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Ventilatory strategies in the neonatal and paediatric intensive care units

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EDUCATIONAL AIMS

- To understand mechanical ventilation, techniques available and limitations of application in children
- From a literature review with an emphasis on practical application, to be informed of recent advances in therapy

KEYWORDS
mechanical ventilation;
paediatric;
neonatal;
non-invasive mechanical ventilation;
high frequency ventilation

Summary Mechanical ventilation is a common form of support in the modern day intensive care unit (ICU). In order for the clinician better to understand and apply mechanical ventilation, it is important that they understand the physiological principles of ventilation. This review describes these basic concepts; parameters of mechanical ventilation, high frequency ventilation and non-invasive ventilation. An overview of ventilatory strategies for four common diseases seen in paediatric and neonatal ICUs will be discussed.

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INTRODUCTION: WHAT IS MECHANICAL VENTILATION?

Mechanical ventilation is an attempt to mimic the respiratory system's physiological function of gas exchange at the time of either impending or actual respiratory failure. The conceptual appreciation of artificial ventilation has been attributed to Vesalius (1514–1564), who attempted to mimic ventilation by blowing air into a tube in the trachea of a small mammal. Mechanical ventilation was a clinical reality by the 19th century, but the majority of ventilators were negative pressure devices. The first positive pressure ventilator was developed by Emerson and made its appearance early in the 20th century. With the polio epidemic in the 1950s, mechanical ventilation became widely accepted as a form of life support.

Mechanical ventilation is a supportive, non-therapeutic technology used to perform the work of breathing for patients who are unable to do so on their own. As such, the requirement for this type of support is among the most common reasons for admission to an intensive care unit (ICU).[1] Respiratory failure occurs during conditions of inadequate exchange of oxygen and/or carbon dioxide. Such inadequate gas exchange can occur as a result of lung disease, cardiac dysfunction, neurological abnormalities, multiorgan system dysfunction or secondary effects of surgery or cardiopulmonary bypass.

At first glance, ventilators can be separated into two categories: negative pressure ventilators or 'iron lungs' (rarely used in the modern ICU) and positive pressure

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ventilators. Positive pressure ventilation can be instituted with an endotracheal tube or tracheostomy (invasive positive pressure ventilation), or with a mask (non-invasive positive pressure ventilation).

PHYSIOLOGICAL PRINCIPLES

The mechanical function of the respiratory system is best described by the equation of motion, where:

$$\text{Driving pressure(Pdr)} = [\dot{V} \cdot (1/\dot{C})] + [R \cdot V] + [I \cdot V] + k$$

The last term (I·V), which incorporates the accelerative energy of respiration, is considered not to be significant at normal respiratory rates and is therefore discarded. The driving pressure is what causes gas movement through the respiratory system.

Gas moves within the respiratory tract because of a pressure gradient, i.e. gas flows from a higher pressure to a lower pressure. Normal inspiration is accomplished by the integrated contraction of the respiratory muscles resulting in an expansion of the thoracic cavity with a decrease in alveolar pressure (Fig. 1). If the alveolar pressure (Palv) is lower than the pressure at the mouth or nose, then air flows into the lung with a change in lung volume. At all ages, including premature infants, a normal Pdr for the respiratory system during spontaneous ventilation is about 8 cmH_2O (0.8 kPa).[2] Conversely, gas will only flow out of the lungs when the alveolar pressure is higher than atmospheric pressure (Palv > Pbar). Except during heavy exercise, the Palv is generated passively by the elastic recoil forces of the lung and chest wall (Pel). Thus, normal exhalation is passive, does not involve work by the patient's muscles, and the pattern of flow is determined by the mechanical properties of the lung and chest wall. Therefore, this pressure differential, or driving pressure (Pdr), causes gas to flow into the lungs.[2] Mechanical ventilators, which raise the pressure at the opening to the airway, conceptually use Pdr to 'push' air into the lungs (positive pressure ventilation).

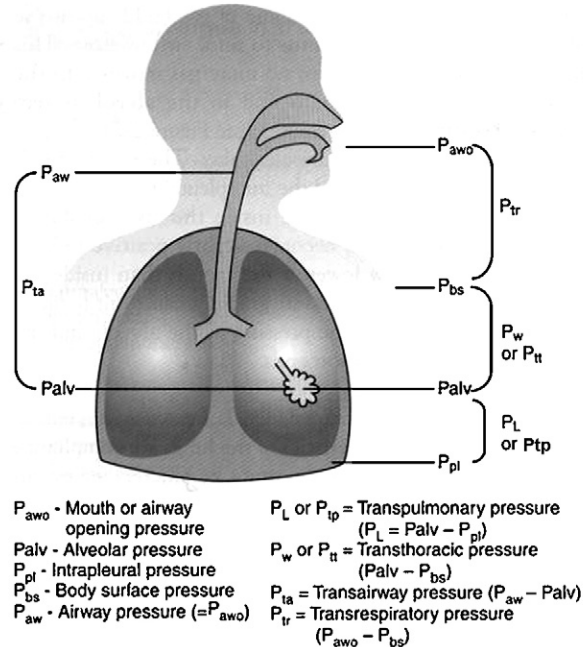

Figure 1 Various pressures and pressure gradients of the respiratory system. (Reproduced from Wilkins et al.,[32] with permission).

There are two major mechanical characteristics of the lung that are important during mechanical ventilation: compliance (describing the viscoelastic forces with the lung tissue) and resistance (friction generated by airflow). Compliance (C) and its reciprocal elastance (E) arise from the deformation of lung tissue and thorax during active inspiration, thus describing the elastic forces that oppose change in lung volume. Two separate forces oppose the inflation of the lung: elastic and frictional forces. Changes in compliance will affect the pressure required to inflate the lungs to the same volume.[3] Therefore, the monitoring of compliance during mechanical ventilation can be a useful tool to describe how the volume of the lung changes for a known pressure change.

Frictional forces result from the resistance of the tissues and organs to the flow of gas as it moves through the airways. Thus, resistance is related to the gas composition (viscosity) and the structure of the airways.[2] While the tissue resistance does not change appreciably in most cases, the resistive component due to the airway can be quite variable depending on the disease state of the patient. Airway resistance depends on gas viscosity, density and flow rate, along with the diameter of the airway lumen (a very large element) and the length of the airway.

In mechanical ventilation, the size (diameter) of the endotracheal tube can be a major determinant of airway resistance. Indeed, a higher airway resistance will mean that more driving pressure must be applied to attain adequate flow of gas to inflate the lung within the time allocation for inspiration.

The lungs are, by nature, heterogeneous with different lung units (acini) having somewhat different compliance and/or resistance. The net effect of this heterogeneity will be that the lung units will fill and empty at different rates (resistive characteristics) such that the distribution of air in the lung is asymmetrical in both volume and sequence. The time taken to fill and empty the lung is referred to as a time constant for the lung (τ): this is the product of the compliance and resistance of the lung, and represents the overall combined performance of the organ. This concept is of importance for expiration in that the Pdr to empty the lung is distributed according to the mechanical properties of the lung. In particular, τ, the product of the resistance and compliance, will determine the expiratory time necessary for deflation of the lung (normally taken as $3 \times \tau$). This concept of τ is especially important when setting the timing parameters for a mechanical ventilator, and particularly in obstructive diseases when the expiratory time may need to be greatly prolonged to allow proper deflation of the lung after an adequate inspiration. Thus, inspiratory timing is determined by the flow characteristics of the ventilator; expiration is determined by the mechanical properties of the lung and τ.

TECHNIQUES OF MECHANICAL VENTILATION

The most commonly used mode of ventilation, called 'bulk modulus', can be described simply by the equation:

$$V_A = f \cdot (V_T - V_D)$$

where V_A represents the minute gas exchange at the alveolus, f is the respiratory frequency, V_T is the size of each breath and V_D is the physiological dead space that does not take part in gas exchange.

Two different types of devices can meter out this bulk modulus mode of mechanical ventilation. The first, a time-cycled, volume-limited ventilator, delivers a set volume of air for each breath (V_T) at a fixed respiratory rate. The tidal volume is normally in the range of 6–8 ml/kg for all ages but the respiratory frequency varies with age. The second type of ventilator, mostly used in neonatology, is the time-cycled, pressure-limited device. Its advantage is that the maximal pressure can be strictly regulated to avoid barotrauma, but the V_T delivered to the lungs is often less well defined, in that it is dependent upon the compliance of the lung remaining constant in dynamic conditions.

Positive end-expiratory pressure

Positive end-expiratory pressure (PEEP) improves functional residual capacity (FRC) and prevents alveolar collapse.[4] A low level of PEEP (0.2–0.3 kPa), supplied by the flow resistive properties of the upper airway, occurs during normal quiet breathing, but clearly increases with speech, singing and playing any reed instrument. A PEEP of 0.3–0.5 kPa should always be supplied during mechanical ventilation and increasing this PEEP will increase mean airway pressure (MAP). This increment in MAP will, in turn, increase oxygenation by improving ventilation–perfusion matching. However, the use of high levels of PEEP (> 1.0 kPa) in normal lungs may be transmitted to the intrathoracic venous return and thus impair venous preload and decrease cardiac output, with negative ramifications on oxygenation. Usually, a minimal level of PEEP is routinely used in all mechanically ventilated patients in order to maintain volume in the alveoli. The level of PEEP needs to be individualized for each patient–disease process dyad.

Intrinsic PEEP

It is possible for a patient to experience PEEP that is not delivered by the ventilator. This is called intrinsic PEEP and may be as high as 1.0–1.5 kPa in magnitude for certain severe disease states (most commonly obstructive airways disease). The most frequent cause of intrinsic PEEP is insufficient time for the patient to exhale completely. This may be due to lung mechanics (high resistance and slow expiratory flow) or the adopted/imposed ventilatory pattern with a short expiratory time (T_E). This increased intrinsic PEEP resets the end-expiratory lung volume, increases the resting lung volume and decreases the available inspiratory capacity. Under these circumstances tidal volume can easily be compromised with decrease in alveolar gas exchange. Elevated PEEP may also increase the risk of air leak, and certainly augments the risk of overdistension of the lung (volutrauma).

Peak inspiratory pressure

Peak inspiratory pressure (PIP) is the principal determinate of tidal volume during pressure-limited ventilation. The Pdr under these circumstances is the difference between PIP and PEEP, with the V_T being determined by the compliance (C). Therefore, alveolar ventilation and carbon dioxide removal are, in part, dependent on both PIP and compliance. Furthermore, any increase in PIP will increase MAP and thus improve oxygenation. However, aggressive ventilation with high PIP can result in damage to alveoli from pressure (barotrauma) or large tidal volumes (volutrauma).

Timing of ventilation

The timing aspects of mechanical ventilation are also dependent upon respiratory rate. For infants, ventilator rates of 60 breaths/min are not infrequent but give a duty cycle (the time for complete breath to occur) of 1 s. In older children and adults, slower rates of ventilation may give a duty cycle of 3 or 4 s. The duty cycle is divided into the inspiratory time (T_I) and expiratory time (T_E), with expiration always longer than inspiration.

Ventilator rate or frequency (f) along with the tidal volume determines the alveolar minute ventilation, and therefore changes in f will affect PCO_2. At f that is higher than physiological, V_T delivery may be impaired. This may also lead to inadvertent PEEP, as there is insufficient time for exhalation.

A normal respiration inspiratory time (T_I) is usually between 0.35 and 0.45 s for full-term babies, and progressively increases with maturation to reach adult values of 1.0–1.4 s by about the age of 8 years, in concert with the normal decrease in respiratory frequency with age.

T_E needs to be adequate for the lung to empty 'passively', i.e. the airflow is determined by the elastic recoil pressure (Pel) of the distended lung, and the mechanics of the tissues and airways unified as the time constant (τ). Approximately $3 \times \tau$ is necessary for adequate deflation of the inflated lung – if inadequate time is allowed, gas is trapped in the lung, with dynamic hyperinflation and intrinsic PEEP. For a ventilated patient, the adequacy of T_E can be easily assessed by auscultation: expiratory airflow should cease before the onset of the next mechanical breath.

GOALS OF VENTILATORY SUPPORT IN THE NEONATAL AND PAEDIATRIC PATIENT

The overall goals of mechanical ventilation are to optimize gas exchange, patient work of breathing and comfort, and at the same time to minimize ventilator-induced lung injury.[5]

There are no specific guidelines for the initiation of mechanical ventilation in neonates and children. Once mechanical ventilation has begun, the clinician can pose three important questions in order to determine the goals of mechanical support:

1. What is the physiological imbalance, i.e. is this primarily an abnormality of ventilation (elevated PCO_2) or of oxygenation (decreased PO_2) or a mix of both?
2. What is understood of the underlying pathology?
3. What physiological goals are to be attained, i.e. blood gas variables, for this patient specifically?[5]

The conceptualization of the underlying pathophysiological process is extremely important to determine approach to ventilatory support. Blood gas analysis provides valuable information about the patient's ventilatory status and is vital in the tailoring of mechanical ventilation. The goals of mechanical ventilation depend on the disease state being treated. The end-point goal of mechanical ventilation should:

1. Provide adequate ventilation;
2. Provide adequate oxygenation;
3. Achieve adequate lung volumes and attempt to improve lung compliance;
4. Maintain FRC, which can contribute to the maintenance of pulmonary compliance.

Hypoventilation (low V_A) may be due to inadequate central respiratory drive (sedation, brain injury), inadequate respiratory muscle pump (muscle and neuromuscular disease, partial paralysis, skeletal abnormalities) or parenchymal lung disease (non-compliant lungs, alveolar or airway disease, or ineffective alveolar gas exchange).

Hypoxaemia, on the other hand, may be due to ventilatory failure or to lack of pulmonary perfusion (low cardiac output state), loss of diffusion area in the lung or increased diffusion distances in the acinar unit. Finally, the problems with oxygenation may be due to 'shunt': either anatomical or physiological, to give ventilation–perfusion inhomogeneity. For example, amongst premature infants, ventilatory support may be necessary only because of prolonged periods of apnoea rather than because of lung disease. Mechanical ventilation should always be provided in such a way as to attempt to reduce injury from oxygen and distending pressure.

NON-INVASIVE POSITIVE PRESSURE VENTILATION

Non-invasive ventilation offers ventilatory support without the need to establish control of the lower airway. This type of ventilatory assistance, which can be removed and then re-applied, is usually accomplished with nasal prongs, or a mask that fits over the nose or the mouth and nose. This mode of ventilatory support was introduced in the late 1980s for adult patients who had nocturnal hypoventilation,[6] but has subsequently migrated to treatment for younger and younger children and is now utilized in neonates and infants in centres with experience in this field. Prior to this technology being available, children who were ventilated for long periods of time needed a tracheostomy, which is still a necessity for those with a primary diagnosis of fixed airway obstruction. Complications from tracheostomies (such as dislodgement) are common and serious in infants; therefore, they should be avoided if at all possible. Moreover, the risk directly attributable to the tracheostomy for dying in the first six months of life is 4%.[7]

The flow-generating devices used for non-invasive ventilatory support should be equipped with leak compensation, i.e. the flow will be increased to compensate for leaks that occur around the mask. These devices should also have the ability to override any measurement of expiratory volume, as this is often lost to leaks.[6] Masks that are used for ventilation can often cause facial skin necrosis, eye irritation and conjunctivitis if not properly fitted. Ideally, non-invasive ventilation should mean that the patient interface, i.e. nasal mask, should have minimal contact with the patient's mucosa. The long-term morbidity of this therapy, especially when commenced in infancy, includes compression of the mid-face facial bones, which may result in some exaggerated mid-face hypoplasia.[6]

Non-invasive ventilation may be provided continuously during an acute illness, or intermittently for chronic respiratory support. Clinical applications for non-invasive ventilation are hypoventilation and hypercapnia associated with neuromuscular disorders or hypoventilation syndromes. In the ICU it can be used in children with weak expiratory muscles who have acute respiratory infections.[6] Immunocompromised patients with chest infections can also benefit from this form of ventilation. It can also be used as a gradual weaning tool/step-down therapy in patients who prove difficult to extubate.

The primary benefit of non-invasive ventilation is that endotracheal intubation is no longer needed. The risk for nosocomial infections due to the lack of invasive airway care for the patient who receives non-invasive mechanical ventilation is lessened. Moreover, these techniques frequently allow avoidance of admission to the ICU, and avoid the use of sedation. Finally, non-invasive ventilation allows for enteral feeding, and earlier and more rapid ambulation and hospital discharge, with consequential diminution of healthcare cost.

Non-invasive ventilation can provide different types of ventilatory support; the simplest being continuous positive airway pressure (CPAP) providing distending pressure to the respiratory airways. For patients who have an adequate spontaneous respiratory rate, CPAP increases FRC and thus pulmonary compliance to maintain adequate alveolar ventilation. CPAP can also be used as a step from mechanical ventilation, and to reduce work of breathing after extubation.

Bi-level ventilatory assistance (BiPAP, bi-level positive airway pressure) provides both a distending pressure, which maintains lung recruitment and FRC, along with an inspiratory pressure, which aids in ventilatory assistance and lung recruitment and expiratory pressure.[6] As long as the patient can maintain control of the airway, respiratory failure of almost any aetiology can be treated with non-invasive BiPAP ventilation. Bi-level pressure ventilation has two modes of application: a spontaneous mode completely dependent and driven by the patient's own respiratory rate, and a 'timed' mode where the device will cycle at a minimum frequency per minute.

There are very few studies that examine the use of non-invasive ventilation in paediatric patients. Padman et al demonstrated the usefulness of non-invasive ventilation to avoid intubation in a group of children with respiratory failure.[8] Kulkarni et al demonstrated that the use of non-invasive positive pressure ventilation was superior to CPAP when used post-extubation in preterm infants.[9] There was no difference in growth; however, the rate of bronchopulmonary dysplasia and long-term oxygen use was lower in the group who received non-invasive ventilation. This was also demonstrated in a group of preterm babies with respiratory distress syndrome (RDS).[10] Regarding its use in treatment of apnoea of prematurity, there is conflicting data,[11,12] with one group demonstrating a clear reduction in bradycardic episodes and another no clear benefit when compared to nasal CPAP.

According to a Cochrane review, non-invasive positive pressure ventilation reduces the incidence of extubation failure in preterm infants.[13] This can also be applied to preterm infants who receive surfactant. In a group of infants born before 30 weeks' gestation, it was shown that treatment with nasal CPAP was superior in terms of days spent in the NICU as well as need for mechanical ventilation after treatment with surfactant.[14,15]

HIGH FREQUENCY VENTILATION

By definition, high frequency ventilation uses a V_T that is less than the patient's V_D (normally V_D is estimated to be $<$ 2 ml/kg). This mode of ventilation uses extremely high respiratory rates and low tidal volumes. Indeed, this mode of ventilation (augmented diffusion) is analogous to the definition of gas diffusion, where:

$$V_A = (A\tfrac{3}{2}\eta\ l)/(P_1 - P_2)$$

where A is the surface area for gas diffusion, η are the properties of the gas, l is the distance for diffusion, and P_1–P_2 is the diffusion pressure across the membrane. Note that in this equation, neither respiratory frequency nor V_T is important for alveolar gas exchange. The theoretical principle related to high frequency ventilation is described in Figs. 2–4.

Currently, there are three different methods to deliver this type of ventilation:

- **High frequency positive pressure ventilation** uses a conventional ventilator at higher than normal respiratory rates (60–100 breaths/min). This strategy is rarely used because of mechanical inefficiencies of the ventilator.

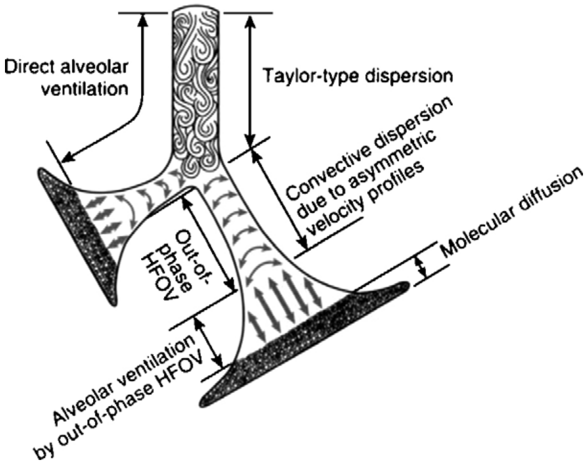

Figure 2 Cross-sectional area of the lung (not to scale) from the airway opening to the alveolar region. Several gas transport mechanisms may operate in various regions of the lung at the same time during high frequency ventilation. Gas velocities slow from the airway opening to the alveolar level. The increase in cross-sectional area from the upper airways to the parenchyma is much greater than shown. Out-of-phase high frequency oscillatory ventilation (HFOV) is also know as 'pendelluft'. (Reproduced from Doss Santos and Slutsky,[33] with permission).

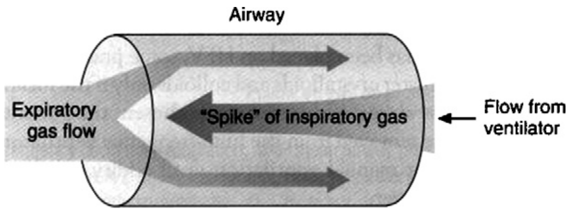

Figure 3 Effect of streaming in high frequency jet ventilation. Pulsations from the jet push the gas forward in the centre; this causes gas along the airway walls to be pushed backward (Reproduced from Doss Santos and Slutsky,[33] with permission).

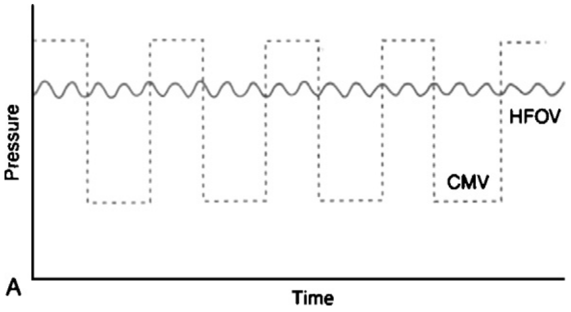

Figure 4 Airway pressure tracing of conventional mechanical ventilation (CMV) which is shown as the dotted line and high frequency oscillatiory ventilation (HFOV) which is represented by the solid line.

- **High frequency jet ventilation** uses a device that injects a jet of air through a small diameter tube near the endotracheal tube. This jet of air draws gas into the lungs and directs it toward the bronchi.[16]
- **High frequency oscillatory ventilation (HFOV)** uses a ventilator with a piston that pushes and withdraws gas rapidly into the lungs. This type of ventilation has both an active inspiratory and expiratory phase.[17] The frequency is very high and measured in Hertz (cycles/s). The range is normally 5–15 Hz with the range 10–15 Hz commonly used in neonates. As the size of the patient increases, the frequency will usually decrease.[17] The latter mode is used commonly in the paediatric and neonatal ICU. In these populations, the high frequency ventilation is defined as a rate > 2.5 Hz (150 breaths/min).

The theoretical advantage of using HFV is that it allows the clinician to recruit lung volume using a relatively high mean airway pressure, thus facilitating oxygenation, but at low tidal volumes and changes in airway pressure (Fig. 4). Moreover, the pressure is attenuated in the airways during HFOV; such that the pressure applied (displayed by the measurement at the proximal airway) does not reach the alveoli[16] and, more importantly, is not transmitted to the pulmonary vascular bed, thus limiting left ventricular preload. This optimizes functional residual capacity but minimizes cyclical stretch of the lungs, which can cause ventilator-induced lung injury.[18] In a large multicentre ($n = 10$) review documented in 2000, 290 paediatric patients who were treated with HFOV were examined.[19] This survey defined the likelihood of survival for various disease states, i.e. RSV lower respiratory tract infection, congenital heart disease and infection, children with pre-existing respiratory conditions, in relation to their oxygenation index after 24 h on HFOV. In patients without pre-existing lung disease, the HFOV was instituted earlier in the clinical course.

Patient–ventilator interaction is important in paediatric ventilation. Children may have difficulty triggering a mechanical ventilator. Optimizing this interaction will improve patient comfort, which may in turn decrease the need for pharmacological sedation. Most current mechanical ventilators offer the option of flow triggering which increases the responsiveness of the ventilator to the patient.

RESTRICTIVE DISEASES AND CHEST WALL RESTRICTION: THE ACUTE RESPIRATORY DISTRESS SYNDROME AND ACUTE LUNG INJURY MODEL

In adults, the mortality rate of acute respiratory distress syndrome (ARDS) is close to 50%.[20] Although there is increased understanding about the mechanisms of this disease, little progress has been made in effective treatments. There is no large randomized controlled trial (RCT) of ARDS in paediatrics, but it is likely that the results of the following adult study can be applied to this group. In a large study of 861 patients with acute lung injury (ALI)/ARDS, the ARDS Network demonstrated that using tidal volumes in the range of 6 ml/kg as opposed to the traditional 12–15 ml/kg significantly decreased mortality (31% versus 39.8%). This strategy using lower tidal volumes also decreased ventilator days in this group.[20] This observation was supported by Mehta and Arnold who demonstrated in children the benefit of using lower tidal volumes and lung volume recruitment manoeuvers.[21]

Other treatment modalities have been tried and studied with less success. In 2004, the Inhaled Nitric Oxide Study group published an RCT of the use of low dose (5 PPM) inhaled nitric oxide (iNO) in patients with ALI. Although iNO did transiently increase PaO_2, it did not decrease mortality or days that mechanical ventilation was needed.[22,23] However, the use of iNO and HFOV in tandem resulted in the greatest change in PaO_2/FiO_2 ratio in children with acute hypoxic respiratory failure.[23]

Although patients with ARDS and/or ALI have pulmonary surfactant dysfunction, the administration of synthetic or natural surfactants does not significantly alter the course of respiratory failure.[24] Outcome measures that were measured were ventilator days, ICU stay and hospital stay. Prone positioning did not significantly affect ventilator days in children with ALI.[25]

OBSTRUCTIVE LUNG DISEASES: THE ASTHMA MODEL

Children who are mechanically ventilated for status asthmaticus are among the most difficult to manage. Increased airway resistance, mucous secretions and oedema lead to air trapping. The main goals during mechanical ventilation are to decrease airway resistance (with pharmacological agents) and to decrease air trapping. Care should be taken to allow enough time for exhalation and to monitor intrinsic PEEP. Indeed, the hyperinflation and the dynamic elevation

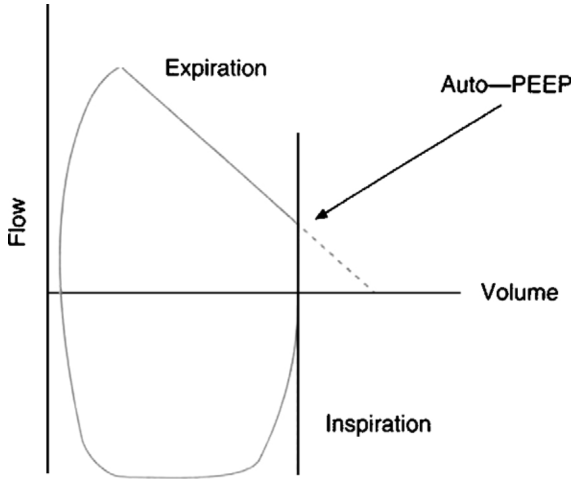

Figure 5 Flow–volume loop reflecting the presence of auto-PEEP. The arrow indicates the amount of flow at the end of exhalation (Reproduced from Doss Santos and Slutsky,[33] with permission).

of PEEP promote pneumothorax, which may occur suddenly because of inhomogeneity of alveolar distension coupled with high intrinsic PEEP (Fig. 5). Mechanically ventilated asthmatics usually are best managed with a low respiratory rate, extended exhalation time and a set PEEP that will match the intrinsic PEEP of the patient.

Consequently, where available, non-invasive ventilation is a preferred option as well in severe asthmatics with respiratory failure. In a study by Beers et al, it was demonstrated that the addition of BiPAP in treating paediatric status asthmaticus is safe and well tolerated. It allowed this group to diminish the number of patients admitted to the PICU. This intervention shows promise as a beneficial adjunct to conventional medical treatments.[26]

RESPIRATORY DISTRESS SYNDROME

RDS is the most common pulmonary disease in premature infants, occurring in 60% of infants born at < 28 weeks' gestation, 30% of those born at 28–34 weeks and $< 5\%$ of those born after 34 weeks.[27] Despite a wealth of knowledge at our disposal about the disease, > 1000 infants succumb to RDS every year in the US.[27]

This was previously referred to as hyaline membrane disease. Infants with RDS lack pulmonary surfactant and, as such, the replacement of the surfactant with either animal or synthetic surfactant is a cornerstone treatment of this disease.

Due to the absence of surfactant, pulmonary compliance is low. Therefore, when instituting mechanical ventilation, PIP should be kept below 20 cmH_2O if possible. If using volume ventilation, a target of 3–5 ml/kg will help avoid over distension. It is useful to attempt non-invasive CPAP or mechanical ventilation to avoid endotracheal intubation. Kugelman et al demonstrated a decrease in the need for endotracheal intubation when non-invasive methods of ventilation were attempted. This study, non-invasive mechanical ventilation offered a slight advantage over CPAP.[28]

If mechanical ventilation is required, HFOV, when introduced immediately after intubation, can reduce the need for surfactant administration and decrease ventilator-induced lung injury.[30] When compared with historical controls, another group demonstrated that very low birth weight infants who were treated early with HFOV had a significantly lower incidence of chronic lung disease.[29]

CONGENITAL DIAPHRAGMATIC HERNIA

Congenital diaphragmatic hernia (CDH) is seen in approximately 1 in 2200 to 1 in 5000 live births. Although long documented, it continues to be one of the most difficult conditions of the newborn to treat. Herniation of the abdominal contents into the chest can cause pulmonary hypoplasia and hypertension. If CDH is suspected, bag and mask ventilation should be avoided in order to diminish abdominal distension that might limit lung expansion. Mechanical ventilation strategies can be used in order to preserve the function of the contralateral lung, i.e. lowest possible PIP with rapid *f*, short TI (inspiratory time) and low PEEP.

Despite the fact that most patients with CDH have pulmonary hypertension, inhaled nitric oxide does not significantly increase survival or diminish ventilator days. Acidosis (pH 7.25 or lower) should be avoided, as this will increase pulmonary vascular pressure. No RCTs in patients with CDH and the use of HFOV versus conventional mechanical ventilation exist. However, early HFOV and delayed surgical repair of the defect were associated with an increase in survival.[31,32]

PRACTICE POINTS

Key Points for mechanical ventilation

- Physiology of respiration
- Application of physiology in mechanical ventilation
- Setting ventilatory parameters
- Alternative/new ventilatory modes

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EDUCATIONAL QUESTIONS

General mechanical ventilation

1. Which statement concerning mechanical ventilation is false?
   a. It is a supportive, non-therapeutic technology
   b. It largely employs negative pressure ventilators in the modern ICU
   c. It is the clinician's attempt to mimic the respiratory system's physiological functions
   d. Optimizing the interaction between the mechanically ventilated patient and the mechanical

ventilator may decrease the need for pharmacological sedation
   e. Ventilation in the ICU is normally done through an endotracheal or tracheostomy tube

Physiology associated with Mechanical Ventilation

2. Which of the following statements concerning the time constant of the lung is true?
   a. The time taken to fill and empty the lung is referred to as a time constant for the lung
   b. The time constant is the quotient of the compliance and resistance of the lung
   c. The time constant will determine the time needed to inflate the lung
   d. Adequate deflation of the lung requires 6 time constants

Non-Invasive Mechanical Ventilation

3. Non-invasive mechanical ventilation can be useful in which of the following situations:
   a. In patients who do not require endotracheal intubation
   b. In patients with poor or no airway control
   c. In patients that require large amounts of sedation
   d. For use during a chronic, but never an acute illness

High Frequency Ventilation

4. When referring to high frequency ventilation, which of the following is true?
   a. It is a mode that uses extremely low tidal volumes and high respiratory rates
   b. The mode of high frequency ventilation most commonly used in neonates is high frequency jet ventilation
   c. It has been demonstrated that very low birth weight infants who were treated early with HFOV had a significantly higher incidence of chronic lung disease
   d. There is no benefit of using high frequency ventilation in tandem with inhaled nitric oxide
   e. It has not been shown to reduce ventilator induced injury in patients with RDS

Asthma and Mechanical Ventilation

5. In the mechanically ventilated asthmatic, which of the following is true?
   a. Intrinsic PEEP should be monitored carefully
   b. High respiratory rates should be used for the control of CO_2
   c. Expiratory time is of little consequence
   d. There is a decrease in mucous production so that endotracheal suctioning is unimportant
   e. Bronchospasm and airway oedema lead to an increase in compliance

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