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FOCUS ON: CONSIDERATIONS DURING THE POSTOPERATIVE PERIOD

Ventilation and ventilators in the ICU: What every intensivist must know

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Summary In the past two decades, important advances have revolutionized the world of mechanical ventilation. A huge volume of research has changed the approach to patients with respiratory failure from high tidal volumes and low PEEP, to a more gentle approach that understands the background of lung pathology and tries to avoid aggravating it. Lung-protective strategies have become a standard part of most intensive care unit ventilation policies. Advances in technology have led to the introduction of newer ventilators and ventilatory modes that respond to changes in patient physiology and patient demand, and promise to provide a more intelligent method of patient care. This article is intended to provide a brief review of the science behind mechanical ventilation as it stands today.

Introduction

Mechanical ventilation remains to be one of the most challenging tasks facing physicians in the ICU. As technology advances, more and more complicated modes and ventilators are introduced. A basic understanding of mechanical ventilation is a must for every physician who is likely to be responsible for a ventilated patient. An ability to make use of the many new advances in this field may lead to more efficient gas exchange, improved patient–ventilator interaction, and therefore better patient comfort, less ventilator days and a more rapid weaning process. Furthermore, speedier weaning of patients can translate into a significant reduction in costs. In a recent study, the mean incremental cost of mechanical ventilation in intensive care unit patients was shown to be $1522 per day.[1]

Historical background

In 1929, Drinker and McKhann introduced the first practical means of ventilatory support, namely the 'iron lung'. This was a device consisting of a chamber in which the patient was laid, covering all but his head and neck. Negative pressure was

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then created using a vacuum pump, enabling expansion of the patient's chest. The drop in intrapulmonary pressure thus created allowed air to flow into the patient's lungs.[2]

Over 20 years later, the polio epidemic led an anaesthetist in Copenhagen, Bjørn Ibsen, to recommend positive pressure ventilation via tracheostomies, thereby reducing the mortality from 84% at the start of the outbreak, to 26%.[3] Interestingly, the ''ventilators'' used at that time were 1400 medical students. In the few years that followed, several ventilators were introduced which allowed setting of a minute volume. This method, besides freeing the medical students, provided a smaller, cheaper, and more practical method of mechanical ventilation, and paved the way for the development of intermittent positive pressure ventilation (IPPV) as we know it today.

Indications for initiation of ventilatory support

Mechanical ventilation, in itself, is not a cure for respiratory failure. It should be thought of as a method to support patients, enabling survival while the cause of the respiratory failure is attended to and reversed. A corollary would be that mechanical ventilation is not indicated in moribund patients in whom alleviation of the precipitating factor is not anticipated. In addition, when considering initiation of mechanical ventilation, much thought must be given to the harmful ventilation-related consequences that may complicate the course of the patient's illness.

The main objectives of initiating mechanical ventilation are: (1) to reverse hypoxaemia; (2) to reverse acute respiratory acidosis, intending to relieve life-threatening acidemia rather than to normalize $PaCO_2$; and (3) to relieve respiratory distress and elevated work of breathing. From these objectives are derived the classical indications for initiation of ventilatory support:

- arterial $PaO_2 < 60$ mmHg (on supplemental oxygen),
- alveolar-to-arterial oxygen difference of more than 350 mmHg (on $FiO_2 = 1$),
- arterial $PaCO_2 > 50$ mmHg (in the absence of chronic disease),
- evidence of elevated WOB:
  - respiratory rate of more than 35 breaths per minute,
  - tidal volume of less than 5 mL/kg,
  - vital capacity of less than 15 mL/kg,
  - maximum inspiratory pressure of less than 25 cm H_2O,
  - presence of retractions or nasal flaring,
  - paradoxical or divergent chest motion.

Modes of mechanical ventilation

A. Common modes of ventilation

Continuous mandatory ventilation (CMV)
This mode delivers a set number of mechanical breaths, with a preset tidal volume, at regular intervals. Classically, the patient is not allowed to breath in between mandatory breaths. This has led to the recommendation that this mode should best be reserved for the patient with no respiratory effort. Indeed, many ventilator brands available today have no true CMV mode; the assist-control mode is provided instead. In the paralysed or apneic patient, assist-control would function as if it were CMV.

Assist-control ventilation (A/C)
In A/C, the ventilator delivers preset breaths with every inspiratory effort the patient initiates. The patient cannot, however, breathe independently of the ventilator. The patient can initiate inspiration and control the breathing frequency; therefore, the respiratory pattern may have irregular intervals depending on the timing of the patient's efforts. If the patient fails to initiate inspiration, the ventilator automatically goes into a backup mode and delivers the preset rate and tidal volume until an inspiratory effort is sensed.

Synchronized intermittent mandatory ventilation (SIMV)
In SIMV, the ventilator delivers preset breaths in coordination with the respiratory effort of the patient. Spontaneous breathing is allowed between breaths. Synchronization of the delivered breath to the inspiratory effort of the patient serves to avoid the potentially hazardous sequelae of delivering a mechanical breath to a patient who is in mid- or end-inspiration. SIMV was initially presumed to provide a means of unloading respiratory muscles; much evidence however has shown that this is not the case.[4] Furthermore, as a mode of weaning, SIMV was found to be inferior to both spontaneous breathing trials and pressure support ventilation (PSV) in several randomized trials.[5–7]

Pressure support ventilation
PSV is a popular mode of mechanical ventilation in spontaneously breathing patients. It is essentially a ventilator-generated flow augmentation of each individual breath that a patient triggers. Only the level of support pressure is set, as opposed to tidal volume and background rate in the previously mentioned modes. The inspiratory flow augmentation continues until the flow rate decreases to 20–25% of the peak flow rate. The decelerating flow pattern thus created allows a more linear flow of gas in the airways, and more efficient gas distribution in-between lung units.

PSV has been used to limit barotrauma and to decrease the work of breathing, which can result from endotracheal tube and breathing circuit resistance. In a comparison between PSV and A/C, patients on PSV showed significantly higher tidal volume, minute ventilation, and inspiratory time in association with a significantly lower pressure in the airway.[8] PSV is further recommended as an adjunct to mechanical ventilation, when A/C or SIMV modes are used.[9]

Pressure control ventilation (PCV)
PCV is a pressure-limited, time-cycled ventilatory mode. The desired pressure level is set, as is inspiratory time and respiratory rate. With every breath, the ventilator delivers an inspiratory flow until the pre-set airway pressure limit is achieved. As with PSV, the flow decelerates throughout inspiration, however the cycle is time, not flow. As with PSV, decreased flow at the end of inspiration results in less turbulent, more laminar flow, and a more even distribution of the breath. This decelerating waveform has long been shown to improve lung mechanics and gas exchange during mechanical ventilation.[10]

PEEP/CPAP
PEEP is the level of positive pressure that is maintained in the airways at the end of expiration. CPAP is an actual mode of ventilation (as opposed to PEEP), and this term is usually referred to when the patient is allowed some degree of spontaneous breathing. Insertion of an endotracheal tube causes an obligatory bypass of the effect of the epiglottic closure on maintenance of functional residual capacity, otherwise known as ''physiological PEEP''. Physiological PEEP is usually considered to be equal to 3–8 cm H_2O, hence the recommendation that a minimum of 5 cm H_2O of PEEP/CPAP should be delivered to each and every intubated patient, to avoid atelectasis.

In patients with increased shunt fraction and consequent hypoxaemia, the application of PEEP has been used to improve gas exchange. How this is achieved is not totally understood, but several mechanisms have been suggested, including the stabilization of small airways,[11] increase in lymphatic drainage, and the decrease of transmural pressure with a resultant decrease of net fluid filtration across the capillary membrane.[12] However, a study by Malo et al.[13] reported a *redistribution* of lung oedema rather than a decrease in the total amount of lung water.

Chronic obstructive pulmonary disease (COPD) is characterized by dynamic hyperinflation and intrinsic PEEP (PEEPi), which may cause patient–ventilator asynchrony and consequently increase the work of breathing. External PEEP is set to counteract the effect of PEEPi on ventilator triggering and therefore may improve the patient's comfort. External PEEP may also stabilize upper airway function during sleep, increase functional residual capacity, or decrease the development of micro- and macroatelectasis.[14]

B. Newer modes of mechanical ventilation

Volume support ventilation (VSV) and volume-assured pressure support (VAPS)
In an attempt to overcome a major pitfall of PSV, these newly available ventilatory modes have been designed to guarantee a minimum tidal volume that is considered acceptable for the patient. In VSV, the ventilator starts with a test breath to determine compliance. Thereafter, it constantly adjusts the pressure support level (in increments of 3 cm H_2O) in order to maintain the required tidal volume. In VAPS, the ventilator initially delivers pressure support, continually comparing the inspired volume to the set tidal volume. If the inspired volume is found to be below target, the pressure-supported component is complemented with a constant-flow volume-controlled breath.

Pressure-regulated volume control (PRVC)
Considered the controlled form of VSV, PRVC entails a test breath to determine compliance, then delivers pressure-controlled breaths, the pressure limit of which is adjusted in order to maintain tidal volume at a preset acceptable level. Like VSV, this is achieved in increments of 3 cm H_2O.

Proportional assist ventilation (PAV)
PAV is a unique form of ventilatory support that aims at coupling the patient's inspiratory drive with the ventilator pressure output. Thus the higher the ventilatory drive, the more pressure the ventilator will generate in that particular breath. Flow,

volume, and pressure are all proportional to patient effort. The patient is allowed to follow whatever breathing pattern he is comfortable with, with a level of ventilatory support that follows his drive. This mode has been reported to improve patient–ventilator synchrony and comfort.[15]

Airway pressure release ventilation (APRV)
APRV is a mode of ventilation that entails a continuous positive airway pressure (CPAP) (termed P_{high}), with intermittent, time-cycled, transient release of pressure to a lower value (P_{low}). Spontaneous ventilation is allowed throughout, independent of ventilator cycle. This mode couples the recruiting effects of CPAP with the superior ventilation/perfusion matching of spontaneous breathing. The intermittent releases act as a supplement for minute ventilation, aiding in CO_2 removal without the threat of overdistension (as with a regular positive pressure breath). Recent studies of APRV[16,17] have shown favourable results on gas exchange and distribution of ventilation, whether used alone, or coupled with prone positioning.

High-frequency oscillatory ventilation (HFOV)
This unique mode of ventilation utilizes rapid respiratory rates, more than four times the normal. At high frequencies, tidal volumes may be less than deadspace. The aim is to hold the lung in a state of recruitment, while maintaining ventilation, probably by facilitated diffusion. Precautions must be taken during HFOV; complications may include pneumothorax, and acute respiratory acidosis should partial endotracheal tube obstruction occur. Overall, HFOV is a ''rescue'' mode of ventilation, to be considered in hypoxemic patients refractory to other more conventional ventilation methods. Although most studies on HFOV have been on its use in neonates and children, several studies on adults[18,19] have demonstrated beneficial effects on oxygenation and ventilation, as well as its safety as a rescue therapy for patients with severe oxygenation failure.

Modes of the future: neurally adjusted ventilatory assist (NAVA)
The ideal method of modifying breath-to-breath ventilator gas delivery would theoretically be based on respiratory centre output. As this is clinically impossible, researchers have strived to reach a feasible alternative. NAVA is a novel system, with a simple yet utterly intelligent idea, depending on electrodes placed in the lower oesophagus that track diaphragmatic activity. This revolutionary system allows ventilatory support to be continually readjusted, according to the varying demands/needs of the patient. Furthermore, it incorporates the breath-to-breath variability that characterizes a natural breathing pattern.[20] This promising invention may provide great changes in the way patients will be ventilated in years to come.

C. Noninvasive ventilation (NIV)

Mechanical ventilation has been shown to be successful when applied noninvasively, through a face or nasal mask, in selected circumstances. Avoiding the hemodynamic sequelae of tracheal intubation, and avoidance of ventilator-associated pneumonia are some of its advantages. The appropriate patients would typically be those with mild to moderate respiratory failure, provided the patient is conscious (and cooperative) enough to follow commands, and not at risk for pulmonary aspiration. Patients who have excessive secretions, are medically unstable, or are at risk for respiratory arrest are not candidates for this modality of ventilatory support.

Although most widely used for acute exacerbation of COPD or asthma, NIV can be used in other types of acute respiratory failure such as acute pulmonary oedema. In a recent meta-analysis, significant reductions in mortality and the need for subsequent mechanical ventilation were associated with NIV in patients with various types of acute respiratory failure, albeit the benefit was more pronounced in the subgroup of patients with COPD.[21]

NIV is most commonly applied as CPAP or biphasic positive airway pressure (BiPAP). BiPAP is basically a form of CPAP that alternates between high and low positive airway pressures, permitting inspiration (and expiration) throughout. Some critical care ventilators have the option for being used noninvasively, avoiding the need for critical care units to purchase further equipment. Mask size, degree of fitting to the patient's face, and type (face mask/nasal mask) are other considerations that may alter the degree of patient benefit and comfort with NIV. Complications of NIV are generally minimal, and include claustrophobia, nasal bridge ulceration, mucosal dryness, eye irritation, and gastric distension.

Complications of mechanical ventilation

Over the past two decades, critical care physicians have become more and more aware that mechanical

ventilation may lead to, or aggravate, lung injury. Common types of injury reported include interstitial emphysema, cyst formation, and pneumothorax. Initially, excessive airway pressures or *barotrauma* was thought to be the primary cause of ventilator-associated lung injury. However, it is currently understood that several other mechanisms of injury may occur in ventilated patients, namely: volutrauma, atelectrauma, and biotrauma.

Barotrauma

In previous eras, mechanical ventilation was instituted using high tidal volumes, in an attempt to avoid atelectasis. Usual tidal volumes used ranged from 10 to 15 mL/kg. Exposing diseased lungs to static alveolar pressures above 50 cm H_2O caused alveolar air leaks resulting in pneumothorax and other forms of extra-alveolar air.[22]

Volutrauma

Dreyfuss et al.[23] introduced the term volutrauma in 1988, after demonstrating that lung volume was the major determinant of increased lung water. In this study the consequences of normal tidal volume ventilation in mechanically ventilated rats at a high airway pressure were compared with those of high tidal volume ventilation at high or low airway pressures. High pressure, low volume-ventilated rat lungs were not different from those of controls. By contrast, the lungs from the groups submitted to high volume ventilation had significant permeability type oedema. It has now become understood that transpulmonary pressures in the excess of 35 cm H_2O can cause overdistension injury to the lungs. Moreover, further research has lead investigators to realize that in severe acute respiratory distress syndrome (ARDS), as little as 30% of lung units may remain healthy. The previous practice of using high tidal volumes, although tolerated by normal lungs, are now known to be the cause of much overdistension injury to these ''baby lungs''.[24]

Atelectrauma

Another factor in the causation of ventilator-associated lung injury appears to be ventilation at very low lung volumes. Repeated opening and closing of airway and alveolar ducts may result in a shear injury, with a consequent significant progression of lung damage.[25] In the patient with very low compliance, in whom tidal volumes must be kept low, addition of sufficient PEEP to maintain end-expiratory lung volume may play a significant role in the avoidance of further lung injury.[26]

Biotrauma

In addition to the previously mentioned mechanisms of ventilator-associated lung injury, mechanical ventilation may initiate mediator-related lung damage. Zhang et al.[27] demonstrated that polymorphonuclear leucocytes can be activated by conventional high-volume mechanical ventilation, as manifested by a significant increase in oxidant production, CD18, and CD63 surface expression, and shedding of L-selectin. They further demonstrated that these findings could be avoided by using lung-protective strategies.

Lung-protective ventilatory strategies

A lung-protective strategy aims to avoid the aforementioned mechanisms of ventilator-associated lung injury. This may be achieved by using low tidal volumes to avoid overdistension (6–8 mL/kg), enough PEEP to avoid atelectasis and atelectrauma, permissive hypercapnia, and (more recently) the implementation of lung recruitment manoeuvres to maintain an ''open lung''.

Researchers have long used pressure–volume curves to study the characteristics of lung compliance. In earlier studies,[28] it was recommended that PEEP should be set to a level slightly above the lower inflection point of the inflation limb. This was in an attempt to maintain lung volumes that would avoid atelectasis, and the damage that ensues. More recent studies[29] have suggested that PEEP could be better targeted according to the slope of deflation-limb compliance, because this measure may more accurately reflect global alveolar closing pressures. That is, setting PEEP levels according to the deflation curve may be a method that points out the pressure below which the lung units start to *close*. This is as opposed to the inflation curve in which the lower inflection point rather denotes the pressure at which the lungs start to *open*.

Permissive hypercapnia is a ventilatory strategy that assigns higher priority to avoiding injurious pressure than to maintaining normal levels of ventilation. Allowing the $PaCO_2$ to rise above baseline values is perhaps the simplest technique for reducing the ventilatory workload, the pressure cost of breathing, or the total number of machine cycles needed per minute. A slow rise of $PaCO_2$ is commonly well-tolerated by patients, and the ensuing mild respiratory acidosis is a small price

to pay in exchange for avoidance of the damaging effects of tidal volumes targeting normocapnia.

The concept of open lung was coined by Amato et al.[30] as they evaluated the impact of a new ventilatory strategy directed at minimizing "cyclic parenchymal stretch." They demonstrated a higher weaning rate in early ARDS patients who received mechanical ventilation based on a new approach, compared to conventional volume-cycled ventilation with tidal volumes of 12 mL/kg, PEEP guided by FiO_2 and hemodynamics, and normal $PaCO_2$ levels. This new approach consisted of maintenance of end-expiratory pressures above the lower inflection point of the pressure–volume curve, a tidal volume of less than 6 mL/kg, peak pressures maintained below 40 cm H_2O, permissive hypercapnia, and stepwise utilization of pressure-limited modes. They could not, however, demonstrate a significantly improved survival.

Another subsequent study,[31] assessed the effects of a lung-protective ventilation strategy that combined both volume- and pressure-limited and open-lung approaches. Patients were randomized to either a conventional study group receiving tidal volumes of approximately 12 mL/kg of measured body weight and mean PEEP of approximately 8 cm H_2O during the first 7 days, or to a lung-protective ventilation group receiving initial tidal volumes of approximately 6 mL/kg of measured body weight. Tidal volumes were decreased further in the lung-protective group if inspiratory airway pressures exceeded 40 cm H_2O. The mean PEEP level in the lung-protective ventilation group was 16.4 cm H_2O during the first 36 h. Recruitment manoeuvres that sustained increases in airway pressure at 35–40 cm H_2O were used in the lung-protective ventilation group to reverse atelectasis of some lung units. Survival, rate of weaning from mechanical ventilation, and frequency of barotrauma events were improved in the lung-protective ventilation group.

Recruitment manoeuvres attempting to "open" the lung and keep it open have been described in a multitude of forms[32–34] and may provide a basis for a readily available and low-cost modality of treatment for patients with ARDS and acute lung injury. The basic idea behind the various designs of these manoeuvres is to inflate the atelectatic lung to a volume that ensures opening of most lung units, and avoiding its return to atelectasis.

Conclusion

Major advances have been made in the field of mechanical ventilation, both in the understanding of the mechanisms of lung injury, and in the tools provided to combat them. Ventilator manufacturers are introducing revolutionary new modes, some of which use complex technology and intricate algorithms, intended to provide better patient comfort, and ultimately aid in patient weaning.[35]

The initial choice of ventilation mode should ultimately be left to the discretion of the attending physician and/or institution policy. In an urgent situation, physicians should be encouraged to use the mode with which they are more familiar. A more comprehensive plan may subsequently be tailored for the patient, according to his condition. However, basic background knowledge of how to use new tools and the mechanisms of lung injury (and protection) can aid in planning patient care, and achieving goals of shorter ventilation days and improved outcomes for patients with respiratory failure.

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