High Frequency Oscillatory Ventilation (Adults) Learning Package

Intensive Care Services
John Hunter Hospital

Name: _____

NSW GOVERNMENT Health
Hunter New England
Local Health Network

Document Authorisation

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Disclaimer

This learning package has been prepared by health care professionals employed in the intensive care unit at the John Hunter Hospital. While all care has been taken to ensure that the information is accurate at time of development, the intensive care unit at the John Hunter Hospital recommends that all information is thoroughly checked before use if utilised in another unit, context or organisation.

Feedback

This self-managed learning package has been added to the intensive care unit intranet (help library) for all to view. We appreciate feedback and encourage you to contact the primary author via e mail (this is located in the authorisation table on the previous page). The primary author may also be prepared to provide further information and insights to the topic.

Introduction

This learning package is designed to provide an introduction to the principles of high frequency oscillatory ventilation. It will facilitate the health care professionals understanding of high frequency oscillatory ventilation and its application within the intensive care unit. It is to be used in conjunction with the related John Hunter intensive care unit ventilation guidelines; HFOV guidelines and in-service education program. These guidelines can be accessed from the intensive care help library.

Learning outcome objectives

Completion of this learning package will enable the Registered Nurse to complete the related HFOV competencies, and therefore demonstrate an understanding of the following:

- Indications and criteria for HFOV
- Assembly of HFOV machine & circuit
- Recruitment manoeuvres
- Nursing management of patient receiving HFOV
- Documentation

Prerequisites for Registered Nurses

In order to complete this package the Registered Nurse must have completed the following John Hunter Hospital intensive care competencies:

- Ripples competencies 1 – 5

Assessment process

When you have completed this package please give it to an accredited assessor for review. Once this has occurred the Registered Nurse can complete the intensive care HFOV competency with an accredited assessor.

Recognition of prior learning

Relevant post graduate qualification will be acknowledged e.g. Graduate Certificate in Critical Care. RPL will be granted at two levels:

1. Registered Nurses who have completed relevant post graduate qualifications will not have to complete this package
2. Registered Nurses who have completed relevant post graduate qualifications and can provide supporting documentation of HFOV competency assessment will not have to complete this package or the HFOV competency

If you feel that you hold relevant qualifications please speak with the CNE or NE in your area.

Conventional Ventilation Strategies

The cornerstone of respiratory support for patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) remains mechanical ventilation.
Although mechanical ventilation is essential it may also result in further harm to the patient known as ventilator induced lung injury (VILI) by both directly injuring lung tissue and causing further release of inflammatory mediators into the systemic circulation (Ferguson & Stewart, 2002; Rose, 2008; Downer & Mehta, 2006)

The following, in isolation or combination, may be contributing factors:

- Lung over-inflation as a result of excess pressure (barotrauma)
- Lung over-inflation as a result of excess volume (volutrauma)
- Repetitive alveolar collapse and recruitment (atelectotrauma)
- Stimulation of the inflammatory response (biotrauma)

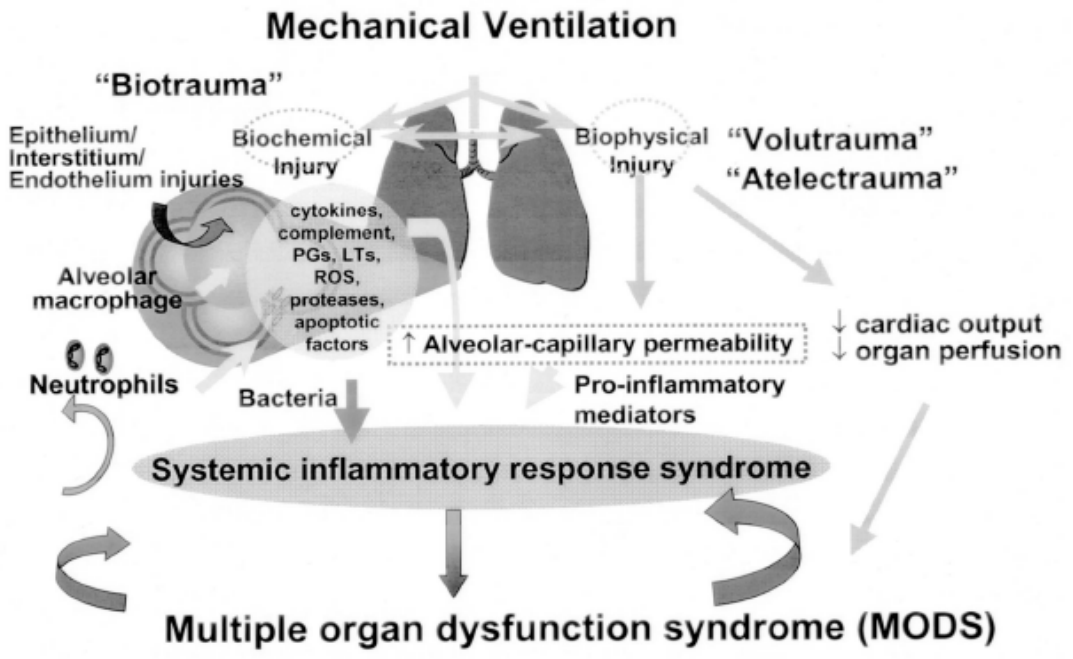
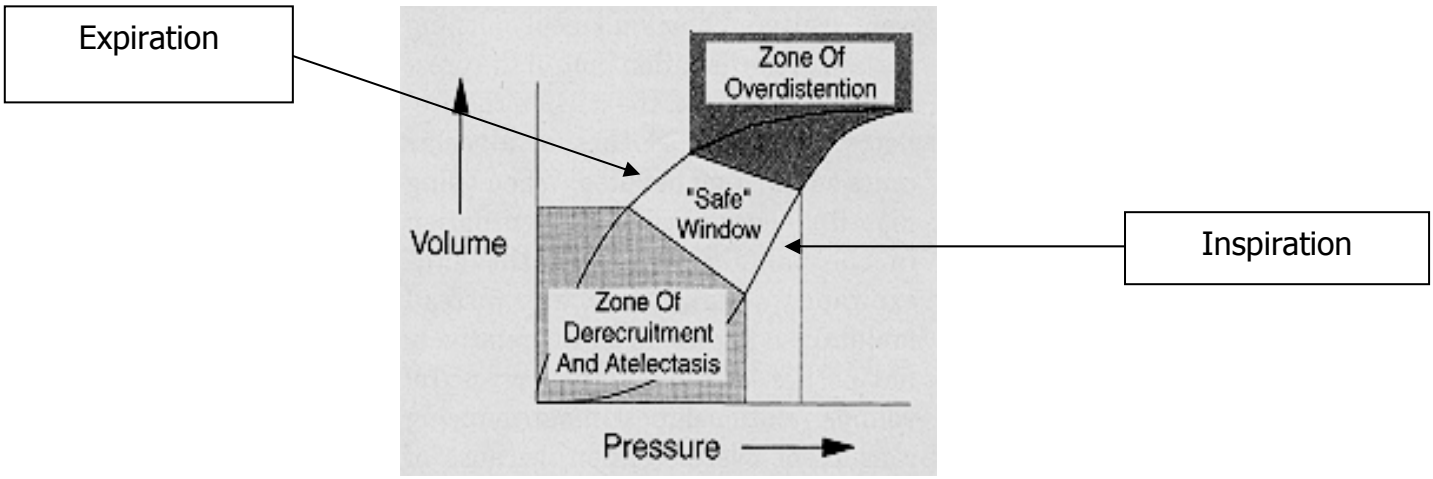


Figure 2. Postulated mechanisms whereby volutrauma, atelectrauma, and biotrauma caused by mechanical ventilation contribute to multiple organ dysfunction syndrome (*MODS*). The potential importance of biotrauma is not only that it can aggravate ongoing lung injury, but also that it can contribute to the development of MODS, possibly through the release of proinflammatory mediators from the lung. Adapted with permission from Slutsky and Tremblay (2).



Pressure volume curve of a moderately diseased lung (Froese, 1997)

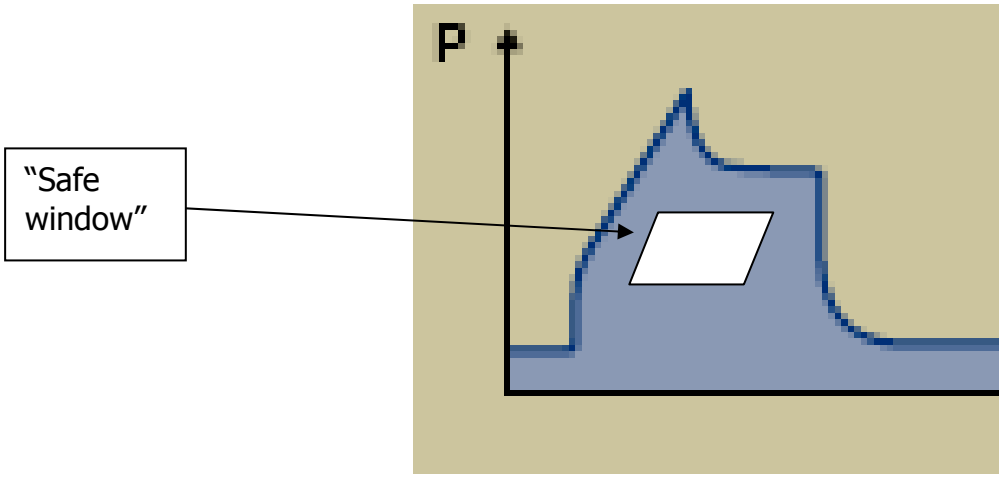
There are two hazardous zones that exist during conventional ventilation. (Fan et al, 2008; Froese, 1997; Stawicki, 2009).

1. The zone of 'over-distension'; too great a pressure and or volume leads to mechanical disruption of alveoli and airways, surfactant degradation and the accumulation of oedema fluid.

2. The zone of 'de-recruitment and atelectasis'; repeated closure and re-expansion of airways and alveoli leads to direct trauma, stimulating an inflammatory response and inhibiting surfactant production.

Ventilation should be targeted within the 'safe window'. However, in reality this 'safe window' area can be difficult to achieve for a number of reasons; (Singh & Ferguson, 2005)

- Diseased lungs are not uniform (the pressure-volume curves will be different for different areas of the lungs
- The 'safe window' area cannot be measured by conventional means
- Ventilation within the 'safe window' may result in inadequate alveolar ventilation causing an increase in paCO2



Pressure curve of a volume delivered breath

In comparison with a pressure volume curve in normal lungs, the patient with ALI/ARDS is exposed to significant periods of cyclical pressure changes that fall outside the 'safe window'. (Derdak, 2001; Haberther & Stocker, 2002; Mehta et al, 2004; Ferguson & Stewart, 2002).

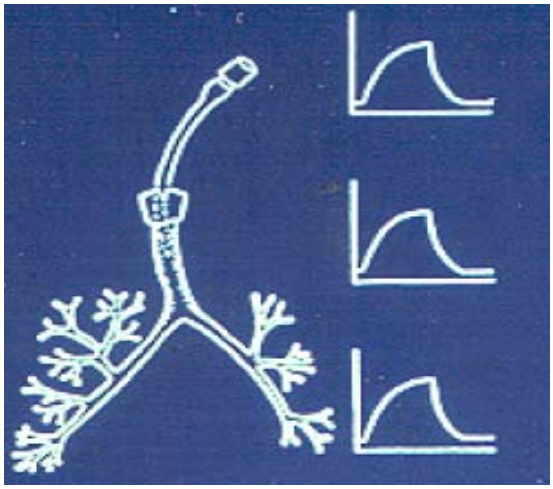
Therefore the optimum aim for ventilation for the patient with ALI/ARDS is to:

- Minimise high peak airway pressures
- Minimise high tidal volumes (to avoid the zone of over distension)
- Optimise PEEP
- Utilise higher mean airway pressures (to avoid the zone of derecruitment)
- Maximise time of ventilation in the 'safe window'.

Conventional Ventilation Limitations in ALI/ARDS

PEEP

Levels of PEEP can be optimised to prevent de-recruitment and tidal volumes can be reduced to prevent barotrauma and volutrauma occurring in the zone of over-distension. However despite the use of smaller tidal volumes, once combined with higher levels of PEEP, high airway pressures may still result. These airway pressures do not only occur at the distal end of the endotracheal tube but they are also transmitted throughout the bronchi, small airways and ultimately the alveoli. (Krishnan & Brower, 2000).



Airway pressure waveforms illustrating pressure transmission throughout distal airways (David et al, 2003)

Tidal volume

The dramatic reductions in tidal volume required to minimise excessive lung distending pressure results in the need for much higher than normal respiratory rates to maintain control over carbon dioxide levels. The ability to deliver these faster rates on a standard ventilator is further hampered by the patient's expiration being passive (that is the ventilator has to wait for the patient to exhale before delivering the next breath) (McCarthy & Dillard, 1990).

This effect can be examined in the table below, displaying ventilator settings that may be used in an 80kg patient. This patient may initially be ventilated with a respiratory rate of 10 breaths and tidal volumes of 640 mL (or 8mL/kg). The tidal volumes are decreased as a lung protective strategy and respiratory rates need to increase dramatically to maintain a desired minute volume and therefore maintain carbon dioxide levels within a desired range. With this strategy the small tidal volumes and dead space will affect paCO2 (Wimbush, 2005).
For example, the average anatomical dead space in an adult is approximately 150 mls, hence with a tidal volume of 160 mls only 10 mls of fresh gas will reach the alveoli.

Minute Volume	Actual tidal volume	Tidal volume mL/kg	Respiratory rate required	Alveolar ventilation
6.4 Litres	640 mL	8	10	4.9 L/min
6.4 Litres	480 mL	6	14	4.6 L/m
6.4 Litres	320 mL	4	20	3.4 L/min
6.4 Litres	160 mL	2	40	0.4 L/min

These difficulties may ultimately lead to a different ventilation strategy being required.

Now read the following journal article:

Esan, A., Hess.D. Raoof, S., George, L., & Sessler, C. (2010). Severe hypoxemic respiratory failure. Chest, 137, (5), 1203-1216.

This article can be located through CIAP via the internet.

After reading the journal article complete the following:

In your own words, describe your understanding of the following lung protective strategies

High PEEP

Low Tidal Volumes

Plateau Pressure Limitations

Adjunct strategies may be implemented in combination with lung protective strategies.

Now read the following chapter:

Cuthbertson, S. & Kelly, M., Support of Respiratory Function. In Elliott, D., Aitken, L., & Chaboyer, W. (2007) *ACCCN's Critical Care Nursing* (1st ed). Elsevier. Australia.

This textbook can be accessed through the Gardiner library on the John Hunter Hospital Campus or borrowed from the ICU Educators office.

In your own words describe the following adjunct strategies:

Permissive Hypercapnia

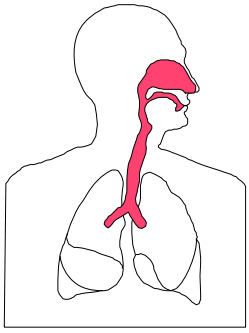
Prone Positioning

Selective pulmonary Vasodilators

Conventional Ventilation and Gas Exchange

Anatomical dead space (V_{Danat}) includes the nose, mouth, trachea, bronchi and bronchioles where no significant gas exchange occurs. Gas transport occurs by bulk delivery of gas and is a result of convection through the airways to the alveolar zones. A proportion of the delivered tidal volume remains in the proximal airways as dead space volume and the tidal volume must be greater than the dead space volume for gas exchange to occur.

Therefore, expiration of all 'old gas' does not occur with each breath. However, if the amount of 'new gas' inspired is greater than the 'old gas' present adequate gas exchange occurs (Chan, 2007).



Anatomic dead space (V_{Danat}) (McCance & Huether, 2002)

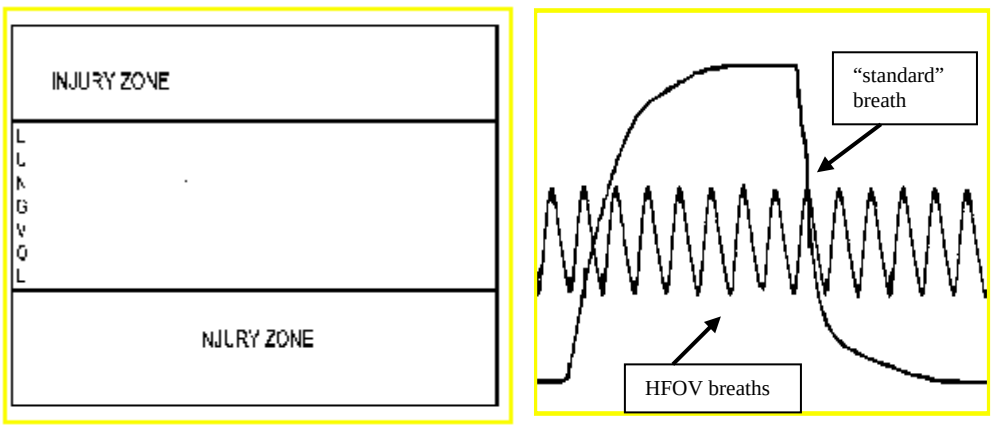
High Frequency Oscillatory Ventilation (HFOV)

High frequency oscillatory ventilation (HFOV) is a mode of assisted ventilation utilising supraphysiologic respiratory rates (called frequency or f) and very small tidal volumes (Chan et al, 2007; Downer & Mehta, 2007).

- Frequency may range between 180-900 breaths per minute
- Tidal volumes range between 0.1-3.0 ml/kg
- HFOV maintains a high mean airway pressure (mPaw)

It is important to note that the very small tidal volumes are often less than anatomical dead space volume.

Oxygenation and carbon dioxide clearance is achieved without the large cyclical pressure changes required in conventional ventilation and without the risk of cyclical lung over-distension or de-recruitment (Fessler et al, 2007).



Comparison of zone of injury to the pressure curve of a volume and HFOV breath (Frose, 1997)

Principles of Operation

Unlike conventional ventilation, HFOV can separate the functions of oxygenation and ventilation (or CO2 clearance) (Ferguson & Stewart, 2002).

- **Oxygenation** is primarily a function of the FiO2 and the mean airway pressure (mPaw)
- **Ventilation** is controlled by frequency, amplitude (ΔP or power) and the I: E ratio.

(Ritacca & Stewart, 2003)

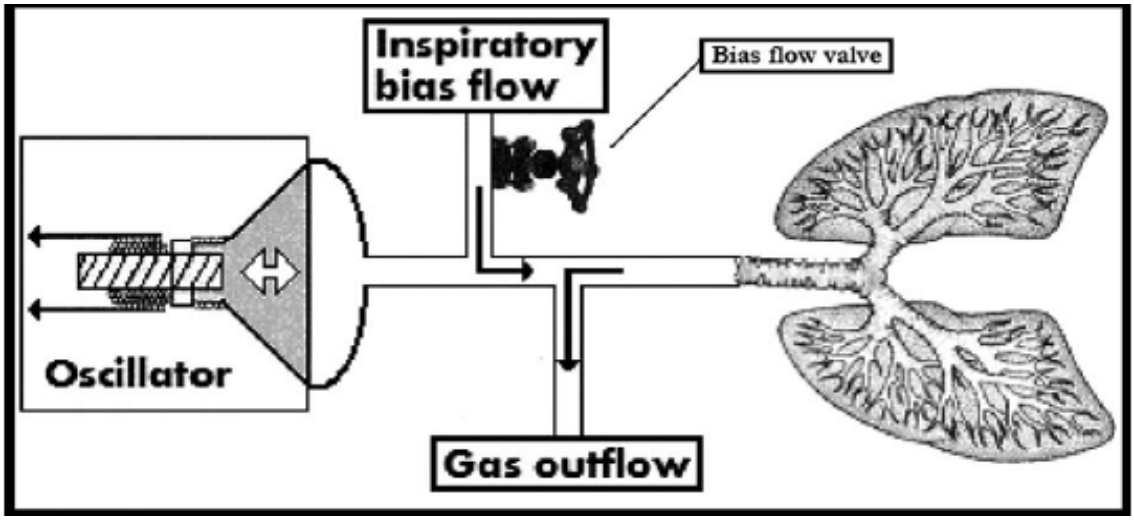
The Bias Flow and Mean Airway Pressure

The Sensormedic 3100B HFOV delivers a continuous flow of gas (called the bias flow) via a stiff and non-compliant ventilator circuit. This in combination with a control valve produces a pressurised patient circuit (the mean airway pressure or mPaw) (Ritacca & Stewart, 2003).

The mean airway pressure is the average pressure to which the lungs are exposed during the respiratory cycle. Mean airway pressure represents the relationship between peak inspiratory pressure (PIP), Positive End Expiratory Pressure (PEEP) and Inspiratory: Expiratory Ratio (I: E) (Rose, 2008).

The mean airway pressure setting can be likened to CPAP and this has led to some authors describing HFOV as "CPAP with a wiggle".

The mPaw during HFOV can be significantly higher than during conventional ventilation (i.e. as high as 40-45 cm H2O) without causing apparent lung injury. This mechanism is unclear but may be related to the minimal lung tissue cyclical stretch associated with HFOV (Downer & Mehta, 2006; Chan et al, 2007).



HFOV ventilator circuit (Stawicki, 2009)

Frequency

The gas in the ventilator circuit is oscillated by means of an electrically driven diaphragm. The frequency (or rate) of oscillation can be varied between 3 and 15 Hertz (1 Hertz = 60 breaths per minute (Hertz, Hz).

In HFOV, the control of CO2 is **inversely** related to the frequency. Increasing the frequency is likely to result in a higher (not lower) PaCo2. This is because a higher frequency (i.e. tidal volume) results in shorter inspiratory times, reducing the duration of the bulk flow of air into the tracheo-bronchial tree (Rose, 2008).

I:E Ratio (% Inspiratory Time)

The % I time setting controls the amount of time the diaphragm spends displacing forward compared to displacing backward. This is the same as the I:E ratio on a conventional ventilator. Unlike conventional ventilators changes to the I:E ratio in HFOV (with high frequencies) result in small differences to time and hence little change in the delivered tidal volume(Rose, 2008).

Example:

At a frequency of 3 Hz (a "breath" occurs every 0.33 seconds).

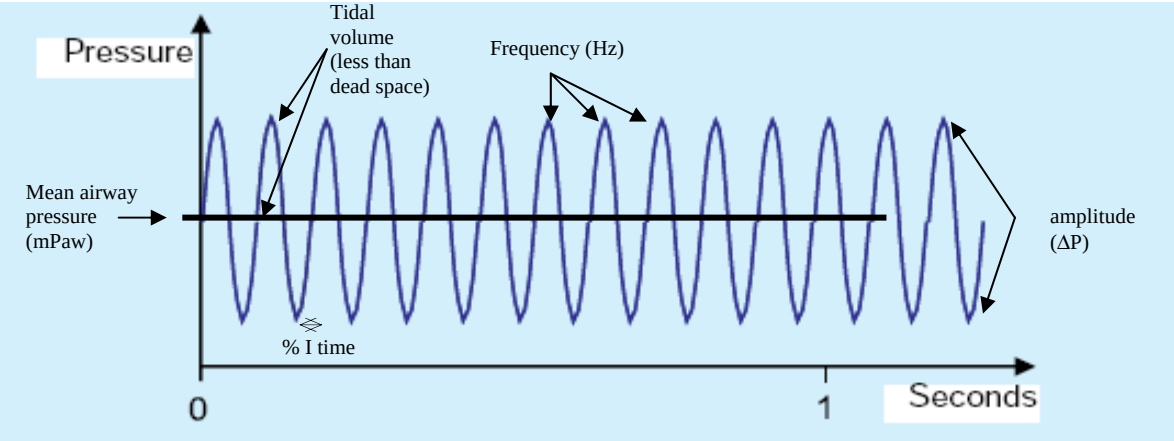
Changing the I:E ratio from 33% to 50% would increase the "inspiratory time" by .0275 per/sec.

Amplitude (Power)

The amplitude, often described as ΔP (delta pressure or power) is a measurement of the piston force which causes the forwards and backwards displacement of the diaphragm from

the resting position. As a result this displacement causes inspiration and expiration to be an active process.

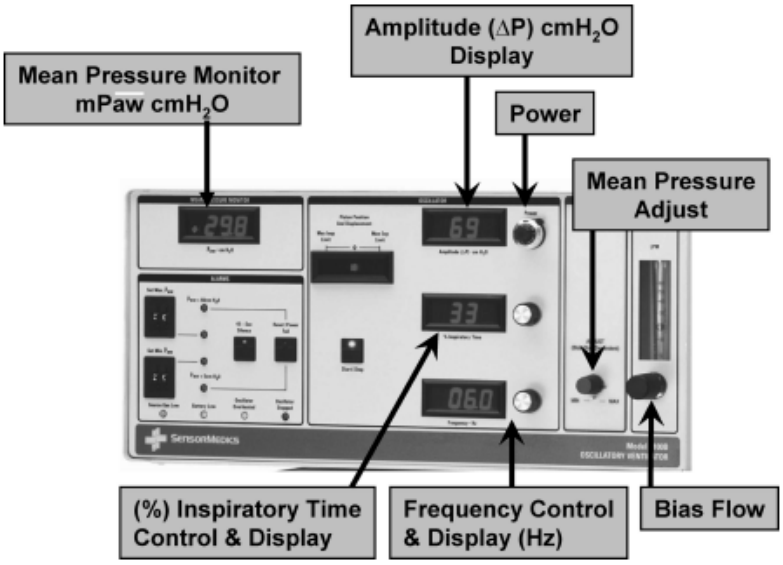
In diagrammatic form this is represented by a peak to trough swing across the mean airway pressure. The amplitude is controlled by the power setting on the 3100B (Derdak, 2001; Scalfaro, 2001; Stawicki, 2009).



HFOV Waveform (Stackow, 1995)

Tidal volume is easily measured during conventional ventilation; however accurate estimations are difficult during HFOV as the tidal volumes are often less than anatomical dead space. Regardless of this difficulty, tidal volume in HFOV is represented by the ΔP (the driving pressure), frequency, airways resistance (mainly from the ETT), and the respiratory system compliance. In the diagram above it is represented as the difference between the peak amplitude and the mean airway pressure (Scalfaro, 2007).

If HFOV is referred to as "CPAP with a wiggle" then the mean airway pressure can be likened to CPAP, and the amplitude is the "wiggle".



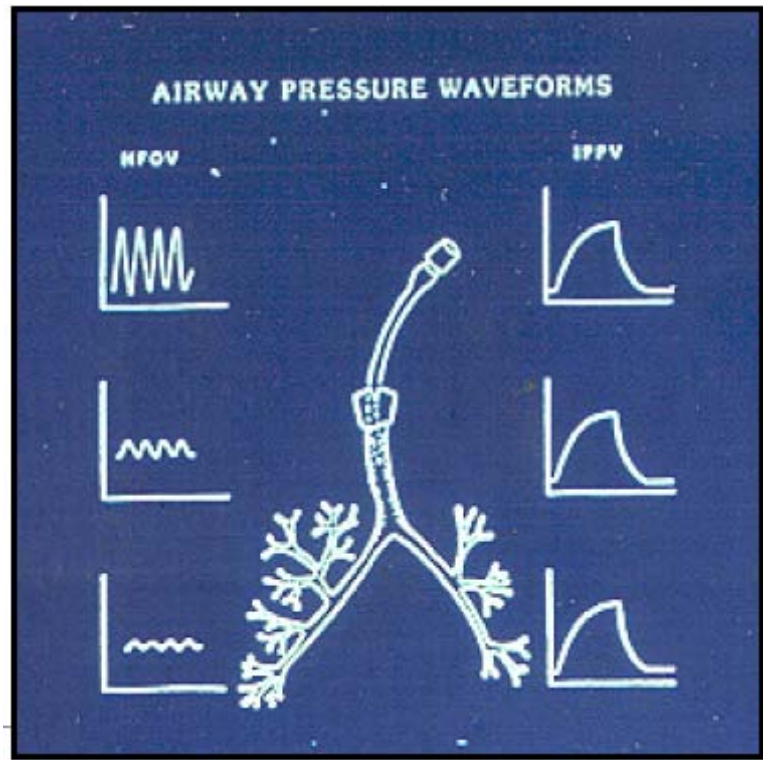
Control dials on the Sensormedic 3100B HFOV (Derdak et al, 2001)

Pressure Attenuation during HFOV

During conventional ventilation, the pressure generated at the endotracheal tube is similar to the pressure generated at the bronchi and alveoli. The ΔP in HFOV is high (and may be higher than peak airway pressure in conventional ventilation); however in HFOV the amount of pressure transmission beyond the endotracheal tube is greatly attenuated (or reduced).
With HFOV the pressure generated at the endotracheal tube is greatly diminished as it travels through the distal airways and reaches the alveoli. This potentially leads to less alveolar distension and may be an advantage as a ventilation strategy for the patient with ARDS/ALI (Ritacca & Stewart, 2003; Derdak, 2003; Branconnier, 2004).

Pressure attenuation is determined by:

- ETT diameter (i.e. resistance)
- oscillator frequency
- airway resistance
- respiratory system compliance



Pressure attenuation through the airway (David et al, 2003)

'Chest Wiggle Factor'

A combination of the amplitude (or delta pressure), frequency and the natural resonant frequency of the lungs results in oscillations transmitted through the lungs and chest wall. Clinically the chest wall and abdomen may be observed to 'wiggle'. This 'wiggle may be observed as a 'wobbling' movement commencing at the chest, abdomen and extending down to the groin.

Due to the difficulty in accurately being able to measure tidal volume in HFOV, some clinicians utilise the intensity of this 'wiggle' as an indicator of adequate ventilation and refer to this as the "Chest Wiggle Factor" (CWF). An absence of CWF has been used as a clinical cue to increase the amplitude setting (Derdak, 2001; Higgins et al, 2005; Rose, 2008; Stawicki et al, 2009).

However, in adults the main focus is the clinical observation of any alteration of CWF (such as a decrease or absence). This may indicate the following:

- change in patient condition
- endotracheal tube obstruction
- right main-stem intubation

- tension pneumothorax

Clinical assessment and investigation is required. The following may be considered:

- comprehensive physical examination
- arterial blood gas
- Chest x-ray

Mechanisms of gas transport & gas exchange during HFOV

Tidal volumes in HFOV are usually less than the anatomical dead space. This challenges the traditional concepts of gas transport and gas exchange as during conventional ventilation.
A number of different convective and diffusive mechanisms have been identified as contributing to the role of gas exchange during HFOV (Pillow, 2005).

Convective	Diffusive
Pendelluft	Pendelluft
Taylor's dispersion	Molecular diffusion
Asymmetric velocity profiles	Asymmetric velocity profiles
Bulk convection	Cardiogenic mixing
Convective ventilation	

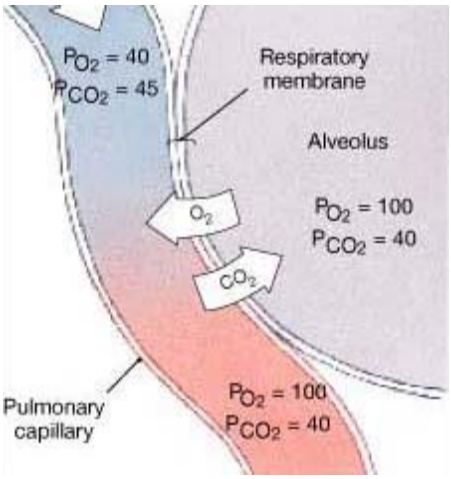
Bulk convection

Bulk convection is simply the bulk movement of matter from one spot to another. Whilst this plays a significant role during conventional ventilation, its plays a relatively small role during HFOV except in some of the most proximal (short path length) gas exchange units where there will be direct alveolar ventilation (Chang, 1984; Ferguson & Stewart, 2002).

Molecular Diffusion

This represents the primary mechanism of gas mixing in the terminal air spaces (as it does in conventional ventilation). It can result from the random motion of gas molecules (Brownian)

and gas transport across the alveolar capillary membrane via diffusion gradients (simple diffusion) (Ferguson & Stewart, 2002; Krishnan & Brower, 2000).



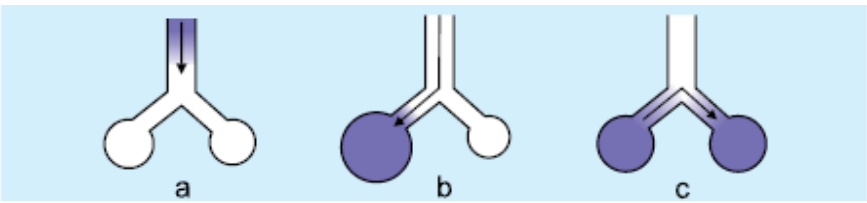
Simple diffusion of gas across the alveolar capillary membrane (McCance & Huether, 2002)

Cardiogenic Mixing

The superimposition of myocardial contraction may promote peripheral gas mixing by promoting the generation of flow within neighbouring parenchymal regions rather than at the airway opening. Although this mechanism may sound marginal some authors suggest this may account for up to half of oxygen uptake during HFOV (Ferguson & Stewart, 2002).

Pendelluft

Not all regions of the lung have the same resistance and compliance, which may result in neighbouring units filling and emptying at different time constants. Due to this asynchrony, alveoli can mutually exchange gas (gas from fast units will empty into slow units). This intraregional gas mixing is know as "pendelluft" and allows very small volumes of fresh gas to reach a large number of alveoli (Chang, 1984; Ferguson & Stewart, 2002).

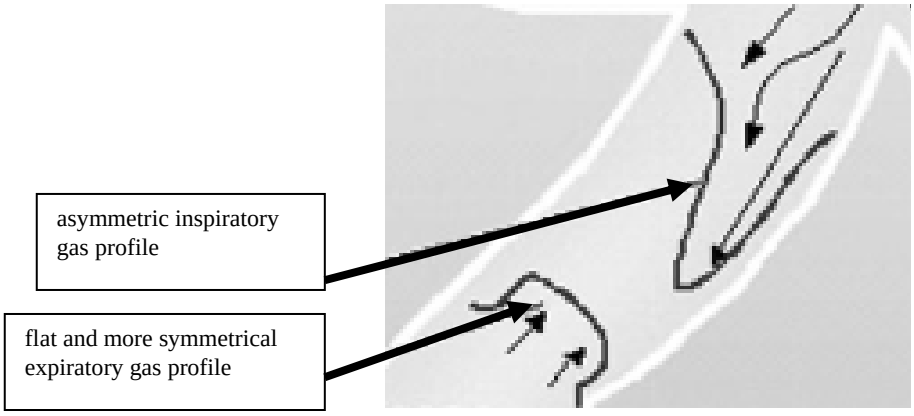


(Stachow, 1995)

a. *beginning of ventilation*
b. *fast unit ventilation*
c. *Gas leaving fast unit subsequently enters a slow unit*

Asymmetric Velocity Profiles

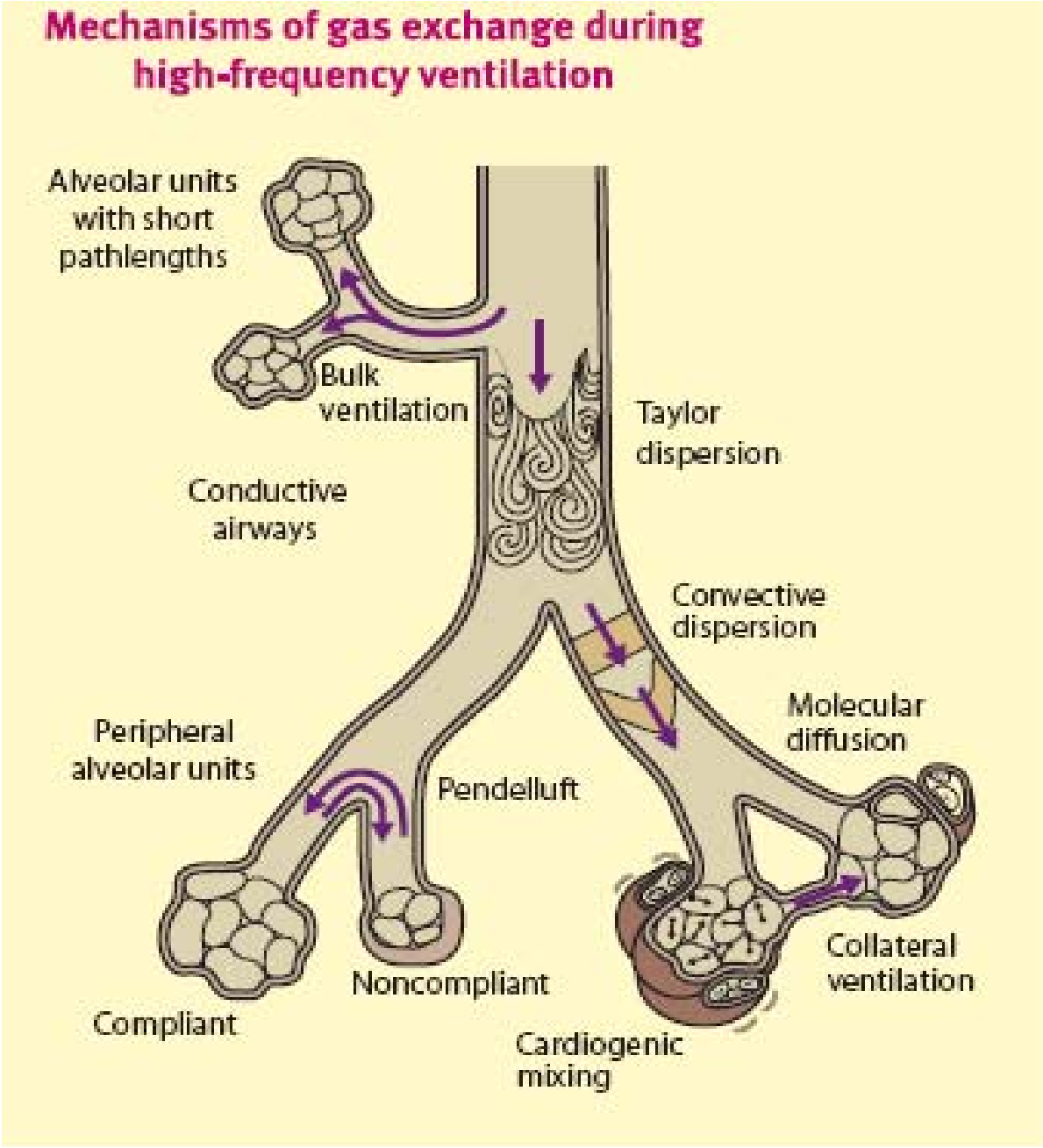
The inspiratory gas velocity profile is asymmetric and parabolic. This leads to gas molecules in the centre of the airway moving faster than those at the periphery. At airway bifurcations there is a skewing of the inspiratory profile streaming 'fresh gas' along inner airway wall, whilst the expiratory velocity profile is flat and more symmetric, streaming "alveolar" air along the outer airway wall. It is the asymmetry of these velocity profiles results in net convective transport of gas. (Chang, 1984; Ferguson & Stewart, 2002).



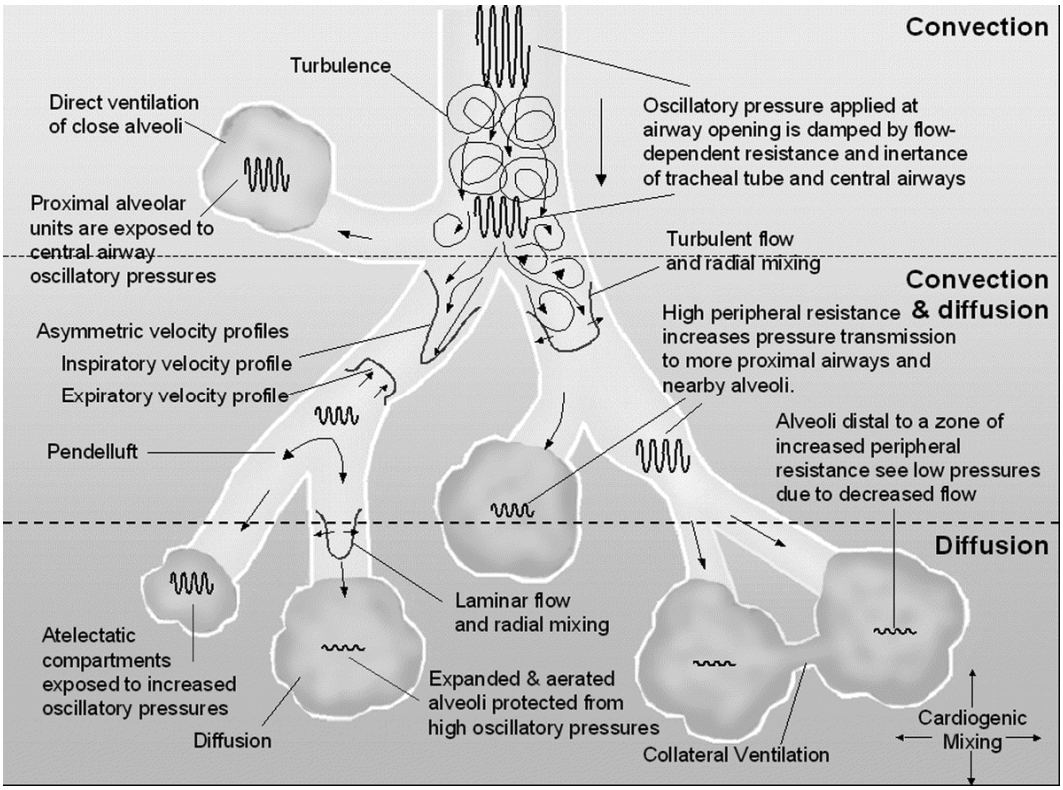
Inspiratory gas velocity profile (Chan, 2007)

Taylor's Dispersion

Longitudinal dispersion or Taylor's dispersion is caused by the interaction between axial flow velocities and radial transport. Gas flow upon inspiration is direct and axial whilst expiratory flows travel along the outer edges of the airways and could be described as radial. The resultant swirling of flow produces turbulence in the conducting airways and lead to increased dispersion of gases and increased gas mixing (Chang, 1984; Ferguson & Stewart, 2002).



Mechanisms of gas transport and gas exchange



Mechanisms of convective and diffusive gas transport during HFOV (Pillow, 2005)

Definition of Terms

Conventional ventilation

Utilises tidal volumes to provide adequate gas exchange in volume control or pressure control modes of ventilation

ALI / ARDS

Acute Lung Injury/Acute Respiratory Distress Syndrome includes diffuse bilateral lung infiltrates and a P/F ratio <200-300 mmHg

HFOV

High Frequency Oscillatory Ventilation is defined as ventilation at a frequency greater than four times the normal respiratory rate

Bias Flow

A continuous flow of gas delivered by the Senormedic 3100B HFOV

Frequency

The rate of oscillation measured in Hertz i.e. 1 Hertz = 60 breaths per minute

Amplitude

Represents the force of piston movement against the diaphragm from a resting position. Often described as ΔP (delta pressure or power)

mPaw

The mean airway pressure

Recruitment Manoeuvres (RM)

Ventilation recruitment manoeuvres including PEEP, suctioning, positioning, ventilation mode, inspiratory holds and pulmonary vasodilators

Workbook

1. Describe in your own words the following HFOV terms.

Frequency

Bias Flow

Amplitude (ΔP, Power)

% Inspiratory time

Mean Airway Pressure (mPaw)

2. State the indications for performing a recruitment manoeuvre whilst a patient is on HFOV

3. Outline the procedure for performing a recruitment manoeuvre

4. List when would you clamp the endotracheal tube?

5. Outline your actions if you observe a decrease or sudden absence of chest wiggle factor

6. What are the criteria for patient selection for HFOV?

7. What are the determinants that directly affect the following in HFOV?

Oxygenation

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Ventilation

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8. State four lung protective strategies that are commonly used in conventional ventilation

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9. State three adjunct strategies that maybe implemented in combination with lung protective strategies

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