INTRODUCTION TO VENTILATION

The primary objective of assisted ventilation is to support breathing until the infant's respiratory efforts are sufficient. Ventilation may be required during the immediate care of the depressed or apnoeic infant, or during prolonged periods of treatment of respiratory failure. Improved survival from advances in neonatal care has resulted in an increased number of infants at risk for chronic lung disease.

Newborns are vulnerable to impaired gas exchange due to their high metabolic rate, tendency for decreased functional residual capacity and decreased compliance, increased resistance and potential for right-to-left shunting through the ductus arteriosus and/or foramen ovale. Hypercapnia and hypoxaemia may coexist, although some disorders may affect gas exchange differentially.

Therefore the goals of mechanical ventilation may be summarised as follows:

- to achieve and maintain adequate pulmonary gas exchange
- to minimise the risk of lung injury
- to reduce the work of breathing
- to optimise infant comfort

Ventilatory failure is defined as the failure of the lungs to eliminate carbon dioxide.

Oxygenation is the diffusion of oxygen across the alveoli, providing the oxygen necessary for cell resistance. Increases in resistance will significantly increase the work that a patient has to make in order to pull in a breath.
Oxygenation failure is defined as a failure of the heart and lungs to provide adequate oxygen for metabolic needs.

Hypercapnia

Hypecapnia is usually caused by hypoventilation or ventilation/perfusion (V/Q) mismatch. Carbon dioxide normally diffuses from the blood into the alveoli. The elimination of Carbon dioxide is dependant on alveolar minute ventilation. Alveolar minute ventilation is the product of tidal volume and frequency. An increase in Tidal Volume or frequency increases alveolar ventilation. However small tidal volumes and higher frequencies are preferred to be able to minimise volutrauma.

Hypoxaemia

Hypoxaemia is usually the result of V/Q mismatch or right-to-left shunting, although diffusion abnormalities and hypoventilation may also decrease oxygenation. Oxygenation is determined largely by the FiO_2 and the mean airway pressure. V/Q mismatch is caused by poor ventilation of alveoli relative to their perfusion. Shunting can be intracardiac (congenital heart disease) or extracardiac (pulmonary). Increasing MAP increases oxygenation by increasing lung volume and improving the V/Q mismatch. However a high MAP may cause alveolar over distension and right-to-left shunting of blood in the lungs. Sometimes increases in PIP and PEEP increases oxygenation better.
Airway resistance is defined as the degree of airflow obstruction in the airways. As resistance in the airways causes obstruction to gas flow, a reduction in the airway size
will have the effect of increasing pressures used to provide artificial support of oxygenation and ventilation.

$$\text{Resistance} = \frac{\Delta\ \text{pressure}}{\Delta\ \text{flow}}$$

Airway resistance depends on the following:

- Radii of the airways
- Length of the airways
- Flow rate
- Density and viscosity of the gas breathed.
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Distal airways contribute less to resistance unless their lumen is decreased by brochospasm, oedema, institial oedema.
Small ET tubes contribute significantly to airway resistance.

Values for normal neonates range from 20 – 40 cmH_2O compared to 50 – 150cm H_2O in intubated neonates.

Lung compliance is the degree of lung expansion per unit of pressure change (work of breathing). Abnormally high or low lung compliance impairs the patient's ability to maintain adequate gas exchange.
Low compliance makes lung expansion difficult, as the lungs are 'stiff' or non-compliant. Volume change is small per unit of pressure change, and the work of breathing is high.
High compliance prevents complete exhalation and causes CO_2 retention because the lungs have a reduced elastic recoil and

become ‘baggy’.

The difficulties caused by both low and high compliance to gas exchange result in the need for ventilation.

$$\text{Compliance} = \frac{\Delta \text{ volume}}{\Delta \text{ pressure}}$$

In neonates with normal lungs the total respiratory system compliance ranges from 0.003 – 0.006 L/cmH_2O.

In infants with RDS this can be as low a 0.0005 – 0.001 L/cmH_2O.

Time Constant

Compliance and resistance describe the time necessary for changes in airway pressure to equilibrate throughout the lungs.

The time constant is a measure of the time needed for alveolar pressure to reach 63% of the change in airway pressure.

Time Constant = Resistance x Compliance

E.g. Healthy newborn with compliance of 0.003L/cmH2O and a resistance of 40cmH2O/L/sec = one time constant of 0.12 seconds.

Lungs that have decreased compliance as in RDS, have a shorter time constant and inflation and deflation are completed faster than normal lungs.

The clinical application of the concept of time constant is that very short inspiratory times may lead to incomplete tidal volume delivery resulting in a decrease in PIP and MAP = hypercapnia and hypoxaemia.
Insufficient expiratory time may cause an increase FRC and inadvertent PEEP i.e. gas trapping.

Peak Inspired Pressure

PIP requirements are determined by the compliance of the respiratory system.
Changes in PIP affect both PaO_2 and $PaCO_2$ due to the effects on tidal volume and alveolar ventilation.
An increase in PIP will improve oxygenation and decrease $PaCO_2$.
However an increase PIP may result in volutrauma.

Blood gas anomalies, chest movement, breath sounds , weight, resistance, time constant and PEEP should also be considered when selecting PIP**.**

PEEP

Adequate PEEP prevents alveolar collapse, maintains lung volume and improves the ventilation – perfusion mismatching.
An increase in PEEP results in an increase in MAP = improved oxygenation.
Very high PEEP may decrease venous return, cardiac output, O_2 transport and increase pulmonary vascular resistance.
An increase in PIP and PEEP will increase oxygenation but usually have opposite effects on CO_2 elimination.

Rate

Changes in rate alter alveolar minute ventilation and $PaCO_2$.
An increase in rate and alveolar ventilation = a decrease in $PaCO_2$.

The goal is providing adequate minute ventilation using minimal mechanical force.
Generally, a high rate, low tidal volume strategy is preferred.

I:E Ratio

The effect of an increase in I:E Ratio is an increase in MAP and improved oxygenation. These changes are not as effective as changes in PIP and PEEP.
Changes in I:E Ratio usually do not alter tidal volumes unless the Ti and Te become too short.

Ti and Te

The effects of changes in Ti and Te on gas exchange are influenced by the relationship of these times to the inspiratory and expiratory time constant.
A Ti 3 – 5 times longer than the time constant of the respiratory system allows relatively complete inspiration.

In infants with CLD a longer Ti = better tidal volumes and CO_2 elimination. CO_2 elimination is usually not affected by changes in I:E Ratio.

FiO2

Changes in FiO_2 alter alveolar oxygen pressure and thereby oxygenation.

Flow

Changes in flow in ventilating infants have not been well studied. The affect on arterial blood gas is probably minimal as long as sufficient flow is used.
Flows of 8 – 12LPM are usually sufficient for neonates.

Normal ventilation volumes guidelines

Preterm infants	4 – 6ml/kg
Term infants	4 – 8ml/kg
Paediatrics	10 – 12ml/kg

Normal blood gas values

Arterial Ph	7.30 – 7.45
PCO_2	4.7 – 6.0kPa
PO_2	6.7 – 9.3kPa
O2 Sat	92% - 95%

There are many conditions requiring the need for assisted ventilation. These can be divided into foetal, maternal and congenital abnormalities and these are universally well known.

Indications for Mechanical Ventilation in the Newborn

At Birth

- Failure to establish spontaneous respiration in spite of mask ventilation
- persistent bradycardia
- Diaphragmatic hernia
- infants < 28 weeks gestation re often electively intubated
- infants < 32 weeks gestation may be intubated to receive Surfactant

In the NICU

Respiratory failure

- 28 weeks and $PaCO_2$ tension 6.7 - 7.3 kPa and/or PaO_2 6.7 - 8 kPa in > 40% O_2. If pH < 7.25 lower limits apply.
- 28 - 34 weeks and $PaCO_2$ 6.7 - 7.0 kPa and/or PaO_2 8.0 kPa in 60% O_2. If pH < 7.25 lower limits apply.
- 35 weeks and $PaCO_2$ > 8 kPa with a pH less than 7.25 and/or PaO_2 < 6 kPa in 80% O_2.

Infants at risk for sudden collapse i.e.

- Frequent apnoea unresponsive to CPAP
- Severe sepsis
- Maintenance of patent airway

To control CO_2

- Severe Asphyxia
- PPHN

MODES OF VENTILATION

Continuous Positive Airway Pressure (CPAP)

CPAP is a positive pressure applied to the airways of a spontaneously breathing infant. The pressure is applied throughout the respiratory cycle.

Positive end expiratory pressure (PEEP) is a pressure applied to the airways during expiration and prevents the collapse of the alveoli at the end of expiration. CPAP and PEEP are administered to infants in acute respiratory distress.
CPAP or PEEP is often used to treat premature infants having episodes of apnoea.

CPAP administered via nasal prongs is the current protocol. It is no longer given via an endotracheal tube, as the work of breathing is very high. The infants tire, become apnoeic and may require intubation. CPAP is regularly used prior to ventilation, and post ventilation.

Continuous Mandatory Ventilation (CMV)

This is a mode of mechanical ventilation, historically called time-limited, pressure cycled ventilation. The rate (bpm), inspiratory time, peak inspired pressure (PIP), positive end expired pressure (PEEP) and FiO_2 are prescribed by the clinician. This mode is usually administered in the acute phase of the illness.

The infant does have the ability to spontaneously breathe as well, and this is referred to as Intermittent Mandatory Ventilation (IMV).

The tidal volume (Vt) is determined by the preset pressure limit, flow, inspiratory time and the infant's lung compliance and airways resistance.

Indications:

- Hypoxaemia with a PaO_2 <6.7kPa when receiving more than 50% O_2.
- Hypercapnia with a Pa CO_2 >8kPa
- Unstable cardiovascular status e.g. bradycardia or hypotension
- Impaired respiratory drive e.g. apnoea
- Excessive work of breathing

Patient Triggered Ventilation (PTV)

In this mode of ventilation inspiration is patient initiated. The breath is then assisted with pressure and oxygen by the ventilator. If the infant fails to initiate (trigger) inspiration, the ventilator automatically delivers a mechanical breath at parameters set by the clinician. This is also referred to as assist/control (A/C). The trigger mechanism can be either airway flow or airway pressure.

The patient controls rate and onset of inspiration. The preset parameters are PIP, PEEP, Ti and FiO_2. There is no termination sensitivity in this mode of ventilation.

Pressure Support Ventilation (PSV)

This mode of ventilation is also a patient triggered mode. The difference between PTV and PSV are as follow:

In PTV the infant initiates inspiration only, and receives assistance for the rest of the respiratory cycle at preset parameters.
In PSV the inspiration is triggered by the infant and is then fully or partially supported by inspiratory pressure. Flow is rapidly delivered to the infant, peaks and then decelerates to a termination sensitivity level. The infant therefore has complete control of the respiratory cycle. Should the infant have episodes of apnoea, back up ventilation is delivered at the preset parameters and the inspiratory assistance is at a 100% of the set PIP.
The advantages of this mode are:

* offers total synchrony between infant and ventilator
* decreased work of breathing
* retraining of respiratory musculature and the ability to wean the infant.

<u>Synchronised Intermittent Mandatory Ventilation (SIMV)</u>

SIMV is another form of patient triggered ventilation. The difference being that the clinician sets a certain number of mechanical breaths which are delivered synchronously with a patient triggered breath. This mode may be used from the onset of ventilation to the completion of the weaning process. The combination of SIMV + PSV makes for a very effective and successful weaning tool.

<u>Volume Controlled Ventilation</u>

This is a form of mechanical ventilation where the inspiratory phase ends when a preset volume of gas has been delivered. Volume controlled ventilation may be used in all modes of conventional ventilation. Although there are the advantages of a consistent tidal volume and the reduction in volutrauma, there are limitations to this mode of ventilation.

<u>Volume limiting ventilation</u>

Although this is not a mode in the true sense of the word, it is an increasingly important option for the various modes of ventilation e.g. CMV/IMV, PSV, SIMV.
A tidal volume, appropriate for the particular infant, is targeted, and then achieved with PIP. In this manner both the volume and pressure are limited thereby reducing both volutrauma and

barotrauma. This in turn reduces the incidence and severity of chronic lung disease.

Although a maximum PIP is set, only the amount required to achieve the targeted tidal volume, is delivered. If there is a change in the infant's condition i.e. resistance or compliance, the PIP automatically adjusts to maintain the targeted tidal volume.

High Frequency Oscillation Ventilation (HFOV)

HFOV can be defined as the delivery of small tidal volumes at very high rates. Both inspiration and expiration are active thereby reducing the incidence of gas trapping.

The way in which HFOV is applied depends on the diagnosis of the infant's disease.

Once it has been established if there is uniform or non-homogeneous pathology, the HFOV strategy can be selected. There are two HFOV strategies:

(i) High Volume. (ii) Low volume

The normal operating frequencies are between 10 and 15Hz i.e., 600–900bpm. The mean airway pressure and recruited lung volume determine oxygenation.

The dp or amplitude influences alveolar ventilation and therefore the removal of CO_2. This mode of ventilation has specific clinician and nursing guidelines.