Initiating Mechanical Ventilation

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Mechanical ventilation is a central facet of intensive care medicine. Technological progress has led to major advances in this field. Initiation of mechanical ventilation remains standard; however, many options are now available to continue mechanical ventilation. These mechanical options include assist-control ventilation, intermittent mandatory ventilation, pressure support ventilation, positive end-expiratory pressure, and high-frequency ventilation. The sophistication of ventilators has also allowed more procedures to be performed during mechanical ventilation, including bronchoscopy and transbronchial biopsy. Complications do occur during mechanical ventilation, but can be minimized by vigilance and use of proper procedures. The recent increase in home care will lead to increased use of alternatives to conventional ventilatory modes. Finally, more well-designed studies are needed as newer techniques are promoted.

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Although rooted in the past, the concept of physically separate intensive care units and the emergence of intensive care as a distinct subject area has proliferated only over the past 20 years. A common ground in all types of intensive care has been the care and management of the patient receiving mechanical ventilation. Perhaps the driving force of the intensive care movement has been the advancement of technological support, of which mechanical ventilation has been in the forefront. We will review this central facet of intensive care medicine, with emphasis on recent developments and current opinion, in two articles. Initiation of mechanical ventilation, mechanical options, procedures, complications, and alternatives to conventional ventilation are covered in this article. A companion article, "Weaning from Mechanical Ventilation," will appear in the next issue of *Journal of Intensive Care Medicine.*

Mechanical ventilation can be loosely defined as the use of any mechanical device that provides either the total or a partial amount of the gas exchange needed to sustain life. There are references to mechanical devices dating back to the 16th century. However, extensive and efficacious clinical use of mechanical ventilation has only occurred in the 20th century. Mechanical devices can be classified as those that exchange gas by external negative pressure and those that deliver positive pressure internally. In the period from the late 1920s to the 1950s negative pressure ventilation came into widespread use. The iron lungs and the cuirasses of this generation were used primarily during the polio epidemics. The modern era of mechanical ventilation features predominantly positive-pressure ventilators. These ventilators have been used for patients with an increasing variety of illnesses since the development of intensive care units in the 1960s and 1970s.

Positive-pressure ventilation, which is the standard of mechanical ventilation today, can be classified into volume-preset and pressure-preset

categories. The pressure-preset ventilators deliver gas once a breath has been initiated until a certain preset pressure is achieved. The volume of gas delivered varies with respiratory compliance. Volume-preset ventilators, on the other hand, deliver a preset volume irrespective of the pressure achieved. They do, however, have a safety pressure limit setting, which allows the volume to be vented externally to protect the patient from excessive barotrauma. The volume-preset ventilators are best suited for continuous ventilation of patients who require long-term ventilatory support. In addition to these conventional methods of ventilation a third method, high-frequency ventilation, has received increased interest in recent years. High-frequency ventilation refers to a mode of ventilation in which frequencies above 60 breaths per minute (bpm) are used with or without bulk delivery of gases to the lung.

Starting Mechanical Ventilation

Indications for Mechanical Ventilation. Mechanical ventilation is indicated when the patient's spontaneous ventilation is not adequate to sustain life. In addition, it is often indicated in critically ill patients to gain control of the patient's ventilation and as prophylaxis for impending collapse of other physiologic functions. Finally, mechanical ventilation is indicated electively for operative cases. Physiologic indications for mechanical ventilation are respiratory or mechanical insufficiency, and gas exchange ineffectiveness. Critical values for these indications are given in Table 1. These guidelines are useful in making the decision to institute mechanical ventilation [1–3]. However, clinical judgment and the trend of these values should be used in the final analysis of whether or not to institute mechanical ventilation. For example, the physician might opt to intubate and mechanically ventilate a refractory asthmatic patient who manifests a "normal" arterial carbon dioxide tension ($PaCO_2$) (i.e., 38–40 mm Hg). In this situation, the patient is unable to maintain a previously (appropriate) subnormal $PaCO_2$. As the $PaCO_2$ becomes (inappropriately) normal, the situation is critical, and abrupt clinical deterioration is imminent. Increasing severity of illness should always predispose a clinician to consider instituting mechanical ventilation. Specifically, mechanical ventilation should be instituted during episodes of severe shock, including septic shock, during cardiopulmonary resuscitation, with the development of the adult respiratory distress syndrome (ARDS), and when severe apnea is likely to be encountered. Mechanical ventilation has been advocated in severe chest trauma to stabilize the chest wall [4,5]. However, in at least one prospective clinical trial it was suggested that management without mechanical ventilation can be as successful [6]. Mechanical ventilation may also be indicated to rest fatigued respiratory muscles. Finally, although it has not been proved in a prospective study, there is general consensus that the institution of mechanical ventilation in severe respiratory failure may minimize further complications and irreversible injury by preserving lung volumes and preventing atelectasis.

Table 1. Indications for Mechanical Ventilation: Critical Values

Measurement	Critical Value
Respiratory mechanics	
Respiratory rate	> 35 bpm
Negative inspiratory force	< −20 mm Hg
Vital capacity	< 10 ml/kg
Minute ventilation	< 3 or > 20 L/min
Gas exchange	
PaO_2 (with supplementary oxygen)	< 55 mm Hg
$PaCO_2$ (acutely)	> 50 mm Hg[a]
$P(A-a)O_2$ (with 100% oxygen)	> 450 mm Hg

[a]Assumes the absence of a metabolic acidosis. In the presence of a metabolic acidosis the patient's respiratory system should compensate by causing a decrease in the $PaCO_2$. The expected $PaCO_2$ can be predicted by the equation $PaCO_2 = 1.5\,[HCO_3^-] + 8 \pm 2$ [96]. For example, if the serum $[HCO_3^-] = 10$ mEq/L then the expected $PaCO_2$ should be 23 ± 2 mm Hg. Acute increases in the $PaCO_2$ above this value may indicate acute respiratory failure and necessitate mechanical ventilation despite the $PaCO_2$ being less than 40 mm Hg.

bpm = breaths per minute; PaO_2 = arterial oxygen tension; $PaCO_2$ = arterial carbon dioxide tension; $P(A-a)O_2$ = alveolar-arterial oxygen tension difference; $[HCO_3^-]$ = bicarbonate concentration.

Initial Ventilatory Settings. When mechanical ventilation is first instituted, we prefer a simple approach to the initial ventilatory settings. In the average adult patient we have found that the 12/12 rule is as efficacious as more complex formulas; therefore, we administer a tidal volume of 12 cc/kg with a backup rate of 12 bpm. Patients are started on assist-control ventilation, which allows them to supplement their ventilation with minimum inspiratory efforts. The intitial fraction of inspired oxygen (FiO_2) is 1.0 or at a level that will ensure an initial arterial oxygen tension (PaO_2) greater than 60 to 70 mm Hg. The rationale for the high initial FiO_2 is that in many situations these patients have a high degree of ventilation-perfusion mismatch and varying degrees of shunt. Once adequate oxygenation is documented with an arterial blood gas measurement, the FiO_2 is rapidly reduced to prevent oxygen

toxicity. Although there are ventilators available that deliver a preset pressure and some that deliver a preset volume [7], in the acute situation ventilation should be initiated with a volume-cycled ventilator. Lack of knowledge of the patient's pulmonary mechanics, and the possibility of dramatic changes in respiratory compliance or resistance in the acute situation, render pressure ventilators inadequate.

Once mechanical ventilation is instituted, levels of arterial blood gases should be measured to document the adequacy of oxygenation and ventilation. We suggest obtaining the first blood gas measurement 10 minutes after initiation of mechanical ventilation. In patients without obstructive lung disease, this period should be adequate for equilibration of PaO_2 [8]. Carbon dioxide equilibrates much more rapidly because of its increased solubility. Although in the patient with obstructive lung disease PaO_2 may not stabilize for 25 minutes [9], a blood gas measurement at the 10-minute point is useful to assess ventilation, and to give an initial indication of the final PaO_2. A subsequent gas measurement can then be obtained at approximately the 30-minute point. The FiO_2 on mechanical ventilation should allow for adequate oxygenation, to a level that has commonly been assumed to be 60 mm Hg [10]. This oxygen tension allows the oxygen saturation to remain in the flat portion of the oxygen–hemoglobin dissociation curve in most clinical conditions [11]. Similarly, ventilation should be adjusted to normalize the $PaCO_2$ between 35 and 40 mm Hg if the patient's response fails to do so by triggering the ventilator. The backup rate should be set on the ventilator at a rate 2 to 3 bpm below the rate required to maintain the desired $PaCO_2$. This will prevent an acute increase in $PaCO_2$ if the patient's response stops triggering the ventilator. In patients with chronic lung disease and carbon dioxide retention, the $PaCO_2$ should be adjusted to the patient's baseline level as indicated by a normal pH in the absence of other acid-base problems. This usually requires the use of a lower tidal volume and rate when choosing the initial ventilator settings. Similarly, when metabolic acidosis is present the desired $PaCO_2$ should be adjusted (if the patient fails to do so) to the $PaCO_2$ expected if normal respiratory compensation for the acidosis was occurring (see Table 1). In this setting a larger initial tidal volume and rate should be used.

A flow rate of approximately 40 L/min should be established to provide an inspiratory-to-expiratory ratio of at least 1 : 3 [12]. Higher flow rates may be required in some patients to meet their inspiratory demands and decrease their inspiratory work [13]. Alternately, lower flow rates may allow for a decrease in airway pressure. This may be useful to potentially reduce barotrauma, or for patients in whom high mean airway pressures are believed to impair venous return and cardiac output. However, as inspiratory flow rates are decreased, the expiratory time available will also decrease. Patients who do not have severe obstructive disease or do not have a marked decrease in elastic recoil can often be ventilated with an inspiration-to-expiration ratio of 1 : 1.5 or even 1 : 1 if necessary. The newer ventilators, such as "sine wave generators" (e.g., ventilators with microprocessing capabilities), allow the inspiratory flow to be given at differing rates over the inspiratory cycle. Whether these various inspiratory curves may be beneficial in certain patients remains to be determined.

The machine pressure required to deliver the preset volume should be noted and a pressure alarm system should be set approximately 10 cm H_2O above this level. If the pressure is exceedingly high, initially above 70 cm H_2O, or if the patient's response continues to supersede the alarm mechanism, mechanical ventilation should be discontinued and the patient managed with ambu bag until the cause of the problem is identified. Selection of equipment and currently available ventilators are discussed in McPherson's and Spearman's excellent book [14].

Inspired Oxygen Concentration. After the patient has been stabilized on the ventilator, the FiO_2 should be decreased to a level that maintains the PaO_2 at 60 to 65 mm Hg or a saturation of 90 to 95% [10]. If the patient has underlying ischemic vascular disease or severe asthma one may choose to maintain the PaO_2 at 80 to 100 mm Hg to prevent intermittent desaturation that might occur during suctioning, with mucous plugging or retained secretions. These adjustments can often be facilitated by use of an ear or finger oximeter that has been documented to correlate with the patient's saturation. Optimizing the FiO_2 as rapidly as possible is important because oxygen is toxic to the lung [15]. The adverse effects of breathing 100% oxygen in humans and animals may occur within 24 hours [16]. These effects include impaired mucociliary clearance, increased epithelial permeability, and impairment of vascular endothelial function. Clinically, pulmonary edema and pulmonary fibrosis may occur. The threshold concentration for oxygen toxicity in humans is not known. However, oxygen toxicity is influenced by both the concentration of oxygen and the duration of exposure. Although any concentration of oxygen above 21% may be toxic, exposure to 40% oxygen for prolonged periods appears to be safe in humans. Concentrations above 50% appear to be progressively more toxic especially as the duration of expo-

sure increases. Recently however, even 40 and 50% oxygen administration for 45 hours was shown to increase lung lavage albumin concentration and the clearance of substances suggesting increased epithelial permeability [17].

Interventions that can be used to decrease the F_IO_2 while maintaining the same PaO_2 are positive end-expiratory pressure (PEEP), decreasing hydrostatic vascular filling pressures in diffuse lung disease, positional changes, or nonsteroidal agents in asymmetric lung disease. Nonsteroidal antiinflammatory drugs presumably increase PaO_2 by augmenting hypoxic vasoconstriction in poorly ventilated areas. The risk of these interventions must be weighed against the risk of oxygen toxicity. If the underlying disease is one that improves rapidly (e.g., cardiogenic pulmonary edema) then the likelihood of oxygen toxicity and the need for intervention is low. However, if the underlying pulmonary dysfunction is likely to be of long duration (e.g., ARDS) early intervention is imperative.

Troubleshooting. The cause of acute respiratory distress in the ventilated patient can often be identified by examining the peak and static pressures on the ventilator manometer [18]. If a ventilator malfunction is present, the patient should be removed from the ventilator and managed with ambu bag until the problem is corrected. Once it is clear that the ventilator is functioning properly, the patient's dynamic and static compliance should be calculated. The pressure manometer on the ventilator will indicate for any given tidal volume and inspiratory flow rate both a peak pressure and a static pressure. The static pressure is assessed just before exhalation (by occluding the exhalation port) when there is no air flow. Dynamic and static compliance are calculated by dividing the tidal volume by the peak and static pressure, respectively. (For an excellent discussion of static compliance, see the article by Demers and colleagues [19].) Changes in these compliance measurements can often help quickly identify a problem that has developed while the patient is on the ventilator [18]. Dynamic compliance is reduced by decreases in chest wall or lung compliance or by increases in airway resistance. Static compliance is reduced by a decrease in chest wall or lung compliance but not affected by airway resistance, because the measurement is made while no air flow is occurring. Therefore, a decrease in dynamic compliance without a change in static compliance indicates a resistance problem, which may result from bronchospasm, retained secretions, or an obstructed ventilator circuit. Symmetric decreases in static and dynamic compliance indicate a problem with lung or chest wall compliance, which may result from tension pneumothorax, right mainstem intubation, pulmonary edema (cardiogenic or noncardiogenic), pneumonia, or atelectasis. In surgical patients, burn patients, or patients with capillary leak syndrome, changes in chest wall compliance must also be considered.

Mechanical Options

Assist-Control Ventilation. Assist-control ventilation has remained the standard mode of ventilation with which other modes are compared. In this mode, the patient is able to trigger the ventilator with an inspiratory effort [3]. When triggered, the machine delivers a preset volume of gas. Therefore, the tidal volume is set by the ventilator and the frequency by the patient. The ventilators in common use today allow a backup respiratory rate to be set, which ensures that a minimum minute ventilation is achieved. This is especially useful in situations in which the patient's response may trigger the ventilator only sporadically or not at all. The major advantage of the assist-control mode is that it allows patients to increase their minute ventilation even if they are weak and debilitated. Patients in unstable condition whose ventilatory needs may rise suddenly (e.g., with onset of sepsis) therefore benefit from this mode. If the patient is agitated or has a central neurologic problem, this mode may predispose the patient to hyperventilation; however, this rarely is a clinical problem.

Intermittent Mandatory Ventilation. Intermittent mandatory ventilation (IMV) is a relatively new mode of mechanical ventilation first advocated in 1973 [20]. In this mode, gas at a preset volume and rate is administered to the patient; however, between machine-delivered breaths, patients are free to supplement their ventilation by their own efforts. The patient's contribution toward the minute ventilation depends on the number and strength of respiratory efforts. Most ventilators in use today have a synchronized IMV mode that allows the preset breath to be delivered when the patient makes a respiratory effort. Synchronized IMV was designed to prevent the patient from getting a double tidal volume (i.e., mandatory machine breath plus spontaneous breath). The assumption has been that synchronized IMV is more comfortable for the patient. However, there are no studies that document an advantage of synchronized IMV over regular IMV.

IMV is probably the most widespread mode of ventilation used. It has rapidly gained clinical acceptance even though there is a paucity of well-designed controlled clinical trials comparing IMV

with the more standard assist-control mode [21–23].

Some of the purported advantages of IMV have recently been questioned. It had been suggested that IMV avoids respiratory alkalosis, which may be a problem with the conventional assist-control mode [24]. However, Culpepper and co-authors [25] studied 18 critically ill patients whom they subjected to one hour of IMV and one hour of assist-control in random sequence. Blood gases and respiratory rates were measured. No clinically significant change in the pH or $PaCO_2$ was noted. Similarly, Hudson and co-workers [26] designed a study to test the efficacy of IMV to compensate for respiratory alkalosis that occurred during assist-control ventilation. In 26 patients, the pH and the $PaCO_2$ rose slightly with IMV, but this rise was not clinically important. They suggested that respiratory alkalosis may be due to increased ventilatory drive, which maintained as long as the patient was able to supplement the IMV ventilation. Importantly, they also noted that carbon dioxide production rose significantly when the patient was switched to IMV despite absence of change in minute ventilation. They speculated that the increase in carbon dioxide production may be due to the increased work of breathing while in the IMV mode. Therefore, it appears that the tendency to develop respiratory alkalosis on mechanical ventilation is usually due to underlying circumstances rather than the mode of ventilation.

The other purported benefits of IMV still await prospective studies comparing IMV with assist-control ventilation. IMV has been claimed to decrease ventilator weaning time [21]. It is clear that most patients wean regardless of the ventilatory mode. Whether IMV is superior or inferior to assist-control in the weaning of the difficult patient remains to be determined. Another area in which IMV and assist-control must be compared is respiratory muscle function. Although respiratory muscle function is thought to be important for adequate ventilation and weaning from the respirator [27], it is unclear that either of these two modes preserves respiratory muscle function or is a better stimulator of respiratory muscles without causing undue fatigue. Other suggested advantages of IMV that have not been proved are that IMV reduces the likelihood of adverse cardiovascular effects from PEEP, requires less frequent sedation and ventilatory monitoring of the patient, and reduces mean airway pressures, resulting in fewer complications [22,23].

In summary, although IMV is used widely and is a useful technique, none of its postulated advantages has been conclusively proved. However, it is a reasonable and safe option. Situations in which IMV would be less preferred are (1) the presence of rapid changes in acid-base status, (2) the desire to decrease respiratory muscle work (i.e., with acute respiratory muscle fatigue), or (3) the need to minimize the proportion of the cardiac output required for respiratory muscle work (e.g., in acute myocardial ischemia with respiratory failure and cardiogenic shock).

Pressure Support Ventilation. Recently several newer mechanical ventilators have offered pressure support as an option. With the pressure support mode, a physician-selected level of positive pressure is maintained during inspiration. This pressure is initiated by a patient's inspiratory effort and it turns off during exhalation. Conceptually, pressure support ventilation is intermittent positive-pressure breathing (IPPB) with a sensing device that delivers the breath at the time the patient makes an inspiratory effort. Pressure support ventilation differs from other modes in that it allows the patient to control respiratory rate and inspiratory time. The pressure support augments the patient's tidal volume and inspiratory flow. Its most touted advantage is that it allows the patient to interact with the ventilator in a more comfortable manner. McIntyre [28] reported more subjective comfort with pressure support in stable patients requiring IMV. He also suggested that pressure support ventilation may decrease the ventilatory work of breathing. Whether these factors are of clinical benefit, especially in the weaning situation, remains speculative. Prakash and Meij [29] have suggested that the addition of pressure support ventilation after cardiac operations has no deleterious effects and may aid weaning. However, their study should be considered preliminary because their patient groups were relatively small. It must be emphasized that pressure support ventilation is contraindicated as the sole source of ventilation in severely ill patients, especially those with impaired respiratory drive. One needs to remember that pressure is the independent variable in this system and that volume is the dependent variable. In unstable situations, if the patient's compliance should decrease, or mucous plugging should occur, minute ventilation could fall precipitously. This relatively new mode of ventilation, however, will probably become more popular in the stable patient and may be beneficial in certain situations.

Positive End-Expiratory Pressure. PEEP is the application of supraatmospheric pressure to the airway during exhalation in the intubated patient. It therefore represents a back pressure against which the lung exhales. PEEP is compatible with any other mode of positive-pressure mechanical ventilation [3]. It can be used with the assist-control mode, with

IMV, with the pressure support mode, and even with high-frequency jet ventilation. Although used for 40 years, it has become popular only in the last 20 years, especially in the treatment of ARDS [30]. Like many new modalities, its popularity rapidly increased as it was promoted for a variety of beneficial effects. Recent evidence, however, has placed PEEP into a more realistic perspective. It is well established that PEEP increases functional residual capacity (FRC), thereby preventing small airway closure and decreasing right-to-left shunt. The magnitude of the increase in FRC depends on the intrinsic lung compliance and the amount of PEEP applied [31]. PEEP appears to be most useful in situations of pulmonary edema, whether from cardiogenic or noncardiogenic (e.g., ARDS) causes.

The contention that PEEP may be beneficial to the lung has no convincing evidence. On the contrary, evidence has been accumulating that PEEP is merely a useful technological mode that will increase PaO_2 and allow for reduction of the FiO_2 [32]. Pepe and colleagues showed that the early application of PEEP at 8 cm H_2O had no effect on the incidence of ARDS in patients at risk for this syndrome [33]. In addition, PEEP did not change the incidence of atelectasis, pneumonia, barotrauma, or mortality. This prospective randomized study of 92 patients provides strong evidence that early PEEP does not prevent ARDS as was suggested in prior clinical studies. Early investigators had also speculated that PEEP may actually drive fluid out of edematous alveoli back into the interstitial compartment. Although PEEP certainly does improve oxygenation and can redistribute intraalveolar edema, there is no evidence to support the contention that PEEP reduces the quantity of extravascular lung water [34]. Finally, although PEEP remains popular in the postoperative setting as a prophylactic against atelectasis, this benefit has not been proved [30]. An early study by Good and colleagues [35] showed no difference in radiologic demonstration of lower lobe atelectasis with PEEP. These authors concluded that although the routine use of low-level PEEP is safe it has no prophylactic value in the prevention of atelectasis. Furthermore, a subsequent study by Tittley and co-workers [36] has suggested that PEEP applied prophylactically after uneventful coronary bypass operations may have deleterious myocardial effects [36]. In 50 patients they found that increasing the level of PEEP decreased cardiac index, stroke index, and left ventricular end-diastolic volume. These changes were reversible with volume loading. However, myocardial oxygen consumption increased with volume loading, and in some patients it was suggested that ischemic metabolism was taking place in the myocardium. These authors thus concluded that prophylactic PEEP may have deleterious effects and should be used cautiously in patients at risk for myocardial ischemic injury.

The goal of PEEP is to increase the PaO_2 in severe hypoxemia, or to allow one to decrease FiO_2 to avoid pulmonary oxygen toxicity [3]. Before PEEP is initiated, baseline hemodynamic and gas exchange measurements should be obtained. The pulmonary static compliance should also be determined. PEEP should be instituted at low levels; we generally recommend 5 cm H_2O. After PEEP has been given for approximately 30 minutes, all hemodynamic, gas exchange, and compliance measurements should be reassessed to guard against an untoward effect of the PEEP. We generally measure arterial oxygen saturation, cardiac output, the mixed venous oxygen saturation, pulmonary static compliance, and pulmonary and sytemic vascular pressures. Oxygen delivery and vascular resistances are calculated. If necessary, PEEP can be increased at increments of 3 to 5 cm H_2O. After each increase the aforementioned variables should be remeasured. It is rare that PEEP levels above 20 cm H_2O are clinically useful. Decrement in cardiac output, which commonly occurs with increasing PEEP, can be ameliorated by the administration of fluids or vasoactive agents. Similarly, PEEP can be discontinued in decrements of 3 to 5 cm H_2O with monitoring of the same aforementioned variables. We believe that the FiO_2 should be decreased before decreasing the PEEP to guard against pulmonary oxygen toxicity. Rapid cessation of PEEP may cause collapse of tenuous alveoli with increased shunt.

The determination of the optimal amount of PEEP remains controversial. Suter and colleagues [37] advocated the measurement of static total lung compliance to determine "best PEEP." They showed that the maximal improvement in pulmonary compliance corresponded with maximal oxygen transport in a study of 15 normovolemic patients. An increase in static compliance is thought to indicate improvement of previously unventilated or partially ventilated alveoli as the FRC increases. Compliance measurements have the advantage of being easy and noninvasive. However, compliance may not parallel the desired endpoint when using PEEP. The optimal PEEP for a given patient will change depending on the goal intended. If the goal is to decrease the FiO_2 to nontoxic levels in a patient with adequate tissue oxygen delivery, the optimal PEEP may be different than for the patient who has inadequate tissue oxygen delivery on 100% FiO_2. In the former situation, the optimal PEEP will be the lowest amount of PEEP that allows the FiO_2 to be decreased to the desired level without causing an unacceptable decrease in tissue oxygen delivery (e.g., cardiac output). In the latter situ-

ation, optimal PEEP will be the level that maximizes tissue oxygen delivery. It should also be remembered that the optimal PEEP determined for a patient is relative to the patient's cardiovascular status. If filling pressures or contractility change, a new level of PEEP may be necessary. Alternately, if PEEP is unsuccessful in obtaining the desired goal given a patient's hemodynamic status, changing the patient's hemodynamic situation (e.g., using vasodilators or inotropes) may improve the effectiveness of PEEP. This approach is time consuming, invasive, and costly because it involves arterial blood gas sampling for calculation of oxygen content, cardiac output determination by a thermodilution catheter, and measurement of mixed venous oxygen saturation. However, because a primary complication of PEEP is a depression of cardiac output secondary to the impedance of systemic vascular return [38] (as well as other mechanisms), this rigorous approach may be warranted. It should also be emphasized that the effect of PEEP on cardiac output cannot be predicted and should be measured.

The application of PEEP can influence vascular pressures in the heart and lungs. If the alveolar pressure exceeds the capillary hydrostatic pressure, then the vascular pressure measured through the pulmonary artery catheter may be altered. This will occur if the pulmonary artery catheter is not located in the dependent portion of the lung. When the pulmonary artery catheter is in one of the upper portions of the lung, the pressure measured may actually reflect the alveolar pressure rather than the pulmonary capillary pressure. The proper positioning of the pulmonary artery catheter in a dependent portion of the lung, confirmed by a cross-table lateral chest roentgenogram, can minimize this problem. When in proper position (zone 3 conditions) the tip of the pulmonary artery catheter should be below the line of entry of the catheter into the thoracic cavity.

In summary, PEEP is a useful supportive adjunct to improve oxygenation and allow for a decrease of supplementary oxygen. There is no evidence to support any direct therapeutic effect to the lungs, and complications are associated with its use, including depression of cardiac output, increased intracranial pressure, and barotrauma. Therefore, it seems rational to use PEEP only in situations in which the PaO_2 demands it.

High-Frequency Ventilation. High-frequency ventilation is a mode of mechanical ventilation that has received much attention over the past 20 years, although the concept of high-frequency ventilation is older. Its current popularity started in the late 1960s when researchers revived the technique to minimize respiratory effects of cardiovascular function. The technique then gained momentum as a possible alternative mode of ventilation, especially for patients with severe respiratory failure [39–40]. Initially, there were some years of confusion about high-frequency ventilation, due to the different modes of high requency that can be used. The three most common techniques of high-frequency ventilation are high-frequency positive-pressure ventilation (HFPPV), high-frequency jet ventilation (HFJV), and high-frequency oscillation (HFO) [41–42]. HFPPV is similar to conventional ventilation except that the rates are much higher, 60 to 100 bpm. HFJV is the most common form of high-frequency ventilation used in the United States today. It consists of a high-pressure gas source with pulsatile jet flow directed into the airway through a small injector port, usually 1 to 2 mm in diameter. This jet then entrains a variable amount of air as it is pulsed into the trachea. HFO consists of a piston moving back and forth at 300 to 2,000 cycles per minute, with the inspiratory and expiratory phase being equivalent. Fresh gas is blown past this piston to provide continual oxygen replenishment and carbon dioxide removal. In the critical care setting HFO can be applied simultaneously with conventional ventilation. The physiologic explanation for the efficacy of high-frequency ventilation defies classic teaching, and is currently the subject of many research investigations.

High-frequency ventilation has certainly generated much interest in both the clinical and research arenas. However, currently there are no specific indications for its use [39]. There have been no controlled prospective trials showing that high-frequency ventilation is superior to conventional ventilation in adults. Most studies to date have investigated physiologic causes and mechanisms of high-frequency ventilation. Some studies have shown that in certain situations (e.g., postoperatively and as an adjunct to weaning) high-frequency ventilation can be equivalent to conventional ventilation [40]. There is some optimism that high-frequency ventilation may be beneficial in the management of bronchopleural fistulas [43]. However, it is of concern that some stable patients have been described who could not be ventilated by HFJV but who were adequately ventilated by conventional ventilation [44]. The reverse finding, long-term successful HFJV in cases where conventional ventilation has failed, is uncommon.

Although currently there are no clear indications for high-frequency ventilation in adults, in clinical practice there are times when high-frequency ventilation is considered. In the United States, HFJV is the mode of choice when high frequency ventilation is used, because HFJV devices are the most

available high-frequency ventilation equipment, and most experience is from use of this mode [42]. However, as HFO becomes clinically available it may be more efficacious in oxygenating a patient who cannot be oxygenated conventionally by HFJV. The following are reasonable situations for the use of high-frequency ventilation: (1) when persistent bronchopleural fistula makes conventional ventilation difficult; (2) with refractory respiratory failure in which conventional ventilation yields diminishing return; (3) during surgical procedures in which lung movement and expansion may be deleterious to the surgery; and (4) to possibly aid the removal of bronchial secretions. One of the major shortcomings of high-frequency ventilation is the difficulty of monitoring the adequacy of ventilation [45]. Rough guidelines are given in Table 2. Blood gas measurements should be obtained within 10 minutes after the start of HFJV, and the patient should be monitored closely, preferably with oximetry. There is also a paucity of data with which to formulate reasonable guidelines on what type of adjustments are necessary during HFJV to achieve the desired outcome. All of the manuevers listed in Table 2, however, increase the potential for air trapping and subsequent barotrauma. Recent studies on HFJV have indicated that increases in the rate of ventilation do not significantly modify minute ventilation. In a lung model, increases in minute ventilation are mainly due to increases in inspiratory-to-expiratory ratio and increases in the driving pressure [46]. Critically ill patients with acute respiratory failure have also been managed with HFJV [47]. Mal and colleagues noted that arterial oxygenation improved as pressure was increased up to 20 cm H_2O. Although there was no difference in PaO_2 with different techniques, there was a marked difference in carbon dioxide elimination. Specifically, most effective carbon dioxide elimination was achieved by increasing the driving pressure rather than increasing the inspiratory-to-expiratory ratio or using a PEEP valve. Mal and associates [47] recommend an inspiratory-to-expiratory ratio of around 0.43 and manipulation of the driving pressure to obtain a satisfactory peak airway pressure and allow for adequate oxygenation and carbon dioxide elimination. It should be noted that this study was done at a respiratory frequency of 250 bpm. Whether these same guidelines can be applied to different respiratory rates is unknown. Finally, in a study done on rabbits [48], Weisberger and colleagues noted that shortening of the inspiratory time requires an increase in the airway pressure to maintain constant ventilation. Shortening of the expiratory time, however, results in air trapping and inadvertent PEEP. The investigators' goal was to maintain constant tidal volume; it was necessary to keep both the inspiratory and expiratory times within a narrow range to achieve this. Weisberger and colleagues also noted that these critical values for inspiratory and expiratory time were dependent on lung compliance.

Table 2. Guidelines for High-Frequency Jet Ventilation

Initial parameters
Rate of 100 bpm
Driving pressure of 30 psi
Inspiratory time of 0.2 sec
Parameter adjustments
To increase CO_2 elimination:
Increase driving pressure
Increase inspiratory time
To improve oxygenation:
Increase driving pressure
Increase inspiratory time
Add PEEP
Use very high rates (800–1,000 bpm)

bpm = breaths per minute; psi = pounds per square inch; PEEP = positive end-expiratory pressure.

Several problems and complications have arisen with high-frequency ventilation. The lack of monitoring techniques to ensure that adequate ventilation is occurring continues to be a primary problem. With conventional ventilation, a known volume is being delivered, and one can be reasonably sure that adequate ventilation is taking place. This assurance is difficult to document with the current status of HFJV. As mentioned previously, it is difficult to predict the direction and magnitude of arterial blood gas changes based on changes made in ventilatory settings [45]. Furthermore, although the airway pressure can be monitored with HFJV, recent reports seem to indicate that the airway pressure may not reflect the alveolar pressure [49–50]. It has also been shown that even with constant airway pressure, lung volume may increase with time [51]. These recent studies point out that it is impossible to predict the amount of barotrauma that the lungs are exposed to during high-frequency ventilation. Although high-frequency ventilation has been promoted as allowing ventilation to take place at lower alveolar pressures, this may or may not be the case. The problem of humidification of the air during HFJV has not been resolved. One popular humidification system is nebulization anterior to the jet. This method is difficult to control or monitor. A recent report [52] also suggests that HFJV may not be innocuous to the tracheal wall. Tracheal injuries with pathology different from that seen with conventional ventilation in animals were described. This finding is troublesome, especially in light of the questionable benefit of HFJV in adults.

HFJV is an intriguing and sometimes fashionable

new mode of ventilation. Its current use is hampered by the lack of controlled studies and the fact that in most patients it has not been shown to be superior to the more familiar and well-studied conventional ventilatory modes. In adults we reserve the use of HFJV for the research laboratory and for desperate situations.

Other Options. Another ventilatory option that has recently been introduced is inverse ratio ventilation (IRV) [53]. In this mode progressively increased inspiratory times are given, with a concomitant decrease in the expiratory phase. Inspiratory-to-expiratory ratios as high as 4 : 1 have been used clinically. This mode has been shown to improve oxygenation while allowing for lower peak airway pressures in a few patients with ARDS [53]. The postulated beneficial effects have been attributed to stabilization of the pulmonary unit and diffusion of gases. The short expiratory phase is said to prevent alveolar collapse. IRV can be used in conjunction with other ventilator modes, including PEEP. Although it has shown promise in the first few patients, IRV has not yet passed the test of time.

Procedures during Mechanical Ventilation

Bronchoscopy and Transbronchial Biopsy. Flexible fiberoptic bronchoscopy has become a common procedure that is performed on some patients while they are being mechanically ventilated. When the proper precautions have been taken, fiberoptic bronchoscopy has been shown to be safe during mechanical ventilation [54–56]. The indications for fiberoptic bronchoscopy during mechanical ventilation are given in Table 3.

The following precautions should be taken when patients on mechanical ventilation undergo bronchoscopy:

(1) The endotracheal tube should be size 8 or larger [56,57]. With an endotracheal tube of smaller diameter, the amount of driving pressure needed to maintain ventilation is too high. Small tubes also predispose patients to air trapping and barotrauma. Quick examination of the tracheobronchial tree may be done, however, with some of the smaller pediatric bronchoscopes in patients with endotracheal tubes smaller than a size 8.

(2) The patient should be ventilated with 100% oxygen for the duration of the bronchoscopy with an increased FiO_2 for 2 to 4 hours postbronchoscopy or until the arterial blood gas measurements indicate stability [54,58]. An ear or finger oximeter is useful to ensure that adequate oxygenation is taking place.

Table 3. Indications for Fiberoptic Bronchoscopy during Mechanical Ventilation

To insert an endotracheal tube
To document endotracheal tube position
To check for tracheal damage
To rule out obstruction of the tube or a major airway
To locate the segment from which mild hemorrhage is occurring
To perform bronchoalveolar lavage or a transbronchial lung biopsy

(3) A trained respiratory therapist should be present at the controls of the mechanical ventilator to adjust flow rates and tidal volumes to ensure adequate ventilation [56].

(4) A swivel adapter with a rubber diaphragm should be used to prevent loss of tidal volume [54, 56].

(5) Continuous echocardiographic monitoring should be done as is standard in intensive care units [54,58].

Mechanically ventilated patients in whom fiberoptic bronchoscopy is contraindicated include (1) those with cardiac instability (e.g., angina or arrhythmias) [59]; (2) those with hemodynamic instability who require large doses of vasopressor agents [60]; (3) those who, despite high levels of FiO_2, are not able to maintain an adequate PaO_2 [55, 59] (the PaO_2 should be at least 100 mm Hg with supplemental oxygen, or the saturation above 95%, before the initiation of bronchoscopy, and during flexible fiberoptic bronchoscopy the saturation as measured by ear or finger oximetry should be maintained above 90%, or the procedure should be discontinued); and (4) those who have massive hemoptysis; if these patients need bronchoscopy, it should be done in the surgical suite with a rigid bronchoscope [59].

Although fiberoptic bronchoscopy may be used to document the presence of secretions and mucous plugging, there is no evidence that suctioning with a fiberoptic bronchoscope is any more beneficial than intensive respiratory therapy. In one prospective study [61], therapy with inhaled bronchodilators, coughing, chest percussion, saline lavage, and suctioning was as effective as fiberoptic bronchoscopy.

Bronchoalveolar lavage can be performed safely during bronchoscopy in mechanically ventilated patients. Bronchoalveolar lavage is especially useful in the immunocompromised patient who likely has a diffuse infectious process [62–65]. Its usefulness in nonimmunocompromised patients with more common infections has not been established. Bron-

choalveolar lavage is also useful to help detect fungal infection and document mycobacterial infection.

Complications of fiberoptic bronchoscopy include, most important, barotrauma. The risk of barotrauma can be minimized by having a respiratory therapist monitor pressures and volumes required to ventilate the patient. A pneumothorax should be excluded immediately after bronchoscopy with fluoroscopy and with an upright chest roentgenogram as soon as possible. An overzealous use of the suction channel can decrease the amount of tidal volume that is delivered to the patient; therefore, suction should be used only for brief periods [55]. Although the levels of arterial blood gases have certainly been shown to change with bronchoscopy during mechanical ventilation, if observed carefully these changes can be minimal and handled by adjustments on the mechanical ventilator.

Although initially considered a contraindication, transbronchial biopsy in patients on mechanical ventilation has recently been reported [58]. Transbronchial biopsy may be useful in patients with diffuse disease and respiratory failure as a first step to establish a diagnosis. Although open-lung biopsy is the standard procedure in this situation, it has the risk of general anesthesia and may not be readily available in all situations. A recent report [58] documented safe transbronchial biopsy during mechanical ventilation in 15 patients. In 5 of those patients a diagnosis was established by transbronchial biopsy. In 2 other patients the brushings and secretions obtained during biopsy were diagnostically important. None of these patients died from the procedure, and only one pneumothorax was encountered, which was managed easily with a chest tube. Hypoxia and hypercapnia were not a problem in this small series. This study suggests that transbronchial biopsy may both be safe and provide useful diagnostic information. It is supported by two animal studies [66,67] in which transbronchoscopic biopsies were done during mechanical ventilation without notable morbidity.

Open-Lung Biopsy. Open-lung biopsy has remained the standard procedure for definitive pathologic identification of a diffuse pulmonary process causing respiratory failure [68,69]. However, as noted previously, we prefer to use transbronchial biopsy and bronchoalveolar lavage as an initial procedure, which can be followed with open-lung biopsy if necessary. Mechanical ventilation does not interfere with the performance of an open-lung biopsy [70]. The decision to perform open-lung biopsy should be made when the tissue diagnosis is considered essential and a transbronchial biopsy, brushings, and bronchoalveolar lavage have not been diagnostic [71–73]. It also should be considered when the severity of the patient's illness or coagulation abnormalities contraindicate transbronchoscopic procedures. The disadvantages of an open-lung biopsy are the necessity for general anesthesia and the requirement of a chest tube for at least 4 hours postoperatively. On the other hand, the likelihood of making the definitive diagnosis still is greater with an open-lung procedure. Numerous authors have suggested sampling a moderately involved area avoiding severely involved lung and normal lung [68,69]. It has also been shown that numerous biopsies of the diffuse process yield an identical diagnosis in the majority of patients [70]. The open-lung biopsy during mechanical ventilation is mostly indicated for the immunosuppressed patient in whom a diagnosis has not been made by other means. Alternately, it may be useful for the patient with a new and aggressive diffuse process who has defied diagnosis. However, in patients who appear to have ARDS on the basis of predisposing risk factors and the clinical status, the open-lung biopsy has not been found helpful [69].

Complications during Mechanical Ventilation

Patients who require mechanical ventilation are at increased risk of morbidity and mortality. Survival appears to decrease with increased duration of mechanical ventilation over a one-month period [74]. Zwillich and co-workers [75] reported an overall survival of only 64% of ventilated patients in a large university center. In a recent study from a community hospital, Witek and co-workers [76] reported only a 60% survival of ventilated patients. They noted that survival decreased to 50% at the time of hospital discharge and to 33% one year after hospitalization. Patient selection is likely to be one component of this decrease in survival, because with increasing critical illness the patient is more likely to require ventilator support. However, a contributing factor may be the incidence of iatrogenic and noniatrogenic complications of mechanical ventilation.

The classic study regarding complications during mechanical ventilation was published in 1974 [75]. Since then there has not been a study of this magnitude, and we believe that its major points are still valid today. A total of 354 patients receiving assisted ventilation were studied prospectively, and 400 individual complications were found. The complications correlating with increased mortality were

right mainstem intubation, endotracheal tube malfunction, and alveolar hyperventilation. These occurred with a 5 to 10% frequency. Other major complications not associated with increased mortality were pneumonia, pneumothorax, atelectasis, gastric distention, and ventilator malfunction. The authors suggested that careful attention to the complications can minimize their appearance. Several recent articles have addressed other specific complications. Craven and co-authors [77] recently defined risk factors for nosocomial pneumonia and fatalities during mechanical ventilation. Factors shown to be associated with the development of nosocomial pneumonia include admission into the intensive care unit in autumn, the presence of an intracranial pressure monitor, and the use of cimetidine. The authors suspected that the decreased chest physiotherapy and suctioning in the patients with intracranial monitors may have led to the second of these factors being present. They also did not find an increase in the incidence of nosocomial infections when ventilator tubing was changed every 48 hours as opposed to every 24 hours. In their study, nosocomial pneumonia was not an independet risk factor for death, although 55% of patients with nosocomial pneumonia died, compared with the overall mortality of 41%. The overall incidence of nosocomial pneumonia of 21% was probably a conservative estimate because the criteria for defining pneumonia were rather stringent. Sixty-one percent of the nosocomial pathogens were gram-negative, which puts the patient at risk for aminoglycoside therapy and, therefore, drug-induced renal failure. One of the interesting speculations was that an increasing gastric pH caused by cimetidine or antacids may result in greater gastric colonization and, therefore, increased propensity toward pneumonia. Indeed, in a study by Pingleton and co-authors [78], 18 patients on mechanical ventilation were studied to determine if tracheal colonization could be traced to bacteria from the oropharynx or the stomach. They found that every patient over the course of 5 days had positive gastric cultures. In 12 of the 16 patients whose tracheas were colonized, the colonizing organism originated in the stomach or oropharynx. These authors thus described a very high incidence of bacterial stomach colonization in patients being mechanically ventilated and receiving tube feedings. These studies raise the possibility that, although the use of antacids and gastric histamine blockers should decrease the risk of gastrointestinal bleeding, they may potentiate the development of nosocomial pneumonia.

Craven and co-authors showed in another study [79] that nebulizers used for medication in mechanical ventilator circuits have a high contamination rate. This was especially the case if the nebulizers were cleaned or disinfected only every 24 hours rather than after every treatment. They therefore suggested that nebulizers be cleaned or disinfected after every treatment.

Mechanically ventilated patients are at increased risk for barotrauma. This risk is increased when PEEP is added. When a pneumothorax is discovered while a patient is being mechanically ventilated, there is general consensus that the immediate placement of a chest tube is indicated. The risk of tension pneumothorax with positive-pressure ventilation, especially in patients being administered PEEP, is too great to allow for observation. Conservative management of the pneumothorax by insertion of a chest tube placed to water seal and external suction results in reexpansion of the affected lung in most cases. Occasionally, a persistent bronchopleural fistula can complicate the pneumothorax. In a recent review of 1,700 cases, Pierson and co-workers [80] noted that bronchopleural fistula occurred in only 2% (39) of mechanically ventilated patients. However, in these 39 patients the overall mortality was 67%. Poor prognosis with bronchopleural fistula appears to be related to its occurrence late in the course of a patient's illness and to increasing volume of the leak. In most patients conventional ventilation can be adjusted to allow adequate ventilation. Usually this is done by monitoring the size of the leak and compensating by increasing the minute ventilation. If conservative management of a bronchopleural fistula does not result in its closing, more aggressive management may be indicated. This subject has been reviewed extensively by Pierson [81]. Techniques suggested include the application of PEEP to the chest tube, occluding the chest tube during inspiration, ventilating the lungs independently, repairing the leak site directly, or using HFJV [82]. We caution the movement to aggressive management techniques if conservative management can stabilize the patient.

An alternative to the traditionally placed chest tube is the placement of small intrathoracic catheters with a one-way valve, the Heimleich valve [83]. These catheters include an external chamber where soft rubber tubing permits air to escape only the pleural space. The tube collapses when pleural pressures are less than atmospheric pressure, thus preventing air entry into the pleural space. These chest tubes are much more comfortable for the patient and yet may allow for adequate removal of pleural air. In general, Heimleich valves should be reserved for the stable patient. For the ventilated patient, the small-diameter chest tube should be connected to a standard collection device with external suction.

Some evidence exists from both animal and human studies that persistent bronchopleural fistulas tend to be prolonged if the patient is on chronic steroid therapy [84]. We therefore suggest that if the patient requires a chest tube in this situation, its potential benefit must be carefully weighed against the risks of steroid therapy.

Other complications of mechanical ventilation are well known. The depression of cardiac output secondary to decreased venous return has been repeatedly described and can be minimized by limiting the inspiratory phase of the positive-pressure ventilator. Positive-pressure ventilation has also been shown to predispose the patient to fluid retention with edema formation secondary to increase in antidiuretic hormone [85]. Mechanical ventilation can be complicated by massive gastric distention. There is also a high incidence of stress ulcer–induced upper gastrointestinal bleeding in patients who require mechanical ventilation. This complication can be reduced by the vigorous use of antacids, H_2 antagonists, or both. It is likely that sucralfate, which has the potential advantage of decreasing stress ulceration and bleeding without increasing gastric pH, would also be effective.

Alternatives to Conventional Ventilatory Modes

Noninvasive modalities of ventilatory support have become a reasonable alternative or adjunct to positive-pressure ventilation in some patients, especially for home care. In general, these forms of mechanical ventilation are reserved for persons who have some respiratory function but often borderline respiratory reserves, especially during the night. Although these modalities were pioneered before the advent of positive-pressure ventilation, their resurgence in today's health care market is due to social and economic pressures that are making home ventilation increasingly desirable [86,87].

The classic negative-pressure ventilator is the iron lung, also known as the Drinker tank. This device reached prominence during the polio epidemic in the 1950s and is still used occasionally to ventilate patients with neurologic problems. In general, all negative-pressure ventilators work best in patients with neuromuscular diseases who have normal lung compliance. None of the negative-pressure ventilators works well if compliance is decreased or resistance is increased in the lungs. A newer version of the iron lung is a portable, lightweight, easy-to-use full-body chamber, which overcomes some of the bulkiness and weight of the iron lung [86]. The primary disadvantage of the iron lung is lack of access to the patient and the requirement of outside assistance to get into and out of the lung. The main advantage is its dependability and mechanical simplicity.

Because of the problems with the iron lung, a chest shell respirator, also known as the cuirass, was developed. This is also commonly called the "turtle shell." The cuirass is frequently better tolerated by patients and offers better patient access than the iron lung. Its primary disadvantage is the need for customizing to provide adequate fit [88]. Although unable to provide the magnitude of negative pressure that can be achieved with the iron lung, the cuirass is often effective in supplementing respiration, primarily at night.

To get around the problems with cuirass custom fitting, the "poncho" was developed. This is an airtight plastic jacket that comes with a fence-like grid that is placed over the chest. Negative pressure is applied with an electronic pump. Its major disadvantages are the difficulty of achieving a good seal and getting into and out of the poncho, which is especially difficult for the patient with neuromuscular disease.

One disadvantage of the iron lung negative-pressure system is the potential for lower extremity pooling of blood with decreased venous return, resulting in a decrease in cardiac output. Patients must also be cautioned not to eat and drink while using a negative assist device because of the great risk of aspiration. Negative-pressure ventilators are contraindicated in patients with upper airway obstruction.

An intriguing new technique that may be useful as a ventilatory assist device is high-frequency chest wall oscillation [89]. Investigators have noted that rapid oscillation of the body surface can result in adequate gas exchange with relatively lower alveolar and airway pressures. In animal studies [90], the animal was placed in a chamber that was rapidly oscillated by an external piston. High-frequency chest oscillation has also been applied to humans; a modified blood pressure cuff is attached to the lower thorax and subsequently inflated and deflated at rapid rates [91, 92]. This method has been effective in maintaining gas exchange in anesthetized rabbits [90]. Calverley and co-authors [89] recently demonstrated that, although high frequency chest wall oscillation causes a decrease in spontaneous minute ventilation, stable $PaCO_2$ is maintained. They suggest that high-frequency chest wall oscillation may assist ventilation without the need for the invasive endotracheal tube. Their 10 normal subjects tolerated the high-frequency chest oscillation without any untoward effects. High-frequency oral oscillation has also been superimposed on tidal breathing in patients with chronic

obstructive pulmonary disease, using a loudspeaker attached to a mouthpiece. This resulted in a decrease in the minute ventilation spontaneously generated by patients to maintain adequate gas exchange [92]. Although high-frequency chest wall or oral oscillation will unlikely provide total ventilatory support in most patients, it will possibly assist gas exchange, especially in patients with chronic disease. Its major attractiveness is the fact that it is noninvasive and appears to be relatively benign. However, the physiology of high-frequency chest wall or oral oscillation and its effectiveness under various disease states must be clinically confirmed.

The pneumobelt is another ventilatory assist device that can be used in some patients [86]. The device wraps around the abdomen and attaches to a positive-pressure source. With inflation, the belt pushes on the abdominal contents, raising the diaphragm and thus causing exhalation. When the pneumobelt is deflated, the abdominal contents drop, thereby causing inspiration. The pneumobelt is primarily used as an assist device when the patient is in the sitting position. We have found it particularly useful in patients with incomplete spinal cord section and respiratory insufficiency.

The rocking bed is used infrequently as a ventilatory assist device [87]. When the bed rocks back and forth, the shifting of the abdominal contents moves the diaphragm up and down, causing adequate tidal volumes in some patients. It is useful only in patients with normal or near normal lung compliance and airway resistance. Although we have had situations in which it provided total ventilatory support, its use is obviously limited by its cumbersomeness and its rocking motion. Although some patients do adapt to the rocking bed, motion sickness and patient care are still paramount problems. In the patient, however, who needs a ventilatory assist device, especially at night, and refuses a tracheostomy or is claustrophobic in the iron lung, the rocking bed may be a reasonable alternative.

Diaphragmatic pacing may be an alternative for people who need chronic ventilatory support for long periods [93–95]. The primary use of diaphragmatic pacing is in patients with cervical cord injury or severe alveolar hypoventilation. It does require an intact phrenic nerve bilaterally. The implanted components consist of the receiver, which receives energy and stimulus information from the external source, and the electrodes, which are affixed to the phrenic nerve and receive their stimulus from the receiver. An external transmitter, either a tabletop or pocket model, is used to set the correct number and strength of the electrical impulses. The technique has been used more often in children and young adults. Although phrenic nerve pacing by electrical stimulation has been used for the past 15 years and has successfully treated respiratory paralysis, certain disadvantages are present. Intermittent rest periods must be used to prevent damage to the phrenic nerve and the diaphragm. A diaphragmatic pacemaker failure in a patient with total respiratory paralysis can be disastrous without an alarm system that the patient can activate. Sedatives and tranquilizers can still depress respiration, and diaphragmatic fatigue still may occur in some patients and may cause irrepairable damage to the diaphragm. Despite its drawbacks, phrenic pacing is an alternative to conventional ventilation that may become more prominent as technical advances are made.

Summary

The technological explosion of the 1970s and early 1980s has added many alternatives in the field of mechanical ventilation. More options and techniques are available now than ever before. Although most of these options are reasonably safe and effective, it is disturbing that there is a lack of prospective controlled clinical data comparing the new modalities. As more new techniques are discovered and promoted, it is hoped that their application will be tempered with clinical data documenting their effectiveness. The requirement to demonstrate efficacy of new techniques with adequate studies is especially necessary now with the economics of health care delivery under increasing scrutiny. The pressure to accept the lowest-cost alternative may become stronger in the future despite the fact that it may not always be the most desirable. Finally, although ventilation in most patients is fairly standardized and successful, more clinical trials are needed to examine ventilation modes and gas exchange mechanisms in the small subset of patients with fulminant acute respiratory failure.

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