Measurement of Air Trapping, Intrinsic Positive End-Expiratory Pressure, and Dynamic Hyperinflation in Mechanically Ventilated Patients

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Introduction

Severe airflow obstruction is a common cause of acute respiratory failure. Dynamic hyperinflation affects tidal ventilation, increases airways resistance, and causes intrinsic positive end-expiratory pressure (auto-PEEP). Most patients with asthma and chronic obstructive pulmonary disease have dynamic hyperinflation and auto-PEEP during mechanical ventilation, which can cause hemodynamic compromise and barotrauma. Auto-PEEP can be identified in passively breathing patients by observation of real-time ventilator flow and pressure graphics. In spontaneously breathing patients, auto-PEEP is measured by simultaneous recordings of esophageal and flow waveforms. The ventilatory pattern should be directed toward minimizing dynamic hyperinflation and auto-PEEP by using small tidal volume and preserving expiratory time. With a spontaneously breathing patient, to reduce the work of breathing and improve patient-ventilator interaction, it is crucial to set an adequate inspiratory flow, inspiratory time, trigger sensitivity, and ventilator-applied PEEP. Ventilator graphics are invaluable for monitoring and treatment decisions at the bedside. Key words: dynamic hyperinflation, intrinsic positive airway pressure, mechanical ventilation, positive end-expiratory pressure, work of breathing, hyperinflation, waveforms. [Respir Care 2005;50(1):110–123. © 2005 Daedalus Enterprises]
that imposes a substantial mechanical load on the respiratory system. Airflow obstruction develops when airway diameter is narrowed by bronchospasm, mucosal or interstitial edema, exudates of inflammatory cells, mucus, and (among patients with COPD) dynamic airway collapse during expiration.1,2

During severe airflow obstruction episodes, increased expiratory efforts simply raise alveolar pressure without improving expiratory airflow. In exacerbations of COPD and asthma, alveolar ventilation is preserved at the expense of increased work of breathing (WOB). If tidal volume (VT) is high or if expiratory time is short because of a high respiratory rate, the lung cannot deflate to its usual resting equilibrium volume between breaths. Several pathophysiologic consequences result from the increase in lung volume and alveolar pressure: (1) end-expiratory lung volume exceeds predicted functional residual capacity (FRC), causing dynamic hyperinflation, (2) respiratory system compliance decreases, (3) the respiratory muscles progressively operate in an unfavorable part of their length-tension curve. Finally, breathing takes place in the upper and less compliant part of the lung pressure-volume relationship, near total lung capacity. In that setting, if exhaustion has not yet occurred, spontaneous breathing cannot be sustained for long, and the patient is at high risk of sudden respiratory arrest.1,3–6

**Description of Dynamic Hyperinflation and Intrinsic Positive End-Expiratory Pressure**

In normal subjects, lung volume at end-expiration approximates the relaxation volume of the respiratory system. However, in patients with airflow obstruction, the end-expiratory lung volume may exceed predicted FRC. Indeed, lung emptying is slowed and expiration is interrupted by the next inspiratory effort, before the patient has reached the static equilibrium volume.3,4 This is termed *dynamic hyperinflation* and is affected by VT, expiratory time, resistance, and compliance (Fig. 1).4,7–9 This phenomenon, also called intrinsic positive end-expiratory pressure (auto-PEEP), was first described by Bergman10 in 1972 and Jonson et al11 in 1975. Its clinical implications and measurement technique during mechanical ventilation were further described by Pepe and Marini9 in 1982.

**External Factors**

Most patients with asthma and COPD have dynamic hyperinflation and auto-PEEP during mechanical ventilation. However, dynamic hyperinflation and auto-PEEP can occur in the absence of expiratory flow limitation.12 Under conditions of high minute volume (V̇E) and/or increased equipment expiratory resistance (eg, a mucus-narrowed endotracheal tube or heat-and-moisture exchanger),13,14 the lungs do not have enough time to reach normal FRC, so expiratory flow driven by the difference in pressure between alveoli and the airway opening is still present at end-expiration, despite the fact that airways are open.15

**Intrinsic Factors: Dynamic Airway Collapse**

Elastic and resistive loads affect WOB. Elastic WOB increases when lung or chest wall compliance is reduced or when there is dynamic hyperinflation with auto-PEEP. COPD patients ventilated for exacerbations of airflow obstruction experience dynamic airway collapse and flow-limitation during tidal breathing.12,16 Anatomical abnormalities in COPD include regional differences in airway caliber, loss of lung elasticity, and alterations in alveolar
Compression and critical closure takes place in the small airways, and air trapping occurs distal to the site of critical closure.\(^4,9,12,17\) In that setting, increasing expiratory effort raises pleural and alveolar pressure to the same extent, without improving expiratory airflow. Although flow limitation is usually an active process, it can occur during passive deflation (ie, during tidal ventilation) if alveolar pressure exceeds airway pressure in deformable small airways.\(^12\) In patients receiving mechanical ventilation there is a significant correlation between expired-carbon-dioxide slope, respiratory-system resistance, and auto-PEEP, which suggests that dynamic hyperinflation originates from sequential emptying of slow hypercapnic units.\(^18\) Carbon dioxide elimination is impaired by flow-resistance, and the degree of airway obstruction modulates the rate of P\(_{CO_2}\) increase during expiration (Fig. 2).

**Lung Mechanics: Difference Between Asthma and COPD**

Acute changes in lung mechanics from severe bronchospasm due to asthma attacks are similar to those in COPD exacerbations. However, the pathophysiology of asthma differs substantially from that of COPD. A main feature of advanced COPD is increased airway collapsibility due to destruction of the lung parenchyma and loss of lung elastic recoil, whereas the main features of asthma are increased thickness of airway walls (due to inflammation) and decreased collapsibility, despite considerable reduction in airway caliber.\(^19–22\)

**Hemodynamic Effects of Auto-PEEP**

The hemodynamic consequences of auto-PEEP in an airflow-obstructed patient may be equal to or worse than the effects of a similar degree of PEEP applied to a patient with normal lungs. In the presence of highly compliant lungs, a high fraction of increased alveolar pressure is transmitted to the intrathoracic vessels. In their original description, Pepe and Marini\(^9\) found that esophageal pressure decreased by at least 50% of the measured auto-PEEP when mechanical ventilation was discontinued. Increased mean intrathoracic pressure decreases venous return and reduces the preload of both ventricles. It also decreases left-ventricle compliance and may increase right-ventricle afterload because of high pulmonary vascular resistance. In clinical practice, pulmonary capillary wedge pressure has generally been considered a valid index of left-ventricle filling pressure, but in COPD the auto-PEEP-induced increase in intrathoracic pressure may falsely increase pulmonary capillary wedge pressure and right-atrial pressure, despite normal transmural pressure (pulmonary capillary wedge pressure minus esophageal pressure) preload, which can lead to mistakes in hemodynamic management. Rogers et al\(^23\) reported on a patient who had severe COPD and developed refractory circulatory arrest, with sinus rhythm,

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*Fig. 2. Flow, carbon dioxide, tracheal pressure, and airway pressure in a patient with severe airflow obstruction. After a long expiratory time, intrinsic positive end-expiratory pressure (auto-PEEP) is still present (end-expiratory occlusion maneuver), indicating severe dynamic hyperinflation.*
after intubation and positive-pressure ventilation. That case illustrated why the sudden increase of dynamic hyperinflation (due to a short expiratory time set on the ventilator) was responsible for the observed electromechanical dissociation (Fig. 3). Therefore, with severe airflow obstruction, a brief discontinuation of mechanical ventilation may be required to measure true pulmonary capillary wedge pressure or to differentiate severe auto-PEEP from other causes of severe hypotension.

Identification and Measurement of Auto-PEEP in Passive Patients

Simple observation of real-time graphics of airflow and airway pressure at the point of end-expiration is the key to identifying dynamic pulmonary hyperinflation in relaxed or well-adapted patients receiving mechanical ventilation. Whenever the end-expiratory flow is far from zero, the respiratory system is dynamically hyperinflated (Fig. 4). Single-breath flow-volume loops provide similar information. The presence of airflow at end-expiration indicates that the alveolar pressure is higher than the atmospheric pressure or higher than the applied PEEP. Auto-PEEP can be measured by performing an end-expiratory occlusion or by simultaneous observation or recording of the airway pressure and airflow (see Fig. 4). Auto-PEEP measured with end-expiratory occlusion is called static auto-PEEP and is higher than auto-PEEP measured by simultaneous recording of airflow and airway pressure at end-expiration, which is called dynamic auto-PEEP. The difference is because dynamic auto-PEEP reflects the end-expiratory pressure of the lung units with short time constants and rapid expiration, while units with long time constants are still emptying. The end-expiratory occlusion maneuver provides time to equilibrate lung units.

Fig. 3. Airway pressure (Pao), esophageal pressure (Pes), and lung volume waveforms in an experimental animal receiving manual (bag) ventilation during cardiopulmonary resuscitation. During rapid manual inflations, the expiratory time is shorter and lung volume and intrathoracic pressure is higher. ΔV = change in volume.

Fig. 4. Flow and airway pressure (Paw) waveforms from a patient with chronic obstructive pulmonary disease, receiving mechanical ventilation. Rapid end-inspiratory and end-expiratory occlusions (arrows) allow assessment of alveolar pressures in static conditions. auto-PEEP = intrinsic positive end-expiratory pressure.
that have different regional auto-PEEP, and the value obtained after 2–3 seconds of end-expiratory occlusion is the mean value after equilibration, provided the airway is open (Figs. 5 and 6). End-expiratory occlusion can be performed manually, using the software incorporated in the ventilator, which can perform rapid occlusion exactly at the end of expiration.

Some ventilators can measure the dynamic-hyperinflation-induced trapped gas. In patients receiving ventilator-applied PEEP, the difference between the pressure obtained after an end-expiratory occlusion and the applied PEEP is the auto-PEEP. The measurement of static compliance of the respiratory system needs to be corrected for the presence of auto-PEEP, or the true value of static compliance will be underestimated (Fig. 7).

The mechanics of the respiratory system can also be measured without the need for special maneuvers or particular flow patterns, by using the least squares fitting technique, with near-relaxed patients.

The increase in lung volume due to applied PEEP or dynamic hyperinflation can be measured by passive exhalation, disconnecting the patient from the ventilator, or prolonging the expiratory time to FRC. At FRC, connecting the patient to the ventilator causes the opposite phenomenon: the inspired VT will be greater than the exhaled VT until lung volume stabilizes. In the absence of applied PEEP, trapped gas above FRC corresponds to the increase in end-expiratory lung volume due to dynamic hyperinflation. In the presence of applied PEEP, lung volume at end-expiration above FRC corresponds to the sum of end-expiratory lung volume due to dynamic hyperinflation plus the increase in lung volume induced by the applied PEEP (Fig. 8).

**Identification and Measurement of Auto-PEEP in Spontaneously Breathing Patients**

Auto-PEEP can be present in a spontaneously breathing patient (ie, who is triggering the ventilator). In a spontaneously breathing patient, auto-PEEP results in less positive or more negative mean intrathoracic pressure than does fully controlled mechanical ventilation. The main consequences are patient-ventilator asynchrony and increased WOB. In spontaneously breathing patients, auto-PEEP is determined by simultaneously recording esophageal pressure and airflow tracings. Dynamic auto-PEEP is measured at end-expiration as the negative deflection of esophageal pressure to the point of zero flow (Fig. 9).

Expiratory muscle contraction transmits an increase in pressure to the intrathoracic space, which further raises auto-PEEP. In these circumstances, the decrease in pleural (esophageal) pressure in early inspiration could be in part attributed to expiratory-muscle relaxation rather than to inspiratory-muscle contraction. Consequently, the part due to expiratory-muscle contraction (determined from an abdominal pressure signal) needs to be subtracted from the drop in esophageal pressure (Fig. 10).

The expiratory-muscle component of auto-PEEP becomes negligible at high auto-PEEP in patients with COPD. In spontaneously breathing patients receiving mechanical ventilation, the patient makes an active inspiratory effort against a positive alveolar pressure (auto-PEEP) at the same time that the expiratory muscles relax. Ventilator triggering takes place when auto-PEEP has been counterbalanced.

**Ventilatory Strategy**

Once intubation has been performed, attention should be directed to avoiding high auto-PEEP. Sedation may lower mean arterial pressure, but positive-pressure ventilation in airflow-obstructed patients can also compromise venous return, and intubation may be followed by cardiovascular collapse. Accordingly, the initial ventilator settings should be directed toward avoiding high mean intrathoracic pressure and auto-PEEP. The patient should be disconnected from the ventilator to check for a rise in blood pressure in cases of ventilator-related hypotension.

Defining the appropriate ventilatory strategy for patients with airflow obstruction requires understanding the relationship between hyperinflation and the rate of lung emptying. Hubmayr et al showed how insufficient expiratory flow produces dynamic hyperinflation during total ventilatory support. Assuming a 1-L VT initiated from the static equilibrium volume at a respiratory rate of 20 breaths/min
and a duty cycle of 0.33 gives the patient 2 seconds to exhale. If airflow is obstructed and mean maximum expiratory flow is only 0.25 L/s, the patient can exhale only 0.5 L (half of the previous VT), so the next breath will take place at a higher lung volume. According to the flow-volume relationship of that patient, a higher mean expiratory flow can be achieved at the new lung-volume, but it will be insufficient to empty the lungs adequately. The steady-state condition, in which the time available for expiration (2 s) is adequate to exhale 1 L would be reached when the increase in lung volume resulted in a maximum mean expiratory flow of 0.5 L/s (Fig. 11). The problem would be avoided by selecting a VT of 0.5 L. The ventilatory pattern should minimize both dynamic hyperinflation and mean intrathoracic pressure, which increase the risk of barotrauma and cardiovascular compromise.

Flow, Tidal Volume, and Respiratory Rate in Asthma and COPD

Optimal ventilatory patterns can be achieved with different combinations of VT, respiratory rate, and inspiratory flow. Tuxen and Lane studied the effects of the ventilatory pattern on the degree of hyperinflation, airway pressure, and hemodynamics in patients with severe airflow obstruction. They found that dynamic hyperinflation could increase end-inspiratory lung volume by as much as 3.6 ± 0.4 L above the apneic FRC when VT was increased and/or when expiratory time was decreased, either by an increase...
in respiratory rate (and hence V˙E) or by a decrease in inspiratory flow (at a constant V˙E). Pulmonary hyperinflation was associated with increased alveolar, central venous, and esophageal pressure, as well as with systemic hypotension. Tuxen and Lane found that, at constant V˙E, mechanically ventilated flow-obstructed patients exhibited the lowest degree of dynamic hyperinflation when ventilated at high inspiratory flow and long expiratory time. Small V{T} and higher respiratory rate seemed preferable to higher V{T} at a lower respiratory rate. Above all, V˙E was the main determinant of hyperinflation. The main goal of the ventilatory pattern was to ensure low V˙E and an expiratory time long enough to allow lung-emptying, together with high peak flow, at the expense of increased peak airway pressure. Although high inspiratory flow exposes robust proximal bronchi to greater pressure, the increase in peak flow reduces alveolar hyperinflation, so there is less hypotension. Finally, measured auto-PEEP may underestimate end-expiratory alveolar pressure in severe asthma, and marked pulmonary hyperinflation may be present despite low measured auto-PEEP, especially at low respiratory rates. This phenomenon may be due to widespread airway closure that prevents accurate assessment of alveolar pressure at end-expiration.

Current recommendations indicate that the ventilatory strategy for patients with acute asthma should favor relatively small V{T} and higher inspiratory flow, to preserve expiratory time and minimize hyperinflation, barotrauma, and hypotension. That objective can be achieved with an inspiratory flow of 80–100 L/min, V{T} of 6–10 mL/kg, peak airway pressure approaching 40–45 cm H₂O, and alveolar plateau pressure not higher than 25–30 cm H₂O. The respiratory rate should be 8–12 breaths/min, to achieve the least possible hyperinflation (auto-PEEP < 10 cm H₂O), and to maintain pH in an acceptable range, if possible. For COPD exacerbations the indications for invasive mechanical ventilation and the ventilatory strategies are similar to those for asthma, but patients with COPD often have less structural airflow obstruction than patients with asthma, at the point when they require ventilatory support. In most cases, patients can be rested adequately with V{T} of 9–10 mL/kg and a respiratory rate of 14–16 breaths/min in assisted/control mode. In both COPD and asthma, ventilator-trigger sensitivity should be minimal.

**Particular Features of Acute Respiratory Distress Syndrome**

In patients who have acute respiratory distress syndrome (ARDS), the factors associated with auto-PEEP and dynamic hyperinflation (in the absence of expiratory flow limitation) are marked increase in expiratory resistance and the use of high V˙E. Recently, Koutsoukou et al., using a negative-expiratory-pressure technique and flow-volume diagrams, showed that expiratory flow limitation and auto-PEEP were present at zero PEEP in semirecumbent patients with ARDS (Fig. 12). Auto-PEEP values ranged from 0.4 to 7.7 cm H₂O, suggesting that the majority of patients with ARDS have small-airway closure and concomitant auto-PEEP. Patients with ARDS present with decreased lung volume, and breathing at low lung volume promotes airway closure and air trapping, with further reduction in the expiratory flow reserve. Interestingly, applied PEEP and inhaled bronchodilators abolish the expiratory-flow-limitation.

The ARDS Network study found that V{T} of 6 mL/kg (compared to V{T} of 12 mL/kg) reduced mortality by 22% among patients with ARDS. The ARDS Network low-V{T} strategy implies the use of increasing ventilatory support (elevated respiratory rate) to maintain carbon dioxide clearance at normal levels. To date, 3 studies—that did not precisely replicate the ARDS Network study—have shown that an increase in respiratory rate (to avoid V{T}-reduction hypercapnia-increase) may induce substantial gas trapping and auto-PEEP in patients with ARDS. Therefore, it is necessary to pay special attention to monitoring graphic displays of pressure and flow with ARDS patients who have expiratory flow limitation and who are ventilated with short expiratory times, in order to detect inadvertent high total PEEP.

**The Role of Applied PEEP**

To initiate inspiratory flow during patient-triggered breaths, the patient must first counterbalance auto-PEEP. Because that counterbalancing pressure is provided by the inspiratory muscles, auto-PEEP acts as a threshold load for
each inspiratory effort. To alleviate the breathing efforts that auto-PEEP imposes on the respiratory muscles, end-expiratory alveolar pressure can be counterbalanced with applied PEEP. To initiate a ventilator breath, pleural pressure must first reverse the positive recoil pressure present at end-expiration, and the ventilator’s trigger sensitivity must be set appropriately.\textsuperscript{16,41,63} Severely hyperinflated patients with poor muscle function may exhibit some ineffective inspiratory efforts between ventilator-aided breaths. In that situation, applied PEEP (usually about 80% of the baseline auto-PEEP) improves inspiratory-muscle effectiveness.\textsuperscript{16,17,19,41,64} Applied PEEP similar to the level of auto-PEEP should have no effect on alveolar pressure.\textsuperscript{4} Patients ventilated for COPD exacerbation experience dy-
namic airway collapse and flow limitation. Under those conditions, critical closure of the airways occurs. Applying PEEP at a magnitude similar to auto-PEEP causes no impairment in expiratory airflow or increments in alveolar pressure or lung volume. The physiology of this phenomenon is explained by the analogy of the waterfall (Fig. 14).4,17,19 Furthermore, counterbalancing auto-PEEP with a set, applied PEEP has no effect on gas exchange and does not impair hemodynamics or right-ventricular function in patients with COPD.19,65

In contrast to COPD patients, applying PEEP during total ventilatory support of a patient who has dynamic hyperinflation with fixed airflow obstruction due to severe asthma and without airway collapse may produce potentially dangerous increases in lung volume, airway pressure, and intrathoracic pressure, causing circulatory compromise.66 Although some clinical studies67,68 have reported improved airway function (without untoward effects) with continuous positive airway pressure or with noninvasive ventilation and PEEP among patients with acute asthma, the use of PEEP during total ventilatory support of a patient with acute asthma is controversial. Furthermore, with a sedated, well-adapted, nonhypoxemic COPD patient who is receiving controlled mechanical ventilation and exhibiting auto-PEEP, applied PEEP gives no clinical benefit,

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**Fig. 13.** Physiologic variables in a patient with acute respiratory distress syndrome (ARDS) receiving mechanical ventilation according to the ARDS Network’s traditional-tidal-volume (VT) strategy and low-VT strategy.58 The increasing respiratory rate used with the low-VT strategy resulted in higher functional residual capacity (FRC) than the traditional-VT strategy and increased total positive end-expiratory pressure (PEEP_total) above the nominal PEEP set on the ventilator. Paw = airway pressure, PEEP_external = PEEP applied by the ventilator (same as PEEP_nominal), EELV = end-expiratory lung volume, Vr = resting volume. (From Reference 59, with permission.)

**Fig. 14.** Flow and airway pressure (Paw) waveforms from a patient with flow obstruction due to chronic obstructive pulmonary disease. Applying positive end-expiratory pressure (PEEP) that is equal to the static intrinsic PEEP (auto-PEEP) does not increase lung volume (as measured by absence of modification of peak Paw and expiratory flow). Increasing applied PEEP above auto-PEEP elevates intrathoracic pressure (high P Paw), which increases lung volume (increased peak expiratory flow). ZEEP = zero end-expiratory pressure.
unless clinical conditions other than dynamic hyperinflation favor its application.

During acute airflow obstruction, to select an adequate ventilatory pattern and optimal PEEP, it may help the clinician to measure peak pressure, alveolar (plateau) pressure, and auto-PEEP, to calculate the respiratory system’s compliance and resistance, and to periodically control end-expiratory lung volume.69,70

Dynamic Hyperinflation and Patient/Ventilator Interaction

The term patient-ventilator interaction describes the various events that occur during patient-triggered ventilation and how those events influence patient-ventilator synchrony and, thus, patient comfort. Once mechanical ventilation has begun, optimal patient-ventilator interaction must be accomplished to link the ventilator’s output to the patient’s requirements. Adequate selection of inspiratory flow, inspiratory time, and trigger sensitivity is critical for reducing the patient’s WOB.36,37,63

The WOB is determined by the patient’s ventilatory drive and muscle strength. Synchrony with a patient-triggered breath depends on the ventilator’s ability to meet the patient’s flow demand. The ventilator’s peak flow setting must be greater than the patient’s inspiratory flow demand, or the patient is forced to work against the resistance of the ventilator circuit and against his own internal impedance to flow and chest expansion.71,72 Marini et al73 demonstrated that WOB during assisted mechanical ventilation may equal that found during spontaneous breathing if the inspiratory flow setting is inadequate. To avoid high inspiratory effort and reduce patient-ventilator asynchrony, the pressure necessary to trigger the ventilator must be minimal. As mentioned above, the difficulty that auto-PEEP causes in triggering the ventilator represents an elastic threshold load and often manifests as intermittent failure of patient effort to trigger the ventilator. With dynamic hyperinflation, only the most vigorous efforts trigger the ventilator, and ineffective inspiratory-muscle contractions can be observed. This is a common phenomenon in patients with airflow obstruction, and it can cause muscle fatigue, impair the muscles, and increase dyspnea. Therefore, auto-PEEP must be reduced by increasing the time available for expiration or by reducing $V_{E}$.36,37,74,75 Another approach is to add applied PEEP, as described above. Among patients with severe obstruction, the incidence of ineffective triggering does not differ between the pressure-triggered and flow-triggered systems built into modern ventilators.37,76

The pressure and flow waveforms displayed on the ventilator monitor can alert the clinician if the patient’s inspiratory effort is insufficient to trigger the ventilator (Fig. 15). This is particularly important at high levels of ventilator assistance. Breaths that precede nontriggering efforts have shorter respiratory cycle times and expiratory times, so elastic recoil pressure builds up within the thorax, in the form of auto-PEEP.77,78 A full description of the factors that affect patient-ventilator interactions is beyond the scope of this review. However, dynamic hyperinflation can be generated or aggravated by certain manipulations of the ventilator settings (Fig. 16). Inadequate increase in inspiratory flow causes immediate and persistent tachypnea, which shortens expiratory time.77,79,80 Further, the switch from inspiration to expiration on the ventilator should match the patient’s breathing pattern. Neural inspiratory time is often shorter or longer than the inflation time set on the ventilator. That type of asynchrony is very uncomfortable; it causes ineffective triggering or double-triggering, and it aggravates dynamic hyperinflation, increasing the burden on the respiratory muscles.35,36,78,81,82 Clinical observation of the patient and of the flow and pressure waveforms is the best and simplest way to optimize patient-ventilator interaction.

Auto-PEEP and Weaning Failure

Failure of the respiratory-muscle pump is probably the most common cause of failure to wean from mechanical ventilation.83,84 Indeed, in comparison to COPD patients who tolerate spontaneous breathing trials and are successfully extubated, COPD patients who fail spontaneous breathing trials exhibit immediate rapid and shallow breathing and progressive worsening of pulmonary mechanics, with inefficient carbon dioxide clearance.85 Deterioration in respiratory system mechanics in patients who fail spontaneous breathing trials is characterized by an increase in auto-PEEP and inspiratory resistance and a decrease in dynamic lung compliance. Thus, inefficient carbon dioxide clearance in the failing group appears to be a consequence of worsening of pulmonary mechanics, with increased energy expenditure and rapid shallow breathing, because the decrease in $V_{E}$ increases dead space.85

Components of the ventilator circuit, including endotracheal tubes, can increase resistance to airflow and the resistive component of the WOB during spontaneous breathing trials. The important factors are the size of the tube, deposition of secretions in the tube, kinking/curvature of the tube, and the presence of elbow-shaped parts or a heat-and-moisture exchanger in the circuit. Moreover, the components of the ventilator circuit can add dead space. Those factors increase muscle work load and reduce alveolar ventilation for a given $V_{E}$.13,86–88 During spontaneous breathing trials, clinicians should be aware that the aforementioned instrumental elements may be unsuitable for difficult-to-wean patients.
Fig. 15. Flow, airway pressure (Paw), and esophageal pressure (Pes) waveforms from a patient with severe chronic obstructive pulmonary disease, receiving pressure-support ventilation. Ineffective inspiratory efforts occur during both mechanical inspiration and expiration and can be easily identified on the flow waveforms (arrows). (From Reference 37, with permission.)

Fig. 16. Airway pressure (Paw), flow (V˙), esophageal pressure (Pes), and gastric pressure (Pga) waveforms from a representative patient with chronic obstructive pulmonary disease, receiving mechanical ventilation with zero positive end-expiratory pressure (PEEP) (left panel) and with PEEP of 5 cm H2O (right panel). Inspiratory esophageal swings and work of breathing were maximal during ventilation with zero PEEP. Moreover, one ineffective inspiratory effort was identified. Applying PEEP markedly reduced inspiratory efforts, intrinsic PEEP, and patient-ventilator asynchrony. (Adapted from Reference 74, with permission.)
Summary

Dynamic hyperinflation and auto-PEEP are common problems in patients receiving full or partial ventilatory support, as well as in those ready to be weaned from the ventilator. The clinician needs to fully understand the physiology of dynamic hyperinflation and auto-PEEP, so as to choose appropriate ventilator settings. Ventilator graphics are invaluable for monitoring and treatment decisions with patients receiving mechanical ventilation.

REFERENCES


**Discussion**

**Bigatello:** I’m sure I’m not the only one in the audience who is very interested in the value of auto-PEEP in the ARDS Network trial patients, and you mentioned the study by de Durante et al., who applied the ARDS Network’s ventilation criteria to a new group of patients and did detect some auto-PEEP. I seem to remember that a subset of the ARDS Network trial centers looked for auto-PEEP, though I don’t think those data are published. Now I’m not sure which one is right.

Do you have an opinion about that? Of course, whether these patients have auto-PEEP makes a big difference in our thinking about the ARDS Network trial ventilator settings. What is very surprising is that these patients did so well with a lower PEEP than what we generally use, but the reality may be that Durante et al are right and those patients did have a higher PEEP than the set PEEP. So do you have any insight about who’s right?

**REFERENCE**


**Blanch:** I have 2 comments on that. First, there are actually 3 papers that indicate that auto-PEEP can occur at the respiratory rates used in the treatment arm of the ARDS Network trial.1–3

Second, in patients with ARDS, flow limitation has also been shown using low-flow negative aspiration during expiration.4 Therefore, flow limitation can be present in a substantial number of patients with ARDS. In fact, older age, smoking history, or other unknown lung diseases may coexist in patients with ARDS that would favor airflow limitation and be exacerbated by high respiratory rate. Smoking history is not a usual exclusion criteria in big mechanical ventilation trials. Moreover, patients with ARDS may have secretions and airway collapse at low PEEP levels.

Finally, applied PEEP counterbalances autoPEEP, and airflow limitation is relieved in the majority of patients and, consequently, auto-PEEP is not usually found at high PEEP levels. That’s why total PEEP might not coincide with applied PEEP in ARDS patients ventilated at high respiratory rates.

**REFERENCES**


MacIntyre: John Marini taught me that there are only 3 factors that determine auto-PEEP. Number one is minute ventilation—it doesn’t matter whether it’s from respiratory rate or tidal volume. Minute ventilation. Number two is the I-E [inspiratory-expiratory] ratio. Number three is the expiratory time constants. And that’s it.

So it doesn’t surprise me that if you take patients who have a little bit different time constants—maybe they do have as little as you’re suggesting; maybe there is a little more airway obstruction in the European population than in the American population because of smoking habits. The ARDS Network study did not tightly limit the I-E ratio, and you can certainly manipulate the I-E ratio to produce more or less risk of air trapping. And if you drive up the respiratory rate and the minute ventilation, you get air trapping in ARDS patients.

You’re right: the ARDS Network trial did not mandate measurement of auto-PEEP, but there were a couple of centers that did it informally, and on occasion they found auto-PEEP, but it was not a common occurrence. I think it was a slightly different patient population and perhaps slightly different ventilator settings that produced the different results that have been published.¹

REFERENCE


Blanch: I agree with you that it is a mistake to believe that prolonging the expiratory time may relieve auto-PEEP, when the main factor is minute ventilation. However, when additional resistance was measured in the context of incremental levels of PEEP, additional resistance markedly increased at PEEP of 10 cm H₂O or higher. That increase can be caused by a viscoelastic effect or by a pendelluft effect among lung regions that have different time constants. Certainly, pendelluft is associated with auto-PEEP, but that is just a physiologic comment.

Bigatello: Pelosi et al¹ studied increased resistance in ARDS patients. They partitioned in early airway resistance and the late viscoelastic resistance. I think it was all viscoelastic component. That high volume might be what causes that slight resistance increase in ARDS.

REFERENCE


Harris: Marcel Amato has some unpublished data about PEEP in obstructive lung disease that shows that lung volume and expiratory lung volume can actually decrease with increased PEEP in certain situations of obstructive lung disease. Presumably that’s because if you overcome auto-PEEP, you can increase expiratory flow and decrease relaxation lung volume. I’m wondering if you have any data or experience with that?

REFERENCE


Dhand: There are data that even in patients with ARDS there is an increase in the airway resistance.¹² This is responsive to bronchodilators, and it is the initial or the minimal resistance that improves after the administration of bronchodilators. I don’t think that you should totally discount airway involvement in ARDS. I think there are several reasons for the airways to be obstructed in patients with ARDS. With the level of inflammation going on in the lung, it would be very easy to imagine that the small airways would probably be more than in a similar kind of inflammatory process, and that the lung involvement in ARDS is not totally restricted to the alveolar region. Therefore, having auto-PEEP in some patients, I think, would be fairly reasonable.

REFERENCE
