REVIEW ARTICLES

CURRENT CONCEPTS

MECHANICAL VENTILATION

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THAT positive-pressure mechanical ventilation can save lives was proved during the poliomyelitis epidemics of the 1950s.[1] Since that time there has been a growing increase in the use of ventilatory support, and it has been closely associated with the development of critical care medicine. Early ventilators were used in conjunction with neuromuscular blocking agents to provide controlled ventilation. Today, most machines are triggered by the patient, and there is growing awareness of the complexity of patient–ventilator interaction.[2-4] There is also increasing recognition that ventilators can induce subtle forms of lung injury,[5] which has led to a reappraisal of the goals of ventilatory support.[6] With advances in computer and electronic technology, ventilators have changed markedly in appearance, and there is an array of options that is increasingly intimidating.[7,8] However, the fundamental principles of ventilatory treatment of the critically ill patient remain unchanged, although there are several new nuances in their application.

OBJECTIVES

The objectives of mechanical ventilation are shown in Table 1. Positive-pressure ventilation can be lifesaving in patients with acute severe hypoxemia or worsening respiratory acidosis (or both) that is refractory to more conservative measures. In patients with severe cardiopulmonary distress for whom the effort of breathing is intolerable, mechanical ventilation substitutes for the action of the respiratory muscles. In some patients the respiratory muscles account for as much as 50 percent of total oxygen consumption.[9] In such circumstances, mechanical ventilation allows precious stores of oxygen to be rerouted to other tissue beds that may be vulnerable. In addition, the reversal of respiratory-muscle fatigue, which may have a role in the development of acute ventilatory failure, depends on adequate rest of the respiratory muscles. Positive-pressure ventilation can reverse and prevent atelectasis, and by allowing inspiration at a more compliant region of the pulmonary pressure–volume curve, it can decrease the work of breathing.[10] Improvements in pulmonary gas exchange and pressure–volume relations and relief from excessive respiratory work provide an opportunity for the lungs and airways to heal. However, positive-pressure ventilation can also decrease cardiac output and initiate or aggravate alveolar damage. The dangers of ventilator-induced lung injury have led to a reappraisal of the objectives of mechanical ventilation.[6] Rather than strive for normal arterial-blood gas values, it is probably better to accept a certain degree of respiratory acidosis, and possibly even hypoxemia, to avoid high inflation pressures. Likewise, it may be better to risk oxygen toxicity than to use high pressures to achieve a decrease in the fraction of inspired oxygen (FiO_2).

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VENTILATOR MODES AND SETTINGS

Because controlled ventilation with the abolition of spontaneous breathing rapidly leads to atrophy of respiratory muscles,[11] assisted modes that are triggered by the patient's inspiratory efforts are preferred. The most common triggered modes are assist-control ventilation, intermittent mandatory ventilation, and pressure-support ventilation.[4,12] With assist-control ventilation, the ventilator delivers a breath either when triggered by a patient's inspiratory effort or independently if such an effort does not occur within a preselected period. With intermittent mandatory ventilation, the patient receives periodic positive-pressure breaths from the ventilator at a preset volume and rate, and (unlike the situation with assist-control ventilation) spontaneous breathing is also allowed. Spontaneous breathing is achieved by means of a demand valve that can considerably increase the work of breathing.[2] Pressure-support ventilation differs from assist-control ventilation and intermittent mandatory ventilation in that the physician sets a level of pressure (rather than volume) to augment every spontaneous effort.[13,14] Airway pressure is maintained at a preset level until the patient's inspiratory flow falls below a certain level (e.g., 25 percent of peak flow). Tidal volume is determined by the level of pressure set, the patient's effort, and pulmonary mechanics.

The settings of the ventilator are based on the patient's size and condition, and they require repeated reassessment. The risk of toxic effects of oxygen is minimized by using the lowest FiO_2 with which satisfactory arterial oxygenation can be achieved. The usual goal is an arterial oxygen tension (PaO_2) of 60 mm Hg or an oxygen saturation of 90 percent, since higher values do not substantially enhance tissue oxygenation.

Over the past 20 years, it has been customary to use tidal volumes (10 to 15 ml per kilogram of body weight) that are two to three times higher than nor-

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Table 1. Objectives of Mechanical Ventilation.

Improve pulmonary gas exchange
Reverse hypoxemia
Relieve acute respiratory acidosis
Relieve respiratory distress
Decrease oxygen cost of breathing
Reverse respiratory-muscle fatigue
Alter pressure–volume relations
Prevent and reverse atelectasis
Improve compliance
Prevent further injury
Permit lung and airway healing
Avoid complications

mal. This approach is being challenged by convincing data from experiments indicating that alveolar overdistention can produce endothelial, epithelial, and basement-membrane injuries that are associated with increased microvascular permeability and lung rupture.[5,15,16] To minimize this risk, one would ideally like to monitor alveolar volume, but this is not feasible. A reasonable substitute is to monitor the peak alveolar pressure as estimated from the plateau pressure, which is measured in a relaxed patient by briefly occluding the ventilatory circuit at end-inspiration.[17] The incidence of ventilator-induced lung injury increases markedly when the plateau pressure is high. Although data are incomplete, there is a growing tendency to lower the tidal volume delivered to 5 to 7 ml per kilogram (or less) in order to achieve a plateau pressure no higher than 35 cm of water. Since this may lead to an increase in the arterial carbon dioxide tension ($PaCO_2$), the strategy is termed permissive hypercapnia or controlled hypoventilation.[18] It is important to focus on the pH rather than the $PaCO_2$ when using this approach. If the pH falls below 7.20, some physicians recommend intravenous bicarbonate, but this is of unproved benefit and a subject of controversy.[18] In patients with severe asthma requiring mechanical ventilation, uncontrolled studies suggest that permissive hypercapnia results in lower mortality than conventional ventilation,[19,20] and the same may be true in patients with the adult respiratory distress syndrome, although the documentation is less complete.[21]

The rate of ventilation that is set depends on the mode. With assist-control ventilation, the backup rate should be about four breaths per minute less than the patient's spontaneous rate; this ensures that the ventilator will continue to supply an adequate volume should the patient have a sudden decrease in output from the respiratory centers. With intermittent mandatory ventilation, the rate should be high at first and then decreased gradually in accordance with the patient's tolerance. With pressure-support ventilation, the rate is not set.

Most ventilators are triggered by a change in airway pressure, and their sensitivity is usually set at -1 to -2 cm of water. With poorly responsive demand valves, however, the actual pressure generated by the patient may be considerably higher.[2,22] If the trigger setting is too sensitive, the ventilator will cycle too frequently ("autocycle"), and severe respiratory alkalosis may result. Flow triggering is available with some ventilators, and it appears to require less work by the patient than pressure triggering.[23] In some patients, especially those with chronic obstructive pulmonary disease and a high minute ventilation, gas trapping develops in the alveoli, and the patients have a positive end-expiratory pressure (PEEP).[24,25] This so-called auto-PEEP makes triggering the ventilator more difficult, since the patient needs to generate a negative pressure equal in magnitude to the level of auto-PEEP in addition to the level of sensitivity selected.[25] This is one of the factors that may account for a patient's inability to trigger the ventilator despite obvious respiratory efforts.[22] Auto-PEEP commonly goes undetected, because it is not registered on the pressure manometer of the ventilator, since the latter is open to the atmosphere. Occluding the expiratory port of the circuit at the end of expiration in a relaxed patient causes the pressure in the lungs and circuit to equilibrate, and the level of auto-PEEP will be displayed on the manometer.[24]

An inspiratory-flow rate of 60 liters per minute is used with most patients during assist-control ventilation and intermittent mandatory ventilation. In patients with chronic obstructive pulmonary disease, better gas exchange is achieved at a flow rate of 100 liters per minute, probably because the resulting increase in expiratory time allows more complete emptying of gas-trapped regions.[26] If the flow rate is insufficient to meet a patient's ventilatory requirements, the patient will strain against his or her own pulmonary impedance and that of the ventilator, with a consequent increase in the work of breathing.[3,27] It is helpful to examine the contour of the wave form for airway pressure when the flow rate and the sensitivity of the trigger are adjusted (Fig. 1). Ideally, the wave form should show a smooth rise and a convex appearance during inspiration. In contrast, a prolonged negative phase with excessive scalloping of the tracing indicates unsatisfactory sensitivity and flow settings.[3,27]

Few aspects of ventilatory management have been more controversial than PEEP.[28,29] In patients with the adult respiratory distress syndrome, PEEP usually produces a substantial increase in PaO_2. This is primarily due to a reduction in intrapulmonary shunting as a result of a redistribution of lung water from the alveoli to the perivascular interstitial space.[30] Contrary to previous thinking, PEEP does not decrease the total amount of extravascular lung water. Provided the improvement in PaO_2 is not offset by a decline in cardiac output, FiO_2 can be decreased, which is the principal therapeutic effect of PEEP. The addition of PEEP also influences lung mechanics. Patients with acute lung injury commonly have a decreased end-expiratory lung volume, and thus tidal breathing occurs on the low, flat portion of the pressure–volume curve. By shifting tidal breathing to a more compliant

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portion of the curve, PEEP can reduce the work of breathing.[10] In patients with an airflow limitation and auto-PEEP who have difficulty triggering the ventilator, the addition of external PEEP (to a level not exceeding the level of auto-PEEP) can help to counteract this problem, since to trigger the ventilator the alveolar pressure needs to be decreased only below the level of external PEEP, rather than below zero.[31,32] In addition to the injury induced by high inflation pressures, mechanical ventilation at a low end-expiratory lung volume aggravates lung injury in laboratory animals, possibly because of shear stresses associated with the repeated closing and opening of alveolar units.[5,6,33,34] Adding about 10 cm of water of PEEP to splint the lung open appears to reduce this problem. It is not known whether this type of shear injury occurs in patients.

Selecting the mode and settings of the ventilator is a dynamic process that is based on a patient's physiologic response rather than a fixed set of numbers. The settings require repeated readjustment over the period of dependency on the ventilator, and such iterative interaction requires careful respiratory monitoring. The key variables in monitoring, most of which can easily be measured at the bedside, are shown in Table 2.[17] In complex situations, such as the case of patients with the adult respiratory distress syndrome who require high levels of PEEP, a pulmonary-artery catheter may be required to help titrate fluid or vasopressor therapy. Monitoring also helps in showing how soon ventilatory support can be discontinued.[11]

Alternative Methods

New methods of ventilatory support are constantly being introduced. These include high-frequency ventilation, inverse-ratio ventilation, airway-pressure-release ventilation, proportional-assist ventilation, and extracorporeal carbon dioxide removal.[6,12] Although these techniques are exciting from a theoretical and physiologic viewpoint, there is no firm evidence that they improve outcomes in patients.

There is also a resurgence of interest in the delivery of positive-pressure ventilation with face or nasal masks. This approach is particularly helpful in the long-term treatment of patients with chest-wall and neuromuscular disorders.[35,36] It can also improve pulmonary gas exchange rapidly in patients with acute ventilatory failure.[35,36] As compared with conventional treatment in patients who have an acute exacerbation of chronic obstructive pulmonary disease, noninvasive ventilation may reduce the need for endotracheal intubation[37] and may decrease mortality.[36] Patients need to be carefully selected for this therapy, since the mask can cause claustrophobia, and it may be dangerous in patients who cannot protect their airways or who have copious secretions. In addition, this approach can be very time-consuming for nurses and respiratory therapists.[38] Although noninvasive ventilation is very promising, carefully controlled studies are needed to define the appropriate indications and contraindications, the optimal duration of therapy, and the morbidity and mortality associated with this approach as compared with standard mechanical ventilation and endotracheal intubation.

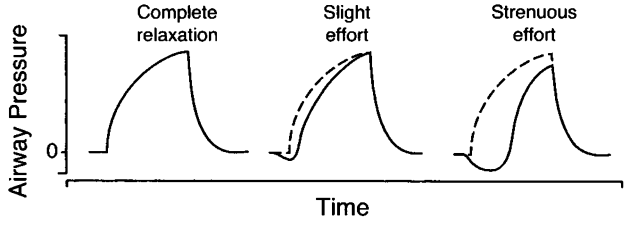

Figure 1. Airway-Pressure Wave Forms Recorded during Assist-Control Ventilation.

The tracings represent changes in airway pressure during inspiration in a completely relaxed patient and in patients making a slight effort and a strenuous effort to breathe. The distance between the dashed line (representing controlled ventilation) and the solid line (representing spontaneous breathing) is proportional to the patient's work in breathing.

Table 2. Variables Used in the Monitoring of Patients Receiving Mechanical Ventilation.

Gas exchange
Arterial oxygen tension or saturation
Arterial carbon dioxide tension and pH
Airway pressure
Peak inspiratory pressure
Plateau (end-inspiratory occlusion) pressure
PEEP, external and auto
Wave form of pressure
Breathing pattern
Minute ventilation
Tidal volume
Respiratory frequency
Hemodynamic function
Blood pressure
Urinary output
Cardiac output
Pulmonary-artery occlusion pressure
Chest film
Endotracheal-tube position
Signs of barotrauma
Signs of pneumonia

Adjunctive Therapy

Mechanical ventilation can be an uncomfortable and frightening experience, and the patient needs constant reassurance. The development of sudden distress in a previously calm patient ("fighting the ventilator") suggests the occurrence of a new and potentially serious complication.[39] The ventilator should be disconnected and the patient given manual ventilation with an FiO_2 of 1.0 while a systematic search is undertaken for the cause. If the distress is due to poor coordination of a patient's respiratory efforts with the rhythm of the ventilator, the problem can usually be resolved by careful adjustment of the ventilator settings plus the administration of analgesic and sedative agents. In some instances, a neuromuscular blocking agent may be needed. The duration of such blockade should be kept to a minimum because of the catastrophic consequences of accidental disconnection from the ventilator. In addition, recent reports indi-

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cate that as many as 70 percent of patients with certain characteristics may have prolonged weakness after the discontinuation of these agents.[40]

Most acute parenchymal processes affect the lung in a nonhomogeneous manner.[41] As a result, alterations in posture can markedly influence gas exchange.[42,43] A trial-and-error approach to body positioning — in particular, switching from the supine to the prone position — can produce substantial improvement in oxygenation. Changing a patient's posture from supine to semirecumbent also lowers the risk of aspiration of gastric contents.[44]

Problems in the circuit of the ventilator, such as a narrow endotracheal tube, a poorly functioning demand valve, certain characteristics and locations of the humidifier, and valves with high expiratory resistance, can markedly increase the work of breathing and predispose patients to barotrauma.[2,45,46] To avoid such problems, the plumbing of the circuit needs to be evaluated repeatedly by a knowledgeable person. In addition, adequate humidification and suctioning are needed to prevent secretions from blocking the tracheal tube or producing atelectasis. General aspects of patient care include oral hygiene, nutritional supplementation, the administration of bronchodilators, and measures to prevent thromboembolism and decubitus ulcers.

Avoidance of Complications

Modifying the ventilator settings and the therapy to minimize the risk of complications is a constant goal in the care of patients who are dependent on mechanical ventilation. Careful monitoring is required in order to identify patients at risk and detect the earliest evidence of a problem.[17] Patients should be evaluated repeatedly with regard to the possibility of discontinuing mechanical ventilation, since complications are generally related to the duration of support.[47] Table 3 provides an abbreviated list of the major complications.

When the pressure in the cuff of a tracheal tube exceeds the critical pressure for perfusion of the tracheal mucosa (about 25 mm Hg), serious injury becomes likely. To minimize this problem, the lowest possible cuff pressure needed to achieve a seal should be used (preferably <15 mm Hg).[48] Barotrauma in the form of pneumothorax, pneumomediastinum, or subcutaneous emphysema occurs in 10 to 20 percent of patients receiving mechanical ventilation.[49,50] The term "barotrauma" is probably a misnomer, since alveolar overdistention appears to be the primary mechanism rather than high peak airway pressure.[51] The classic apicolateral location of a pneumothorax is less common in patients receiving mechanical ventilation (largely because of their posture), with the result that the diagnosis is often missed.[52] Positive-pressure ventilation usually lowers cardiac output, primarily as a result of decreased venous return (Fig. 2).[53] Conversely, this form of ventilation can increase cardiac output in patients with impaired myocardial contractility[54] because there is a decrease in left ventricular afterload due to increased intrathoracic pressure. Nosocomial pneumonia occurs in about 30 percent of patients receiving mechanical ventilation and is associated with a more than twofold increase in mortality.[47,55] The risk increases with the duration of ventilatory support, at a rate of about 1 percent per day.[47]

Table 3. Complications of Mechanical Ventilation.

Toxic effects of oxygen
Endotracheal-tube complications
Laryngeal injury
Tracheal stenosis
Tracheomalacia
Endobronchial intubation
Sinusitis
Volume-induced alveolar injury
Barotrauma
Decreased cardiac output
Pneumonia
Psychological problems

Discontinuation of Ventilatory Support

Although the discontinuation of mechanical ventilation is easy in patients requiring short-term support, it can be quite difficult in patients recovering from a

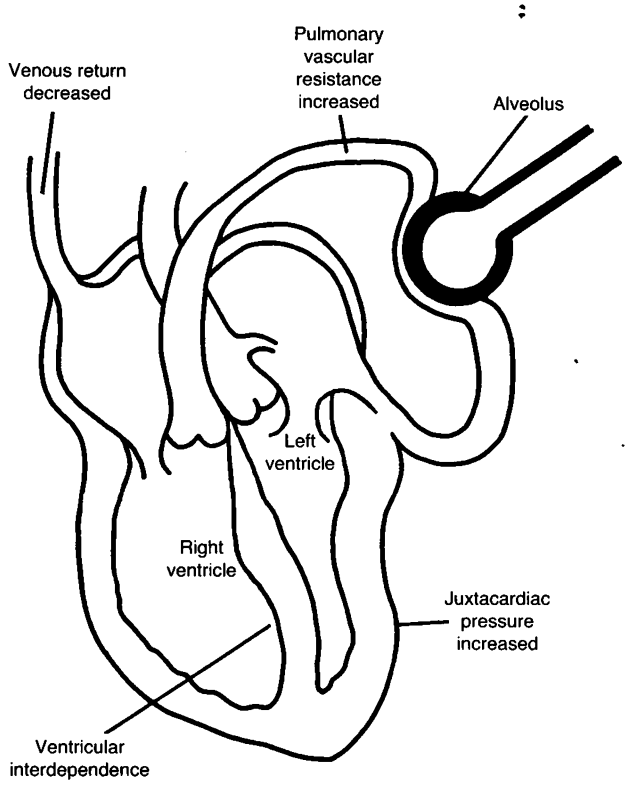

Figure 2. Factors Responsible for the Decrease in Cardiac Output during Positive-Pressure Ventilation.

An increase in intrathoracic pressure compresses the vena cava and thus decreases venous return. Alveolar distention compresses the alveolar vessels, and the resulting increases in pulmonary vascular resistance and right ventricular afterload produce a leftward shift in the interventricular septum. Left ventricular compliance is decreased by both the bulging septum and the increased juxtacardiac pressure resulting from distended lungs.

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major episode of acute respiratory failure.[11] Weaning such patients from the ventilator is a major clinical challenge and constitutes a large portion of the workload in an intensive care unit. The initiation of the weaning process requires careful timing. If it is delayed unnecessarily, the patient remains at risk for ventilator-associated complications.[47,51] If it is performed prematurely, severe cardiopulmonary decompensation may further delay extubation.

In general, the discontinuation of mechanical ventilation is not contemplated in a patient with cardiopulmonary instability or a PaO_2 of less than 60 mm Hg with an FiO_2 of 0.40 or higher. However, satisfactory oxygenation does not reliably predict successful weaning. Instead, the outcome of a weaning trial is more commonly determined by the ability of the respiratory muscles to cope with an increased respiratory workload.[56,57] Measurements of maximal inspiratory pressure, vital capacity, and minute ventilation have traditionally been used to judge a patient's readiness for weaning, but they have limited predictive accuracy.[11,58] The ratio of respiratory frequency to tidal volume during one minute of spontaneous breathing appears to be a more accurate predictor[58]; a value of less than 100 breaths per minute per liter indicates that weaning is likely to be successful. Indexes such as this serve only as a guide, however, and do not replace the need for careful clinical assessment.

Weaning techniques include trials of spontaneous breathing through a T-tube circuit and gradual reductions in the level of intermittent mandatory ventilation or pressure-support ventilation. Although there is considerable controversy about relative merits, the intrinsic superiority of any one technique has not been clearly demonstrated, and the manner in which a technique is used is likely to be more important. Finally, a patient's ability to protect the upper airway and clear secretions must be evaluated before extubation.

In conclusion, advances in biomedical engineering have resulted in better ventilators and improved methods of monitoring patients, and the goals and hazards of mechanical ventilation are better defined. Mechanical ventilation saves lives but can result in serious complications, and thus it should be discontinued at the earliest possible time.

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