Review article

Basic neonatal and paediatric mechanical ventilation

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Abstract

Neonatal and paediatric ventilation is highly challenging in respiratory care. Management of the 'little ones' on ventilator is a difficult task. A strong knowledge about basic respiratory physiology and lung mechanics of newborns, gives us the easier application of providing both noninvasive and invasive ventilation. Respiratory distress is mainly assessed by Downes' score, clinical conditions and arterial blood gases. Different modes of ventilation describe the easier ability for gentler ventilation and lung protective strategy. Humidification is essential as the upper airway is bypassed and it can prevent hypothermia. This review will explore the ventilatory support in neonates and paediatric age group.

Keywords: Neonatal, mechanical ventilation, pulmonary physiology, modes of ventilation, humidification.

Introduction

Neonatal and paediatric mechanical ventilation is currently one of the most clinically challenging situations in respiratory care. The neonatal and paediatric population encompasses a broad range of weight, ages, sizes and diseases. Therefore, ventilator practices can vary widely. Children are not small adults and infants are not small children.[1] To manage neonatal and paediatric ventilation effectively, one must combine the principles and knowledge as how airway anatomy, pulmonary physiology and pathophysiology are affected by various diseases. One of the most common reasons for intensive care admission is need for mechanical ventilation for acute or impending respiratory failure.

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Since the introduction of mechanical ventilation in neonatology in the 1960s, mortality rates of preterm infants have decreased dramatically.[2] The introduction of neonatal intensive care, including mechanical ventilation, during the 1960s and its widespread application in the 1970s was associated with increased survival of very low birth weight infants and was shown to be more cost effective in infants of 1 to 1.5 kg birth weight compared with infants weighing less than 1 kg.[3] Assessment of respiratory distress and clinical condition of these neonates is essential in order to support them with positive pressure ventilation. Different modes of ventilation describe the easier ability for gentler ventilation and lung protective strategy. Application of humidification is essential as the upper airway is bypassed.

Basic concepts

Anatomy: Infants are nose breathers because of the large omega-shaped soft epiglottis, positioned high in the larynx (at the level of C4 in the infants versus C6 in the adult). This tends to obscure the

How to cite this article: Shilla E, Lewis L. Basic neonatal and paediatric mechanical ventilation. Ind J Resp Care 2012; 1:21-7.

laryngeal inlet. Partial or complete occlusion of the nasopharyngeal airway may increase the work of breathing and contribute to respiratory failure. The narrowest part of the infant airway is cone shaped cricoid cartilage in the subglottic region. The cross-sectional area between the vocal cords is widened during inspiration and narrowed during expiration. This action allows infant to generate intrinsic positive end expiratory pressure (PEEP) grunting to stabilise their cartilaginous, relatively elastic and flaccid chest, which has a tendency to collapse. Infant's ribs are aligned horizontally allowing for less anteroposterior movements during respiration. Breathing is primarily diaphragmatic. The soft and compliant chest wall can cause two specific pathological conditions: i) rapid and serious impairment of breathing efficiency and ii) inward distortion of the rib cage and waste of energy through sucking in of the rib cage rather than air.

Basic understanding of pulmonary mechanics is necessary when ventilating an infant or a child. The most important elements are elastic recoil, compliance, resistance and time constant.

Elastic recoil: Newborn has low elastic recoil of the chest because it is nonossified and has low total muscle mass, with a low percentage of slow-muscle fibres in both the diaphragm and intercostal muscle. Very little airway pressure is needed to expand the chest wall during inspiration. This explains why muscle relaxation is rarely needed in the young infant during controlled ventilation. The major force contributing to elastic recoil in the newborn is the surface tension at the air-liquid interface in distal bronchioles and alveoli. As described by Laplace's law ($P=2T/r$), the pressure (P) needed to counteract the tendency of bronchioles to collapse is directly proportional to the surface tension (T) and inversely proportional to the radius (r). A decrease in surface tension in surfactant deficiency state (hyaline membrane disease) or after inactivation of surfactant (acute respiratory distress syndrome) increases the tendency of distal airways to collapse.

Compliance: Lung compliance is very low (3.5 ml/cm H_2O) at birth and increases rapidly during the first week to about 5-6 ml/cm H_2O. Reduced lung compliance is seen in neonates with congenital pathologies characterised by small lung volumes with primary or secondary surfactant deficiency, with restrictive lung disease and with bronchopulmonary dysplasia. Reduced compliance also can be seen in obstructive lung disease combined with high lung volumes, such as asthma and bronchiolitis.

Resistance: Airway resistance refers to the inherent capacity of the air conducting system (airway and endotracheal tube) and tissues to oppose airflow and is expressed as change in pressure per unit change in flow. It depends on the radius and length of the airways and gas flow rates. Airway resistance in spontaneously breathing infants is normally 20-30 cm H_2O/L/s. Values in intubated infants are 50-150 cm H_2O/L/s consequent to a narrow endotracheal tube (ETT).[4] In hyaline membrane disease (HMD), the resistance is usually normal while in the case of meconium aspiration syndrome, the airway resistance is markedly increased.

Time constant: The concept of time constant in the lung represents how fast pressure equilibrates between the circuit and the alveoli. The time constant is a mathematic and physiologic concept that is not consistently applied clinically. It is the product of multiplying compliance and resistance. The time constant relates to both inspiratory and expiratory filling of the lungs. Mouth pressure or proximal pressure equilibrates with alveolar pressure in three or five time constants. In a healthy newborn this is 0.33 s. In premature infants with respiratory distress syndrome with decreased lung compliance, time constant can be as short as 0.05 s.[5] Hence the pressure equilibrium will occur in 0.15-0.25 s, which is the minimal inspiratory time required to ensure complete delivery of the tidal volume. In meconium aspiration syndrome, the airway resistance is high resulting in longer time constant. An intervention to improve compliance (surfactant treatment or recruitment manoeuvres) produces a longer time constant by improving compliance.[6]

Mechanical ventilation

Indications: The three major indications for mechanical ventilation in the paediatric and neonatal age group are to secure the upper airway, improve gas exchange and reduce work of breathing. Common objectives of ventilation are three-fold: i) To support gas exchange by improving alveolar ventilation in order to achieve acceptable oxygenation, ii) to restore and maintain adequate functional residual capacity in order to prevent or reopen atelectasis, improve oxygenation and lung compliance and iii) to reduce work of breathing in the presence of high airway resistance and /or reduced compliance, which causes ineffective spontaneous breathing.

Respiratory failure is diagnosed by two or more criteria from the following clinical and laboratory criteria:

Clinical criteria: Retraction, grunting, respiratory rate more than 60 breaths/minute in neonates or more than 35 breaths/minute in children, central cyanosis, intractable apnoea, decreased activity and movement.

Laboratory criteria: $PaCO_2 > 60$ mm Hg, $PaO_2 < 50$ mm Hg or $SpO_2 < 80\%$ with FiO_2 of 1.0, pH < 7.25.

Retractions typically indicate a significant loss of lung volume. The infant then attempts to recruit alveolar volume by increasing respiratory efforts but the excessively complaint neonatal chest wall make this effort somewhat futile in most cases. Grunting often accompanies retractions, particularly in the neonate with respiratory distress syndrome (RDS). Grunting is an expiratory effort against a partially closed glottis that elevates the end-expiratory pressure in an attempt to increase residual volume and oxygenation. Retraction and grunting are the ominous signs of respiratory failure in neonates and infants. If significant retraction and grunting are observed in an infant, early ventilatory assistance should be considered.

Modes of ventilation can be pressure, volume or flow controlled. Ventilation can be fully supported or partially supported.

Control mode (CMV): To attain complete control, the patient-triggering mechanism is made inactive and all breaths are delivered at a preset volume or pressure, frequency and inspiratory flow rate.[7] Patient should be paralysed or sedated to avoid asynchrony between ventilator inflation and patient breathing efforts. The disadvantage of this mode is that asynchrony may cause complication such as pneumothorax. Such situation may occur in acute respiratory distress syndrome (ARDS) or asthma.

Assist/Control Mode (A/C): The volume or pressure, frequency and inspiratory flow rate are preset and the ventilator supports every breath. The patient is allowed to use his/her own ventilatory drive to trigger the ventilator and receive every breath at the preset volume or pressure. If the patient fails to take a breath during the specific period, the ventilator delivers the defined breath at a preset rate. A/C is then defined as patient triggered and machine triggered continuous mandatory ventilation.[8] The advantage of the A/C mode is that every breath delivered to the patient, whether patient or machine triggered, has a guaranteed volume or pressure. The disadvantage is that in neonates, infants and small children with high respiratory rate, hyperventilation, hyperinflation and respiratory alkalosis can occur. The work of breathing may be increased, especially for patients who are not breathing in synchrony with the ventilator or who are 'fighting the ventilator'. If the sensitivity of the triggering mechanism is not adequately set, then the patient's inspiratory effort may be increased and result in an increase in oxygen consumption.[9]

Synchronised intermittent mandatory ventilation (SIMV): The ventilator presets a rate for mandatory breaths and attempts to synchronise the breaths with the patient's spontaneous effort. If no patient effort is sensed within the specific window of time, a mandatory breath is given. Airway pressure or flow is usually the triggering mechanism for SIMV. Small volumes and rapid rates characterise an infant's spontaneous breathing effort, which makes synchronising ventilatory inflation difficult. With the latest advances in sensor technology, SIMV is now a feasible option in neonates and infants.[10,11]

Pressure support ventilation (PSV): Pressure support ventilation is a flow-cycled but time-limited mode that supports each spontaneous breath. It is patient triggered ventilation where patient initiates inspiration, expiration, frequency and determines to a certain extent minute ventilation. This enhances patient's comfort and ventilatory synchrony. To use this mode the neonate needs to have sufficient ventilatory drive. This mode comes with a back up mode when apnoea is detected.[12]

Continuous Positive Airway Pressure (CPAP): It can be used as a noninvasive type of ventilation. It is a gentle method of providing respiratory support to spontaneously breathing neonates. The pressure applied is continuous, during both inspiration and expiration. CPAP is used to maintain lung expansion under conditions that cause alveoli and small airways to collapse or filled with fluid.[13] This constant application of positive pressure helps to open up the alveoli and increase the functional residual capacity (FRC) of the lung. Hence, better gas exchange and recruitment takes place. The condition where CPAP can be applied includes RDS, postextubation, meconium and other aspiration syndromes, pulmonary oedema, pneumonia, congestive heart failure, resuscitation in delivery room, high chest compliance and pulmonary haemorrhage. CPAP can also be used to treat apnoea of prematurity whereby it may stimulate breathing or help maintain airway patency. CPAP can be delivered by mechanical ventilator CPAP, bubble nasal CPAP and infant flow nasal CPAP.

Setting ventilator parameters

Peak inspiratory pressure (PIP): It is the positive pressure that opens up the alveoli. PIP is regulated by a combination of flow rate, respiratory rate and I:E (inspiratory : expiratory) ratio. For an appropriate PIP, the infant's age, weight, gestational age, severity of the disease, lung compliance, airway resistance and time constant of the lung must be considered. Excess of PIP may lead to air leak. Initial PIP may be determined by manual ventilation with a pressure gauge by taking the average PIP required to obtain bilateral chest movement, colour and oxygen saturation. PIP can be adjusted to achieve a desired tidal volume.

A PIP of < 20 cm H_2O is considered low whereas PIP > 20 cm H_2O is considered high. Low PIP is associated with fewer side effects and may allow normal lung development to occur more rapidly. Ventilation may be insufficient and if PIP is too low, generalised atelectasis may occur.

High PIP may help to re-expand atelectasis, increase oxygenation (PaO_2) and carbon dioxide (CO_2) elimination and reduce pulmonary vascular resistance. However, the use of high PIP impedes venous return, reduces cardiac output acutely and in the long term, is associated with chronic lung disease.

Fraction of inspired oxygen (FiO_2): Appropriate use of O_2 is highly therapeutic in most cases of neonatal cardiopulmonary disease. In addition to relieving hypoxaemia, its action as a pulmonary vasodilator in case of persistent pulmonary hypertension of the neonates may be invaluable.[14] Excessive variation in O_2 administration has been implicated as one of the provocative factors for retinopathy of prematurity with subsequent retinal scarring and loss of vision, as well as in bronchopulmonary dysplasia, leading to O_2 or ventilatory dependency.[15, 16] Accurate measurement of O_2 administration and PaO_2 or oxygen saturation is mandatory in any neonate requiring O_2 therapy.

A simple method to determine the FiO_2 needed for a desired arterial oxygen tension PaO_2 is derived from the arterial-to-alveolar oxygen tension ratio. Assuming constant barometric pressure and stable lung conditions, the equation simplifies to:

$$FiO_2 \text{ desired} = PaO_2 \text{ desired} \times FiO_2 \text{ known} / PaO_2 \text{ known}$$

Since prolonged exposure to high levels of oxygen may be toxic, the lowest acceptable FiO_2 should be used.[17] However, high levels of oxygen may be necessary to correct hypoxaemia when treating severe lung disease or persistent pulmonary hypertension of the newborn.

Tidal volume: The tidal volume control on a microprocessor ventilator adjusts flow or time to derive an increase in tidal volume. Research has shown that the largest contribution to ventilator-induced lung injury results from lung over distension caused by excessive tidal volume (volutrauma), followed by the repetitive opening and closing of the terminal lung units (atelectrauma).[18] Hence it is emphasised that the setting of lower tidal volume improves tidal volume monitoring and setting of adequate PEEP level. The tidal volume setting for neonates varies from 4 to 6 ml/kg in low birth weight premature infants, 5 to 8 ml/kg in term infants, and 7 to 10 ml/kg in paediatric age group and adolescent patients.[18,19] Lower volumes have been shown to be effective for patients with restrictive lung disease such as RDS and ARDS.[20,21] Tidal volume should be corrected for compressible volume loss or a proximal flow sensor should be used. During pressure controlled ventilation, increasing the pressure limit may also increase tidal volume. An increase in pressure limit causes the initial flow to increase. The result is larger decelerating flow waveform and a larger tidal volume.

Flow rate: The flow rate will directly affect mean airway pressure. Inspiratory flow should be set to match the peak inspiratory demands and depends largely on the patient's spontaneous effort, work of breathing or patient-ventilator synchrony. Too much or not enough flow can increase the work of breathing or cause dyssynchrony.[22,23] The flow required for spontaneous breathing is provided by means of continuous flow or a demand valve that is triggered by the patient's inspiratory effort.[9]

Frequency: The respiratory rate can determine the minute ventilation and CO_2 elimination. The frequency varies with the disease condition of the lung, complications, infants' age and clinical response. Generally taking the basic knowledge, as the compliance decreases, time constant decreases and faster is the respiratory rate. Higher rates are commonly used in persistent pulmonary hypertension (PPHN) to induce respiratory alkalosis and decrease pulmonary vascular resistance (PVR). Caution needs to be exercised with higher rates of more than 60 breaths per minute (bpm) in neonates because it may lead to air trapping, inadvertent PEEP, decreased venous return and cardiac output.[24] For infant, toddler, child and adolescent, the respiratory rate ranges from 25-40 bpm, 20-35 bpm, 18-25 bpm and 12-20 bpm respectively.[26]

Inspiratory time and I:E ratio: Inspiratory time and I:E ratio directly affect the mean airway pressure. The inspiratory time usually set for neonates is 0.25-0.5 s, in infants 0.60-0.90 s and for toddlers and children its 1-1.2 s.[25] An I:E ratio of 1:2 to 1:3 is usually considered. It should be kept as physiological as possible.

Positive end expiratory pressure (PEEP): PEEP is the pressure applied at the end of expiration. PEEP improves gas exchange by improving PaO_2, recruiting collapsed alveoli, increasing functional lung volume, decreasing intrapulmonary shunting and improving lung compliance.[26] A conservative but acceptable PaO_2 is 45 to 65 mm Hg for neonates younger than 33 weeks of gestation and 60 to 80 mm Hg for paediatric patients at an FiO_2 of 0.40 to 0.50. PEEP usually begins with 3 to 5 cm H_2O, with an increase made in increments of 2 cm H_2O. [27,28]

Mean airway pressure: Mean airway pressure is the most critical factor that determines the optimal gas exchange as it correlates with lung volume. Improvement of PaO_2 directly relates to increase in airway pressure. Mean airway pressure determines the area under the curve (pressure time waveform) from the beginning of inspiration to the beginning of the next inspiration divided by the total cycle time. An equation for this is as follows:

$$MAP = PIP \times T_i / TCT + PEEP \times T_e / TCT$$

Where MAP = Mean airway pressure; PIP = Peak inspiratory pressure; T_i = inspiratory time; TCT = total cycle time; T_e = Expiratory time; PEEP = Positive end expiratory pressure.[29]

Initial ventilator settings

Depends on underlying lung condition, gestation and weight of the neonate, the PIP is set based on

the chest rise and retractions on clinical examination and severity of lung condition (*Table 1*).

Table 1: Setting of peak inspiratory pressure based on lung condition.

Lung condition	PIP (cm H_2O)
Normal	12
Mild disease	13-15
Moderate disease	16-20
Severe disease	20-25

PEEP is set between 4-6 cm H_2O. Neonates with hyaline membrane disease will require higher PEEP to open up the collapsed alveoli. Ventilator rate (VR) must be adjusted between 40-60 breaths /min and based on underlying lung pathology. Concentration of inspired oxygen (FiO_2) is adjusted based on target saturation. The target saturation for preterm neonates below 32 weeks of gestation will be 88-92%.

Lung protective strategy: It is essential to choose the correct mode and appropriate ventilation strategy to enhance patient ventilatory synchrony, reduce leak and decrease days of ventilation.[30] Optimal lung volume and pressure is the key to lung protection. Permissive hypercapnoea is another ventilator strategy which allows the $PaCO_2$ to be a little higher than normal. This is a valuable tool for lung protection in premature neonates. A study done by Kraybill *et al* showed that infants ventilated with higher $PaCO_2$ had lower risk of developing chronic lung disease.[31]

Humidification: Humidification is essential when the tracheal tube bypasses the normal humidifying, filtering and warming system of the upper airway. Therefore, heat and humidity must be provided to prevent hypothermia, drying of airway secretions and necrosis of airway mucosa. Filtration of dry gases before humidification also is needed because of the contamination sometimes found in medical gas lines. A heated water humidifier is necessary to ensure that inspired gases are delivered at or near body temperature.

A modern servo-controlled heated humidifier, with its high and low temperature alarms and heated wires preventing accumulation of condensation, should provide adequate humidification with proper operation. Heated wire circuits are adopted because of the frequency with which condensation needed to be drained and because of infection control considerations. The heated wire circuits are intended to enable the clinician to heat the gas inside the circuit to a temperature above that at which it left the humidifier, ensuring adequate absolute humidity without condensation in the circuit.

Summary

The different modes of ventilation and ventilation strategies are designed to prevent ventilatory induced lung injury (VILI). New ventilators are equipped with patient-triggered ventilation which has more patient-ventilator synchrony to improve gas exchange and recruitment of lungs. Future neonatal and paediatric ventilation appears to be better and more promising. Ventilator management protocols are essential to improve the care of these delicate patients.

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