Liberation From Mechanical Ventilation*  
A Decade of Progress

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Multiple complications associated with mechanical ventilation mandate that clinicians expeditiously define and reverse the pathophysiologic processes that precipitate respiratory failure and then, detect the earliest point that a patient can breathe without the ventilator. Over the past decade, numerous laboratory and clinical studies have been reported that may inform transformation of the “art of weaning” to the science of liberation. We review these studies and use them to formulate a systematic approach to assure early, safe, and successful liberation of patients from mechanical ventilation.

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Key words: hypoxemia; hypercapnia; liberation; mechanical ventilation; respiratory failure; weaning

Abbreviations: \( CO_2 = \) carbon dioxide; CPAP = continuous positive airway pressure; CRF = compliance, rate, oxygenation, pressure; Crs = respiratory system compliance; MV = maximum voluntary ventilation; NIPPV = noninvasive positive-pressure ventilation; \( P_{0.1} \) = airway occlusion pressure; PEEP = positive end-expiratory pressure; pHi = intramucosal pH; PImax = maximum inspiratory pressure; PSV = pressure-support ventilation; RF = respiratory failure; ROC = receiver operating characteristic; RVR = rate:volume ratio (respiratory rate:tidal volume ratio); SIMV = synchronized intermittent mandatory ventilation; \( V_A \) = alveolar ventilation; \( V_{CO_2} \) = carbon dioxide output; \( V_d \) = dead space ventilation; \( V_e \) = minute volume

Respiratory failure (RF) is defined as the failure of the lungs and respiratory pump to exchange oxygen and carbon dioxide (\( CO_2 \)) adequately. Hypoxemic (type 1) RF results from flooding or collapse of alveoli, which leads to intrapulmonary shunt and inadequate arterial oxygenation despite generous concentrations of inspired oxygen. Hypercapnic (type 2) RF results from inability to sustain sufficient alveolar ventilation to eliminate the \( CO_2 \) produced metabolically. Perioperative RF, a special case of types 1 and 2, results when postoperative pain and recumbency result in atelectasis and hypoxemia or when medications to alleviate pain reduce respiratory drive leading to hypercapnia. Shock-related RF is another special case in which the underperfused respiratory muscles are unable to compensate for the acidosis resulting from inadequate global tissue perfusion. Mechanical ventilators substitute for the respiratory pump until these disturbances have been reversed adequately to allow resumption of spontaneous breathing and gas exchange.

Although modern mechanical ventilators are versatile tools to stabilize the conditions of patients in RF, treatment with them should be withdrawn promptly when no longer necessary, so as to reduce the likelihood of nosocomial complications.1,2 To minimize the duration of mechanical ventilation, the clinician should (1) define and treat the underlying cause of RF, and (2) discontinue machine support on the earliest possible day. We cannot overemphasize the importance of the first step, which is reviewed in detail in pulmonary and critical care textbooks. The second step, commonly referred to as “weaning,” is the subject of this review.

In 1987, Hall and Wood3 suggested that weaning, which implies the gradual withdrawal of mechanical ventilation, is unnecessary in most patients. Further,
the use of this term tends to divert the physician’s attention from the treatment of the patient and shift it inappropriately to manipulating the ventilator. They proposed that the ultimate goal is not to wean but, rather, to liberate the patient from the machine. Over the ensuing decade, numerous investigators have examined methods of identifying readiness for liberation and studied whether weaning is necessary. We herein review these developments and propose a simple, pathophysiologic approach to expeditious liberation of the patient from mechanical ventilation.

**Identification of Readiness for Liberation: Weaning Parameters**

Weaning parameters are objective criteria used to predict the readiness of patients to successfully sustain spontaneous ventilation and maintain adequate oxygenation. Further, these parameters give insight into the mechanisms of ventilator dependence in individual patients by revealing the general pathophysiologic mechanism of ventilatory failure (i.e., reduced neuromuscular competence, decreased respiratory system compliance [Crs], or increased flow resistance). Some diseases primarily affect neuromuscular function to reduce pump capacity (e.g., Guillain-Barré, myasthenia gravis, steroid myopathy), while others secondarily cause pump failure by overloading the respiratory muscles (e.g., acute severe asthma, pulmonary fibrosis). Not infrequently, critical illness both reduces pump capacity (e.g., sepsis, electrolyte imbalances, narcotics) and increases the ventilatory load (e.g., bronchospasm, pulmonary edema, hypermetabolism). Since the patient’s oxygenation is readily apparent from arterial blood gas analysis or pulse oximetry, most weaning parameters focus on the ability to maintain an acceptable PaCO2. We review below most of the commonly used weaning parameters.

A more direct method of assessing readiness is to simply initiate a trial of unassisted breathing. To consider a patient for a trial of spontaneous breathing, oxygenation must be adequate (PaO2 >60 mm Hg) on a concentration of oxygen (≤50%) that can be increased should some decrement in gas exchange occur during the transition, at nominal levels of positive end-expiratory pressure (PEEP ≤5.0 cm H2O). The patient should be in hemodynamically stable condition and, preferably, not excessively tachycardic. Additionally, excessive respiratory muscle loads such as severe bronchospasm in asthmatics (airways resistance >20 cm H2O/L/s) should be reversed before considering a patient for liberation from mechanical ventilation, since excessive loads are likely to precipitate failure.

**Measures of Neuromuscular Function**

The capacity of the respiratory muscles to perform work against mechanical and metabolic loads depends on intact neuromuscular function. Neuromuscular parameters suggesting poor function may predict weaning failure, but normal function does not necessarily predict success, especially if coupled with excessive loads.

**Maximal Inspiratory Pressure (PImax, Negative Inspired Force):** The maximal pressure generated on a voluntary (or involuntary) inspiratory effort from functional residual capacity is commonly used to test respiratory muscle strength. Normal subjects did not develop task failure when subjected to an inspiratory resistive load if they generated <40% of PImax with each breath.4 In this study, subjects breathed in a constrained manner as to maximize diaphragmatic breathing. It is commonly assumed that if a critically ill patient can generate adequate ventilation while keeping the breath-by-breath inspiratory pressure below 30% of PImax, the patient does not require a ventilator. However, whether the findings from short-term inspiratory loading in normal subjects can be extrapolated to critically ill patients, who may have less ventilatory endurance, is unknown.

Measurement of the voluntary PImax requires patient effort and coordination and is difficult to perform reproducibly in many intubated critically ill patients. Accordingly, in clinical practice, the measured pressure is not necessarily the maximal inspiratory pressure. In 100 predominantly surgical patients being assessed for discontinuation of mechanical ventilation, a PImax ≥25 cm H2O was associated with successful extubation while all patients with a PImax <22 cm H2O failed extubation,5 though another study failed to reproduce these results.6

In 100 medical patients recovering from RF, the predictive value of this single test was low.7 A common method for assessing predictive value of a test is to measure the area under the receiver operating characteristic (ROC) curve. ROC curves are derived by plotting sensitivity vs 1—specificity and are independent of outcome frequencies and threshold values. An area of 1.0 (100%) under an ROC curve suggests that the test is a perfect predictor, while an ROC area of 0.5 suggests that a test is no better than random chance in predicting outcomes. In this study, the ROC area for PImax was 0.61 (Table 1). In another series of 100 medical patients being weaned from the ventilator, the ROC area for the PImax was 0.68,8 while it was 0.62 in 183 postoperative patients.9 Thus, the PImax cannot be used by itself to predict reliably the outcome of weaning. In part, this may relate to the difficulty in assuring cooperation from patients who are so ill.
Table 1—Predictive Characteristics of the PImax

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Patients</th>
<th>Sens</th>
<th>Spec</th>
<th>PPV</th>
<th>NPV</th>
<th>ROC Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yang and Tobin⁷</td>
<td>100</td>
<td>Medical</td>
<td>1.0</td>
<td>0.14</td>
<td>0.60</td>
<td>1.0</td>
<td>0.61</td>
</tr>
<tr>
<td>Pmax ≤20 cm H2O</td>
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<tr>
<td>Pmax ≤30 cm H2O</td>
<td></td>
<td></td>
<td>0.56</td>
<td>0.21</td>
<td>0.58</td>
<td>0.55</td>
<td></td>
</tr>
<tr>
<td>Chatla et al⁴</td>
<td>100</td>
<td>Medical</td>
<td>0.90</td>
<td>0.26</td>
<td>0.67</td>
<td>0.60</td>
<td>0.68</td>
</tr>
<tr>
<td>Pmax ≤20 cm H2O</td>
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<td>0.67</td>
<td>0.69</td>
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<tr>
<td>Pmax ≤30 cm H2O</td>
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<td></td>
<td>0.96</td>
<td>0.07</td>
<td>0.92</td>
<td>0.14</td>
<td></td>
</tr>
<tr>
<td>Jacob et al⁴</td>
<td>183</td>
<td>Surgical</td>
<td>0.96</td>
<td>0.07</td>
<td>0.92</td>
<td>0.14</td>
<td>0.62</td>
</tr>
</tbody>
</table>

*Sens = sensitivity; Spec = specificity; PPV = positive predictive value; NPV = negative predictive value.

Truwi and Marini¹⁰ have described a method for measuring PImax that does not depend on cooperation. The airway is occluded for 20 to 25 s with a one-way valve that allows the patient to exhale, but not inhale, a maneuver that leads to increasing inspiratory effort and a PImax that is generally greater than that measured by other techniques. This is still a PImax that is less than the maximal pressure that the inspiratory muscles are capable of generating during a tetanic involuntary contraction and may thus be an underestimate of true muscle strength. Even when such steps are not taken (and we do not use this method routinely), we believe the PImax is useful by helping to identify patients with potentially treatable respiratory muscle weakness. Recently, experimental methods utilizing electrical or magnetic stimulation of the phrenic nerves to achieve diaphragmatic contractions have been pioneered and may allow more accurate estimation of respiratory muscle strength. Note, however, that even a normal muscle with a normal PImax will fatigue if coupled with excessive loads,⁴ so that this single measure of neuromuscular competence will never be an adequate single weaning parameter.

Airway Occlusion Pressure: The airway occlusion pressure (P0.1) is the pressure measured at the airway opening 0.1 s after inspiring against an occluded airway. This index is a measure of respiratory drive; abnormally elevated drive during spontaneous breathing could result from a mismatch of mechanical load and neuromuscular function. In 20 medical patients, P0.1 was >4.2 cm H2O in weaning failures while patients with P0.1 ≤4.2 cm H2O were successfully extubated.¹¹ In another study of 12 patients with COPD, all patients who failed to wean had P0.1 >6.0 cm H2O, while those who were successful had P0.1 <6.0 cm H2O.¹² However, these findings (in relatively small, selected samples of patients) have not been reproduced in other studies. Moreover, since the P0.1 requires instrumentation that is not present on most ventilators, its practical clinical utility remains unclear.

Vital Capacity: The vital capacity is used in many hospitals to assess single-breath respiratory muscle performance. A value >10 mL/kg has been suggested to predict weaning success, but this has not been proven to be clinically useful.⁶,¹³ A vital capacity <10 mL/kg is commonly used as the threshold for intubating and mechanically ventilating patients with worsening neuromuscular diseases (eg, Guillain-Barré syndrome, myasthenia gravis), although to our knowledge, there are no data to demonstrate improved outcomes in patients treated with this strategy.

Maximum Voluntary Ventilation: The MVV is measured with a spirometer for 12 to 15 s during which the patient ventilates maximally. In one study, patients whose resting minute volume (Ve) was <10 L/min and whose MVV was greater than twice resting Ve were successfully liberated. However, seven patients who could not double their resting Ve were also successfully liberated;⁵ thus the discriminative value of the MVV remains unproved.⁶

A variety of other indexes of respiratory muscle function, including the inspiratory time/total cycle time, inspiratory pressure-time product (the time integral of the difference between esophageal pressure and chest wall recoil pressure during inspiration), the ratio of electromyographic power in high and low frequency bands, rib cage/tidal volume ratio, tension-time index (the product of the mean inspiratory pressure normalized for maximal pressure generation and inspiratory time/total cycle time), inspiratory effort quotient, and maximal relaxation rate are tools that are undergoing clinical investigation. Most of these require equipment that is not routinely available and their utility has not been validated in prospective clinical trials.

Measures of Respiratory Muscle Load

For clinical purposes, three respiratory muscle loads should be considered: Ve, Crs, and airways resistance. The presence of these loads can be inferred from the mechanical ventilator, since the Ve is directly measured and abnormalities of compli-
ance and resistance affect the peak airway pressure. Intrinsic PEEP (also called auto-PEEP or dynamic hyperinflation), one of the causes of abnormal Crs, is not readily evident by examining the ventilator, but rather requires special maneuvers to detect (see below and Fig 1). One might expect that the higher the load, the poorer the weaning outcome. Yet even normal loads can precipitate ventilatory failure in patients with neuromuscular insufficiency. Thus, respiratory muscle loads must be interpreted in relation to respiratory muscle strength.

**Minute Volume:** Ve is the total ventilation in liters per minute. The arterial PCO₂ is proportional to \( \dot{VCO}_2/V_A \), and \( V_A = Ve - Vd \), where \( \dot{VCO}_2 \) is total body CO₂ production, \( V_A \) is alveolar ventilation, and \( Vd \) is dead space ventilation each minute. Any process that increases \( \dot{VCO}_2 \) or \( Vd \) increases the Ve required to maintain a normal PCO₂, thus loading the respiratory muscles. \( \dot{VCO}_2 \) is determined by the rate of total body metabolism and it increases with exercise, sepsis, burns, fever, multiple organ failure, and hyperthyroidism. \( \dot{VCO}_2 \) in itself is not a good predictor of weaning outcome, but a higher \( \dot{VCO}_2 \) loads the respiratory muscles. Similarly, a higher dead space fraction increases the Ve required to maintain \( V_A \), but it has not discriminated between weaning successes and failures.

A \( Ve < 10 \text{ L/min} \) has been associated with weaning success. However, in two prospective studies that included a combined total of 200 medical patients, \( Ve \) was a poor predictor of weaning outcomes with ROC areas=0.54 and 0.40. A similar prospective study of 183 surgical patients revealed an ROC area of 0.54. High values of \( Ve (>15 \text{ to } 20 \text{ L/min}) \) may help identify patients who are unlikely to be liberated, but lower values are not helpful in predicting success.

**Respiratory System Compliance:** The static compliance of the respiratory system (Crs,st) describes the pressure-volume relationship of the lungs and chest wall (\( \Delta V/\Delta P \)). The pressure needed (\( \Delta P \)) to inflate the lungs by a given volume (\( \Delta V \)) is found by

![Figure 1](http://journal.publications.chestnet.org/pdaccess.ashx?url=/data/journals/chest/21828/)

**Figure 1.** Determination of intrinsic PEEP. At end-expiration, the alveolar pressure is normally atmospheric. In the presence of significant airflow obstruction, however, exhalation is incomplete and the alveolar pressure may still be positive. Since the expiratory limb of the ventilator is open to atmosphere (or the PEEP valve) during expiration, the airway pressure gauge shows only atmospheric pressure (or the set PEEP), not the abnormally elevated alveolar pressure. By closing the expiratory limb of the ventilator at end-expiration, the alveolar pressure can be measured.
subtracting the end-expiratory pressure from the plateau pressure (the airway pressure when the expiratory valve is briefly occluded at end-inspiration). The dynamic compliance (\(C_{rs,\text{dyn}}\)) is calculated similarly, except that \(AP\) is found by subtracting the end-expiratory pressure from the peak airway pressure. A very stiff respiratory system should predispose to weaning failure. However, dynamic and static compliances are not good predictors, as judged by ROC areas of 0.67 and 0.68, respectively.\(^7\)

**Airways Resistance:** A very high airways resistance (eg, >20 cm H\(_2\)O/L/s determined as the peak dynamic airway pressure minus the plateau airway pressure divided by the inspiratory flow rate during constant flow ventilation) is likely to cause liberation failure even with normal respiratory muscles. However, to our knowledge, no studies have examined whether there is a value of airway resistance that precludes successful unassisted breathing. Nevertheless, we do not consider liberating patients with asthma until the airways resistance is less than about 20 cm H\(_2\)O/L/s.

**Changes in Respiratory Load During the Trial of Spontaneous Breathing:** In recent investigations Jubran and Tobin\(^{21,22}\) studied the pathophysiologic basis of acute respiratory distress in patients with COPD who failed a trial of weaning from mechanical ventilation. In comparison to the group successfully weaned, those who failed on discontinuation of the ventilator developed rapid shallow breathing with lower values of dynamic lung compliance and higher levels of intrinsic PEEP. This increment in intrinsic PEEP raises the work of breathing as discussed further below, forcing the respiratory muscles to perform undue work. Interestingly, the passive mechanics of the lung and chest wall were not significantly different between patients who were successfully liberated from mechanical ventilation and those who were not. The authors speculated that the pattern of breathing selected by the patients who failed, when coupled to their significant airflow obstruction, resulted in dynamic hyperinflation, diminished compliance, and muscle dysfunction related to large lung volumes. In this group of patients with COPD, lung mechanics that deteriorated during the period of spontaneous breathing appeared more important for predicting weaning failure than the lung mechanics measured during full ventilatory support.

Accordingly, the three elements of respiratory muscle load (resistance, compliance, and \(Ve\)) are not useful to assess readiness for liberation—at least not as measured prior to a trial of spontaneous breathing. In patients who fail a trial of spontaneous breathing, measurement of these parameters (see below) guides the physician to correct the reversible elements of elevated respiratory muscle loads that can contribute to subsequent successful liberation.

**Measuring the Effect of Weaning on Other Organs**

**Gastric Mucosal \(\text{PCO}_2\):** Respiratory loads are frequently increased and respiratory muscle efficiency may be reduced\(^23\) in some patients with respiratory failure. The transition to spontaneous breathing is expected to increase the proportion of total body oxygen delivery demanded by the respiratory muscles. This demand is met by an increase in blood flow to the actively contracting muscles of the respiratory system. In patients with marginal cardiopulmonary reserve, weaning may result in a "steal" of blood flow from other tissues. The development of gastric intramucosal acidosis during weaning might identify such patients. In one study of 29 medically ill patients,\(^24\) the \(\text{PCO}_2\) of gastric juice was measured before and during weaning, and gastric intramucosal pH (pHi) was computed using the Henderson-Hasselbalch equation. Eighteen patients were successfully extubated after pressure-support weaning and 11 patients failed. Gastric pH did not change during weaning in successful extubations, while it decreased in 9 of 11 failures. Moreover, values of pHi >7.3 during weaning or that changed by <0.09 predicted success significantly better than other conventional weaning parameters. Another study of 26 COPD patients confirmed the value of a gastric

### Table 2—Predictive Characteristics of the Spontaneous \(Ve\)*

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Patients</th>
<th>Sens</th>
<th>Spec</th>
<th>PPV</th>
<th>NPV</th>
<th>ROC Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yang and Tobin(^7)</td>
<td>100</td>
<td>Medical</td>
<td>0.31</td>
<td>0.61</td>
<td>0.50</td>
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</tr>
<tr>
<td>(Ve \leq 10 \text{ L/min})</td>
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<td></td>
<td>0.78</td>
<td>0.18</td>
<td>0.55</td>
<td>0.38</td>
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<tr>
<td>Chahilla et al(^a)</td>
<td>100</td>
<td>Medical</td>
<td>0.79</td>
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<td>0.48</td>
<td>0.54</td>
</tr>
<tr>
<td>(Ve \leq 15 \text{ L/min})</td>
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<td></td>
<td>0.97</td>
<td>0.11</td>
<td>0.65</td>
<td>0.67</td>
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</tr>
<tr>
<td>Jacob et al(^a)</td>
<td>183</td>
<td>Surgical</td>
<td>0.76</td>
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<td>0.93</td>
<td>0.13</td>
<td>0.54</td>
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<tr>
<td>(Ve \leq 10 \text{ L/min})</td>
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</tbody>
</table>

*For explanation of abbreviations, see Table 1 footnotes.*
pHi threshold of 7.30 when measured after a 20-min trial of spontaneous breathing via T-piece. In addition, a threshold value for gastric mucosal PCO₂ of 60 mm Hg (when measured before the trial) was also predictive. These studies did report fundamental differences that are not clearly reconciled. The former study did not report a difference between baseline pHi between weaning successes and weaning failures, while the latter did. Thus, this technique should be examined prospectively in a larger cohort of patients before being applied clinically.

**Integrative Indexes**

**Oxygen Cost of Breathing:** The oxygen cost of breathing can be estimated by subtracting total body oxygen consumption during controlled ventilation from that measured during unassisted breathing. Although one study suggested that the oxygen cost of breathing was a lesser percentage of total body oxygen consumption (8%) in patients who were successfully liberated as compared with those who failed (21%), others have failed to demonstrate significant differences. Since patients can perform significant respiratory muscle work during controlled ventilation, the assumption on which calculation of the oxygen cost of breathing is based is probably invalid, and this index remains of unproven value.

**Compliance, Rate, Oxygenton, Pressure (CROP):** The CROP is an index that integrates measurements of dynamic Crs, spontaneous respiratory frequency, arterial/alveolar oxygenation (PaO₂/PaO₂), and PImax in the following equation:

\[ \text{CROP} = \text{dynamic Crs} \times \text{PImax} \times (\text{PaO}_2/\text{PaO}_2)/\text{frequency} \]

Yang and Tobin found that a CROP >13 had a sensitivity of 0.81, specificity of 0.57, positive predictive value of 0.71, negative predictive value of 0.70, and ROC area of 0.78. Accordingly, the CROP offers a reasonably accurate predictor of weaning outcome. However, the CROP is somewhat cumbersome to use in the clinical setting as it requires measurement of five variables and subsequent calculation of the index. A simpler and more accurate alternative is available.

**Index of Rapid Shallow Breathing (RVR):** One index has now been studied in a large number of patients and appears to have predictive utility that is superior to other commonly used parameters. The RVR measures the respiratory rate/tidal volume ratio (breaths/min/L) during a 1-min T-piece trial and 105 breaths/min/L is the threshold that best predicts weaning outcome. Tobin and colleagues first noted that patients who failed to wean developed significantly more rapid (32 vs 21 breaths/min) and shallow (194 vs 398 mL/breath) breathing than those who succeeded. Yang and Tobin then performed a prospective study of 100 medically ill patients that demonstrated that the RVR was a significantly better predictor of weaning outcomes (ROC area=0.89) than the PImax or Ve (Table 3). Epstein confirmed the excellent sensitivity (92%) but found lower specificity (22%) of the RVR in medical patients. Poor specificity related to nonpulmonary factors such as congestive heart failure or glottic edema, which are not measured in the RVR. Chatila and colleagues studied 100 medical patients being weaned from mechanical ventilation using a method that was nearly identical to that of Yang and Tobin. The predictive value of the RVR measured after only 1 min (ROC area=0.74) was significantly less than when it was remeasured after 30 min of spontaneous breathing (ROC area=0.92; Table 3). Patients who failed tended to breathe more rapidly and shallowly during the course of a trial, whereas those who succeeded tended to breathe more slowly. Jacob and colleagues studied 183 postoperative patients finding that the RVR was superior to Ve and PImax in predicting weaning outcomes.

The principal defect in the RVR is that it produces excessive false positives (patients with RVR <105 breaths/min/L who fail); specificities from the various studies range from 11 to 64%. Accordingly, an RVR <105 breaths/min/L does not necessarily ensure successful liberation from mechanical ventilation, but should prompt a spontaneous breathing trial of 30 to 120 min to further assess patient readiness. The RVR should not be measured until sedative and narcotic effects have adequately abated and the patient triggers >2 to 3 breaths/min above the ventilator set rate. Slow, shallow breathing (even

### Table 3—Predictive Characteristics of the RVR*

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients</th>
<th>Sens</th>
<th>Spec</th>
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<th>NPV</th>
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<td>0.64</td>
<td>0.78</td>
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<td>0.76</td>
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</table>

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CHEST / 114 / 3 / SEPTEMBER, 1998 891

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with RVR <100 breaths/min/L) suggests that respiratory drive is not fully developed, which could also contribute to false positives. The RVR has not been validated in patients who have been ventilated long term and it may not be as accurate in this population of patients. Finally, since no study (to our knowledge) has extubated all patients after obtaining the RVR, reported sensitivities of this screening tool may be overestimated. A recent study suggests that the RVR may vary significantly with gender (higher in women) and endotracheal tube size (proportional to tube diameter). Nevertheless, with all of these cautions in mind, the RVR may be one of the better indexes for predicting weaning, is easily applied at the bedside, and can be readily employed to identify candidates for a spontaneous breathing trial.

THE LOGISTICS OF LIBERATION

Preparation

As the patient recovers from respiratory failure, the physician should anticipate the possibility of spontaneous breathing trials. Sedatives should be adjusted in advance to facilitate sleep during the night, but to allow maximum alertness and cooperation in the morning. Short-acting agents such as propofol or midazolam facilitate such a rapid reduction in the level of sedation. Gastric feeding should be held to allow time for stomach emptying in case a successful spontaneous breathing trial leads to extubation. It is preferable for patients to sit upright to reduce the work of breathing. A nurse, respiratory therapist, or physician should carefully observe the patient for signs of failure (see below).

Modality

Pressure-support, continuous positive airway pressure (CPAP), and T-piece trials are the most common methods used to test readiness for liberation from mechanical ventilation. The choice of whether to use pressure support or to allow the patient to breathe without inspiratory assistance through the endotracheal tube remains largely a matter of physician preference. The endotracheal tube imposes a resistive load on the respiratory muscles that is inversely related to its cross-sectional diameter. Thus, some clinicians advocate use of 5 to 8 cm H2O pressure support to offset this imposed load. However, some investigators have found that this level of pressure support may unload the respiratory muscles beyond the conditions of spontaneous breathing and thus contribute to an overestimation of the patient’s ability to breathe spontaneously. Nevertheless, two large studies have suggested that the rate of extubation failure is similar after successful T-piece or pressure-support (5 to 7 cm H2O) trials, suggesting that either approach is acceptable. In one of these trials, a direct comparison of T-piece vs pressure support at 7 cm H2O in a diverse group of 484 patients, the percentage of patients failing the trial was significantly higher in those randomized to T-piece (22% vs 14%). Once extubated, there was no difference in the proportion remaining extubated after 48 h. The authors concluded that either method was suitable for assessing a patient’s ability to breathe, but speculated that the somewhat higher failure rate during T-piece trials might lead to continued (and unnecessary) ventilation in a small number of patients who could be successfully extubated.

Success is measured by subjective comfort, physiologic stability (no significant increment in heart rate, BP, or respiratory rate), and an arterial blood gas value that does not demonstrate acute respiratory acidosis or hypoxemia (defined as PaO2 <60 mm Hg on 50% inspired oxygen). Patients who have successful breathing trials should be considered for extubation (see below).

APPROACH TO THE PATIENT WHO FAILS

When a patient fails a trial of spontaneous breathing, clinicians frequently focus on specific ventilator regimens aimed at improving respiratory muscle function. To date, and to our knowledge, no study has suggested that the ventilator can be used to expedite the recovery from respiratory failure. This is not surprising, since the reason for persistent ventilatory failure (impaired drive, inadequate neuromuscular competence, or excessive load) lies within the patient, not within the machine. Accordingly, the clinician should turn attention to the patient and seek reversible contributors to weaning failure (Table 4). Only when the patient has improved will liberation be possible.

The clinical signs of weaning failure include tachypnea, tachycardia, hypertension, mental status changes, and subjective distress. These signs are due to (1) difficulty maintaining gas exchange, (2) cardiovascular events, and/or (3) noncardiopulmonary factors.

Difficulty Maintaining Gas Exchange

Hypercapnia: Acute hypercapnia during weaning frequently results from an imbalance between respiratory pump capacity and load (Fig 2). Normal individuals who are subjected to resistance loading exhibit rapid shallow breathing as a sign of impending respiratory failure. In many patients, rapid
shallow breathing during a trial of unassisted breathing signals strength-load imbalance that can eventuate in hypercapnia if the trial goes on for too long. When rapid shallow breathing is accompanied by other signs of weaning failure, the trial of spontaneous breathing should be stopped so as to avoid respiratory muscle exhaustion. Acute respiratory acidosis or prolonged rapid shallow breathing during a weaning trial should prompt identification of reversible causes of respiratory pump insufficiency or elevated load (Table 4).

Table 4—Reversible Factors Contributing to Ventilatory Failure*

<table>
<thead>
<tr>
<th>Factors</th>
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<tbody>
<tr>
<td>Improve neuromuscular competence</td>
</tr>
<tr>
<td>Treat sepsis</td>
</tr>
<tr>
<td>Nutritional support without overfeeding (aim to achieve a normal prealbumin)</td>
</tr>
<tr>
<td>Replace K⁺, Mg²⁺, PO₄⁻ to normal</td>
</tr>
<tr>
<td>Assure periods of respiratory muscle rest; avoid exhausting breathing trials</td>
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<tr>
<td>Limit the use of neuromuscular blocking drugs</td>
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<tr>
<td>Consider stopping aminoglycoside therapy</td>
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<tr>
<td>Consider</td>
</tr>
<tr>
<td>Neurologic disease/occult seizures</td>
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<tr>
<td>Hypothyroidism</td>
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<tr>
<td>Oversedation</td>
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<tr>
<td>Critical illness myopathy/polyneuropathy</td>
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<tr>
<td>Steroid myopathy</td>
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<tr>
<td>Investigational/unproven</td>
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<tr>
<td>Anabolic steroids</td>
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<tr>
<td>Growth hormone</td>
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<tr>
<td>Aminophylline</td>
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<tr>
<td>Reduce respiratory load</td>
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<tr>
<td>Resistance</td>
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<tr>
<td>Bronchodilators</td>
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<tr>
<td>Corticosteroids</td>
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<tr>
<td>Removal of excess airway secretions</td>
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<tr>
<td>Treatment of upper airway and endotracheal tube obstruction</td>
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<tr>
<td>Compliance</td>
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<tr>
<td>Treat pneumonia</td>
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<tr>
<td>Treat pulmonary edema</td>
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<tr>
<td>Reduce intrinsic PEEP</td>
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<tr>
<td>Drain large pleural effusions</td>
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<tr>
<td>Evacuate pneumothoraces</td>
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<tr>
<td>Treat ileus</td>
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<tr>
<td>Decompress abdominal distention</td>
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<tr>
<td><strong>Respiratory</strong></td>
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<tr>
<td>Treat sepsis</td>
</tr>
<tr>
<td>Antipyretics</td>
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<tr>
<td>Avoid overfeeding</td>
</tr>
<tr>
<td>Correct metabolic acidoses</td>
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<tr>
<td>Reduce intrinsic PEEP</td>
</tr>
<tr>
<td>Bronchodilators</td>
</tr>
<tr>
<td>Maintain least PEEP possible</td>
</tr>
<tr>
<td>Resuscitate shock and hypovolemia</td>
</tr>
<tr>
<td>Identify and treat pulmonary embolism</td>
</tr>
</tbody>
</table>

*Reversible contributors to ventilatory failure should be sought daily to speed recovery.

Figure 2. The coupling of respiratory neuromuscular capacity and respiratory muscle loads. This diagram summarizes the components of neuromuscular competence and respiratory muscle loads, and illustrates processes that can affect the strength-load balance leading to ventilatory failure. (Reproduced with permission from Manthous and Siegel.95)

Measure and Reverse Reduced Neuromuscular Competence

Most patients who fail trials of spontaneous breathing have adequate drive as reflected by tachypnea and distress. When the Pco₂ rises substantially and the patient fails to demonstrate tachypnea, accessory muscle use, and subjective distress, reduced drive should be suspected.

Two relatively common situations arise in which hypercapnia during a spontaneous breathing trial is not accompanied by physiologic decompensation, does not signal a failed trial, and should not prompt termination of the trial: (1) compensation for metabolic alkalosis,40 and (2) return to a chronic hypercapnic state following iatrogenic overventilation as in patients with COPD or sleep apnea. Most other patients who do not exhibit signs of ventilatory
failure in the face of rising Pco₂ have impaired consciousness due to sedatives or brain injury. Occasional patients have hypothyroidism or subclinical status epilepticus,41 two conditions worth considering because they are treatable.

Critical illness is frequently accompanied by processes that reduce respiratory pump capacity. Malnutrition and electrolyte deficiencies can contribute to respiratory muscle weakness. The polynuropathy of critical illness may reduce respiratory pump function and prolong the need for mechanical ventilation.42,43 Sepsis may also contribute to respiratory muscle weakness44-47 and weaning failure.48 Medications that are commonly used in the treatment of critically ill patients such as corticosteroids49-51 and neuromuscular blockers52-54 also negatively affect respiratory muscle function. Prolonged mechanical ventilation may contribute to respiratory muscle atrophy, though few data exist to support this hypothesis.

In occasional patients, we speculate that reduced neuromuscular competence represents inadequate rest of the respiratory muscles (persistent fatigue). This may be present in patients who have only recently (<48 hours previously) been intubated after days or weeks of deterioration. In such patients, full rest of the respiratory muscles for the 24 to 48 hours on the ventilator may be useful to restore maximal function. Another possible example of fatigued respiratory muscles is the patient who performs inordinate amounts of ventilatory work, despite being connected to a ventilator. Even during “full” mechanical ventilation, such as with the assist-control mode, patients often perform large amounts of ventilatory work. This is especially likely when the patient’s drive is high (as in sepsis), when the ventilator settings are not well matched to the patient (for example, inspiratory flow rate too low), or when intrinsic PEEP is present and not matched by machine PEEP (discussed further below). Optimizing patient-ventilator synchrony is important both during periods of exercise and of rest.55,56

**Measure and Reverse Elevated Loads**

In patients who fail due to strength-load imbalance, we quantify the three elements of respiratory muscle load (resistance, compliance, and Ve) each day so as to define and treat reversible processes. Respiratory system resistance can be measured when patients are ventilated passively during volume ventilation as follows: (peak airway pressure-plateau pressure)/inspiratory flow rate. Bronchospasm and excessive airway secretions are the most common reasons for high airway resistance.

Total respiratory system static compliance can be easily measured as follows: tidal volume/(plateau pressure-PEEP). Low Crs, usually considered <40 mL/cm H₂O, can result from a stiff chest wall, stiff lungs, flooded or atelectatic alveoli, or intrinsic PEEP. Intrinsic PEEP is not readily detected by examining the inspiratory airway pressures and must be sought by special maneuvers. When patients are being passively ventilated, a brief end-expiratory pause reveals the elevated alveolar pressure (Fig 1). When patients are actively breathing, intrinsic PEEP can be recognized (but not quantitated) by persistent expiratory flow at end-expiration when the ventilator is equipped with a flow waveform display.

Pressure triggering of the demand valve on the ventilator may also impose a significant load on the spontaneously or even “fully” ventilated patient.55,57,58 In part, this results from the mechanical function of these valves, but at times it is related to airway obstruction in the patient. In those patients with sufficient airflow obstruction to cause intrinsic PEEP, the valve is not pressure triggered until respiratory muscle contraction first counterbalances the residual positive alveolar pressure (that is, the intrinsic PEEP), and then further reduces alveolar pressure to lower the proximal airway pressure beyond the triggering value. This inspiratory threshold load raises the work of breathing. In such patients, addition of machine PEEP or CPAP to nearly match intrinsic PEEP can reduce this inspiratory load on breathing, usually without further hyperinflating the patient.59-62 It is difficult to measure accurately the amount of intrinsic PEEP in spontaneously breathing patients by the expiratory pause technique, however, because of active expiration. Thus attempting to approximately match the degree of intrinsic PEEP by externally applied PEEP must be done with caution. Flow triggering rather than pressure triggering the machine demand valve has not been shown to be superior to pressure triggering in all studies.63-67

The third element of load, namely the Ve, can be readily measured either with a spirometer during unassisted breathing or by the ventilator during mechanical ventilation. Ve loads may represent respiratory alkalosis (eg, due to anxiety, acidemia, or sepsis), excess dead space (eg, as in emphysema, pulmonary embolism), or elevated CO₂ production (eg, due to fever, overfeeding).

**Hypoxemia:** Hypoxemia (PaO₂ <60 mm Hg or saturation <90% on 50% inspired oxygen) can occur during weaning for several reasons. Obesity and recumbency predispose to a lower functional residual capacity that can contribute to atelectasis and subsequent hypoxemia during the transition to unassisted breathing. This propensity can be minimized if patients sit up during spontaneous breathing trials. Respiratory muscle weakness (see above) and drugs
(sedatives or narcotics) may also contribute to shallow breathing and atelectasis. Lung injury, a common complication of critical illness, is associated with surfactant depletion and increased propensity for atelectasis during the withdrawal of positive-pressure ventilation. Thus, many pulmonary factors can contribute to hypoxemia during weaning. Hypoxemia can also result from cardiovascular changes during weaning, such as cardiogenic pulmonary edema, arrhythmias, or decreased cardiac output (which reduces the mixed venous saturation).

**Cardiovascular Events**

The transition from positive-pressure ventilation to unassisted breathing redistributes centrally blood and edema pooled peripherally and raises left ventricular afterload, both of which may precipitate cardiogenic pulmonary edema.\(^{68-75}\) Catecholamine surges during weaning\(^ {76}\) may also induce increases in heart rate, BP, or arrhythmias. Tachycardia is a common cardiovascular manifestation of weaning failure. In patients with coronary artery disease, weaning can trigger cardiac ischemia that may also contribute to clinical signs of weaning failure.\(^ {77-79}\)

**Noncardiopulmonary Factors**

**Ventilator and Circuit Elements:** The ventilator and its circuitry can contribute to weaning failure by two mechanisms: (1) by imposing exhausting respiratory muscle work during “rest” periods, as described above, when the ventilator is not adjusted appropriately to the patient, and (2) by presenting an unnecessary respiratory load during a spontaneous breathing trial. An example of the second mechanism is elevated endotracheal tube resistance. Endotracheal tube resistance (normally 5 to 10 cm H\(_2\)O/L/s) increases over time, as the tube becomes encrusted with secretions and its cross-sectional area reduced. In general, the resistance of these tubes as reported by the manufacturer is less than resistances measured in the patient. This problem is often suspected when a large difference between the peak and plateau airway pressures fails to respond to bronchodilators. Another source of unnecessary load is seen when a spontaneous breathing trial is conducted by using the ventilator CPAP setting (rather than T-piece). The inspiratory valve triggered sensitivity is often left at ~2 cm H\(_2\)O (the default value on many ventilators is ~1 or ~2 cm H\(_2\)O) rather than being turned to zero, so that the patient has to lower the proximal airway pressure to ~2 cm H\(_2\)O to generate any inspiratory flow, even though this triggers no assistance from the ventilator.

**Psychological Issues:** Anxiety can manifest as rapid shallow breathing, tachycardia, and hypertension, which are interpreted as weaning failure by caregivers. Anxiety can be reduced by carefully explaining the weaning process to the patients and then reassuring them during spontaneous breathing trials. Sedatives (eg, small doses of haloperidol) are occasionally helpful, but should be used cautiously because they reduce respiratory drive and can mask the underlying causes of weaning failure. We emphasize that anxiety is usually a consequence of weaning failure rather than a cause of it.

**Weaning Strategies**

To our knowledge, there are no studies definitively proving that respiratory muscle training, through the use of decremental ventilatory support, hastens the recovery to unassisted breathing. Thus, weaning, that is the gradual withdrawal of ventilatory support intended to slowly strengthen the muscles, is of unproven benefit and may serve only to prolong mechanical ventilation (see below).

Two large multicenter studies have assessed the role of “weaning” strategies in expediting liberation from mechanical ventilation. Brochard and colleagues\(^ {36}\) studied 456 medical-surgical patients being considered for weaning. Three hundred forty-seven patients (76%) were successfully extubated after a single 2-h T-piece trial. One hundred nine patients who failed an initial trial of spontaneous breathing were randomized to be weaned by one of three strategies: (1) T-piece trials of increasing length until 2 h could be tolerated; (2) synchronized intermittent mandatory ventilation (SIMV) with attempted reductions of 2 to 4 breaths/min twice a day, until 4 breaths/min could be tolerated; and (3) pressure-support ventilation (PSV) with attempted reductions of 2 to 4 cm H\(_2\)O twice a day until 8 cm H\(_2\)O could be tolerated. Patients randomized to the three strategies were similar with regard to disease severity and duration of ventilation before weaning. There was no difference in the duration of weaning between the T-piece and SIMV groups, but PSV led to significantly shorter weaning compared with the combined T-piece and SIMV cohorts. These authors concluded that “the outcome of weaning from mechanical ventilation was influenced by the ventilatory strategy chosen, and the use of PSV resulted in significant improvement compared with other strictly defined weaning protocols using T piece or SIMV.”

Esteban and colleagues\(^ {80}\) performed a similar study of 546 medical-surgical patients. In this study, 416 (76%) patients were successfully extubated on their first day of weaning after a T-piece trial. The 130 patients who failed were randomized to undergo weaning by (1) once a day T-piece trial, (2) two or
more T-piece or CPAP trials each day as tolerated, (3) PSV with attempts at reduction of 2 to 4 cm H₂O at least twice a day, and (4) SIMV with attempts at reducing 2 to 4 breaths/min at least twice a day. Patients assigned to the four groups were similar with regard to demographic characteristics, acuity of illness, and cardiopulmonary variables. The weaning success rate was significantly better with once-daily and multiple T-piece trials than for PSV and SIMV. PSV was not superior to SIMV. The median duration of weaning was 5 days for SIMV, 4 days for PSV, and 3 days for the T-piece regimens. These authors concluded that “a once-daily trial of spontaneous breathing led to extubation about three times more quickly than intermittent mandatory ventilation and about twice as quickly as pressure-support ventilation.”

These seminal studies provide strong evidence for the following assertions:

1. The majority of patients with RF can be successfully extubated on their first attempt of spontaneous breathing. Gradual withdrawal of ventilatory support would only have served to prolong the period of ventilator dependence in a majority (76%) of patients. In fact, some of these patients surely could have been liberated sooner had physicians recognized earlier their readiness to breathe.

2. In patients who fail their initial attempt at spontaneous breathing, SIMV weaning is inferior to other approaches and serves to prolong the duration of mechanical ventilation.

**Exubation**

The physician should distinguish between liberation (no need for the ventilator) and extubation (no need for the endotracheal tube). Aside from coupling the patient to the mechanical ventilator, the endotracheal tube also maintains and protects the airway and allows removal of secretions from patients whose cough and airway protective mechanisms are inadequate. Thus, after a patient has performed a successful trial of unassisted breathing (is liberated), one must make a second judgment about whether the artificial airway is still required.

One consideration is whether the patient has an upper airway lesion that would collapse to a critically small aperture after extubation. Patients at risk of this problem include those who were initially intubated for upper airway stenosis and stridor and those who have had a traumatic or prolonged intubation. A number of studies have suggested that the ability to breathe around a deflated endotracheal tube cuff or the presence of a cuff leak >110 mL during volume-cycled ventilation predicts an adequate airway diameter. However, lack of a cuff leak does not absolutely predict extubation failure.

Assessing the patient’s mental status, airway protective mechanisms, ability to cough, and character of secretions requires experience and judgment. Many clinicians delay extubation in patients with a weak cough or excessive, tenacious secretions. Patients with severe cerebral vascular accidents frequently present with this constellation and when to extubate such patients remains a difficult, unsolved clinical problem. Swallowing is abnormal in >30% of general ICU patients following extubation. Accordingly, all patients, and especially those with altered mentation or cerebral vascular accidents, should be carefully observed for aspiration of oropharyngeal secretions, especially when they first attempt drinking and eating.

Postextubation stridor due to glottic edema, tracheal stenosis, or laryngospasm may complicate extubation. Pulmonary edema can develop in some of these patients because large negative intrathoracic pressures during inspiration can dramatically increase left ventricular afterload. Nebulized racemic epinephrine and parenteral corticosteroids aid in increasing airway aperture. Heliox may be used to temporarily reduce upper airway resistance in selected patients who do not require immediate reintubation. Finally, mask CPAP may stent the airway, reduce the work of breathing, and provide time for inflammation and edema to subside.

Noninvasive face-mask positive-pressure ventilation (NIPPV) may prevent the need for reintubation in those who appear to be failing immediately after extubation. In one prospective study, 39 patients with COPD and normal sensorium who had failed a T-piece trial were randomized to pressure-support weaning or extubation with NIPPV. Patients assigned to NIPPV were more likely to be liberated from mechanical ventilation and succeeded 5 days sooner than those who were weaned using pressure support. Thus, NIPPV may be useful in cooperative patients who appear to be failing extubation and who do not require immediate reintubation for impending respiratory arrest. It should be remembered that NIPPV is merely supportive therapy, not a treatment of the underlying cause of ventilatory failure. When NIPPV succeeds in staving off reintubation, the physician must continue to identify and treat reversible causes of decreased neuromuscular competence and elevated load.

We sometimes extubate patients who fail to meet our usual criteria for liberation. Some examples include the following:

1. When an endotracheal tube has been in place
for >7 days; endotracheal tube resistance increases with time and could contribute to failed breathing trials.

2. When the patient experiences repeated episodes of bronchospasm upon awakening from sedation; the endotracheal tube can cause reflex bronchospasm in some individuals. One approach in patients with asthma is to rapidly awaken and extubate them when the respiratory mechanics have improved, rather than waiting for the patient to demonstrate his or her ability to sustain spontaneous breathing with the tube in.

3. When patients become overwhelmingly anxious when awakened to breathe through the endotracheal tube and the amount of sedative required to make them comfortable causes hypoventilation. We are particularly careful to assure that cardiopulmonary reasons for failure have been reversed in these patients.

4. When patients with restrictive chest wall disease (eg, obesity) repeatedly desaturate every time PEEP is decreased to <10 cm H2O; some obese patients require >5 to 10 cm H2O to prevent atelectasis while intubated yet maintain adequate oxygenation when extubated.

5. When patients with chronic severe restrictive or obstructive lung disease breathe rapidly and shallowly (with RVR >125 breaths/min/L); for some patients with end-stage disease, rapid shallow breathing is their chronic baseline. Waiting excessively for a successful trial of spontaneous breathing through the endotracheal tube risks missing a window of opportunity.

In these relatively rare situations, extubation should not be performed casually. We consider “breaking the rules” outlined in this article only after numerous failed trials of unassisted breathing and after treating reversible causes of failure. The clinical risks associated with failure and reintubation must be weighed against those of continued mechanical ventilation. These unusual patients should be extubated only with personnel who are skilled at endotracheal intubation nearby, should reintubation be required. In addition, NIPPV should be available at the bedside as it may avert the need for reintubation in carefully selected patients.90

A Practical Approach

At many institutions, patients remain bound to ventilators for longer than necessary because clinicians either do not obtain weaning parameters in a timely manner or are hesitant to liberate even when parameters and breathing trials are favorable. Mechanical ventilation can cause complications.1,2 However, there is also risk associated with extubation failure. The relative risks of each vary from patient to patient and from institution to institution. A simple approach can minimize both risks by asking daily: Can my patient be liberated from the ventilator? The use of interdisciplinary weaning teams89,92 or respiratory therapist-driven protocols83 may expedite successful liberation by actively addressing this question each day.

Ely and colleagues84 prospectively randomized 300 mechanically ventilated medical patients to receive algorithm-guided attention to weaning or routine care. All patients were assessed daily to delineate recovery to adequate oxygenation (PaO2/fraction of inspired oxygen >200 on ≤5 cm H2O PEEP), hemodynamic stability, and an RVR ≤105 breaths/min/L (measured on CPAP) and adequate cough. When all of the above criteria were met, the patient was placed on a 2-h T-piece or CPAP (5 cm H2O) trial. If they passed this trial of spontaneous breathing, a note was left in the charts of patients assigned to the treatment group suggesting that they were ready for liberation. Patients in the experimental group were liberated 1.5 days more rapidly than patients treated only by their physicians. The median time to liberation after satisfaction of the criteria was 2 days shorter in the treatment group. This reduction in ventilator days reduced the ICU costs of care by $5,000 per patient. These results cannot necessarily be generalized to all institutions since “routine care” (the control group in this study) likely varies considerably between hospitals. However, this study supports the concept that daily use of validated screening tools to measure the very first day of readiness reduces the duration of mechanical ventilation.

A Simple and Practical Algorithm

The algorithm presented in Figure 3 distills the literature reviewed in this article and our own experience into a simple bedside approach to liberation. If the patient is in hemodynamically stable condition and triggering the ventilator with PaO2 >60 mm Hg on an inspired oxygen of ≤50% and PEEP ≤7.5 cm H2O, perform a 2- to 3-min T-piece trial and measure the RVR. If the RVR is ≤125 breaths/min/L, place the patient on a T-piece trial (40 to 50% oxygen) of 30 to 120 min. This threshold is used because roughly half of patients whose RVR is between 100 and 125 can be successfully liberated whereas very few patients with RVR >125 can be.8 If the patient is comfortable without tachycardia or hypertension, the oxygen saturation remains >90%, and the RVR stays <125 breaths/min/L at 30 min, obtain an arterial blood gas and, if it is favorable, consider a trial of extubation (see above). Patients
who still require an artificial airway can continue to breathe as much as they tolerate off of the ventilator until they are ready for extubation. Patients with an initial RVR >125 breaths/min/L and those who fail the trial of spontaneous breathing should be assessed as to the pathogenesis of their ventilatory failure as discussed above. Meanwhile, the particular ventilator settings are probably unimportant as long as the patient gets adequate rest. T-piece or pressure-support trials should be performed daily until the patient can tolerate >30 min of unassisted breathing.

**Conclusions**

A decade has passed since Hall and Wood\(^3\) suggested that the term "weaning" from mechanical ventilation should be replaced with "liberation," to
emphasize that most patients do not require a prolonged period of gradual withdrawal of this potentially dangerous therapy. Many studies have been conducted in the interim that support the following conclusions:

1. The majority of mechanically ventilated patients can be liberated from the machine after a brief trial of spontaneous breathing when their underlying diseases have been identified and they are responding to treatment;

2. Most weaning parameters are helpful in identifying causes of RF rather than as predictors of who will succeed at spontaneous breathing.

3. The duration of ventilator dependence can be reduced by using validated, clinical parameters, especially the rapid shallow breathing index, each day to identify the first day of readiness for liberation.

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