An introduction to mechanical ventilation

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INTRODUCTION
One of the main interventions offered in an Intensive Care Unit (ICU) is advanced respiratory support. An understanding of the indications and types of mechanical ventilation is therefore essential for anyone working in this environment.

INDICATIONS FOR MECHANICAL VENTILATION
The main indication for mechanical ventilation is respiratory failure. However, other clinical indications include:

- prolonged postoperative recovery,
- altered conscious level,
- inability to protect the airway, or
- exhaustion, when the patient is likely to develop respiratory failure.

The aim of mechanical/artificial ventilation is to improve gas exchange, to reduce the work of breathing and to avoid complications while maintaining optimal conditions for the patient to recover from their underlying illness. Whatever the indication for respiratory support, the underlying condition of the patient must be reversible; otherwise subsequent weaning may not be possible.

RESPIRATORY FAILURE
This is the primary indication for respiratory support. It occurs when pulmonary gas exchange is sufficiently impaired to cause hypoxaemia (PaO₂ less than 8kPa), with or without hypercarbia. The causes of respiratory failure are diverse and the problem may occur due to disease at the alveolar/endothelial interface (e.g. pulmonary oedema) or failure of the respiratory pump mechanism, resulting in inadequate minute ventilation (e.g. flail segment accompanying fractured ribs). It is difficult to define specific criteria for commencing mechanical ventilation and the decision is a clinical one, influenced by different factors for each patient.

Indications to consider include:

- Respiratory rate > 35 or < 5 breaths per minute,
- Exhaustion with a laboured pattern of breathing,
- Hypoxia - central cyanosis, SaO₂ < 90% on oxygen or PaO₂ < 8kPa,
- Hypercarbia - PaCO₂ > 8kPa,
- Decreasing conscious level (Glasgow Coma Score < 8),
- Significant chest trauma,
- Tidal volume < 5ml.kg⁻¹ or vital capacity < 15ml.kg⁻¹.

Causes of respiratory failure

Inadequate gas exchange
- Pneumonia, pulmonary oedema, acute respiratory distress syndrome (ARDS).

Inadequate breathing
- Chest wall problems e.g. fractured ribs, flail chest,
- Pleural problems e.g. pneumothorax, haemothorax,
- Respiratory muscle failure e.g. myasthenia gravis, poliomyelitis, tetanus,
- Central nervous system depression e.g. drugs, brain stem compression, head injury.

Obstructed breathing
- Upper airway obstruction e.g. epiglottitis, croup, oedema, tumour,
- Lower airway obstruction e.g. asthma and bronchospasm.

Other indications for ventilation
Patients in this category are ventilated to assist in the management of other, non-respiratory conditions and may include:

- Control of intracranial pressure in head injury,
- Airway protection following drug overdose,
- Following cardiac arrest,
- For recovery after prolonged major surgery or trauma,
- As support when other organs systems are failing – e.g. severe shock or acidosis requiring aggressive therapy.
TYPES OF MECHANICAL VENTILATION

The most commonly used type of artificial ventilation is intermittent positive pressure ventilation (IPPV). The lungs are intermittently inflated by positive pressure, generated by a ventilator, and gas flow is delivered through an oral tracheal or tracheostomy tube.

Tracheal intubation is usually achieved by the oral route although nasal intubation may be better tolerated by the patient during prolonged ventilation. Although more secure, nasotracheal intubation is technically more challenging and has a higher incidence of bleeding and infective complications such as sinusitis. Tracheal intubation not only allows institution of IPPV, but also reduces dead space and facilitates airway suctioning. However, it is also possible to deliver positive pressure ventilation to cooperative patients in a non-invasive manner through a tight-fitting face or nasal mask (Non-invasive ventilation, NIV).

In general, there are two main modes of ventilation commonly in use in ICU - modes where the ventilator delivers a preset tidal volume, and those that deliver a preset inspiratory pressure, during each inspiration. Modern ventilators allow different modes of ventilation and the clinician must select the safest and most appropriate mode of ventilation for the patient.

Volume-controlled ventilation (or volume-cycled ventilation)

The ventilator delivers a preset tidal volume regardless of the pressure generated. The lung compliance (stiffness) of the lungs determines the airway pressure generated, so this pressure may be high if the lungs are stiff, with the resultant risk of barotrauma. Barotrauma describes rupture of the alveoli resulting in pneumothorax and mediastinal emphysema, but also describes acute lung injury that can result from over-distension of alveoli (volutrauma).

Pressure-controlled ventilation (or pressure-preset ventilation)

The ventilator delivers a preset target pressure to the airway during inspiration. The resulting tidal volume delivered is therefore determined by the lung compliance and the airway resistance.

MODES OF VENTILATION

Overview

Modern ventilators have a variety of modes that can be selected depending on the patient’s illness. For example (see Figure 1) a patient with severe respiratory disease (whether primary or secondary to other disease), who requires ventilation, will initially require full ventilation with mandatory breaths; they will be heavily sedated and may require paralysis. As another example, one of the primary goals of ventilation in a severely head-injured patient is to achieve a low-normal CO₂ level, which requires a controlled minute volume, delivered by fully controlled ventilation.

As the patient’s respiratory disease improves, the patient will generate their own respiratory rate and require less positive pressure support during each breath. So, as their clinical state improves, the mode and settings of the ventilator are adjusted to reflect this. In time the low level of support they require will indicate that they are in a fit state for extubation and withdrawal of respiratory support. Figure 1 shows a simplified summary of this process in a theoretical patient.

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**Figure 1.** Summary of modification of ventilator mode and settings, mirroring improvement in a theoretical patient’s clinical state and respiratory performance; CMV - controlled mandatory ventilation; PSV - pressure support ventilation; ASB - assisted spontaneous breathing; SIMV - synchronised intermittent mandatory ventilation.
In some resource-poor settings, the ventilator used in ICU may offer only a mandatory mode. This is suitable for full ventilation of heavily sedated and paralysed patients, but is poorly tolerated as patients improve, wake up and begin to breathe for themselves. Gradual weaning through SIMV and pressure support ventilation is not possible and so weaning must be achieved through daily ‘sedation holds’ to see how the patient copes when breathing without ventilatory support, receiving supplemental oxygen from a T-piece. Many of the more modern ICU ventilators, with advanced modes of ventilation, are unsuited to an environment where malfunction is more likely (due to heat, humidity and dust), piped or cylinder air and oxygen may not be available and spare parts and servicing are unavailable or not affordable. Machines designed to run from an electric power source and using oxygen from an oxygen concentrator are available; an example is the HT50® ventilator (Newport Medical Instruments, California), however long term use is limited by damage to more fragile parts of the breathing circuit, such as the expiratory valve.

**Intermittent mandatory ventilation (IMV)**

This is a combination of spontaneous and mandatory ventilation. Between the mandatory controlled breaths, the patient can breathe spontaneously and unassisted. IMV ensures a minimum minute ventilation, but there will be variations in tidal volume between the mandatory breaths and the unassisted breaths.

**Synchronised intermittent mandatory ventilation (SIMV)**

With SIMV, the mandatory breaths are synchronised with the patient’s own inspiratory effort which is more comfortable for the patient.

**Pressure-support ventilation (PSV) or assisted spontaneous breathing (ASB)**

A preset pressure-assisted breath is triggered by the patient’s own inspiratory effort. This is one of the most comfortable forms of ventilation. The preset pressure level determines the level of respiratory support and can be reduced during weaning. There are no mandatory breaths delivered, and ventilation relies on the patient making some respiratory effort. There is, however, no back up ventilation should the patient become apnoeic, unless this mode is combined with SIMV. The name given to this mode of ventilation is determined by the manufacturer of each machine.

**Positive end-expiratory pressure (PEEP)**

PEEP should be used with all forms of IPPV. A positive pressure is maintained during expiration, preventing collapse of the distal airways, minimising damage to alveoli by repeated deflation and re-inflation, and also improving the compliance of the lung. PEEP improves arterial oxygenation and, in severe disease (e.g. ARDS), higher levels of PEEP cause sequentially improved oxygenation. However, PEEP causes a rise in intrathoracic pressure and can reduce venous return and so precipitate hypotension, particularly in hypovolaemic patients. With low levels of PEEP (5-10cmH₂O) these effects are usually correctable by intravenous volume loading. In its simplest form PEEP can be achieved using an adjustable valve on the expiratory limb of the breathing circuit. PEEP valves are available that attach to the Ambu-E valve of a simple circuit, used with an Oxford bellows.

**Continuous positive airway pressure (CPAP)**

CPAP is effectively the same as PEEP but in spontaneously breathing patients. Effective delivery of CPAP requires a source of oxygen in excess of the maximal inspiratory flow in inspiration (usually about 30L.min⁻¹). This is difficult to achieve where the sole source of oxygen is an oxygen concentrator. CPAP is useful for patients with poor oxygenation, but gives no ventilatory support, so does not generally improve CO₂ clearance.
**INITIATING MECHANICAL VENTILATION**

The act of sedating, paralysing and intubating a critically ill patient is challenging and can result in severe cardiac and/or respiratory compromise or even death. Choose the drugs that you are most familiar with, but aim to use a fraction of the dose that the patient would require when well. Ketamine is useful as an induction agent as it confers some degree of haemodynamic stability. Some intensivists favour a combination of fentanyl and midazolam. Induction of anaesthesia rapidly obviates the production of endogenous catecholamines in patients who have a high work of breathing; this, for example in otherwise young fit asthmatics with an acute exacerbation, may precipitate profound haemodynamic compromise. For patients on the verge of cardiovascular collapse, it is sometimes safest to intubate using only local anaesthesia, applied topically to the airway and larynx.

When initiating artificial ventilation, the aim is to provide the patient with a physiological tidal volume and ventilatory rate that is adjusted to allow for the demands of their pathological condition. Recommendations for initial ventilator settings are generally derived from the ARDSnet study, that showed that a ‘lung protective ventilation strategy’ (in ARDS) reduced the contribution that机械 ventilation made to lung trauma during critical illness.\(^2\)

Recommendations for initial ventilator settings are generally derived from the ARDSnet study, that showed that a ‘lung protective ventilation strategy’ (in ARDS) reduced the contribution that mechanical ventilation made to lung trauma during critical illness.\(^2\) Bear in mind that it is very difficult to adequately replicate the respiratory compensation of a patient with a severe metabolic acidosis, with mechanical ventilation. Acidosis is likely to worsen in the initial period after intubation and commencement of mechanical ventilation.

Suggested initial ventilator settings are:

- \(\text{FiO}_2\) 1.0 initially but then reduce – aim for \(\text{SaO}_2\) 93-98%,
- PEEP 5cmH\(_2\)O,
- Tidal volume 6-8ml.kg\(^{-1}\),
- Inspiratory pressure 20cmH\(_2\)O (15cmH\(_2\)O above PEEP),
- Frequency 10-15 breaths per minute,
- Pressure support (ASB) 20cmH\(_2\)O (15cmH\(_2\)O above PEEP),
- I:E ratio 1:2,
- Flow trigger 2L.min\(^{-1}\),
- Pressure trigger -1 to -3 cmH\(_2\)O.

These settings should be titrated against the patient’s clinical state and level of comfort. Some conditions require particular consideration. Patients with severe bronchospasm are at risk of dynamic hyperinflation (‘breath-stacking’) - a prolonged expiratory phase means that the next inspired breath occurs before full expiration has taken place. The result is high intra-thoracic pressures, with worsening lung compliance and haemodynamic collapse. Initial ventilation should be by hand, using a bag-valve-mask, with auscultation to ensure expiration is complete - the required ventilatory rate to allow this may be as slow as 3 to 4 breaths per minute. For all patients in whom effective ventilation is difficult due obstructive disease or due to poor compliance, the \(\text{CO}_2\) should be allowed to rise in order to avoid excessive high pressure ventilation. This *permissive hypercapnea* is tolerated until it causes a dangerous level of acidosis.

**OPTIMIZING OXYGENATION**

When settling a patient on the ventilator, it is sensible practice to initially set the \(\text{FiO}_2\) at 1.0 (100%) and then wean rapidly to a \(\text{FiO}_2\) adequate to maintain \(\text{SaO}_2\) of > 93%. An \(\text{FiO}_2\) of greater than 0.6 for long periods should be avoided if possible because of the risk of oxygen-induced lung damage.

**PEEP**

Strategies to improve oxygenation (other than to increase \(\text{FiO}_2\)) include increasing the mean airway pressure by either raising the PEEP to 10cmH\(_2\)O or, in pressure-preset ventilation modes, by increasing the mean airway pressure by either raising the PEEP to 10cmH\(_2\)O or, in pressure-preset ventilation modes, by increasing the peak inspiratory pressure. However, care should be taken to avoid very high inflation pressures (above 35cmH\(_2\)O) as this may cause barotrauma to the lungs. In severe hypoxia, it may be possible to improve oxygenation by increasing the PEEP further to 15cmH\(_2\)O (or above) and using small (6-8ml.kg\(^{-1}\)) tidal volumes more frequently. However, this may cause a reduction in blood pressure and may be poorly tolerated by the patient, requiring intravenous fluid loading and inotropic or vasopressor therapy. The PEEP strategy employed in the ARDSnet trial is widely used as a guide to application of appropriate levels of PEEP and is shown in Box 1.\(^2\)

**Altering the I:E ratio**

Because oxygenation is largely determined by the mean airway pressure through the respiratory cycle, prolonging the inspiratory time may improve this. This is achieved by increasing the I:E ratio (to 1:1) or even inverting the ratio (e.g. to 1.5:1 or 2:1). Heavy sedation and paralysis are usually required for this. Ensure that sufficient time is allowed for expiration.

**Lung recruitment strategies**

Improvements in oxygenation can be achieved by exposing the lungs to a higher pressure for a short period of time. An example of such a lung recruitment manoeuvre is to apply high CPAP at 40cmH\(_2\)O for 40 seconds.

**Prone positioning**

Placing the patient face down (prone) whilst well sedated may improve oxygenation by re-expanding collapsed alveoli and improving the distribution of blood perfusion in the lung relative to ventilation. In this position, patient monitoring and care is difficult, and this approach should be undertaken with caution. There is a high risk of dislodging tubes or cannulae whilst rolling, and the patient should

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**Box 1 Guide to acceptable levels of PEEP**

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[1] Update in Anaesthesia | www.anaesthesiologists.org
not remain in the prone position for more than 18 hours in every 24-hour period. Patients should have all pressure areas protected (eyes, nose, neck, shoulders, thorax, pelvic area, knees) whilst allowing free diaphragmatic and abdominal movement to prevent high abdominal pressures.

**Airway pressure release ventilation (APRV)**
The ventilator alternates a high PEEP (e.g. 20cmH₂O) for long periods (e.g. 3-4 seconds), with low PEEP (e.g. 5cmH₂O) for short periods (e.g. 1 second). This maintains recruitment of lung tissue, and the patient can take further breaths during the high pressure period. However carbon dioxide removal can be difficult and achieving optimal sedation for the patient to breathe on top of the ventilator breaths can be problematic.

Other methods of ventilation which may improve oxygenation, are detailed at the end of the article.

**OPTIMISING CARBON DIOXIDE ELIMINATION**
Carbon dioxide elimination is improved by increasing minute ventilation, either by increasing the tidal volume or the respiratory rate. Aiming for a normal level of carbon dioxide may require high minute volumes and can be hard to achieve in sick patients. The PaCO₂ is usually allowed to rise, causing a respiratory acidosis. This is termed *permissive hypercapnia* and can be accepted as long as the blood pH does not fall below 7.20. This level of acidosis is usually well tolerated.

**Sedation**
Most patients require sedation in order to tolerate the endotracheal tube. Ideally, only light sedation should be given so that the patient can understand and cooperate with ventilation, while continuing to make some respiratory effort.

**PROBLEMS DURING MECHANICAL VENTILATION**

**‘Fighting the ventilator’**
When a patient starts to breathe out of phase with the ventilator or becomes restless or distressed during IPPV, there is a fall in the delivered tidal volume, due to a rise in respiratory resistance. This may result in inadequate ventilation and hypoxia. Factors to consider include:

- **Patient factors** - breathing against the ventilator’s inspiratory phase, breath holding and coughing.
- **Decreased pulmonary compliance** - pulmonary pathology, including oedema or infection and pneumothorax.
- **Increased airway resistance** - bronchospasm, aspiration, excess secretions
- **Equipment** - ventilator disconnection, leak, failure. ET tube blocked, kinked, dislodged.

**Management of a patient ‘fighting the ventilator’**
Is the patient hypoxic? If yes - follow ABC:

- Is the endotracheal tube patent and correctly positioned? Reintubate if necessary.
- Give 100% O₂ by manual ventilation via self-inflating bag.
- Check chest expansion is adequate.
- Auscultate chest to assess bilateral air entry.
- Check heart rate and blood pressure.
- Check ventilator and apparatus for disconnection/leak/failure.

**Diagnosing the problem**

**High airway pressure due to blocked ET tube**
- The patient may be biting the tracheal tube - insert oral airway and sedate patient.
- Tube is blocked by secretions - suction with catheter and consider irrigation with 5ml saline. Change ET tube if necessary.
- ET tube over-inserted into right main bronchus - pull tube back.

**High airway pressure due to intrapulmonary factors**
- Is there evidence of bronchospasm? Ensure ET tube not over-inserted, stimulating the carina. Give bronchodilators.
- Is there evidence of pneumothorax, haemothorax, lung collapse or pleural effusion? Examine, request chest Xray and treat appropriately.
- Is there pulmonary oedema? Treat with diuretics, treat cardiac failure or arrhythmias.

**Sedation/analgesic factors**
- Is the patient hyperventilating due to hypoxia or hypercarbia (cyanosis, tachycardia, hypertensive and sweating). Increase FiO₂ and increase the mean airway pressure with PEEP. Increase minute ventilation (if hypercarbic).
- Coughing, discomfort or pain (raised heart rate and blood pressure, sweating and grimacing). Look for causes of discomfort, e.g. endotracheal tube irritation, full bladder, pain. Review analgesia and sedation. Change ventilation mode to one better tolerated e.g. SIMV, PSV. Neuromuscular blockade - only if all other options explored.
- Ideally sedation is delivered by infusion pumps - commonly propofol is used with an infusion of an opioid such as morphine. Where pumps are not available regimes of intramuscular benzodiazepine and intramuscular opioid are used, although this technique is associated with periods of over-sedation and periods of under-sedation.

**PROVIDING OPTIMAL VENTILATION AND PREVENTING HARM**
There is no single correct form of ventilation - each clinician has their favourite method, depending on the clinical circumstances. However, mechanical ventilation can cause harm, so where possible the following should be considered:

- Check the ET tube cuff pressure if possible, and aim to keep between 30-60cmH₂O. If there is a leak from the ETT, then the
cuff is either deflated or damaged and the ET tube should be replaced.

- Position the patient 30° head up, to reduce oesophago-gastric reflux and the risk of ventilator-associated pneumonia.
- Keep peak inspiratory pressure less than 35cmH2O, regardless of the mode of ventilation.
- Aim to have a peak plateau pressure of less than 30cmH2O.
- Tidal volumes should be 6-8ml.kg⁻¹ of ideal body weight.
- Avoid high respiratory rates if possible - these can worsen atelectrauma.
- Avoid hyperoxia as far as possible – aim SaO₂ 93-98%, or PaO₂ 8-10kPa.
- Use all of the monitoring that you have available in order to ensure that the patient remains haemodynamically stable whilst ventilated. Set the alarms to provide you with information on clinically relevant changes in the measured variables. In particular, it is recommended that, where available, all ventilated patients are monitored with capnography in order to detect problems with ventilation early.
- Aim to avoid the patient fighting the ventilator, especially in the early stages of their illness.
- Provide a sedation break every day, unless maintaining optimal ventilation is absolutely critical (e.g. when prone or using neuromuscular blockade).
- Maintain a negative fluid balance in ARDS using diuretics, unless there is critical renal function which cannot be supported. This will not be possible early in a septic illness where volume resuscitation is paramount.
- Use prophylaxis for GI ulcers – although these raise the gastric pH and make ventilator-associated pneumonia more likely, the mortality from GI bleeding is high. Nasogastric feed should be used to prevent gastric ulcers and to mitigate weight loss in critically ill patients.
- Use thromboprophylaxis for venous thrombosis. Pulmonary embolism is common and high risk in critically ill patients. A combination of compression stockings, mechanical calf pumps and pharmacological prophylaxis is best if possible.
- Maintain oral hygiene, preferably using chlorhexidine mouthwash. These reduce the oral flora and aim to reduce the incidence of ventilator-associated pneumonia
- Reduce ventilator settings, sedation and other organ support whenever possible during weaning.

**WEANING FROM VENTILATION**

There are a number of complications associated with mechanical ventilation, including barotrauma, pneumonia and decreased cardiac output. For these reasons, it is essential to discontinue ventilatory support as soon as the patient improves. Indeed in most resource-poor settings, prolonged ventilation is unsustainable and inappropriate.

Weaning is indicated when the underlying condition is resolving. Many patients are ventilated for a short period or time, for example those recovering from major surgery, whereas others are ventilated for many days (e.g. for ARDS). During long periods of prolonged ventilatory support, the respiratory muscles weaken and atrophy. As a consequence, the speed of weaning is often related to the duration and mode of ventilation. Assisted modes of ventilation and good nutritional support are important to prevent atrophy of the respiratory muscles.

Patients recovering from prolonged critical illness are at risk of developing critical illness polyneuropathy. In this condition, there is both respiratory and peripheral muscle weakness, with reduced tendon reflexes and sensory abnormalities. Treatment is supportive. There is evidence that long-term administration of some aminosteroid muscle relaxants (such as vecuronium) may cause persisting paralysis. For this reason, vecuronium should not be used for prolonged neuromuscular blockade.

**Indications for weaning**

The decision to start weaning is often subjective and based on clinical experience. However, there are some guidelines that may be helpful:

- Underlying illness is treated and improving.
- Respiratory function:
  - Respiratory rate < 35 breaths per minute,
  - FiO₂ < 0.5, SaO₂ > 90%, PEEP < 10cmH₂O,
  - Tidal volume > 5ml.kg⁻¹,
  - Vital capacity > 10ml.kg⁻¹,
  - Minute volume < 10L.min⁻¹.
- Absence of infection or fever.
- Cardiovascular stability, optimal fluid balance and electrolyte replacement.

Prior to weaning, there should be no residual neuromuscular blockade and sedation should be minimised so that the patient can be awake, cooperative and in a semirecumbent position. Weaning is likely to fail if the patient is confused, agitated or unable to cough.

**Modes of weaning**

There is debate over the best method for weaning and no one technique has been found to be superior to others. There are several different approaches.

**Unsupported spontaneous breathing trials**

The machine support is withdrawn and a T-Piece (or CPAP) circuit can be attached intermittently for increasing periods of time. The patient gradually takes over the work of breathing, with shortening rest periods back on the ventilator.

**Intermittent mandatory ventilation (IMV) weaning**

The ventilator delivers a preset minimum minute volume which is gradually decreased as the patient takes over more of the respiratory workload. The decreasing ventilator breaths are synchronised to the patient’s own inspiratory efforts (SIMV).

**Pressure support weaning**

In this mode, the patient initiates all breaths and these are ‘boosted’ by...
the ventilator. This weaning method involves gradually reducing the level of pressure support, thus making the patient responsible for an increasing amount of ventilation. Once the level of pressure support is low (5-10 cmH₂O above PEEP), a trial of T-Piece or CPAP weaning should be commenced.

**Failure to wean**
During the weaning process, the patient should be observed for early indications of fatigue or failure to wean. These signs include distress, increasing respiratory rate, falling tidal volume and haemodynamic compromise, particularly tachycardia and hypertension. At this point it may be necessary to increase the level of respiratory support as, once exhausted, the respiratory muscles may take many hours to recover.

It is sensible to start the weaning process in the morning to allow close monitoring of the patient throughout the day. In prolonged weaning, it is common practice to increase ventilatory support overnight to allow adequate rest for the patient.

**Tracheostomy in the intensive care unit**
The commonest indication of tracheostomy in an ICU setting is to facilitate prolonged artificial ventilation and the subsequent weaning process. Tracheostomy allows a reduction in sedation and thus increased cooperation with weaning. It also allows effective tracheobronchial suction in patients who are unable to clear pulmonary secretions, either due to excessive secretion production or due to weakness following critical illness. Oral hygiene is improved and the shorter tracheostomy tube aids weaning.

**OTHER METHODS OF VENTILATION**
Some patients have such severe respiratory illness that the techniques above cannot provide sufficient oxygen to prevent organ failure. In this situation there are a number of other techniques that may be used, although an improvement in mortality for these techniques has not been shown.

**High frequency oscillatory ventilation**
This mode maintains high mean airway pressures (24-40 cmH₂O) with very fast respiratory oscillations (3-15 Hz). Therefore there is no ‘tidal volume’, as the volume of gas moving with each oscillation is very small. The method of gas flow in this mode is very complex and cannot be compared to normal mechanical ventilation. Problems include hypercapnia, thick tenacious secretions with mucous plugging, barotrauma, the requirement for heavy sedation and neuromuscular blockade and hypotension from increased intra-thoracic pressure necessitating fluid loading and inotropic support.

**CONCLUSION**
The ability to offer short term ventilatory support for patients with reversible respiratory failure is a major feature of intensive care management. This article has outlined the very basics of ventilatory management. Each clinician must become familiar with the machines available to them and develop strategies to institute and wean ventilation safely. It is vital that each unit has clearly defined criteria to decide which patients will benefit from ventilatory support. In resource poor settings prolonged ventilation does not represent appropriate use of medical resources and for each patient there must a good prospect for successful and timely weaning of ventilation.

**REFERENCES**