

Chapter 1

The patient with acute lung injury (ALI)

Julie Hamilton

Introduction

Acute respiratory distress syndrome (ARDS) is the severest form of acute lung injury (ALI) and presents one of the greatest challenges to health professionals within critical care. This scenario focuses on the knowledge and skills necessary to manage the complex needs of a patient with ARDS.

Patient scenario

Lee Kuan Yew, a 68-year-old gentleman who weighs 75 kg, was admitted to the intensive care unit (ICU) following intubation in the Accident and Emergency (A&E) department for acute respiratory failure. He had been unwell for four days with shortness of breath, pleuritic pain, fever and rigours and presented to the A&E with tachypnoea, followed by dyspnoea and progressive hypoxaemia and hypercarbia. Physical examination revealed focal findings of consolidation. His past medical history was unremarkable, but he smoked 30 cigarettes per day for the past 40 years. Three days following his admission to the ICU, Lee Kuan Yew remains sedated, intubated and mechanically ventilated. Assessment findings can be seen in Table 1.1.

Reader activities

Having read this scenario, consider the following:

- How do Lee Kuan Yew's symptoms suggest that he has an ALI?
- Consider the possible causes of Mr Kuan Yew's lung injury. Outline the factors that make him at risk for the development of ALI.
- What stage of ALI do you think Mr Kuan Yew is in? Explain this using relevant pathophysiology.
- Analyse the blood gas presented. Consider the possible causes of Mr Kuan Yew's altered results.

2 *Critical Care Nursing: Learning from Practice*

- Do you agree with the current ventilation strategy? How would you manage Mr Kuan Yew's respiratory function?
- What therapies other than conventional ventilation can be utilised in the management of ARDS?

Table 1.1 Assessment findings.

Ventilator settings	<ul style="list-style-type: none"> • Ventilated on synchronised intermittent mandatory ventilation–volume control (SIMV-VC) • 50% oxygen (FiO₂ 0.5) • PEEP 5 cmH₂O • Preset tidal volume (Vt) 600 mL • Respiratory rate set at 14 bpm (no spontaneous effort) • Peak airway pressure (PAP) 31 cmH₂O • Inspiratory:expiratory ratio 1:2
Clinical findings	<ul style="list-style-type: none"> • Bilateral air entry with coarse crackles throughout • Frothy pink sputum obtained on endotracheal suctioning • Bilateral infiltrates on chest X-ray
Arterial blood gases	<ul style="list-style-type: none"> • pH 7.34 • PaCO₂ 6.5 kPa • PaO₂ 10.8 kPa • HCO₃⁻ (standard) 20 mmol/L • Base excess -3 • PaO₂/FiO₂ 21.6 kPa • SpO₂ 92%
Haemodynamic data	<ul style="list-style-type: none"> • Heart rate (HR) 115 bpm (sinus) • Mean arterial blood pressure (MABP) 59 mmHg • Central venous pressure (CVP) 9 mmHg • Tympanic temperature 38.7°C • Feels peripherally warm to touch • 500 mL colloid administered over last 24 hours • Urine output 40 mL/h • Nasogastric feeding in progress at 80 mL/h
Laboratory results:	<ul style="list-style-type: none"> Sodium 137 mmol/l Potassium 4.5 mmol/L Lactate 2.8 mmol/L Glucose 8.2 mmol/L Haemoglobin (Hb) 10.5 g/L Raised white cell count (WCC) and C-reactive protein (CRP) level

Definitions of acute lung injury (ALI) and acute respiratory distress syndrome (ARDS)

Adult respiratory distress syndrome was first described by Ashbaugh et al. in 1967 as a clinical syndrome different from other types of acute respiratory failure, with clinical characteristics of tachypnoea, hypoxaemia resistant to supplemental oxygen, diffuse alveolar infiltrates and decreased pulmonary compliance (Ashbaugh et al. 1967).

Since its initial description in 1967, the criteria for defining ALI/ARDS have changed several times. In 1988 Murray and colleagues proposed a definition which described

Table 1.2 American–European Consensus Conference Definitions of ALI/ARDS.

	Onset	Chest X-ray	Left-ventricular pressure	PaO ₂ /FiO ₂
ALI	Acute onset	Bilateral infiltrates on chest X-ray	No clinical evidence of left-atrial hypertension or PAOP <18 mmHg	<40 kPa
ARDS	Acute onset	Bilateral infiltrates on chest X-ray	No clinical evidence of left-atrial hypertension or PAOP <18 mmHg	26.6 kPa

Source: From Bernard et al. (1994).

whether the syndrome was in an acute or chronic phase, the physiological severity of pulmonary injury and the disorder associated with the development of the lung injury (Murray et al. 1988). In 1994, recognising that the study of ALI and ARDS was still hindered by the lack of a simple, uniform definition, the North American–European Consensus Conference (NAECC) published further revised definitions (Bernard et al. 1994) (see Table 1.2).

From the NAECC definition it can be deduced that Mr Kuan Yew has ARDS. He has developed acute respiratory failure requiring ventilation; he is hypoxaemic with a PaO₂/FiO₂ ratio of 21.6 kPa and has bilateral infiltrates on chest x-ray (see Figure 1.1).

Mr Kuan Yew does not have a pulmonary artery catheter *in situ* to enable determination of the pulmonary artery occlusion pressure (PAOP); however, he has no history of cardiac disease and clinically shows no signs of left-atrial hypertension such as a raised central venous pressure (CVP), although the latter can be normal in left-atrial hypertension. Mr Kuan Yew is also demonstrating symptoms of severe sepsis (see Chapter 4 for further information on sepsis), a common co-existing condition.

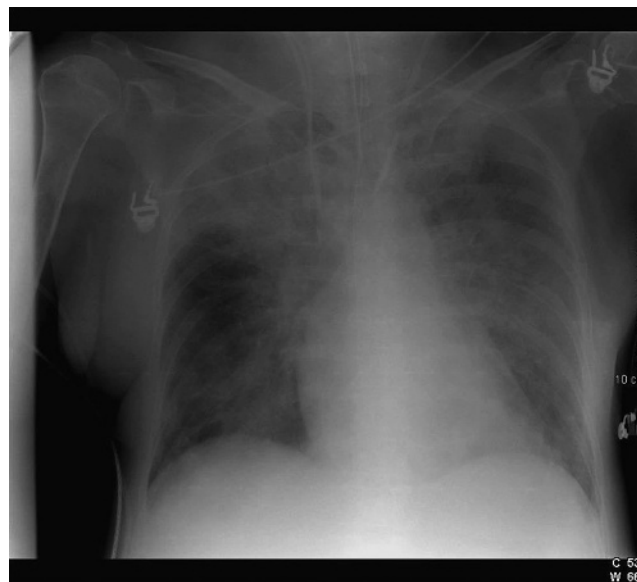


Figure 1.1 Chest X-ray. (X-ray courtesy of Dr Duncan Wyncoll, Consultant Intensivist, Guy's and St Thomas' NHS Foundation Trust, London.)

Pathophysiology of ALI/ARDS

Pathology

In 1972 the National Institute of Health estimated the incidence of ARDS at 60 cases per 100 000 population per year (National Heart and Lung Institute, National Institute of Health (NHL, NIH) 1972). Several robust studies since then have demonstrated a wide range of incidence rates of ARDS from 1.5 to 8.3 cases per 100 000 per year (Villar and Slutsky 1989; Garber et al. 1996). Although it could therefore be considered a rare disease, the mortality of ARDS is high, estimated to be between 34% and 65% (Estenssoro et al. 2002; Herridge et al. 2003). The incidence of ALI, however, appears more common with many patients within high dependency settings having a PaO₂/FiO₂ of <40 kPa. It is therefore essential that critical care nurses have an understanding of the pathophysiology and management of ALI and ARDS. The major cause of death in patients with ALI/ARDS is multiple organ failure and irreversible respiratory failure, with 84% of deaths occurring more than three days after the onset of ALI/ARDS caused by multi-system organ failure (Ware and Matthay 2000).

Acute lung injury is a term used to describe the response of the lungs to a broad range of insults with ARDS representing the most severe end of the spectrum. Its pathophysiology is driven by an aggressive inflammatory reaction which results in widespread changes throughout the lung. A broad variety of precipitating causes are recognised and these can be differentiated into those which cause injury to the lung directly and those which cause injury indirectly (see Table 1.3). A number of endogenous anti-inflammatory mechanisms are also initiated to counteract the effects of the aggressive pro-inflammatory response; however, these responses may be excessive and contribute to a state of immunoparesis (Doyle et al. 1995).

Epidemiological literature indicates that the major risk factor for the development of ALI and ARDS is severe sepsis; 18–40% of patients with sepsis will develop ALI/ARDS, followed by pneumonia, aspiration of gastric contents, multiple blood transfusions, multiple trauma and pregnancy-related ALI/ARDS (Villar and Slutsky 1989; Ware and Matthay 2000).

From Mr Kuan Yew’s clinical history and initial presentation it appears that he may have developed an acute lung injury and subsequent ARDS from a direct cause such as lobar pneumonia. It is also important, however, to note that as Mr Kuan Yew is mechanically

Table 1.3 Risk factors for ARDS.

Direct causes	Indirect causes
<ul style="list-style-type: none"> ● Aspiration of gastric contents ● Pneumonia ● Near drowning ● Lung trauma, e.g. blast injury, lung contusions ● Inhalation injury ● Fat emboli 	<ul style="list-style-type: none"> ● Sepsis ● Massive blood transfusion ● Disseminated intravascular coagulation ● Pancreatitis ● Cardiopulmonary bypass ● Pregnancy-related ARDS

ventilated and is critically ill, he is at a significant risk of developing a nosocomial infection and secondary sepsis (Vincent et al. 1995), a major risk factor for the development of ARDS, and at present he is indeed demonstrating signs of severe sepsis.

ALI and ARDS cause diffuse alveolar damage affecting all parts of the alveolus, including the epithelium, the endothelium and the interstitial space. It is a progressive condition with the pathological changes typically described as passing through three overlapping phases – an inflammatory or exudative phase, a proliferative phase and a fibrotic phase (Ware and Matthay 2000).

Exudative phase

Lasting for up to seven days following the onset of symptoms, the exudative or acute phase of ALI/ARDS is characterised by the influx of protein-rich oedema fluid into the alveolar air spaces, as a result of increased permeability of the alveolar–capillary membrane and the formation of hyaline membranes. The hyaline membranes contain necrotic epithelial cells, plasma proteins which have been deposited in the alveolar space as part of the inflammatory exudate that leaks across the alveolar–capillary membrane, immunoglobulin and complement. The alveolar–capillary barrier has focal areas of damage and the alveolar wall is oedematous. Neutrophils are increasingly found within the capillaries, interstitium and eventually airspaces. As the process of damage progresses, there is extensive necrosis of type 1 alveolar epithelial cells and further hyaline membrane formation (Figures 1.2a and 1.2b).

These pathological changes can be seen in Mr Kuan Yew's clinical picture by the presence of pulmonary oedema and his deterioration in lung function. Flooding of the alveoli with protein-rich fluid and debris has caused a decrease in lung compliance, reflected in the high airway pressures. It has also caused a significant reduction in the diffusion of oxygen, leading to a reduced arterial oxygen saturation and PaO₂. Fluid-filled

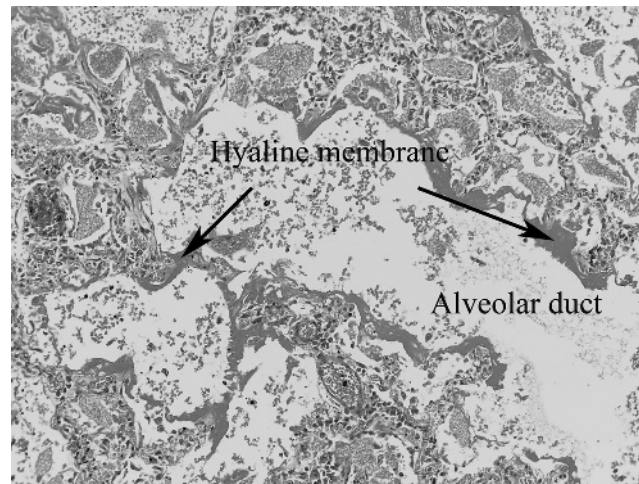


Figure 1.2 (a) Histopathology slide of lung tissue. (*Continued*)

6 Critical Care Nursing: Learning from Practice

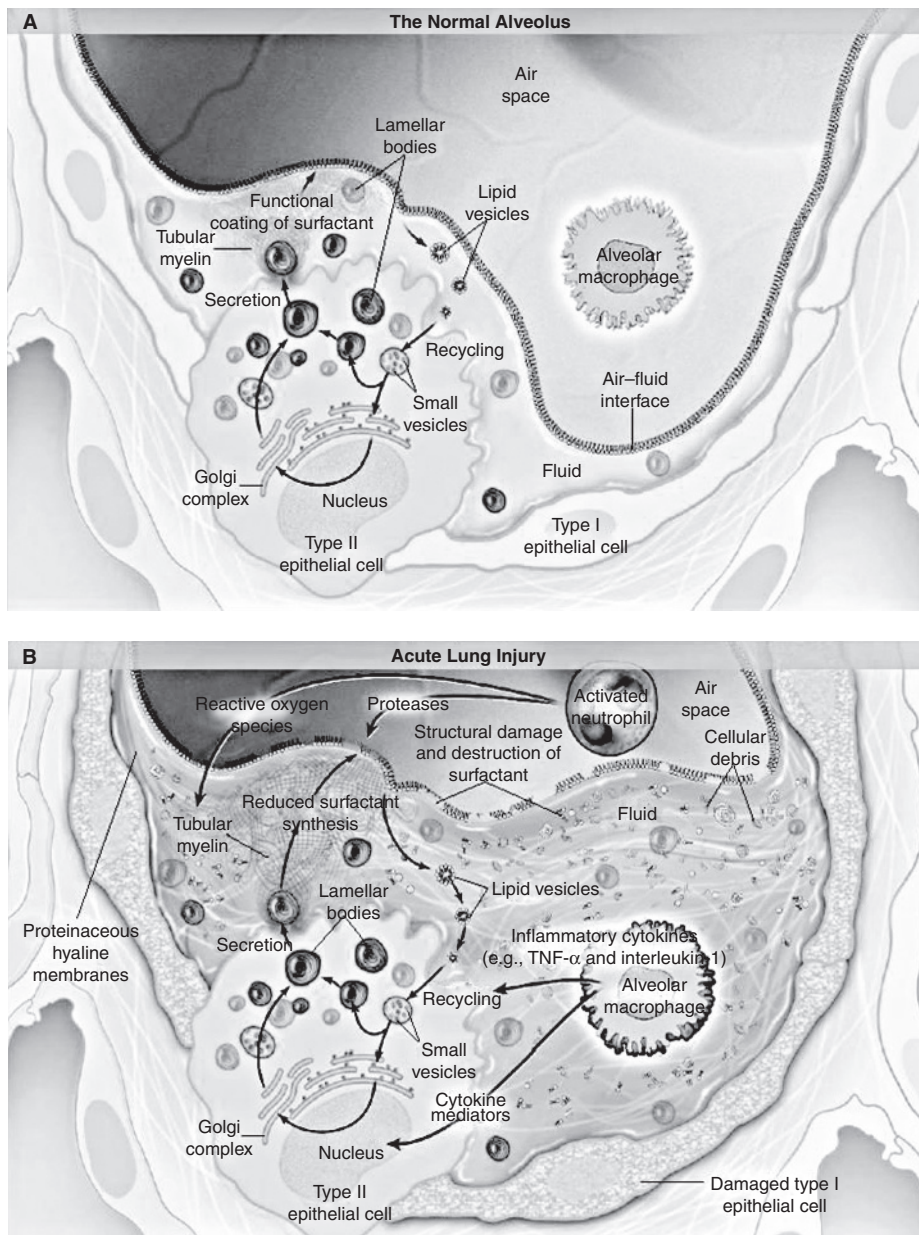


Figure 1.2 (Continued) (b) Diagrammatic illustration of cellular changes in ARDS. (Baudouin 2004). Used with permission of Massachusetts Medical Society.

and collapsed alveoli result in the development of a right to left intra-pulmonary shunt. The negative effects of this on Mr Kuan Yew's gas exchange are further compounded by loss of the normal compensatory hypoxic pulmonary constriction.

Proliferative phase

The proliferative phase is characterised by organisation of the hyaline membranes by proliferating fibroblasts, cell debris and inflammatory cells (Ware and Matthay 2000). Necrosis of type 1 alveolar cells exposes areas of the epithelial basement membrane and the lumens of the alveoli fill with leucocytes, red blood cells and fibrin. Type 2 alveolar cells, which are responsible for the production of surfactant, are also damaged but some proliferate along the alveolar wall in an attempt to cover damaged areas of the epithelium and differentiate into type 1 cells. Pulmonary oedema is less prominent at this stage; however, alveolar collapse becomes more marked and the alveolar ducts become narrowed and distorted. This then leads to a further increase in the degree of intrapulmonary shunt, leading to a further deterioration in gas exchange, and hypoxaemia resistant to oxygen therapy.

At this stage the process can be reversed and the lung parenchyma may return to normal. However, in some cases the damage is severe and the hyaline membranes become incorporated into the walls of the revised alveoli (Ware and Matthay 2000).

Fibrotic phase

The fibrotic phase can begin as early as ten days following the insult and is characterised by progressive thickening of the vasculature walls and an increase in the amount of lung collagen (Ware and Matthay 2000). Fibrosis results in a further reduction in lung compliance, increasing the work of breathing, decreasing the tidal volume and resulting in the retention of CO₂. As a result of the destruction of some alveoli and interstitial thickening, gas exchange is reduced and this contributes to further hypoxaemia and ventilator dependence.

Pathogenesis of ALI/ARDS

Inflammation

As a result of the initiation of an inflammatory response, there is increased leucocyte production and mobilisation to the inflamed site. Mediator cascades including the production of cytokines, chemokines, free radicals and complement and coagulation pathway components are also activated. There is also an anti-inflammatory response.

The neutrophil is the dominant leucocyte involved in the pro-inflammatory response. Neutrophils cause cell damage by the production of free radicals, pro-inflammatory mediators and proteases, and excessive quantities of these products, including cytokines, have been found in patients with ARDS (Chollet et al. 1996). The inflammatory response

8 *Critical Care Nursing: Learning from Practice*

is in part driven by cytokines. Two of the major pro-inflammatory cytokines are tumour necrosis factor- α (TNF- α) and interleukin-1 (IL-1). The actions of these include (1) recruitment and localisation of macrophages to the lung parenchyma, (2) stimulation of other inflammatory cytokines such as IL-6 and IL-8 and (3) adherence of neutrophils to endothelium. Cytokines and other pro-inflammatory mediators such as endotoxin and thrombin have also been implicated in the increased vascular permeability that contributes to pulmonary oedema in ALI/ARDS (Ware and Matthay 2000).

This inflammatory response leads to surfactant dysfunction in ALI/ARDS (Baudouin 1997), with destruction and loss of type 2 cells resulting in decreased synthesis and recirculation of surfactants. Additionally, leakage of protein-rich fluid into the alveoli during the development of ALI/ARDS, as seen in Mr Kuan Yew's clinical picture, contaminates the surfactant, resulting in a further reduction in its ability to function. The degree to which lack of surfactant contributes to the pathogenesis of ALI/ARDS, however, remains unclear.

Fibroproliferative response and resolution of ARDS

The fibroproliferative response is part of a normal repair process; however, if not closely regulated, it can have serious consequences such as lung fibrosis. Mediators such as TNF- α and products of the coagulation cascade such as thrombin, fibrin and factor Xa fuel the fibrotic response and stimulate local fibroblasts to migrate, replicate and produce excessive amounts of connective tissue.

In some patients pulmonary fibrosis does not completely resolve and can lead to problems with weaning from mechanical ventilation. There does not appear to be a uniform response to injury in that some patients develop ALI, some develop ARDS and some do not develop pulmonary symptoms at all. The reason for this may lie in genetics and recent evidence suggests that there is a genetic susceptibility both to sepsis and ARDS (Wax and Angus 2000).

Holistic assessment and detailed management of all issues related to total patient care are fundamental in caring for patients such as Mr Kuan Yew who have ARDS. In early ARDS, however, difficulties with oxygenation can be the major physiological challenge requiring careful assessment, titration of therapy and meticulous monitoring.

Tests and investigations

Continuous pulse oximetry

Continuous pulse oximetry has become a vital part of monitoring the critically ill patient as it is readily available, is non-invasive and can be used in many different settings. Pulse oximeters shine red and infrared light through a finger or ear lobe using a probe. The proportion of light absorbed allows the amount of oxygenated and deoxygenated haemoglobin to be estimated. The pulsatile component of absorption corresponds to arterial blood, and therefore, arterial oxygen saturation can be deduced. When arterial oxygen saturations are greater than 80%, current pulse oximeters can detect arterial

oxygen saturations to within a few percentage points and their use would, therefore, be beneficial in the assessment of Mr Kuan Yew's oxygenation. However, they are less accurate when arterial oxygen saturations are lower. It is also important to be aware that inaccurate values may be obtained in patients with shock, due to poor peripheral perfusion, by carboxyhaemoglobin, by low levels of haemoglobin and by the use of some dyes such as methylene blue which absorb wavelengths of light used by some pulse oximeters. After ruling out the possibility of any of these contraindications in Mr Kuan Yew's case, pulse oximetry will be a useful tool to rapidly detect periods of arterial hypoxaemia.

Arterial blood gas analysis

Arterial blood gas analysis is considered the gold standard in the assessment and management of ARDS patients such as Mr Kuan Yew. To ensure accuracy, however, it is important that the health professional obtaining the sample is aware of several key points. The sample must be taken and processed as quickly as possible to eliminate aerobic contamination; the current FiO_2 and temperature of the patient should be recorded at the time of sampling, the latter to allow temperature correction, and the sample rapidly analysed in a calibrated blood gas machine. If the arterial blood gas results are to reflect current ventilatory support, the sample should not be obtained until 15–20 minutes following any manipulation of ventilator settings. PaO_2 , PaCO_2 and pH are measured during arterial blood gas analysis. Oxygen saturation may be measured by a co-oximeter built into a blood gas machine or estimated from the PaO_2 based on the oxygen-dissociation curve corrected for temperature, PaCO_2 and pH. This estimate is considered reasonably accurate for oxygen saturations greater than 80% but is significantly erroneous at lower saturations.

$\text{PaO}_2/\text{FiO}_2$ measurement

The sole use of PaO_2 in assessing Mr Kuan Yew has limitations. As a result calculation of the ratio of PaO_2 to FiO_2 is now commonly used as an additional measurement (see Chapter 2 on weaning for how to calculate the $\text{PaO}_2/\text{FiO}_2$ ratio). The usefulness of the $\text{PaO}_2/\text{FiO}_2$ ratio is clearly demonstrated in Mr Kuan Yew's arterial blood gas results. At first glance a PaO_2 of 10.8 kPa could appear to be an acceptable level; however, when the FiO_2 is taken into consideration, it is clear from the NAEECC definition that he has ARDS as his $\text{PaO}_2/\text{FiO}_2$ ratio is 21.6 kPa.

Evidence-based management of a patient with ALI/ARDS

Airway and breathing

In patients like Mr Kuan Yew who have severe ARDS, the hallmark respiratory abnormality is hypoxaemia which gradually becomes more resistant to supplemental oxygen therapy as the condition progresses. Maintaining adequate arterial oxygenation is therefore a goal given a high priority and usually requires assisted/mechanical ventilation.

10 *Critical Care Nursing: Learning from Practice*

Assisted ventilation is generally carried out invasively via an endotracheal tube. However, a small subset of patients may be candidates for non-invasive ventilation (Hilbert et al. 2001). Non-invasive positive pressure ventilation (NIPPV) is finding increasing application in the management of acute respiratory failure in the high-dependency setting and it may be postulated that it would be successful in carefully chosen patients with ALI. It may aid in the recruitment of collapsed and fluid-filled alveoli, thereby reducing intrapulmonary shunt, and could also facilitate unloading of the respiratory muscles, reducing the work of breathing. It is important to highlight, however, that patients with ALI and ARDS are also frequently haemodynamically unstable, have severe hypoxaemia or have a rapidly progressive course of disease. Therefore, although NIPPV has been shown to be beneficial in some patients, there is little published experience or evidence of its benefits in patients with ARDS. It may therefore not be a good first choice for Mr Kuan Yew.

Approaches to mechanical ventilation

The pathophysiology of ARDS has been presented earlier in this chapter; however, it is important to highlight some important features which are relevant when discussing Mr Kuan Yew's ventilatory management. Computerised tomographic scanning (CT) has demonstrated that consolidation of lung tissue in ARDS is not uniform but rather is concentrated in dependent lung regions, leaving non-dependent areas relatively aerated. This distribution of aerated lung, described as 'baby lung' (Gattinoni et al. 1987), has important implications for mechanical ventilation strategies.

Traditional methods of mechanically ventilating patients with ALI and ARDS gave priority to the maintenance of oxygenation, while minimising the use of high concentrations of oxygen, and providing sufficient ventilation to maintain arterial pH and PaCO₂ within normal limits. These goals were achieved by the administration of increased levels of positive end expiratory pressure (PEEP) to enable a decrease in the FiO₂, and the use of relatively large tidal volumes of 10–15 mL/kg. This approach, however, results in high inspiratory pressures in patients who already have decreased lung compliance. The application of tidal volumes of 10–15 mL/kg can also lead to over-inflation of the normal 'baby lung' which has been shown to cause local damage and further inflammation (Dreyfuss and Sauman 1998). Present understanding of ventilator-induced lung injury suggests that a traditional mechanical ventilation strategy such as this, using high tidal volumes and is likely to enhance Mr Kuan Yew's lung injury. Lung injury is caused by excessive volumes rather than high airway pressure (Dreyfuss et al. 1988) and even healthy animals ventilated with high tidal volumes for several hours develop pulmonary oedema that is histologically identical to that seen in ARDS. Furthermore, in animal models with ALI, large lung volumes have been shown to cause increased oedema accumulation and cytokine production (Tremblay et al. 1997). Although evidence in humans is lacking, it is likely that ventilating with high tidal volumes results in similar effects.

Four randomised controlled trials of 'lung-protective' ventilation, directed at preventing over-distension of the lung in ARDS, have been published over the past ten years (Brochard et al. 1998; Stewart et al. 1998; Brower et al. 1999; Acute Respiratory Distress Syndrome Network (ARDSNet) 2000). Of these the ARDSNet (2000) study is the largest and the

Table 1.4 Summary of ARDSNet (2000) low tidal volume strategy.

Variable	Settings
Ventilator mode	Volume assist control
Set tidal volume (mL/kg)	Aim for 6 mL/kg (if baseline tidal volume > 8 mL/kg, then set initial tidal volume at 8 mL/kg and reduce by 1 mL/kg every 2 h until 6 mL/kg)
Rate (breaths/min)	Set to approximate baseline rate of 6–35 breaths/min but not >35 breaths/min
Pressure (cmH ₂ O)	Aim for Pplat <30 cmH ₂ O or peak pressure <35 cmH ₂ O
Inspiratory flow rate (L/min)	Above patient demand (>80 L/min)
Inspiratory:expiratory ratio	1:1–1.3
PaO ₂ (kPa)	7.3–10.7
SpO ₂ (%)	88–95
PEEP and FiO ₂	Incremental FiO ₂ /PEEP combinations have been suggested with PEEP range from 5 to 24 cmH ₂ O (see Table 1.3)
pH	7.30–7.45

Source: Adapted from the NIH NHLBI ARDSNet low tidal volume ventilation strategy (ARDSNet 2000).

only one to date to demonstrate a mortality benefit of a lung-protective strategy in ARDS patients. Eight hundred and sixty one patients were randomised into two groups. One group received a tidal volume of 6 mL/kg if the plateau pressure (Pplat) did not exceed 30 cmH₂O and 4–5 mL/kg if the Pplat exceeded 30 cmH₂O and the other group received tidal volumes of 10–12 mL/kg if the Pplat did not exceed 50 cmH₂O and tidal volumes as low as 4 mL/kg if the Pplat exceeded 50 cmH₂O. A 9% mortality difference was observed in those patients who received the lower tidal volume ventilation strategy. Although the design of the ARDSNet trial has been heavily criticised, the ARDSNet lower tidal volume strategy has become accepted as the standard on which to base the ventilatory management of patients with acute lung injury (see Table 1.4 for protective lung ventilation protocol from the ARDSNet study) and this is how Mr Kuan Yew’s ventilation should be managed.

Volume control versus pressure controlled ventilation

Traditionally, invasive mechanical ventilation has been provided by volume controlled modes, as in the case of Mr Kuan Yew, whereby a preset tidal volume is delivered at a preset rate and inspiratory flow. Volume control, has the benefit of maintaining a constant tidal volume and hence minute ventilation and PaCO₂ under changing respiratory system conditions and easy detection of changes in lung mechanics. Over the past decade, however, in light of research demonstrating the non-homogenous distribution of consolidation in ARDS and the focus on limiting alveolar distension, there has been a trend towards the use of pressure controlled modes of ventilation.

With pressure control, a decelerating inspiratory flow is applied to a preset pressure limit, allowing the critical care team to select both inspiratory and expiratory pressures with the advantage of limiting pressure to a set level. The critical care nurse has to be

12 *Critical Care Nursing: Learning from Practice*

particularly vigilant when caring for a patient on pressure control ventilation as changes in lung compliance are not as easily detected. Close observation of the tidal volume and PaCO₂ is essential to detect changes in lung mechanics.

As a result of technological advances in mechanical ventilators, the distinction between volume- and pressure-controlled modes of ventilation has become slightly blurred. Parameters can now be adjusted within each of the different modes, such as pressure limitation within a volume-controlled mode of ventilation. The critical care teams are therefore faced with a number of different modes from which to choose. Several studies have attempted to compare the benefits of various modes; however, the majority of them have been too small to enable detection of an outcome benefit of either. In the ARDSNet (2000) study, a mortality benefit was detected between two groups of patients receiving volume-controlled ventilation which may suggest that it is more important to concentrate on the actual settings rather than the particular mode of ventilation.

Regardless of the mode of ventilation chosen, it is clear from the ARDSNet (2000) trial that we should aim for a tidal volume of 6 mL/kg, limiting the peak pressure to 35 cmH₂O or plateau pressure <30 cmH₂O if receiving volume-controlled ventilation (see Table 1.4).

Mr Kuan Yew is currently being ventilated on a volume-controlled mode which is acceptable when considering recent evidence. However, he is receiving greater than 6 mL/kg of tidal volume. In order to prevent further deterioration in Mr Kuan Yew's lung function and ventilator-induced lung injury, it would therefore be advisable to gradually decrease his preset tidal volume to closer to that suggested by the ARDSNet trial. When considering the most appropriate tidal volume, it is important to highlight that the ARDSNet (2000) study used predicted body weight which is based on the patient's sex and height rather than actual body weight.

Permissive hypercapnia

With traditional methods of mechanically ventilating patients with ALI/ARDS, attempts were made to maintain a normal PaCO₂ and acid–base balance. Reducing Mr Kuan Yew's tidal volume to 6 mL/kg, as advocated in the ARDSNet (2000) study, may result in an increase in his PaCO₂ and a corresponding decrease in pH, leading to a respiratory acidosis. Over the past ten years, increasing evidence suggests that allowing the arterial PaCO₂ to increase above 6 kPa, termed permissive hypercapnia, is safe when used in conjunction with a low-tidal volume, low-pressure ventilation strategy, as long as the pH remains >7.3. Although acidaemia has many physiological effects such as depression of myocardial contractility, systemic vasodilation, increased intracranial pressure and cellular metabolic dysfunction, these have not been demonstrated to be clinically significant. However, permissive hypercapnia is unlikely to be appropriate in patients who have a raised intracranial pressure. The question for the critical care nurse and critical team is therefore which puts the patient at more risk: a high PaCO₂ or alveolar distension? Current evidence would suggest that it is the latter. It is important, however, that the critical care nurse remains vigilant in monitoring the PaCO₂ and pH via arterial blood gas analysis, and responds to the results in a timely and appropriate manner.

Use of positive end expiratory pressure (PEEP)

PEEP has been shown to improve oxygenation in several ways, encouraging movement of fluid from the alveoli into the interstitial spaces, recruitment of small airways and collapsed alveoli and increasing functional residual capacity (FRC). Its application is now advocated during all modes of mechanical ventilation. Its use is particularly imperative for Mr Kuan Yew, not only as an adjunct to improve oxygenation, but also to prevent further ventilator-induced lung injury. It has been suggested that lung damage can be induced at low lung volumes as well as high lung volumes as a consequence of the production of shearing forces which can occur with the opening and closing of alveoli at low lung volumes during mechanical ventilation. The application of PEEP should reduce the volume of reopening–collapsing tissue and hence reduce the degree of damage (Gattinoni et al. 1995).

Mr Kuan Yew is currently receiving a PEEP of 5 cmH₂O. Controversy exists over what level to set the PEEP in patients with ALI/ARDS and indeed in respiratory failure in general. One method which has been used is to assess the pressure–volume relationship of the lungs (see Figure 1.3).

In theory, setting the PEEP above the lower inflection point (LIP) may prevent derecruitment and hence low-lung volume ventilator-associated injury; however, although the pressure–volume curve could give a physiological illustration of the mechanics of Mr Kuan Yew’s lung, applying this technique in the clinical setting is difficult. Modern ventilators commonly display the pressure–volume relationship of the respiratory system but this is obtained under dynamic conditions where elasticity and resistance of the respiratory system as a whole are considered. In acute respiratory failure, the impairment of the respiratory mechanics involves mainly the elastic component of the respiratory system. As a consequence, the measurement of respiratory pressure–volume curves should be done under static or semi-static conditions in order to eliminate the resistive component.

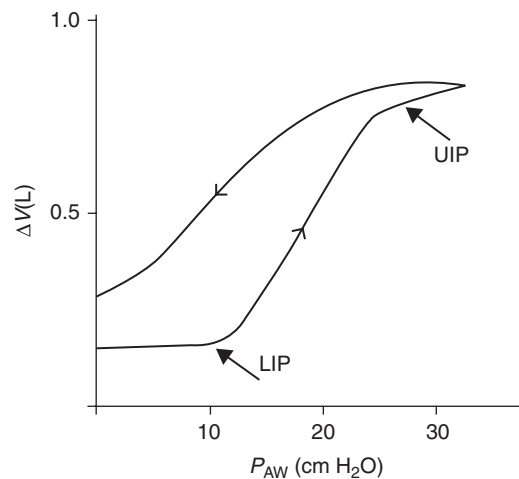


Figure 1.3 Pressure–volume relationship. (Adapted from Russell J and Walley K (eds) (1999) *Acute Respiratory Distress Syndrome: A Comprehensive Clinical Approach*. Cambridge University Press, Cambridge.)

Table 1.5 Titration of PEEP and FiO₂ in patients with ARDS.

FiO₂	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12
FiO₂	0.7	0.8	0.9	0.9	0.9	1.0	1.0	1.0
PEEP	14	14	14	16	18	20	22	24

Source: Adapted from the NIH NHLBI ARDSnet low tidal volume ventilation strategy (ARDSNet 2000)

Several research studies have attempted to quantify the appropriate level of PEEP for patients with ALI/ARDS. A small randomised controlled trial conducted by Amato and colleagues in 1998 suggested that high levels of PEEP should be adopted. Although this study demonstrated that the group of patients receiving higher than normal levels of PEEP had significantly lower mortality, it was difficult to conclude that the benefit was due to the high PEEP as there were many variables. The ALVEOLI study (ARDSNet 2004) conducted more recently found no difference in mortality between patients given higher levels of PEEP and those receiving traditional levels.

In light of the lack of conclusive evidence, a common approach is to choose the minimum level of PEEP likely to limit derecruitment, such as 10–15 cmH₂O. The ARDSNet trial protocol (ARDSNet 2000) also provides a useful guide to setting levels of PEEP in relation to FiO₂ (see Table 1.5) and this can be used by experienced critical care nurses when titrating FiO₂ and PEEP levels to oxygenation levels.

Prevention of ventilator associated pneumonia

It is important for the critical care nurse to appreciate that as an intubated patient, Mr Kuan Yew is at a high risk of developing nosocomial pneumonia. Preventing hospital-acquired infection is always important; however, the development of pneumonia associated with the use of mechanical ventilation is of particular concern. Ventilator-associated pneumonia (VAP) is an infection of the airways that develops more than 48 hours following intubation. It is the leading cause of death amongst hospital-acquired infections, exceeding the rate of death due to central line infections, severe sepsis and respiratory tract infections in the non-intubated patient. Hospital mortality of mechanically ventilated patients who do not develop VAP is 32% compared to 46% for ventilated patients who do develop VAP (Ibrahim et al. 2001). In addition, VAP increases the length of time patients spend on the ventilator, stay in the ICU and stay in hospital following discharge from the ICU (Rello et al. 2002), resulting in an estimated additional cost of approximately £20 000 to a hospital admission.

Prevention of VAP is therefore an essential component of caring for Mr Kuan Yew and he should be nursed according to the ventilator care bundle (Tablan et al. 2004). The ventilator care bundle is a series of evidence-based interventions which when implemented together aim to improve the outcome of ventilated patients. At the time of writing, a care bundle focusing specifically on preventing VAP was also undergoing consultation (DH 2010). The key components of both of these bundles are:

- Elevation of the head of the bed
- Daily ‘sedation hold’
- Peptic ulcer prophylaxis

- Deep vein thrombosis prophylaxis
- oral hygiene with 2% chlorhexidine
- subglottic aspiration
- ventilator tube management
- tracheal tube cuff pressure monitoring.

A degree of debate exists between the literature and clinical practice in relation to the angle to which the head of the bed should be elevated. A meta-analysis by Hess (2005) concluded that the semi-recumbent position was the most effective position for preventing VAP, semi-recumbent being defined as elevation of the head of the bed to 45 degrees. A study by Grap et al. (2005) published at the same time as Hess (2005) suggested that elevation of the head of the bed at >30 degrees did not result in a statistically significant increase in the incidence of VAP. More recently, a prospective multicentre trial tested elevations of 45 and 10 degrees. The authors concluded that elevation to 45 degrees was not feasible, finding that in reality the mean elevation for ventilated patients was in fact closer to 30 degrees (Van Nieuwenhoven et al. 2006). On this basis elevation of the head of the bed to at least 30 degrees is the current recommendation (DH 2007).

Mr Kuan Yew's condition, as presented in the initial assessment, could be managed effectively by the aforementioned ventilatory strategies. If his condition were to deteriorate, however, with worsening oxygenation and ventilation, several adjuncts to conventional ventilation strategies have been described in the literature, which could be considered for him. These are discussed below.

Inverse ratio ventilation

Inverse ratio ventilation (IRV), which can be employed with either pressure- or volume-controlled modes of ventilation, involves prolongation of the inspiratory time, resulting in inspiratory/expiratory (IE) ratios of 1:1 or 2:1. Most studies using IRV demonstrate an improvement in the PaO₂ (Tharratt et al. 1988) with proposed mechanisms of improvement being related to increased alveolar recruitment, an increase in mean airway pressure and a more even distribution of mechanical ventilation. Unfortunately, not all patients respond positively and the use of IRV also has important implications physiologically. The critical care nurse is required to monitor the patient closely as lengthening the inspiratory time may result in a significant increase in the amount of gas that is trapped at the end of expiration, resulting in hyperinflation and an increase in the amount of intrinsic PEEP generated. These effects can then lead to a reduction in cardiac output in a patient who may already be compromised haemodynamically. With a lack of a clearly defined role IRV is usually adopted in those patients in whom hypoxaemia is refractory to more conventional approaches.

Recruitment manoeuvres

The use of recruitment manoeuvres evolved from traditional 'sighs' which are breaths two or three times greater than normal resting tidal volumes. Sighs occur approximately three to four times per hour in normal healthy subjects and increase the function of surfactants in stabilising the alveoli and preventing their collapse. The justification for use of recruitment manoeuvres in Mr Kuan Yew would be to recruit partially collapsed and

16 *Critical Care Nursing: Learning from Practice*

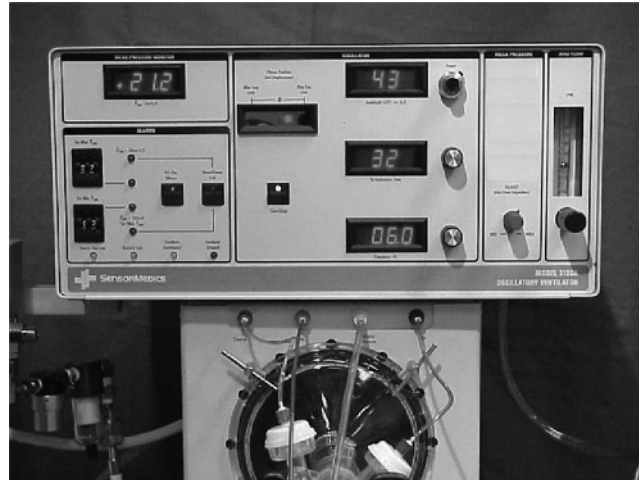


Figure 1.4 High-frequency oscillatory ventilator.

fluid-filled alveoli. These re-inflated alveoli could then be kept open by the application of PEEP, with the aim of improving oxygenation. Different methods of undertaking a recruitment manoeuvre have been described in the literature. Evidence that recruitment manoeuvres alone improve mortality, length of stay in ICU or ventilator-free days is, however, lacking. Complications of the manoeuvres have also been noted with transient hypotension and decreased SpO₂ being described. With this in mind the routine use of recruitment manoeuvres is not advocated within the literature and, if carried out, should be done so by an experienced member of the critical care team.

High-frequency oscillatory ventilation (HFOV)

Over the past few years there has been renewed interest in the use of HFOV (Figure 1.4) involving the administration of very small tidal volumes of approximately 80 mL at frequencies approaching 300 breaths per minute. The low tidal volume is generated by the movement of an oscillator within a ventilator circuit, similar to that used with continuous positive airway pressure (CPAP), and can be altered by adjusting the frequency of breaths, the IE ratio and the amplitude of the oscillator.

The repetitive movement of the oscillator results in an active expiratory phase which differs from conventional ventilators where expiration is passive and dependent on the elastic recoil properties of the lungs and chest wall. An active expiratory phase has been shown to aid in the clearance of CO₂, a common problem in patients such as Mr Kuan Yew who are ventilated using a low-tidal volume ventilation strategy. It is important that the critical care nurse is aware that altering the frequency of breaths in HFOV has the opposite effect on PaCO₂ when compared with conventional ventilation; that is, decreasing the frequency in HFOV can result in a decrease in PaCO₂ due to a slight increase in the tidal volume. Oxygenation is controlled simply by altering the mean airway pressure or the FiO₂.

On commencement of HFOV, the mean airway pressure is gradually increased to encourage lung recruitment. It is important during this stage that the critical care nurse closely monitors the SpO₂ as if the lung becomes over-distended, oxygen saturations can deteriorate. It is also imperative that the patient's haemodynamic status is closely monitored as increasing the mean airway pressure can result in an increase in intrathoracic pressure and decrease in venous return, with a subsequent decrease in cardiac output. Once it is felt that optimal recruitment has occurred, the mean airway pressure is then gradually decreased to an appropriate level.

HFOV has been used extensively and very successfully in neonates, but unfortunately, its application in the ventilation of adults with ALI/ARDS remains unclear. A study of the use of HFOV in adults, the MOAT trial, published in 2002 demonstrated a trend towards decreased mortality when compared to conventional ventilation (Derdak et al. 2002). The patients receiving conventional ventilation, however, were not ventilated using the ARDSNet (2000) guidelines and there were no significant differences in mortality at 30 days or 6 months. Furthermore, the mean airway pressure was significantly higher in the conventional ventilation group which raises the possibility of an increased risk of ventilator-induced lung injury in the control group. In order to obtain a definitive answer as to the application of HFOV in clinical practice, further research is clearly necessary to compare HFOV to the ARDSNet (2000) protocol and the recently commenced High Frequency Oscillation in ARDS (OSCAR) trial will hopefully provide the answer. Details of this trial, comparing conventional positive pressure ventilation with high HFOV for adults with ARDS can be found on the OSCAR website: <http://duncanyoung.net/index.php>.

Kinetic therapy

Kinetic therapy involves the use of specialised beds with low-air loss technology which can turn the critically ill patient in an arc of between 40 and 90 degrees (Figure 1.5). Rotation redistributes localised dependent oedema, helping to mobilise pulmonary secretions, therefore reducing atelectasis and respiratory tract infections associated with ALI/ARDS. Research studying the impact of kinetic therapy in patients with ALI/ARDS, however, is sparse.

A study by Pape et al. (1994) looked at the effects of kinetic therapy on lung function and pulmonary haemodynamics in patients with post-traumatic ARDS. Rotational therapy was commenced when the PaO₂/FiO₂ was 150 mmHg to angles of 30–62 degrees laterally with a two-minute pause in each position. Pape et al. (1994) demonstrated a significant improvement in oxygenation in the group receiving kinetic therapy with no notable adverse haemodynamic effects. Patients in this study were not commenced on kinetic therapy until they had severe hypoxaemia. The study was also conducted in a very specific group of patients. It is therefore difficult to apply these results to the general population of patients with ARDS. Further, the authors do not specify the exact angle patients were rotated to.

More recently McLean (2001) hypothesised that kinetic therapy could decrease the incidence of refractory hypoxaemia in a group of patients with risk factors for ALI/ARDS. Patients were rotated to 45 degrees laterally for a minimum of 18 hours per day. McLean

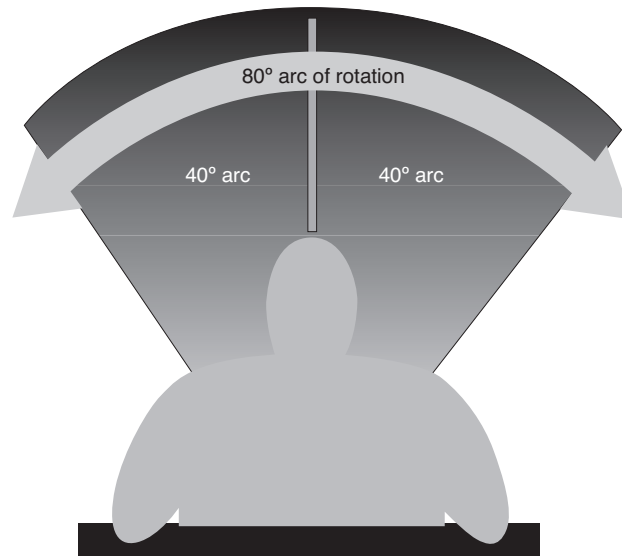


Figure 1.5 Diagrammatic representation of kinetic therapy.

(2001) concluded that kinetic therapy did have a positive effect on the lung function of severely injured trauma patients at risk of atelectasis, ALI and ARDS.

Kinetic therapy appears to have a role in the management of the critically ill patient with pulmonary complications resulting from prolonged immobilisation, who is at risk of VAP (NICE and NPSA 2008; MacIntyre et al. 1999). Further work is required, however, to establish the statistical significance of this therapy in influencing gas exchange in patients with ALI/ARDS.

Prone positioning

Mr Kuan Yew is currently being nursed in the supine position. However, multiple studies over the past 20 years have demonstrated an improvement in $\text{PaO}_2/\text{FiO}_2$ in two-thirds of patients with ARDS placed in the prone position (Langer et al. 1988; Pappert et al. 1994; Gattinoni et al. 2001) (Figure 1.6). Hypotheses offered to explain this improvement in oxygenation have included an increase in FRC, a change in the position of the heart and diaphragm allowing increased recruitment of lung units, increased clearance of secretions and redistribution of perfusion. Current thinking suggests that any improvement in oxygenation is mostly related to changes in the pleural pressure gradient from the dorsal to the ventral surface on turning to the prone position (Lamm et al. 1994). The gradient of pleural pressure from negative ventrally to positive dorsally is not completely reversed on turning prone which leads to a more even distribution of ventilation and improvement in ventilation/perfusion (V/Q) matching.

Unfortunately, although an improvement in oxygenation has been observed, a multi-centre randomised controlled trial published in 2001 failed to demonstrate any differences in clinical outcome between those nursed in the supine position and those nursed prone (Gattinoni et al. 2001). A recent systematic review by Alsaghir and Martin, which



Figure 1.6 Patient placed in the prone position.

included five randomised controlled trials, further supports these findings (Alsaghir and Martin 2008). Turning a critically ill patient prone also has practical implications for the critical care nurse. A team of at least five members of the staff are required to safely turn the patient, and special attention and vigilance is required to prevent the development of pressure necrosis of the face, ears and genitals and to ensure maintenance and patency of the airway. With the lack of a randomised controlled trial supporting the routine use of the prone position, its use as an adjunct to mechanical ventilation strategies in patients with severe hypoxaemia has become a rescue therapy in many critical care units. However, the systematic review by Alsaghir and Martin (2008) did note a significant reduction in mortality in those with a higher illness severity, perhaps suggesting that it should be considered more frequently.

Inhaled nitric oxide/prostacyclin

Initially described almost 15 years ago, the use of inhaled nitric oxide (NO) as a potential therapy for ALI/ARDS has received a great deal of interest in the literature. Given continuously, via inhalation during mechanical ventilation, it selectively vasodilates pulmonary capillaries and arterioles which perfuse ventilated alveoli (Figure 1.7). This results in the diversion of blood from under-ventilated alveoli, subsequently improving V/Q matching and oxygenation. As a vasodilator, NO also has a function in decreasing pulmonary hypertension which can be present in patients with ARDS as a consequence of increased pulmonary vascular resistance caused by various pro-inflammatory mediators, hypoxia or thrombi. The systemic effects of inhaled NO are minimal due to its rapid inactivation on binding with haemoglobin, and there is little risk posed to the critical care team caring for patients receiving it as any NO present in exhaled gas is rapidly absorbed by scavenging systems. Unfortunately, as NO is rapidly deactivated by haemoglobin, any interruption in

20 *Critical Care Nursing: Learning from Practice*

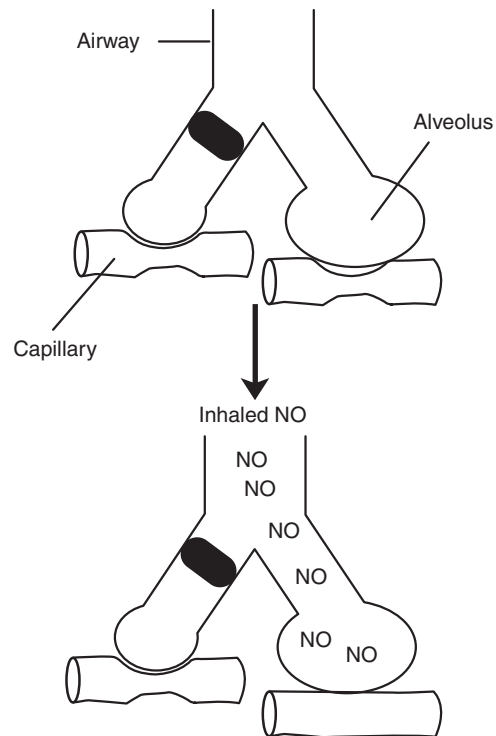


Figure 1.7 Schematic illustrating the vasodilatory effect of inhaled nitric oxide.

its supply, e.g. patient transport or supply exhaustion, can lead to a sudden decrease in PaO₂ or rebound pulmonary hypertension which may precipitate right heart failure.

Randomised controlled trials of the use of NO in ARDS have shown that although NO temporarily improves oxygenation and reduces pulmonary artery pressure in the majority of patients, similarly to other therapies its use is not associated with an improved patient outcome (Rossaint et al. 1995; Bigatello et al. 1994). Inhaled NO should not, therefore, be considered as a standard treatment for Mr Kuan Yew.

Prostacyclin is an endothelium-derived prostaglandin vasodilator which inhibits platelet aggregation and neutrophil adhesion when administered intravenously. Nebulised prostacyclin produces similar effects to inhaled NO with minimal side effects and without measureable platelet dysfunction and appears to provide the same degree of improvement in oxygenation as NO (Dahlem et al. 2004). Similarly to inhaled NO there are, however, no large randomised controlled trials demonstrating an outcome benefit of its use in adults with ARDS.

Extracorporeal membrane oxygenation (ECMO)/extracorporeal CO₂ removal (ECCO₂R)

During ECMO venous blood is removed via a cannula in the inferior vena cava or right atrium, passed through a heart/lung machine and returned to either the right atrium

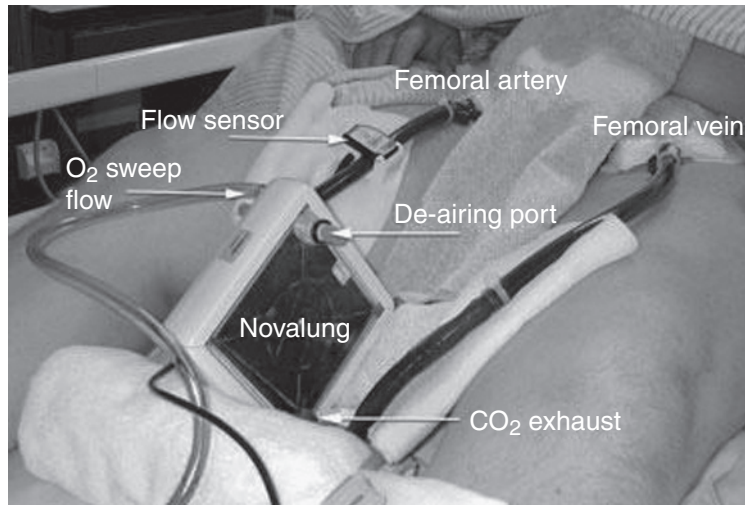


Figure 1.8 Novalung. (McKinlay, 2008). Reproduced with permission from John Wiley & Sons Ltd.

(veno-venous bypass) or aorta (veno-arterial bypass). The use of extracorporeal gas exchange techniques such as ECMO or ECCO₂R in patients with ARDS is viewed as an attractive strategy since it allows the lung to remain at rest, hence preventing any further lung damage, whilst allowing adequate oxygenation and ventilation. ECMO has proven mortality benefit in neonatal ARDS; however, this benefit has not been demonstrated thus far in adult clinical studies. A randomised prospective controlled study of ECMO in 180 adult patients with ARDS (the CESAR trial) has recently been conducted in the UK and early results suggest that there is a survival benefit if it is used early. Further details of this trial can be found on the CESAR website: www.cesar-trial.org/.

ECCO₂R involves the use of an extracorporeal veno-venous circuit with lower blood flows and with oxygenation still occurring via the patient's lungs. One such system is the Novalung (Bein et al. 2006) (Figure 1.8). However, randomised controlled trial of ECCO₂R compared with conventional support in patients with severe ARDS reported no significant difference in survival (Morris et al. 1994).

Several centres have recently reported observational studies demonstrating high survival rates in adult patients managed with extracorporeal support. These encouraging results should, however, be interpreted in the context of a trend towards improved survival, generally in patients with ARDS.

Steroids

Corticosteroids reduce the production of a large number of inflammatory and profibrotic mediators and the importance of steroid therapy in the resolution of lung inflammation in animal models became apparent 20 years ago. Unfortunately, trials of high-dose steroid

22 *Critical Care Nursing: Learning from Practice*

therapy have failed to show a survival benefit in patients with early ARDS and, in fact, some trials have shown an increase in infection rates and mortality.

The use of steroids in late ARDS (7–14 days from diagnosis) has been more closely studied in recent years. The rationale behind this interest is that much of the scarring that occurs during this phase of the illness is as a consequence of unattenuated inflammation that can cause severe damage to the affected alveoli. It has also been postulated that the use of steroids can have an effect on the fibrotic process seen in late ARDS. Unfortunately, however, a recent large multicentre prospective randomised controlled trial conducted by the ARDSNet failed to support the routine use of methylprednisolone for persistent ARDS (National Heart, Lung and Blood Institute (NHLBI) ARDSNet 2006a). In addition they demonstrated that commencement of methylprednisolone two weeks after the onset of ARDS may increase the risk of death. The routine use of corticosteroids in patients with ARDS is therefore not currently supported by the literature.

Circulation

Major emphasis is likely to be placed on ventilatory strategies for Mr Kuan Yew since hypoxaemia and hypoxia are of particular concern. ARDS, however, does affect the cardiovascular system, and cardiovascular management can have an effect on physiological status and outcome for several reasons. Firstly, oxygen delivery to the tissues is dependent not only upon arterial oxygen saturation but also upon the oxygen-carrying capacity of the blood and cardiac output. The management of cardiac output and haemoglobin is therefore as important as maintenance of arterial oxygen saturations via mechanical ventilation strategies. Mr Kuan Yew currently has a normal haemoglobin level, and infusion of packed red cells to increase his oxygen-carrying capacity would therefore not be indicated at this time. It is important, however, to ensure that he has an adequate cardiac output. Secondly, as previously discussed, ARDS is characterised by a degree of intra-pulmonary shunt, and in this situation, mixed venous oxygen saturations have an impact on arterial oxygen saturations. Cardiac output plays a key role in determining mixed venous oxygen saturations (SvO_2) and it is therefore imperative to ensure that an adequate cardiac output and SvO_2 is maintained. Thirdly, the initial stage of ARDS is characterised by pulmonary oedema which results in a decrease in oxygenation. A low PAOP may decrease the rate of oedema formation; therefore, finding the lowest possible PAOP which gives an adequate cardiac output may be a focus of cardiovascular management for Mr Kuan Yew. The measurement of PAOP would, however, require the insertion of a pulmonary artery catheter and the benefits of this are questionable when one considers the risks associated with its use.

The initiation of mechanical ventilation can also have an effect on the cardiovascular system. Pulmonary hypertension associated with ARDS may be exacerbated by the administration of positive pressure and this effect, combined with the direct application of positive pressure to the major vessels within the thoracic cavity, can significantly decrease venous return, leading to a reduction in cardiac output.

From the data presented in the initial assessment, Mr Kuan Yew is demonstrating signs of cardiovascular insufficiency. He has a mean arterial blood pressure (MABP) of <65 mmHg and a tachycardia of 115 beats per minute and is pyrexial. He is also warm to

touch, suggesting that he is peripherally dilated with a low systemic vascular resistance. The management of Mr Kuan Yew's cardiovascular function presents a major challenge in that, as a consequence of ARDS, he also has non-cardiogenic pulmonary oedema, caused by increased pulmonary capillary permeability. On the one hand, intravenous fluid administration is critical to maintain an appropriate intravascular volume to ensure haemodynamic stability and organ perfusion. On the other hand, excessive fluid administration could worsen any pulmonary oedema and compromise gas exchange further. Fluid management practices within critical care units can vary greatly and are often guided by established practice ranging from the liberal 'wet' approach which prioritises perfusion to the very conservative or 'dry' approach which aims to prevent pulmonary oedema.

An associated challenge in managing Mr Kuan Yew's fluid status is the decision as to what is the most effective method of monitoring fluid status. Proponents of the pulmonary artery catheter argue that it is essential to measure the PAOP and cardiac output. However, others argue that the use of a pulmonary catheter carries a significant mortality risk and that fluids can be adequately managed using a central venous catheter in conjunction with clinical assessment. The FACTT study (Fluid and Catheter Therapy Trial), conducted by the NHLBI ARDS network (NIHB, ARDSNet 2006b) concluded that although there was no difference in mortality, patients receiving a conservative fluid strategy demonstrated improved lung function and had shorter duration of mechanical ventilation and intensive care stay. These results support the use of a conservative fluid management strategy for Mr Kuan Yew. With regards to the use of a pulmonary artery catheter versus a central venous catheter, although there were no differences in outcomes such as mortality, length of stay or number of ventilated days, patients with pulmonary artery catheters had twice as many catheter-related complications, and investigators concluded that the use of a pulmonary artery catheter is not indicated in the routine management of patients with ALI/ARDS. Alternative monitoring techniques such as use of a pulse-induced contour cardiac output (PiCCO; Cottis et al. 2003) (Figure 1.9) which measures both extravascular

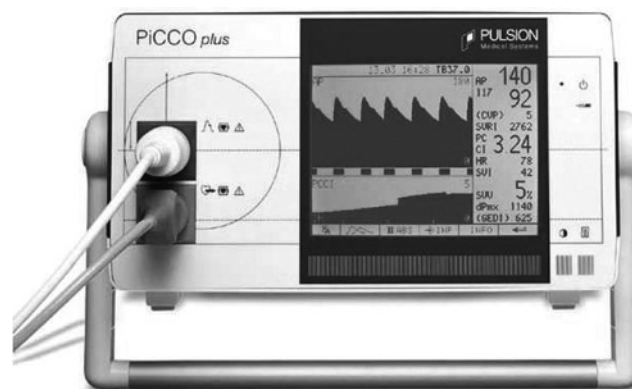


Figure 1.9 PiCCO plus monitor and example of information obtained. Reproduced with permission from Pulsion Medical UK Ltd.

24 *Critical Care Nursing: Learning from Practice*

and intravascular lung water (EVLW and IVLW) may also be used to enable more accurate measurement of the fluid status.

When deciding which fluid to administer, there is currently no conclusive evidence to suggest that choosing either a colloid or a crystalloid results in a significant improvement in outcome in patients with ARDS. The recent SAFE study, although not specifically related to patients with ARDS, suggested that either a colloid or a crystalloid may be used (Finfer et al. 2004). Regardless of the fluid chosen, it is essential that the critical care nurse pays strict attention to fluid balance whilst also closely monitoring gaseous exchange and the effects of the fluid infusion on Mr Kuan Yew's haemodynamic status.

Infused vasopressors and/or inotropic drugs are frequently used to increase a low MABP or cardiac output; however, the use of these should follow adequate and safe fluid resuscitation. Mr Kuan Yew is displaying signs of severe sepsis and the haemodynamic changes seen in ARDS patients are indistinguishable from those seen in sepsis and septic shock (please refer to Chapter 4 for further information on the cardiovascular management of sepsis).

Disability of the nervous system

In 2001, Van Den Berghe et al. demonstrated that strict attention to tight glycaemic control can reduce mortality in the critically ill patient. A continuous intravenous infusion of actrapid may therefore be considered in order to reduce Mr Kuan Yew's blood glucose to within the recommended level (see Chapter 7 for more information on blood glucose management).

Mr Kuan Yew will require sedation and analgesia in order to tolerate the ventilatory support he is receiving. It is important that the critical care nurse assesses pain and sedation requirements frequently in order to prevent over/under-sedation. As part of the ventilator care bundle discussed earlier in this chapter, it is also important that a daily sedation hold is implemented where possible. This will also facilitate a more accurate neurological assessment. A minority of patients with refractory hypoxaemia who are receiving more advanced forms of mechanical ventilation may also require the administration of neuromuscular blocking agents to decrease skeletal muscle oxygen consumption, improve thoracic compliance and favour redistribution of blood flow to vital organs (see Chapters 10 and 11 for further aspects of managing pain, sedation and neuromuscular blockade in the critically ill).

Exposure/environment

It is important when managing Mr Kuan Yew that a holistic approach to care is used as tissue hypoxia can lead to problems with skin integrity and this may be compounded by the use of multiple drug therapies. Respiratory and haemodynamic instability may also render Mr Kuan Yew difficult to turn and reposition. Strict attention to and assessment of the condition of his skin is therefore imperative.

As a patient who is critically ill and is in the ICU, Mr Kuan Yew is also at risk of developing further secondary infection and strict attention to infection control measures is therefore considered a high priority. These measures should include a high standard

of hand hygiene, attention to line and wound care and effective eye and oral care (see Chapter 4 for further information on infection control).

Conclusion

This scenario has discussed the complex challenges associated with the care of a patient with ARDS. The condition does not just affect the respiratory system and a holistic approach to management, including titration of fluid support, titration of medication and a focus on general aspects of caring for a critically ill patient, is imperative.

Key learning points

- ARDS is the most severe form of a spectrum of respiratory disease and presents a great challenge to the multi-professional team within critical care.
- An evidence-based approach is essential, with strict attention being paid to appropriate ventilatory strategies as an initial priority and also the use of care bundles.

Critical appraisal of research paper

Drakulovic et al. (1999) Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: a randomised trial. *The Lancet* **354**(9133), 1851–1858.

This prospective, randomised, controlled trial investigated whether use of the semi-recumbent position can reduce the incidence of nosocomial pneumonia in mechanically ventilated patients. Eighty-six intubated and mechanically ventilated patients in one medical and one respiratory ICU in a tertiary referral hospital in Spain were randomly assigned to supine ($n = 47$) or semi-recumbent ($n = 39$) position. The frequency of clinically suspected and microbiologically confirmed nosocomial pneumonia was assessed in both groups together with other known risk factors. These included the presence of enteral feeding, mechanical ventilation for seven days or more and a Glasgow Coma score of less than nine. Results of the study demonstrated that the frequency of clinically suspected nosocomial pneumonia was lower in the semi-recumbent group than in the supine group (8% vs. 34%, $p = 0.003$). This difference was also seen with microbiologically confirmed pneumonia (5% of those in semi-recumbent position vs. 23% of those in supine position, $p = 0.018$). The authors concluded that the semi-recumbent position reduces the risk and frequency of nosocomial pneumonia, especially in patients receiving enteral nutrition. They also concluded that the risk of nosocomial pneumonia is increased by decreased level of consciousness and long-duration mechanical ventilation.

Reader activities

1. Read the research article written by Drakulovic et al. (1999).
2. Using the critical appraisal framework in Appendix I, consider the methodological quality of the paper.

26 *Critical Care Nursing: Learning from Practice*

3. Reflect on this aspect of your own practice and the implications for future practice management that this paper arises.

A commentary on this paper has been provided by the chapter author in Appendix II.

References

- Acute Respiratory Distress Syndrome Network (ARDSNet) (2000) Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *The New England Journal of Medicine* **342**, 1301–1308.
- Alsaghir A, Martin C (2008) Effect of prone positioning in patients with acute respiratory distress syndrome: a meta-analysis. *Critical Care Medicine* **36**(2), 603–609.
- Amato M, Barbas C, Medeiros D (1998) Effect of a protective ventilation strategy on mortality in acute respiratory distress syndrome. *The New England Journal of Medicine* **338**, 347–354.
- Ashbaugh D, Bigelow D, Petty T, Levine B (1967) Acute respiratory distress in adults. *Lancet* **2**, 319–323.
- Baudouin S (1997) Surfactant medication for acute respiratory distress syndrome. *Thorax* **52**, S9–S15.
- Bein T, Philipp A, Dorlac W, Taeger K, Nerlich M, Schlitt H (2006) A new pumpless extracorporeal interventional lung assist in critical hypoxaemia/hypercarbia. *Critical Care Medicine* **34**, 1372–1377.
- Bernard G, Artigas A, Brigham K, Carlet J, Falke K, Hudson L, Lamy M, Legall J, Morris A, Spragg R (1994) The American-European Consensus Conference on ARDS. *American Journal of Respiratory and Critical Care Medicine* **149**, 818–824.
- Bigatello LM, Hurford WE, Kacmarek RM, Roberts JD, Zappol WM (1994) Prolonged inhalation of low concentrations of nitric oxide in patients with severe adult respiratory distress syndrome. Effects on pulmonary haemodynamics and oxygenation. *Anaesthesiology* **80**(4), 761–770.
- Brochard L, Roudot-Thoraval F, Roupie E, Delclaux C, Chastre Jean, Fernandez-Mondejar E, Clementi E, Mancebo J, Factor P, Matamis D, Ranieri M, Blanch L, Rodi G, Mentec H, Dreyfuss D, Ferrer M, Brun-Buisson C, Tobin M, Lemaire F (1998) Tidal volume reduction for prevention of ventilator induced lung injury in acute respiratory distress syndrome. *American Journal of Respiratory and Critical Care Medicine* **158**(6), 1831–1838.
- Brower R, Shanholtz C, Fessler H, Shade, White P, Wiener C, Teeter J, Dodd J, Almog Y, Piantadosi S (1999) Prospective randomised controlled clinical trial comparing traditional versus reduced tidal volume ventilation in acute respiratory distress syndrome patients. *Critical Care Medicine* **27**(8), 1492–1498.
- Chollet-Martin S, Jourdain B, Gibert C, Elbim C, Chastre J, Gougerot-Pocidal MA (1996) Interactions between neutrophils and cytokines in blood and alveolar spaces during ARDS. *American Journal of Respiratory and Critical Care Medicine* **154**, 594–601.
- Cottis R, Magee N, Higgins DJ (2003) Haemodynamic monitoring with pulse-induced contour cardiac output (PICCO) in critical care. *Intensive and Critical Care Nursing* **19**(5), 301–307.
- Dahlem P, van Aalderen W, de Neef M, Dijkgraaf M, Bos A (2004) Randomised controlled trial of aerosolized prostacyclin therapy in children with acute lung injury. *Critical Care Medicine* **32**, 1055–1057.
- Department of Health (DH) (2007) *Saving Lives: reducing infection, delivering clean and safe care*. Crown copyright, London. Available online at: www.dh.gov.uk/. Accessed 6 August 2009.

- Department of Health (DH) (2010) High impact intervention no.5; care bundle to reduce VAP. Available online at: www.nric.org.uk. Accessed 26th November 2010.
- Derdak S, Mehta S, Stewart E, Smith T, Rogers M, Buchman T, Carlin B, Lowson S, Granton J (2002) Multi-centre oscillatory ventilation for acute respiratory distress syndrome trial. High frequency oscillatory ventilation for acute respiratory distress syndrome in adults: a randomised controlled trial. *American Journal of Respiratory and Critical Care Medicine* **166**, 801–808.
- Drakulovic MB, Torres A, Bauer TT, Nicolas JM, Nogue S, Ferrer M (1999) Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: a randomised trial. *Lancet* **354**(9133), 1851–1858.
- Doyle R, Szaflarski N, Modin G, Wiener-Kronish J, Matthay M (1995) Identification of patients with acute lung injury. Predictors of mortality. *American Journal of Respiratory and Critical Care Medicine* **152**, 1818–1824.
- Dreyfuss D, Soler P, Basset G (1988) High inflation pressure pulmonary oedema: respective effects of high airway pressure, high tidal volume and positive end expiratory pressure. *American Review of Respiratory Disease* **137**, 1159–1164.
- Dreyfuss D, Sauman G (1998) Ventilator induced lung injury: lessons from experimental studies. *American Journal of Respiratory and Critical Care Medicine* **157**, 294–323.
- Estenssoro E, Dubin A, Laffaire E (2002) Incidence, clinical course, and outcome in 217 patients with acute respiratory distress syndrome. *Critical Care Medicine* **30**, 2450–2456.
- Finfer S, Bellomo R, Boyce N, French J, Myburgh J, Norton R (2004) A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *The New England Journal of Medicine* **350**, 2247–2256.
- Garber B, Hebert P, Yelle J, Hodder R, McGowan J (1996) Adult respiratory distress syndrome: a systematic review of incidence and risk factors. *Critical Care Medicine* **24**(4), 687–695.
- Gattinoni L, Pesenti A, Avalli L, Rossi F, Bombino M (1987) Pressure-volume curve of total respiratory system in acute respiratory failure. Computed tomographic scan study. *American Review of Respiratory Diseases* **136**, 730–736.
- Gattinoni L, Pelosi P, Crotti S (1995) Effects of positive end expiratory pressure on regional distribution of tidal volume and recruitment in adult respiratory distress syndrome. *American Journal of Respiratory and Critical Care Medicine* **151**, 1807–1814.
- Gattinoni L, Tognoni G, Pesenti A (2001) Effect of prone positioning on the survival of patients with acute respiratory failure. *The New England Journal of Medicine* **345**, 568–572.
- Grap M, Munro C, Hummel R, Elswick RK, McKinney J, Sessler C (2005) Effect of backrest elevation on the development of ventilator-associated pneumonia. *American Journal of Critical Care* **14**(4), 325–332.
- Herridge M, Cheung A, Tansey C (2003) One-year outcomes in survivors of the acute respiratory distress syndrome. *The New England Journal of Medicine* **348**, 683–693.
- Hess D (2005) Patient positioning and ventilator-associated pneumonia. *Respiratory Care* **50**(7), 892–897.
- Hilbert G, Gruson D, Vargas F (2001) Non-invasive ventilation in immunosuppressed patients with pulmonary infiltrates, fever and acute respiratory failure. *The New England Journal of Medicine* **344**, 481–484.
- Ibrahim E, Tracy L, Hill C, Fraser V, Kollef M (2001) The occurrence of ventilator-associated pneumonia in a community hospital: risk factors and clinical outcomes. *Chest* **120**(2), 555–561.
- Lamm W, Graham M, Albert R (1994) Mechanism by which the prone position improves oxygenation in acute lung injury. *American Journal of Respiratory and Critical Care Medicine* **150**, 184–193.

28 *Critical Care Nursing: Learning from Practice*

- Langer M, Mascheroni D, Marcolin R, Gattinoni L (1988) The prone position in ARDS patients: a clinical study. *Chest* **94**, 103–107.
- MacIntyre N, Helms M, Wunderink R, Schmidt G, Sahn S (1999) Automated rotational therapy for the prevention of respiratory complications during mechanical ventilation. *Respiratory Care* **44**, 1447–1451.
- McLean B (2001) Rotational kinetic therapy for ventilation/perfusion mismatch. *Critical Care Nursing in Europe* **1**, 113–118.
- Morris A, Wallace C, Menlove R, Clemmer T, Orme J, Weaver L (1994) Randomised clinical trial of pressure controlled inverse ratio ventilation and extracorporeal CO₂ removal for adult respiratory distress syndrome. *American Journal of Respiratory and Critical Care Medicine* **149**, 295–305.
- Murray J, Matthay M, Luce J (1988) An expanded definition of the adult respiratory distress syndrome. *American Review Respiratory Disease* **138**, 720–723.
- National Heart and Lung Institute (NHLI), National Institute of Health (NIH) (1972) *Respiratory Distress Syndromes: task force report on problems, research approaches, needs*. US Government Printing Office, Washington, DC, **73-432**: 165–180.
- National Heart, Lung and Blood Institute (NHBL) Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network (2004) Higher versus lower positive expiratory end pressures in patients with acute respiratory distress syndrome. *The New England Journal of Medicine* **351**, 327–336.
- National Heart, Lung and Blood Institute (NHBL) Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network (2006a) Efficacy and safety of corticosteroids for persistent acute respiratory distress syndrome. *The New England Journal of Medicine* **354**(16), 1671–1684.
- National Heart, Lung and Blood Institute (NHBL) Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network (2006b) Pulmonary artery versus central venous catheter to guide treatment of acute lung injury. *The New England Journal of Medicine* **354**, 2213–2224.
- National Institute for Health and Clinical Excellence (NICE), National Patient Safety Agency (NPSA) (2008) Technical patient safety solutions for ventilator associated pneumonia in adults. Available online at: www.nice.org.uk. Accessed 26th November 2010.
- Pape H, Regel G, Borgmann W, Sturm J, Tscherne H (1994) The effect of kinetic positioning on lung function and pulmonary haemodynamics in post traumatic ARDS: a clinical study. *Injury* **25**, 51–57.
- Pappert D, Rossaint R, Slama K (1994) Influence of positioning on ventilation-perfusion relationships in severe adult respiratory distress syndrome. *Chest* **106**, 1511–1555.
- Rello J, Ollendorf D, Oster G (2002) VAP Outcomes Scientific Advisory Group. Epidemiology and outcomes of ventilator-associated pneumonia in a large US database. *Chest* **122**(6), 2115–2121.
- Rossaint R, Falke K, Lopez F, Slama K, Pison U, Zapol W (1993) Inhaled nitric oxide for the adult respiratory distress syndrome. *The New England Journal of Medicine* **328**, 399–403.
- Rossaint R, Gerlach H, Schmidt-Ruhnke H, Pappert Dirk, Lewandowski K, Steudel W, Falke K (1995) Efficacy of inhaled nitric oxide in patients with severe ARDS. *Chest* **107**, 1107–1110.
- Stewart T, Meade M, Cook D (1998) Evaluation of a ventilation strategy to prevent barotrauma in patients at high risk for acute respiratory distress syndrome. *The New England Journal of Medicine* **338**, 355–361.
- Tablan O, Anderson L, Besser R, CDC Healthcare Infection Control Practices Advisory Committee (2004) Guidelines for preventing health care-associated pneumonia, 2003: Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. *MMWR Recommendations and Reports* **53**(RR-3), 1–36.
- Tharratt R, Allen R, Albertson T (1988) Pressure controlled inverse ratio ventilation in severe adult respiratory failure. *Chest* **94**, 755.

- Tremblay L, Valenza F, Ribeiro S, Li J, Slutsky A (1997) Injurious ventilatory strategies increase cytokines and c-fos m-RNA expression in an isolated rat lung model. *Journal of Clinical Investigation* **99**, 944–952.
- Van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R (2001) Intensive insulin therapy in critically ill adults. *The New England Journal of Medicine* **345**, 1359–1367.
- Van Nieuwenhoven C, Vandenbroucke-Grauls C, Van Tiel F, Joore H, Van Schijndel R, Van der Tweel I, Ramsay G, Bonten M (2006) Feasibility and effects of the semi-recumbent position to prevent ventilator associated pneumonia: a randomized study. *Critical Care Medicine* **34**, 396–402.
- Villar J, Slutsky A (1989) The incidence of the adult respiratory distress syndrome. *American Review of Respiratory Disease* **140**, 814–816.
- Vincent J, Bihari D, Suter P, Bruining H, White J, Nicolas-Chanoin M, Wolff M, Spencer R, Hemmer M (1995). The prevalence of nosocomial infection in intensive care units in Europe. Results of the European Prevalence of Infection in Intensive Care (EPIC) Study. *Journal of American Medical Association* **278**, 639–644.
- Ware L, Matthay M (2000) The acute respiratory distress syndrome. *The New England Journal of Medicine* **342**(18), 1334–1350.
- Wax R, Angus D (2000) The molecular genetics of sepsis. In: *Yearbook of Intensive Care and Emergency Medicine*, Vincent JL (ed). Springer, Berlin, pp. 3–17.