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INTRODUCTION

The vast majority of patients who are admitted to an Intensive Care Unit (ICU) will need artificial ventilation (Jones et al 1998). The usual means through which this is achieved will be via positive pressure ventilation. Gas is delivered under positive pressure, allowing alveoli expansion and gas exchange (Adam and Osborne1997).

However, the effects of this non-physiological approach to ventilation are numerous and can be detrimental. Further more, in diseased lungs positive pressure ventilation may not always provide adequate CO2 clearance or oxygen delivery and may even result in alveolar/lung damage due to ventilating at high airway pressures (MacIntyre and Branson 2001).

An alternative approach to conventional ventilation has emerged over the last decade and is known as High Frequency Ventilation.

PATHOPHYSIOLOGY

In order to understand the benefits of artificial ventilation, it is first important to understand respiratory failure.

Patients need to be intubated and ventilated in order to treat and manage respiratory failure (Oh 1997), of which there are two types

Type1: hypoxaemia without CO2 retention. These will include asthma, pneumonia, pulmonary oedema and pulmonary embolism.

Type2: hypoxaemia with CO2 retention. These will include chronic bronchitis,

post operative hypoxaemia, chest injuries and chronic lung disease.

Along with patients suffering from respiratory failure, there are certain patients who need ventilatory support for other medical reasons. Post operative ICU admissions for 'waking, warming and weaning' are

not uncommon (Adam and Osborne 1997) and certain maxillofacial surgical patients require a period of post operative care/management on ICU, during which time the patient is kept sedated and ventilated.

CONVENTIONAL VENTILATION vs HFOV

Once a patient has been identified as needing artificial ventilation, they are intubated and placed on a ventilator and ventilated using positive pressure. Gases are delivered to the patient using pressure to inflate the lungs, expand the alveoli and allow for gas exchange and oxygenation (Weavind and Wenker 2000). Such delivery can be by means of pressure cycled, volume cycled and/or time cycled. However, the point to remember here is that whatever the mode of conventional ventilation used, they will all use positive pressure to deliver gas and achieve their ventilatory goals.

This use of positive pressure ventilation has its side effects (Fort et al 1997). These are briefly described below;

> Decreased cardiac output: Inspiratory pressure are higher than normal and will reduce venous return. Further, the use of Positive End Expiratory Pressure (PEEP) will further decrease venous return and, thus, cardiac output.
>
> Decreased urine out put: As the cardiac output fall, the kidneys attempt to retain fluid.
>
> Risk of ventilator associated pneumonia.

Risk of tracheal and lung damage if gases are not humidified.

Lung trauma due to high or increasing airway pressures.

It is the potential risk of barotrauma which HFOV attempts to deal with, and which will now be dealt with in more detail.

Patients who develop Acute Respiratory Distress Syndrome (ARDS) will have reduced lung compliance and increases in their lung resistance (Simma et al 2000). Ventilating patients with either decreased lung compliance and/or increased lung resistance can lead to alveolar and lung damage and exacerbate their respiratory problems (Simma et al 2000, Weavind and Wenker 2000). HFOV is generally considered to be of benefit for patients with diseased lungs for a number of reasons;

1. It uses SMALLER tidal volumes than conventional ventilation. To try to deliver a constant tidal volume to a patient with increasingly 'stiff' lungs results in further lung complications. HFOV reduces this risk by

delivering small tidal volumes.

2. HFOV keeps the lungs/alveoli open at a constant, less variable, airway pressure. This prevents the lung 'inflate-deflate', inflate-deflate' cycle, which has been shown to damage alveoli and further complicate lung disease (Fort et al 1997).
3. Along with the above lung protection strategy, it is believed that HFOV may enhance gas mixing and improve ventilation/perfusion (V/Q) matching (Fort et al 1997).

Thus, patients who are at risk of further lung damage due to increases in airway pressure secondary to increases in resistance and decreases in compliance, may benefit from HFOV. When conventional ventilation fails to safely and adequately provide respiratory support, HFOV can be considered an alternative.

HIGH FREQUENCY OSCILLATORY VENTILATION

Essentially, HFOV provides small tidal volumes usually equal to, or less than, the dead space; 150 millilitres, at a very fast rate (Hertz-Hz) of between 4-5 breaths per second. The delivery of tidal volumes of dead space or less at very high frequencies enables the maintenance of a minute volume. Lungs are kept open to a constant airway pressure via a mean pressure adjust system. Further, HFOV allows for the decoupling of oxygenation from ventilation: it allows the clinician to separately adjust either oxygenation or ventilation.

The core of a HFOV system will be a piston assembly. Cairo and Pilbeam (2000) describe the working of such a piston assembly very well;
"Such a system will incorporate an electronic control circuit, or square-wave driver, which powers a linear drive motor. This motor consists of an electrical coil within a magnet, similar to a permanent magnet speaker. When a positive polarity is applied to the square-wave driver, the coil is driven forward. The coil is attached to a rubber bellows, or diaphragm, to create a piston. When the coil moves forward, the piston moves toward the patient airway, creating the inspiratory phase. When the polarity becomes negative, the electrical coil and the attached piston are driven away from the patient, creating an active

expiration."

Since tidal volumes are so low, gas transport mechanisms other than conventional bulk flow must be invoked to explain gas and CO2 flow. This will be explained a later.

Along with the above mentioned amplitude which provides ventilatory volumes, a Mean Pressure Adjust control knob allows for adjustments in mean airway pressure (Paw). This control varies the resistance placed on a mushroom shaped control valve on the patient circuit at the terminus of the expiratory limb. This allows the clinician to manipulate the Paw. Adjusting the Paw enables lung recruitment, keeps lungs and alveoli open at a consent pressure, thus avoiding lung expansion/collapse, lung expansion/collapse which is detrimental to the lungs. Research has also shown that increasing the Paw during HFOV does not effect cardiac out put, unlike conventional ventilation, and increases oxygenation (Fort et al 1997). The mean pressure adjust control is Bias Flow dependent. Bias flow is the rate at which the flow of gas, through the oscillator, is delivered to the patient.

The speed at which the oscillator runs is set by manipulating the frequency. The frequency control sets the breaths per minute in Hertz (Hz). One Hz is equal to one breath per second, i.e., 60 breaths per minute. A frequency of 5 Hz gives a frequency of 5 breaths per second, or 300 breaths per minute. An important point to remember is that as frequency is increased, the excursion of the piston is limited by the time allocated for each breath cycle. Thus, changes in frequency will effect Paw and the amplitude.

In conjunction with amplitude, mean airway adjust, bias flow, and frequency control, an oscillator will usually also allow for the inspiratory time to be adjusted. The inspiratory time will be displayed as % Inspiratory Time. Further, as with conventional ventilators, alarm limits can also be set.

USES FOR HIGH FREQUENCY OSCILLATORY VENTILATION

The use of HFOV in neonates and paediatric patients is well researched and established (Goldsmith and Karotkin 1998). However, its use with adults has only relatively recently been realised. Research is now being conducted into its use with adult patients.

The conceptual advantages of using HFOV are: smaller tidal volumes, a constant, less variable, airway pressure and the fact that nonbulk-flow mechanisms may improve V/Q matching. HFOV is used to avoid conventionally ventilating atelectasis prone lungs in ARDS (Clark et al 1994). Over distention of the lungs and ongoing atelectasis contribute to progressive lung injury which arises not directly from the disease process itself, but from the impact of the ventilator patterns used to support gas exchange during the course of the illness by conventional ventilation (Isabey et al 1984). Atelectasis can be halted, and even reversed, during HFOV, while avoiding the over distention so commonly seen with conventional ventilation (Froese 1997, Tseng et al 1998, MacIntyre and Branson 2001).

Thus, HFOV is used to minimize ventilator-related lung injuries in ARDS. The protective strategy of a constant airway pressure, with smaller tidal pressure swings, preventing over distention, are reasons why HFOV is used.

In addition to this better alveoli recruitment strategy, the rapid flow pattern may enhance gas mixing and improve V/Q matching. However, since tidal volumes are smaller than usual, gas transport mechanisms other than conventional bulk flow transport must be discussed to explain oxygen and CO2 flow. There are a number of mechanisms to explain gas transport under these non-physiologic conditions. The following have been suggested by Weavind and Wenker (2000):

Bulk flow can still provide conventional gas delivery to proximal alveoli with low regional dead space volumes.

Coaxial flow. Gas in the centre flows inward, while gas on the periphery flows outward. This can develop because of the asymmetric low profile of high velocity gases.

Taylor dispersion can produce a mixing of fresh and residual gas along the front of a flow of gas through a tube.

Pendelluft can mix gases between lung regions having different impedances.

Augmented molecular diffusion can occur at the alveolar level secondary to the added kinetic energy from the oscillations

The importance of each of these is debated. It has been suggested by MacIntyre (1998) that perhaps all of the above may be operative simultaneously during HFOV.

The combination of these non-physiological, non bulk flow gas mechanisms and a constant airway pressure, are the advantages of HFOV over conventional ventilation. Improvements in V/Q matching and the preventing of over distention have led HFOV to be viewed as an alterative to conventional positive pressure ventilation. In a study by Fort et al (1997) HFOV was evaluated in terms of safety and effectiveness in patients with ARDS and with whom conventional ventilation had failed. This prospective study (n=17) included patients who had failed conventional ventilation, had very high peak inspiratory pressure (peak pressure of 54.3 +/- 12.7cm H2O), a PaO2/FiO2 ratio of 68.6 +/- 21.6 and positive end expiratory pressure of 18.2 +/- 6.9cm H2O. HFOV was instituted after varying periods of conventional ventilation (5.12 +/- 4.3 days). A lung volume recruitment strategy was employed, consisting of incremental increases in mean airway pressures to achieve a PaO2 of > or to 8.0 kPa. During the study 13 patients demonstrated improved gas exchange and an overall improvement in PaO2/FiO2 ratio. Cardiac output was not compromised in any of the patients, despite increases in mean airway pressure. The authors of the study maintain that HFOV is both safe and effective in adult patients with severe ARDS failing conventional ventilation. They do, however, acknowledge the need for continual research into HFOV in adult patients who fail conventional ventilation.