Breathing Frequency and Pattern Are Poor Predictors of Work of Breathing in Patients Receiving Pressure Support Ventilation*

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Objective: To evaluate the relationships between directly measured work of breathing (WOB) and variables of the breathing pattern commonly used at the bedside to infer WOB for intubated, spontaneously breathing patients treated with pressure support ventilation (PSV).

Design: In vivo measurements of the WOB were obtained on a consecutive series of adults. Breathing frequency (f), tidal volume (Vt), the index of rapid, shallow breathing (ΔV/T), the duration of respiratory muscle contraction expressed as the ratio of inspiratory time over total respiratory cycle time (Ti/Ttotal), and a breathing pattern score (applied to approximately 50% of the patients) which ranks f, Vt, sternocleidomastoid muscle activity, substernal retraction, and abdominal paradox on a scale were variables of the breathing pattern were also measured. The greater the breathing pattern score, the lower the WOB and vice versa.

Setting: Surgical ICUs in two university teaching hospitals.

Patients: Sixty-seven adults (42 men and 25 women, aged 20 to 78 years) who had acute respiratory failure from various etiologies were studied. All patients were breathing spontaneously receiving continuous positive airway pressure and PSV.

Interventions: Intraesophageal pressure (indirect measurement of intrapleural pressure) was measured with an esophageal balloon integrated into a nasogastric tube. Vt was obtained by positioning a flow sensor between the "Y" piece of breathing circuit and the endotracheal tube. Data from these measurements were directed to a bedside respiratory monitor (BiCore; Allied Healthcare Products; Riverside, Calif) that calculates WOB using the Campbell diagram. Patients received PSV at levels deemed reasonable to unload the respiratory muscles. All measurements were obtained after 15 to 20 min at each level of PSV, averaged over 1 min, and then variables of the breathing pattern were regressed with directly measured values for WOB.

Results: All breathing pattern variables poorly predicted WOB as evidenced by the low values for the coefficients of determination (r²). Breathing frequency correlated positively with WOB (r=0.47, p<0.001) and predicted or explained only 22% (r²=.22) of the variance in WOB. Vt correlated negatively and ΔV/T and Ti/Ttotal each correlated positively with WOB. However, these variables predicted only 20 to 27% of the variance in WOB. The breathing pattern score correlated negatively with WOB and predicted only 43% of the variance in WOB. A prediction model taking all variables into consideration using multiple regression analysis predicted only 50% of the variance in WOB; thus, it too was a poor to moderate predictor of WOB.

Conclusion: Our data reveal that WOB should be measured directly because variables of the breathing pattern commonly used at the bedside appear to be inaccurate and misleading inferences of the WOB. The clinical implication of these findings involves the traditional and empirical practice of titrating PSV based on the breathing pattern. We do not imply that the patient's breathing pattern should be ignored, nor undermine its importance, for it provides useful diagnostic information. It appears, however, that relying primarily on the breathing pattern alone does not provide enough information to accurately assess the respiratory muscle workload. Using the breathing pattern as the primary guideline for selecting a level of PSV may result in inappropriate respiratory muscle workloads. A more comprehensive strategy is to employ WOB measurements and the breathing pattern in a complementary manner when titrating PSV in critically ill patients. (CHEST 1995; 108:1338-44)

CPAP=continuous positive airway pressure; f=breathing frequency; ΔV/T=ratio of breathing frequency over tidal volume; PSV=pressure support ventilation; Ti/Ttotal=ratio of inspiratory time over total respiratory cycle time; Vt=tidal volume; WOB=work of breathing

Key words: breathing frequency; breathing pattern; chest wall compliance; esophageal pressure; mechanical ventilation; pressure support ventilation; respiratory failure; respiratory monitor; respiratory muscle loading; work of breathing

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Spontaneous breathing frequency (f) and a patient’s breathing pattern in general are commonly used to infer the work of breathing (WOB) or respiratory muscle afterload during ventilatory support. A normal or tolerable WOB is inferred when f is approximately 15 to 25 breaths/min for adults and accessory respiratory muscles, such as the sternocleidomastoid muscles, are not contracting. It is recommended that f should be taken into consideration when titrating pressure support ventilation (PSV) to unload the respiratory muscles and decrease the WOB of patients with increased workloads. For example, if the f for an adult were approximately 35 to 40 breaths/min and the sternocleidomastoid muscles were contracting at a specific level of PSV, then it would be inferred that WOB is abnormally increased and that the level of PSV should be increased. Following an increase in PSV, if the f decreases to approximately 15 breaths/min and sternocleidomastoid muscle activity is absent, then it would be inferred that the new level of PSV is appropriate to unload the respiratory muscles and that the WOB is in a more tolerable range. Observing the breathing pattern and inferring whether the patient’s WOB is appropriate is the conventionally used approach for selecting a clinically acceptable level of PSV. Ostensibly, the approach is subjective and may be considered “educated guesswork.”

The precision of using f and the breathing pattern to infer WOB when applying PSV is in question. In several reports, f did not correlate with WOB during weaning from ventilatory support. For example, Brochard et al reported that, following application of decremental levels of PSV, f was fairly constant at about 23 breaths/min, while WOB ranged from 0.55 to 1.10 J/L in patients with respiratory failure. Nathan et al observed f to be fairly constant at about 22 breaths/min, while WOB ranged from 0.31 to 1.04 J/L in patients receiving PSV prior to extubation. Petros et al showed that while patients breathed spontaneously during PSV, with continuous positive airway pressure (CPAP) or with a T-piece, WOB ranged from 0.87 to 1.78 J/L, while f was fairly constant at approximately 22 breaths/min. In another study, Tokioka et al reported that f was fairly constant at 25 to 30 breaths/min, while WOB ranged from 0.2 to 0.75 J/L following decremental levels of PSV.

A need exists to evaluate the accuracy of predicting WOB based on f and related assessments of the breathing pattern in critically ill patients who are receiving ventilatory support. We hypothesized that objective, real-time measurement of a patient’s total WOB (physiologic plus imposed work) is a more accurate, and thus, appropriate, method of assessing the respiratory muscle workload. To test this hypothesis, we evaluated the relationships between directly measured WOB and variables of the breathing pattern commonly used at the bedside to infer WOB for intubated, spontaneously breathing patients receiving PSV who were diagnosed as having respiratory failure.

### Materials and Methods

Sixty-seven adults (42 men and 25 women) admitted to the surgical ICU who were diagnosed as having acute respiratory failure from various etiologies were studied after obtaining informed consent from the patient’s family. The study was approved by the Institutional Review Boards at Shands Hospital at the University of Florida Medical Center and at the Jackson Memorial Medical Center at the University of Miami. Patients in the study population were diagnosed as having moderate to severe forms of respiratory failure based on airway pressures, pulmonary mechanics, radiologic examination, and blood gas exchange. Causes of respiratory failure included sepsis, cirrhosis, pneumonia, congestive heart failure, pulmonary edema, ascites, cirrhositis, or abdominal and chest wall blunt trauma from either gunshot, stab wounds, or motor vehicle accidents. A few patients had COPD. Age and weight ranged from 20 to 77 years and 40 to 180 kg, respectively. All patients received PSV at levels deemed reasonable to unload the respiratory muscles and lesson the WOB based on observing the patients’ breathing. All patients had either an endotracheal or a tracheostomy tube in place and were breathing spontaneously receiving CPAP from 5 to 15 cm H2O. Endotracheal and tracheostomy tube sizes ranged from 6.5 to 10.0 mm internal diameter. Applied levels of CPAP were based on established criteria of applying a nontoxic fractional inspirated oxygen concentration (FiO2 of 0.5 or less) to achieve an arterial oxygen saturation of 97% or more.

Several types of mechanical ventilators were used (Hamilton Veolar; Reno, Nev; Puritan-Bennett 7200; Carlsbad, Calif; Bird 6400 ST; Palm Springs, Calif; and Siemens 900 C; Schaumberg, III). All patients were studied using the ventilator chosen by the medical and respiratory care personnel. The patient-trigger sensitivity setting for all ventilators was set at -2 cm H2O. Standard diameter corrugated tubing and heated wick-type humidifiers were used with all ventilators.

At all levels of PSV, the following breathing pattern variables (inferences of WOB) were measured: f; tidal volume (Vt); the index of rapid shallow breathing, expressed as the ratio of f over Vt (f/Vt); the duration of respiratory muscle contraction or the portion of the respiratory cycle when the inspiratory muscles are active, expressed as the ratio of inspiratory time over total respiratory cycle time (Ti/Ttot); and a breathing pattern score that we derived that ranks the following five variables each on a scale from 0 to 2: f; Vt; and the degree of sternocleidomastoid muscle activity, subcostal retraction, and abdominal paradox (Table 1). The total score ranged from 0 to 10, with 10 inferring a normal or tolerable WOB.

#### Table 1 — Breathing Pattern Score (Inference of WOB)*

<table>
<thead>
<tr>
<th>Variable Rank</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>f, breaths/min</td>
<td>&gt;30</td>
<td>&gt;20-30</td>
<td>≤10-20</td>
</tr>
<tr>
<td>Vt, mL/kg</td>
<td>&lt;4</td>
<td>4-8</td>
<td>&gt;8-10</td>
</tr>
<tr>
<td>Sternocleidomastoid muscle activity</td>
<td>Severe (noted by observation)</td>
<td>Mild (noted by palpation only)</td>
<td>Absent</td>
</tr>
<tr>
<td>Subcostal retraction</td>
<td>Severe</td>
<td>Mild</td>
<td>Absent</td>
</tr>
<tr>
<td>Abdominal paradox</td>
<td>Severe</td>
<td>Mild</td>
<td>Absent</td>
</tr>
</tbody>
</table>

*Score range=0 to 10; a score of 10 is an inference of a normal WOB of respiratory muscle load. The lower the score, the greater the WOB and vice versa.
It is inferred that the greater the breathing pattern score, the lower the WOB and vice versa. The selected variables and scoring range are based on our collective experience at quantifying changes in the breathing pattern. The variables used in the breathing pattern score are commonly used by many clinicians to assess breathing. Only 30 patients at one of the institutions (University of Florida) were evaluated with the breathing pattern score.

WOB performed by the patient was also directly measured at all levels of PSV. Total measured WOB, which includes physiologic (elastic and resistive) work and the imposed resistive work of the breathing apparatus (endotracheal tube, breathing circuit tubing, and ventilator’s demand-flow system) during spontaneous ventilation was calculated by integrating the change in intraesophageal pressure (indirect measurement of intrapleural pressure) and Vr.9

Intraesophageal pressure was measured (relative to atmospheric pressure) by the nasal insertion of an esophageal balloon that is integrated into a nasogastric tube (Smart Cath, Nasogastric Balloon Catheter; Bicore; Allied Healthcare Products; Riverside, Calif). Correct position was confirmed with an occlusion test as described by Baydur et al.10 following occlusion of the airway opening, the changes in pressure at the airway and in the esophagus are close to identical during spontaneous inspiratory efforts. Vr was obtained by integrating the flow signal of a pneumotachograph (flow sensor) positioned between the “Y” piece of the breathing circuit and the endotracheal tube. Airway pressure was measured from a catheter attached to the pneumotachograph; this was used to measure CPAP and PSV. Data from these measurements and from the measurement of chest wall compliance were processed with a portable, commercially available respiratory monitor (Bicore, model CP-100) that calculates WOB by the method described by Campbell11 and Agostoni et al12 (Fig 1). This is a well-established method that has been used by many investigators to assess WOB during ventilatory support.13-16

The nasogastric tube with the esophageal balloon and the pneumotachograph are components of the monitor, which also provides real-time measurements of f, f/Vt, and Ti/Ttot. The degree of sternocleidomastoid muscle activity, subternal retraction, and abdominal paradox were ranked based on clinical presentation, ie, observation and palpation by a board-certified anesthesiologist who was an attending physician in critical care medicine.

Accuracy in measuring chest wall compliance requires a relaxed and mechanically ventilated patient. Patients were initially given 1 to 2 mg of midazolam for relaxation and then the mechanical ventilator rate was increased transiently to about 12 breaths/min. Under these conditions of mechanical inflation with a preselected Vr esophageal pressure increases. The monitor integrates the changes in esophageal pressure and volume to produce a pressure-volume loop moving in a counterclockwise direction, the slope of which was taken as the chest wall compliance. This compliance value was stored in the monitor’s computer memory. Measured chest wall compliance values were 109±37 mL/cm H2O. Next, 0.2 mg of flu-
mazelnol was given to reverse the effects of the midazolam so that
the patient resumed spontaneous breathing. The dose was repeated
if complete reversal of sedation was not achieved in 10 min. The
mechanical ventilator rate was decreased to zero and PSV reapplied.

The monitor places the static chest wall compliance line at the
deflection in baseline airway pressure that marks the onset of each
spontaneous inhalation. Normally, this deflection and the start of
inspiratory flow coincide. The development of intrinsic positive
end-expiratory pressure, however, produces a threshold load re-
quiring the respiratory muscles to generate sufficient force to
counteract the opposing positive recoil pressure before flow begins.17 Under these conditions, the static chest wall compliance
line is placed before flow commences and is displaced horizontally
from the zero flow point and the lung compliance line. Separation
of the two compliance lines increases the area enclosed within the
Campbell diagram and, in turn, increases elastic and thus, total
WOB. At the onset of each spontaneous inhalation, the monitor
measures intrinsic positive end-expiratory pressure as the change in
intraesophageal pressure required to effect a change in the baseline
airway pressure.

WOB performed by the ventilator to inflate the respiratory sys-
tem during PSV, which was not measured in this study, may be
calculated by the monitor; pressure measured at the airway open-
ing is integrated with Vt to produce a pressure-volume loop. The
area circumscribed within this loop is defined as the work per-
formed by the ventilator.

In a validation study of the accuracy of the monitor,18 WOB was
measured by using the monitor and simultaneously using conven-
tional equipment to construct a Campbell diagram. The relationship
between the two sets of measurements of WOB was nearly perfect
(r2=0.99, p<0.001). Bias was minimal (−0.05 J/L) and precision was
excellent (+0.03 J/L).

All measurements (over 200 determinations) were obtained af-
after 15 to 20 min at each level of PSV, 5 to 10 per test, averaged over
1 min, and then recorded. Measurements were recorded only if they
could be consistently reproduced during each set of tests; thus, in-
ternal test consistency (reliability of measurement) was ensured. When
appropriate, arterial blood gas and hemodynamic data were
collected at various levels of PSV. All variables or inferences of
WOB were regressed with directly measured values for WOB at all
levels of PSV. Data were analyzed by linear, polynomial (second
order), and multiple regression analyses. Alpha was set at 0.05 for
statistical significance.

RESULTS

Measured values for WOB ranged from 0 to 2.2 J/L. The level of
PSV applied ranged from 5 to 50 cm H2O. All breathing pattern variables poorly predicted WOB as evidenced by the low values for the coefficients
determination (r2). For each variable, r2 predicts or explains the amount of variance in WOB. A variable with an r2 value between 0.64 and 0.81 is considered
high and thus, a fairly good predictor.19 The f correlated positively with WOB and predicted only 22% of
the variance in WOB (Table 2 and Fig 2). Vt correlated negatively and for f/Vt and Ti/Ttot each correlated positively with WOB. However, these variables predicted 20 to 27% of the variance in WOB (Table 2). The breathing pattern score correlated negatively with
WOB and it predicted only 43% of the variance in
WOB (Table 2, Fig 3).

By using multiple regression analysis, a prediction
to consider. Only three variables were significant predic-
tors of WOB: breathing pattern score, Ti/Ttot, and
Vt. However, because it predicted only 50% of the
variance in WOB, the model was a poor to moderate
d predictor of WOB (Table 3).

For most ventilatory conditions, all patients were in
hemodynamically stable condition and blood gas ex-
change was within acceptable ranges.

DISCUSSION

The main finding of this study is that, for adults with
abnormal pulmonary mechanics and loaded respira-
tory muscles who are in respiratory failure and being
treated with PSV, the f and the breathing pattern are
poor inferences of the WOB. Our study reveals that
WOB should be measured directly because f, for ex-
ample, appears to be an inaccurate and misleading
variable from which to infer the respiratory muscle
workload. The clinical implication of these findings

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<tr>
<th>Table 2—Relationships Between Directly Measured WOB and Variables Used to Predict or Infer WOB</th>
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<tr>
<td>Predictor Variables</td>
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<tr>
<td>---------------------</td>
</tr>
<tr>
<td>f</td>
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<tr>
<td>Vt</td>
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<tr>
<td>Index of rapid, shallow breathing (f/Vt)</td>
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<tr>
<td>Duration of respiratory muscle contraction, expressed as Ti/Ttot</td>
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<td>Breathing pattern score</td>
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Figure 2. Relationship between WOB and spontaneous f is depicted. Although a positive and significant correlation was found (r=0.47, p<0.001), spontaneous f predicted or explained only 22% of the variance in WOB, ie, a poor predictor. Within a clinically acceptable range of 15 to 25 breaths/min, some patients performed no work (predisposing to disuse respiratory muscle atrophy), others performed work in a normal range, and some performed work in a fatiguing range (predisposing to respiratory muscle fatigue).
Phases of a respiratory support system are illustrated in Figure 1. Performing work of breathing (WOB) in a normal range brings into question the traditional practice of titrating PSV based on breathing pattern, as this may result in inappropriate respiratory muscle workloads. This point is illustrated in Figure 2. While receiving PSV and breathing at a clinically acceptable range between 15 and 25 breaths/min, the WOB for some patients was either zero, in a normal range (0.3 to 0.6 J/L) (Fig 2), or at an abnormally high range. Similar findings were observed with the breathing pattern score (Fig 3). Disuse respiratory muscle atrophy may result when the muscles are totally unloaded and performing no work for an extended period. Respiratory muscle fatigue is likely to result when the muscles are forced to perform workloads at an abnormally high range for too long a period. With either severe respiratory muscle atrophy or fatigue, patients may become dependent and not wean from ventilatory support.

Our findings are in accordance with other studies. Silas et al reported that the index of rapid shallow breathing (f/Vt) as described by Tobin et al was a poor predictor of WOB because it predicted only 16% of the variance in WOB in patients before extubation. The index of rapid shallow breathing predicted only 20% of the variance in WOB in our patients. Most of our patients were not ready for weaning from ventilatory support but were treated during the initial-to-late phases of respiratory failure. Although regarded as an inference of the respiratory muscle workload, the index of rapid shallow breathing does not appear specific enough to infer WOB when applying PSV. Moreover, regarded to be an accurate predictor of weaning failure, the index has also been criticized as poorly predicting extubation outcome. The index lacked sufficient sensitivity and specificity to differentiate between patients who were successfully extubated and those who were not.

Because it predicted only 50% of the variance in WOB, the prediction model is not considered a clinically acceptable method of inferring WOB. Other factors explaining the variance in WOB not included in the model may be carbon dioxide minute production, oxygen consumption, lung and chest wall compliances, airway resistance, physiologic dead space, and peak inspiratory flow rate demand. A more comprehensive prediction model, including the aforementioned variables, may increase the value of $r^2$ and, thus, its predictive ability. However, it is easier to measure WOB directly than to measure and regress a number of physiologic variables. Predicting WOB based on a host of clinical variables, even assuming that all measurements are obtained correctly, appears dicey.

We do not dispute the fact that as pulmonary mechanics deteriorate, the respiratory muscles are loaded, WOB increases, and the breathing pattern changes. Specifically, these changes are vagally mediated by afferent or sensory fibers (load sensors) in the lungs and respiratory tract. Three types of afferent fibers that modulate the breathing pattern have been described: (1) slowly adapting receptors; (2) rapidly adapting receptors (also termed “deflation,” “cough,” or “irritant” receptors), both of which are pulmonary stretch or mechanoreceptors; and (3) chemosensitive or C-fiber endings. Slowly adapting receptors are found in the bronchial smooth muscle fibers, rapidly adapting receptors in the superficial layers of the respiratory tract mucosa, and C-fibers in the airway epithelium. These receptors detect changes in pulmonary mechanics and thoracic gas volume (functional residual capacity).

Following a decrease in lung compliance (increase in respiratory muscle load), an increase in discharge

### Table 3—Prediction (Multiple Regression) Model for WOB

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Coefficient</th>
<th>Standard Error</th>
<th>t-Value</th>
<th>p-Value</th>
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<tr>
<td>f/Vt</td>
<td>-0.44</td>
<td>0.02</td>
<td>-21.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Ti/Ttot*</td>
<td>-0.21</td>
<td>0.003</td>
<td>-6.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Vt</td>
<td>+2.13</td>
<td>0.21</td>
<td>10.0</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*Vt, the duration of respiratory muscle contraction expressed as Ti/Ttot, and the breathing pattern score were the only significant (p<0.05) predictors (regressors) in the model, where f is breathing frequency (breaths/min) and f/Vt is the index of rapid shallow breathing (breaths/min-L). The factor “Institution” (University of Florida and University of Miami) is not shown because it also was not a significant regressor. The model predicts or explains only 50% of the variance in WOB, i.e., a relatively poor to moderate prediction model.
activity from the stretch receptors increases. Similar responses follow increases in total resistance. C-fiber endings are activated by many substances produced in the lungs, such as histamine, bradykinin, and some prostaglandins. Some sympathetic afferents may also be activated by increases in mechanical loads. Affenter discharge signals from the sensory fibers are directed back via the vagus nerve to the central respiratory controllers in the central nervous system, modifying their output signal, which in turn, modifies the breathing pattern. Stimulation of these receptors produces patterns of rapid, shallow breathing to minimize large changes in intrapleural pressure. Patients with loaded respiratory muscles breathe at a faster rate and a smaller Vt to minimize the WOB—the so-called minimal WOB or least average force concept, which produces the most energy-efficient combination of Vt and f. This may be described as optimal because, when the frequency is too low, much elastic work is required to produce large Vt; when f is too high, much resistive work is required, as well as work uselessly done in ventilating the dead space with each breath. This mechanism also functions to protect the respiratory muscles from exhausting, fatiguing contractions that could lead to muscle fiber splitting, hemorrhage, and self-destruction.

A local-load compensator mechanism involving muscle spindle receptors and motor neurons in the intercostal muscles and diaphragm has also been described. This mechanism regulates muscle contraction to obtain the “desired” change in length, and, thus, Vt. The demand for a given change in length is transmitted from the muscle receptors to the motor neurons. As a result, a change in mechanical load (eg, increased airway resistance) to the contracting muscle leads to compensatory adjustments that increase the activity of the motor neurons.

Unquestionably, this is a sophisticated, physiologic, load-sensing, and load-compensating mechanism that functions during spontaneous breathing. Whether this mechanism functions in the same manner during assisted spontaneous breathing with PSV is unknown. However, some altered role of the pulmonary stretch receptors in mediating the response seems likely, particularly when ventilatory support results in larger than normal Vt (this is often the case during PSV) and restoration of functional residual capacity by applying CPAP. The focal point or question of this argument is, can the aforementioned physiologic, load-sensing, and load-compensating mechanism be relied on alone to predict or infer respiratory muscle workloads accurately for all patients in respiratory failure treated with PSV? Our study and others reveal that the breathing pattern is not an accurate predictor of WOB and may provide preliminary evidence that this physiologic mechanism is indeed affected during positive pressure ventilatory support. There is also evidence that the perceived inspiratory effort sensation during spontaneous breathing (how the patient feels, degree of comfort) is not related to fatiguing or nonfatiguing diaphragmatic contractions. Thus, how a patient “feels” may also not be a reliable inference of respiratory muscle workloads.

A logical deduction is that WOB should be measured directly. With easy-to-operate, portable bedside respiratory monitors, real-time measurements of WOB are readily obtainable. Directly measured values for WOB may serve as objective and quantifiable data for applying PSV to achieve specific goals, ie, to partially or totally unload the respiratory muscles. A total WOB of 2 J/L (approximately 300% above normal) while a patient receives 10 cm H2O PSV, for example, would be considered by many clinicians to be a potentially fatiguing workload. PSV can be increased until the respiratory muscles are partially unloaded and the WOB is in a more tolerable or perhaps normal range. Totally unloading fatigued respiratory muscles and allowing them to rest and recover is appropriate under some circumstances. Muscle fatigue is reversible with rest and is the most important way to treat it. The time for respiratory muscles to recover from chronic fatigue is estimated to be at least 24 h. Some patients with COPD and respiratory muscle fatigue may benefit by decreasing the WOB to zero and totally unload the muscles for approximately 24 h. Subsequently, PSV may be decreased so that the WOB is in a more normal range and the respiratory muscles are partially unloaded. The load tolerance for each patient may be accurately and objectively determined rather than subjectively inferred (guessed) by observing the breathing pattern only.

In our experience over a 3-year period, directly measured WOB data were useful for selecting appropriate levels of PSV for approximately 50% of our patients. These data were particularly helpful when we were uncertain whether the respiratory muscles were properly unloaded for patients with moderate-to-advanced respiratory failure during weaning from ventilatory support when measurements of imposed and physiologic WOB were useful in identifying those patients who could be successfully extubated. Patients with milder forms of respiratory failure usually do not need this type of monitoring. In others, however, the measurements appear essential to guide therapy appropriately.

The study was specifically designed not to involve a homogeneous subset of patients and ventilatory equipment. Conclusions and clinical implications of such a study would be limited in scope. Rather, a more heterogeneous study involving patients with a variety of diagnoses and using various types of ventilators allows more general conclusions to be drawn.
In summary, we contend that the traditional and somewhat subjective practice of inferring a level of WOB or respiratory muscle afterload during PSV based on bedside assessments of the breathing pattern alone appears inaccurate. We do not undermine the importance of assessing a patient’s breathing pattern and do not imply that it should be ignored. On the contrary, the breathing pattern provides useful diagnostic information and should be followed in patients receiving ventilatory support. Changes in the f and pattern are the result of changes in the workload performed by the respiratory muscles. However, we have shown that relying primarily on the breathing pattern alone does not provide enough information to accurately assess the patient’s WOB when titrating PSV. WOB measurements and breathing pattern should be used in a complementary manner when titrating PSV in critically ill patients.

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