Weaning from Mechanical Ventilation

Melvin L. Morganroth, MD,
and Cyril M. Grum, MD

For most patients who require mechanical ventilation weaning and extubation is simple. In these patients a variety of strategies can be successful. In addition, simple criteria may predict when the patient is ready for extubation. For the small group of patients who require prolonged mechanical ventilation, however, controversy exists about how best to remove ventilator support by weaning, and available data are sparse. Much of the controversy has centered on T-piece weaning versus intermittent mandatory ventilation. To date no controlled study has demonstrated the superiority of either intermittent mandatory ventilation or T-piece weaning in difficult-to-wean patients. In the evolution of this controversy, concern has developed over the potential for increased inspiratory work and expiratory resistance that may be associated with certain intermittent mandatory ventilation systems. The possibility that significant inspiratory work may occur during assist-control ventilation has also been demonstrated. Respiratory muscle weakness and fatigue is likely important in failure to wean. Other possible causes are failure of the cardiovascular system and impaired ability of the lung to carry out gas exchange. In this article we first examine criteria and techniques for weaning short-term ventilator patients. We then examine criteria to begin the weaning process in prolonged ventilation patients, potential causes of failure to wean, and techniques that can be used to remove ventilator support from patients who are difficult to wean.

Much literature has been devoted to techniques and criteria for weaning and extubation of patients from mechanical ventilation. For most patients who require ventilatory support, weaning and extubation can be easily accomplished by a variety of techniques [1–4]. At one referral center 77.2% of all surviving patients were weaned from the ventilator within 72 hours of the onset of mechanical ventilation, and 91% were weaned within 7 days [1]. Less than 10% of ventilated patients potentially posed problems in weaning from mechanical ventilation. Similarly, at a community hospital, few surviving patients required prolonged ventilatory support [2]. In easy-to-wean patients, Sahn and Lakshminarayan [5] described simple criteria that are predictive of successful discontinuation of ventilator support. For the small group of patients who require prolonged mechanical ventilation, however, minimal data are available. In these patients criteria to determine weaning ability or which measurements to follow are not clearly defined. Furthermore, no controlled trials are available to compare the different weaning techniques proposed. In this article we first address routine weaning of the patient who has not required prolonged ventilator support. We then examine the difficult-to-wean patient and discuss criteria to begin the weaning process, potential causes of failure to wean, and available weaning techniques.

Routine Weaning

The patient undergoing routine weaning is awake and alert and is clinically stable or improving. Typically the patient has been on the ventilator for fewer than 7 days and required ventilator support for a reversible process (e.g., anesthesia for cardiac surgery or drug overdose). The patient should be able to maintain an adequate arterial oxygen tension (PaO_2; of greater than or equal to 60 mm Hg) on an oxygen fraction less than or equal to 0.4

Table 1. Spontaneous Ventilatory Measurements That Predict Successful Weaning in Patients Requiring Short-Term Ventilation

Measurement	Value Predicting Successful Extubation
Resting minute ventilation	$\leq$ 10 L
Negative inspiratory force	$\geq$ -30 cm H_2O
Maximum voluntary ventilation	$\geq$ 2 times resting minute ventilation

without positive end-expiratory pressure. For these patients criteria have been established that predict successful discontinuation of mechanical ventilation [5]. These criteria (Table 1) are easily obtained. The amount of ventilation the resting patient requires to maintain PaO_2 and arterial carbon dioxide tension ($PaCO_2$) is measured (resting minute ventilation, or $\dot{V}E$). Indexes of respiratory strength or ventilatory reserve are also measured (i.e., negative inspiratory force or maximum voluntary ventilation). Sahn and Lakshminarayan [5] examined these criteria in a prospective study of 100 patients ventilated for multiple causes (61% postoperative, 15% chronic obstructive pulmonary disease [COPD], 9% pulmonary edema, 5% pneumonia). Any patient with a $\dot{V}E$ less than or equal to 10 L and a maximum voluntary ventilation greater than or equal to two times $\dot{V}E$ or a negative inspiratory force (NIF) greater than or equal to -30 cm H_2O could be successfully extubated (76 of 100 patients, mean NIF = -31.5 cm H_2O). Seven of the 24 patients (29%) who did not meet the criteria (mean NIF = -28 cm H_2O), however, still were successfully extubated. The patients who could not be extubated (17 of 24) had a mean NIF of -17 cm H_2O. Thus, these criteria are specific but not sensitive. Therefore, failure to satisfy these ventilatory criteria or other measures that have been used to indicate ability to wean (e.g., functional residual capacity, work of breathing, or dead space to tidal volume ratio) does not preclude successful extubation. Because patients who fail to meet these criteria are likely to fail weaning, however, they should be observed very carefully during a weaning attempt.

One successful approach for routine weaning has been to allow the patient to breathe on a T-piece circuit with a fraction of inspired oxygen of 0.4 to 0.5 [1,5]. If the patient is comfortable and maintains a normal or acceptable cardiac rhythm and respiratory rate, arterial blood gases are obtained after 15 to 30 minutes. If the resultant oxygenation is acceptable (PaO_2 $\geq$ 60 mm Hg and similar to the PaO_2 on the ventilator) and there is no significant carbon dioxide retention ($<$ 5 mm Hg increase in $PaCO_2$) or acute decrease in the arterial

pH, the patient is immediately extubated after suctioning. If a patient demonstrates the ability to support unassisted respiration on a T-piece circuit and meets the spontaneous ventilatory criteria in Table 1, it is extremely unlikely that the patient will require reintubation, barring a new event or the development of stridor postextubation. Importantly, a large percentage of patients who do not meet the criteria in Table 1 but demonstrate the ability to support unassisted ventilation on a T-piece circuit can still be successfully extubated. Using this technique, more than 90% of patients who survive mechanical ventilation can be successfully extubated within 7 days of the onset of ventilator support [1].

Other techniques have also been successfully used to wean short-term ventilator patients. These include intermittent mandatory ventilation (IMV) [3,4], high-frequency jet ventilation [6], and pressure support ventilation [7,8]. When weaning by IMV, the patient is switched to an IMV setting at 8 to 12 breaths per minute and a tidal volume of 10 to 12 ml/kg. The rate of the ventilator is then decreased progressively at 1- to 4-hour intervals as long as spontaneous respiratory efforts are adequate, the arterial pH is greater than 7.30, and PaO_2 is adequate [4]. Patients are considered completely weaned and extubated when they are clinically stable and able to maintain for 1 hour an arterial pH greater than 7.30 and adequate oxygenation on an IMV rate equal to 0. High-frequency jet ventilation has also been used to wean patients from ventilator support by progressively decreasing driving pressure in 5 psi steps at 4-hour intervals [6]. When blood gases are adequate with a driving pressure of 15 psi, the ventilator is discontinued, and the patients are placed on a T-piece circuit and are subsequently extubated. Similarly, pressure support ventilation has been used to wean patients by progressive decreases in inspiratory pressure [7,8]. None of these techniques have been demonstrated to be superior to T-piece weaning, however, in terms of either success rate or decreased time of ventilator support. More importantly, these patients can likely be successfully weaned by whatever technique is chosen. New techniques and research need to be directed toward patients who require prolonged mechanical ventilation and who are difficult to wean.

Weaning from Prolonged Mechanical Ventilation

Difficulty arises when a patient cannot support unassisted ventilation with an endotracheal tube in place or fails extubation following weaning trials by

any or all of the methods listed previously. Typically, these patients have underlying pulmonary disease, neurological disease, severe systemic disease, or multiorgan failure. For example, patients ventilated after organ transplant or with COPD spent 3.0 to 5.2 times as many days on the ventilator as compared with all other patients who survived mechanical ventilation (Nett L, Morganroth ML, Petty TL. Unpublished data). Although these patients represent a small fraction of those supported by mechanical ventilation, they often remain on the ventilator for a prolonged period of time. Importantly, their hospital survival rate is similar to that for all patients who are ventilated. In a retrospective study, although only 2% of patients ventilated received more than 30 days of mechanical ventilation (30–100 days), their hospital survival was 70%, which is similar to the overall hospital survival of 71% for all patients ventilated [9]. Sivak [10] found a 73% survival rate in patients who required prolonged mechanical ventilation. Because patients who require prolonged mechanical ventilation have a high hospital survival rate, it is important to develop a strategy to initiate weaning and to ensure that the weaning trials will progress.

Criteria to Begin the Weaning Process. A major problem in the patient who requires prolonged mechanical ventilation is deciding when to initiate the weaning process by whatever technique is chosen. Data are sparse because most centers have few such patients. Furthermore, controlled studies are exceedingly difficult in this group of diverse patients. It has been suggested that NIF greater than −25 cm H_2O, forced vital capacity greater than 12 to 15 ml/kg, shunt fraction of less than 20%, alveolar to arterial gradient of less than 300% on 100% oxygen, or a V_D/VT less than 0.6 may be used to judge the ability to wean patients receiving long-term mechanical ventilation [10–12]. There are no controlled data, however, to confirm the usefulness of these measurements in patients receiving prolonged mechanical ventilation. Furthermore, Gilbert and colleagues [13] found that the forced vital capacity or spontaneous respiratory rate before a weaning trial did not predict in which patients respiratory distress would likely develop when placed on a T-piece circuit [13]. The utility of spontaneous ventilatory measures (NIF, forced vital capacity to tidal volume ratio [FVC/TV], respiratory rate, V_E) to assess the ability of prolonged ventilator patients to wean was examined in a retrospective study [9]. This study found that none of the patients met criteria that predict successful weaning in short-term ventilator patients. Furthermore, the patients' spontaneous ventilatory measurements did not change between the period when they could

Table 2. Means of Spontaneous Ventilatory Measurements During Unsuccessful and Progressive Weaning Periods

Measurement	Unsuccessful Wean Period	Progressive Wean Period
Respiratory rate	33 ± 1.6	29 ± 2.6
Tidal volume (mL)	391 ± 39	403 ± 27
Minute ventilation (L/min)	12.2 ± 1.7	11.1 ± 1.2
Negative inspiratory force (cm H_2O)	−25 ± 2.7	−27 ± 4.0
Forced vital capacity (mL)	702 ± 57	795 ± 69
Forced vital capacity to tidal volume	1.79 ± 0.24	1.89 ± 0.24

Data are taken from 8 patients who had multiple determinations during both periods. *P* values for all measurements (paired *t* test) failed to approach significance. (From Morganroth and colleagues [9]. Used with permission.)

not be weaned from the ventilator to the period when they could be progressively weaned (Table 2). Thus, measurements of a patients' spontaneous ventilatory ability have not been demonstrated to be useful in predicting when a prolonged ventilation patient is ready to begin weaning trials.

It has been suggested that weaning can be inhibited by multiple factors including fever, infection, arrhythmias, sedation, poor nutrition, psychological status, pain, and decreased level of consciousness [11,14–16]. From clinical experience these factors seem to be important and are supported by a retrospective study [9]. In that study a decrease in the underlying number and severity of medical, psychological, and respiratory problems correlated with the ability of prolonged ventilation patients to wean. There is general agreement that attention needs to be directed to these patients' multiple problems, and that the correction of these problems may facilitate the weaning process. Clear criteria to begin the weaning process, however, have not been prospectively evaluated in these patients. At present, that decision depends on the clinical judgment of the responsible physician aided by the respiratory therapist and nurse caring for the patient. A major problem in developing criteria to predict successful weaning is that the precise reason why these patients fail to wean is not known.

Potential Causes of Failed Weaning. Four potential causes of failure to wean are: 1) respiratory muscle weakness; 2) inadequate respiratory drive; 3) inability of the lung to carry out gas exchange; and 4) failure of the cardiovascular system.

The maximal pressure that can be generated by the respiratory muscles is usually below normal in patients who require prolonged mechanical ventilation [9,10]. This decrease could be due to mechanical disadvantage consequent to increased lung volume, weakness, acute or chronic fatigue, or a combination of these factors. Disorders of the central nervous system, peripheral nerves, neuromuscular junction, or muscles themselves can result in respiratory muscle weakness.

We focus on common factors that can lead to muscle weakness and considerations related to mechanical ventilation and weaning, including malnutrition, electrolyte disturbances, disuse atrophy, and inspiratory muscle fatigue. For a detailed review of this subject, refer to Braun and colleagues' [17] report of a conference examining respiratory muscle function in acute respiratory failure. Malnutrition may lead to loss of skeletal muscle mass and has been associated with a decrease in inspiratory muscle strength [18]. In these patients inspiratory mouth pressures increased following hyperalimentation. It is possible that chronic disease states associated with a catabolic state may also impair respiratory muscle structure and function. Another consequence of malnutrition or some acute and chronic disease states is electrolyte disturbances. Electrolyte disturbances that may impair respiratory muscle function include hypophosphatemia, hypocalcemia, and hypokalemia. Hypophosphatemia occurs frequently in patients with acute respiratory illness [19] and has been associated with respiratory failure [20]. Hypophosphatemia has also been demonstrated to impair the contractile function of the diaphragm. Transdiaphragmatic pressure (P_{di}) during bilateral supramaximal stimulation of the phrenic nerves in a group of patients with hypophosphatemia (.55 ± .21 nmol) was 9.75 ± 3.8 cm H_2O [21]. Following administration of 10 nmol of phosphate, P_{di} increased to 17.25 ± 6.5 cm H_2O. Thus, correction of hypophosphatemia and malnutrition has been demonstrated to cause clinically significant improvements in respiratory muscle function. Hypocalcemia and hypokalemia have also been associated with impaired respiratory muscle function. Aubier and associates [22] showed that hypocalcemia is associated with reduced diaphragmatic contractility in dogs. A clinical case demonstrated hypoventilation probably resulting from respiratory muscle weakness in association with severe hypokalemia complicating treatment of diabetic ketoacidosis [23]. Thus, it is important to monitor and maintain normal serum levels of potassium, calcium, and phosphate as well as reverse catabolic states in patients who are weaning from mechanical ventilation.

It is unclear whether disuse atrophy contributes to respiratory muscle weakness in patients receiving ventilator support. Human skeletal muscle, however, clearly develops disuse atrophy when rested. Fifty percent of patients will lose 2% of muscle strength per day in a casted leg [24]. The amount of work necessary to prevent this decrease in strength, however, was only one stimulation a day to 10% of the muscle's maximum tension [24]. Whether the work involved in triggering the ventilator on assist-control [25] or breathing on an IMV circuit [26] is sufficient to prevent disuse atrophy is unknown. The respiratory muscle work present during mechanical ventilation may be sufficient to prevent disuse atrophy, however, because no decrement was seen over time in NIF or FVC/TV in a group of patients who required prolonged mechanical ventilation [9]. It should also be noted that normal adults use less than 5% of their ventilatory reserve in the process of breathing. This small percentage of maximum energy expenditure, however, is adequate to avoid the development of disuse atrophy of the diaphragm.

Respiratory muscle fatigue can cause inspiratory muscle weakness in humans [27,28]. Respiratory muscle fatigue may also contribute to weaning failure in patients requiring mechanical ventilation [29]. Cohen and colleagues found electromyographical evidence of muscle fatigue in patients failing a weaning trial. In these patients the following sequence was observed (Fig 1). Initially, there was a shift in the electromyograph power spectrum accompanied by an increase in respiratory rate. This was followed by abdominal paradox and respiratory alternans as well as an increase in $PaCO_2$. Finally, a severe respiratory acidosis and decrease in $\dot{V}E$ and respiratory rate occurred. Chronic inspiratory muscle fatigue may also contribute to muscle weakness and respiratory failure. Marino and Braun [30] demonstrated that 5 months of nocturnal ventilation in patients with lung, chest wall, or neuromuscular disease resulted in an increase in FVC, maximum voluntary ventilation, and activity level. There was a decrease in the number of hospitalizations and reversal of right heart failure. These patients also decreased their $PaCO_2$ from 54 ± 8 mm Hg to 42 ± 5 mm Hg. These changes were presumed secondary to nocturnal rest of the inspiratory muscles and relief of chronic fatigue.

Although respiratory muscle fatigue may occur in normal humans breathing against a high respiratory load [28] and in other patients with acute and chronic respiratory failure, other causes of carbon dioxide retention during failed weaning trials are possible. Recently, Swartz and Marino [31] demonstrated that carbon dioxide retention occurred dur-
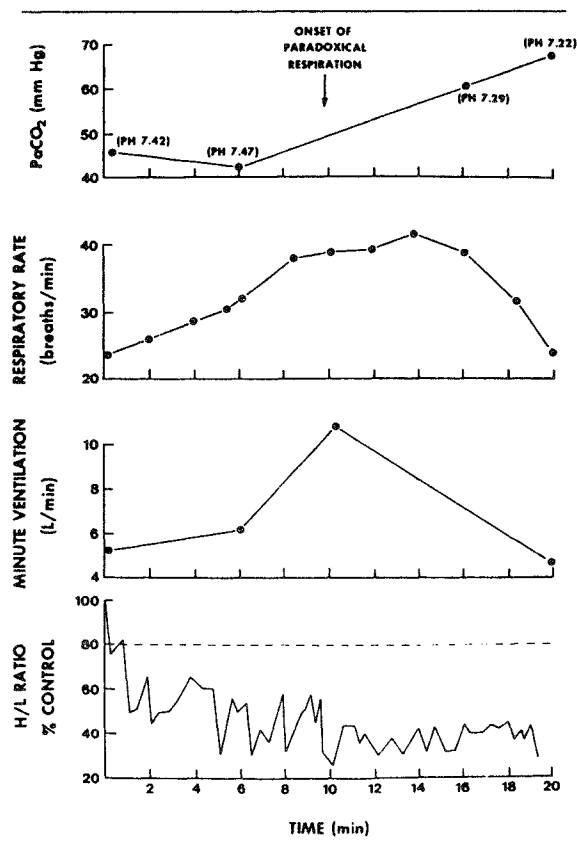
Fig 1. Sequence of changes in $PaCO_2$, respiratory rate, minute ventilation, and high/low ratio of the diaphragm in a patient during a 20-minute attempt at discontinuation of ventilator support. The initial change was the decrease in high/low ratio, followed by a progressive increase in respiratory rate. The $PaCO_2$ decreased initially, and the patient became alkalemic. Paradoxic abdominal displacements were not noted until after a substantial increase in respiratory rate and minute ventilation occurred. Hypercapnia and respiratory acidosis did not develop until after abdominal paradox and alternation between rib cage and abdominal breaths were noted. Just before artificial ventilation was reinstituted, there was a sharp decrease in respiratory frequency and minute ventilation. (From Cohen and associates [29]. Used with permission.)

Fig 2. Measurements of ΔP_{di} and $PaCO_2$ during weaning trials terminated for hypercapnia. Initial measurements were recorded within the first 5 minutes following discontinuation of the ventilator. Final measurements were recorded when the period of ventilator discontinuation terminated. Note that the increase in $PaCO_2$ is associated with the significant increase in ΔP_{di}. (From Swartz and Marino [31]. Used with permission.)

ing failed weaning trials, but this was not associated with decreased diaphragmatic pressure generation (Fig 2). The failure of transdiaphragmatic pressure to decrease, however, does not prove that diaphragmatic fatigue was not occurring. The force of diaphragmatic contraction (P_{di}) may be maintained even though decreased contractility (fatigue) occurs if the stimulus for the diaphragm to contract (chemical and central drive) is increased. Whether diaphragmatic fatigue occurred in Swartz and Marino's patients is uncertain because diaphrag-
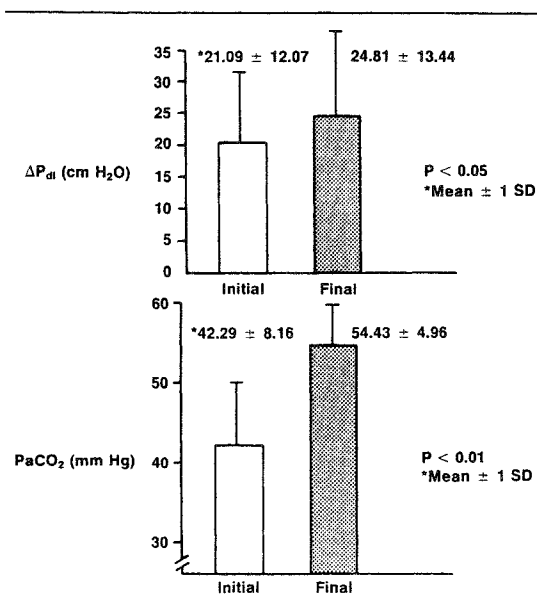
matic contractility, which required measurement of P_{di} in relation to a known stimulus, was not determined. Causes other than inspiratory muscle fatigue, however, potentially may have been responsible for the increase in $PaCO_2$. Increased carbon dioxide production by inefficient muscles, increased dead space to tidal volume ratio, or increased ventilation to perfusion mismatching are other potential causes for the hypercapnea that developed. Impaired ability of the lung to carry out gas exchange, as evidenced by an increased alveolar to arterial gradient, may also result in hypoxemia during weaning.

Hypercapnia and hypoxemia may also develop as a result of causes other than lung parenchymal dysfunction or respiratory muscle weakness. Central alveolar hypoventilation may occur. This may be primary or idiopathic or may result from secondary causes such as hypothyroidism, malnutrition, cerebral infarcts, as well as other central nervous system pathology. For a detailed review of the causes of extrapulmonary respiratory failure, refer to Pratter and Irwin's [32] recent review. Alternatively, situations with high levels of carbon dioxide production may lead to hypercapnia in a patient
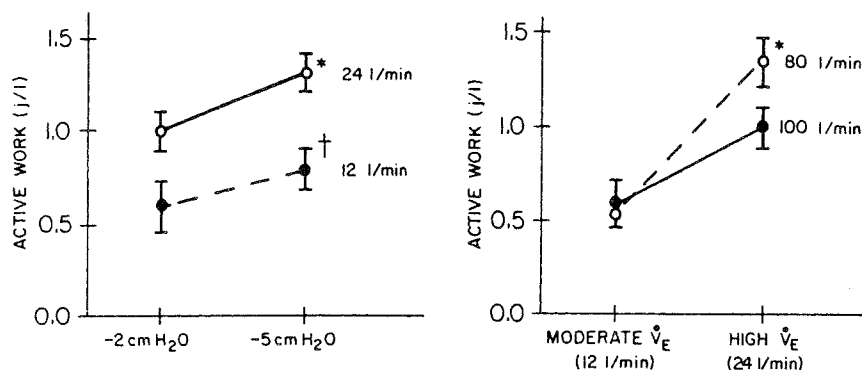
Fig 3. The graph on the left shows influence of trigger sensitivity on energy expended by the subject during assisted mechanical ventilation at two levels of minute ventilation ($\dot{V}E$), MA − 1 and flow setting = 100 L/min, positive end-expiratory pressure = 0 cm H_2O. Data points are expressed as mean ± SE. The signs * and † = significantly different from corresponding value at − 2 cm H_2O sensitivity. The graph on the right shows influence of peak flow setting on energy expended by the subject during assisted mechanical ventilation at two levels of $\dot{V}E$. MA − 1, trigger sensitivity = − 2 cm H_2O, positive end-expiratory pressure = 0 cm H_2O. (From Marini and colleagues [25]. Used with permission.)

with an inadequate respiratory reserve. For example, this may occur in patients given intravenous hyperalimentation, especially if excess carbohydrate is used. An elevation in carbon dioxide production may lead to an inability to maintain the patients' baseline $PaCO_2$ before the current episode of acute respiratory failure. Pulmonary or extrapulmonary respiratory failure secondary to many causes may contribute to the failure of weaning in prolonged ventilator patients.

Cardiovascular system failure is another cause of failure to wean in addition to respiratory system failure. Mechanical ventilation can provide support for a failing heart as well as a failing respiratory system [33–35]. This can occur by positive pressure ventilation, causing increased intrapleural pressure and diminishing right ventricular preload. In addition, left ventricular preload may be decreased because left ventricular filling may be impaired by increasing alveolar pressure. Finally, increased intrathoracic pressure may cause decreased left ventricular afterload [33]. In patients being weaned from mechanical ventilation who have underlying cardiac dysfunction, removal from positive pressure ventilation can result in increased pulmonary capillary wedge pressure and decreased cardiac output [34,35]. This may precipitate hypoxemia,

pulmonary dysfunction, and failure of weaning. Similarly, in patients with respiratory failure, a large fraction of cardiac output may be required for respiratory work [36]. In patients with low cardiac output states, the heart may not be able to supply the output necessary to meet these needs, which may lead to respiratory muscle dysfunction. In addition, respiratory work may precipitate cardiac ischemia and dysfunction in patients with primary cardiac disease [37].

Many different mechanisms may be responsible for a patient's failure to wean, and different mechanisms may be operative at different times or in different patients. One of the greatest difficulties in patients failing to wean is that the mechanism for each patient is often unrecognizable. Measurement of pulmonary capillary wedge pressure, cardiac output, oxygen consumption, carbon dioxide production, or transdiaphragmatic pressures may aid in elucidating the mechanisms responsible for a failed weaning trial. Correction of factors present that can increase the patient's respiratory requirements (e.g., bronchospasm, secretions, fever) or decrease muscle strength (e.g., hypophosphatemia, muscle fatigue, low cardiac output state) may facilitate weaning. It has been shown that successful progressive weaning in prolonged ventilation patients correlates with scores that demonstrate a decrease in respiratory requirements [9]. Finally, in patients with decreased muscle strength, it is clinically impossible to distinguish between respiratory muscle weakness and respiratory muscle fatigue. Therefore, most patients should be rested to eliminate muscle fatigue that may be present before initiating weaning. The duration of rest required, however, is unclear. The rate of recovery from muscle fatigue may depend on the cause of the fatigue and require 15 minutes, an hour, or a day [36,38,39]. Injurious fatigue has also been described, however, in which an inflammatory infiltrate and muscle fiber necrosis

is present [40]. In this situation more than 7 days was required for a return of muscle strength to baseline levels. The best way to rest the patient is also unclear. Placing the patient on assist-control may not eliminate the respiratory work required (Fig 3), particularly if inspiratory flow rates are low or if the trigger sensitivity is high [25,41]. IMV systems may entail added inspiratory work and expiratory resistance [26,42]. Furthermore, work clearly must be done on the IMV system if the patient is required to provide a portion of the $\dot{V}e$ necessary to maintain alveolar ventilation. One practical approach is to rest the patient on an assist-control mode with adequate inspiratory flow rates and minimum trigger sensitivity until the patient is clinically stable and negative inspiratory force is stable. Whether the amount of respiratory work involved in this approach is detrimental (perpetuating fatigue) or beneficial (preventing the development of disuse atrophy) requires investigation.

Once it is clinically decided that the patient is adequately rested, and underlying factors that may be inhibiting weaning have been addressed, the process of weaning may be attempted by one of several techniques.

Weaning Procedures in the Difficult-To-Wean Patient. Minimal data are available, and controversy exists about how best to remove ventilator support by weaning. Much controversy has centered on classic T-piece weaning trials versus IMV weaning trials since 1973, when the latter was introduced as a mode of weaning by Downs [43]. To date, no controlled study has demonstrated the superiority of either IMV or classic T-piece weaning in difficult-to-wean patients. It is likely that if the same care and attention to the weaning process is carried out, however, both techniques can succeed. If muscle training to increase muscle strength and endurance is important to weaning, then the following should be implemented. First, to increase the functional capacity of a muscle the muscle must be stressed to a critical level [44]. Therefore, the amount of time the patient must be placed on a T-piece circuit or the fraction of $\dot{V}e$ the patient provides on IMV must be adequate to stress the respiratory muscles to the point of early, mild fatigue. A rest period should then follow. The best way to identify early fatigue is not known. It is known, however, that arterial blood gases are neither sensitive nor specific identifiers of muscle fatigue. $PaCO_2$ elevation is a late finding in inspiratory muscle fatigue and may even occur as a result of other causes [29, 31]. Furthermore, recovery from severe failure manifested by an acute respiratory acidosis may take 24 hours. Whether electromyographical or

transdiaphragmatic pressures are clinically useful is not known. At present one approach is to use heart rate, respiratory rate, NIF, $\dot{V}e$, and FVC [1]. Early fatigue may be indicated by an increase in heart rate, respiratory rate, and $\dot{V}e$ and a decrease in NIF and FVC. Assessment of thoracoabdominal motion for the presence of respiratory alternans and abdominal paradox may also be helpful to assess fatigue of the respiratory muscles [29].

Second, to increase a muscle's functional capacity, a progressive increase in the training stimulus is required [44]. Therefore, with T-piece weaning, the amount of time on the T-piece circuit needs to be progressively increased to find the new critical level that produces early, mild fatigue as weaning progresses. Similarly, during IMV the portion of $\dot{V}e$ that the patient has to generate needs to be progressively increased.

Third, the effect of conditioning is transient [44]. Improvements in muscle strength are reversible if the training program is interrupted or ceases. Therefore, for T-piece or IMV weaning, a progressive structured program is required. Care must be taken to ensure that the weaning schedule is not interrupted by other necessary procedures such as physical therapy, nursing care, or diagnostic procedures. Often this entails creating a schedule for the patients' daily activities. In the following section considerations relative to T-piece and IMV weaning are reviewed.

T-Piece Weaning. T-piece weaning has been reviewed in detail elsewhere [1]. In brief, before initiating the weaning process, all patients have their ventilator settings adjusted to ensure a PaO_2 of 60 to 70 mm Hg and a $PaCO_2$ that is appropriate for the patient. To facilitate transition from the ventilator to the T-piece circuit, tidal volumes on the ventilator are adjusted to approximate each patient's spontaneous tidal volume. Before being placed on the T-piece circuit, the patients' secretions are removed from the airway and endotracheal tube by suctioning. The patient is then placed in a chair or in Fowler's position in bed. Oximetry or arterial blood gases are measured to ensure that the patients' PaO_2 on the T-piece circuit are greater than 60 mm Hg. Attempts are then made to begin weaning the patients for 5 to 10 minutes without mechanical ventilatory assistance. Weaning is accomplished by slowly increasing the time periods off the ventilator with rest periods of 1 or 2 hours separating individual weaning trials. Patients are allowed to sleep while receiving mechanical ventilatory assistance from 10 P.M. until 7 A.M. until the final stages of weaning. Sleep time may be increased by decreasing the frequency of vital sign
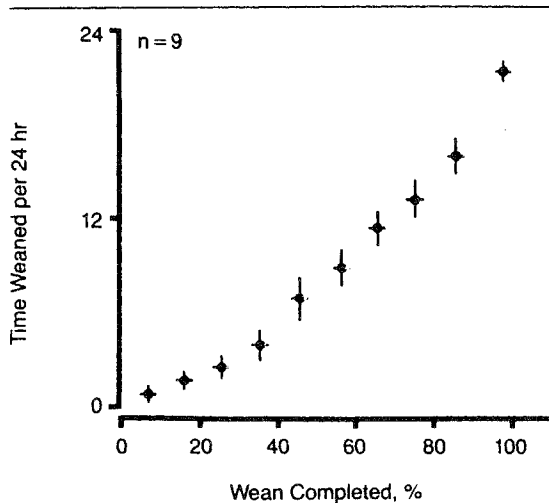
Fig 4. Shown for all patients (N = 9) is the number of hours without mechanical ventilatory assistance per 24 hours, during progressive weaning period versus percentage of wean completed. Percentage of wean completed was calculated by dividing number of days into progressive weaning period by total duration (number of days) of progressive weaning period and expressing result as percentage. (From Morganroth and associates [9]. Used with permission.)

Fig 5. The peak expiratory resistance encountered during spontaneous intermittent mandatory ventilation *(IMV)* is presented. The resistance was significantly greater with demand systems as a group ($p < 0.01$). (From KL Christopher and colleagues [26]. Used with permission.)

checks and other stimulation to the patient between 10 P.M. and 7 A.M. Sedatives are generally avoided, but if necessary those with short half-lives are used. The end point of individual weaning trials and the rate of progression of the length of the weaning trials are determined by the length of time a patient can wean before signs of early fatigue develop. Early fatigue is judged by changes in heart rate and spontaneous ventilatory measurements that occur while the patient is on the T-piece circuit. Techniques such as ambulation physical therapy, biofeedback, and patient involvement in their own care (personal hygiene, suctioning) are used in all patients to facilitate the weaning process. Weaning is considered complete when the patient is able to tolerate 24 hours without mechanical ventilatory assistance and without signs of fatigue. Using this technique 82% (9 of 11) of patients who required prolonged mechanical ventilation (lasting 30 to 100 days) were successfully weaned from the ventilator [9]. After completing a successful 24-hour wean, no patient required another period of prolonged ventilator support during the following 2 months. Although the time required to wean these patients was variable (11–43 days), the course of the progression of the wean was similar in all patients. Once progressive weaning had begun, the
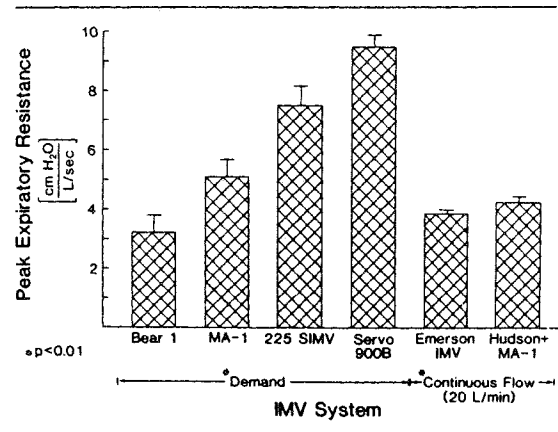
amount of time required to complete the weaning process could be predicted (Fig 4). When a patient was able to tolerate 7.1 ± 1.3 hours of a 24-hour period off the ventilator, 45 ± .5% of the total time required to wean the patient had elapsed.

IMV Weaning. The technique of weaning by IMV has been reviewed elsewhere [43,45]. Supportive measures as listed under T-piece weaning (e.g., biofeedback, physical therapy) should also be used with IMV weaning. The IMV rate is initially determined by the amount of ventilatory support required to maintain an adequate $PaCO_2$ without demonstration of fatigue. The IMV rate is then progressively decreased as tolerated by the patient. The IMV rate is usually decreased once every 24 hours in these difficult-to-wean patients. This is different than the more frequent decreases in IMV rate (every 1 to 4 hours) that are made when weaning short-term ventilation patients. The early detection of respiratory decompensation may be important as discussed under T-piece weaning. This is likely better detected by increased respiratory rate and decreased NIF rather than the development of an acute respiratory acidosis (e.g., acute increased $PaCO_2$). It also may be useful to allow the patient to rest on the ventilator at night by increasing the IMV rate.

When using an IMV system to wean a patient, it is important to note that some systems may impose increased expiratory resistance (Fig 5) and require high negative inspiratory pressures to initiate flow during spontaneous ventilation between mechani-
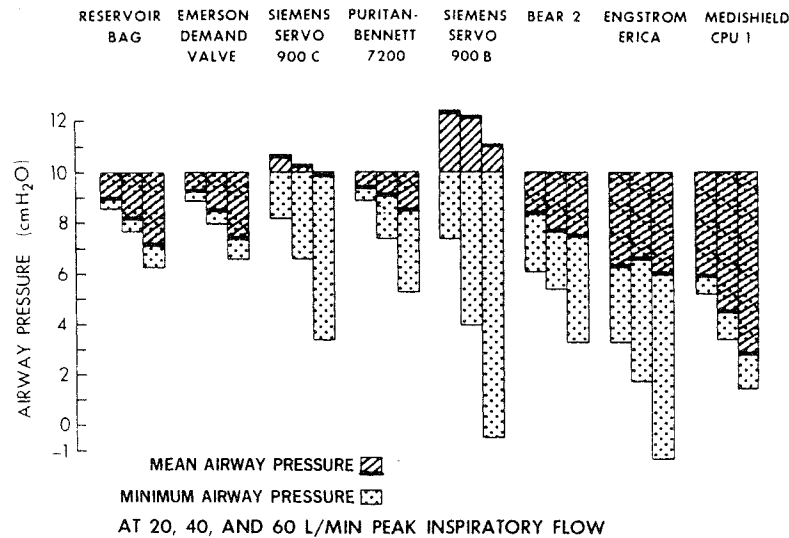
Fig 6. Mean airway pressure and minimum airway
pressure for each continuous positive airway
pressure delivery system at inspiratory flows of 20 L/
min, 40 L/min, and 60 L/min, respectively. The systems were
studied at 10 cm H_2O end-expiratory pressure. (From
Katz and associates [42]. Used with permission.)

cal breaths [26,42]. Demand flow and continuous
flow are the two types of IMV systems commonly
available to deliver airflow for the spontaneous
breaths a patient takes between machine-delivered
fixed tidal volume breaths. The demand flow sys-
tem uses a one-way valve that must be opened by
negative pressure generated by the patients' in-
spiratory effort before airflow becomes available
for spontaneous breaths. This has the potential to
allow better synchronization of the patient with the
ventilator and the possibility of decreasing inspira-
tory work if delivered flow exceeds the patients'
demand. Many of the demand systems tested, how-
ever, imposed a potentially significant added bur-
den in terms of effort required to open the demand
valve and overcome the additional resistance of the
ventilator circuit. Some demand systems [42], how-
ever, do not demonstrate added expiratory resis-
tance (e.g., Emerson demand valve, Puritan Bennett
7200) or inspiratory work (e.g., Puritan Bennett
7200, Emerson demand valve, Siemens Servo
900 C). The mean and minimum airway pressures
for the continuous positive airway pressure (CPAP)
delivery systems studied by Katz and colleagues [42]
are shown in Figure 6. The continuous flow sys-
tem delivers a constant flow of humidified gas for
spontaneous breaths between machine-delivered
breaths. This system may eliminate the increased

negative pressure required to deliver flow; how-
ever, if the flow rates are inadequate to match the
inspiratory flow demands, these systems might also
impose increased respiratory work. Thus, when an
IMV system is used to wean a prolonged ventilator
patient, it is important to understand the system
used because different systems may have the poten-
tial to inhibit or facilitate the weaning process.

Other Weaning Techniques. High-frequency jet
ventilation [6] and inspiratory resistive training
[46,47] have been used successfully to wean ven-
tilator-dependent patients. An assessment of the
utility of these techniques in the prolonged ventila-
tion patient requires controlled prospective stud-
ies. Pressure support ventilation [7] has been
successfully used to wean short-term ventilation
patients, but its use has not been reported in the
difficult-to-wean patient. External chest wall or oral
oscillation [48] are techniques that have the poten-
tial to decrease the respiratory work required by a
patient, but use of these techniques in patients be-
ing weaned from prolonged mechanical ventilation
has not been reported.

Other Considerations

Tracheostomy. Airway complications of an endo-
tracheal tube compared with a tracheostomy tube
are not different in the first 21 days [49]. The trache-
ostomy tube, however, is still the preferred airway
in long-term ventilation patients. The tracheostomy
tube allows better patient comfort, facilitates mouth
care, and permits the patient to eat and talk. In
situations where a small endotracheal tube has

been used (< 8 mm), the tracheostomy tube may result in a lower airway resistance [50].

Pharmacological Therapy. Use of steroids, theophylline, and beta$_2$ agonists may facilitate weaning if airway resistance and edema are present. Discovery of an inflammatory myopathy or myasthenia may be treatable by pharmacological intervention. If an inappropriate metabolic alkalosis is present (i.e., not compensatory for a respiratory acidosis), it may be corrected with acetazolamide, which may also result in a decrease in PaCO$_2$ [51].

Cardiac Failure Inhibiting Weaning. In patients with cardiac disease, cardiac output may decrease and pulmonary capillary wedge pressure may increase when a patient is switched to IMV or a T-piece circuit. Treatment of the underlying cardiac disease may facilitate weaning. In addition, the use of IMV with CPAP or a T-piece circuit with CPAP may prevent cardiac deterioration during weaning [35]. This technique may entail added respiratory work for the patient, however, and may be deleterious in the patient with borderline respiratory muscle function.

Nutrition. Malnutrition occurs frequently in patients receiving ventilator support [15] and can result in decreased respiratory muscle strength. Thus, it is important to provide adequate caloric intake for the patient by the enteral route or if necessary by intravenous hyperalimentation. Potential complications of this therapy, however, include increased carbon dioxide production, especially if excessive carbohydrate is used [52,53], pneumothorax, hemothorax, and catheter sepsis for intravenous hyperalimentation or the possibility of increased gastric bacterial colonization [54] and subsequent nosocomial pneumonia [55] for enteral hyperalimentation. The insertion of narrow-bore nasogastric tubes may also cause complications [56], including pneumomediastinum, pneumothorax, pleural effusion, empyema, hemothorax, and perforation of the esophagus.

General Care. Many patients die not from the underlying disease but from complications that occur during hospitalization. Catheter sepsis, urinary tract infections, nosocomial pneumonia, pulmonary embolii, gastrointestinal bleeding, drug-induced renal failure, and other iatrogenic and noniatrogenic complications contribute to mortality. Thus, it is important in patients who are likely to be in the intensive care unit for 1 to 3 months or

longer to minimize invasive procedures and to attempt to prevent the multiple complications that occur (e.g., subcutaneous heparin to prevent pulmonary emboli). Prophylactic measures used, however, may also produce complications. For example, antacids and H$_2$ antagonists decrease the incidence of gastrointestinal bleeding in ventilated patients but also increase gastric pH. The increased pH may result in increased gastric colonization and, thus, may increase the incidence of nosocomial pneumonia [55,57].

Conclusion

Most patients can be weaned from the ventilator by a variety of techniques and do not pose a clinical problem. A small fraction of patients, however, require prolonged ventilatory support. In these patients a systematic approach is required for successful weaning by whatever technique is chosen. It should also be stressed that, although these patients can be weaned from the ventilator, their ultimate outcome may be poor. In one series prolonged ventilation patients experienced a 1-year survival of only 30% (3 of 10) [9]. All patients who survived for at least 1 year were ventilated for reversible infectious processes (pneumococcal pneumonia, tuberculosis, botulism). This was less than Sivak's [10] reported 1-year survival of 73% (11 of 15 patients) but similar to the 2-year survival of 28% (11 of 39 patients) reported by Davis and associates [58] in prolonged ventilation patients. Therefore, it is important to decide if a potentially reversible process is present before instituting supportive care with mechanical ventilation.

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