Manual hyperinflation — effects on respiratory parameters

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ABSTRACT

Background and Purpose. Manual hyperinflation (MH) of the lungs is commonly used by physiotherapists in the treatment of intubated mechanically ventilated patients with the aim of increasing alveolar oxygenation, reversing atelectasis or mobilizing pulmonary secretions. However, the efficacy of MH, used in isolation, has not been clearly established. Method. This randomized, controlled trial investigated the effects of MH on lung compliance ($C_l$), the arterial oxygen to fraction of inspired oxygen ratio ($P_{A\text{O}_2}:FIO_2$) and the alveolar–arterial oxygen tension difference ($A-a$)PO$_2$ in 100 medically stable, mechanically ventilated subjects who had undergone coronary artery surgery (CAS). Post-CAS subjects were used for this study as they constitute a large, homogeneous and accessible group. Subjects were randomized to either a control group (non-MH group) or to a treatment group (MH group) which received MH within four hours of surgery. Results. After four minutes of MH there were significant improvements in $C_l$, $P_{A\text{O}_2}:FIO_2$ and ($A-a$)PO$_2$ with values remaining above baseline measures at 60 min post-intervention. The mean improvement in $C_l$ was 6 ml/cmH$_2$O (approximately 15%), 56 mmHg for $P_{A\text{O}_2}:FIO_2$ (approximately 17%) and 29 mmHg for ($A-a$)PO$_2$ (approximately 17%) immediately post-intervention. No significant changes in mean $C_l$, $P_{A\text{O}_2}:FIO_2$, or ($A-a$)PO$_2$ were seen in the non-MH group. Conclusions. MH performed in the stable ventilated patient significantly increased $C_l$ and $P_{A\text{O}_2}:FIO_2$ and decreased ($A-a$)PO$_2$, but the clinical significance of this improvement is unclear. Further investigations are required to validate the findings of this study as well as to determine the therapeutic value of MH on patient outcome.

Key words: lung compliance, manual hyperinflation, oxygenation
INTRODUCTION

Manual hyperinflation (MH), as performed by physiotherapists, was first described by Clement and Hubsch (1968). The technique of MH is also referred to as ‘bag squeezing’ or ‘bagging’ and involves the delivery of a volume of gas to patients’ lungs via a manual resuscitation circuit. MH is commonly used by physiotherapists in the treatment of intubated patients with the aim of increasing alveolar oxygenation, reversing atelectasis or mobilizing pulmonary secretions (Jones, 1997). Despite the widespread use of the technique (Jones et al., 1992a; King and Morrell, 1992; Hodgson et al., 1999) and many studies which have described both beneficial and detrimental effects of MH (Eales, 1989; Eales et al., 1995; Enright, 1992; Goodnough, 1985; Gormezano and Branthwaite, 1972; Jones et al., 1992b; Ntoumenopoulos et al., 1998; Paratz and Burns, 1993; Singer et al., 1994), a standardized regimen for delivery of MH has not been defined. As some studies have reported detrimental effects from MH, such as barotrauma or haemodynamic instability, it is important to investigate the effects of the technique and its ability to achieve desired objectives.

By use of a randomized, controlled trial design and a patient population of medically stable, ventilated post-coronary artery surgery (CAS) patients, the present study investigated whether MH achieved the objective of improving the lung function of intubated patients. Specifically, the effects of MH on lung compliance ($C_L$) and arterial oxygenation were examined.

METHOD

Subjects

One hundred subjects in the surgical intensive care unit (ICU) of a major tertiary hospital (Royal Perth Hospital) who had undergone elective or semi-urgent CAS were randomized to either a control group (non-MH group), or to a treatment group (MH group), which received MH within four hours of surgery. Randomization during pre-operative recruitment was via selection, without replacement, of a group identification tag from a box by an independent person. Post-CAS patients were chosen as subjects for this study as they provided a large homogeneous sample with stable haemodynamic and respiratory parameters at the time of physiotherapy interventions. Further, such patients are predominantly free of significant pre-existing lung pathology or respiratory problems that are likely to influence the measurement of the dependent variables to a significant degree. The Ethics Committees of Royal Perth Hospital (RPH) and Curtin University of Technology granted approval for this trial. Written, informed consent was obtained from subjects before surgery.

Using formulae for sample size determination (Portney and Watkins, 1993), statistical power of 0.9 and an alpha ($\alpha$) of 0.05, a minimum of 36 subjects for each group was necessary to establish significance for a 15% improvement in the dependent variables.
Exclusion criteria

An inability to understand written or spoken English was an exclusion criterion. Any subject whose past medical history included conditions that may have influenced $C_L$, such as severe asthma, severe chronic airflow limitation (CAL) (subjects who recorded less than 60% for the forced expiratory volume in one second [FEV₁] to forced vital capacity (FVC) ratio on pre-operative pulmonary function testing were excluded) or bronchiectasis, was excluded.

Withdrawal criteria

Any subject who had unstable cardiovascular status (systolic blood pressure [SBP] < 100 or > 180 mmHg; or mean arterial pressure [MAP] < 60 or > 110 mmHg), arrhythmias which compromised cardiovascular function, presence of a pneumothorax, excessive blood loss from subcostal catheters (> 100 ml/hour) or high levels of respiratory support (fraction of inspired oxygen [FIO₂] > 0.7 and positive end expiratory pressure [PEEP] > 7.5 cmH₂O) was withdrawn.

Variables and measurements

MH was performed in a standardized manner by one experienced physiotherapist (SP) as follows:

- Subjects were maintained in supine position with the bed flat throughout the measurement period.
- MH was performed with the same FIO₂ as delivered by the ventilator for each subject.
- MH was delivered via a Mapleson B circuit with a 2-l rebreathing bag and Irwin valve. A manometer was incorporated into the MH circuitry to ensure peak airway pressure did not exceed 40 cmH₂O during MH.
- MH was performed with an inspiratory pause of approximately 2–3 seconds and inspiratory:expiratory ratio of approximately 1:2 utilizing a one-handed technique and at a rate of 10–12 breaths/min for a period of 4 min.

Dependent variables

1. Static $C_L$ was measured by use of the formula:

$$\frac{V_T-k}{PIEP-PEEP}$$

where: $V_T =$ tidal volume; $k =$ compressible volume of ventilator; PIEP = peak inspiratory end pressure; PEEP = positive end expiratory pressure (Siemens, 1983).
2. The arterial oxygen to fraction of inspired oxygen ratio \( (P_aO_2:FIO_2) \) was derived from arterial blood gas (ABG) analysis and the \( FIO_2 \). A calibrated Radiometer ABL System 625 (Radiometer Medical, Copenhagen, Denmark) was used for ABG analysis and the \( FIO_2 \) was read from the Servo 300 ventilator digital display (Siemens–Elema, Life Support Systems Division, Sweden).

3. The alveolar–arterial oxygen tension difference \( [(A−a)PO_2] \) was calculated from the formula:

\[
P_AO_2 − P_aO_2
\]

The \( P_AO_2 \) (alveolar oxygen tension) was estimated using the modified alveolar gas equation:

\[
P_AO_2 = (P_B−P_{H_2O}) \times FIO_2 − PaCO_2/R
\]

where \( P_B = \) barometric pressure; \( H_2O = \) water vapour pressure (usually 47 mmHg); \( PaCO_2 = \) arterial carbon dioxide pressure; \( R = \) respiratory exchange ratio (assumed to be 0.8) (Tobin, 1988, p. 1627; Oh, 1997, p. 964).

Values for \( PaO_2 \) and \( PaCO_2 \) were derived from ABG analysis and the \( FIO_2 \) was read from the Servo 300 ventilator digital display (Siemens–Elema, Life Support Systems Division, Sweden).

For the purpose of this study calculations of \( C_L \) were simplified by assuming the end expiratory lung pressure to be the same as \( PEEP \) and thereby excluding any intrinsic \( PEEP \). As subjects were receiving a controlled mode of ventilation (generally synchronized intermittent mandatory ventilation with a respiratory rate of 10 or 12 breaths/min) and were not making any respiratory efforts of their own during the data collection period, it was felt that there should not be any significant limitations on passive exhalation that may have contributed to the development of intrinsic \( PEEP \). As \( V_t \) can vary by a few millilitres from breath to breath, the average of three consecutive breaths was used for the compliance calculations.

**Procedures**

Subjects were studied on one occasion. In an attempt to standardize the collection of dependent variables, all patients who consented to the study were ventilated postoperatively on one particular Servo 300 ventilator until the data collection period had concluded.

Testing commenced approximately 3–4 hours after surgery. Operative and descriptive data were recorded. Subjects were positioned supine with the bed flat and then suctioned via the endotracheal tube (ETT) to ensure no excess pulmonary secretions were present in the central airways, as this may have influenced the dependent variables (Chatila et al., 1995). Subjects then had no changes to their management for 15 min prior to the testing period. Two sets of baseline
measurements of the dependent variables were obtained at five-minute intervals before commencement of MH. At each data collection point, measures of $C_L$ were taken prior to arterial blood sampling.

For subjects assigned to the intervention group, MH was applied. If PEEP of $>5 \text{ cmH}_2\text{O}$ was required, a PEEP valve calibrated to the same PEEP was incorporated into the MH circuitry. Upon completion of MH subjects were reconnected to the ventilator on the same settings as pre-treatment. Measurements of the dependent variables were repeated immediately after MH and then at 5, 10, 20, 30 and 60 min after cessation of MH.

Subjects assigned to the control group had the same measurements of the dependent variables but did not receive MH or other physiotherapy intervention. These subjects were maintained on the same ventilator settings, with no intervention, for the equivalent amount of time (4 min) that the intervention group received MH.

Medication, patient position and ventilatory support were kept constant during the period of measurement. No ETT suctioning was performed during the testing.

**Statistical analyses**

Data were stored on a Microsoft Excel 97 (Microsoft Corporation) spreadsheet and analyses performed using SPSS® 6.1 for Windows™ statistical package. Chi-square tests and t-tests for independent samples were used to investigate the success of the randomization process in achieving two comparable groups. Levene's test was performed to determine if equality of variances between the groups existed. Fisher's $r$ to $z$ correlation and repeated measures analysis of variance (ANOVA) were performed to establish baseline stability of the dependent variables. Repeated measures ANOVA on the baseline data when separated by group were also performed.

A repeated measures ANOVA was then performed to examine differences of dependent variables before and after MH. Repeated measures ANOVA using polynomial contrast (metric = 0, 5, 10, 20, 30, 60) were performed on the post-intervention data in order to investigate the behaviour of the dependent variables over time.

To avoid increased chance of type 1 errors from repeated measures with multiple comparisons, a more stringent level of significance ($p < 0.01$) was chosen.

**RESULTS**

Ninety-four subjects completed the study. Six subjects were withdrawn (all prior to commencement of post-operative data collection): three due to cardiovascular instability, one subject was extubated in the operating room before transfer to ICU, one due to excessive respiratory support requirements ($\text{FiO}_2$ 1.0 and PEEP 7.5 cmH$_2$O) and one subject died intra-operatively. Five of the six subjects withdrawn had been randomized to the MH group.

Pre-operative data are provided in Table 1 and operative and post-operative data are provided in Table 2. With the exception of ‘Age’ no significant differences
between groups were present for demographic and operative variables. Data for the dependent variables are shown in Table 3 and figures 1–3.

No significant differences were present between the baseline measures for the dependent variables, with the exception of the $P_{aO_2}:FIO_2$ ratio ($F_{1,91} = 10.0; p = 0.002$). Observed differences of the mean values of $P_{aO_2}:FIO_2$ for the baseline

<table>
<thead>
<tr>
<th>Variable</th>
<th>MH group (n = 45)</th>
<th>Non-MH group (n = 49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M/F)</td>
<td>36/9</td>
<td>39/10</td>
</tr>
<tr>
<td>Age (years)*</td>
<td>61.0 (9.6)</td>
<td>66.0 (8.8)**</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.4 (4.2)</td>
<td>27.8 (4.5)</td>
</tr>
<tr>
<td>FEV₁ (l)</td>
<td>2.66 (0.7)</td>
<td>2.46 (0.7)</td>
</tr>
<tr>
<td>FEV₁ (% pred)</td>
<td>88.0 (18.0)</td>
<td>89.0 (22.0)</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>3.23 (0.9)</td>
<td>3.07 (0.7)</td>
</tr>
<tr>
<td>FVC (% pred)</td>
<td>85.0 (15.0)</td>
<td>89.0 (19.0)</td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td>82.2 (8.6)</td>
<td>79.7 (8.4)</td>
</tr>
<tr>
<td>Respiratory past medical history***</td>
<td>37 (82.2)</td>
<td>39 (79.6)</td>
</tr>
<tr>
<td>nil</td>
<td>4 (8.9)</td>
<td>5 (10.2)</td>
</tr>
<tr>
<td>asthma</td>
<td>2 (4.4)</td>
<td>2 (4.1)</td>
</tr>
<tr>
<td>other</td>
<td>2 (4.4)</td>
<td>3 (6.1)</td>
</tr>
<tr>
<td>Smoking history***</td>
<td>11 (24.5)</td>
<td>14 (28.6)</td>
</tr>
<tr>
<td>non-smoker</td>
<td>2 (4.4)</td>
<td>3 (6.1)</td>
</tr>
<tr>
<td>current smoker</td>
<td>6 (13.3)</td>
<td>3 (6.1)</td>
</tr>
<tr>
<td>Ex &lt;6/52</td>
<td>26 (57.8)</td>
<td>29 (59.2)</td>
</tr>
<tr>
<td>Chronic sputum production***</td>
<td>4 (8.9)</td>
<td>5 (10.2)</td>
</tr>
<tr>
<td>yes</td>
<td>30 (66.7)</td>
<td>28 (57.1)</td>
</tr>
<tr>
<td>left ventricular function***</td>
<td>12 (26.7)</td>
<td>18 (36.7)</td>
</tr>
<tr>
<td>good</td>
<td>3 (6.7)</td>
<td>3 (6.1)</td>
</tr>
</tbody>
</table>

* Data for age, BMI and lung function are means (SD) and range.
** $p<0.01$.
*** Number of subjects with percentages in parenthesis.
MH = manual hyperinflation; SD = standard deviation; BMI = body mass index; kg = kilogram; m = metre; FEV₁ = forced expiratory volume in one second; l = litre; FVC = forced vital capacity; % = per cent; %pred = percentage of predicted normal value; CAL = chronic airflow limitation; Ex<6/52 = ceased smoking within the last six weeks; Ex>6/52 = ceased smoking over six weeks ago.
measures for both groups (Table 3) were <10 mmHg and would not be considered clinically important. For the variable P O2:FIO2 there were no significant differences between baseline measures for the two groups. A correlation of 0.96 or higher for the baseline measures was found. As a result of these analyses it was considered that stability of the baseline for the dependent variables was present.

Differences were present between the MH and non-MH groups following the intervention period (figures 1–3). Analysis of data from all eight data collection points revealed a significant difference was present and that the two groups behaved differently.

Lung compliance improved markedly immediately post-intervention in the MH group and remained above baseline at 60 min post-intervention while varying very

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**TABLE 2: Operative and post-operative data for the 94 subjects who completed the study**

<table>
<thead>
<tr>
<th>Variable</th>
<th>MH group (n = 45)</th>
<th>Non-MH group (n = 49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean number of grafts*</td>
<td>3.4 (0.8)</td>
<td>3.2 (0.9)</td>
</tr>
<tr>
<td></td>
<td>2–5</td>
<td>1–5</td>
</tr>
<tr>
<td>Conduit used:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LIMA ± SVG</td>
<td>38</td>
<td>45</td>
</tr>
<tr>
<td>SVG only</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>LIMA + radial artery</td>
<td>4</td>
<td>Nil</td>
</tr>
<tr>
<td>BIMA</td>
<td>2</td>
<td>Nil</td>
</tr>
<tr>
<td>Cardiopulmonary bypass time</td>
<td>87.4 (29.4)</td>
<td>90.6 (25.3)</td>
</tr>
<tr>
<td>(min)</td>
<td>21–156</td>
<td>45–153</td>
</tr>
<tr>
<td>Aortic cross clamp time (min)</td>
<td>49.2 (19.3)</td>
<td>51.5 (16.6)</td>
</tr>
<tr>
<td></td>
<td>14–99</td>
<td>21–106</td>
</tr>
<tr>
<td>Post-op (min)</td>
<td>200.6 (41.5)</td>
<td>198.5 (39.0)</td>
</tr>
<tr>
<td></td>
<td>160–450</td>
<td>170–450</td>
</tr>
<tr>
<td>FIO2</td>
<td>0.49 (0.07)</td>
<td>0.49 (0.06)</td>
</tr>
<tr>
<td></td>
<td>0.30–0.85</td>
<td>0.40–0.60</td>
</tr>
<tr>
<td>PEEP**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>nil</td>
<td>16 (35.6)</td>
<td>22 (44.9)</td>
</tr>
<tr>
<td>5 cmH2O</td>
<td>26 (57.8)</td>
<td>24 (49.0)</td>
</tr>
<tr>
<td>other</td>
<td>3 (6.6)</td>
<td>3 (6.1)</td>
</tr>
<tr>
<td>Ventilation (min)***</td>
<td>585</td>
<td>720</td>
</tr>
<tr>
<td></td>
<td>270–2510</td>
<td>300–12855</td>
</tr>
</tbody>
</table>

*Values are means (SD) and range.
**Values are numbers of subjects with percentages in parentheses.
***Values are median and range.
MH = manual hyperinflation; SD = standard deviation; LIMA = left internal mammary artery; SVG = saphenous vein graft; BIMA = bilateral internal mammary artery; min = minute; Post-op = time since admission to ICU from operating room to the time data collection commenced; FIO2 = fraction of inspired oxygen at time of data collection; PEEP = positive end expiratory pressure; cmH2O = centimetres of water; Ventilation = time from admission from theatre to removal of endotracheal tube.
TABLE 3: Descriptive data (mean (SD)) for the dependent variables for the 94 subjects who completed the study

<table>
<thead>
<tr>
<th>Variable/Group</th>
<th>Baseline 1</th>
<th>Baseline 2</th>
<th>Immediate</th>
<th>5</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>C_L (ml/cmH2O)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MH (n = 45)</td>
<td>38.43 (9.02)</td>
<td>38.76 (9.27)</td>
<td>44.72 (15.26)</td>
<td>43.07 (14.13)</td>
<td>42.38 (11.09)</td>
<td>40.62 (10.74)</td>
<td>40.85 (10.65)</td>
<td>40.52 (9.62)</td>
</tr>
<tr>
<td>Non-MH (n = 49)</td>
<td>40.15 (9.38)</td>
<td>41.15 (10.21)</td>
<td>40.99 (11.37)</td>
<td>41.17 (10.57)</td>
<td>41.15 (10.21)</td>
<td>40.94 (10.02)</td>
<td>40.93 (9.64)</td>
<td>41.24 (9.94)</td>
</tr>
<tr>
<td>PaO2:FIO2 (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MH (n = 45)</td>
<td>272.4 (79.9)</td>
<td>275.6 (86.0)</td>
<td>329.7 (79.0)</td>
<td>303.5 (77.9)</td>
<td>298.6 (79.8)</td>
<td>315.4 (80.7)</td>
<td>310.9 (79.8)</td>
<td>315.9 (85.1)</td>
</tr>
<tr>
<td>Non-MH (n = 49)</td>
<td>253.9 (81.3)</td>
<td>261.4 (83.1)</td>
<td>259.7 (79.9)</td>
<td>262.9 (83.2)</td>
<td>268.5 (87.0)</td>
<td>270.5 (82.7)</td>
<td>275.4 (83.9)</td>
<td>288.3 (86.0)</td>
</tr>
<tr>
<td>(A–a)PO2 (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MH (n = 45)</td>
<td>176.8 (62.3)</td>
<td>176.6 (64.6)</td>
<td>147.3 (59.9)</td>
<td>159.8 (51.1)</td>
<td>160.7 (52.0)</td>
<td>154.9 (58.9)</td>
<td>157.3 (58.1)</td>
<td>151.0 (61.3)</td>
</tr>
<tr>
<td>Non-MH (n = 49)</td>
<td>182.8 (60.3)</td>
<td>179.9 (60.8)</td>
<td>180.7 (59.1)</td>
<td>179.3 (58.7)</td>
<td>177.1 (60.4)</td>
<td>176.4 (58.8)</td>
<td>174.3 (59.4)</td>
<td>168.3 (63.0)</td>
</tr>
</tbody>
</table>

MH = manual hyperinflation; min = minutes; C_L = lung compliance; ml = millilitres; cmH2O = centimetres of water; PaO2 = oxygen tension in arterial blood; FIO2 = fraction of inspired oxygen; mmHg = millimetres of Mercury; (A–a)PO2 = alveolar–arterial oxygen tension difference.
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little over time in the non-MH group (Figure 1). Significant differences in $C_L$ were found between groups ($F_{7,92} = 9.21; p < 0.001$). The changes in $C_L$ over time were not significant ($F_{1,92} = 0.19; p = 0.665$). Compared to baseline, a mean improvement of approximately 6 ml/cmH$_2$O (15%) occurred after MH, and this is considered
clinically important. No significant changes in mean $C_L$ were seen in the non-MH group. As outlined in Table 3, considerable variability was evident with all data collection points in both groups for $C_L$ and this may temper any finding for this variable.

Significant differences in $P_{O_2}:FIO_2$ were found between groups (Figure 2) ($F_{7,91} = 10.4; p < 0.001$). Over time the changes in $P_{O_2}:FIO_2$ were not significant ($F_{1,92} = 6.52; p = 0.012$). The mean improvement in $P_{O_2}:FIO_2$ was approximately 56 mmHg (17%) immediately post-intervention in the MH group with improvements over baseline measures being maintained at 60 min. This improvement is considered clinically important. In the non-MH group a gradual improvement in $P_{O_2}:FIO_2$ over time was evident; this improvement is most likely to represent natural recovery following general anaesthesia.

A similar trend was seen with $(A−a)P_o2$ (Figure 3) which improved by a mean of approximately 29 mmHg (17%) immediately following MH, with improvements over baseline measures maintained at 60 min. Significant differences in $(A−a)P_o2$ were found between groups ($F_{7,91} = 11.5; p < 0.001$). The changes with $(A−a)P_o2$ over time were not significant ($F_{1,92} = 3.1; p = 0.082$). These results are considered clinically important. A small but non-significant improvement over time was present in the non-MH group, which once again can be attributed to the natural post-anaesthetic recovery.

For all the variables no significant difference in the way the groups behaved over time was detected suggesting that any variation seen in the variables was not due to the group allocation. It is inferred that any changes seen in the variable are therefore

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**FIGURE 3**: Mean $(A−a)P_o2$ by group: MH = manual hyperinflation; $(A−a)P_o2$ = alveolar–arterial oxygen tension difference; mmHg = millimetres of Mercury; min = minute.
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a result of the intervention provided. In both the MH and the non-MH group significant differences in all variables were apparent over time.

For $C_L$, linear and quadratic trends were present for the MH group but no significant trends were found in the non-MH group. With $(A-a)PO_2$ and $P_{O_2}:FIO_2$ the trend of the changes in the MH group was quadratic in nature but linear in the non-MH group.

Neither group exhibited the same significant trends for any of the variables. As it was determined that group allocation was not a significant factor in how the variable changed over time, the significant differences in how the groups behaved may be attributed to the intervention given to those in the MH group.

**DISCUSSION**

This study is the first to investigate whether MH, used in isolation and performed by physiotherapists, improved $C_L$, $P_{O_2}:FIO_2$ and $(A-a)PO_2$ for a sample of stable, mechanically ventilated subjects. The mean improvement in $C_L$ of approximately 15% (6 ml/cmH$_2$O) seen in this study immediately post-MH is comparable to the increases of 16% (6.5 ml/cmH$_2$O) reported by Jones et al. (1992b) following a treatment regimen which included MH in the treatment of subjects with respiratory failure. However, Jones et al. (1992b) did not report the specific length of time MH was applied. Eales et al. (1995) reported that $C_L$ did not change significantly in subjects post-cardiac surgery following six MH breaths and suction. Differences in the dose of MH applied may be a possible reason for the discrepancies between the findings of Jones et al. (1992b), Eales et al. (1995) and the present study. Similarly, the type of MH circuit, operator technique and operator characteristics, and patient type are likely to have influenced the delivery of MH.

The improvement in $C_L$ in the MH group may have been secondary to recruitment of more functioning alveolar units. The application of MH may utilize intercommunicating channels, or collateral ventilation within the lungs, to facilitate the mobilization of secretions and the recruitment of atelectatic lung units (Anderson et al., 1979), thereby improving FRC. Similarly, delivering an increased $V_T$ via MH may generate adequate transpulmonary pressure gradients to overcome alveolar collapse. Maintenance of this gradient for an appropriate length of time, via an inspiratory hold during MH, may influence the distribution of ventilation (Bindslev et al., 1981; Nunn, 1987), allow time for alveolar inflation or enlargement, as well as the recruitment or unfolding of interdependent collapsed alveoli.

Although a statistically significant change in $C_L$ was detected and the trends of change varied between the MH and non-MH groups, only improvements found in $C_L$ in the MH group immediately post-MH, 5 and 10 minutes post-MH are likely to be considered clinically significant. However, over the entire period of data collection no statistically or clinically significant differences between the groups existed for $C_L$. Perhaps the reason why the improvements in $C_L$ were not sustained following MH is because the subjects were returned to a controlled mode of ventilation with
monotonous $V_T$ and an absence of sighs (Bartlett et al., 1973; Bendixin et al., 1963; Meyers et al., 1975).

Prior to the present study there have been no controlled studies published investigating the effects of MH alone on oxygenation. The observed improvements post-MH in $P_{aO_2 \cdot FIO_2}$ and $(A\cdot a)PO_2$ could be considered clinically important for some subjects, but due to the stringent guarding against type 1 errors (significance accepted at $p < 0.01$) the improving trend in mean values post-MH did not reach significance. This finding is in keeping with the results of a meta-analysis of studies using MH, as part of physiotherapy interventions, in which it was suggested that whilst MH may have augmented patients' ability to match ventilation and perfusion, this was not to a significant degree (Barker, 1994). Three other studies found that MH, as part of a treatment regimen, had no significant effect on $P_{aO_2}$ or $P_{aO_2 \cdot FIO_2}$ in subjects post-cardiac surgery (Gormenzano and Branthwaite 1972; Rhodes, 1987; Eales et al., 1995). In studies investigating the effect of MH on oxygenation for non-CAS populations, Jones et al. (1992b) reported a significant increase in $S_{aO_2}$, of approximately 2%, immediately following MH in subjects with respiratory failure. No studies were identified that report $(A\cdot a)PO_2$ after MH.

The lack of improvement over time in measures of oxygenation following MH could be related to the dose of MH applied as MH was performed for a single period of four minutes. As yet, there is no consensus or guidelines in the literature as to the appropriate dose of MH (in terms of length of time of application, number of repetitions or standardized description of the technique) that is required to achieve improvements in alveolar oxygenation, reverse atelectasis or mobilize pulmonary secretions (Hodgson et al., 1999). Four minutes was chosen for this study as it reflected the clinical application of MH by physiotherapists at the RPH. However, at the RPH it is usual for a number of repetitions of approximately four minutes of MH to be incorporated into a single physiotherapy treatment. Perhaps the lack of clinically important changes seen in this study is a factor of the study design only having a single period of four minutes of MH.

The failure to measure $V_T$ during MH is a further limitation to this study, as the ability to improve $C_v$ and arterial oxygenation by MH may be related to the $V_T$ delivered. Practical considerations prevented the recording of $V_T$ during the MH; however, it can be inferred that as subjects received MH from the same experienced physiotherapist, the delivery of MH to all subjects is likely to have been consistent (Smith et al., 1997).

It should be restated that the post-CAS group was used for this study as it represented a large homogeneous sample with stable respiratory and haemodynamic parameters at the time of testing. The authors do not wish to advocate that MH is an essential part of the management of patients post-CAS. Indeed, previous studies have not shown physiotherapy interventions to be of any additional benefit in preventing post-operative pulmonary complications post-CAS (Jenkins et al., 1989; Stiller et al., 1994; Stiller et al., 1995). It is suggested that this study be replicated in other patient populations to establish if the improvements found in $C_v$ and oxygenation in the post-CAS group are evident in those more likely to receive MH as part of their physiotherapy management.
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It is also important to highlight that this study did not attempt to measure broader patient outcome. Although improvements in $C_L$ and oxygenation were found after MH, the effect on patient outcome from this improvement is unknown. Further investigation into the length of time of application of MH, the number of repetitions of MH, or the provision of a standardized description of the technique that is required to achieve improvements in alveolar oxygenation, reverse atelectasis or mobilize pulmonary secretions would be invaluable to physiotherapists.

CONCLUSION

Manual hyperinflation, applied for four minutes by an experienced physiotherapist, to a large homogeneous group (MH group) of stable mechanically ventilated subjects (post-CAS), resulted in a significant increase in $C_L$. Lung compliance improved markedly immediately post-intervention and remained above baseline measures at 60 min post-intervention (although not significantly), whilst varying very little over time in a similar group of subjects who did not receive MH (non-MH group).

Statistically and clinically important improvements in oxygenation were found immediately post-intervention in the MH group, with $P_{aO_2}:FIO_2$ improving 56 mmHg (approximately 17%) and $(A-a)PO_2$, decreasing 29 mmHg (approximately 17%). Improvements in oxygenation were maintained at 60 min. A small improvement in oxygenation over time was present in the non-MH group, which can be attributed to the natural post-anaesthesia recovery.

It is concluded that MH performed in the stable ventilated patient following CAS significantly increased $C_L$ and $P_{aO_2}:FIO_2$ and decreased $(A-a)PO_2$, but the clinical significance of this improvement on patient outcome is unclear. Further investigations are required to validate the results of this study as well as to determine the therapeutic value of MH on patient outcome.

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