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Mechanical ventilation strategies

Martin Keszler

Department of Pediatrics, Alpert Medical School of Brown University, Women and Infants Hospital of Rhode Island, 101 Dudley Street, Providence, RI 02905, USA

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A B S T R A C T

Although only a small proportion of full term and late preterm infants require invasive respiratory support, they are not immune from ventilator-associated lung injury. The process of lung damage from mechanical ventilation is multifactorial and cannot be linked to any single variable. Atelectrauma and volutrauma have been identified as the most important and potentially preventable elements of lung injury. Respiratory support strategies for full term and late preterm infants have not been as thoroughly studied as those for preterm infants; consequently, a strong evidence base on which to make recommendations is lacking. The choice of modalities of support and ventilation strategies should be guided by the specific underlying pathophysiologic considerations and the ventilatory approach must be individualized for each patient based on the predominant pathophysiology at the time.

1. Introduction

Despite appropriate emphasis on non-invasive respiratory support when feasible, mechanical ventilation remains an important therapy in the sickest infants. Although frequently life-saving, invasive mechanical ventilation has many untoward effects on the cardiovascular system, brain and lungs. Whereas preterm infants are most vulnerable, term newborns are not immune from these adverse effects [1,2]. The endotracheal tube (ETT) acts as a foreign body, quickly becoming colonized and acting as a portal of entry for pathogens, increasing the risk of ventilator-associated pneumonia and late onset sepsis [3]. For these reasons, avoidance of mechanical ventilation in favor of non-invasive respiratory support remains the most important step in preventing neonatal morbidity. When mechanical ventilation is required, the goal is to liberate the patient from invasive ventilation as soon as feasible in order to minimize ventilator-associated lung injury (VALI) and other ventilator-related complications.

2. Ventilator-associated lung injury

Many terms have been used to describe the mechanism of injury in VALI. Barotrauma refers to damage caused by pressure. The conviction that pressure is the key determinant of lung injury has led clinicians to focus on limiting inflation pressure, sometimes to the point of precluding adequate ventilation. However, there is convincing evidence that high pressure by itself, without correspondingly high volume, does not cause lung injury. Rather, injury related to high inflation pressure is mediated through the tissue stretch, resulting from excessive tidal volume (V_T) or from regional overdistention when ventilating a lung with extensive atelectasis. Dreyfuss and colleagues demonstrated more than twenty years ago that severe acute lung injury occurred in small animals ventilated with large V_T, regardless of whether that volume was generated by positive or negative inflation pressure [4]. In contrast, animals exposed to the same high pressure but with an elastic bandage constraining chest excursion to limit V_T delivery suffered substantially less acute lung injury. In a similar study, Hernandez et al. showed that animals exposed to pressure of 45 cmH_2O did not show evidence of acute lung injury when their chest and abdomen were enclosed in a plaster cast [5]. Volutrauma is injury caused by over-distention and excessive stretch of tissues, which leads to disruption of alveolar and small airway epithelium, resulting in acute edema, outpouring of proteinaceous exudate, release of proteases, cytokines and chemokines, which in turn lead to activation of macrophages and invasion of activated neutrophils. Collectively, this latter process is referred to as biotrauma. Another important concept is that of atelectrauma, or lung damage caused by tidal ventilation in the presence of atelectasis [6]. Atelectrauma causes lung injury via several mechanisms. The atelectatic portion of the lungs experiences increased surfactant turnover and high critical opening pressure. Shear forces at the boundary between aerated and atelectatic parts of the lung cause structural tissue

E-mail address: mkeszler@wihri.org.

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damage. Ventilation of injured lungs using inadequate end-expiratory pressure results in repeated alveolar collapse and expansion with each inflation, which rapidly injures the lungs. Finally, when a large portion of the lungs is atelectatic, gas entering the lungs will preferentially distend the aerated portion of the lung, which is more compliant than the atelectatic lung with its high critical opening pressure. This fact is evident from LaPlace's law and corroborated by experimental evidence, showing that the most injured portion of the lung was the aerated non-dependent lung [7].

2.1. How can we reduce VALI?

As is evident from the prior discussion, the process of lung damage from mechanical ventilation is multifactorial and cannot be linked to any single variable. Consequently, any approach to reducing lung injury must be comprehensive and begin with the initial stabilization of the infant in the delivery room [8].

2.2. Non-invasive respiratory support

There is little doubt that, in general, avoiding mechanical ventilation will reduce iatrogenic lung injury, but this issue has been studied much more thoroughly in preterm infants [9], with little available information regarding the use of non-invasive support in late preterm and term newborns. Only observational studies are available and thus there is wide variation in practice style. Nonetheless, based on sound physiologic principles, it is reasonable to attempt initial support with non-invasive modalities in an effort to prevent progression to more severe illness and need for mechanical ventilation.

3. Strategies of mechanical ventilation

Mechanical ventilators are devices designed to replace or augment the patient's inadequate respiratory effort. Ventilators are simply tools in our hands and we need to employ them thoughtfully in order to optimize outcomes. There are many devices and modes of ventilation to choose from, with limited high-quality data to guide the clinician's choice. The goal of mechanical ventilation is to maintain acceptable gas exchange with a minimum of adverse effects and to wean from invasive support as expeditiously as possible. Because of the wide range of clinical conditions of neonatal patients, no simple rules can define indications for intubation and initiation of mechanical ventilation. Similarly, “cookbook” settings that are often provided in texts on this subject have limited utility. Instead, choice of modalities of support and ventilation strategies should be guided by the specific underlying pathophysiologic considerations. In the following paragraphs, I review basic concepts of synchronized mechanical ventilation and general concepts of lung-protective ventilation strategies, followed by a discussion of specific strategies suitable for treatment of term and late preterm infants with respiratory failure.

3.1. Basic modalities of synchronized ventilation

Despite lack of a strong evidence base, the use of synchronized mechanical ventilation has become standard in our neonatal intensive care units (NICUs), but there is no clear consensus about which modality of synchronization is optimal. Synchronization of ventilator inflations with the infant's spontaneous breaths makes it possible to minimize sedation and muscle paralysis and to maximally utilize the patient's spontaneous respiratory effort. Whereas allowing the patient to breathe spontaneously during mechanical ventilation has clear advantages, it makes managing mechanical ventilation more challenging for the clinician. In order to employ assisted ventilation optimally, the clinician must understand the complex interaction between the awake, spontaneously breathing infant and the various modalities of synchronized ventilation. A key ingredient is an appreciation of the additive nature of the patient's own inspiratory effort and the positive pressure generated by the ventilator. The V_T entering the infant's lungs is driven by the sum of the negative inspiratory effort of the infant and the positive inflation pressure from the ventilator, which together constitute the transpulmonary pressure.

3.2. Synchronized intermittent mandatory ventilation (SIMV)

This is a basic synchronized modality that provides a user-set number of inflations in synchrony with the infant's breathing. If no spontaneous effort is detected during a trigger window, a mandatory inflation is delivered. Spontaneous breaths in excess of the set ventilator rate are not supported. In small preterm infants this results in uneven V_T values and high work of breathing because of the high airway resistance of the narrow ETT, coupled with the limited muscle strength and mechanical disadvantage of the infant's excessively compliant chest wall, but this is much less of a problem with term and late preterm infants. SIMV allows the operator to set the ventilator rate as well as inflation pressure and PEEP. Weaning is accomplished by gradual lowering of both rate and inflation pressure.

3.3. Assist control (AC)

Assist control is a modality that supports every spontaneous breath (“assist”) and provides a backup minimum rate of ventilator inflations in case of apnea (“control”). AC is time-cycled and can be pressure or volume controlled. Supporting every breath leads to more uniform V_T and lower work of breathing than SIMV. The goal is to have the infant and the ventilator work together, resulting in lower ventilator pressure. A backup ventilator rate provides a minimum rate in case of apnea and should be set just below the infant's spontaneous rate, usually at 30–40 inflations per minute. A backup rate that is too low will result in excessive fluctuations in minute ventilation and oxygen saturations during periods of apnea. Because the infant controls the inflation rate, gradual withdrawal of support is accomplished by lowering the peak inflation pressure, reducing the support provided to each breath and allowing the infant to gradually take over the work of breathing.

3.4. Pressure support ventilation (PSV)

A variety of modalities are referred to as PSV, a situation that greatly complicates communication. In specialty neonatal ventilators, pressure support ventilation is a flow-cycled and pressure-controlled mode that supports every spontaneous breath just like AC but is flow-cycled. Flow cycling means that inflation is terminated when inspiratory flow declines to a preset threshold, usually 5–20% of peak flow, eliminating inspiratory hold (prolonged inflation time, T_I) and providing more complete synchrony with less fluctuation in intrathoracic and intracranial pressure that occurs when infants exhale during inspiratory hold. Conveniently, PSV automatically adjusts T_I to be appropriate for the changing lung mechanics of the patient. Changing from time-cycled AC to PSV typically results in a shorter T_I and therefore lower mean airway pressure (P_{AW}). Therefore, unless positive end-expiratory pressure (PEEP) is adjusted to maintain P_{AW}, changing to PSV may lead to atelectasis. As with triggering, a substantial leak around the ETT may affect flow cycling.

Similar to AC, a backup rate will maintain a minimum inflation

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