Anesthesia for Patients Requiring Advanced Ventilatory Support

James M. Blum, MD*, Ross Blank, MD, Lauryn R. Rochlen, MD

DEFINITIONS AND PATHOLOGY

Respiratory failure has a multitude of causes and generally results in a combination of poor oxygenation and ventilation; however, oxygenation is the primary concern as hypercarbia is rarely life threatening and typically manageable. The definition of hypoxia is defined as an inadequate supply of oxygen. The quantification of hypoxia is more challenging. A widely accepted, yet controversial, method of quantifying hypoxemia is the A-a gradient, which is calculated by subtracting the amount of oxygen
dissolved in blood (PaO2) from alveolar oxygen tension (PAO2).\textsuperscript{1} The A-a gradient involves substantial arithmetic because calculating the PAO2 requires the alveolar gas equation:

$$\text{PaO}_2 = (760 - 47) \times \text{FiO}_2 - \text{PaCO}_2/0.8$$

Hence, the PaO2/FiO2 (P/F ratio) has become a common method of describing hypoxia, and is used as a primary criterion in the definition of certain types of lung pathology.\textsuperscript{2} A common concern of the A-a gradient and P/F ratio is the lack of incorporation of other parameters that affect oxygenation other than the FiO2. This has led to the development of the oxygenation index (OI):

$$\text{OI} = (\text{FiO}_2 \times \text{mean airway pressure})/\text{PaO}_2$$

However, thus far, it has not been used as a primary measure in defining disease states.\textsuperscript{3}

The pathology causing hypoxia is important as there are ventilator strategies that have been shown to provide improvement in long-term survival in acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). The definitions of these disorders are shown in Box 1. In these disorders, the ARDSnet ARMA trial showed that ventilation with 6 mL/kg predicted body weight (PBW) with ventilator plateau pressures <30 cm H2O using a specifically prescribed positive end-expiratory pressure (PEEP) and FiO2 reduced the likelihood of mortality.\textsuperscript{4} These specific settings are available at http://www.ardsnet.org.

To date, there is a lack of large-scale, randomized trial data supporting survival benefits of other ventilation strategies in ALI/ARDS or other pathologies in the adult population. Despite this, it is commonly accepted that VILI affects long-term outcomes and occurs through a variety of methods including barotrauma (too much pressure applied to the lung), volutrauma (too much volume per breath), and atelectrauma (constant opening and closing of lung units).\textsuperscript{5} This has promoted the use of open lung strategies (OLS) to prevent trauma to the lung while providing maximal oxygenation to the patient.\textsuperscript{6} Many OLS are promoted, and many of these involve advanced ventilator modes. However, to understand these modes, one must comprehend the basic physiology of mechanical ventilation.

**THE PHYSIOLOGY OF MECHANICAL VENTILATION**

*Mechanics*

At its most basic level, mechanical ventilation is positive-pressure ventilation in which the ventilator creates positive pressure (relative to atmospheric pressure) at the airway opening. This results in the flow of gas from the airway opening to the lungs and an increase in the volume of the elastic respiratory system (lungs and chest wall). Positive-pressure ventilation is in contrast to spontaneous breathing during which the muscles of respiration create negative intrathoracic pressure. In mechanical and

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<th>Box 1</th>
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<td><strong>Definition of ARDS/ALI</strong></td>
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<td>Acute respiratory failure</td>
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<td>Diffuse bilateral infiltrates on chest radiograph</td>
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<td>Absence of left atrial hypertension</td>
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<tr>
<td>Hypoxemia (P/F ratio &lt;200 for ARDS, P/F ratio &lt;300 for ALI)</td>
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spontaneous ventilation, expiration is a passive process dependent on the elastic recoil of the respiratory system.

The interplay of pressure, flow, volume, and the mechanical properties of the respiratory system is summarized by the equation of motion:

$$\Delta\text{Pressure} = \text{Flow} \times \text{Resistance} + \Delta\text{Volume}/\text{Compliance}$$

In this equation, $\Delta$Pressure refers to the total pressure generated by the respiratory muscles and that applied to the airway opening. The equation shows that the total pressure can be divided into a component of airways resistance ($\text{Flow} = \Delta P/R$ by Ohm’s Law) and an elastic component ($\text{Compliance} = \Delta V/\Delta P = 1/\text{Elastance}$). The relevant $\Delta V$ is the tidal volume ($V_t$).7

Monitoring patients during mechanical ventilation involves measuring pressure at the airway opening. There is no simple way to directly measure pressure in the alveoli or the likely more important transpulmonary pressure (defined as the difference in pressure from inside the alveolus to outside the alveolus; ie, in the pleural space).8 This is shown in Fig. 1.

For patients who are deeply sedated and/or pharmacologically paralyzed, the contribution of the respiratory muscles is nil. In such cases, $\Delta P$ can be measured at the airway opening as peak inspiratory pressure (PIP) − end-expiratory pressure. When ventilator settings or expiratory mechanics lead to PEEP, $\Delta P$ becomes PIP − PEEP. Thus, rearranging the equation of motion gives

$$\text{PIP} = \text{Flow} \times R + V_t/C + \text{PEEP}$$

Fig. 1. Pressures in the respiratory system. (From Hess DR, Bigatello LM. The chest wall in acute lung injury/acute respiratory distress syndrome. Curr Opin Crit Care 2008;14:95; with permission.)
Thus, there are many possible causes for high PIPs during mechanical ventilation. Specifically, high inspiratory flow, high airways resistance, high tidal volume, low compliance, and high PEEP (either applied or intrinsic) can all contribute to high PIP.

In an effort to simplify the assessment of airway pressure, the equation of motion can be simplified by an end-inspiratory pause. During such a pause, the inspiratory and expiratory valves of the ventilator circuit close and gas flow ceases. The airway pressure drops to a plateau pressure less than PIP during this pause such that
\[
\Delta \text{Pressure} = P_{\text{PLAT}} - \text{PEEP} = \text{Flow} \times R + \frac{\text{VT}}{C}
\]
\[
P_{\text{PLAT}} - \text{PEEP} = \frac{\text{VT}}{C} \text{ because Flow} = 0.
\]

Determination of \( P_{\text{PLAT}} \) gives an approximation of alveolar pressure assuming that there is a static column of gas between the airway opening and the alveoli during the pause. It also allows for calculation of the compliance of the respiratory system. Accordingly, the difference between PIP and \( P_{\text{PLAT}} \) corresponds to the \( \text{Flow} \times R \) term and allows for calculation of resistance for a given value of flow.

**Management of Ventilation and Oxygenation**

Management of ventilation and CO\(_2\) clearance with mechanical ventilation is relatively straightforward. Minute ventilation is the product of tidal volume and respiratory rate and these 2 parameters can be directly controlled with older and newer operating room (OR) ventilators. Moreover, continuous monitoring of end-tidal CO\(_2\) in the OR allows for real-time feedback on the adequacy of ventilation. Situations may arise during which CO\(_2\) clearance is inadequate despite seemingly adequate minute ventilation. This situation may occur with increased CO\(_2\) production (eg, sepsis, malignant hyperthermia) or increased dead space (eg, extensions of the breathing circuit distal to the Y-piece or abnormalities in the pulmonary circulation).

Traditional ventilator teaching stresses that F\(_{\text{IO2}}\) and PEEP are the 2 primary variables that influence oxygenation. The former is intuitive and follows from the alveolar gas equation. The effect of PEEP is more complicated. The alveolar gas equation shows the factors that determine the partial pressure of oxygen in the alveolus but does not address how such oxygen enters the circulation. The latter process depends intimately on ventilation-perfusion (V/Q) matching. At one extreme, when V/Q = 0, blood flows from the right to the left side of the heart without encountering any ventilated alveoli. This situation, called shunt, always exists at a low level as a result of anatomic factors such as the bronchial and Thebesian circulations. Pathologic shunts include intracardiac right-to-left shunts, pulmonary arteriovenous malformations, and, most commonly, blood flow through poorly ventilated lung as seen in atelectasis, pneumonia, and pulmonary edema. At the other extreme, dead space, where V/Q approaches infinity, regions of the lungs are ventilated but not well perfused and minimal gas exchange can occur. In reality, the lungs constitute a heterogeneous collection of units with a spectrum of V/Q ratios that can be modified by mechanical ventilation.

The intent of applied PEEP is to prevent cyclic atelectasis and maintain alveoli continuously above their respective closing pressures. When effective, this will reduce or eliminate regions of intrapulmonary shunt and improve V/Q matching and oxygenation. However, PEEP can paradoxically worsen oxygenation if regional over distention redistributes perfusion from high to low V/Q units. Also, the increase in pulmonary vascular resistance associated with high airway pressures may favor passage of blood through an intracardiac shunt and worsen oxygenation. Exactly how to apply PEEP and how much PEEP to apply remain controversial questions. Moreover, PEEP can have other adverse effects, namely an increase in intrathoracic pressure that limits venous return and barotrauma.
A useful concept when considering the effects of PEEP and other ventilator settings on oxygenation is that of mean airway pressure (mPaw). The typical beneficial effect of PEEP on oxygenation may be attributed to an increase in mPaw that reduces intrapulmonary shunt. mPaw is simply the airway pressure averaged over the respiratory cycle. With conventional ventilator settings, PEEP largely determines mPaw because more time is spent in the expiratory than the inspiratory phase when the I/E ratio is $\sim 1:2$. Ventilator modes that maintain a high inspiratory pressure and lengthen the inspiratory time (and thus increase or invert the I/E ratio) result in a further elevation in mPaw. This is the basis of the further improvements in oxygenation that may be seen with advanced ventilatory modes. All the adverse effects noted for applied PEEP are also relevant to elevations in mPaw created by adjustments in ventilator settings.

In addition, adjustments that increase mPaw and thereby limit the expiratory time may increase the likelihood of intrinsic or auto-PEEP. Auto-PEEP refers to the persistence of expiratory flow (and therefore of an expiratory pressure > the applied PEEP) when the patient or ventilator initiates the next inspiration. Auto-PEEP is commonly seen in patients with obstructive lung disease who exhibit delayed expiration. Auto-PEEP can also be induced in patients with or without obstructive disease when they are subjected to rapid respiratory rates, high tidal volumes, and/or shortened expiratory times. For a patient on a mechanical ventilator, auto-PEEP is most easily detected by observation of the flow versus time waveform and evidence that expiratory flow does not return to zero before the subsequent inspiration. Auto-PEEP can be measured during an end-expiratory pause. During volume-control ventilation (VCV), auto-PEEP may be detected by a progressive increase in PIP caused by progressive hyperinflation. During pressure-controlled ventilation (PCV), there will be a progressive loss of tidal volume as the PIP is fixed. Auto-PEEP may also prevent ventilator triggering by not allowing patients to generate the negative pressure or inward flow necessary to open the inspiratory valve in modes that allow for spontaneous breaths. Most importantly, auto-PEEP can aggravate the deleterious hemodynamic and barotraumatic effects of applied PEEP and mPaw caused by further increase of intrathoracic pressures.7

**ADVANCED MODES OF VENTILATION**

When deciding which mode of ventilatory support to apply to a hypoxic patient, it is vital to keep in mind that responses are not always predictable and may be highly variable between patients. This is particularly true for the patient in respiratory failure with other comorbidities and concomitant organ dysfunction. It is important to monitor the patient’s response to a particular ventilator setting by continued assessment of pulmonary compliance and serial arterial blood gases. Patients with respiratory failure who are difficult to ventilate and oxygenate on conventional VCV modes may benefit from PCV in which strict pressure limitation can be achieved. With close monitoring of VCV, similar results to PCV can be obtained; however, advanced ventilators with servo-controlled valves are able to provide additional control and volumes at lower pressures not obtainable with VCV.

**PCV**

Features specific to pressure-controlled modes contribute to improved gas exchange and patient-ventilator interaction.9 The most basic settings of this mode include a respiratory rate, $FIO_2$, PEEP, drive pressure ($\Delta P$), and I/E ratio. Inspiration can be pressure or time cycled. Tidal volumes are generated by the pressure difference achieved from beginning to end of inspiration and vary with the airway resistance.
and compliance. The inspiratory time is determined by the clinician and can be adjusted based on the patient’s lung mechanics. A large portion of tidal volume is delivered early in the cycle resulting in higher average mPaw compared with VCV, although adjusting the flow rate and using additional ventilator settings may reduce this increase and result in lower pressures for equal tidal volumes.

PCV offers several advantages for the patient in severe respiratory failure. The concept of pressure-limited ventilation maintains PIP less than a specified value, decreasing the risk of macro- and microscopic barotraumas. The decelerating flow pattern of PCV produces a more uniform distribution of ventilation with the lung fields, which is beneficial in heterogeneous lungs. Another potential benefit of the decelerating flow pattern is that most of the tidal volume is delivered early on in inspiration. This provides higher mPaw over the course of the respiratory cycle. Higher mPaw results in improved oxygenation.\textsuperscript{10,11}

PCV may be especially beneficial in patients with ALI and ARDS. The variable flow rates used in PCV improve patient work of breathing and limit high PIP. The increased mPaw leads to improved oxygenation, which is often difficult in these patients. Shorter expiration times may lead to increased auto-PEEP, which can also lead to improved oxygenation.

The disadvantages and limitations of using PCV also warrant discussion. Improvement in oxygenation from an increase in mPaw depends on the amount of lung tissue available for recruitment. In highly damaged and inflamed lung in which compliance is poor, it is difficult to achieve adequate ventilation while maintaining pressure limits. When lung damage is heterogeneous, normal lung regions may be overinflated and at risk for VILI. Maximum flow rates early during inspiration expose the distal airways to high shearing, also increasing the risk of VILI. As lung compliance improves, PCV provides a mechanism for the introduction of volutrauma if the ventilator settings are not changed.

**Airway Pressure Release Ventilation and Biphasic Positive Airway Pressure**

Quests to improve oxygenation, pulmonary mechanics and reduce VILI have led to the development of more advanced modes of ventilation that can commonly be seen in use in ICUs. Two such modes are airway pressure release ventilation (APRV) and biphasic positive airway pressure (BIPAP). Both modes are extensions of PCV that exhibit similar concepts and physiology as PCV. These modes are typically reserved for patients with difficult to manage ALI or ARDS, and their implementation is often limited by lack of clinician comfort and knowledge with managing the ventilator settings and potential patient-ventilator interactions and patient responses.

Barriers to using APRV or BIPAP may be related to confusion regarding the definitions and parameters of these 2 modes, which may differ between institutions due to branding by ventilator companies. A review of definitional criteria by Rose and Hawkins\textsuperscript{12} which examined 50 studies and 18 discussion articles investigating APRV and/or BIPAP, revealed discrepancies in the descriptions of APRV and BIPAP.

Such ambiguity makes the use and discussion of these modes difficult and often frustrating. They found that further confusion is caused by ventilator branding and reference terms for ventilator setup. This study found that APRV and BIPAP are described in the literature as the same mode, on a continuum, or as distinctively different modes. The major distinction between modes found in this review was the mean duration of expiration time, which was approximately 3 times longer in BIPAP compared with APRV (\textsuperscript{Fig. 2}). Inspiratory pressure (P\textsubscript{high}) and expiratory pressure (P\textsubscript{low}) were reported as similar for either mode.

Original descriptions of these modes were published within 2 years of each other. APRV was first described in 1987 by Stock and Downs\textsuperscript{13} from the United States...
and was described as continuous positive airway pressure (CPAP) with an intermittent release phase. BIPAP was then described in 1989 by the European team of Baum and colleagues as a mode that combined PCV with spontaneous ventilation.

APRV and BIPAP are both pressure-limited, time-cycled modes (Figs. 3 and 4). The basic settings are similar to those of PCV; however, instead of the drive pressure being set, a high pressure \( P_{\text{high}} \) (i.e., PIP) and a low pressure \( P_{\text{low}} \) (i.e., PEEP) are set. Ventilation occurs by alternating between 2 pressure levels, \( P_{\text{high}} \) and \( P_{\text{low}} \). A key difference between these modes and PCV is that these modes allow for spontaneous breathing independent of ventilator cycling using an active inspiratory valve. A spontaneous breath can be taken by the patient at any point in the respiratory cycle. If the patient is not breathing spontaneously, both modes are virtually identical to conventional PCV, perhaps with an extended I time. The degree of ventilatory support is determined by time spent at both pressure levels and the subsequent tidal volume achieved. As mentioned earlier, the primary distinction between modes is that there are no restrictions on the duration of time spent at \( P_{\text{low}} \) or release pressure in APRV. Original descriptions of APRV used durations of \( P_{\text{low}} \) less than or equal to

![Fig. 2. Airway pressure release ventilation. (From Frawley PM, Habashi NM. Airway pressure release ventilation: theory and practice. AACN Clin Issues 2001;12(2):235; with permission.)](image1)

![Fig. 3. Airway pressure release ventilation. (From Frawley PM, Habashi NM. Airway pressure release ventilation: theory and practice. AACN Clin Issues 2001;12(2):238; with permission.)](image2)
1.5 seconds. The time spent at $P_{low}$ ($T_{low}$) is set for adequate carbon dioxide removal, and not so long as to lead to de-recruitment. $P_{low}$ can be titrated exactly like PEEP to be maintained higher than the lower inflection point on the compliance curve, allowing for optimal lung mechanics.

One of the major advantages of APRV and BIPAP is improved distribution of gas to dependent regions of the lung as a result of the effects of spontaneous ventilation, leading to improved V/Q matching. This is caused by movement of posterior sections of the diaphragm that occurs during spontaneous ventilation, but is absent during mechanical breaths. In addition, spontaneous ventilation reduces the amount of positive-pressure ventilation required during the respiratory cycle, which can result in improved hemodynamics by reducing the negative cardiovascular effects of positive pressure. There is also evidence supporting an increase in glomerular filtration rate and sodium excretion, as well as improved liver function as a result of spontaneous ventilation. Patients with left ventricular dysfunction may not tolerate the increased preload and left ventricular afterload that accompanies spontaneous breaths. Although hemodynamic instability may be disadvantageous in certain patient populations, there are no absolute contraindications to using APRV or BIPAP.

Another proposed advantage is improved patient-ventilator interaction. Dyssynchrony may increase the work of breathing, leading to an increase in oxygen consumption and reductions in effective ventilatory support. Improved synchrony with the ventilator results in decreased need for sedation and neuromuscular blockade, another potential advantage.

Despite the evidence supporting the beneficial effects of APRV and BIPAP, there are also questions that remain to be answered in future trials. Suggestions that spontaneous breaths taken while at higher pressure levels may result in even higher inflation volumes and pressure, aggravating VILI need to be examined further. There are also some who question the need for less sedation and neuromuscular blockade as some data suggest patients with severe ARDS should not breath spontaneously. One major outstanding concern is how to standardize the application of APRV and BIPAP to assist clinicians in determining optimal settings and making adjustments.

The specific advantages seen with the application of inverse ratio PCV, APRV, or BIPAP become especially useful when caring for the patient with ALI/ARDS that is difficult to manage. The nearly constant airway pressures achieved favor alveolar recruitment, which should improve oxygenation. Short expiratory times promote ventilation in healthier alveoli, again increasing oxygenation and V/Q matching. Some
arguments for spontaneous ventilation can also be extrapolated to this patient population. Lower requirements for sedation may be beneficial because of decreased effects of sedating medications on other organ functions and less accumulation. Minimal sedation will also allow acute complications, such as changes in mental status, to be more easily recognized. The avoidance of neuromuscular blockade reduces the likelihood of myopathy of critical illness. Despite these potential benefits, there is no large-scale randomized data suggesting improved long-term outcomes from the use of any of the ventilatory modes.

**High-Frequency Oscillatory Ventilation**

High-frequency oscillatory ventilation (HFOV) works by providing exceptionally small tidal volumes at a high mPaw,\(^{19}\) which is believed to be lung protective as it avoids the atelectrauma from other higher volume forms of ventilation and maintains open lung to help improve oxygenation. HFOV is similar to other forms of jet ventilation, but in HFOV, there is an active expiratory component that improves ventilation.\(^ {20}\)

HFOV devices operate with a continuous flow of fresh gas (bias flow) of 30 to 60 L/min and a CPAP valve keeping the mPaw typically between 25 and 35 cm H\(_2\)O,\(^ {21}\) which provides oxygenation to the patient. Ventilation is provided by a piston-driven vibrating diaphragm. The rate of the diaphragm is set somewhere between 3 and 10 Hz. The power set on the diaphragm determines the tidal volume of each breath and is reflected in the oscillatory pressure amplitude (\(\Delta P\)).

Typically, the settings on HFOV include the F\(_{\text{IO}_2}\), the mPaw, diaphragm Hz and \(\Delta P\). Oxygenation is controlled through increasing the mPaw and F\(_{\text{IO}_2}\) if required. Ventilation is controlled through the \(\Delta P\) and Hz. Contrary to initial beliefs, a decrease in Hz actually improves ventilation by allowing a larger V\(_T\). It cannot be predicted how patients will respond to HFOV at various settings.

When initiating HFOV, patients are started with a setting of 100% F\(_{\text{IO}_2}\) and a mPaw about 5 cm H\(_2\)O greater than that found on a conventional ventilator. Patients are frequently recruited using recruitment maneuvers at the initiation of HFOV. If oxygenation does not improve, the mPaw is increased up to levels as high as 45 cm H\(_2\)O. Once life-sustaining oxygenation has been achieved, the frequency and power are adjusted to help minimize the amount of hypercarbia. If patients remain hypercarbic, it is not uncommon to deflate the endotracheal tube cuff to improve ventilation. As the patient improves, the F\(_{\text{IO}_2}\) is typically weaned to nontoxic levels (<50%) and then the mPaw is gradually decreased. Conversion from HFOV to a conventional ventilator is considered when mPaw is <25 cm H\(_2\)O.

For patients to tolerate HFOV, substantial sedation is required. Patients do not tend to tolerate spontaneous respiration while on HFOV. If spontaneous breaths cannot be extinguished using sedation alone, neuromuscular blockade is warranted. HFOV also has marked effects on the cardiovascular system with potentially profound hypotenion.\(^ {22,23}\) This is likely because the mPaw used tends to decrease preload profoundly; however, attempts to treat preload with increased volume administration risks reducing oxygenation. Judicious use of volume with the early administration of vaso-pressors seems reasonable in the management of these patients.

The overall effect of HFOV to date seems to be improved oxygenation. There are no large-scale randomized trials showing improved survival with HFOV.\(^ {21}\) This is most likely because of its use as a salvage strategy when conventional ventilation does not provide adequate support. Data show that with the initiation of HFOV, the Pa\(_{\text{O}_2}\)/F\(_{\text{IO}_2}\) ratio typically improves. In 1 multicenter trial, there was a nonsignificant trend toward survival in the HFOV population compared with a conventional ventilation
The upcoming OSCILLATE trial which will be enrolling patients with early ARDS may provide greater insight into potential improved outcomes with HFOV.

**Extracorporeal Membranous Oxygenation**

Extracorporeal membranous oxygenation (ECMO) represents the final possible salvage modality in patients with severe respiratory failure. ECMO is the technique of providing long-term cardiopulmonary bypass support typically for days to weeks when conventional and/or unconventional ventilator modalities result in continuing hypoxia, profound hypercarbia, and/or hemodynamic compromise. ECMO is only a supportive therapy that provides time for other curative measures to act, including the body’s natural immune system and time. In general survival for adult patients undergoing ECMO for respiratory support is between 40% and 70%.25

There are 2 primary forms of ECMO, venovenous (VV) and venoarterial (VA). In VV ECMO, deoxygenated blood is extracted from the patient, oxygenated extracorporally, and returned to the patient’s venous circulation close to the right atrium. This process results in a higher venous PO2, which is then circulated through the lungs and then to the rest of the body using the patient’s native cardiac function. VV ECMO is accomplished by either 2 single lumen catheters typically placed in the internal jugular and femoral veins or newer venovenous double lumen catheters (VVDL) placed in the internal jugular vein.

VA ECMO is designed to provide respiratory and cardiac support using catheters placed in the venous and arterial circulatory systems. Blood is withdrawn from the venous circulation, oxygenated, and returned to the patient’s arterial circulation. Depending on the age and condition of the patient, a variety of methods for access to the patient’s vasculature are available. In the adult, femoral venous and femoral arterial cannulation is preferred.

Randomized trials of adult ECMO are rare, similar to most high-technology/surgical therapies. During the original trial at 9 centers, 48 patients were managed using conventional ventilation and 42 patients received VA ECMO.26 Survival in each group was 4 patients, which was not significantly different. Criticisms of the trial were: the technology that was implemented was immature, the centers involved were inexperienced, and the intervention of ECMO support occurred too late to affect survival.

In 2009, Peek and colleagues27 published the results of the Conventional Ventilation or ECMO for Severe Adult Respiratory Failure (CESAR) trial. This trial randomized 180 patients with severe ARDS (Murray score >3 or pH <7.20) to care either at a tertiary care center or a single ECMO center. Of patients referred to the ECMO center 75% received ECMO therapy. Survival was 63% at 6 months for those patients referred to the ECMO center and 47% for those treated at tertiary care centers. Six of the patients in the ECMO group died in or pending transfer to the ECMO center. Criticisms of this trial include the lack of a standardized protocol for care at the tertiary care centers and a high rate to transfer mortality in the ECMO referral group.28

Historically, ECMO has been a limited resource. This has forced providers to seriously consider potential ECMO candidates long-term prognosis before the initiation of therapy. Each center has their own criteria for the initiation of ECMO; however, the requirement of anticoagulation and blood component transfusion for ECMO limits the patients who are eligible for therapy. Predictors of poor outcome for respiratory ECMO include advanced patient age, increased pre-ECMO ventilation duration, and diagnosis.29 Absolute contraindications to ECMO include ongoing terminal disease that will not resolve or stabilize, inability to anticoagulate, intracranial hemorrhage, and refusal to receive blood products.
Special preparations by the OR anesthesia and intensive care teams are required when the patient with severe respiratory failure on an advanced mode of ventilation needs a surgical procedure. It is imperative that communication regarding the patient’s underlying condition and respiratory status occurs between the 2 teams. The ICU team should be responsible for optimizing the patient’s oxygenation, ventilation, and hemodynamics before the patient goes to the OR.

Concerns for the OR anesthesia begin with how to safely transport the patient from the ICU to the OR. Most commonly, oxygenation and ventilation of an intubated ICU patient going to the OR occurs via resuscitation bag with high-flow oxygen and the addition of a PEEP valve. The patient in respiratory failure may not tolerate this mode of ventilation. If there is any question about the patient’s safety for transport with a resuscitation bag, consider transporting the patient with an ICU ventilator. Newer models of ICU ventilators are lighter and easier to transport, provided enough personnel are available to assist. In the event an ICU travel ventilator is not available, every attempt should be made to bring the OR to the patient if possible, or consider an alternate technique for managing the situation. For example, a large intraabdominal fluid collection may be managed with percutaneous drainage rather than an exploratory laparotomy.

In addition, there is evidence that moving patients from the ICU is associated with an increased incidence of ventilator-associated pneumonia (VAP). This increase in VAP rate may in part be caused by the patient being supine during transfer and transport, and patient movement causing shifting of respiratory secretions. Aggressive suctioning, using sterile in-line suction, checking for endotracheal tube cuff leaks, and maintaining the head of bed elevated may help to minimize this risk.

Once in the OR, the team must now focus on how to proceed with ventilation, oxygenation, the anesthetic plan, and management of hemodynamics. The patient in respiratory failure requiring an advanced mode of mechanical ventilation presents unique challenges to the anesthesia team. One of the main goals when providing care for such a patient should be to avoid additional lung injury leading to further deterioration in oxygenation. Careful attention should be paid to the preoperative PEEP settings and flow dynamics, and every effort should be made to replicate this environment in the OR. Most anesthesia machines in the OR, however, are not able to achieve the pressure limitations and flow characteristics of the ICU ventilators. In the case of the patient who may not tolerate ventilation with a conventional anesthesia machine, consider using the ICU ventilator in the OR.

If it is possible to use the anesthesia machine, the focus now shifts on how to best anesthetize this patient. Volatile agents are known to inhibit hypoxic pulmonary vasoconstriction. Increasing intrapulmonary shunt will likely result in decreased oxygenation, which is not good in an already compromised patient. Conversely, experimental evidence has shown that inhalation agents (specifically sevoflurane) may actually be protective to injured lung by modulating the inflammatory response caused by activation of alveolar macrophages. This concept is still theoretic. Until stronger evidence emerges, it remains most prudent to arrange for a total intravenous technique.

The effect of spontaneous ventilation on oxygenation during ventilation with APRV or BIPAP was discussed earlier. Surgical procedures requiring paralysis and abolition of spontaneous efforts may be detrimental. In this instance, the need for paralysis should be discussed with the surgical team, keeping in mind that the patient’s best interests are paramount.
Hemodynamics as they relate to mechanical ventilation must also be considered. Positive-pressure ventilation and spontaneous ventilation during the respiratory cycle have significant effects on the patient’s cardiovascular status. How the patient’s hemodynamics react to alterations in airway pressures and the respiratory cycle must be monitored. These patients should have invasive monitors in place or placed intraoperatively to assist the anesthesiologist in assessing the patient’s responses. Of utmost importance is the patient’s intravascular volume status; it is highly recommended to avoid volume overload and favor hypovolemia in this population. It has been shown that in patients with ARDS fluid balance is directly correlated with mortality. Aggressive fluid resuscitation may worsen alveolar damage and inflammation. Overall, the early implementation of vasopressors and/or inotropes seems prudent in this population in the OR. If the patient is receiving continuous renal replacement therapy (CRRT), and the procedure is expected to last more than 90 minutes, it would be prudent to continue to provide CRRT in the OR.

In the event a patient needs a surgical procedure on HFOV, it is imperative to fully discuss the entire procedure in detail with all providers involved. Frequently, these patients are not moveable because of hypoxia or profound risk of technical complications during transport. For smaller procedures, it is reasonable to bring the OR to the patient, rather than risking moving the patient from the ICU. If the procedure is complex, requires specialized equipment, or has potentially high blood loss, it is then reasonable to move the patient from the ICU. If the patient is on HFOV, it is frequently possible to transport the patient using 100% FiO2, high airway pressures, and inverse ratio ventilation. The goal should be to obtain an equivalent mPaw using a conventional ventilator to that provided on HFOV. This is usually not possible using the traditional low tidal volume strategies that have been proved to improve survival, and these settings are purely seen as salvage strategies. The acceptance of lower than normal SpO2 as low as 80% with PaO2 into the low 50s is also reasonable in these situations. Once in the OR, it is reasonable to resume treatment with HFOV during the procedure.

If a patient requires surgery while on ECMO, multiple provisions must be made by the anesthetic team. Bleeding and transport risks are the primary threat to the ECMO patient. Because of the high risk of bleeding, almost all surgical procedures should be completed in the OR. If a patient is on VV ECMO, it is reasonable to hold anticoagulation and attempt to normalize coagulation before surgery. This is because the risk from small venous emboli is very small relative to the risk of bleeding. VA ECMO patients are at a profound risk of stroke or other embolic events if they are not anticoagulated for prolonged periods of time. In the event a VA patient goes to the OR, a serious discussion of the risks and benefits of reducing anticoagulation must take place between all services. Regardless of the form of ECMO, there must be profound attention by the surgical service to achieving hemostasis during the procedure. The anesthesiologist should have all medications required for a cardiac bypass, including heparin and protamine. The anesthesiologist should have a firm understanding of the recommended ventilator settings that should be implemented in the event of ECMO circuit failure.

Despite the distinct challenges that patients with respiratory failure present to the anesthesiologist, a safe and smooth perioperative course can be achieved if the principles discussed in this article are followed.

REFERENCES


