Current status of new modes of mechanical ventilation

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Current approaches to managing patients requiring ventilatory support have focused on a lung protective strategy. This approach limits peak alveolar pressure and tidal volume, and allows hypercapnia. Although hypercapnia is tolerated by many patients, in some the acute acidosis markedly complicates clinical management. Tracheal gas insufflation (TGI) has been designed as an adjunct to conventional ventilation to decrease $PaCO_2$. Although no commercial TGI systems are available, TGI holds great promise and can be expected to be available comercially in the future. Pressure ventilation has become the ventilatory approach of the 1990s, whether pressure support or pressure control. However, problems associated with varying tidal volumes have resulted in manufacturers developing ventilatory modes that combine the beneficial effects of both pressure and volume ventilation.

Key Words: *Tracheal gas insufflation, Ventilatory support*

État actuel des nouveaux modes de ventilation mécanique

RÉSUMÉ : Les approches courantes de la prise en charge des patients nécessitant un support ventilatoire privilégient une stratégie de protection des poumons. Ce type d'approche limite la pression alvéolaire maximale et le volume courant, et tolère une hypercapnie. Si l'hypercapnie est tolérée par de nombreux patients, pour d'autres, l'acidose aiguë complique énormément leur prise en charge clinique. Un système permettant d'insuffler du gaz dans la trachée a été conçu pour servir d'appoint à la ventilation classique dans le but de diminuer la $PaCO_2$. Bien que ce système ne soit pas disponible sur le marché, il est très prometteur et devrait être vendu prochainement. Dans les années 90, on a privilégié la ventilation par pression, soit de soutien ou contrôlée. Cependant, devant les problèmes associés à la fluctuation des volumes courants, les fabricants ont mis au point des modes de ventilation qui associent les effets bénéfiques de la pression et du volume.

Over the past 10 years, a number of different modes of mechanical ventilation have been introduced, in addition to changes in the philosophy by which we apply mechanical ventilation. Of primary concern today is the prevention of ventilator-induced lung injury. Along with this concern has come a change in the level of carbon dioxide considered to be acceptable in critically ill patients (permissive hypercapnia) and the introduction of adjunct therapies (tracheal gas insufflation [TGI]) designed to reduce carbon dioxide. In addition, the focus of ventilator delivery has moved from volume to pressure. Pressure support and pressure control have become the standards for ventilatory modes.

VENTILATOR-INDUCED LUNG INJURY

Mechanical ventilation is a nonphysiological process. Pressure, volume and fraction of inspired oxygen beyond the levels that the lung normally tolerates are frequently used. As a result, lung injury may be caused or extended by the process of mechanical ventilation. Lung injury may be manifest in two forms: gross barotrauma or parenchymal injury similar to acute respiratory distress syndrome (ARDS) (Table 1).

Three conditions must usually be present for gross barotrauma to develop: disease; high transpulmonary pressure; and overdistension (1). The precise pressures and volumes having a high likelihood for the development of barotrauma are unknown. However, because the maximum transpulmon-

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TABLE 1
The spectrum of lung injury induced by mechanical ventilation

- Atelectasis
- Alveolar hemorrhage
- Alveolar neutrophil infiltration
- Alveolar macrophage accumulation
- Decreased compliance
- Denudement of basement membrane
- Detachment of endothelial cells
- Emphysematous changes
- Gross pulmonary edema
- Hyaline membrane formation
- Intracapillary blebs
- Interstitial edema
- Interstitial lymphocyte infiltration
- Pneumonia
- Subcutaneous emphysema
- Systemic gas embolism
- Tension cyst formation
- Type II pneumocyte formation

ary pressure gradient developed by healthy individuals is about 35 to 40 cm H_2O, it seems reasonable to expect that the probability of barotrauma will increase if pressure is applied above this level (2).

Numerous animal studies (eg, in rats, sheep, dogs and pigs) have demonstrated parenchymal damage after relatively short periods of mechanical ventilation when peak airway pressures are maintained at about 45 cm H_2O (3,4). An important finding of these studies was that the extent of the damage was decreased if positive end-expiratory pressure (PEEP) was maintained above the inflection point on the compliance curve of the lung (3) or if the thorax was strapped (preventing hyperinflation because of decreased chest wall compliance) (4). These data have lead most authorities on mechanical ventilation to recommend limiting end-inspiratory plateau pressure and thus the resulting inflation volume (5). The term 'volutrauma' has been used to describe the lung injury induced by mechanical ventilation to emphasize that it is local overdistension that causes lung injury and not pressure per se (2). If local overdistension is limited by strapping of the thorax (or any other mechanism that decreases chest wall compliance), no injury develops despite high alveolar pressure. From a practical perspective, most have indicated that peak alveolar pressure (end-inspiratory plateau pressure) should be kept below 35 cm H_2O (5).

PERMISSIVE HYPERCAPNIA

Permissive hypercapnia is the deliberate limitation of ventilatory support to avoid regional or global overdistension, allowing $PaCO_2$ to rise to levels greater than normal (50 to 100 mmHg) (6). Allowing $PaCO_2$ to rise to these levels should be considered when the only alternative is a potentially dangerous increase in peak alveolar pressure. The potential adverse effects of elevated $PaCO_2$ are listed in Table 2 (6). Most of the more important clinical problems occur

TABLE 2
Physiological effects of permissive hypercapnia

- Shift in the oxyhemoglobin dissociation curve to the right
- Decreased alveolar PO_2
- Both stimulation and depression of the cardiovascular system
- Stimulation of ventilation
- Dilation of vascular bed
- Increased intracranial pressure
- Anesthesia ($PaCO_2$ 200 mmHg)
- Decreased renal bloodflow ($PaCO_2$ 150 mmHg)
- Leakage of intracellular potassium ($PaCO_2$ 150 mmHg)
- Alteration of the action of pharmacological agents (a result of intracellular acidosis)

at $PaCO_2$ levels above 150 mmHg. However, even small increases in $PaCO_2$ increase cerebral bloodflow, and permissive hypercapnia is generally contraindicated when intracranial pressure is increased (eg, acute head injury). Elevated $PaCO_2$ also stimulates ventilation, but patients are usually sedated and paralyzed in the settings where permissive hypercapnia is maintained.

Permissive hypercapnia may adversely affect the oxygenation status of some patients. Elevated $PaCO_2$ and low pH shift the oxyhemoglobin dissociation curve to the right, decreasing the affinity of hemoglobin for oxygen and decreasing oxygen loading in the lungs, but facilitating the unloading of oxygen at the tissues. In addition, as illustrated by the alveolar gas equation, an increase in alveolar PCO_2 results in a decrease in alveolar PO_2. For each $PaCO_2$ rise of 1 mmHg, PaO_2 decreases by about 1 mmHg. Whenever permissive hypercapnia is used, optimal efforts to maximize oxygenation should be attempted.

The effect of carbon dioxide on the cardiovascular system is more difficult to predict because carbon dioxide elicits competing responses from the cardiovascular system (7). Carbon dioxide directly stimulates or depresses some parts of the cardiovascular system, but opposite effects can occur via stimulation of the autonomic nervous system. It is thus difficult to predict the precise response of the cardiovascular system to permissive hypercapnia in any given patient (7). However, clinically an increase in PCO_2 normally causes pulmonary hypertension. Dosage of pharmaceutical agents affecting the cardiovascular and autonomic nervous systems may need to be adjusted in the presence of permissive hypercapnia, but this is due to the resulting acidosis and not to elevated PCO_2 (6).

The primary factor limiting the use of permissive hypercapnia is pH. Patients without primary cardiovascular disease or renal failure usually tolerate a pH of 7.20 to 7.25, and younger patients may tolerate an even lower pH (6). The specific acceptable minimal pH needs to be determined on an individual patient basis. Allowing PCO_2 to rise gradually from the onset of ventilation allows gradual renal compensation without severe acidosis. Abrupt changes in ventilator strategies that result in rapid and marked elevation of $PaCO_2$ are more poorly tolerated.

Whether alkalizing agents should be administered to manage acidosis induced by permissive hypercapnia is debatable. In the setting of cardiac arrest, sodium bicarbonate use has been questioned because of the resulting increased intracellular acidosis (8). Its use in permissive hypercapnia, however, has not been extensively studied. One can expect a short term increase in carbon dioxide load when sodium bicarbonate is administrated, which is exhaled over time if the level of ventilation is held constant. However, whether the use of alkalizing agents has any effect on an overall tolerance of permissive hypercapnia is not known.

TGI: TGI is an adjunct to mechanical ventilation used in settings of elevated $PaCO_2$ (9). A secondary flow of gas (4 to 12 L/min) is injected distal to the tip of the endotracheal tube but proximal to the carina through a small bore catheter. TGI is proposed to lower $PaCO_2$ by reducing dead space ventilation via washout of carbon dioxide from the large airways at end-expiration, injection of part or all of the tidal volume (V_T) at the trachea and enhanced gas mixing by the high velocity gas flow injected (10). Application can be either continuous or during expiration only. Preliminary data indicate that $PaCO_2$ is decreased in direct proportion to TGI flow and that TGI is more effective the greater the baseline $PaCO_2$ (10). Of concern is that TGI elevates peak alveolar pressures, increases V_T and causes auto-PEEP (11). As a result, it appears that expiratory phase TGI or volume-adjusted TGI would be the safest approach to TGI (11). With volume-adjusted TGI, V_T during volume-controlled ventilation is decreased by the TGI volume delivered during the inspiratory phase. Although TGI is promising, it must be considered experimental; problems with humidification, system over-pressure, ability to monitor changes in peak alveolar pressure and auto-PEEP must be solved before TGI can be recommended for general clinical use.

PRESSURE- VERSUS VOLUME-TARGETED VENTILATION

There are distinct advantages as well as disadvantages of both pressure targeting and volume ventilation (Table 3). The decision to employ one or the other approach is generally based on personal bias, and which of the advantages and disadvantages are considered most important. Review of the literature with a focus on well-defined, controlled studies indicates that there are no differences in physiological effects, development of barotrauma or acute lung injury, or outcome between pressure and volume ventilation regardless of the inspiratory:expiratory (I:E) ratio used (12,13). This is particularly true when pressure ventilation is contrasted to volume ventilation with a decelerating flow waveform and an end-inspiratory plateau (14).

Pressure-targeted ventilation – advantages and disadvantages: The major advantage of pressure-targeted ventilation is that peak inspiratory and alveolar pressures are maintained at a constant level. This may decrease the likelihood of localized over-distension with associated barotrauma and acute lung injury. In addition, pressure ventilation is able to respond on a breath-to-breath basis to changes in ventilatory demand, thus increasing patient-ventilator synchrony and reducing patient effort. The major disadvantage is that V_T varies as impedance changes, increasing the likelihood of blood gas alterations and making it more difficult to identify major alterations in impedance rapidly.

TABLE 3
Advantages and disadvantages of pressure- and volume-targeted ventilation

Pressure-targeted ventilation
Advantages
• Peak alveolar pressure is limited
• Flow responds to patient demand
• Increased patient-ventilator synchrony
Disadvantages
• Tidal volume variable
• $PaCO_2$ variable
Volume-targeted ventilation
Advantages
• Tidal volume constant
• $PaCO_2$ constant
• Easily identifiable changes in peak inspiratory pressure as impedance changes
Disadvantages
• Peak alveolar pressure variable
• Inability to respond to changes in patient ventilatory demand

Volume-targeted ventilation – advantages and disadvantages: The major advantage of volume-targeted ventilation is the delivery of a constant V_T. This ensures a consistent level of alveolar ventilation and results in easily identifiable changes in peak inspiratory pressure as impedance to ventilation changes. However, with volume ventilation, peak alveolar pressure may change dramatically as impedance changes, potentially increasing the risk of ventilator-induced lung injury. In addition, volume ventilation is unable to respond to changes in patient demand. As a result, patient-ventilator dyssynchrony and increased patient effort can be anticipated with volume-targeted ventilation.

Combined pressure/volume modes: A number of manufacturers have developed modes (pressure augmentation, volume support, pressure-regulated volume control) of ventilation that combine the beneficial aspects of both pressure and volume ventilation and limit the disadvantages of each. Preliminary data indicate that these approaches are successful in marrying the two targets (15,16). As a result, based on current literature, one must speculate whether standard volume ventilation is ever indicated. In both the assisted and controlled ventilated patient, pressure targeted or combined pressure- and volume-targeted approaches appear to be better at preventing circumstances associated with ventilator-induced lung injury and improving patient-ventilator synchrony.

INVERSE RATIO VENTILATION

As discussed earlier, no differences have been reported between volume and pressure ventilation compared at normal or inverse I:E ratios (12,13). However, these studies have helped to focus attention on the methods available to increase

mean airway pressure in order to improve oxygenation. This discussion has particular relevance in the ARDS patient in whom oxygenation is a particular problem. Of primary concern is setting PEEP at a level that ensures recruitment of lung units (about 12 to 15 cm H_2O). Once PEEP is established at this level, oxygenation is directly related to mean airway pressure. Extending inspiratory time is one method of increasing mean airway pressure without increasing peak alveolar pressure. The emphasis should not be establishing a specific I:E ratio but establishing the mean airway pressure that allows the oxygenation target to be met. Inspiratory time extension should be limited by the development of auto-PEEP (17). Once auto-PEEP starts to develop, increases in inspiratory time should stop and other approaches (set PEEP) to increasing mean airway pressure should be used. Auto-PEEP should be avoided because it results in a much less uniform increase in lung unit total PEEP and functional residual capacity than applied PEEP (16). Because auto-PEEP depends on local lung unit time constants, lung units that are most stiff have the least auto-PEEP, whereas lung units that are most compliant have the greatest increase in auto-PEEP (17).

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