Acute Respiratory Distress Syndrome
Pathophysiology, current management and implications for physiotherapy

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Key Words
Respiratory distress syndrome – acute, physical therapy, patient positioning, postural drainage, ventilation – mechanical

Summary
Acute respiratory distress syndrome (ARDS) is an intensive care condition first described more than three decades ago. Recent advances in the understanding of its pathology and management are reviewed. Of particular relevance to physiotherapists are the characteristic metabolic, haemodynamic and respiratory consequences of ARDS. These have implications for physiotherapy practice as chest physiotherapy has been demonstrated to have metabolic, haemodynamic and respiratory effects. Application of chest physiotherapy may exacerbate or attenuate these effects. However, there is a lack of research in this area.

On the other hand, body positioning has enjoyed much attention recently. The prone position is strongly advocated as an inexpensive alternative to new mechanical ventilatory modes in improving oxygenation. Despite a large body of research on ARDS, it continues to carry a mortality rate of up to 70%. Therefore, there is a need for continuing research efforts on investigating its response to various therapeutic and supportive measures.

Introduction
Adult (or acute) respiratory distress syndrome (ARDS) was first described 30 years ago (Ashbaugh et al, 1967). In this original description, 12 out of 272 patients who were admitted to the intensive care unit (ICU) developed acute onset of severe dyspnoea, hypoxaemia, cyanosis and decreased respiratory system compliance, despite supplemental oxygen and mechanical ventilation. Since then, a large body of basic and clinical research on ARDS has been generated with the aim to elucidate its aetiology and to optimise its treatment. The purpose of this paper is to review the current understanding of ARDS, its pathophysiology, management and the implications for physiotherapy.

Definition
Ashbaugh and colleagues (1967) originally referred to the condition as ‘acute respiratory distress’. In a subsequent report, this was described as ‘adult respiratory distress syndrome’ (Petty and Ashbaugh, 1971), a term that has since become established in the medical diagnostic vocabulary. Of the 12 patients described by Ashbaugh et al (1967), one was a child. It is now recognised that ARDS can affect not only adults, but also children (Katz, 1987; Davis et al, 1993; Sarnaik and Lieh-Lai, 1994; Paulson et al, 1995). It was therefore proposed that ARDS be considered as a form of acute lung injury (ALI) (Murray et al, 1988; Beale et al, 1993). The American-European Consensus Conference on ARDS recommended that ARDS, now known as acute respiratory distress syndrome, be defined as a ‘syndrome of inflammation and increased permeability’ associated with characteristic clinical, physiological and radiological changes (Bernard et al, 1994). These changes include an acute onset of deteriorating oxygenation expressed in terms of the ratio of arterial oxygen tension to fraction of inspired oxygen (PaO$_2$/FiO$_2$). For ARDS, the PaO$_2$/FiO$_2$ is defined as less than 200 mm Hg, while for ALI it is less than 300 mm Hg. Thus, ARDS is a more severe form of ALI. This definition also requires evidence of bilateral lung field infiltrates on chest roentgenograms; exclusion of left atrial or pulmonary capillary hypertension as a cause, though not necessarily the consequence, of ARDS; as well as the presence of one or more risk factors (Bernard et al, 1994).

Incidence, Mortality and Risk Factors
The reported incidence of ARDS varies between centres. The annual incidence per 100,000 population has been reported to be 1.5-3.5 cases in a Spanish island (Villar and Slutsky, 1989), 4.5 in the United Kingdom (Webster et al, 1988), 7.3-9.3 in Australia (Nolan et al, 1997) and 7.5 in the United States of America (Murray, 1977). While it is likely that the incidence may also depend on resuscitation practices among intensive care units (ICUs), a figure between 1.5 to 5.3 per 100,000 may be considered acceptable (Garber et al, 1996).

Despite much research on various treatment strategies, ARDS continues to carry a high mortality rate (Lee et al, 1994). Regardless of management practices, its mortality rate ranges from 30% to 70% (Ashbaugh et al, 1967; Villar and Slutsky, 1989; Lee et al, 1994; Doyle et al, 1995; DiRusso et al, 1995; Milberg et al, 1995; Nolan et al, 1997). Age was found to be associated with higher mortality in one retrospective study.
that the condition incurred a daily ICU charge of US$2,430 per patient per day (Bellamy and Oye, 1984). Seven out of 39 patients with ARDS survived in this survey. Survivors cost significantly less (US$1,683) than the non-survivors (US$2,760). The high costs and poor outcomes of ARDS are of great concern and a reason for ongoing therapeutic trials.

Sepsis, multiple blood transfusions, near drowning, pneumonia, aspiration of gastric contents, multiple trauma and drug overdose are some of the risk factors associated with ARDS (Pepe et al, 1982; Fowler et al, 1983; Hudson et al, 1995). Moreover, non-pulmonary organ system dysfunction appears to be prevalent in ARDS, with high incidence of renal, hepatic, gastrointestinal and cardiac involvement (Dorinsky and Gadek, 1989). Mortality is known to increase with the presence of risk factors, especially sepsis and non-pulmonary organ system dysfunction (Doyle et al, 1995). For example, sepsis accounts for about one-third (Montgomery et al, 1985) to over two-thirds (Hudson et al, 1995) of all ARDS deaths. Similarly, infection in the lungs, pleura or abdomen (Seidenfeld et al, 1986), gastric aspiration (Hudson et al, 1995) and non-pulmonary organ system dysfunction (Bell et al, 1983; Montgomery et al, 1985) have also been associated with high mortality in ARDS. Respiratory failure, however, accounts for only 10% of all ARDS deaths (Montgomery et al, 1985).

There is a large number of inflammatory cells, such as neutrophils, mononuclear cells, pulmonary intravascular macrophages and platelets. A combination of various inflammatory cells, humoral mediators and nitric oxide (NO) metabolism may be involved in causing the inflammatory response and its sequelae (Fulkerson et al, 1996). Macrophages and platelets are involved in the release of mediators such as cytokines, products of arachidonic acid metabolism, oxygen free radicals and complements such as C5a (Rinaldo and Rogers, 1982). Cytokines such as tumour necrosis factor-alpha (TNF-α) and interleukins (ILs) are elevated in plasma and bronchoalveolar lavage fluid in patients with ARDS (Nash et al, 1991; Suter et al, 1992; Meduri et al, 1995). They cause neutrophil adhesion on the endothelium, thus potentiating the inflammatory responses (Wortel and Doerschuk, 1993). The toxic reactive oxygen species, produced or enhanced by high FiO₂ and reperfusion of hypoxic tissues, also mediate lung injury (Beale et al, 1993). Pulmonary vasoconstriction occurs as a result of alveolar hypoxia and circulating vasoactive substances (Zapol et al, 1977; Zapol and Snider, 1997; Greene, 1986; Artigas et al, 1987) such as arachidonic acid products, platelet-activating factors and complements. Inducible NO synthase is an enzyme found in both epithelium and endothelium which produces NO in response to cytokines and endotoxin (Barnes and Belvisi, 1993). Excess NO may cause further capillary leak and plasma exudation.

In the first few hours of an animal model of lung injury, 20–25% of the increased extravascular lung water has been shown to drain into the pleural space, causing pleural effusion (Wiener-Kronish et al, 1988). Radiologically, there is evidence of diffuse consolidation, pleural effusions (Aberle and Brown, 1990) and about 30% incidence of pneumothorax (Gattioni et al, 1994). During this stage, the patient becomes progressively dyspnoeic and tachypnoeic, often requiring ventilatory assistance.

During the intermediate stage, the lungs become heavier and may be two to three times the normal
lung weight (Gattinoni et al., 1988a). The weight of the oedematous lungs exerts a vertical gradient of decreasing transpulmonary pressure, causing compression atelectasis of lung units in the dependent regions (Gattinoni et al., 1988a; Pelosi et al., 1996). As the oedema accumulates, fibrin is formed, causing thrombotic obstruction in the pulmonary vessels and resulting in pulmonary arterial hypertension (PAH) (Greene, 1986; Artigas et al., 1987). An increased right ventricular afterload is associated with PAH (Coetzee et al., 1996) and compensatory Frank-Starling mechanism operates to maintain right ventricular stroke volume (Dhainaut and Brunet, 1990). Poor right ventricular contractility, as in septic shock, may undermine this mechanism, resulting in right ventricular failure (Dhainaut and Brunet, 1990). Surfactant depletion and abnormalities have also been observed (Petty et al., 1979; Seeger et al., 1990; Li et al., 1991) and noted to correlate with the severity of ARDS (Pison et al., 1989). This surfactant dysfunction may contribute to the decrease in lung compliance, which has been found to be significantly lower than that in the early stage (Gattinoni et al., 1994).

The late ARDS stage is marked by widespread lung damage. Histologically, there is diffuse fibrosis (Lamy et al., 1976) and presence of hyaline membranes (Ashbaugh et al., 1967). Significant decrease in respiratory compliance and an increase in intrapulmonary shunts, dead space ventilation (Gattinoni et al., 1994) and airway resistance have been reported (Pelosi et al., 1995) although the latter is reversible by nebulised salbutamol (Morina et al., 1997). The increasing fibrotic changes may be responsible for the alterations in gas exchange and lung mechanics. The incidence of pneumothorax was reported to be 87% (Gattinoni et al., 1994). Often sepsis or infection is superimposed in the late stage of ARDS, and may be associated with high mortality rate.

Gattinoni and colleagues (1993) have used computed tomographic scans to characterise the lung structure in ARDS. Previously, ARDS was believed to be diffuse lung damage with a homogeneous and generalised distribution of atelectasis and damage to the alveolar-capillary barrier (Maunder et al., 1986). Gattinoni and colleagues (1988a, 1991) have however demonstrated that most of the lung collapses were found in the dependent zones. Based on these and other studies (Maunder et al., 1986; Gattinoni et al., 1987, 1988b, 1993), three areas in ARDS lungs have been distinguished: (1) an area of irreversibly damaged lung units; (2) an area of relatively recruitable collapsed alveoli; and (3) a very small area of normal undamaged alveoli. These undamaged lung units have been termed the ‘baby lung’ because they occupy a very small proportion, approximately 15 to 20%, of normal adult lung (Gattinoni et al., 1988a).

The oxygen transport system is also altered in ARDS. Normally when oxygen delivery (DO,) to the tissues decreases, as it does in ARDS (Meade et al., 1994), oxygen consumption (VO,) remains constant while tissue oxygen extraction increases. When DO, decreases to a critical level, VO, will then decrease linearly as DO, decreases. This is known as physiological dependence of VO, on DO,.

In ARDS, a ‘pathological’ dependence of VO, on DO,, a linear relationship between these two variables, has been noted whereby oxygen extraction does not increase with decreasing DO, (Knoss, 1993; Russell and Phang, 1994). Furthermore, DO, is caused by decreased cardiac contractility rather than preload and afterload (Stelzer et al., 1994a). Therefore, strategies to enhance DO, by inotropes and catecholamines have been used (Yu et al., 1995). However, calculation errors in the determination of VO, and DO, have been shown recently to account for the linear relationship established by previous studies (Hanique et al., 1994; Phang et al., 1994; Stelzer et al., 1994b). Nevertheless, at least one recent study, using direct measurements by indirect calorimetry, has demonstrated a VO,-DO, linear relationship in patients with ARDS and sepsis (Yu et al., 1996).

**Management: Current Practices**

Lack of controlled randomised trials of specific therapeutic strategies explains why the current principle of management is still largely supportive (Kolleff and Schuster, 1995). The main goals of supportive treatment are to ensure adequate gas exchange and tissue perfusion, while minimising any possible iatrogenic or secondary lung injury. Treatment of specific predisposing and risk factors is also a goal in the overall management of ARDS.

**Mechanical Ventilation**

The initial settings, the application of positive end-expiratory pressure (PEEP) and various types of ventilatory modes have been the topics of many theoretical speculations and fewer investigations of rigorous designs. The application of increasingly high levels of PEEP recruits previously collapsed alveoli; while low levels of PEEP distend and aerate normal undamaged alveoli (Gattinoni et al., 1987, 1988b, 1993). But at high levels of PEEP, the normal alveoli have been found to be overly distended (Gattinoni et al., 1993). Overdistention, or ‘volutrauma’ has been shown to be associated with pneumothorax in ARDS (Finfer and Rocker, 1996). High supplemental oxygen therapy has also been used, but the ensuing
The iatrogenic effects of high levels of PEEP and FiO₂ have resulted in the development of many different ventilatory strategies.

One strategy to avoid volutrauma to the 'baby lung' employs the use of lower tidal volumes (as low as 6 ml/kg body weight) and/or ventilatory rate, thus permitting the arterial carbon dioxide tension to rise. Permissive hypercapnia aims to reduce peak airway distending pressure (and hence volutrauma) associated with high tidal volume (Kacmarek and Hickling, 1993; Tuxen, 1994). This has been shown to decrease the incidence of pulmonary infection (Lee et al., 1990), improve respiratory system compliance (Leatherman et al., 1991), decrease minute ventilation (McIntyre et al., 1994), increase oxygen delivery (Kiiski et al., 1992; Thorens et al., 1996), facilitate earlier ventilator weaning (Amato et al., 1995), prevent a decrease in cardiac output (CO) (Leatherman et al., 1991; Kiiski et al., 1992), and cause lower mortality (Hickling et al., 1990). However, it has also been associated with elevated cardiac index, intrapulmonary shunt and mean pulmonary arterial pressure (Thorens et al., 1996).

Another strategy commonly used to reduce the peak airway pressures is inverse-ratio ventilation (IRV) where the inspiration:expiration (I:E) ratio is increased. However, recent controlled randomised trials have shown no additional benefits, in terms of oxygenation, lung mechanics and haemodynamics, over the conventional volume-cycled ventilation with normal I:E ratio (Mancebo et al., 1994; Mercat et al., 1997). In fact, IRV has been found to decrease CO (Mercat et al., 1997).

Airway pressure release ventilation is another form of ventilatory mode that aims to reduce the peak airway pressure. It is a form of continuous positive airway pressure (CPAP) for spontaneously breathing patients, with periods when the airways rapidly depressurise allowing expiratory gases to empty and fresh gas to refill the airways to the pre-set CPAP. It has been shown to have lower peak airway pressure, higher oxygenation and lower intrapulmonary shunt when compared to volume-cycled IRV (Sydow et al., 1994).

Extracorporeal ventilatory support has been tried in some centres. Prospective clinical randomised trials have shown that both extracorporeal membrane oxygenation (Zapol et al., 1979) and extracorporeal carbon dioxide removal (Morris et al., 1994) offered no additional benefits over conventional ventilatory support.

Partial liquid ventilation with perfluorocarbons has been used and studied in some centres. The lungs are filled with perfluorocarbon to functional residual capacity (FRC) while gas ventilation is effected by conventional mechanical ventilator. Surface tension is reduced with this approach, thereby recruiting more lung volume and facilitating better gas exchange (Hernan et al., 1996). Studies involving both animal models (Hernan et al., 1996; Hirschl et al., 1996a) and patients with severe ARDS on extracorporeal support (Hirschl et al., 1996b; Kazerouni et al., 1996) have shown improved gas exchange and lung mechanics without any complications.

**Fluid Management**

Fluid therapy has been a topic of much debate within the medical community (Schuster, 1995; Sporn, 1996). Some studies have shown lower mortality and improved outcomes of fluid support to increase DO₂ to supranormal states (Shoemaker et al., 1988; Yu et al., 1993), while others have shown better outcomes with fluid restriction (Mitchell et al., 1992). It has been suggested that fluid therapy is best if individually tailored (Fulkerson et al., 1996).

**Pharmacological Interventions**

Pharmacological interventions aim to interfere with the inflammatory process, reduce PAH and replenish surfactant. Recent randomised controlled trials have shown better oxygenation, reduced levels of inflammatory markers or improved survival with pharmacological interventions, including surfactant replacement (Weg et al., 1994; Gregory et al., 1997); antioxidants such as N-acetylcysteine and procysteine (Bernard et al., 1997); vasodilator such as nitric oxide (Walmrath et al., 1996; Doering et al., 1997) and prostacyclin (Walmrath et al., 1996); substances which enhance the hypoxic pulmonary vasoconstriction such as phenylephrine (Duering et al., 1997) and almitrine (Wyszcki et al., 1994; Benzing et al., 1997; Jolliet et al., 1997); as well as inflammatory mediator-inhibitors such as prostaglandins E₃ (Abraham et al., 1996). Corticosteroids have been shown to have no effect on improving survival in one randomised clinical trial (Bernard et al., 1987). Similarly, gut decontamination has not been found effective in reducing the incidence of ARDS (Cerra et al., 1992). Immunological therapy by administration of anti-endotoxin antibody (Bigatello et al., 1994) as well as cytokine antagonists (Opal et al., 1997) do not appear to improve survival when compared with conventional approaches.

**Body Positioning**

It has been suggested that the upright position could be important in improving the FRC in ARDS, a condition with a reduced FRC (Dean, 1996a).
The aim of body positioning in ARDS is to use gravity to optimise the ventilation-perfusion ratio, thereby improving gas exchange and oxygenation. However, to date, there has been no research evidence in favour of the upright position for people with ARDS. One study reported that PaO2/FiO2 remained unchanged when the semi-recumbent position was compared to the supine position in ARDS patients (Bittner et al., 1996). Moreover, respiratory compliance was reduced in the semi-recumbent compared to the supine position in this study.

The lateral decubitus position has been shown consistently to improve oxygenation (PaO2, and/or PaO2/FiO2) in intubated and ventilated ICU patients with acute respiratory failure due to unilateral lung involvement (Ibáñez et al., 1981; Prokocimer et al., 1983; Rivara et al., 1984; Gillespie and Rehder, 1987). There is no clear advantage of this position for patients with radiographic appearance of bilateral lung field infiltrates (Nelson and Anderson, 1989). Furthermore, in patients with ARDS superimposed with marked SIR and moderate right ventricular dysfunction, the lateral decubitus position for just 15 minutes has been shown to cause significant haemodynamic changes (Bein et al., 1996). Left side lying appears to induce increased pre-load resulting in higher than normal CO; while right side lying causes a significant decrease in pre-load and right ventricular end-diastolic volume with a resultant systemic hypotension (mean arterial pressure fell from 85 mm Hg in supine to 72 mm Hg) (Bein et al., 1996). Thus, in applying the lateral decubitus position in patients with ARDS, it is important to be aware of and to monitor the haemodynamic variables, especially where CO may be compromised.

The prone position has been found to cause better arterial oxygenation in patients with ARDS (Piehl and Brown, 1976; Douglas et al., 1977). Since 1988, there has been a growing interest in the prone position (Langer et al., 1988; Gattinoni et al., 1991; Brüssel et al., 1993; Pappert et al., 1994; Hörmann et al., 1994; Murdoch and Storman, 1994; Vollman and Bander, 1996; Chatte et al., 1997; Stocker et al., 1997; Fridrich et al., 1997; Mure et al., 1997). All except four of these studies involved manual turning of the patients into prone, usually involving three to four medical, nursing or auxiliary staff (see table). All except one study (Vollman and Bander, 1996) did not randomise the assignment of patients into the supine (control) and prone position. Despite the lack of randomised trials, the prone position has consistently resulted in higher oxygenation in all studies (see table), reduced intrapulmonary shunts (Brüssel et al., 1993; Pappert et al., 1994; Hörmann et al., 1994; Fridrich et al., 1997), lowered mortality (Stocker et al., 1997) and decreased FiO2 (Brüssel et al., 1993; Mure et al., 1997). No haemodynamic complications have been reported (Gattinoni et al., 1991; Brüssel et al., 1993; Pappert et al., 1994; Hörmann et al., 1994; Murdoch and Storman, 1994; Vollman and Bander, 1996; Chatte et al., 1997). However, dependent oedema in the face is to be expected (Douglas et al., 1977; Brüssel et al., 1994; Chatte et al., 1997; Fridrich et al., 1997; Mure et al., 1997). A semi-prone position has also been described in five case studies (Schmitz, 1991) with similar results.

Animal models of ALI have provided some explanations for these observations. A re-distribution of regional ventilation is likely to occur in adopting the prone position. Transpulmonary pressure exceeding airway opening pressure was observed to increase in the non-dependent lungs in canine models of lung injury (Lamm et al., 1994). Increased normal ventilation-perfusion units in the dorsal lung regions (Lamm et al., 1994) and reduced shunt perfusion (Albert et al., 1987) were shown to account for the improvement in oxygenation in patients lying prone. Furthermore, in the prone position, the weight of the heart, the mediastinum (Margulies and Rodarte, 1990) and the upper abdomen (Liu et al., 1990) no longer compress the underlying compromised lungs as in the supine position.

Therefore, it appears that there is quite strong evidence of enhanced oxygenation in ARDS patients placed in the prone position. Further investigations should focus on the effect of upright position, the optimal frequency and duration of the prone position, and the cost-effectiveness of this position when compared to the other modalities to improve oxygenation.

Finally, the effect of continuous turning, or 'kinetic therapy' has been investigated in Europe and North America (Gentiello et al., 1988; Choi and Nelson, 1992; Pape et al., 1994). A meta-analysis of research studies on kinetic therapy in critically ill patients appeared to suggest that this treatment modality had no significant effect on reducing the incidence of ARDS in a pooled sample size of 419 patients across five studies (Choi and Nelson, 1992). Pape et al (1994) compared a prospective cohort of post-trauma ARDS patients subjected to continuous turning with a historical cohort of ARDS patients who had been nursed only in the supine position. Significant improvement in PaO2/FiO2 with an accompanying decrease in pulmonary shunt was reported. The extravascular lung water was also significantly lower in the 'kinetic' group than the 'supine' control group. There were no significant
### Effects of the prone position in patients with ARDS

<table>
<thead>
<tr>
<th>Studies</th>
<th>No of subjects</th>
<th>Manual or mechanical turning</th>
<th>Duration of position (hours)</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piehl and Brown (1976)</td>
<td>5</td>
<td>Mechanical</td>
<td>Not stated</td>
<td>Not studied</td>
</tr>
<tr>
<td>Douglas et al (1977)</td>
<td>6</td>
<td>Both</td>
<td>4 and 33</td>
<td>CO↑ in two patients</td>
</tr>
<tr>
<td>Langer et al (1988)</td>
<td>13</td>
<td>Manual</td>
<td>2</td>
<td>None observed</td>
</tr>
<tr>
<td>Hörmann et al (1994)</td>
<td>7</td>
<td>Mechanical</td>
<td>12 over 6½ days</td>
<td>No haemodynamic changes</td>
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<tr>
<td>Murdoch and Storman (1994)</td>
<td>7</td>
<td>Manual</td>
<td>½</td>
<td>No haemodynamic changes</td>
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<tr>
<td>Vollman and Bander (1996)</td>
<td>15</td>
<td>Mechanical</td>
<td>½</td>
<td>No haemodynamic changes</td>
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<tr>
<td>Chatte et al (1997)</td>
<td>32</td>
<td>Manual</td>
<td>4</td>
<td>78% had PaO2/FiO2 ↑ more than half of these maintained improvements on return to supine</td>
</tr>
<tr>
<td>Fridrich et al (1997)</td>
<td>20</td>
<td>Manual</td>
<td>½ each turn; total 96 after four turns</td>
<td>Improvement up to 4th prone turn.</td>
</tr>
<tr>
<td>Mura et al (1997)</td>
<td>13</td>
<td>Manual</td>
<td>Individualised 2 to 36 at one time</td>
<td>PaO2/FiO2 ↑; PawO2 ↓. Mortality 12%&lt; comparable groups reported elsewhere</td>
</tr>
</tbody>
</table>

**FiO2** = fraction of inspired oxygen; ↑ = increase; ↓ = decrease; CT scan = computed tomographic scan; CO = cardiac output; SaO2 = oxygen saturation; PaO2/FiO2 = arterial oxygenation tension to fraction of inspired oxygen ratio; PaO2 = arterial oxygen tension; PawO2 = alveolar-arterial oxygen difference; Q/V = interpulmonary shunt.
differences in the haemodynamic variables between both groups. The authors speculated that the improvements could be due to better drainage and removal of bronchial secretions, re-inflation of collapsed dependent alveoli, mobilisation of interstitial fluid into the capillary system to arrest pulmonary oedema and optimisation of the ventilation-perfusion ratio in previously shunted areas (Pape et al, 1994). Kinetic therapy, therefore, appears to have short-term clinical benefits but it does not reduce the incidence of ARDS.

The Role of Physiotherapy

The role of physiotherapy in modifying or exacerbating the inflammatory responses in ARDS has never been investigated. Some, if not the majority, of the inflammatory processes in ARDS are also found during exercise. As a stressor to the immune system, strenuous aerobic and eccentric exercises in top athletes were found to increase significantly the production of cytokines, such as TNF-α and the ILs, both during and after exercises (Cannon et al, 1991; Northoff and Berg, 1991; Kverno et al, 1992; Rivier et al, 1994). This increase in plasma cytokines has been implicated as the source of symptoms, such as fever, following intense exercise bouts (Northoff and Berg, 1991). Physiotherapy has been described as a form of stressor in the ICU setting (Weissman and Kemper, 1993). Positioning and mobilisation have also been advocated as a form of physical stressor in the ICU setting to combat the adverse effects of bed rest (Dean, 1996b). However, the effects of physiotherapy as an ICU stressor on the immune system and the inflammatory response in ALI and ARDS can so far only be inferred from the research literature on exercise immunology.

Physiotherapy, in terms of chest wall percussion and vibration as well as manual hyperinflation, to enhance mucociliary clearance in patients with ARDS has also never been studied, although it has been suggested to be of value (Takala, 1997). Nevertheless, a fair number of researchers have documented adverse metabolic, haemodynamic and respiratory effects of these techniques. Since ARDS has potential or known metabolic, haemodynamic and respiratory deficits, application of physiotherapy techniques must be based on weighing the risks-and-benefits scale. Any combination of postural drainage with or without manual techniques of either percussion, vibration and/or suctioning has been shown to significantly increase VO₂ (Weissman et al, 1984; Swinamer et al, 1987; Klein et al, 1988; Weissman and Kemper, 1993; Harding et al, 1994; Dean et al, 1996), cause cardiac arrhythmias (Hammon et al, 1992), elevate circulating catecholamines (Aitkenhead et al, 1984), increase rate-pressure product and CO (Aitkenhead et al, 1984; Weissman et al, 1984; Klein et al, 1988; Weissman and Kemper, 1993; Harding et al, 1993, 1994, 1995; Cohen et al, 1996) and reduce PaO₂ (Huseby and Hudson, 1976; Tyler et al, 1980; Connors et al, 1980; Harding et al, 1994). Manual hyperinflation has also been shown to decrease CO (Singer et al, 1994), which may further compromise an already poor DO₂ state in ALI and ARDS. Furthermore, tissue extraction is said to be the main mechanism to compensate for the increase in VO₂ during and following chest physiotherapy, as in exercise (Weissman and Kemper, 1991, 1993). In ARDS, however, any increase in VO₂ will require an increase in DO₂ instead of tissue extraction (Knox, 1993). It should also be recognised that any of these physiotherapy modalities with known metabolic, haemodynamic and respiratory effects may further exacerbate the similar effects of permissive hypercapnia and IRV, besides ARDS itself.

In contrast, two studies which included patients with ARDS among other diagnoses have shown beneficial effects of physiotherapy. Mackenzie and colleagues (1980), studying a sample of 42 ICU patients which included two patients with ARDS, found improved total lung/thorax compliance. This was supported by another study which also included two patients with ARDS in a cohort of 19 ICU patients (Mackenzie and Shin, 1985). In both studies, no control group was included. The clinical significance of these results is therefore questionable. If there are areas of recruitable collapsed alveoli, especially in the early acute stages (Gattinoni et al, 1994), and reduced mucociliary clearance in ARDS (Reynolds, 1987), then theoretically physiotherapy techniques could be of benefit. However, this remains to be determined.

Conclusion

The condition known as ARDS has been researched extensively over the past three decades since it was first described. There is consistent evidence of a multi-aetiological involvement of inflammatory cells and mediators, leading to inflammatory responses, membrane permeability and destruction of endothelial and epithelial lung cells. The mortality of ARDS remains high despite efforts in investigating the optimal therapeutic modalities. Recently, there has been a strong focus on body positioning as a supportive measure to improve arterial oxygenation, and thereby gas exchange, in these patients. The contribution of physiotherapy techniques in these patients remains speculative and awaits to be further investigated with regard to their potentially adverse or beneficial roles.
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