C3.
Hyperinflation in the ICU

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Key Points

1.
The respiratory consequences of hyperinflation in spontaneously breathing, mechanically ventilated patients, are increased work of breathing, overdistention of the ventilatory pumps (pump insufficiency), and wasted or ineffective efforts.

2.
The hemodynamic consequences of hyperinflation are decreased venous return, cardiac output and hypotension. These events are more pronounced in patients in intensive care units under mechanical ventilator support.

3.
The reduction of hyperinflation is the prime aim of the management of respiratory failure in intensive care units. However, this is not always feasible, since ventilator support modes may potentially increase overdistention of the respiratory system.

4.
Therapeutic approaches should include decreasing the level of extensive ventilator assistance by using the appropriate extrinsic PEEP and prolonging the time of expiration by reducing machine inspiratory timing (Ti) or by increasing the expiratory trigger threshold.

5.
Reducing wasted efforts of the patient is another complimentary therapeutic strategy.

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INTRODUCTION

The consequences of hyperinflation in spontaneously breathing mechanically ventilated patients are respiratory (*i.e.*, increased work of breathing, overdistention of the respiratory system and wasted or ineffective efforts) and hemodynamic compromise. In the passive patient (exhibiting no respiratory muscle activity) under controlled mechanical ventilation, the consequences of hyperinflation are overdistention and hemodynamic compromise.

Respiratory Consequences of Hyperinflation

The pathophysiology of hyperinflation of the respiratory system in spontaneously breathing mechanically ventilated patients can be illustrated using the Campbell diagram.

Campbell diagram

The Campbell diagram is constructed by plotting the dynamic relation between pleural pressure (measured with an esophageal balloon) and lung volume during breathing in relation to the passive pressure-volume curves of the lung $Pel_{(L)}$ and the chest wall $Pel_{(cw)}$ (**Figure 1**). The $Pel_{(cw)}$ is constructed by connecting the values taken by the esophageal pressure during passive inflation (*i.e.*, with no respiratory muscle activity) when the airways are closed at different lung volumes. Unfortunately, as this is difficult to do, since it requires passive inflation and often muscle paralysis, a theoretical value for the slope of this curve is frequently used. However, if a patient is passively ventilated and an esophageal balloon is placed, a true value for the volume–pressure relationship of the chest wall during passive tidal breathing can be obtained. This passive pressure–volume relationship can be used as a reference value for subsequent calculations when the patient develops spontaneous inspiratory efforts. Any change in esophageal pressure is referred

Normal breath from the relaxation volume of the respiratory system with expiratory muscle use

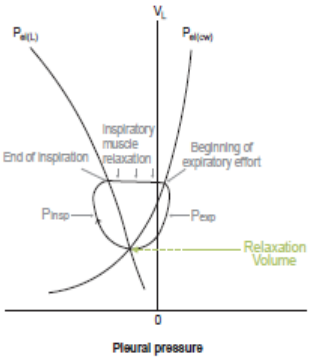

Figure 1. In normal subjects, inspiration starts from the relaxation volume of the respiratory system, where the passive pressure-volume curves of the lung ($Pel_{(L)}$) and chest wall ($Pel_{(cw)}$) intersect. Inspiratory muscle action results in pressure development (P_{insp}) on the left of the pressure-volume curve of the chest wall ($Pel_{(cw)}$). Inspiratory flow, and thus increases in lung volume (V_L) take place on the left of the pressure-volume curve of lung and coincide with the beginning of inspiratory muscle action. Inspiration ends on the pressure-volume curve of the lung and the inspiratory muscles relax (so that pressure returns on the pressure volume curve of the chest wall). In the case shown, expiration is active so that pressure develops on the right of the pressure volume curve of the chest wall due to activity of expiratory muscles (P_{exp}). This returns volume back to the relaxation volume of the respiratory system.

to this line in the Campbell diagram in order to calculate the true muscular pressure developed by the patient. In normal subjects, inspiration starts from the relaxation volume of the respiratory system (V_r), where the $Pel_{(L)}$ and $Pel_{(cw)}$ intersect (*i.e.*, where the tendency of the lung to recoil inward is equal to the tendency of the chest wall to expand; **Figure 1**). Inspiratory muscle action results in pressure development (P_{insp}) on the left of the $Pel_{(cw)}$. Inspiratory flow,

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and thus increases in lung volume (V_L), take place on the left of the $Pel_{(L)}$ and coincide with the beginning of inspiratory muscle action. At any volume, the horizontal distance between the $Pel_{(cw)}$ and $Pel_{(L)}$ represents the portion of inspiratory muscle action devoted to expanding the lung at this volume with open airways (elastic pressure), and the portion on the left of the $Pel_{(L)}$ represents the pressure dissipated to generate airflow (resistive pressure). Inspiration ends on the $Pel_{(L)}$ (point of zero flow) and the inspiratory muscles relax so that pressure returns on the $Pel_{(cw)}$. Expiration is usually passive, and the respiratory system returns to its relaxation volume along the $Pel_{(cw)}$; however, in patients with respiratory distress, such as mechanically ventilated chronic obstructive pulmonary disease (COPD) patients, expiration is frequently active. In the case of active expiration, pressure develops on the right of the $Pel_{(cw)}$ due to activity of expiratory muscles (P_{exp}). This returns volume back towards the relaxation volume of the respiratory system.

In mechanically ventilated patients, inspiratory muscle action has to overcome the trigger sensitivity (P_{tr}) of the ventilator (horizontal distance between the $Pel_{(L)}$ curve and P_{tr}; **Figure 2**) before it results in inspiratory flow and thus increases in V_L. Thus, the beginning of inspiratory effort does not coincide with the beginning of inspiratory flow. Instead, the initial inspiratory effort (orange line in **Figure 2**) is horizontal (no flow, only pressure development) until it crosses the P_{tr} line at which time it deviates upward, indicating inspiratory flow, and thus increases in V_L.

Work of breathing

Measuring work of breathing (WOB) is a useful approach to calculate the total expenditure of energy by the respiratory muscles and/or the ventilator. In general, the work performed during each respiratory cycle is mathematically expressed as $WOB = \int Pressure \times Volume$, *i.e.*, the area on a pressure–volume diagram (**Figure 1-2**). Esophageal pressure is usually taken as a surrogate for intrathoracic (pleural) pressure. Esophageal pressure swings during inspiration are needed to overcome two forces: the elastic forces of the lung parenchyma and chest wall, and the resistive forces generated by the movement of gas through the airways. One can calculate these two components (elastic and resistive) by comparing the difference between esophageal pressure during the patient's effort during the breath and the pressure value in passive

Normal breath overcoming ventilator trigger from the relaxation volume of the respiratory system with expiratory muscle use

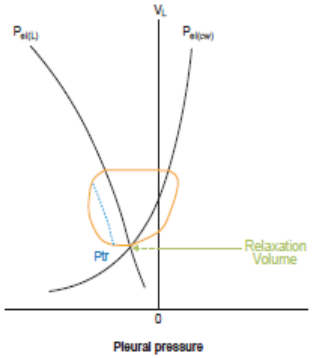

Figure 2. In mechanically ventilated patients, inspiratory muscle action has to overcome the trigger sensitivity of the ventilator (horizontal distance between the $Pel_{(L)}$ curve and P_{tr}) before it results in inspiratory flow and thus increases in V_L. Thus, the beginning of inspiratory effort does not coincide with the beginning of inspiratory flow. Instead, the initial inspiratory effort (orange line) is horizontal (no flow, only pressure development) until it crosses the P_{tr} line at which time it deviates upward indicating inspiratory flow and thus increases in V_L. In the case shown, the trigger sensitivity is pressure triggering.

conditions, represented by the static $Pel_{(cw)}$. The area between the $Pel_{(L)}$ and $Pel_{(cw)}$ represents the elastic work of breathing. The area on the left of the $Pel_{(L)}$ represents the resistive work of breathing. The area on the right of the $Pel_{(cw)}$ represents the work of breathing of the expiratory muscles.

The WOB is normally expressed in joules (J). One J is the energy needed to move 1L of gas through a 10-cmH_2O pressure gradient. The work per liter of ventilation (J/L) is the work per cycle divided by the tidal volume (expressed in liters). In a healthy subject the normal value is around 0.35 J/L. Lastly, WOB can be expressed in *work per unit of time*, multiplying joules per cycle by the respiratory rate (expressed in breaths per minute) to obtain the power of breathing (joules/minute). In a healthy subject the normal value is around 2.4 J/min.

As illustrated by the Campbell diagram, hyperinflation increases the WOB: the higher the End-expiratory lung volume (EELV), *i.e.*, the higher the intrinsic positive end-expiratory pressure (PEEPi), the larger the area of the work of breathing on the Campbell diagram. This is because the $Pel_{(L)}$ and $Pel_{(cw)}$ both deviate as lung volume increases. Consequently, with a constant tidal volume (V_T), the higher the EELV, the larger the elastic component of the work of breathing, and thus the total work of breathing. Intrinsic positive end-expiratory pressure can be quite high, especially in patients with COPD, and may represent a high proportion of the total WOB. For example, a patient who displaces 0.5L of tidal volume through a 7-cmH_2O pressure gradient will perform an amount of work of 0.35J/cycle. If nothing else changes except that this patient develops 5 cmH_2O of PEEPi, 0.25J will be required to counterbalance this, meaning that the total WOB will be 0.60J (0.35 + 0.25), which represents around 40% of the total work required for the inspiration.

Overdistention

In patients with hyperinflation, inspiration starts from an increased EELV, which predisposes to overdistention. The higher the hyperinflation and the higher the inspiratory pressure or volume delivered by the ventilator, the higher the risk of overdistention. The presence of overdistention can be inferred from the airway pressure over time tracing of the ventilator screens when late inspiratory upstroke develops (**Figure**). (Editor: **Figure number missing**)

Wasted efforts

Wasted or ineffective efforts are inspiratory efforts that fail to trigger the ventilator. Nearly 25% of mechanically ventilated patients exhibit ineffective efforts, which are even more frequent in COPD patients. Wasted efforts are a major cause of patient-ventilator dyssynchrony that increase the energy expenditure of the respiratory muscles and may injure them. Understanding their pathophysiology is essential to properly adjust the ventilator settings to attenuate or eliminate them. Wasted efforts should be searched for before any change in ventilator settings is implemented during assisted modes of mechanical ventilation, since any ensuing (after the change in ventilator settings) increase in ventilator rate might be caused by the attenuation of wasted efforts (the ventilator rate now approaching the patient's respiratory controller rate) and not by the development of respiratory distress with the new settings.

How can wasted efforts be detected at the bedside?

Wasted efforts can be clinically detected when the breaths delivered by the ventilator (measured rate on the ventilator display) are less than the number of inspiratory efforts of the patient (on clinical examination) over the same time interval. On modern ventilator screens, ineffective efforts can be detected as abrupt airway pressure drops simultaneous to an abrupt decrease in expiratory flow (from the flow trajectory established earlier during expiration) caused by the inspiratory effort, and not followed by a machine breath (**Figure 3**). Monitors that can automatically detect wasted efforts are available.

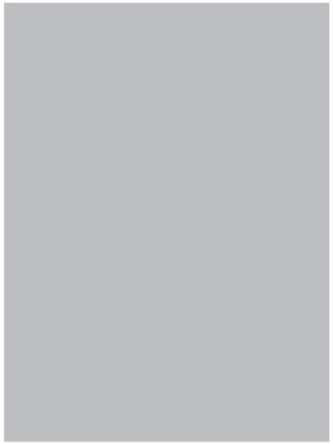

Figure 3. Upper panel: Esopageal (P_{eso}) and airway (P_{ao}) pressures and flow at the tracheostomy in a patient with wasted efforts (black arrows) on flow-controlled, volume-cycled (assist/control) mode. Wasted efforts can be detected as abrupt airway pressure drop simultaneous to an abrupt decrease in expiratory flow (from the flow trajectory established earlier during expiration) caused by the inspiratory effort and not followed by a machine breath. The units are cm H_2O for the pressure tracing and L/min for flow. The patient's inspiratory efforts are identified by the negative P_{eso} swings. The PEEP is set at 0. Pao appropriately drops to 0 during expiration, demonstrating little circuit or valve resistance. Wasted efforts (black arrows) are evident, with one triggered breath (white arrow) every two to three inspiratory efforts. Prolonged expiratory flow is due to airflow limitation. Lower panel: PEEP is now increased to 10 cm H_2O so the P_{eso} during expiration is now 10 cm H_2O. There is persistent flow at end-expiration, thus PEEPi is still present. Wasted efforts (black arrows) have been reduced with one breath triggered every other inspiratory effort. Peak inspiratory pressure and the P_{eso} have increased slightly compared to the upper panel, most likely indicating a higher end-expiratory lung volume and total PEEP level.

Why are spontaneously breathing mechanically ventilated COPD patients prone to develop wasted/ineffective efforts?

In COPD patients with hyperinflation, inspiration starts from an increased end-expiratory lung volume (**Figure 4**). Inspiratory muscle action has to overcome the PEEPi (red

Breath from increased end-expiratory lung volume due to the presence of dynamic hyperinflation in COPD

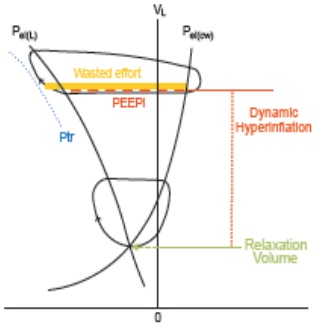

Figure 4. In COPD patients with dynamic hyperinflation, inspiration starts from an increased end-expiratory lung volume. Inspiratory muscle action has to overcome the intrinsic positive end expiratory pressure (PEEPi, horizontal distance between the between the $Pel_{(L)}$ and $Pel_{(cw)}$) before it results in inspiratory flow and thus increases in volume. In mechanically ventilated patients, inspiratory muscle action has to overcome PEEPi plus the trigger sensitivity (P_{tr}) before it results in inspiratory flow and thus increases in volume (V_t). When the magnitude of inspiratory muscle action is less than the sum of PEEPi + P_{tr}, this inspiratory effort (orange horizontal line) cannot trigger the ventilator, and consequently does not result in inspiratory flow and thus increases in volume (V_t). This inspiratory effort is called ineffective or wasted.

dashed line, horizontal distance between the $Pel_{(L)}$ and $Pel_{(cw)}$) before it results in inspiratory flow and thus increases in V_L. In mechanically ventilated patients, inspiratory muscle action has to additionally overcome the Ptr of the ventilator (horizontal distance between the $Pel_{(L)}$ and P_{tr}) before it results in inspiratory flow and thus increases in V_L; thus, in mechanically ventilated COPD patients, inspiratory muscle action has to overcome PEEPi plus the P_{tr}. When the magnitude of inspiratory muscle action is less than the sum of PEEPi plus P_{tr}, this inspiratory effort (**Figure 4**, orange line) cannot trigger the ventilator, and consequently does not result in inspiratory flow and thus increases in V_L. This inspiratory effort is called "ineffective" or "wasted".

Hyperinflation not only increases PEEPi, but at the same time renders the respiratory muscles weaker for a variety of reasons: (see chapter …) (Editor: Chapter number missing)

The greater the degree of hyperinflation, the higher the PEEPi, and the weaker become the respiratory muscles. The net effect is a greater predisposition to wasted efforts.

Is the Campbell diagram useful for estimating the work of breathing during wasted efforts?

The Campbell diagram is not useful to estimate inspiratory work of breathing when inspiratory triggering does not happen (*i.e.*, during wasted efforts): work is physically defined as the area subtended in a pressure/volume loop. Since there is no inspiratory volume, the work is zero (albeit muscles, indeed, consume energy). The energy expenditure during non-triggered wasted inspiratory efforts can be estimated by the pressure/time product: the product of the pressure developed by the inspiratory muscles (difference between the measured esophageal pressure and the $Pel_{(cw)}$) multiplied by the time of muscle contraction (*i.e.*, neural inspiratory time [Ti]).

How are ventilator settings affecting the incidence of wasted/ineffective efforts?

Excessive ventilator support predisposes to ineffective efforts irrespective of the mode of mechanical ventilation used. This is because, in the case of COPD, excessive pressure or volume delivered by the ventilator results in increased EELV, and this combined with the long time constant of the respiratory system (which retards lung emptying) and/or a short imposed expiratory time (in case of volume or pressure control) results in further increased EELV before the next inspiratory effort begins (**Figure 5**, green curve).

At the same time, excessive ventilator assistance reduces inspiratory muscle effort, via either a phenomenon called neuromechanical inhibition (the main mechanism most likely being the Hering–Breuer reflex), and/or by producing alkalemia via excessive CO_2 reduction in patients with chronic bicarbonate elevation, thus reducing the drive to the respiratory muscles.

The ensuing inspiratory effort is inadequate to overcome PEEPi plus Ptr; thus, this inspiratory effort fails to trigger the ventilator (ineffective or wasted effort) (**Figure 5**, red line). This is, of course, exaggerated in the presence of respiratory muscle weakness. The next expiratory effort (**Figure 5**, blue curve) decreases the EELV. When EELV decreases to a level where the ensuing inspiratory effort exceeds PEEPi plus Ptr, the ventilator is triggered again to deliver a machine breath (**Figure 5**, purple curve). The breath-to-breath variability in breathing pattern contributes to the variability in the EELV and thus to the frequency of ineffective efforts.

During assist volume or pressure control mechanical ventilation, prolonged imposed inspiratory time (machine Ti) greater than the patient's neural Ti results in a situation where the ventilator is inflating the patient long after the inspiratory muscles have stopped their

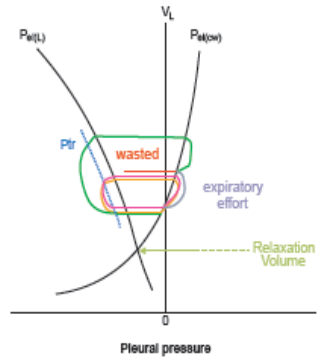

Figure 5. In the case of COPD presented in this figure, the first inspiratory effort (orange line) triggers the ventilator. In the next breath that triggered the ventilator, excessive ventilator assistance (either pressure or volume) resulting in large tidal volume, combined with the long time constant of the respiratory system (which retards lung emptying) and/or a short imposed expiratory time (in case of volume or pressure control) result in further increased end-expiratory lung volume before the next inspiratory effort begins (green line). The ensuing inspiratory effort despite being equal to the previous effective effort is inadequate to overcome PEEPi + P_{tr} due to the increased EELV. Thus this inspiratory effort fails to trigger the ventilator (wasted or ineffective effort, red line). The following expiratory effort (blue line) decreases the end-expiratory lung volume. When the end-expiratory lung volume decreases to a level where the ensuing inspiratory effort exceeds PEEPi +P_{tr}, the ventilator is triggered again to deliver a machine breath (purple line).

contraction, *i.e.*, during the neural expiration. This results in increased EELV and a shorter time available for expiration, both of which increase EELV (more hyperinflation) and thus predispose to wasted efforts.

During pressure support ventilation, a low expiratory trigger threshold (percentage of peak inspiratory flow at which the ventilator cycles to expiration) might lead to pressure support being delivered well beyond the patient's neural Ti. The next inspiratory effort (controlled by the patient's respiratory controller) begins during the early phase of ventilator expiration, *i.e.*, at an increased VL. This effort might not be sufficient to overcome PEEPi plus Ptr, and thus, this inspiratory effort also fails to trigger the ventilator.

Therapeutic approach to overdistention

An obvious solution to reduce overdistention is to decrease the level of excessive ventilator assistance (*i.e.*, the delivered pressure or volume). This will decrease EELV. However, this might not be always clinically feasible, since it might lead to respiratory distress and derangement of blood gases.

Another not mutually exclusive approach is to decrease hyperinflation by prolonging the expiratory time. With a constant VT, decreasing hyperinflation will decrease overdistention:

1. During assist volume or pressure control, reducing machine Ti (or equivalently increasing the inspiratory flow in volume control) may prolong expiratory time (if the respiratory frequency, and thus the total time of the respiratory cycle, does not change) and will reduce hyperinflation.
2. During pressure support, increasing the expiratory trigger threshold will stop the breath earlier, *i.e.*, at a lower EELV, and will prolong expiratory time and thus, will decrease hyperinflation.

Therapeutic approach to wasted efforts: What ventilator adjustments should be done in the presence of wasted/ineffective efforts?

One solution to reduce the frequency of wasted efforts is to decrease the level of excessive ventilator assistance (*i.e.*, the delivered pressure or volume). This will decrease the EELV and with a given (or even

increased) time for expiration will reduce EELV-hyperinflation and will reverse the pathophysiology presented above. In clinical practice one may start gradually decreasing the level of applied pressure during assist pressure control or pressure support in steps of 2 cm H_2O or the delivered volume in assist volume control by 10%. However, this might not be always clinically feasible, since it might lead to respiratory distress and to derangement of blood gases.

During assist volume or pressure control reducing machine Ti (or equivalently increasing the inspiratory flow in volume control) may prevent ventilator delivery beyond the patients' neural Ti. This will prolong expiratory time and will reduce wasted efforts. In practice, machine Ti may be reduced in steps of 10%. This titration may be stopped when ineffective triggering is eliminated or the patient shows poor tolerance.

During pressure support, increasing the expiratory trigger threshold will stop the breath earlier, and thus at a lower EELV, and will prolong expiratory time. Both will reduce wasted efforts. Practically, the insufflation time may be gradually reduced, by increasing the cycling-off criterion in steps of 10%; if ineffective triggering persists at the highest value (which depends on the ventilator), the insufflation time may be further reduced by adjusting the maximal insufflation time in steps of 0.2s from the mean insufflation time; this titration may be stopped when ineffective triggering is eliminated or the patient shows poor tolerance.

Another solution is the application of PEEP. The addition of an amount of external PEEP lower than the PEEPi (**Figure 6**, red dotted lines) offers part of the pressure required to overcome PEEPi plus P_{tr} (**Figure 6**). The inspiratory effort starts closer to the $Pel_{(L)}$ (the horizontal distance between this point and the Pel(cw) being the applied PEEP [PEEP external]). The inspiratory effort is now adequate to trigger the ventilator (orange curve).

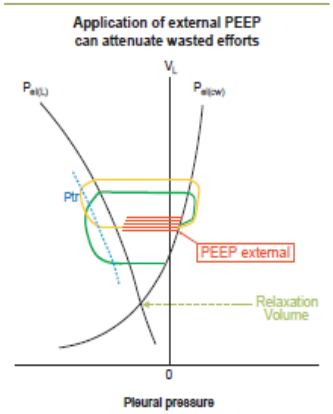

Figure 6. In the presence of increased EELV (green line), addition of an amount of external PEEP lower than the PEEPi (red dotted lines) offers part of the pressure required to overcome PEEPi+P_{tr}. The inspiratory effort starts closer to the passive pressure-volume curve of the lung ($Pel_{(L)}$). The horizontal distance between this point and the passive pressure- volume curve of the chest wall ($Pel_{(cw)}$) is the applied PEEP (PEEP external). The inspiratory effort is now adequate to trigger the ventilator (orange line).

In clinical practice, one may begin by applying small amounts of PEEP (e.g., 2 cmH_2O) and gradually increase the applied PEEP until wasted efforts obtain their minimal value. Increasing the applied PEEP above a certain value, i.e. the value of PEEPi (which is, however, difficult to measure), will increase the end inspiratory lung volume and will start to predispose again to wasted efforts. The greater the increase of applied PEEP above PEEPi, the more likely the reappearance or increase in the number of wasted efforts.

Measurement of PEEPi

It becomes obvious that an accurate measurement of PEEPi is needed. In passive

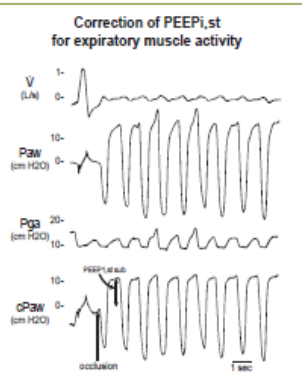

Figure 7. Recordings of Flow ($\dot{V}$), P_{aw}, P_{ga} and on line "corrected" airway pressure (cP_{aw}) in a representative actively expiring patient during airway occlusion. cP_{aw} is obtained by subtracting the P_{ga},exp rise from P_{aw}. Note a consistent end-expiratory plateau in cP_{aw} despite marked variability in P_{ga} swings. From this plateau, the PEEPi,st sub is measured.

mechanically ventilated patients, PEEPi is routinely measured under static conditions (PEEPi,st, or static PEEPi) as the plateau in airway pressure during a prolonged end-expiratory airway occlusion. In spontaneously breathing mechanically ventilated patients estimation of PEEPi is difficult without the placement of esophageal balloons and gastric balloons. In actively breathing patients on assisted ventilation, PEEPi has been assessed dynamically from records of esophageal pressure (Pes) obtained with the placement of an esophageal balloon. The decrease in Pes needed to abruptly bring expiratory flow to zero during unoccluded breathing is taken as dynamic PEEPi (PEEPi,dyn).

However, expiratory muscle activity (which causes an expiratory rise in gastric pressure measured with a gastric balloon [Pga,exp rise]) can increase the end-expiratory alveolar pressure independently of dynamic hyperinflation, leading to an overestimation of PEEPi. In spontaneously breathing and actively expiring patients, PEEPi,st can be corrected for expiratory muscle contraction by synchronously subtracting the expiratory rise in gastric pressure (Pga,exp rise) from the end-expiratory airway pressure (Paw) occurring during airway occlusion (**Figure 7**, PEEPi,st sub).

In the case that PEEPi,dyn is part of the decrease in Pes preceding inspiration, which

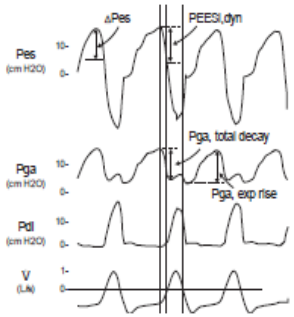

Figure 8. Tracings of esophageal pressure (P_{es}), gastric pressure (P_{ga}), transdiaphragmatic pressure (Pdi) and flow ($\dot{V}$) in a patient actively expiring during a spontaneous breathing trial through the ventilator (pressure support of 6 cm H_2O). The three vertical lines are passed through the onset of inspiratory muscle activity (i.e., beginning of P_{es} decay) and the beginning and the end of inspiratory flow, respectively. Note the large increase of P_{ga} and P_{es} during expiration due to expiratory muscle recruitment. ΔP_{es} is the increase in end-expiratory P_{es} relative to its level at the onset of spontaneous breathing trial, when expiratory muscle activity was nil. P_{ga},total decay represents the abrupt decrease in Pga from its maximum end-expiratory value to its minimum value at the beginning of inspiration due to relaxation of the abdominal muscles. P_{ga},exp rise is the P_{ga} rise from its minimal end-inspiratory level to the maximal level at end expiration. The decay in P_{es} between the first two vertical lines represents dynamic intrinsic positive end-expiratory pressure (PEEPi,dyn).

is measured as PEEPi,dyn, is actually due to relaxation of the expiratory muscles, rather than contraction of the inspiratory muscles to counterbalance PEEPi. The amount of pressure due to expiratory muscle activity that should be subtracted from the measured PEEPi,dyn to obtain the actual ("true") PEEPi,dyn elicited by dynamic hyperinflation is either the increase in gastric pressure over the course of expiration (Pga,exp rise) attributed to expiratory muscle contraction or the total decrease in gastric pressure observed at the beginning of inspiration attributed to expiratory muscle relaxation (Pga,total decay); the latter being somewhat more accurate than the former (**Figure 8**).

HEMODYNAMIC CONSEQUENCES OF HYPERINFLATION

At the end of a normal expiration, alveolar and airway pressures are zero relative to atmosphere, and esophageal pressure is negative (around -5 cmH_2O in normal conditions). However, in the presence of hyperinflation (because of either dynamic airway collapse or inadequate time to exhale), the alveolar pressure remains positive throughout expiration. This positive alveolar pressure is transmitted to the pleural-intrathoracic cavity to varying degrees depending on the compliance of the lung. In the case of COPD patients, the lungs are compliant, so most of the positive alveolar pressure is transmitted to the intrathoracic cavity. This increases the mean intrathoracic pressure during the whole respiratory cycle. The effect is more pronounced in the passive patient, because during inspiration (pressure or flow delivery by the ventilator), the intrathoracic pressure becomes even more positive than the pressure existing at the end of expiration. In the spontaneously breathing mechanically ventilated patient, the inspiratory effort of the patient decreases the mean inspiratory pressure and thus the mean intrathoracic pressure. However the average, *i.e.*, the mean intrathoracic pressure, is still positive. The more positive the mean intrathoracic pressure, the more pronounced its hemodynamic effects. The hemodynamic effects of hyperinflation are the result of a complex interaction between changes in preload, secondary to changes in the venous return, right—left ventricle interactions, direct effects of lung inflation and changes in afterload. At moderate degrees of hyperinflation, decreased venous return is the main mechanism leading to decreased cardiac output. The hyperinflated lung compresses the pericardium and especially the pericardial fossa, increasing their pressure (pericardial and juxtacardial respectively). This pressure is transmitted to the right atrial cavity increasing the right atrial pressure, which is the downstream pressure for venous return. However, hyperinflation with the resulting PEEPi also elevates the upstream pressure driving venous return (*i.e.*, the mean systemic pressure) by both reflex and mechanical means, independent of the abdominal pressure. The positive intrathoracic pressure also changes the resistive and elastic properties of peripheral veins, increases venous resistance, and directly compresses the inferior vena cava. The net effect of all these phenomena is a decrease in the venous return.

At the same time that hyperinflation decreases the preload of the right ventricle, it also increases its afterload, *i.e.*, the pulmonary vascular resistance. The more lung volume increases above the relaxation volume of the respiratory system, the more the intra-alveolar vessels are compressed, creating a starling resistor phenomenon. This dominates over vessel recruitment, resulting in increased pulmonary vascular resistance. The combination of decreased preload and increased afterload of the right ventricle results in decreased stroke volume, and thus in decreased preload of the left ventricle. Despite the fact that the elevations in intrathoracic pressure decrease the transmural pressures of the left heart, and thus its afterload, the net effect is a decrease in cardiac output and/or hypotension.

Of course, the effects of hyperinflation on hemodynamics also depend on the contractile function of the right and left heart and the volume status of the patient. The more hypovolemic (volume depleted) a patient is, the more vulnerable they become to the effects of hyperinflation on the venous return and the right ventricular afterload; thus, the more vulnerable the patient to the development of hypotension and decreased cardiac output.

In spontaneously breathing mechanically ventilated patients, application of external PEEP is beneficial to counterbalance intrinsic PEEP (see above). The hemodynamic effects of this PEEP application depend on its magnitude. Positive end-expiratory pressure amounting to 85-90% of PEEPi have no hemodynamic effects, whereas levels of PEEP above this level and especially well above PEEPi (e.g., by 5cmH_2O) decrease cardiac output. It is needless to say that levels of PEEP that are higher than PEEPi are counterintuitive, and should not be used.

Suggested Reading

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