Advances in Respiratory Monitoring During Mechanical Ventilation*

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This review provides an update on the various techniques that are available to monitor patients during mechanical ventilation with an emphasis on clinical observations and applications in critically ill patients.

Key words: elastance; pressure-time product; pulse oximetry; resistance

Abbreviations: 
ABG = arterial blood gas; ΔR = additional resistance; Edyn,L = dynamic elastance of the lung; Edyn,rs = dynamic elastance of the respiratory system; Est,rs = static elastance of the respiratory system; f = respiratory frequency; Hb = reduced hemoglobin; O₂Hb = oxyhemoglobin; P₀₁ = mouth occlusion pressure at 0.1 s after onset of inspiratory effort; Paw = airway pressure; PEEP = positive end-expiratory pressure; PEEPi = intrinsic positive end-expiratory pressure; Peso = esophageal pressure; Petco₂ = end-tidal Pco₂ concentration; Pga = gastric pressure; Pinit = initial pressure; Ppeak = peak pressure; Pplat = plateau pressure; PTP = pressure-time product; Rmax = maximum resistance; Rmin = minimum resistance; SaO₂ = arterial oxygen saturation; SpO₂ = pulse oximeter estimate of arterial oxygen saturation; Vₜ = tidal volume; WOB = work of breathing

Several advances in monitoring gas exchange, neuromuscular capacity, respiratory mechanics, and patient effort during mechanical ventilation have occurred in recent years. Monitoring these parameters is helpful in minimizing ventilator-induced complications, optimizing patient-ventilator interaction, and determining a patient’s readiness for the discontinuation of mechanical ventilation.

**GAS EXCHANGE**

With the proliferation of pulse oximeters in different locations of the hospital throughout the 1980s, several investigators demonstrated that episodic hypoxemia is much more common than previously suspected, with an incidence ranging from 20 to 82%.

In patients admitted to a general medical service, Bowton et al found that patients who experienced hypoxemia (O₂ saturation < 90% for ≥ 5 min) during the first 24 h of hospitalization had a mortality rate more than three times higher than patients who did not experience desaturation. Whether or not the early detection and treatment of episodic hypoxemia can affect patient outcome remains to be answered.

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**Pulse Oximetry**

Pulse oximetry is based on two physical principles: (1) the presence of a pulsatile signal generated by arterial blood, and (2) the fact that oxyhemoglobin (O₂Hb) and reduced hemoglobin (Hb) have different absorption spectra. Currently available oximeters use two light-emitting diodes that emit light at the 660 nm (red) and the 940 nm (infrared) wavelengths. These two wavelengths are used because O₂Hb absorbs less light than Hb, while the reverse occurs in the infrared region. The ratio of absorbencies at these two wavelengths is calibrated empirically against direct measurements of arterial oxygen saturation (SaO₂) in volunteers, and the resulting calibration algorithm is stored in a digital microprocessor within the pulse oximeter. During subsequent use, the calibration curve is used to generate the pulse oximeter’s estimate of arterial oxygen

Accuracy: The accuracy of commercially available oximeters varies widely, probably because of the different algorithms employed in signal processing. Oximeters commonly have a mean difference (bias) of < 2% and an SD (precision) of < 3% when SaO₂ is ≥ 90%. Accuracy of pulse oximeters deteriorates when SaO₂ falls to 80% or less. In a study of 54 ventilator-dependent patients, the bias ± precision of oximetry was 1.7 ± 1.2% for SaO₂ values > 90%,
and it increased to $5.1 \pm 2.7\%$ when $\text{S}a_2\text{O}_2$ was $\leq 90\%$.5

**Limitations:** Pulse oximeters employ only two wavelengths of light, and thus can distinguish only two substances, Hb and $\text{O}_2\text{Hb}$. Elevated carboxyhemoglobin and methemoglobin levels can cause inaccurate oximetry readings.1 Anemia does not appear to affect the accuracy of pulse oximetry: in nonhyperoxic patients with acute anemia (mean Hb, $5.2 \pm 0.3$ [SEM] g/dL), pulse oximetry was accurate in measuring $\text{O}_2$ saturation with a bias of only 0.53%.6 Moreover, in patients with sickle cell anemia who presented with acute vaso-occlusive crisis, Ortiz et al7 found that pulse oximetry overestimated $\text{S}a_2\text{O}_2$ by an average of 3.4%; the error of $\text{SpO}_2$ was never enough to misdiagnose either hypoxemia or normoxemia in such patients.

Motion artifact continues to be a significant source of error and false alarms.1,8 In a recent prospective study in an ICU setting, $\text{SpO}_2$ signals accounted for almost half of a total of 2,252 false alarms9 (Fig 2). Various methods have been employed to reject motion artifact, but have met with little success. An innovative technologic approach, termed Masimo signal extraction technology (Masimo SET; prototype), was recently introduced to extract the true signal from artifact due to noise and low perfusion.10 This technique incorporates new algorithms for processing the pulse oximeter’s red and infrared light signals that enable the noise component, which is common to the two wavelengths, to be measured and subtracted. When tested in healthy volunteers during standardized motion, Masimo SET exhibited much lower error rates (defined as percentage of time that the oximeter error exceeded 5, 7, and 10%) and dropout rates (defined as the percentage of time that the oximeter provided no $\text{SpO}_2$ data) than did the Nellcor N-200 and Nellcor N-3000 oximeters (Nellcor Puritan-Bennett Corp; Pleasanton, CA).11 In 50 postoperative patients, the pulse oximeter’s alarm frequency was decreased twofold with a Masimo SET system vs a conventional oximeter (Nellcor N-200).12

**Clinical Applications:** Moller et al13 conducted the first prospective, randomized study of pulse oximetry on the outcome of anesthesia care in 20,802 surgical patients. A 19-fold increase in the detection of hypoxemia (defined as an $\text{SpO}_2 < 90\%$) was noted in the oximeter group vs the control group. Myocardial ischemia was more common in the control group than in the oximetry group (26 and 12 patients, respectively). However, pulse oximetry did not decrease the rate of postoperative complications or mortality.

Pulse oximetry can assist with titration of the fraction of inspired oxygen concentration ($\text{F}\text{io}_2$) in ventilator-dependent patients, although the appropriate $\text{SpO}_2$ target depends on a patient’s pigmenta-
tion.5 In white patients, a $\text{SpO}_2$ target value of 92% predicts a satisfactory level of oxygenation, whereas in black patients, this target may result in significant hypoxemia. While a higher target $\text{SpO}_2$ value of 95% avoids hypoxemia in black patients, some will have $\text{PaO}_2$ values as high as 198 mm Hg; if such patients

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**Figure 1.** Red (R) and infrared (IR) scaled alternating current signals at ($\text{S}a_2\text{O}_2$) of 0, 85, and 100%. The numeric value of the red-to-infrared ratio (R/IR) can be easily converted to $\text{S}a_2\text{O}_2$. Reprinted with permission from Wukitsch et al.6

**Figure 2.** Number of false alarms for devices used to monitor respiratory rate, mean systemic BP from an arterial catheter, heart rate from an ECG, heart rate measured by pulse oximetry (P Ox), and $\text{SpO}_2$. Forty-five percent of all false alarms were from the $\text{SpO}_2$ signal. Based on data from Tsien and Fackler.6
receive a high $F_{1O_2}$ to achieve the $SpO_2$ target of 95%; oxygen toxicity may result.

The potential usefulness of pulse oximetry as a screening tool that could supplement or supplant respiratory rate as a "pulmonary vital sign" was recently investigated in > 12,000 adult patients in the triage area of an emergency department.\textsuperscript{14} The relationship between $SpO_2$ and respiratory rate (counted while auscultating breath sounds for 1 min) revealed correlation coefficients of 0.378 to \(-0.454\), with a weighted mean of \(-0.160\) (ie, a weak inverse relationship between $SpO_2$ and respiratory rate). The study confirmed previous observations that respiratory rate alone is not accurate in detecting hypoxemia.

Cost-effectiveness: In an emergency department, a recent report showed that the number of unjustified arterial blood gas (ABG) samples (as determined by independent experts) during a 2-month period decreased from 29% when pulse oximetry was unavailable to 12% when oximetry was available; the number of justified ABGs did not change.\textsuperscript{15} Inman et al\textsuperscript{16} examined the effect of implementing pulse oximetry without any specific algorithm for its appropriate use. They studied 148 patients before the implementation of oximetry in their ICU and 141 patients after its implementation. The number of ABG samples decreased from 7.2 to 6.4 per patient per day—a reduction of only 10.3%, compared with average reductions of 39% in the previous studies.\textsuperscript{1} This suggests that in the absence of explicit guidelines, the pulse oximeter was used in addition to, rather than instead of, ABG samples.

Pulse oximetry is probably one of the most important advances in respiratory monitoring. Perhaps the major challenge facing pulse oximetry is whether this technology can be incorporated effectively into diagnostic and management algorithms that can improve the efficiency of clinical management in the ICU.

Capnography

The end-tidal $PCO_2$ concentration (PET$CO_2$) is the value of exhaled gas taken at the plateau of the CO$_2$ waveform. In healthy subjects, PET$CO_2$ is usually 1 mm Hg (range, up to 5 mm Hg) less than Pa$CO_2$.\textsuperscript{17} Consequently, PET$CO_2$ can be employed as a continuous, indirect measure of Pa$CO_2$. Hoffman et al\textsuperscript{18} obtained simultaneous measurements of PET$CO_2$ and Pa$CO_2$ in 20 intubated patients with respiratory failure 5 to 10 min after altering settings on the mechanical ventilator. The correlation between PET$CO_2$ and Pa$CO_2$ was good ($r = 0.78$). However, the correlation between changes in PET$CO_2$ and changes in Pa$CO_2$ from baseline was considerably weaker ($r = 0.58$). Importantly, four patients demonstrated a trend in PET$CO_2$ opposite in direction to the trend in the Pa$CO_2$. Likewise, Hess et al\textsuperscript{19} found that the change in PET$CO_2$ incorrectly indicated the direction of change in Pa$CO_2$ in 43% of patients being weaned from mechanical ventilation following cardiac surgery.

Respiratory Neuromuscular Function

Airway Occlusion Pressure

Measuring mouth occlusion pressure at 0.1 s after onset of inspiratory effort against an occluded airway (P$_{O.1}$) provides a measure of respiratory drive. In ventilator-dependent patients, P$_{O.1}$ has been shown to correlate significantly with work of breathing (WOB) during pressure-support ventilation (PSV; $r = 0.87$).\textsuperscript{20} Several studies have indicated that an elevated P$_{O.1}$ predicted weaning failure, but the threshold separating success from failure differed among the studies.\textsuperscript{21,22}

Breathing Pattern

Minute ventilation should be partitioned into tidal volume (V$_T$) and respiratory frequency (f). In healthy subjects, f is approximately 17 breaths/min and V$_T$ is approximately 400 mL.\textsuperscript{17} An elevated frequency is often the earliest sign of impending respiratory distress, and the degree of elevation is proportional to the severity of the underlying lung disease. Rapid shallow breathing is a common finding in patients who fail a trial of weaning from mechanical ventilation,\textsuperscript{23} and this can be quantitated in terms of the f/V$_T$ ratio; a value > 100 breaths/min/L suggests that a trial of weaning is unlikely to be successful.\textsuperscript{24} Rapid shallow breathing has been considered a useful strategy to avoid fatigue during a failed weaning trial. However, rapid shallow breathing develops immediately following the discontinuation of mechanical ventilation and does not progress with time—a response that is difficult to attribute to fatigue. Moreover, data in patients failing a weaning trial indicate a poor correlation between f/V$_T$ and the tension-time index, a crude index of impending respiratory muscle fatigue. To serve as a compensatory strategy to avoid fatigue, f/V$_T$ should have a negative correlation with tension-time index, whereas $r$ was found to be 0.08.\textsuperscript{25}

Maximal Inspiratory Airway Pressure

Global inspiratory muscle strength is assessed by measuring maximal inspiratory pressure while the patient makes a maximum inspiratory effort against
an occluded airway, preceded by complete exhalation to residual volume. To obtain more reproducible recordings, a two-step modification was introduced consisting of a one-way valve to ensure that inspiration begins at a low lung volume and maintaining the period of occlusion for 20 s.\textsuperscript{26,27}

Maximal inspiratory pressure is one of the standard measurements employed to determine a need for the continuation of mechanical ventilation. Values that are more negative than $-30$ cm H\textsubscript{2}O are thought to predict weaning success, while values that are less negative than $-20$ cm H\textsubscript{2}O are predictive of weaning failure. However, these criteria are frequently falsely positive and falsely negative.\textsuperscript{24}

**Respiratory Mechanics**

Measurements of respiratory mechanics in a relaxed ventilator-dependent patient can be obtained using the technique of rapid airway occlusion during constant flow inflation.\textsuperscript{28} Rapid airway occlusion at the end of a passive inflation produces an immediate drop in both airway pressure (Paw) and transpulmonary pressure ($P_l$) from a peak value ($P_{\text{peak}}$) to a lower initial value ($P_{\text{init}}$) followed by a gradual decrease until a plateau ($P_{\text{plat}}$) is achieved after 3 to 5 s\textsuperscript{29,30} (Fig 3). $P_{\text{init}}$ is measured by back extrapolation of the slope of the latter part of the pressure tracing to the time of airway occlusion.\textsuperscript{28} $P_{\text{plat}}$ on the Paw, $P_l$, and pleural pressure (Pes) tracings represents the static end-inspiratory recoil pressure of the total respiratory system, lung, and chest wall, respectively.

**Elastance**

The end-inspiratory airway occlusion method is clinically used to measure the static compliance of the respiratory system or its reciprocal, elastance of the respiratory system (Est,rs), according to the following equation\textsuperscript{31}:

$$\text{Est,rs} = \frac{(P_{\text{plat}} - \text{PEEPi})}{V_t}$$

where $P_{\text{plat}}$ is plateau pressure obtained after occluding the airway, PEEPi is intrinsic positive end-expiratory pressure (PEEP), and $V_t$ is tidal volume. Using an esophageal balloon catheter, Est,rs can be partitioned into its lung and chest wall components by dividing $[P_{\text{plat}} - \text{PEEPi}]$ by $V_t$ on the $P_l$ and Pes tracings, respectively (Fig 3).

In mechanically ventilated patients with acute respiratory failure secondary to COPD or pulmonary edema, Est,rs is higher than in normal subjects.\textsuperscript{30} Static lung elastance is higher in patients with pulmonary edema than in patients with COPD, whereas static chest wall elastance was similar in both patient groups.\textsuperscript{30}

**Dynamic Compliance**

An index that commonly is referred to as effective dynamic compliance, or the dynamic characteristic, can be derived by dividing the ventilator-delivered $V_t$ by $[\text{peak Paw} - \text{PEEP}]$. This index is not a measure of true thoracic compliance because peak Paw includes all of the resistive and elastic pressure losses of the respiratory system and endotracheal tube. Alternatively, dynamic elastance of the respiratory system (Edyn,rs) can be obtained by dividing the difference in Paw at points of zero flow by the delivered $V_t$.\textsuperscript{31} Accordingly, Edyn,rs can be computed according to the formula:

\begin{figure}
\centering
\includegraphics[width=\textwidth]{flow_paw_p_l_pes_tracings.png}
\caption{Flow (inspiration upwards), Paw, $P_l$, and Pes tracings in a representative patient during passive ventilation. An end-inspiratory occlusion produced a rapid decline in both Paw and $P_l$ from $P_{\text{peak}}$ to a lower $P_{\text{init}}$, followed by gradual decrease to $P_{\text{plat}}$. Reprinted with permission from Jubran and Tobin.\textsuperscript{29}}
\end{figure}
Edyn,rs = \text{Pinit} - \text{PEEPi/Vt}

Edyn,rs can be partitioned into its lung (Edyn,L) and chest wall components by dividing \([\text{Pinit} - \text{PEEPi}]\) on \(P_l\) and \(Pes\) tracings, respectively (Fig 3). In a recent study,\(^29\) Edyn,rs was found to be similar in patients with COPD who went on to fail a trial of spontaneous breathing and in a control group who tolerated the trial and were extubated. In both groups, Edyn,rs was predominantly influenced by Edyn,L because the values of chest wall dynamic elastance were normal. Edyn,L was significantly higher in the failure group than in the success group, but the individual values showed a considerable overlap among the patients in the two groups, thus limiting its usefulness in signaling a patient’s ability to sustain spontaneous ventilation.

**Pressure-Volume Curves**

A pressure-volume curve of the respiratory system can be constructed in a paralyzed patient by measuring the airway pressure as the lungs are progressively inflated with a 1.5- to 2-L syringe. A lower inflection point and an upper inflection point may be seen on the pressure-volume curve.\(^32\) The lower inflection point is thought to reflect the point at which small airways or alveoli reopen, corresponding to closing volume. In patients with acute lung injury, some investigators have recommended that PEEP should be set at a pressure slightly above the lower inflection point.\(^33\) In a prospective, randomized study in 28 patients, Amato et al.\(^34\) compared an “open-lung approach”—consisting of a lower \(V_t\) (\(< 6 \text{ mL/kg}\)) with PEEP individually titrated to be consistently above the inflection point on the static pressure-volume curve of the respiratory system on a PEEP of 0 cm H\(_2\)O—with a conventional approach consisting of \(V_t\) of 12 mL/kg and a low PEEP level. A significant improvement in oxygenation and mechanics together with a higher weaning rate were observed in the open-lung approach. The authors reported that when the study was extended to 48 patients, mortality was significantly reduced in the group treated with the new approach\(^35\) (Fig 4). Two multicenter, randomized studies looking only at the effects of reducing \(V_t\) did not find any significant difference in mortality,\(^36,37\) indirectly suggesting that the individual titration of PEEP in the Amato study\(^34\) may be an important factor.

**Resistance**

Airway resistance can be measured in ventilator-dependent patients by using the technique of rapid airway occlusion during constant flow inflation.\(^28,29,31\) The maximum resistance (Rmax) and minimum resistance (Rmin) of the total respiratory system, lung, or chest wall can be computed by dividing \([P\text{peak} - P\text{plat}]\) and \([P\text{peak} - \text{Pinit}]\) from the Paw, \(P_l\), and Pes tracings, respectively, by the flow immediately preceding the occlusion. The additional resistance (\(\Delta R\)) of the respiratory system, lung, or chest wall can be calculated as Rmax – Rmin for the respiratory system, lung, or chest wall, respectively. Rmin is considered to reflect ohmic airway resistance, while \(\Delta R\) reflects both the viscoelastic properties (stress relaxation) and time-constant inhomogeneities within the respiratory tissues (\(pendelluft\)).\(^28\)

In patients who subsequently underwent a trial of spontaneous breathing, patients who went on to fail a weaning trial and those who were successfully weaned did not have different total Rmax values.\(^29\) In both groups, the increased resistance originated almost totally in the lungs, with minimal contribution from the chest wall. In both groups, pulmonary flow resistance was mainly due to Rmin, which reflects ohmic airway resistance. \(\Delta R\) of the lung was not different in the two patient groups, but the value in the failure group was two times higher than in normal subjects, suggesting increased dynamic dissipations caused by time-constant inhomogeneity within the lungs.

Measurements of airway resistance are helpful in assessing the response of patients to bronchodilator therapy. In a study of ventilator-dependent patients with COPD, Dhand et al.\(^38\) showed that a significant decrease in airway resistance occurred after 4 puffs of bronchodilator were given, with no additional effect after the addition of 8 and 16 puffs (cumulative doses of 12 and 28 puffs.) In a separate group of patients with COPD, the bronchodilator effect of 4 puffs was sustained for at least 60 min.\(^39\)
Intrinsic PEEP

The static recoil pressure of the respiratory system at end expiration may be elevated in patients receiving mechanical ventilation. This positive recoil pressure, or intrinsic PEEP (static PEEPi), can be quantified in relaxed patients by using an end-expiratory hold maneuver on a mechanical ventilator immediately before the onset of the next breath.

PEEPi poses a significant inspiratory threshold load that has to be fully counterbalanced by increasing inspiratory muscle effort in order to generate a negative pressure in the central airway and trigger the ventilator. Thus, PEEPi adds to the triggering pressure such that the total inspiratory effort needed to trigger the ventilator is the set trigger sensitivity plus the level of PEEPi. This is one of the factors that may account for the not infrequent observation of a patient who is unable to trigger a ventilator despite obvious respiratory effort.

In a recent study of ventilator-dependent patients, Leung et al observed that ineffective triggering occurred with all assisted modes of mechanical ventilation. These ineffective efforts were significantly related to resistance ($r = 0.85$), elastance ($r = -0.61$), and static PEEPi ($r = 0.77$) (Fig 5). Moreover, the breaths preceding nontriggering efforts had shorter respiratory cycle times and expiratory time and higher PEEPi than breaths preceding triggered efforts. These findings suggest that ineffective triggering did not result from a decrease in the magnitude of effort, but rather from inspiratory efforts that were premature and insufficient to overcome the elevated elastic recoil pressure associated with dynamic hyperinflation.

In a spontaneously breathing patient, an esophageal balloon catheter system can be used to measure PEEPi during unoccluded breathing (dynamic PEEPi). This is achieved by calculating the negative deflection in esophageal pressure from the start of inspiratory effort to the onset of inspiratory flow. To obtain valid measurements, both the inspiratory and expiratory muscles need to be relaxed at end expiration. Two methods have been proposed to distinguish between the contribution of elastic recoil and expiratory muscle activity to PEEPi, with the latter being estimated from measurement of either

![Figure 5](image_url)

**Figure 5.** Relationship between wasted effort (quantitated as wasted PTP) and resistance, elastance, and PEEPi in 11 ventilator-dependent patients. Wasted PTP significantly correlated with resistance, elastance, and PEEPi. On multiple linear regression analysis, 93% of the variance in wasted PTP among patients could be explained by these three variables. Based on data from Leung et al.
the increase in gastric pressure (Pgas) over the course of expiration or the decrease in Pgas at the onset of the next expiration. In a recent report, Parthasarathy et al compared the accuracy of these two approaches in healthy volunteers in whom airflow limitation was induced with a Starling resistor. The results indicate that expiratory rise in Pgas correlated better with transversus abdominus electromyographic activity using needle electrodes (r = 0.7 to 0.95) than did early inspiratory decrease in Pgas (r = 0.04 to 0.53).

Work of Breathing

The mechanical WOB can be calculated by measuring the generation of intrathoracic pressure due to contraction of the respiratory muscles (or a ventilator substituting for them) and the displacement of gas volume. Coussa et al found that inspiratory work was approximately twofold greater in patients with COPD receiving controlled mechanical ventilation than in healthy control subjects, and the difference between the two groups was almost completely explained by PEEPi. Likewise, in patients with COPD receiving PSV, PEEPi accounted for 63% of the total amount of patient effort. A number of investigators have examined the usefulness of respiratory work measurements in predicting the outcome of a trial of weaning from mechanical ventilation. These studies show that patients can tolerate only a very small fraction of the maximum possible workload. Furthermore, WOB appeared to be higher in ventilator-dependent patients compared with ventilator-independent patients. Unfortunately, the predictive value of respiratory work as an index of weaning outcome remains to be determined.

Pressure-Time Product

A significant limitation of measurements of respiratory work is that they underestimate energy expenditure during isometric contractions. To overcome this problem, many investigators have measured pressure-time product (PTP) during mechanical ventilation. This is calculated as the time integral of the difference between esophageal pressure (Pes) measured during assisted breathing and the recoil pressure of the chest wall measured during passive ventilation with VT and flow settings that are identical to the assisted breaths. While this can be achieved satisfactorily during assist-control ventilation and intermittent mandatory ventilation, a problem arises during PSV because lung volume and inspiratory flow vary from breath to breath in this mode. To overcome this problem, a modified approach in the calculation of PTP has been described. First, an estimated recoil pressure of the chest wall is quantitated on a breath-by-breath basis by multiplying chest wall elastance (measured during passive ventilation) by lung volume. Then PTP is calculated as the time integral of the difference between the Pes tracing and the recoil pressure of the chest wall (Fig 6).

An element of uncertainty exists with the measurement of PTP, however, because the rapid decrease in Pes before the onset of inspiratory flow may result from inspiratory muscle activity needed to overcome the threshold imposed by dynamic hyperinflation and/or cessation of expiratory muscle activity. To deal with this issue, an upper- and lower-bound inspiratory PTP can be calculated that includes the entire possible range of muscular activity (Fig 6). To calculate upper-bound inspiratory PTP, the estimated elastic recoil pressure of the chest wall was set equal to Pes at the onset of the rapid decrease in Pes. To calculate lower-bound inspiratory PTP, the estimated elastic recoil pressure of the chest wall is set equal to Pes at the onset of inspiratory flow.

In patients with COPD, a marked and progressive decrease in upper-bound PTP was observed during graded levels of PSV, but the response among

![Figure 6. Pes (continuous line) and estimated recoil pressure of the chest wall (Pescw, interrupted line) tracings in a patient receiving PSV. Top, A, pressure tracings have been superimposed so that Pescw is equal to Pes at the onset of the first inspiratory effort, and the integrated difference (hatched area) represents upper-bound inspiratory PTP. Bottom, B, pressure tracings have been superimposed so that Pescw is equal to Pes at the first moment of transition from expiratory to inspiratory flow, and the integrated difference (hatched area) represents lower-bound inspiratory PTP. Reprinted with permission from Jubran et al.](image-url)
patients was quite variable, with a coefficient of variation of up to 96%. Evidence of expiratory effort, quantitated by an expiratory PTP, was seen in many patients, and this increased as PSV was increased. Moreover, several patients displayed expiratory muscle activation during late inflation, indicating that the patient was fighting the ventilator (Fig 7). This was more common in patients who had elevated time constants and who required more time for inspiratory flow to fall to the threshold value required for termination of inspiratory assistance by the ventilator (25% of peak inspiratory flow).

Measurements of PTP were recently obtained in 17 ventilator-supported patients with COPD who failed a trial of spontaneous breathing and in 14 patients who tolerated such a trial and were extubated. At the onset of the trial, upper-bound PTP in the failure and success groups did not differ. Throughout the course of the trial, upper-bound PTP was higher in the weaning failure group (Fig 8). A similar pattern of change was observed for lower-bound PTP.

**Figure 7.** Recordings of flow, Paw, and transversus abdominis electromyogram (EMG) in a patient with COPD receiving pressure support of 20 cm H2O. The onset of expiratory muscle activity (vertical line) occurred when mechanical inflation was only partly completed. Reprinted with permission from Farthasarathy et al.

**Figure 8.** Measurements of upper-bound (UB) and lower-bound (LB) PTP/min in 17 ventilator-supported patients with COPD who failed a trial of weaning and 14 patients who tolerated the trial and were extubated. Between the onset and end of a trial of spontaneous breathing, both indexes of PTP increased in the failure group (p < 0.0001 in both instances) and in the success group (p < 0.006 in both instances). Over the course of the trial, the failure group had higher values of both upper-bound PTP/min (p < 0.006) and lower-bound PTP/min (p < 0.02) than the success group. Bars represent ±1 SEM). Reprinted with permission from Jubran and Tobin.

**Conclusions**

Several devices can be used to monitor a patient’s gas exchange function, respiratory neuromuscular capacity, respiratory mechanics, and breathing effort during mechanical ventilation. Use of the derived information permits the physician to better tailor ventilator settings to an individual patient’s requirements with the promise of enhancing patient comfort. In addition, such measurements are helpful in characterizing the pathophysiology of a patient’s respiratory disorder, minimizing the risk of ventilator-induced complications, and determining the patient’s readiness for the discontinuation of ventilator support.

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