An evaluation of a single chest physiotherapy treatment on mechanically ventilated patients with acute lung injury

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ABSTRACT Background and Purpose. Acute lung injury is a lung pathology that presents frequently on the intensive care unit. Chest physiotherapy, in the form of endotracheal suction, alternate side-lying and manual hyperinflation, is usually given to patients with this condition with the intention of removing retained pulmonary secretions and recruiting collapsed distal lung units. Despite this common practice there is insufficient research on the effects of chest physiotherapy in patients with acute lung injury being ventilated mechanically. The aim of the present study was to further understanding of the effects of three modes of treatment in chest physiotherapy in an acute lung injury patient group. Method. This randomized, controlled trial investigated all mechanically ventilated patients with acute lung injury admitted to the adult intensive care unit at Guy’s and St Thomas’ NHS Trust between August 1996 and July 1997, who matched the inclusion criteria. Patients were randomized into one of three treatment groups: Group 1 (suctioned only); Group 2 (positioned and suctioned); and Group 3 (positioned, manually hyperinflated and suctioned). Baseline and 10, 30 and 60 minutes’ post-treatment data were recorded for dynamic pulmonary compliance, arterial blood gases and haemodynamic variables. Results were analysed by use of an SPSS software package with a repeated-measures analysis of variance (ANOVA). Results. Eighteen patients fitted the inclusion criteria. Significant changes were observed in both PaCO₂ (p = 0.026) and dynamic compliance (p = 0.019) over time for all three groups. The arterial oxygen to fraction of inspired oxygen ratio (PaO₂/FiO₂) did not alter significantly in any of the groups. With respect to other oxygenation parameters, mixed venous oxygen saturation (SvO₂) showed a significant difference between the groups. Heart rate (HR) and systemic blood pressure (BP) showed statistically significant, but not clinically significant differences over time. Conclusions. Patients with acute lung injury are notably complex to nurse and may require protracted physiotherapy intervention, which may take many forms. As de-recruitment was the single most important event that occurred in the present study population, a prescriptive chest physiotherapy approach to treating mechanically ventilated patients with acute lung should be questioned and adapted accordingly.

Key words: acute lung injury, chest physiotherapy, mechanical ventilation, recruitment
INTRODUCTION

Chest physiotherapy is typically given to patients who are ventilated mechanically to aid the clearance of retained pulmonary secretions, to recruit collapsed distal lung units and to optimize the matching of ventilation and perfusion. The definition of ‘chest physiotherapy’ in the context of patients being ventilated mechanically may vary, but in the UK mostly embodies a combination of endotracheal suction, positioning and manual hyperinflation.

Clinical trials have been conducted in which these techniques have been used on mechanically ventilated patients with trauma, post-operative cardiac surgery and acute lobar atelectasis, respectively (Mackenzie et al., 1980; Eales et al., 1995; Stiller et al., 1996). Other clinical trials have focused on manual hyperinflation alone (Patman et al., 1998; McCarren and Chow, 1998; Patman et al., 2000). This has meant that there is insufficient research on the effects of chest physiotherapy in mechanically ventilated patients with acute lung injury.

Considering that acute lung injury is a pathology that presents commonly on the intensive care unit, frequently induced by sepsis and aspiration (Beale et al., 1993; Doyle et al., 1995; Protopapas and McLuckie, 1996), it would be difficult to support the routine use of chest physiotherapy techniques in this patient group when they have not been sufficiently investigated. To the knowledge of the authors, no published trials are available in which the effects of chest physiotherapy on this patient group have been considered. The aim of the present study was to further understand the effects of three modes of physiotherapy (endotracheal suction, positioning and manual hyperinflation) that are commonly used in chest physiotherapy in the acute lung injury patient group.

METHOD

Subjects

All mechanically ventilated patients with acute lung injury admitted to the adult intensive care unit at Guy’s Hospital were considered for the trial. Permission to conduct the study was obtained from the local ethics committee and assent was obtained from the nearest relative or next of kin of all the patients. The trial took place between August 1996 and July 1997. Patients had to meet the following inclusion criteria:

- Over 18 years of age.
- A Murray score* between 0 and 2.5 (Table 1).
- Intubated and ventilated mechanically (via an oral endotracheal tube) on pressure support ventilation via a Siemens Servo Elema 900 C ventilator.
- Adequately sedated on a combination of benzodiazepines and opioids.
- Haemodynamically stable, with a mean arterial pressure >60 mmHg and no acute cardiac dysrhythmias.
- Have an intra-arterial catheter and pulmonary artery catheter connected to a Hewlett Packard monitor (model 66S). The indwelling pulmonary artery catheter was also connected to a mixed venous oxygen saturation (SvO₂) monitor (Baxter Vigilance monitor VG S2).

*Barker and Adams* If used with ‘at risk diagnoses’, the Murray score, according to the American/European Consensus Conference Definition (Bernard et al., 1994) has sufficient power to identify acute lung injury and acute respiratory distress syndrome (ARDS).
TABLE 1: The Murray score — components and individual values of the lung injury score

<table>
<thead>
<tr>
<th>Chest roentgenogram score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
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<tbody>
<tr>
<td>No alveolar consolidation</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Alveolar consolidation confined to 1 quadrant</td>
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<tr>
<td>Alveolar consolidation confined to 2 quadrants</td>
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<td>Alveolar consolidation confined to 3 quadrants</td>
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<td>Alveolar consolidation in all 4 quadrants</td>
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<table>
<thead>
<tr>
<th>PaO$_2$:FiO$_2$ (mmHg)</th>
<th>0</th>
<th>1</th>
<th>2</th>
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<tr>
<td>≥300</td>
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<td>225–299</td>
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<td>175–224</td>
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<td>100–174</td>
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<td>&lt;100</td>
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<thead>
<tr>
<th>PEEP (cm H$_2$O)</th>
<th>0</th>
<th>1</th>
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<tr>
<td>≥5</td>
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<td>6–8</td>
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<td>9–11</td>
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<td>12–14</td>
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<td>≥15</td>
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<table>
<thead>
<tr>
<th>Compliance (ml/cm H$_2$O)</th>
<th>0</th>
<th>1</th>
<th>2</th>
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<td>≥80</td>
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<td>60–79</td>
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<td>40–59</td>
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<td>20–39</td>
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<td>≥15</td>
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The final value is obtained by dividing the aggregate sum by the number of components that were used: No lung injury, score = 0; Mild to moderate lung injury, score = 0.1–2.5; Severe lung injury (ARDS), score >2.5. (Murray et al., 1988). PaO$_2$:FiO$_2$ = arterial oxygen to fraction of inspired oxygen ratio; PEEP = positive end expiratory pressure.

**Procedure**

Concealed, opaque envelopes were used to randomly allocate all patients to one of three treatment groups:

- **Group 1**: Patients were positioned in the 30° head-up, supine position. They were then pre-oxygenated to a fraction of inspired oxygen (FiO$_2$) of 1.0 for three minutes and received endotracheal suction.

- **Group 2**: Patients were positioned and pre-oxygenated to an FiO$_2$ of 1.0 for three minutes as for Group 1. The patients were then positioned in both the left and right lateral decubitus positions with head of bed at 0° elevation and received endotracheal suction in these positions.

- **Group 3**: Patients were treated as for Group 2, with the addition of six manual hyperinflation breaths before suctioning, by use of the Mapleson C system on an oxygen flow rate of 15 l/min. A Wright’s spirometer was positioned in the circuit of the manual hyperinflation system, in order to ensure that 1.5× the set tidal volume was delivered. The bag was compressed with both hands and an
inspiratory hold of two seconds was applied at the end of inspiration for each breath.

The positions chosen reflect current practice on the intensive care unit at Guy’s and St Thomas’ NHS Trust and were based on understanding the pathology of acute lung injury, where atelectasis occurs in dependent zones. Positioning to change dependency of the lung bases is maximally achieved in the prone position, but this strategy is more commonly reserved for severe lung injured patients (those with acute respiratory distress syndrome). Since acute lung injury is infrequently associated with high secretion load, conventional postural drainage positioning, targeting specific lobes, was not used.

The procedures, as described above, were repeated until the patients were clinically clear of secretions on auscultation. The treatment time was then recorded. The patients in groups 2 and 3 were returned to the 30° head-up, supine position once the procedures had been completed. The same two senior physiotherapists were used to perform the treatment procedures. One operator was consistently used to perform the manual hyperinflation and the other to perform the suction, although both physiotherapists were used to position the patients. Nurses were used as independent observers to record parameters.

The following parameters were recorded:

- Arterial blood gases on an Instrumentation Laboratory 1420 blood gas analyser.
- FiO₂.
- Inspired tidal volume (TV).
- Peak airway pressure (from the Siemens ventilator display panel).
- Positive end expiratory pressure (PEEP).
- Heart rate (HR).
- Systemic blood pressure (BP).
- Pulmonary capillary wedge pressure (PCWP).
- Mixed venous oxygenation saturation (SvO₂).

All indices were measured before treatment with the patient in the 30° head-up supine position to obtain baseline values. Patients were then returned to this position after treatment (if the treatment included a change in position) and monitoring of variables was performed at 10, 30 and 60 minutes after treatment. Transducers were re-zeroed after all changes in position. After 60 minutes the trial was complete and any care that was then deemed necessary in the patients’ management was resumed.

Calculations were made of the PaO₂:FiO₂ ratio, and the dynamic compliance by use of the formula:

\[ C = TV - kP_{\text{PEAK}} - \text{PEEP} \]

where \( C \) = dynamic compliance, \( k \) = compressible volume of ventilator, considered negligible on the Servo 900C (Siemens, 1983) and \( P_{\text{PEAK}} = \) Peak airway pressure. When \( TV \) and \( P_{\text{PEAK}} \) were read, three consecutive recordings were made and the mean value used in the calculation.

Results were analysed by use of the SPSS 6.1 software package. A repeated-measures analysis of variance (ANOVA) with one between-case factor and one within-case factor was also conducted. The behaviour of all the groups together over time was also analysed. For each measured variable the group mean and standard deviation values were calculated by use of the Microsoft Excel 7.0 software, at pre-treatment and then at 10, 30 and 60 minutes post-treatment. Significance was accepted as \( p<0.05 \).
RESULTS

Eighteen patients fitted the inclusion criteria; one was excluded as a result of an asystolic cardiac arrest with successful resuscitation. Table 2 shows demographic data, Murray scores, treatment time, and temperature and ventilation parameters for the 17 patients who completed the trial. Of these 17 patients, six presented with sepsis following laparotomy, five with aspiration pneumonia, four with community acquired pneumonia, one with pancreatitis and one with sepsis from a central line infection.

Oxygenation

SvO₂

SvO₂ did not change significantly over time \((p = 0.106)\) for each group, and did not show a difference between the groups over time \((p = 0.788)\). However, there was a significant difference between the groups \((p = 0.03)\) with Group 2 showing a lower SvO₂. Mean values for groups 1, 2 and 3 at pre-treatment and 10, 30 and 60 minutes post-treatment are shown in Figure 1.

Partial pressure of carbon dioxide in arterial blood \((\text{PaCO}_₂)\)

\(\text{PaCO}_₂\) values showed a significant difference over time \((p = 0.026)\) with increases observed at the 10-minutes post-treatment measurement and returns to near baseline values at the 60-minutes post-treatment measurement. This difference is represented in Figure 2. There was no significant difference between the groups over time \((p = 0.564)\) nor between the groups \((p = 0.780)\).

TABLE 2: Mean \((±SD)\) values describing the study group and total population in terms of demographics

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>73 (±2.6)</td>
<td>70 (±7.4)</td>
<td>70 (±16.3)</td>
<td>70 (±10.9)</td>
</tr>
<tr>
<td>Sex</td>
<td>7 males</td>
<td>10 females</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days in study</td>
<td>2.8 (±2.4)</td>
<td>4.4 (±1.1)</td>
<td>4.1 (±4.5)</td>
<td>3.8 (±3.1)</td>
</tr>
<tr>
<td>Murray scores</td>
<td>1.9 (±0.4)</td>
<td>2.35 (±0.2)</td>
<td>2.14 (±0.2)</td>
<td>2.13 (±0.3)</td>
</tr>
<tr>
<td>Treatment time (min)</td>
<td>4.8 (±2.8)</td>
<td>14.8 (±2.3)</td>
<td>17.4 (±2.1)</td>
<td>12.9 (±5.9)</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>37.2 (±1.1)</td>
<td>37.2 (±1.4)</td>
<td>37.8 (±0.4)</td>
<td>37.4 (±0.4)</td>
</tr>
<tr>
<td>FiO₂ (%)</td>
<td>0.42 (±0.1)</td>
<td>0.47 (±0.1)</td>
<td>0.4 (±0.1)</td>
<td>0.43 (±0.1)</td>
</tr>
<tr>
<td>PEEP (cm H₂O)</td>
<td>8 (±2.0)</td>
<td>8 (±2.5)</td>
<td>8 (±2.2)</td>
<td>8 (±2.2)</td>
</tr>
<tr>
<td>PSV</td>
<td>12 (±6)</td>
<td>17 (±6)</td>
<td>13 (±5)</td>
<td>14 (±3)</td>
</tr>
<tr>
<td>SSS</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>5 (±0.8)</td>
</tr>
</tbody>
</table>

SD = standard deviation; FiO₂ = fraction of inspired oxygen; PEEP = positive end expiratory pressure; PSV = pressure support ventilation; SSS = Sheffield sedation score.
FIGURE 1: Mean values of $\text{SvO}_2$ for groups 1, 2 and 3, showing pre-treatment and 10, 30 and 60 minutes post-treatment observations: $\text{SvO}_2$ (per cent) = mixed venous oxygen saturation.

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-treatment</th>
<th>10</th>
<th>30</th>
<th>60</th>
</tr>
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<tbody>
<tr>
<td>Group 1</td>
<td>74.4</td>
<td>73</td>
<td>73</td>
<td>72.8</td>
</tr>
<tr>
<td>Group 2</td>
<td>62.6</td>
<td>63.2</td>
<td>61</td>
<td>59.6</td>
</tr>
<tr>
<td>Group 3</td>
<td>71.8</td>
<td>73.3</td>
<td>70.2</td>
<td>70.9</td>
</tr>
</tbody>
</table>

FIGURE 2: Mean values of $\text{PaCO}_2$ (mmHg) for groups 1, 2 and 3, showing pre-treatment and 10, 30 and 60 minutes’ post-treatment observations: $\text{PaCO}_2 = \text{partial pressure of carbon dioxide in arterial blood.}$

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-treatment</th>
<th>10</th>
<th>30</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>44.43</td>
<td>44.69</td>
<td>43.412</td>
<td>42.378</td>
</tr>
<tr>
<td>Group 2</td>
<td>45.63</td>
<td>48.138</td>
<td>47.89</td>
<td>46.436</td>
</tr>
<tr>
<td>Group 3</td>
<td>46.9</td>
<td>49.4</td>
<td>47.9</td>
<td>47.3</td>
</tr>
</tbody>
</table>
$\text{PaO}_2: \text{FiO}_2$ ratio

No significance was demonstrated in the $\text{PaO}_2: \text{FiO}_2$ ratio over time, for the groups over time and between the groups.

**Dynamic compliance measurement**

Dynamic compliance displayed a significant difference over time ($p = 0.019$) with a decrease observed at the 10-minutes’ post-treatment measurement. No significance was demonstrated between the groups over time ($p = 0.311$). A $p$-value of 0.073 observed for difference between the groups shows that dynamic compliance was not significantly different between the groups (given $p<0.05$), but since 0.073 approximates to 0.05, it may suggest that each group had slightly different compliance — an observation commensurate with a sample not matched at randomization. These results are shown graphically in Figure 3 where mean values are plotted.

**Haemodynamic variables**

**Heart rate**

Heart rate displayed a significant difference over time ($p = 0.012$) as well as a difference between the groups over time ($p = 0.035$). However, there was no significant difference in heart rate between the groups ($p = 0.640$). Mean values for groups 1, 2 and 3 at pre-treatment and at 10, 30 and 60 minutes’ post-treatment are plotted in Figure 4.

**Systemic blood pressure**

Systemic blood pressure displayed a significant difference over time ($p = 0.002$) with an initial fall, followed by an increase to above

![FIGURE 3: Mean values of dynamic pulmonary compliance for groups 1, 2 and 3, showing pre-treatment and 10, 30 and 60 minutes’ post-treatment observations: ml/cm H$_2$O = millilitres per centimetres of water.](image-url)
FIGURE 4: Mean values for heart rate for groups 1, 2 and 3, showing pre-treatment and 10, 30 and 60 minutes’ post-treatment observations: HR = heart rate; bpm = beats per minute.

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-treatment</th>
<th>Post-treatment (minutes)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Group 1</td>
<td>85.8</td>
<td>89</td>
</tr>
<tr>
<td>Group 2</td>
<td>97.4</td>
<td>107</td>
</tr>
<tr>
<td>Group 3</td>
<td>97.1</td>
<td>99.7</td>
</tr>
</tbody>
</table>

FIGURE 5: Mean values for mean arterial blood pressure for groups 1, 2 and 3 showing pre-treatment and 10, 30 and 60 minutes’ post-treatment observations: MAP = mean arterial blood pressure; mmHg = millimetres of mercury.

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-treatment</th>
<th>Post-treatment (minutes)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Group 1</td>
<td>76.44</td>
<td>76.76</td>
</tr>
<tr>
<td>Group 2</td>
<td>79.36</td>
<td>72</td>
</tr>
<tr>
<td>Group 3</td>
<td>72.5</td>
<td>68.8</td>
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</tbody>
</table>
baseline values by the 60 minutes’ post-treatment measurement. This is shown in Figure 5 where mean arterial pressures are plotted. No significance was demonstrated between the groups over time \( (p = 0.276) \) or between the groups \( (p = 0.427) \).

**Pulmonary capillary wedge pressure.**

No significance was demonstrated in pulmonary capillary wedge pressure over time, for the groups over time and between the groups.

**DISCUSSION**

The respiratory physiotherapist working on the intensive care unit often encounters patients with acutely injured lungs. The temptation is to treat these patients conventionally using a combination of suction, positioning and manual hyperinflation, the aim being to improve arterial blood gases and pulmonary compliance by the clearance of retained large airway secretions and the recruitment of peripheral lung units. The results of the present study challenge this notion. For the purposes of the present study, we chose not only to emulate a typical treatment pattern of practice but also to quantify the derecruitment that patients with acute lung injury may experience after such physiotherapy interventions. The original research question was not about whether or not chest physiotherapy is useful in the intensive care unit, but to quantify the effects of a single treatment, typically used in the UK. Short-term outcomes were used to assess the effects of the intervention, to monitor patients for longer than an hour, keeping all things constant during this period, is impractical in the intensive care environment where other therapies and interventions have to be carried out. Our outcomes were therefore not positioned on the long-term effects of receiving chest physiotherapy or not. Clearly, this would have involved a very different study design requiring a more homogenous group of patients and would have required many more patients to have sufficient power to show that the physiotherapy alone had any effect on mortality or even ventilator-free days.

Ventilation strategies within the intensive care unit are now directed at optimal recruitment of the injured, non-compliant lung and protecting it from the shear stresses, and thus further damage, induced by continual alveolar collapse and reinflation episodes (Dreyfuss et al., 1988; Amato et al., 1995).

The continual disconnection of the patients from the mechanical ventilation, whether for suction or manual hyperinflation, results in disruption of the PEEP and subsequent de-recruitment (Jonson et al., 1999). Significant changes were observed in both PaCO\(_2\) \( (p = 0.026) \) and dynamic compliance \( (p = 0.019) \) over time for all three groups. At the 10 minutes’ post-treatment measurement PaCO\(_2\) increased, suggesting a decrease in alveolar ventilation that can be explained by loss of recruitment of the injured lung. PaCO\(_2\) values gradually returned to pre-treatment levels over the next hour indicating slow re-recruitment with controlled PEEP. Dynamic compliance measurements demonstrated the same mass effect for the groups over time with no particular differences between the groups, confirming a derecruitment event. Similarly, dynamic compliance measurements slowly returned towards baseline values once the patient was reconnected to the ventilator on controlled PEEP.

Patients in Group 3 responded the same as patients in groups 1 and 2 with respect to dynamic compliance and PaCO\(_2\), which contradicts the expectation of improved recruitment with manual hyperinflation. A
trade-off clearly exists between disconnection with the resultant loss of recruitment and attempting to achieve recruitment with manual hyperinflation.

Manual hyperinflation has been the focus of many clinical trials (Goodnough, 1985; Novak et al., 1987; Eales, 1989; Jones et al., 1992; Eales et al., 1995), however, these trials have all considered the therapeutic effects of self-inflating manual resuscitation bags (MRB). Moreover, a meta-analysis of these studies has suggested that these self-inflating bags may have limited therapeutic value (Barker and Eales, 2000). Common practice in intensive care units within the UK involves the use of oxygen-powered manual resuscitation bags, for example the Mapleson C re-breath systems. There is scant literature on the therapeutic value of these systems. One trial identified investigated the effects of a Mapleson B system on respiratory parameters (Patman et al., 2000). These researchers looked at coronary artery surgery patients who received manual hyperinflation for four minutes within the first four hours of operation. They reported significant increases in lung compliance and PaO₂:FiO₂ ratio, but were unclear about the clinical relevance of these observations.

The present study is unique in that it considered the therapeutic value of the Mapleson C re-breath system, combined with positioning and endotracheal suction in patients with acute lung injury. The role of manual hyperinflation, as used in the present study in the context of acute lung injury, was not convincing. Despite small sample sizes, notoriously a problem in randomized controlled clinical trials (Sackett and Cook, 1993), it would be reasonable to postulate that if manual hyperinflation was a potent ‘recruiter’ in this patient group, its effects may have been detected. A distinction has to be made between the use of manual hyperinflation for recruiting the injured lung and manual hyperinflation to recruit acute lobar collapse, where it is indeed the treatment of choice (Stiller et al., 1996).

Since we postulated that there was de-recruitment in the present study group, it is not surprising to find that the PaO₂:FiO₂ ratio showed no significant improvement in any of the groups. With respect to other oxygenation parameters, SvO₂ showed a significant difference between the groups, with Group 2 having a lower mean value than groups 1 and 3. Considering that the patients in Group 2 had higher lung injury scores (mean Murray score of 2.35 compared with groups 1 and 2, see Table 2), the observations made for SvO₂ are in keeping with a sample of patients who were not matched at randomization. The \( p \) value of 0.073 for dynamic compliance values between the groups further supports this observation. As none of the statistics for ventilation and oxygenation parameters demonstrate significant difference between the groups this observation does not change the overall implication of the study.

Since alterations in pulmonary wedge pressure (PCWP) can occur in side-lying, and thus measuring PCWP in side-lying is not recommended (Cason et al., 1990), measurements were taken with patients in the supine position. There was no significant effect on PCWP in the present study group. It is appreciated that since all the patients had indwelling pulmonary artery catheters, cardiac output measurements could have been made, but at the time that the present study was conducted, continuous cardiac output measurement technology was not available on the intensive care unit at Guy’s and St Thomas’ NHS Trust. Measuring cardiac output by thermodilution techniques, therefore, would have required extra personnel and additional boluses of fluid to be administered, the logistics of
which were not satisfactory at the time the trial was conducted. Haemodynamic variables that were altered during the course of the study were heart rate and systemic blood pressure, which showed significant differences over time with heart rate also showing difference between the groups over time. The latter is not readily explained. Blood pressure changes observed for all groups indicate that manual hyperinflation and position changes caused no additional disturbance than suction alone. The patients in the present study were not paralysed and so the haemodynamic disturbances observed with handling, position changes and coughing on suction are realistic. Physiotherapy and haemodynamic disturbance may well be correlated to some extent with some initial reports of ‘danger’ (Laws and McIntyre, 1969) and later reports of falls in cardiac output associated with manual hyperinflation (Singer et al., 1994). Our data showed that disturbance did indeed occur; however, the clinical relevance of these changes was not significant since patients included in the trial did not experience severe hypotension, hypertension or cardiac dysrhythmias and the responses may well have been attenuated as suggested by Klein et al. (1988) with high sedation levels (mean and standard deviation Sheffield sedation score = 5 (±0.8)). We are of the opinion that despite sedation levels, chest physiotherapy can be practised safely in the intensive care unit so that severe haemodynamic disturbance is avoided.

The traditional physiotherapy approach warrants review in patients with acute lung injury. Adaptations can be made through alterations to suction practice, the exploitation of positioning alone and the judicious use of manual hyperinflation.

Endotracheal suction practice may warrant the use of closed-circuit suction. More importantly, accurate assessment should suggest whether suction is indicated at all. We all appreciate that retained secretions should be cleared, it is the manner and frequency in which they are cleared that becomes an important consideration. Positioning, as a technique, can be a significant variable in clinical trials concerned with pulmonary function (Dean and Ross, 1992). The present trial was not concerned with measurement of the effects of positioning alone, but more with alternate side-lying positioning as a component of conventional physiotherapy management. Alternate side-lying reflects only one approach open to the intensive care unit physiotherapist. Other positioning strategies may form an integral part of the management of patients with acute lung injury or acute respiratory distress syndrome and should be exploited (for example, head-down tilt and prone).

As regards manual hyperinflation, there is a need to further quantify anecdotal clinical evidence for the therapeutic benefit of Mapleson C re-breath systems and the potential for exploring recruitment manoeuvres during physiotherapy treatment. Furthermore, the option not to use this mode of treatment in certain clinical situations rests only with the astute clinician.

It is recognized that the present study has limitations. These relate to the small sample size and that only a single treatment within a series of potential treatments was investigated. This makes it difficult to extrapolate too widely about the findings.

CONCLUSION

Disconnection of patients with acute lung injury from mechanical ventilation during physiotherapy treatment can result in significant derecruitment of the lungs and altered physiology. The use of manual hyperinflation does not appear to override the loss of PEEP and the derecruitment effects. How-
ever, patients with acute lung injury are notably complex to nurse and may require protracted physiotherapy intervention, which may take on many forms, that is, manual hyperinflation may have a role in the later or recovery stages of the acute lung injury. Furthermore, the rehabilitative needs of these patients, and the commensurate physiotherapy input, must not be ignored. The importance of assessing patients individually and then modifying their treatment accordingly cannot be overstated.

Since the present study considered only three techniques, it is not proposed that chest physiotherapy intervention in patients with acute lung injury is not indicated. Rather, a prescriptive, historical chest physiotherapy approach to treating mechanically ventilated patients with acute lung should be questioned in light of the potential for de-recruiting injured lungs.

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