

# Advances in Respiratory Monitoring During Mechanical Ventilation\*

Amal Jubran, MD FCCP

**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. (CHEST 1999; 116:1416–1425)**

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

**Abbreviations:** ABG = arterial blood gas;  $\Delta R$  = additional resistance;  $E_{dyn,L}$  = dynamic elastance of the lung;  $E_{dyn,rs}$  = dynamic elastance of the respiratory system;  $E_{st,rs}$  = static elastance of the respiratory system;  $f$  = respiratory frequency; Hb = reduced hemoglobin;  $O_2Hb$  = oxyhemoglobin;  $P_{0.1}$  = mouth occlusion pressure at 0.1 s after onset of inspiratory effort;  $P_{aw}$  = airway pressure; PEEP = positive end-expiratory pressure; PEEPi = intrinsic positive end-expiratory pressure;  $P_{es}$  = esophageal pressure;  $P_{ETCO_2}$  = end-tidal  $PCO_2$  concentration;  $P_{ga}$  = gastric pressure;  $P_{init}$  = initial pressure;  $P_1$  = transpulmonary pressure;  $P_{peak}$  = peak pressure;  $P_{plat}$  = plateau pressure; PSV = pressure support ventilation; PTP = pressure-time product;  $R_{max}$  = maximum resistance;  $R_{min}$  = minimum resistance;  $SaO_2$  = arterial oxygen saturation;  $SpO_2$  = pulse oximeter estimate of arterial oxygen saturation;  $V_T$  = 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%.<sup>1</sup> In patients admitted to a general medical service, Bowton et al<sup>2</sup> found that patients who experienced hypoxemia ( $O_2$  saturation  $< 90\%$  for  $\geq 5$  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.

\*From the Division of Pulmonary and Critical Care Medicine, Edward Hines Jr., Veterans Affairs Hospital and Loyola University of Chicago Stritch School of Medicine, Hines, IL  
Correspondence to: Amal Jubran, MD, Division of Pulmonary and Critical Care Medicine, Edward Hines, Jr., Veterans Affairs Hospital, Route 111N, Hines, IL 60141

## 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 ( $O_2Hb$ ) and reduced hemoglobin (Hb) have different absorption spectra.<sup>1</sup> 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  $O_2Hb$  and Hb have different absorption spectra at these particular wavelengths. In the red region,  $O_2Hb$  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 ( $SaO_2$ ) 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 saturation ( $SpO_2$ )<sup>3</sup> (Fig 1).

**Accuracy:** The accuracy of commercially available oximeters varies widely, probably because of the different algorithms employed in signal processing.<sup>1</sup> Oximeters commonly have a mean difference (bias) of  $< 2\%$  and an SD (precision) of  $< 3\%$  when  $SaO_2$  is  $\geq 90\%$ .<sup>4</sup> Accuracy of pulse oximeters deteriorates when  $SaO_2$  falls to 80% or less. In a study of 54 ventilator-dependent patients, the bias  $\pm$  precision of oximetry was  $1.7 \pm 1.2\%$  for  $SaO_2$  values  $> 90\%$ ,

SaO <sub>2</sub>	660 nm (RED)	940 nm (IR)	R IR
0%			~3.4
85%			1.0
100%			.43

FIGURE 1. Red (R) and infrared (IR) scaled alternating current signals at (SaO<sub>2</sub>) of 0, 85, and 100%. The numeric value of the red-to-infrared ratio (R/IR) can be easily converted to SaO<sub>2</sub>. Reprinted with permission from Wukitisch et al.<sup>3</sup>

and it increased to  $5.1 \pm 2.7\%$  when SaO<sub>2</sub> was  $\leq 90\%$ .<sup>5</sup>

**Limitations:** Pulse oximeters employ only two wavelengths of light, and thus can distinguish only two substances, Hb and O<sub>2</sub>Hb. Elevated carboxyhemoglobin and methemoglobin levels can cause inaccurate oximetry readings.<sup>1</sup> Anemia does not appear to affect the accuracy of pulse oximetry: in nonhypoxic patients with acute anemia (mean Hb,  $5.2 \pm 0.3$  [SEM] g/dL), pulse oximetry was accurate in measuring O<sub>2</sub> saturation with a bias of only 0.53%.<sup>6</sup> Moreover, in patients with sickle cell anemia who presented with acute vaso-occlusive crisis, Ortiz et al<sup>7</sup> found that pulse oximetry overestimated SaO<sub>2</sub> by an average of 3.4%; the error of SpO<sub>2</sub> 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.<sup>1,8</sup> In a recent prospective study in an ICU setting, SpO<sub>2</sub> signals accounted for almost half of a total of 2,525 false alarms<sup>9</sup> (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.<sup>10</sup> 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 dur-

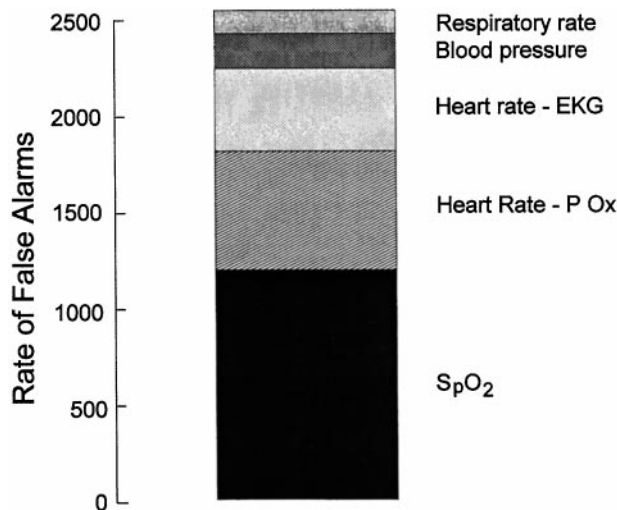


FIGURE 2. Number of false alarms for devices used to monitor respiratory rate, mean systemic BP from an arterial catheter, heart rate from an ECG, heart rate measured by pulse oximetry (P Ox), and SpO<sub>2</sub>. Forty-five percent of all false alarms were from the SpO<sub>2</sub> signal. Based on data from Tsien and Fackler.<sup>9</sup>

ing 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 SpO<sub>2</sub> data) than did the Nellcor N-200 and Nellcor N-3000 oximeters (Nellcor Puritan-Bennett Corp; Pleasanton, CA).<sup>11</sup> 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).<sup>12</sup>

**Clinical Applications:** Moller et al<sup>13</sup> 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 SpO<sub>2</sub> < 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 (FIO<sub>2</sub>) in ventilator-dependent patients, although the appropriate SpO<sub>2</sub> target depends on a patient's pigmentation.<sup>5</sup> In white patients, a SpO<sub>2</sub> 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 SpO<sub>2</sub> value of 95% avoids hypoxemia in black patients, some will have PaO<sub>2</sub> values as high as 198 mm Hg; if such patients

receive a high  $\text{FIO}_2$  to achieve the  $\text{SpO}_2$  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.<sup>14</sup> The relationship between  $\text{SpO}_2$  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  $\text{SpO}_2$  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.<sup>15</sup> Inman et al<sup>16</sup> 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.<sup>1</sup> 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  $\text{PCO}_2$  concentration ( $\text{PETCO}_2$ ) is the value of exhaled gas taken at the plateau of the  $\text{CO}_2$  waveform. In healthy subjects,  $\text{PETCO}_2$  is usually 1 mm Hg (range, up to 5 mm Hg) less than  $\text{PaCO}_2$ .<sup>17</sup> Consequently,  $\text{PETCO}_2$  can be employed as a continuous, indirect measure of  $\text{PaCO}_2$ . Hoffman et al<sup>18</sup> obtained simultaneous measurements of  $\text{PETCO}_2$  and  $\text{PaCO}_2$  in 20 intubated patients with respiratory failure 5 to 10 min after altering settings on the mechanical ventilator. The correlation between  $\text{PETCO}_2$  and  $\text{PaCO}_2$  was good ( $r = 0.78$ ). However, the correlation between changes in  $\text{PETCO}_2$  and changes in  $\text{PaCO}_2$  from baseline was considerably

weaker ( $r = 0.58$ ). Importantly, four patients demonstrated a trend in  $\text{PETCO}_2$  opposite in direction to the trend in the  $\text{PaCO}_2$ . Likewise, Hess et al<sup>19</sup> found that the change in  $\text{PETCO}_2$  incorrectly indicated the direction of change in  $\text{PaCO}_2$  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 ( $\text{P}_{0.1}$ ) provides a measure of respiratory drive. In ventilator-dependent patients,  $\text{P}_{0.1}$  has been shown to correlate significantly with work of breathing (WOB) during pressure-support ventilation (PSV;  $r = 0.87$ ).<sup>20</sup> Several studies have indicated that an elevated  $\text{P}_{0.1}$  predicted weaning failure, but the threshold separating success from failure differed among the studies.<sup>21,22</sup>

### Breathing Pattern

Minute ventilation should be partitioned into tidal volume ( $\text{VT}$ ) and respiratory frequency ( $f$ ). In healthy subjects,  $f$  is approximately 17 breaths/min and  $\text{VT}$  is approximately 400 mL.<sup>17</sup> 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,<sup>23</sup> and this can be quantitated in terms of the  $f/\text{VT}$  ratio; a value > 100 breaths/min/L suggests that a trial of weaning is unlikely to be successful.<sup>24</sup> 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  $f/\text{VT}$  and the tension-time index, a crude index of impending respiratory muscle fatigue. To serve as a compensatory strategy to avoid fatigue,  $f/\text{VT}$  should have a negative correlation with tension-time index, whereas  $r$  was found to be 0.08.<sup>25</sup>

### 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.<sup>26,27</sup>

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<sub>2</sub>O are thought to predict weaning success, while values that are less negative than  $-20$  cm H<sub>2</sub>O are predictive of weaning failure. However, these criteria are frequently falsely positive and falsely negative.<sup>24</sup>

### 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.<sup>28</sup> Rapid airway occlusion at the end of a passive inflation produces an immediate drop in both airway pressure ( $P_{aw}$ ) and transpulmonary pressure ( $P_l$ ) from a peak value ( $P_{peak}$ ) to a lower initial value ( $P_{init}$ ) followed by a gradual decrease until a plateau ( $P_{plat}$ ) is achieved after 3 to 5 s<sup>29,30</sup> (Fig 3).  $P_{init}$  is measured by back extrapolation of the slope of the latter part of the pressure tracing to the time of airway occlusion.<sup>28</sup>  $P_{plat}$  on the  $P_{aw}$ ,  $P_l$ , and pleural pressure ( $P_{es}$ ) 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<sup>31</sup>:

$$Est_{rs} = (P_{plat} - PEEP_i) / V_T$$

where  $P_{plat}$  is plateau pressure obtained after occluding the airway,  $PEEP_i$  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  $[P_{plat} - PEEP_i]$  by  $V_T$  on the  $P_l$  and  $P_{es}$  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.<sup>30</sup> Static lung elastance is higher in patients with pul-

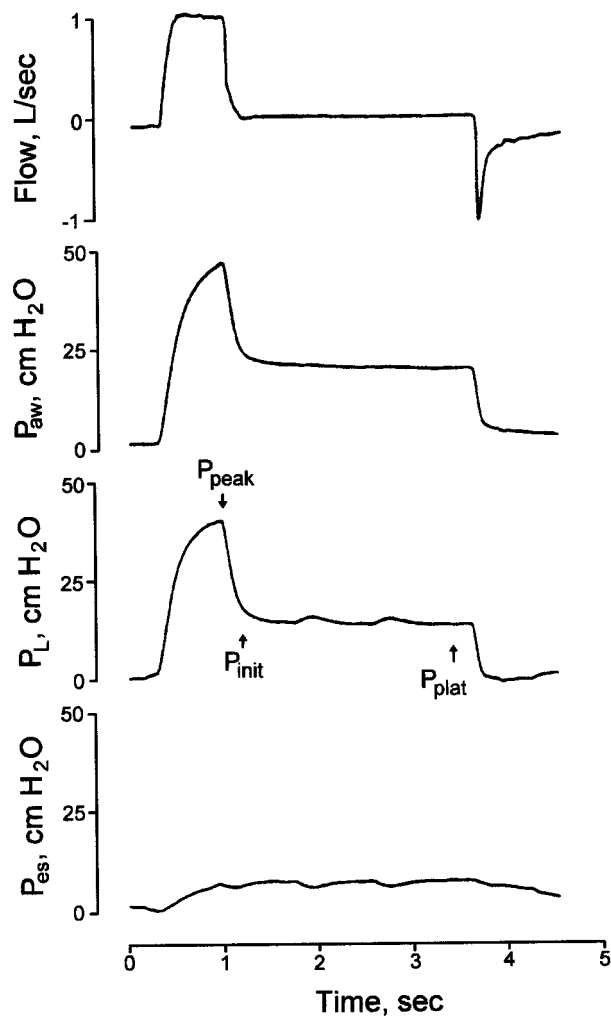


FIGURE 3. Flow (inspiration upwards),  $P_{aw}$ ,  $P_l$ , and  $P_{es}$  tracings in a representative patient during passive ventilation. An end-inspiratory occlusion produced a rapid decline in both  $P_{aw}$  and  $P_l$  from  $P_{peak}$  to a lower  $P_{init}$ , followed by gradual decrease to  $P_{plat}$ . Reprinted with permission from Jubran and Tobin.<sup>29</sup>

monary edema than in patients with COPD, whereas static chest wall elastance was similar in both patient groups.<sup>30</sup>

#### 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  $[\text{peak } P_{aw} - PEEP]$ . This index is not a measure of true thoracic compliance because peak  $P_{aw}$  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  $P_{aw}$  at points of zero flow by the delivered  $V_T$ .<sup>31</sup> Accordingly,  $Edyn_{rs}$  can be computed according to the formula:

$$E_{dyn,rs} = P_{init} - PEEP_i/V_T$$

$E_{dyn,rs}$  can be partitioned into its lung ( $E_{dyn,L}$ ) and chest wall components by dividing  $[P_{init} - PEEP_i]$  on  $P_1$  and  $P_{es}$  tracings, respectively (Fig 3). In a recent study,<sup>29</sup>  $E_{dyn,rs}$  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,  $E_{dyn,rs}$  was predominantly influenced by  $E_{dyn,L}$  because the values of chest wall dynamic elastance were normal.  $E_{dyn,L}$  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.<sup>32</sup> 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.<sup>33</sup> In a prospective, randomized study in 28 patients, Amato et al<sup>34</sup> compared an "open-lung approach"—consisting of a lower  $V_T$  ( $< 6$  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  $H_2O$ —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<sup>35</sup> (Fig 4). Two multicenter, randomized studies looking only at the effects of reducing  $V_T$  did not find any significant difference in mortality,<sup>36,37</sup> indirectly suggesting that the individual titration of PEEP in the Amato study<sup>34</sup> 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.<sup>28,29,31</sup> The maximum resistance ( $R_{max}$ ) and minimum re-

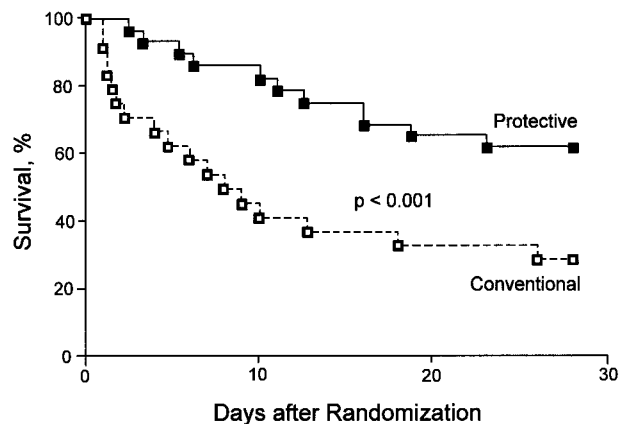


FIGURE 4. Actuarial 28-day survival among 53 patients with ARDS assigned to protective or conventional mechanical ventilation. After 28 days, 11 of 29 patients (38%) in the protective-ventilation group had died, compared with 17 of 24 (71%) in the conventional-ventilation group ( $p < 0.001$ ). Reprinted with permission from Amato et al.<sup>35</sup>

sistance ( $R_{min}$ ) of the total respiratory system, lung, or chest wall can be computed by dividing  $[P_{peak} - P_{plat}]$  and  $[P_{peak} - P_{init}]$  from the  $P_{aw}$ ,  $P_1$ , and  $P_{es}$  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  $R_{max} - R_{min}$  for the respiratory system, lung, or chest wall, respectively.  $R_{min}$  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*).<sup>28</sup>

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  $R_{max}$  values.<sup>29</sup> 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  $R_{min}$ , 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<sup>38</sup> 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.<sup>39</sup>

### Intrinsic PEEP

The static recoil pressure of the respiratory system at end expiration may be elevated in patients receiving mechanical ventilation.<sup>31</sup> This positive recoil pressure, or intrinsic PEEP (static PEEP<sub>i</sub>), 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.

PEEP<sub>i</sub> 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, PEEP<sub>i</sub> 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 PEEP<sub>i</sub>. 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.<sup>40,41</sup>

In a recent study of ventilator-dependent patients, Leung et al<sup>40</sup> observed that ineffective triggering occurred with all assisted modes of mechanical ventilation. These ineffective efforts were signifi-

cantly related to resistance ( $r = 0.85$ ), elastance ( $r = -0.61$ ), and static PEEP<sub>i</sub> ( $r = 0.77$ ) (Fig 5). Moreover, the breaths preceding nontriggering efforts had shorter respiratory cycle times and expiratory time and higher PEEP<sub>i</sub> 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 PEEP<sub>i</sub> during unoccluded breathing (dynamic PEEP<sub>i</sub>).<sup>42</sup> 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 PEEP<sub>i</sub>, with the latter being estimated from measurement of either

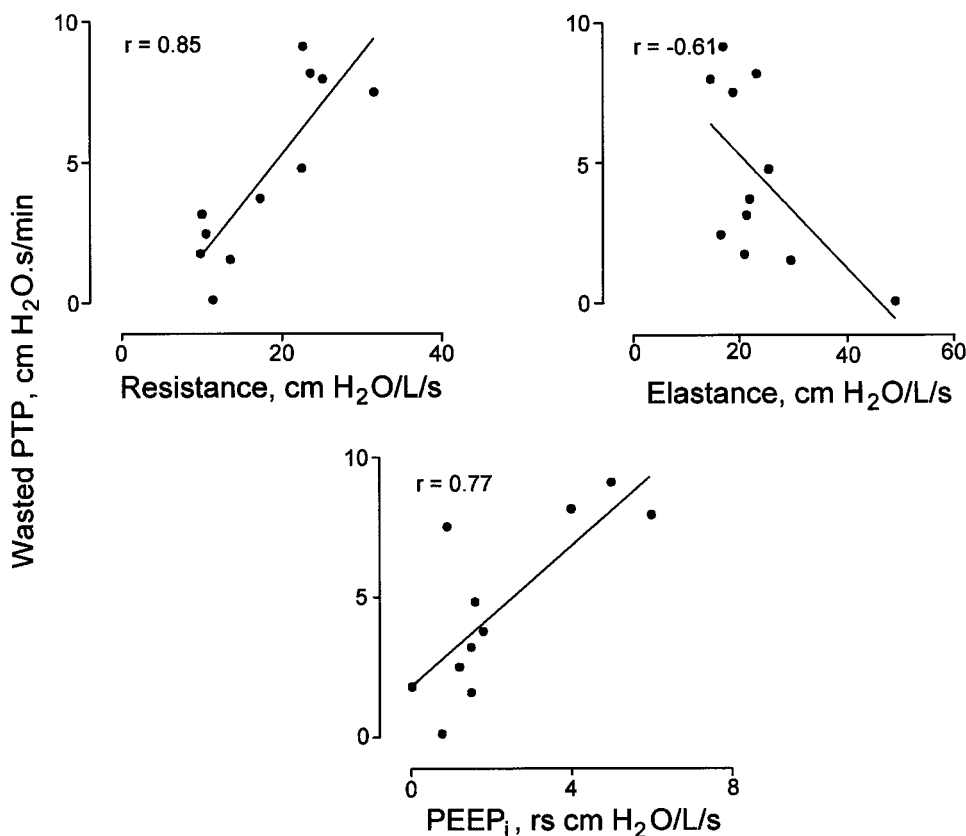


FIGURE 5. Relationship between wasted effort (quantitated as wasted PTP) and resistance, elastance, and PEEP<sub>i</sub> in 11 ventilator-dependent patients. Wasted PTP significantly correlated with resistance, elastance, and PEEP<sub>i</sub>. On multiple linear regression analysis, 93% of the variance in wasted PTP among patients could be explained by these three variables. Based on data from Leung et al.<sup>40</sup>

the increase in gastric pressure (Pga) over the course of expiration<sup>43</sup> or the decrease in Pga at the onset of the next expiration.<sup>44</sup> In a recent report, Parthasarathy et al<sup>45</sup> 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 Pga correlated better with transversus abdominus electromyographic activity using needle electrodes ( $r = 0.7$  to  $0.95$ ) than did early inspiratory decrease in Pga ( $r = 0.04$  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<sup>46</sup> 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.<sup>47</sup>

A number of investigators have examined the usefulness of respiratory work measurements in predicting the outcome of a trial of weaning from mechanical ventilation.<sup>48</sup> 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.<sup>49,50</sup> 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 de-

scribed.<sup>47</sup> 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

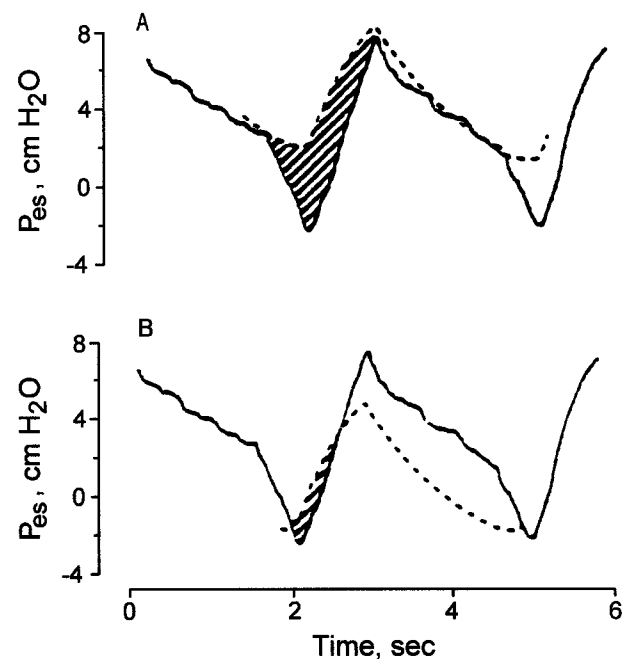


FIGURE 6. Pes (continuous line) and estimated recoil pressure of the chest wall (Pescw, interrupted line) tracings in a patient receiving PSV. *Top, A*, pressure tracings have been superimposed so that Pescw is equal to Pes at the onset of the first inspiratory effort, and the integrated difference (hatched 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.<sup>47</sup>

patients was quite variable, with a coefficient of variation of up to 96%.<sup>47</sup> 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).<sup>45,47</sup> 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.<sup>25</sup> 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.

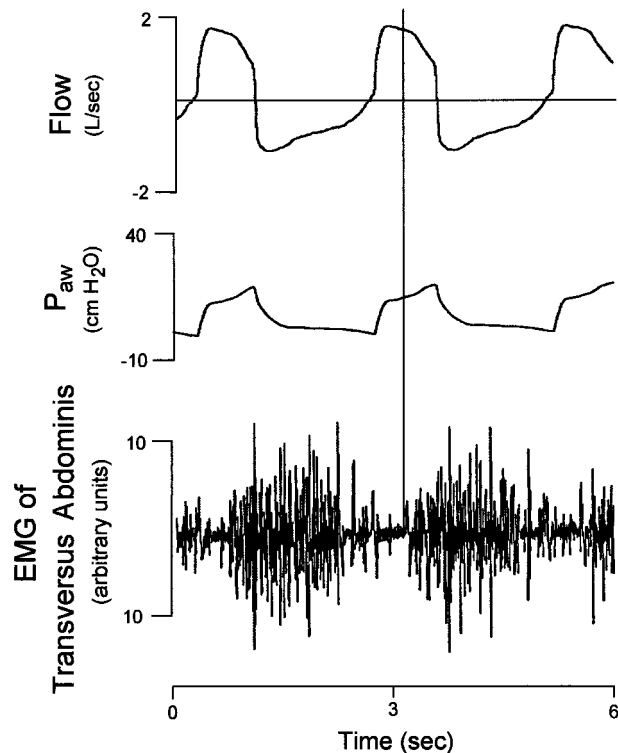


FIGURE 7. Recordings of flow,  $P_{aw}$ , and transversus abdominis electromyogram (EMG) in a patient with COPD receiving pressure support of 20 cm  $H_2O$ . The onset of expiratory muscle activity (vertical line) occurred when mechanical inflation was only partly completed. Reprinted with permission from Parthasarathy et al.<sup>45</sup>

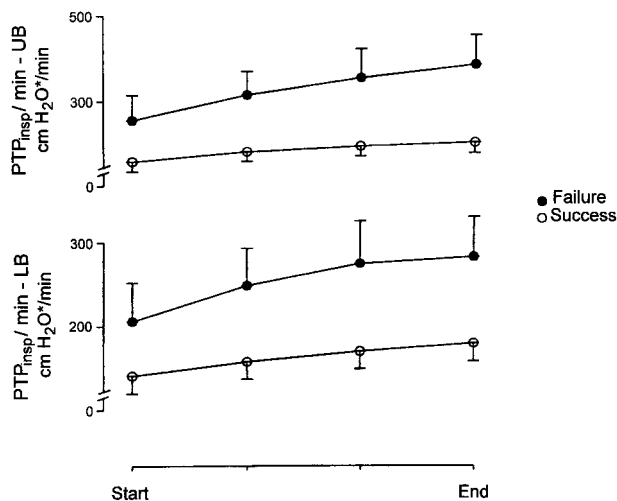


FIGURE 8. Measurements of upper-bound (UB) and lower-bound (LB) PTP/min in 17 ventilator-supported patients with COPD who failed a trial of weaning and 14 patients who tolerated the trial and were extubated. Between the onset and end of a trial of spontaneous breathing, both indexes of PTP increased 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  $\pm 1$  SEM). Reprinted with permission from Jubran and Tobin.<sup>25</sup>

## 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.

## REFERENCES

- 1 Jubran A. Pulse oximetry. In: Tobin MJ, ed. Principles and practice of intensive care monitoring. New York, NY: McGraw-Hill, 1998; 261-287
- 2 Bowton DL, Scuderi PE, Haponik EF. The incidence and effect on outcome of hypoxemia in hospitalized medical patients. *Am J Med* 1994; 97:38-46
- 3 Wukitisch MW, Peterson MT, Tobler DR, et al. Pulse oximetry: analysis of theory, technology, and practice. *J Clin Monit* 1988; 4:290-301
- 4 Webb RK, Ralston AC, Runciman WB. Potential errors in pulse oximetry: II. Effects of changes in saturation and signal quality. *Anesthesia* 1991; 96:207-212

- 5 Jubran A, Tobin MJ. Reliability of pulse oximetry in titrating supplemental oxygen therapy in ventilator-dependent patients. *Chest* 1990; 97:1420–1425
- 6 Jay GD, Hughes L, Renzi FP. Pulse oximetry is accurate in acute anemia from hemorrhage. *Ann Emerg Med* 1994; 24:32–35
- 7 Ortiz FO, Aldrich TK, Nagel RL, et al. Accuracy of pulse oximetry in sickle cell disease. *Am J Respir Crit Care Med* 1999; 159:447–451
- 8 Rheineck-Leyssius AT, Kalkman CJ. Influence of pulse oximeter lower alarm limit on the incidence of hypoxaemia in the recovery room. *Br J Anaesth* 1997; 79:460–464
- 9 Tsien CL, Fackler JC. Poor prognosis for existing monitors in the intensive care unit. *Crit Care Med* 1997; 25:614–619
- 10 Pollard V, Prough DS. Signal extraction technology: a better mousetrap [editorial]? *Anesth Analg* 1996; 83:213–214
- 11 Barker SJ, Shah NK. Effects of motion on the performance of pulse oximeters in volunteers. *Anesthesiology* 1997; 86:101–108
- 12 Dumas C, Wahr JA, Tremper KK. Clinical evaluation of a prototype motion artifact resistant pulse oximeter in the recovery room. *Anesth Analg* 1996; 83:269–272
- 13 Moller JT, Pedersen T, Rasmussen LS, et al. Randomized evaluation of pulse oximetry in 20,802 patients: I. Design, demography, pulse oximetry failure rate and overall complication rate. *Anesthesiology* 1993; 78:436–444
- 14 Mower WR, Sachs C, Nicklin EL, Safa P, Baraff LJ. A comparison of pulse oximetry and respiratory rate in patient screening. *Respir Med* 1996; 90:593–599
- 15 Le Bourdelles G, Estagnasie P, Lenoir F, et al. Use of a pulse oximeter in an adult emergency department: impact on the number of arterial blood gas analyses ordered. *Chest* 1998; 113:1042–1047
- 16 Inman KJ, Sibbald WJ, Rutledge FS, et al. Does implementing pulse oximetry in a critical care unit result in substantial arterial blood gas savings? *Chest* 1993; 104:542–546
- 17 Jubran A, Tobin MJ. Monitoring during mechanical ventilation. *Clin Chest Med* 1996; 17:453–473
- 18 Hoffman RA, Ershowsky P, Krieger BP. Determination of auto-PEEP during spontaneous and controlled ventilation by monitoring changes in end-expiratory thoracic gas volume. *Chest* 1989; 96:613–616
- 19 Hess D, Schlottag A, Levin B, et al. An evaluation of the usefulness of end-tidal PCO<sub>2</sub> to aid weaning from mechanical ventilation following cardiac surgery. *Respir Care* 1991; 36:837–843
- 20 Alberti A, Gallo F, Fongaro A, et al. P<sub>0.1</sub> is a useful parameter in setting the level of pressure support ventilation. *Intensive Care Med* 1995; 21:547–553
- 21 Capdevila XJ, Perrigault PF, Perey PJ, et al. Occlusion pressure and its ratio to maximum inspiratory pressure are useful predictors for successful extubation following T-piece weaning trial. *Chest* 1995; 108:482–489
- 22 Sassoon CSH, Mahutte CK. Airway occlusion pressure and breathing pattern as predictors of weaning outcome. *Am Rev Respir Dis* 1993; 148:860–866
- 23 Tobin MJ, Guenther SM, Perez W, et al. The pattern of breathing during successful and unsuccessful trials of weaning from mechanical ventilation. *Am Rev Respir Dis* 1986; 134:1111–1118
- 24 Yang K, Tobin MJ. A prospective study of indexes predicting outcome of trials of weaning from mechanical ventilation. *N Engl J Med* 1991; 324:1445–1450
- 25 Jubran A, Tobin MJ. Pathophysiological basis of acute respiratory distress in patients who fail a trial of weaning from mechanical ventilation. *Am J Respir Crit Care Med* 1997; 155:906–915
- 26 Marini JJ, Smith TC, Lamb V. Estimation of inspiratory muscle strength in mechanically ventilated patients: the measurement of maximal inspiratory pressure. *J Crit Care* 1986; 1:32–38
- 27 Multz AS, Aldrich TK, Prezant DJ, et al. Maximal inspiratory pressure is not a reliable test of inspiratory muscle strength in mechanically ventilated patients. *Am Rev Respir Dis* 1990; 142:529–532
- 28 Bates JHT, Rossi A, Milic-Emili J. Analysis of the behavior of the respiratory system with constant inspiratory flow. *J Appl Physiol* 1985; 58:1840–1848
- 29 Jubran A, Tobin MJ. Passive mechanics of lung and chest wall in patients who failed and succeeded in trials of weaning. *Am J Respir Crit Care Med* 1997; 155:916–921
- 30 Polese G, Rossi A, Appendini L, et al. Partitioning of respiratory mechanics in mechanically ventilated patients. *J Appl Physiol* 1991; 71:2425–2433
- 31 Rossi A, Polese G, Milic-Emili J. Monitoring respiratory mechanics in ventilator-dependent patients. In: Tobin MJ, ed. *Principles and practice of intensive care monitoring*. New York, NY: McGraw-Hill, 1998; 553–596
- 32 Brochard L. Respiratory pressure-volume curves. In: Tobin MJ, ed. *Principles and practice of intensive care monitoring*. New York, NY: McGraw-Hill, 1998; 597–616
- 33 Matamis D, Lemaire F, Harf A, et al. Total respiratory pressure-volume curves in the adult respiratory distress syndrome. *Chest* 1984; 86:58–66
- 34 Amato MBP, Barbas CSV, Medeiros DM, et al. Beneficial effects of the “open lung approach” with low distending pressures in acute respiratory distress syndrome: a prospective randomized study on mechanical ventilation. *Am J Respir Crit Care Med* 1995; 152:1835–1846
- 35 Amato MBP, Barbas CSV, Medeiros D, et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. *N Engl J Med* 1998; 338:347–354
- 36 Brochard L, Roudot-Thoraval F, Roupie E, et al. Tidal volume reduction for prevention of ventilator-induced lung injury in acute respiratory distress syndrome. *Am J Respir Crit Care Med* 1998; 158:1831–1838
- 37 Stewart TEM, Meade O, Cook DJ, et al. Evaluation of a ventilation strategy to prevent barotrauma in patients at high risk for acute respiratory distress syndrome. *N Engl J Med* 1998; 338:355–361
- 38 Dhand R, Jubran A, Tobin MJ. Efficacy of bronchodilator delivered by metered-dose inhaler in ventilator-supported patients with COPD. *Am J Respir Crit Care Med* 1995; 152:129–136
- 39 Dhand R, Duarte AG, Jubran A, et al. Dose response to bronchodilator delivered by metered-dose inhaler in ventilator-supported patients. *Am J Respir Crit Care Med* 1996; 154:388–393
- 40 Leung P, Jubran A, Tobin MJ. Comparison of assisted ventilator modes on triggering, patient effort, and dyspnea. *Am J Respir Crit Care Med* 1997; 155:1940–1948
- 41 Nava S, Bruschi C, Rubini F, et al. Respiratory response and inspiratory effort during pressure support ventilation in COPD patients. *Intensive Care Med* 1995; 21:871–879
- 42 Haluszka J, Chartrand DA, Grassino AE, et al. Intrinsic PEEP and arterial PCO<sub>2</sub> in stable patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1990; 141:1194–1197
- 43 Lessard MR, Lofaso F, Brochard L. Expiratory muscle activity increases intrinsic positive end-expiratory pressure independently of dynamic hyperinflation in mechanically ventilated patients. *Am J Respir Crit Care Med* 1995; 151:562–569
- 44 Appendini L, Patessio A, Zanaboni S, et al. Physiologic effects of positive end-expiratory pressure and mask pressure support during exacerbations of chronic obstructive pulmonary

- disease. *Am J Respir Crit Care Med* 1994; 149:1069–1076
- 45 Parthasarathy S, Jubran A, Tobin MJ. Cycling of inspiratory and expiratory muscle groups with the ventilator in airflow limitation. *Am J Respir Crit Care Med* 1998; 158:1471–1478
- 46 Coussa ML, Guerin C, Eissa NT, et al. Partitioning of work of breathing in mechanically ventilated COPD patients. *J Appl Physiol* 1993; 75:1711–1719
- 47 Jubran A, Van de Graaff WB, Tobin MJ. Variability of patient-ventilator interaction with pressure-support ventilation in patients with COPD. *Am J Respir Crit Care Med* 1995; 152:129–136
- 48 Tobin MJ, Van de Graaff WB. Monitoring of lung mechanics and work of breathing. In: Tobin MJ, ed. *Principles and practice of mechanical ventilation*. New York, NY: McGraw-Hill, 1994; 967–1003
- 49 Giuliani R, Mascia L, Recchia F, et al. Patient-ventilator interaction during synchronized intermittent mandatory ventilation: effects of flow triggering. *Am J Respir Crit Care Med* 1995; 151:1–9
- 50 Imsand C, Feihl F, Perret MD, et al. Regulation of inspiratory neuromuscular output during synchronized intermittent mandatory ventilation. *Anesthesiology* 1994; 80:13–22