Prolonged mechanical ventilation

An approach to weaning

Edward D. Sivak, M.D.

Department of Pulmonary Disease

The decision to intubate and begin mechanical ventilation in patients with acute respiratory failure is based on the observation of signs and symptoms of hypoxemia or hypercarbia or both. The decision is less thought-provoking when the patient has stopped breathing under circumstances such as cardiac arrest, drug overdose, or fatigue and retention of secretions. In less acute circumstances, the question of when and how to wean a patient from mechanical ventilation should be raised even before the patient is intubated. The two main reasons for this: (1) there are terminal diseases that can be prolonged unmercifully by instituting mechanical ventilation, and (2) knowledge of the predisposing causes of respiratory failure will determine the therapeutic approach.

The purpose of this review is threefold. The first is to describe 15 patients with respiratory failure hospitalized in the Medical Intensive Care Unit of the Cleveland Clinic. The second is to present a logical approach to weaning from mechanical ventilation, and the third is to emphasize the necessity of using a physiological approach to the problem of weaning rather than through trial and error.

Patients

All patients initially had presenting symptoms of respiratory failure and had received mechanical ventilation for at least 2 weeks before weaning or were specifically referred to the Cleveland Clinic to be weaned. Of 15 patients, eight were referred from within a 300-mile radius. Three could not be completely weaned (patients 1, 4, and 7). Eleven patients are still living; two patients died of respiratory failure 6 months and one year after initial weaning (patients 2 and 9, respectively). One patient died of septic shock subsequent to abdominal surgery 5 months after the initial weaning was completed (patient 10). Patient 1 has atrophic diaphragmatic muscles from amyotrophic lateral sclerosis and requires mechanical ventilation at home only during the hours of rest. Patient 13 required a permanent tracheostomy to aid in the clearance of secretions, and patient 7 died 11 months after initial evaluation in another hospital where he had been transferred after he could not be successfully weaned.

The diagnosis, sex, age, and preweaning arterial blood gases are listed in *Table 1*. The method of weaning, weaning time, and postweaning arterial blood gases are listed in *Table 2*. The most frequently encountered problems, which singly or in combination complicated the weaning process, are listed in *Table 3*.

Discussion

Before the question of when to discontinue ventilation can be answered, the cause of the respiratory failure must be determined. Acute respiratory failure, regardless of whether it is associated with underlying chronic respiratory distress, can be divided into the three categories with respective subdivisions listed below.[1]

1. Ventilatory failure with normal lungs. (a) Insufficient respiratory center function (metabolic alkalosis, drugs, cerebrovascular accident, old bulbar poliomyelitis, blunted response to CO_2). (b) Insufficient chest wall function (neuromuscular disease—polymyositis, acid maltase deficiency, myasthenia gravis,

Table 1. Diagnosis and preweaning arterial blood gases

				Preweaning arterial blood gases				
Patient	Diagnosis	Sex	Age	FiO_2	pH	HCO_3	pCO_2	pO_2
1	Amyotrophic lateral sclerosis	M	57	.40	7.38	39	63	77
2	Amyotrophic lateral sclerosis	M	72	.60	7.50	32	44	53
3	Amyotrophic lateral sclerosis	M	69	.35	7.48	26	37	76
4	Diaphragmatic paralysis	F	61	.30	7.38	27	47	110
5	Polymyositis	F	40	.30	7.45	27	40	101
6	COPD (pontine CVA)	M	56	.35	7.40	28	47	108
7	Eaton-Lambert syndrome	M	74	.35	7.47	28	41	106
8	COPD (metabolic alkalosis)	F	62	.40	7.38	46	78	80
9	COPD (metabolic alkalosis)	M	65	.55	7.45	33	49	63
10	COPD (metabolic alkalosis)	F	63	.25	7.43	32	50	77
11	COPD	M	61	.35	7.35	31	58	105
12	COPD	M	63	.40	7.53	33	40	75
13	COPD	M	59	.35	7.50	26	35	67
14	Bullous emphysema	F	54	.45	7.50	33	43	65
15	Bullous emphysema	F	40	.30	7.42	37	59	52

COPD = chronic obstructive pulmonary disease, CVA = cerebrovascular accident.

Table 2. Method and duration of weaning and postweaning arterial blood gases

Patient	Method	Duration, days	Postweaning arterial blood gases				
			FiO_2	pH	HCO_3	pCO_2	pO_2
1	Graduated	20	3 L/min	7.39	26	45	84
2	Graduated	66	3 L/min	7.37	32	56	88
3	Graduated	17	4 L/min	7.43	23	36	70
4	Expiration	...	...	...	...	...	...
5	Graduated	150	0.30	7.42	29	46	99
6	Graduated	25	0.21	7.43	27	42	52
7	Not weaned	...	0.21	7.50	25	33	66
8	Graduated	20	0.21	7.44	28	43	59
9	Graduated	5	4 L/min	7.40	29	47	80
10	Graduated	17	2 L/min	7.48	28	40	57
11	Graduated	5	0.21	7.43	29	45	69
12	Graduated	35	0.21	7.41	26	43	73
13	Graduated	1	2 L/min	7.51	24	32	56
14	IMV	9	2 L/min	7.37	26	46	69
15	IMV	12	0.28	7.43	32	49	76

IMV = intermittent mandatory ventilation.

Table 3. Problems complicating the weaning process

Problem*	Number	Percent
Excess secretions	7	47
Obscure cause of respiratory failure	5	33
Metabolic alkalosis	3	20
Malnutrition	3	20
Severe bullous disease	1	7
Gastrointestinal bleeding	1	7

* Either singly or in combination.

myasthenic syndrome, amyotrophic lateral sclerosis, Guillain-Barré syndrome, trauma with unstable chest wall, kyphoscoliosis, obesity).

2. Primarily failure to excrete CO_2 (chronic obstructive lung disease, asthma, cystic fibrosis).

3. Primarily failure to oxygenate blood (adult respiratory distress syndrome of various causes, cardiogenic pulmonary edema, end-stage fibrotic lung disease).

The decision on when to discontinue mechanical ventilation should be made when the inciting process that caused the respiratory failure has reversed to the point where the patient can begin to become self-supporting. This decision should be based on the presence of (1) adequate respiratory drive, (2) sufficient respiratory muscle strength and endurance, and (3) sufficient lung parenchyma for adequate gas exchange. All too frequently, however, it is difficult to make an objective physiologic evaluation at the bedside, necessitating more practical bedside evaluation.

Adequate respiratory drive. The normal drive to breathe is mediated by the stimulus from hydrogen ions in the cerebrospinal fluid surrounding the medullary chemoreceptors. The stimulus to breathe can be severely blunted in metabolic alkalosis.[2, 3] Thus, usually a patient should have a normal acid-base and electrolyte picture before mechanical ventilation is discontinued. If a patient has acute respiratory failure superimposed upon underlying obstructive lung disease, all efforts should be made to rule out precipitating causes that alter the normal drive to breathe. In patients 8, 9, and 10, metabolic alkalosis blunted the central drive, and in patient

6, localized edema in the area of the brainstem most likely blunted the normal respiratory drive. The initial assumption in each of these cases was that the underlying obstructive lung disease was associated with such severe ventilation perfusion deficits that the patients' baseline conditions were associated with carbon dioxide retention.[4] In each of these patients, minute ventilation was increased to maintain a mild metabolic alkalosis (pH 7.45 to 7.50), thus allowing for a gradual renal excretion of bicarbonate and correction of the blood pH. Other problems such as oversedation, primary alveolar hypoventilation,[5] old bulbar poliomylitis,[6] vasculitis, hypothyroidism,[7] and cerebrovascular accident[8] should be considered.

Sufficient respiratory muscle strength. During the course of clinical evaluation, careful observation for signs of neuromuscular disease is mandatory. Proximal muscle weakness and atrophy should raise the question of polymyositis, underlying malignancy, and malnutrition. Fasciculations suggest motor neuron disease. Progressive weakness that becomes worse with activity suggests myasthenia gravis, and weakness that improves with activity suggests the myasthenia syndrome described by Eaton and Lambert.[9]

The normal motion of the diaphragm on inspiration pushes the anterior abdominal wall outward. A paradoxical inward motion on inspiration suggests diaphragmatic paralysis or weakness from motor neuron disease,[10-12] polymyositis, acid maltase deficiency,[13] idiopathic diaphragmatic paralysis,[14] and, rarely, myasthenia gravis.[15] The clinician must be careful in making the diagnosis of idiopathic diaphragmatic paralysis because any of the above-mentioned conditions can masquerade as diaphragmatic paralysis. For this reason, any patient who has paradoxical motion of the abdomen on inspiration should have electromyographic studies, and appropriate muscle biopsy should be obtained to elucidate the cause of diaphragmatic dysfunction.

Other mechanical problems such as ankylosing spondylitis, severe kyphoscoliosis,[16] chest wall trauma, or old thoracoplasty alter the efficiency of ventilation, which eventually deteriorates to the point where the work of breathing produces more carbon dioxide and consumes more oxygen than the patient is capable of tolerating. Under such circumstances, respiratory failure is the end result. My experience with three such patients is that they functioned well without mechanical ventilation when they were awake. They required ventilation when sleeping, but were relatively symptom-free during the daytime.

When assessing the condition of a patient with myasthenia gravis, particularly in the immediate postoperative period after thymectomy, it is important to be aware of the patient's response to anticholinesterase medications. The patient may have adequate ventilatory parameters, but the condition may deteriorate during the next 8 to 24 hours or between doses of these medications. For this reason, it is advisable that the patient have adequate stable parameters for at least 24 hours before extubation.

When assessing respiratory muscle strength, objective data should be obtained: the work of breathing (the movement of air into the lungs) and the muscles required to do this work (muscles of inspiration). Excess secretions in the airways or a decrease in lung compliance due to atelectasis require increased work. Sufficient respiratory muscle strength is present when the patient can generate a vital capacity of

greater than 12 to 15 ml/kg and the negative inspiratory force is greater than 25 cm of water.[17-19]

Sufficient lung parenchyma. A common problem with gas exchange in the patient recovering from respiratory failure is ventilation perfusion mismatching. This is caused by excess secretions and less commonly by air trapping. Air trapping was so severe in patient 15 that it was necessary to perform a left upper bullectomy before she could be weaned. In each of the cases in which air trapping was a problem, the pCO_2 remained elevated despite adequate minute volume (15 ml/kg) delivered by the ventilator.[4] In each case this was confirmed by large discrepancies between the inhaled minute volume and the exhaled minute volume. This was alleviated by an expiratory pause, low levels of positive end-expiratory pressure and/or vigorous chest physical therapy and postural drainage to remove secretions from small airways. It is imperative that the patient receive enough chest physical therapy and postural drainage to keep the airways patent, thus minimizing the work of breathing.

In the debilitated state, a patient often becomes dependent on a respirator. Respirator muscles become weak and may not permit adequate tidal volume. A patient may often breathe rapidly, but tidal volumes may be no larger than anatomical dead space (1.0 cc/0.45 kg). This type of respiratory pattern effectively reduces alveolar ventilation. A high dead space to tidal volume ratio has been shown to make weaning almost impossible.[20, 21] For this reason, regardless of which method is used to wean the patient, the process should not be started until spontaneous tidal volumes are in excess of 100% of the calculated anatomical dead space of 1 cc/pound (see below).

The frequency of ventilation is also an important factor, particularly when the patient becomes anxious during the weaning process or is under sufficient stress from pain, infection, or postoperative recovery. Ordinarily, the compliance of the lung is equal in the static as well as the dynamic (during breathing) state.[22] However, in all patients in the present series, and for most practical purposes, in the majority of patients who require respiratory support, there are underlying abnormalities that cause differences between static and dynamic compliance.[22] This frequency dependency of compliance is responsible for the abnormalities in gas exchange, which are found when the frequency of respirations increases.[23] This can be objectively demonstrated by a fall in pO_2, a rise in pCO_2, and an increase in the dead space to tidal volume ratio. Compliance itself can be improved by adding small amounts of end expiratory pressure without additional support from the ventilator.[24] The improved compliance lessens the work of breathing and the end-expiratory pressure can lessen the chance of microatelectasis development.

If the disease process is associated with diffuse five-lobe infiltrates on chest roentgenogram, the underlying process causing the problem should be reversed before any weaning process is started. In addition, it is wise to allow the lung to repair the underlying damage to the point where an arterial oxygen tension can be maintained above 60 mm Hg with no end-expiratory pressure and an FiO_2 of less than 0.40. The underlying abnormalities that make gas exchange fall short of this goal are manifested as a widened alveolar-arterial oxygen gradient, and increased venous admixture. Failure to meet these minimum standards implies that sufficient time or treat-

ment has not been afforded to the patient or that end-stage lung disease is present.[25]

Criteria for discontinuing ventilation. Putting theoretical discussion aside, the following criteria should be considered when mechanical ventilation is discontinued.

1. Adequate ventilatory drive to maintain a stable pCO_2 less than 45 mm Hg when placed on "flush." In this respect, it should be kept in mind that severe ventilation-perfusion abnormalities can be associated with a pCO_2 greater than 45 mm Hg, but if the chemical drive to breathe (response to CO_2) is normal, the patient will feel the sensation of dyspnea. If a blunted response to CO_2 is suspected, the underlying cause should be determined and corrected if possible. If it cannot be corrected, the pO_2 should be kept between 55 and 65 mm Hg with or without supplemental oxygen to allow the stimulus of hypoxemia to maintain ventilation.

2. Sufficient respiratory muscle strength to maintain a vital capacity between 12 and 15 ml/kg. This strength is equal to the ability to generate a negative inspiratory occlusion pressure greater than negative 25 cm of H_2O.

3. Sufficient lung parenchyma to allow the pO_2 to be greater than 60 mm Hg on 40% supplemental oxygen or less without ventilatory assistance.

The physician should be familiar with the method used to wean a patient. The two basic methods are graduated periods of time off the ventilator with the patient connected to a "T-piece" and intermittent mandatory ventilation (IMV).[26,27] Neither method has been proved to be superior to the other. In my experience, the graduated method seems to be more effective than IMV in cases of blunted respiratory drive, neuromuscular disease, and prolonged ventilation due to debilitating disease. In each of these situations IMV tends to allow the patient to accommodate to less minute ventilation, retain carbon dioxide, and develop compensatory metabolic alkalosis, which blunts respiratory drive. This is evidenced by the fact that patients 1 through 13 were weaned by the graduated method only after unsuccessful trials of IMV. With prolonged mechanical ventilation, the respiratory muscles become weakened and thus when the weaning process is instituted, the patient's respiratory muscles should be stressed to allow for improvement in strength. This cannot always be done if the patient's stimulus to use the respiratory muscles is decreased by the presence of normal blood gases or if the muscles move only an amount of air equal to anatomic dead space. If one wishes to give maximum stimulation to respiratory muscles, then the graduated method should be used. The patient must be returned to assisted ventilation if the pulse increases more than 25% or if bradycardia develops. Diaphoresis is another sign associated with hypercarbia and hypoxemia and can occur without associated tachycardia, especially in the elderly. These signs are due to increased sympathetic overtone and hypoventilation, respectively. Ideally, it is preferable to use a combination of IMV and the graduated method.

IMV has proved to be convenient in weaning patients who require short-term ventilation. For the patient with a normal drive to breathe and good respiratory muscle strength, it seems to be superior to the graduated method only in terms of allowing the personnel within the intensive care unit the comfort of knowing that the patient is receiving some additional ventilation during the weaning process. These com-

ments are based only on my personal experience. However, to date, there is no published study that can substantiate any other claim. This study should ideally contain matched pairs of patients with similar diseases weaned with one method or the other. Further investigation is thus in order.

If a blunted respiratory drive, neuromuscular disease, or chest wall restriction is a problem, some type of assisted ventilation would be indicated. Further consideration should be given to using ventilation during the hours of rest so that a reasonable life-style can be maintained during the waking hours. Such assisted ventilation can be accomplished by (1) diaphragmatic pacemakers in patients who are quadriplegic or have abnormal respiratory drives,[28, 29] (2) pneumobelt,[30] (3) oscillating bed, and (4) ventilator.

Each of these methods of ventilation is associated with various advantages and disadvantages. When considering the patient for home care ventilation, it is usually wise to consult an individual with experience, because an almost monumental effort is required to achieve satisfactory home care. However, once accomplished, the results are extremely gratifying for both the patient and the physician. This is the case with patient 1, who has been assisted by ventilation at night for the past 2 years at home. During the day he is able to support his own ventilation and has continued with some of the manual chores on the farm.

Summary

The process of weaning a patient from mechanical ventilation is nothing more than respiratory failure in reverse. Each patient must be approached with knowledge of the inciting cause of the respiratory failure. When the more apparent causes are reversed, the clinician should look for more unusual causes of respiratory failure if the patient is not progressing toward maintaining spontaneous ventilation. The more obscure causes usually are due to neurological diseases that have subtle clinical findings. Under such circumstances, neurological consultation can be most helpful.

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