pressure, Ti, and AUC (Figs 1 and 2). However, the utility of the flow-time trace lies in the clinician's ability to determine whether expiratory flow has terminated before the delivery of the next breath. In other words, the flow tracing should recover to the baseline (zero flow) before the tracing begins to rise above baseline, indicating that gas delivery has once again resumed.

Failure to return to the baseline before the initiation of the next positive pressure breath indicates that there is trapped volume that remains within the bronchial tree that cannot exit, which increases end-expiratory lung volume and creates additional pressure termed auto-PEEP. In general, auto-PEEP is thought to be undesirable and can contribute to retardation of venous return, reduction

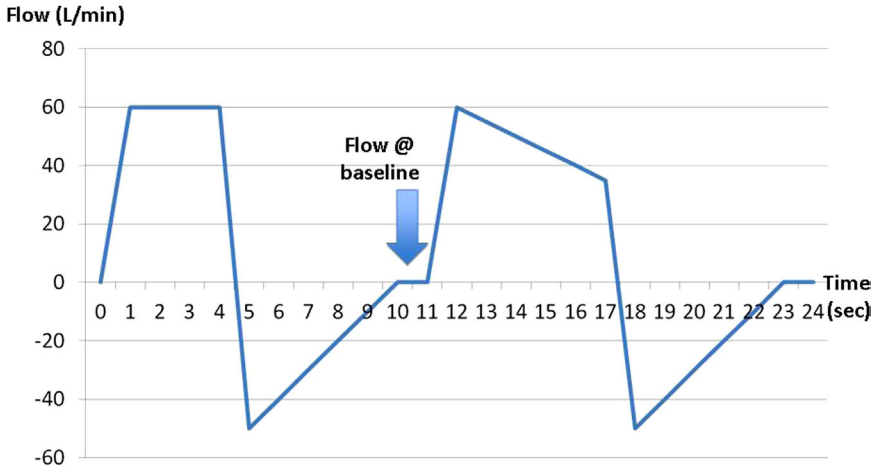

Fig. 2. Square to decelerating waveforms. (Color version of figure is available online.)

of cardiac output, and arterial hypotension, compromising macrocirculatory and microcirculatory flow.[1] Therefore, the clinician should strive to recognize and address auto-PEEP.

Clinical management implications

Recognition of auto-PEEP should prompt ventilator prescription adjustment. Multiple adjustments may be undertaken and depend on what is believed to be the underlying etiology of the auto-PEEP. A major issue is a spontaneous RR in excess of the set rate that of necessity will shorten Te. Sedation or analgesia or both as commonly utilized in critically ill postoperative or injured patients, may solve this problem without requiring any adjustment of the ventilator prescription. One should recall that the set rate may also be inappropriately high and a reduced RR may be required along with an increase in VT to maintain VE.

If this is not the case, then in VCV, reducing the set VT, changing from a decelerating to a square waveform (rare as most patients have some degree of acute lung injury and benefit from the longer Ti to match RTC variations), or increasing flow rate (also rare for the aforementioned reasons) may eliminate auto-PEEP. In fixed inspiratory time PCV, reduced PC or Ti may be required because the combination of the 2 describes the total volume of delivered gas, and of course, the Ti is set by the clinician. In I:E ratio PCV, the PC may need to be reduced for similar reasons, or the I:E ratio may require manipulation to increase the Te (ratio moves further away from 1:1).

Special note is made of advanced modes of ventilation including airway pressure release ventilation (APRV) and high-frequency oscillatory ventilation (HFOV). APRV does not have an external PEEP setting and achieves PEEP by the *deliberate* creation of auto-PEEP termed intrinsic PEEP to differentiate it from the undesirable *additional* auto-PEEP described previously. When using APRV, intrinsic PEEP is the *only* PEEP. HFOV similarly creates auto-PEEP by virtue of its nonconventional method of rather small volume and high-frequency gas-volume delivery. As such, auto-PEEP is unavoidable and only marginally manipulatable as the major indication for HFOV is hypoxemic respiratory failure unresponsive to conventional ventilation; increased pCO_2 is expected as is auto-PEEP but is generally tolerated in favor of achieving oxygenation.[10] With both of these modes, standard waveforms analysis generally does not apply, and these modes are discussed separately.

Pressure-volume (P-V) loop (dynamic)

The dynamic *P-V* curve or loop is readily displayed by most modern ventilators and is a useful gauge as to how the delivered gas is being received by the patient's lung. As it is dynamic and

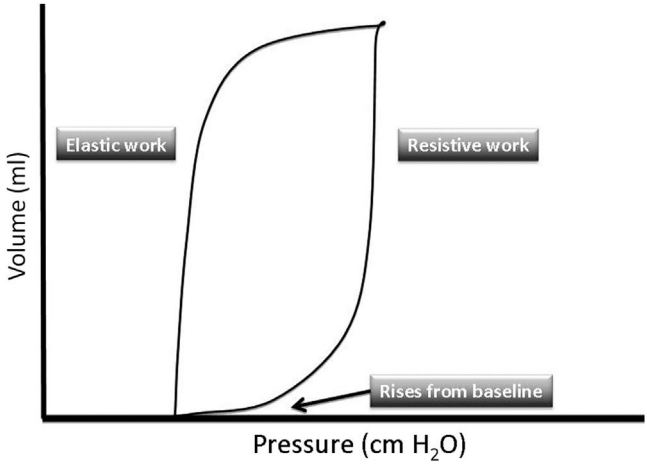

Fig. 3. Dynamic *P-V* loop.

reflects multiple changing parameters in real time, there are several factors that affect its shape. A normal P-V loop is demonstrated in Figure 3. Note that the trace is initiated at the externally set PEEP and should rise smoothly with each incremental increase in the delivered gas volume (and resultant pressure). At the end of inspiration, the curve should reach a nearly vertical peak and then return to baseline describing a nearly mirror course to inspiration. The right side of the trace reflects resistive work whereas the left side reflects elastic work. Inadequate PEEP is demonstrated by the tracing adhering to the baseline before rising, whereas alveolar overdistension is characterized by a flattening of the curve such that increases in pressure no longer translate into increases in volume creating the "bird's beak" trace (Fig 4).

Clinical management implications

Ventilator parameters may be titrated to the shape of the *P-V* curve in concert with measured pressures and values from the arterial or venous blood gas. PEEP may be increased until the trace takes off from baseline without a flat segment, or alternatively decreased while assuring that the trace does not become flat. Alveolar overdistension therapy depends on the mode of ventilation. In VCV, reduced VT is the most common intervention, but one must ensure that the patient is

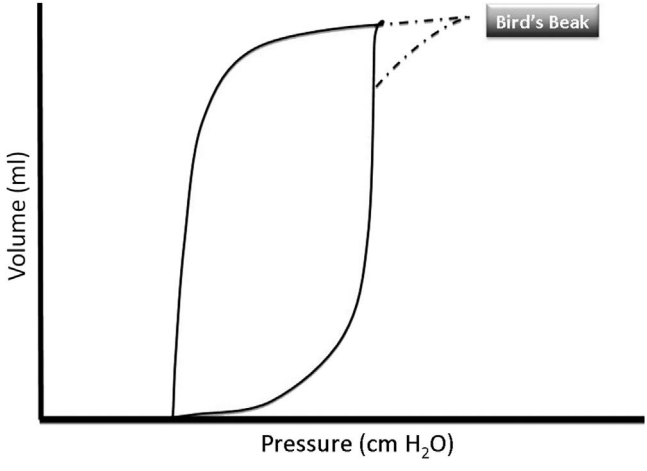

Fig. 4. Overdistension.

not suffering from auto-PEEP as well. Decreased inspiratory flow rate can also address overdistension as the longer Ti may allow additional alveolar units to participate in ventilation. This maneuver effectively increases the available cross-sectional area of ventilatable lung, and reduces the reflected pressure and the proportion of gas that disproportionately distributes into compliant segments. Relatedly, increases in PEEP that increase functional residual capacity may have a similar effect and for similar reasons.

In pressure control ventilation, reduced inspiratory pressure may address overdistension, as can reductions in Ti, if the Ti is already long (different than the shorter Ti achieved during VCV). When in fixed Ti PCV (as opposed to I:E driven PCV), a reduced RR will increase the available Te and reduce or eliminate auto-PEEP and correct overdistension. PEEP adjustments are similar to those in VCV detailed earlier.

In patients with inadequate ventilation (CO_2 clearance) regardless of the mode, the *P-V* curve may serve as a useful guide when increasing VT to increase VE and help ensure that one is not inducing overdistension that was not previously present. Thus, the loops and traces explored in this section are meant to be used in a complementary fashion. Overreliance on a single trace or curve will deprive the clinician of real-time information that provides important insights into how a patient's lung is responding to the ventilator prescription.

P-V loop (static)

The static *P-V* loop is usually obtained only in a research setting because obtaining it requires special circumstances to be satisfied including but not limited to: neuromuscular blockade, zero end-expiratory pressure, square waveform, 100% O_2, and a 3000-mL super syringe.[11] Sequential pressures as a result of sequential deliveries of 100 mL of volume are recorded and plotted with a 2-3-second pause in between serial inflations. These recordings describe a curvilinear plot that identifies a lower curve of reduced compliance, a middle portion of improved compliance that is more linear, and an upper curve of again reduced compliance (Fig 5). Multiple methods of determining both an inflation and deflation curve have been described. Using a variety of mathematical manipulations, an upper and lower inflection point along each curve may be identified.[11] The lower inflection point is believed to represent a pressure 1-2 cm H_2O pressure above which PEEP should be set to maintain open alveolar units. The upper inflection point is believed to represent the maximum tolerable volume and pressure that provides appropriate alveolar gas entry but avoid alveolar overdistension. Although these may be readily measured in a research setting, it is difficult to determine in practice as most patients only demonstrate a dynamic pressure-volume curve (see earlier in the article).

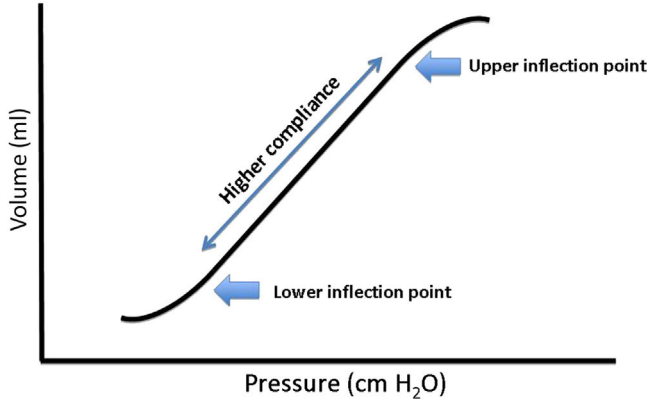

Fig. 5. Static *P-V* loop. (Color version of figure is available online.)

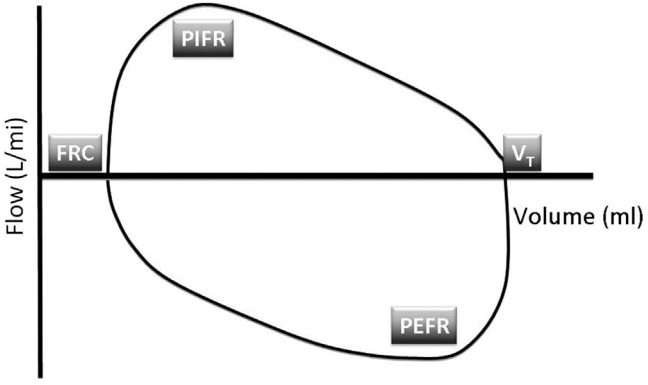

Fig. 6. Flow-volume loop.

Flow-volume loop (dynamic)

This loop is used to help determine the effect of elements that change airway resistance, such as secretions or reactive airway disease (Fig 6). In the spontaneously breathing patient the loop starts at the far left, and the upward deflection represents exhalation that peaks on the left side of the graph and then trends down toward the far right. This is the classic curve that is used to determine forced expiratory volume in 1 second in spontaneously breathing individuals.[12] In the normal setting, approximately 80% of each breath is exhaled within 1 second, with the peak expiratory flow generally achieved within 150 ms. Once the exhalation flow ceases (normal) and the trace returns to the *x*-axis, inspiration is reflected by the deflection below the *x*-axis.

In mechanically ventilated patients, the flow-volume loop is differently arranged. The inspiratory limb starts at the far left, with the starting point representing the functional residual capacity. During inspiration as flow rises, the curve assumes a fairly convex shape and terminates on the right at a point that represents the delivered VT. A mirrored course appears below the *x*-axis during exhalation.

Clinical management implications

Increased airway resistance brings the curve closer to the *x*-axis, allowing one to readily assess the response to bronchodilators (ie, albuterol or magnesium) by watching the downward displacement of the curve with successful therapy (Fig 7). In cases of auto-PEEP, the exhalation trace will not reach the *x*-axis before inspiratory flow begins and will be readily detectable on the graphic. In patients with significant secretions, a saw-tooth pattern to the trace will be

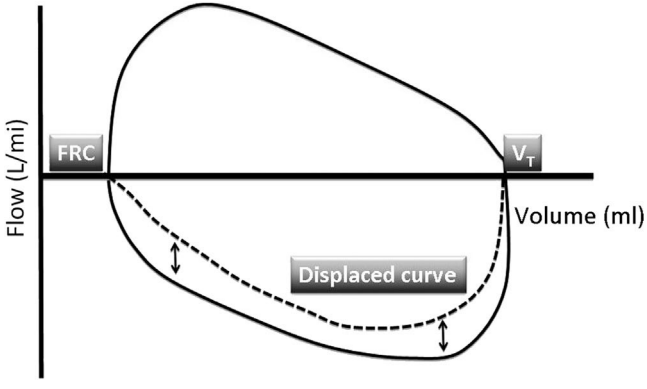

Fig. 7. Airway resistance.

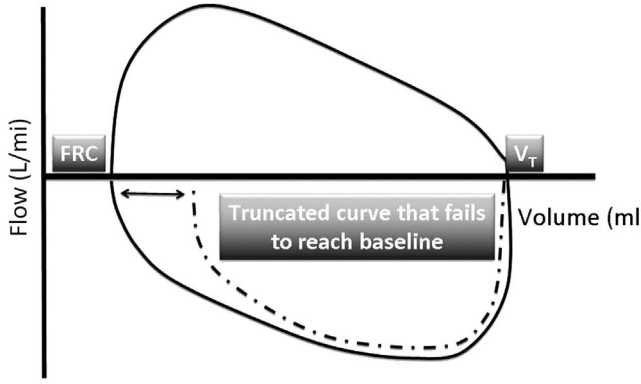

Fig. 8. Air leak detection.

evident. Effective therapy such as suctioning will smooth the trace. Additionally, the flow-volume loop is particularly useful in identifying air leaks as the trace fails to return to baseline during exhalation and instead follows a truncated course (Fig 8). Of course, as with all loops used for analysis, obtaining a baseline and "freezing" it on the screen is particularly useful and serves as a guide to determine the effect of ventilator changes or applied therapeutics.

Limitations

In some ways, limitations of using ventilator derived graphic analysis are akin to those cited for the pulmonary artery catheter (PAC) related to the lack of clinician knowledge regarding what the data represent and how to effectively respond to the data. One must either be present at the bedside to view the waveforms or have the ability to view the waveforms remotely to respond effectively to changes. Remote management of ventilator prescription without being able to view waveforms may deprive the clinician of useful information that would alter the prescribed therapy. Of course, regulatory requirements for reporting do not extend to graphical analysis, leading to the common practice of having the ventilator display present numbers instead of waveforms to facilitate charting, especially for patients who are in isolation for multidrug-resistant organism–related infectious processes. Waveforms are only useful when they are embraced as an integral part of practice.

Conclusions

Understanding ventilator-derived waveforms requires a basic knowledge of pulmonary physiology. However, so armed, the clinician should be able to readily interpret the commonly presented traces and waveforms that reflect how a patient's pulmonary system is responding to the ventilator prescription. Straightforward changes to the prescription or application of common therapeutics are easily implementable and trackable using graphic analysis by clinicians who care for ventilated patients.

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Use and titration of positive end-expiratory pressure

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Introduction

As previously noted, acute respiratory distress syndrome (ARDS) is a pathologic reaction to various insults to the lung or systemic circulation. An essential part of the treatment of ARDS is the appropriate use of mechanical ventilation. Loss of aeration is progressive and may be incompatible with life. Hence, mechanical ventilation is initiated to aid respiratory muscles with the work of breathing and prevent atelectasis.

A panel of experts convened in 2011[1] (European Society of Intensive Care Medicine endorsed by the American Thoracic Society and the Society of Critical Care Medicine) and developed the Berlin Definition. The definition proposed 3 mutually exclusive categories of ARDS based on the degree of hypoxemia: mild (PaO_2/inspired oxygen fraction [F_iO_2] $\leq$300 mm Hg), moderate ($PaO_2/F_iO_2 \leq 200$ mm Hg), and severe ($PaO_2/F_iO_2 \leq 100$ mm Hg), and 4 ancillary variables for severe ARDS: radiographic severity, respiratory system compliance ($\leq$40 mL/cm H_2O), positive end-expiratory pressure (PEEP, $\geq$10 cm H_2O), and corrected expired volume per minute ($\geq$10 L/min).

Alveolar collapse is prevented by positive end-expiratory pressure, an inherent feature of modern ventilators. A higher mean pressure decreases the rate of collapse of diseased units, but it may result in barotrauma in others.

In ARDS, 3 populations of alveoli exist:

(1) *Normal alveoli* are always inflated and engaging in gas exchange,
(2) *Alveoli flooded by fluid and sequestered cellular material* cannot be used for gas exchange, and
(3) *Atelectatic alveoli* can be recruited for gas exchange.

The atelectatic alveoli represent a potentially recruitable population, some of which can be brought into service using minimal positive end-expiratory pressure (PEEP), whereas others can

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