Strategies for ventilatory support

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Since the introduction of manual positive pressure ventilation during the Copenhagen polio epidemic of 1952, a range of mechanical devices and methods of ventilatory support have been developed to provide life-saving respiratory therapy to critically ill patients. In addition to the development of the equipment hardware to enable effective mechanical ventilation, there has been a gradual realisation that a single mode of ventilation is not universally applicable to all patients, individual pathologies, or to individual patients at various stages in the evolution of their pulmonary pathology. While mechanical ventilation can undoubtedly be life-saving, it can also cause lung damage as a result of its non-physiological method of promoting effective gas exchange. This iatrogenic problem is known as ventilator induced lung injury and, although it may be a problem in any patient requiring mechanical ventilation for any reason, is of particular importance to those patients requiring mechanical ventilation as a consequence of trauma. This chapter describes the range of ventilatory support techniques available, the problem of ventilator induced lung injury with specific reference to trauma patients and offers a strategy for ventilatory support in the trauma patient.

Origin of ventilatory support

The birth of mechanical ventilation for respiratory failure resulted from the work of Henry Lassen and Bjorn Ibsen during the 1952 polio epidemic in Copenhagen. They demonstrated a reduction in mortality due to respiratory failure from 90% to 30% as a result of manual intermittent positive pressure ventilation used in place of 'iron lung' systems using externally applied negative pressure. At the height of the epidemic, 70 patients had to be ventilated simultaneously, 24 h per day, until their own spontaneous respiratory effort had improved to the point where they could sustain adequate gas exchange. The apparatus used was simple; a cylinder of oxygen, reducing valve, humidifier, CO$_2$ absorber, one way valve connected to a rubber bag and to the patient via a tracheostomy tube. The tidal volume for each breath was obtained by squeezing the rubber bag, the motive force being provided by teams of
Strategies for ventilatory support

medical and dental students working in shifts. They became adept at varying the tidal volume and rate to produce normocarbia, could vary the pressure generated on bag squeezing as the patient's lung compliance changed and were able to detect and synchronise with the patient's own respiratory effort as the patients made a gradual recovery. It is this flexibility of response that is aspired to by all modern mechanical ventilators, since the patient's ventilatory requirements, ability to tolerate mechanical ventilation, lung mechanics and responses will change with the natural history of the disease producing the need for mechanical ventilation.

The need for ventilatory support in trauma

Inadequate ventilation resulting in hypoxia and hypercapnia as a result of trauma may have many causes but may be divided into thoracic or extra-thoracic origins.

Thoracic trauma

Thoracic trauma is quoted as causing one out of every four deaths from trauma in North America. Blunt trauma is more common than penetrating trauma but both mechanisms of injury may degrade respiratory function by effects on the chest wall, airways or lung parenchyma by contusion, fractures, rupture/penetration, thermal or chemical inhalational injury.

Extra-thoracic trauma

Extra-thoracic trauma causes ventilatory failure by a range of mechanisms of which head injury with alteration of level of consciousness is the most common. Other causes include blood loss from non-thoracic causes sufficient to cause diminished consciousness, overdose of opioid analgesics, high spinal cord injury, thermal injury to upper airway or the effects of multiple trauma on other areas of the body with sepsis or systemic inflammatory response leading to the production of acute respiratory distress syndrome. In addition, ventilatory support after trauma is often initiated electively, e.g. to enable control of intracranial pressure or after prolonged surgical procedures.

Whatever the mechanism of trauma resulting in ventilatory failure, the need for ventilation is determined by careful monitoring for signs of
Trauma

airway obstruction, tachypnoea (adult respiratory rate > 35), use of accessory muscles of respiration, cyanosis or measured hypoxia (PaO$_2$ < 8 kPa on FiO$_2$ = 0.5), measured hypercarbia (PaCO$_2$ > 8 kPa if not chronic hypercapnia) or objective measurements of decreased mechanical reserve such as FEV$_1$ < 10 ml/kg or vital capacity < 15 ml/kg. The detection of cyanosis in the profoundly anaemic, peripherally shut-down trauma patient is difficult; pulse oximetry may be unreliable and gives no indication of carbon dioxide levels. Serial blood arterial blood gas analysis will provide the most useful indication of the need for ventilatory support. Having made the decision to proceed, then ventilatory support is most commonly provided via a definitive airway, i.e. a cuffed tube inserted in the trachea by various routes. The choice of airway, methods of acquisition and the precautions to be taken in the trauma patient requires specialised skill and knowledge.

**Options for ventilatory support**

There is a plethora of means for providing ventilatory support coupled, it seems, with an even greater number of acronyms to describe the various forms of mechanical ventilation. The goal of each is to raise functional residual capacity above the closing capacity (lung volume at which small airways begin to close) thus decreasing micro-atelectasis and, by taking over or supporting the movement of gas with each breath, to decrease the work of breathing in an attempt to reduce the hypoxia and/or hypercapnia of type 1 or 2 respiratory failure. There is no evidenced-based trial that proves one technique to be more effective than another. The changing nature of pulmonary and extra-pulmonary factors in an individual trauma patient means that one particular mode of ventilation may be advantageous only at a given time and that part of the science of success in their management rests with choosing the best mode for each patient at that particular time in their illness.

**Non-invasive respiratory support**

For some patients, non-invasive (i.e. non-intubational) forms of respiratory support (NIRS) are applicable. The types of NIRS most widely used are continuous positive airways pressure applied by mask (mask CPAP), non-invasive positive pressure ventilation by mask (NIPPV) and negative pressure ventilation (NPV).

Consider a patient who has sustained isolated blunt chest trauma resulting in a minor degree of underlying pulmonary contusion and
Strategies for ventilatory support

fractured ribs such that he is hypoxic and, because of the limitation of chest wall movement due to pain, has a degree of hypercapnia. Pulmonary contusion may be defined as localised interstitial oedema and haemorrhage not conforming to anatomical lung segments and directly associated with chest wall injury. It is a relatively common, occurring in 17% of patients with multiple injury (Injury Severity Score > 15) in the Yale Trauma Registry. Alveolar haemorrhage and parenchymal damage are maximal in the first 24 h after injury and hypoxaemia and hypercapnia peak at about 72 h. If the patient is able to breathe spontaneously and hypoxia is the main problem, then a tightly applied face mask with a high volume, low pressure soft plastic rim held in place by a special harness is connected to a circuit delivering and air/oxygen mixture of the required FiO₂. The flow of fresh gas in the circuit must be sufficient to keep the positive pressure set by the expiratory valve (2.5–10 cm of water) almost constant throughout the respiratory cycle with only minimal negative pressure drop during inspiration. Various commercial products using Venturi or bellows systems are available, but consume large amounts of fresh gas. Depending on the degree of pulmonary contusion, then the patient may be appropriate for management unintubated using mask CPAP and intubation avoided in over 85% of patients. For CPAP to be successful, the patient must have adequate analgesia, be alert, co-operative and have no facial injuries including basal skull fractures. Patients often find the tight fitting mask uncomfortable and require short periods of respite on ordinary fixed FiO₂ masks. At higher levels of CPAP (> 7.5 cm of water), the work of breathing against the expiratory valve may result in an elevation of PaCO₂ and CPAP alone is generally not effective in most cases of type 2 respiratory failure. If the patient does not have a nasogastric tube, then air swallowing and gastric dilatation may be problematic and hinder diaphragmatic excursion or provoke regurgitation.

The use of NIPPV and NPV is well documented in the management of patients with non-traumatic causes of ventilatory failure, particularly for home ventilation of patients who are chronic carbon dioxide retainers or have high spinal injury who do not have a permanent tracheostomy but require nocturnal ventilation. NIPPV uses portable electrically powered volume or pressure cycled ventilators to deliver synchronised positive pressure breaths via a tight fitting nasal or full face mask held in place by a harness. NPV requires a made to measure ‘cuirass’ which is worn over the chest and sealed at the neck and waist. This is then connected to a pump which cyclically produces negative pressure within the cuirass by sucking some of the air out. The intrathoracic pressure is exceeded by atmospheric pressure and air flows into the chest. The pump cycles to atmospheric pressure and expiration occurs as a result of the normal elastic recoil of the lungs and chest wall. The device is essentially a cut-down version of the old cylindrical ‘iron lung’. There is
little published data on their use in the acute management of pulmonary trauma and, at present, it would not be possible to recommend their routine use.

**Conventional modes of positive pressure ventilation**

World-wide, there are an enormous number of different types of mechanical ventilators, but they have well-defined fundamental principles and function in well-defined modes.

Intermittent positive pressure ventilation (IPPV) is the basic mode of mechanical ventilation used during balanced anaesthesia and on the Intensive Care Unit. It requires the presence of a definitive airway and the patient must be tolerant to the presence of the airway and the cyclical delivery of a humidified fresh gas mixture the FiO\(_2\) being set by an air-oxygen blender. The ventilator is set to deliver a fixed tidal volume at a fixed rate to produce a fixed minute ventilation required to ensure adequate CO\(_2\) elimination. This mode of ventilation is also known as controlled mandatory ventilation (CMV). The use of positive intrathoracic pressure is not without problems, predominantly effecting the cardiovascular and respiratory systems.

The patient has no option but to accept each ventilator generated breath at the time determined by the ventilator and unless sedated to the point of tolerance, and in some cases, paralysed with neuromuscular junction blocking drugs, might try to breathe against the ventilator decreasing the efficiency of gas exchange or causing iatrogenic injury to the lung and depression of the cardiac output. Over-sedation itself may cause decreased cardiac output by direct drug-induced myocardial depression in addition to the decrease in preload caused by positive intra-thoracic pressure. This effect is particularly marked if the patient is inadequately fluid resuscitated after concurrent haemorrhagic shock, in the elderly or other patient with intercurrent myocardial dysfunction. Drug-induced paralysis for long periods may have damaging effects on neuromuscular function. There is an increased mismatch of ventilation and perfusion. Oliguria, sodium retention and possibly some reduction in the splanchnic blood flow occurs with decreased glomerular filtration rate and excess anti-diuretic hormone production. Thus the life-saving aspects of correcting hypoxia and hypercapnia are not achieved without cost.

The mandatory sedation and obtundation of all of the patient’s spontaneous respiratory effort needed for CMV to be effective has an additional disadvantage in that prolonged sedation ± paralysis results in atrophy of the respiratory muscles in a manner analogous to the atrophy of skeletal muscle seen on prolonged bed rest. This can result in prolonged weaning, *i.e.* delay in liberation from mechanical ventilation.
and return to full spontaneous respiration. Ventilatory modes have been developed to allow preservation of the patient's own respiratory efforts by detecting and synchronising machine breaths with patient inspiration as in synchronised intermittent mandatory ventilation (SIMV). If no inspiration is detected, then a mandatory breath is delivered at a pre-set interval to ensure a minimal level of ventilation. Alternatively, the patient's spontaneous breaths may be detected and supported to a pre-set level of positive pressure, a mode known as pressure support ventilation (PSV). PSV is essentially patient triggered, pressure limited ventilation and can only be used if the patient has a normal intrinsic respiratory rate. These techniques allow less sedation, preservation of respiratory muscle tone, greater cardiovascular stability and avoid the surges in airway pressure which would occur if a mandatory inspiratory breath was imposed as the patient was trying to breathe out. SIMV and PSV are the most commonly used conventional modes of ventilation in modern ICUs.

Ventilator induced lung injury (VILI)

Where trauma has produced significant pulmonary pathology, either directly by severe contusion or indirectly as a result of a cytokine mediated systemic inflammatory response resulting in the development of ARDS, then the lung is particularly at risk of a secondary injury as a result of injudiciously aggressive ventilatory strategies using conventional modes of ventilation. Attempts to achieve 'normal' arterial blood gases have often required the use of very high tidal volumes, high respiratory rates, and high peak inflation pressures. The ventilator is capable of delivering these pre-set variables regardless of the compliance or resistance of the lungs. If all the alveolar sub-units of the lungs had normal and equal compliance the tidal volume would be equally distributed. Diseased lung is non-homogeneous with some units having normal compliance and others having poor compliance with long time constants. Some alveolar sub-units are available to be ventilated and others are not. This dis-homogeneity of diseased lung is seen very clearly by pulmonary CT scanning. As a result of this, the more normal alveolar sub-units are preferentially ventilated and subjected to pressures which cause over-distension. It is not only high pressures, but high tidal volumes can also cause over-distension. Over-distension injury due to excessively high tidal volumes is known as volutrauma and over-distension injury due to excessive pressures is known as barotrauma. In extreme cases of barotrauma, gas from over-distended ruptured alveoli forms interstitial pulmonary emphysema and tracks along the adventitia of intra-pulmonary blood vessels. Eventually the gas bubbles coalesce as
they migrate centrally and eventually mediastinal emphysema occurs. If the process persists then gas bursts through the mediastinal pleura and pneumothorax occurs\textsuperscript{11}. Unrecognised pneumothorax is a serious complication during positive pressure ventilation as the volume of the pneumothorax enlarges with each subsequent tidal breath and tension pneumothorax may rapidly occur. All pneumothoraces must be drained prior to positive pressure ventilation or anaesthesia with nitrous oxide. Prophylactic chest drainage of blunt contusional lung injury is justified since once pneumothorax occurs, positive pressure ventilation may result in prolonged pulmonary leak or formation of bronchopleural fistula. Even without gross pneumothorax the repeated over-distension of alveoli by high tidal volumes can contribute to the deterioration of the underlying lung problem. The cyclical opening and closing of the alveoli results in a shearing injury characteristic of conventional high tidal volume, high inflation pressure ventilation in which injured, atelectatic alveoli are forced open on inspiration and allowed to collapse on expiration. Exudative pulmonary oedema can form as a result of increased alveolar permeability as a result of large tidal volumes producing shearing injury, compliance further decreases and the tidal volume is displaced to more normal alveoli so that the injury is propagated. It is this combination of volutrauma and barotrauma that together constitutes ventilator induced lung injury\textsuperscript{12}. The insult is probably increased by concurrent oxygen toxicity as a result of the use of high FiO\textsubscript{2} in an attempt to preserve adequate arterial oxygen tension\textsuperscript{13}.

**Lung protective ventilatory strategies**

The end-points of the pathophysiology of ventilator induced lung injury (VILI) and the pathophysiology of trauma induced lung injury, either direct or indirect, are identical and it would seem reasonable to prevent exacerbation of lung injury by the adoption of a lung protective ventilatory strategy. The mechanism of VILI would suggest that the avoidance of over-distension, shear stress injury and oxygen toxicity should be the main strategies of such a technique.

**Pressure and volume limitation strategy**

A reduction in volutrauma can theoretically be achieved by the use of low tidal volumes and low peak inspiratory pressures during ventilation. Classically relatively large tidal volumes (10–12 ml/kg) have been used to ensure normocapnia in the range of 4.5–5.5 kPa and, in the presence of the dishomogenous lung of the trauma patient with respiratory
compromise, this leads to over-distension, high peak inspiratory pressures and the production of VILI. Reducing the tidal volume to 6–8 ml/kg and limiting peak inspiratory pressures in the presence of poorly compliant lung results in decreased incidence of over-distension and a reduction in transalveolar pressure. Over-distension does not seem to occur if the transalveolar pressure is kept below 35 cm of water which equates to a plateau airway pressure of 35–45 cm of water. Plateau airway pressure being the pressure measured at the end of inspiration as distinct from the pressure just before the end of inspiration which is generally higher. The downside of pressure limitation (also called pressure controlled ventilation PCV) is that minute ventilation is decreased with subsequent hypercapnia. In addition, the low lung volumes generated mean that alveoli have an increased tendency to collapse which must be countered by a concurrent lung recruitment strategy. This concept of pressure controlled ventilation and 'permissive hypercapnia' has been used in clinical practice and appeared to show improved outcome in ARDS on retrospective analysis and the technique is used widely. Three recent randomised controlled trials of this volume-pressure limited strategy failed to show increased survival in mixed, i.e. trauma and non-trauma, ARDS patients. If the results of the three studies are pooled, then the 95% confidence interval around the pooled estimate of relative risk includes the possibility of important benefit (r 1.11; 95% CI 0.87–1.43). These studies were used with conventional levels of positive end expiratory pressure (PEEP) and the result may be a consequence of not combining pressure limitation with optimal PEEP or other methods of recruiting alveoli or preventing alveolar collapse at end expiration. There are no large prospective trials of the strategy applied specifically to trauma patients.

Lung recruitment strategy

Shear forces induce alveolar damage because of the cyclical opening and particularly subsequent closure. Tidal volume and positive end expiratory pressure (PEEP) interact such that collapse of alveoli at end expiration at low or zero PEEP produces the maximum degree of alveolar injury. The use of PEEP then tends to hold alveoli open, stop them collapsing totally at end expiration and limiting the shear forces applied. The level of PEEP applied to prevent collapse and recruit unopened alveoli is difficult to calculate on an individual basis except from each patient's pressure volume curves by choosing the lower inflection point on the ascending limb or the upper inflection point on the descending limb. Alternative strategies for recruiting closed or semi-closed alveolar subunits involve giving a single large tidal breath and holding end inspiration for 20 s.
Currently, the evidence that the use of such combined lung protective strategies results in a decreased mortality is conflicting\(^1\). The rationale for their use is logical, particularly in trauma patients with co-existing pulmonary contusion but, as yet, no prospective controlled trial data have been published.

**Alternative modes of ventilation**

Taking into account the mechanism of VILI, the theoretical advantages of avoiding over distension and shear injury while promoting lung recruitment, one can formulate a key message for ventilating injured lungs. The aim should be to set a ventilatory mode that will open under-ventilated alveolar units, keep them open for as long as possible to allow optimal gas exchange at volume and transalveolar pressure that will not induce secondary lung injury or produce haemodynamic instability, then allow exhalation to a lung volume and positive end expiratory pressure which prevents alveolar collapse and allows adequate carbon dioxide excretion. Various alternative modes of ventilation can help achieve this goal.

*Pressure controlled inverse ratio ventilation*

Part of this goal may be achieved by the use of pressure controlled ventilation (PCV), but the time for which the distending pressure is applied is important in attempts to achieve all of it. Consideration must be given to the frequency of breaths, the time allowed for inspiration and the time allowed for expiration – the inspiratory to expiratory ratio (I:E ratio). For an alveolar sub-unit of a given compliance, a high pressure for a short time period will produce volume expansion which may be equal to that produced by a lower pressure applied for a longer period of time. PCV may be used with a range of I:E ratios from the ‘normal’ 1:2 to equal ratio 1:1 or even inverse ratios of 2:1 or even 3:1. Pressure controlled inverse ratio ventilation (PCIRV) would seem to offer theoretical advantages in terms of lung protection and recruitment particularly when combined with PEEP in some patients with poor lung compliance and alveoli with long time constants\(^1\). The improvement in oxygenation seen may be due to reduced arteriovenous shunt, decreased ventilation-perfusion mismatch or increased functional residual capacity as a result of intrinsic PEEP developing due to the short expiratory phase. While the development of intrinsic PEEP has some advantages in terms of alveolar recruitment, too much, particularly in the presence of bronchospasm or other obstruction to expiration, may promote the
Strategies for ventilatory support

formation of volutrauma. Where evidence of increasing intrinsic PEEP is found then the expiratory time should increased.

Disadvantages of pressure controlled inverse ratio ventilation
The use of PCIRV is not tolerated by unsedated patients as the inverse I:E ratio is a very uncomfortable pattern of ventilation. High doses of sedative drugs and occasionally neuromuscular junction blocking drugs are required to facilitate the optimal pattern of ventilation and gas exchange. In chest trauma with co-existing blunt myocardial injury sedation to the required depth often requires inotropic support to maintain cardiac output. Hypovolaemic trauma patients may suffer a drop in cardiac output particularly at inverse ratios of 3:1 as a result of the effect of prolonged positive mean intrathoracic pressure on preload. Trauma patients with a head injury and raised intracranial pressure in combination with lung contusion form a not infrequent group of patients where a lung protective ventilatory strategy with permissive hypercapnia is at odds with the need to control PaCO₂ to low normocapnia for intracranial pressure control. This is a particular problem if the injured lungs are so poorly compliant, PEEP levels so high and the inspiratory ratio so short as to generate a very small tidal volume. In these circumstances, the series dead space (the volume of the ventilator tubing from the ‘Y’-piece and endotracheal tube plus anatomical dead space) may represent a large proportion of the tidal volume and result in a significant degree of rebreathing.

Pressure controlled inverse ratio ventilation and tracheal gas insufflation
If PCIRV and PEEP are used in a head injured patient as described above, the series dead space may be washed out by placing a catheter co-axially in the endotracheal tube to lie with its tip just above the level of the carina. Tracheal gas insufflation (TGI) with the fresh gas flow of the same FiO₂ with which the patient is being ventilated flushes out the dead space and reduces PaCO₂ by preventing rebreathing of the gas contained in the dead space with each cyclical ventilator breath. Various modes of TGI exist depending on whether the insufflated gas is applied continuously throughout inspiration and expiration, during inspiration only or during expiration. Confining wash-out to the expiratory phase has theoretical advantages but does require specialised equipment to synchronise catheter gas flow with the expiratory phase. Correct positioning of the catheter is vital, movement of the catheter may cause tracheal mucosal damage and the catheter gas flow must be humidified. For the expiratory washout modes flows of up to 14 l/min have been
used, the other methods need lower flows to prevent over-inflation and gas trapping. Although there have been no formal trials of the system, it may be useful in those trauma patients with head injury and pulmonary injury who need a lung protective mode of ventilation\textsuperscript{17}.

**High frequency ventilation**

High frequency jet ventilation (HFJV) and high frequency oscillation (HFO) are the two types of this mode that are commercially available. HFO uses sub-dead space tidal volumes generated by pistons or electromagnetic diaphragms to produce oscillatory gas flows of 150–3000 breaths/min. The concept of using very low tidal volumes delivered at very high respiratory rates is not new and after initial popularity in the late 1970s and early 1980s the technique fell into disuse after two prospective controlled trials failed to show any advantage over conventional ventilation in terms of morbidity and mortality. The low volumes and low mean airway pressures used are now attracting further investigation, but this time combined with alveolar recruitment regimes to prevent alveolar collapse. HFO combined with recruitment manoeuvres is the mode currently under investigation in both adults and children\textsuperscript{18}. No prospective controlled trials have been published in trauma patients but the concept would appeal in patients with established barotrauma or bronchopleural fistulae where very low mean airway pressures may be advantageous. The disadvantages of HFJV AND HFO are difficulty in humidification, monitoring of delivered pressures and volumes and the potential for a rapid build up airway pressure or air trapping if outflow obstruction occurs during the expiratory phase.

**Prone positioning**

Despite the use of high FiO\textsubscript{2}, PEEP and optimal PCIRV, some patients progressively become more difficult to oxygenate. The next strategy to improve ventilation is to turn the patient prone\textsuperscript{19}. The improved gas exchange was thought to occur as gravity redistributed blood from the dorsal regions of the lungs, where atelectasis had developed, to the anterior segments where alveoli were more recruitable. Recent work has suggested that the improvement is related to changes in regional pleural pressure. In the supine position, there is a gradient increasing negative pressure ventrally which promotes ventilation in ventral lung areas; if the patient is turned prone the pleural pressure becomes more uniform and reduces ventilation perfusion mismatch. Turning the trauma patient
Prone positioning in patients with ARDS and severe respiratory failure is difficult if there is biomechanical instability of the spine or if a large pelvic external fixator is applied, the technique is initially not popular with ICU nursing staff as it is, for the actual turning over, very labour intensive. Four people are needed and great care is required to ensure that the airway and vascular access lines are not dislodged during the turn. Once turned the head should be turned to one side and the shoulders supported on a pillow with the arms by the sides. A standard protocol for turning and nursing should be used and needs to include checking all tubes, lines, etc after turning, eye protection, pressure area care, positioning of arms and legs such that neuropraxia is avoided and the use of a 100 reverse Trendelenberg to reduce the incidence of facial oedema. In a recent series, 78% of patients had improved oxygenation after prone positioning and the improvement often persists after return to the supine position. It is not possible to predict who the responders will be, but it would seem reasonable to target those patients with high resolution pulmonary CT scans showing evidence of dorsal atelectasis and consolidation, particularly if there is decreased lung compliance. The data on the duration of ventilation in the prone position and on how long any benefit lasts are variable. Some units recommend keeping the patient prone overnight, some for only 4 h. Some recommend early use of the position and others only when there is evidence of dorsal atelectasis. Further prospective randomised trials are currently being undertaken.

**Inhaled nitric oxide**

Nitric oxide (NO) is an ultra-short acting pulmonary vasodilator which improves oxygenation by dilating pulmonary vessels adjacent to the best ventilated alveolar units. When given by inhalation, it is preferentially delivered to the more recruited alveoli of the dis-homogenous lung and diffuses rapidly out of the alveoli to the pulmonary capillaries causing relaxation of vascular smooth muscle and dilatation of the blood vessel. It is rapidly bound and inactivated by haemoglobin (in 110–130 ms) so its vasodilator effects are limited to the pulmonary circulation. Since the blood flow past under-ventilated alveoli is reduced as a result of the human hypoxic vasoconstrictor response, the improved blood flow in the NO dilated vessels adjacent to open alveoli produces a net reduction in intrapulmonary shunt and an increased PaO₂. As well as rescuing the critically hypoxic, it enables a reduction in FiO₂ and may help to prevent oxygen toxicity. The UK Consensus Guidelines for the use of inhaled nitric oxide (NO) therapy suggests that it should be used at 5–20 parts per million (ppm) only where oxygenation fails to improve despite optimisation of PCIRV, PEEP and prone positioning and that the dose
used should be the lowest to achieve a 20% improvement in $\text{PaO}_2: \text{FiO}_2$ ratio\(^2\). In high concentrations, > 100 ppm, it is highly reactive and toxic and the delivery system used must conform to *Hospital Technical Memorandum HTM 20/22*. During its use, the level of methaemoglobin in the blood and inspired nitrogen dioxide must be measured so this therapy is expensive and potentially dangerous. The largest phase 2 randomised placebo controlled trial of NO so far, in ARDS patients, demonstrates improved oxygenation but no difference in mortality\(^2\). It does improve oxygenation in the short-term in about 50% of patients but the effect is transient. Larger phase 3 trials and comparisons with other inhaled pulmonary vasodilators, such as nebulised prostacyclin\(^2\), are required.

**Extra-corporeal gas exchange**

Extra-corporeal gas exchange (ECGE) represents the final option if all other avenues of providing life-saving ventilatory support have failed. Partial cardiopulmonary venovenous bypass is initiated using heparin bonded vascular access lines, extra-corporeal oxygenation and carbon dioxide removal achieved using membrane oxygenators. A low volume, low pressure low frequency regimen of ventilation is continued to contribute to respiration. The basic concept is that by providing adequate oxygenation and carbon dioxide removal with minimal ventilation, then the lungs are at rest and that lung healing is more likely to occur. Its use in trauma patients is particularly difficult as any active bleeding is worsened if successful extra-corporeal circulation, oxygenation and CO\(_2\) removal requires anti-coagulation. Currently, in the UK, the availability of ECGE is limited to a few centres carrying out evaluative research and trials and is not recommended in trauma patients.

**Summary**

Trauma patients may suffer pulmonary dysfunction either as a result of direct injury or as a result of injury to other body systems which produce a systemic inflammatory response that promotes development of pulmonary complications, such as sepsis, acute lung injury or acute respiratory distress syndrome. The effective treatment of the respiratory system must occur in parallel with the effective treatment of the patient as a whole and ventilatory support strategies should not be seen in isolation from meticulous fluid balance, support of other failed organ systems, microbiological surveillance and nutrition. Early, safe and
Strategies for ventilatory support

efficient resuscitation may pre-empt or reduce the need for aggressive ventilatory support. Current data would suggest that a step-wise approach is required:

1 Assess the patient’s suitability for continuous positive airways pressure applied by mask; if ventilation is needed, then

2 Initiate ventilation with lung protective modes, specifically pressure controlled ventilation at normal I:E ratio with optimal positive end expiratory pressure and periodic alveolar recruitment techniques.

3 If failure of oxygenation occurs, convert to a pressure controlled inverse ratio mode with continued positive end expiratory pressure and recruitment.

4 Use specific adjuncts such as tracheal gas insufflation if permissive hypercapnia is unacceptable.

5 Use the prone position and assess any improvement of oxygenation.

6 If previous strategies fail, inhaled nitric oxide or nebulised prostacyclin may be used; it is unlikely to provide prolonged benefit, but may buy time for other aspects of the whole patient treatment regimen to work.

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