Volume ventilation in neonates

Samir Gupta

Sundaram Janakiraman

Abstract

Preterm babies are born with immature airways and deficiency of surfactant and a compliant chest wall. This increases the work of breathing with a large proportion of extreme preterm babies requiring invasive ventilation through the endotracheal tube. The improvements in the uptake of antenatal steroids and better perinatal care allow a proportion of these preterm babies to be successfully managed non-invasively. Those ventilated are at risk of iatrogenic lung injury including ventilation induced lung injury. Synchronising the ventilation to the baby's breathing pattern and minimizing lung injury are crucial in reducing complications such as bronchopulmonary dysplasia and long term respiratory morbidity. Volume targeted ventilation in preterm babies has been evaluated in last two decades and is now standard of care. Meta-analysis of trials reports that it improves short-term outcomes to discharge and some complications of prematurity.

Keywords bronchopulmonary dysplasia (BPD); preterm; respiratory distress syndrome (RDS); ventilation; volume ventilation

Introduction

Newborn intensive care facilities and advances in technology have resulted in improved survival of extreme premature babies (23–25 weeks gestation). The EPICURE data from the 1995 and 2005 report an increase in survival of preterm babies born at 23–25 week gestation but with no change in the respiratory morbidity to discharge. Bronchopulmonary dysplasia (BPD) continues to pose challenges with spiralling healthcare costs. This is most likely to be due to the increase in absolute numbers of babies now surviving and discharged home with ongoing morbidity.

Respiratory Distress syndrome in premature babies is due to morphological and biochemical inadequacies and the lack of surfactant. Anatomic immaturity, the compliant chest wall and immaturity of surfactant production pathways in premature babies lead to increase in surface tension of the respiratory units with resultant alveolar collapse, atelectasis, decreased compliance and increase in work of breathing. Early severe and persistent lung disease can result in over 60% babies developing BPD. The risk of BPD is also related to the pattern of respiratory illness. Babies with early severe or progressive lung disease are at significantly higher risk of developing BPD compared to those with minimal lung disease.

Surfactant

Surfactant is produced by the Type II pneumocytes in the alveoli. The surfactant production starts around 22–23 weeks' gestation and peaks around 33–35 weeks. Surfactant has many important properties and these are summarised in Box 1. Mechanical stretch, hyperventilation, maternal catecholamines, cAMP, prostaglandins and leukotrienes are known to stimulate surfactant secretion.

The surfactant production is also increased by antenatal steroid administration, maturity, opiate and alcohol abuse, black race and female gender. Clinical factors that may decrease surfactant production include prematurity, perinatal asphyxia, maternal diabetes, elective caesarean section, maternal hypertension, multiple gestation, erythroblastosis fetalis and male gender.

Respiratory support & RDS

After stabilisation at birth, the need for respiratory support depends on gestation, antenatal steroids and co-morbidity including sepsis and perinatal care. The need for invasive mechanical ventilation is inversely proportional to gestation at birth. Most of the very preterm babies (over 28 weeks) with RDS can be managed with non-invasive respiratory support. However a significant proportion of extreme preterm babies still require mechanical ventilation in the first week of life. With advances in microprocessor technology small volumes of gas can now be accurately delivered to the babies. This delivery of tidal volume can be synchronised with baby's breathing efforts to minimise asynchrony. In the following sections we would discuss the modes and modalities of mechanical ventilation and the evidence supporting use of volume ventilation in preterm babies with RDS.

Assisted ventilation

Assisted ventilation is defined as movement of gas in and out of the lung by an external source directly connected to the patient. The use of Positive Pressure Ventilation with Continuous Distending Pressure is aimed at achieving satisfactory lung expansion throughout the respiratory cycle and overcoming alveolar atelectasis. This facilitates pulmonary gas exchange whilst the surfactant production and the compliance of lung improve. The purpose of mechanical ventilation is to remove carbon dioxide and provide oxygenation. The mechanical ventilation can be

Properties of surfactant

- Maintains functional residual capacity
- Provides a gas permeable protective barrier between air and tissue in lungs
- Helps clearance of foreign material from the lung
- Maintains compliance
- Decreases work of breathing

Box 1

Samir Gupta *DM MD FRCPCH FRCPI is Professor of Neonatology, Durham University; Director, Research & Development and Consultant Neonatologist, University Hospital of North Tees, Stockton, Cleveland, UK. Conflict of interest: Nil.*

Sundaram Janakiraman *FRCPCH is Consultant Neonatologist, University Hospital of North Tees, Stockton, Cleveland, UK. Conflict of interest: Nil.*

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tidal ventilation or non-tidal ventilation, such as high frequency ventilation. In this monograph we will discuss tidal ventilation only.

Using conventional ventilation, the minute ventilation is achieved by delivering set tidal volume at the physiologic rate. The primary modalities of the ventilation can be Pressure or Volume. This is the physiologic modality that the clinician controls by setting the pressure or the volume to achieve minute ventilation. There are however hybrid modes of ventilation now available on newer machines which combines the two modalities.

The modes of mechanical ventilation can be described using the following parameters:

- How each breath is initiated (trigger)? e.g. flow, pressure, EMG
- How gas flow is limited during each breath (limit)? e.g. flow, pressure
- How the breath is terminated (cycling)? This can be volume, time or flow limited

In volume cycling, inspiration ends when a certain volume is reached. In time cycling, inspiration ends when a preset time is reached and in flow cycling, inspiration ends when flow decreases to a chosen percentage of inspiratory flow rate. Thus TCPL (Time cycled, pressure limited ventilation) and other modes of ventilation can be defined using the above variables.

Ventilation parameters

Peak inspiratory pressure (PIP)

The peak inspiratory pressure is the maximum pressure generated by the machine to drive the gas into circuit. This distends the alveoli and the respiratory units. In pressure controlled ventilation the clinician sets the desired pressure to achieve the desired tidal volume. In volume controlled ventilation the clinician sets the desired tidal volume and the ventilator generates the pressure to deliver the set tidal volume.

Inspiratory time (Ti)

This is the time duration when the ventilator pumps the gas into the circuit measured as inspiratory flow of gas. Setting an appropriate inspiratory time is important to minimise flow starvation and inadvertent lung injury and discomfort to the baby. To set the inspiratory time, the intrinsic or extrinsic factors should be considered and the compliance and resistance estimated. One Time Constant (product of compliance and resistance) is defined as the time taken by the alveoli to discharge 63% of its tidal volume. At least three Time constants are required to discharge 95% of the tidal volume and five time constants for 99% of the tidal volume delivery. Thus in babies with surfactant deficiency (low compliance) shorter inspiratory time is appropriate and on the contrary in babies with BPD with high resistance, longer inspiratory time is desirable. While setting the inspiratory time care should be taken for inspiratory to expiratory ratio (I:E ratio) to be 1:2 and not less than 1:1.

Flow

Flow is an important determinant of the ability of the ventilator to deliver desired levels of PIP. The bias flow pressurises the circuit and the machine flow is used to generate pressure and deliver volume. Most of the pressure controlled ventilators set the flow automatically to meet the demand. In volume controlled ventilation the flow is adjusted to set the desired inspiratory time; higher the flow shorter the inspiratory time and *vice versa*. The flow cycling setting terminates the inspiratory flow after delivering the chosen percentage of flow. This is utilised to achieve expiratory synchronisation with the baby's breath. Very short inspiratory time can cause flow starvation and very long inspiratory time results in inspiratory hold of breath.

Positive end expiratory pressure (PEEP)

The continuous distending pressure or PEEP setting depends on the pathophysiology of the disease and treatment goal. The PEEP setting is used to generate functional residual capacity. This facilitates gas exchange and reduces collapse of alveoli at the end of expiration, which in turn minimises lung injury due to repeated collapse below the lung opening pressure. The optimum PEEP setting can be achieved using pressure-volume curve on ventilator graphics and assessing the opening pressure of inspiratory curve. If expiratory time is too short or airway resistance is too high then there is risks of inadvertent PEEP build up, which in turn may contribute to gas trapping and increase in the risk of pneumothorax.

Mean airway pressure (MAP)

Mean airway pressure is computed using various ventilator variables. It depends on peak inspiratory pressure, PEEP, inspiratory time and flow. The oxygenation is proportional to the product of MAP and fractional concentration of inspired oxygen (FiO_2). On the ventilator, pressure (scalar) waveform can be used to see the MAP, which is defined by the area under one respiratory cycle. This is calculated by the summation of all segments on time axis in one cycle (Figure 1).

Ventilation induced lung injury (VILI)

In itself, VILI is multi-factorial (Table 1) and results mainly from injury secondary to the delivery of the gas and level of synchronization between machine and patient. These can be grouped into:

- Inconsistency in tidal volume delivery to the lungs: too little causing atelectasis (atelectotrauma) or too much causing over-distension and stretching (volutrauma),
- Lack of synchronisation between mechanical breaths delivered by the ventilator and babies own spontaneous breaths, and
- Inability to manipulate gas flow delivery (fixed versus variable flow) to meet patient's demand thus causing increased work of breathing (rheotrauma).
- Inadvertent delivery of high pressure (barotrauma)

Pressure vs volume

There are concerns that neonatal lung injury may be more related to the overdistension due to the tidal volume delivered (volutrauma), than pressure induced (barotrauma). When appropriate tidal volume is delivered the pressure is not related to the lung injury. To minimise iatrogenic lung injury, appropriate ventilation modality should be chosen utilizing the mode of ventilation to synchronise with baby's breath and ventilator settings to

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