Introduction

Physiotherapy treatment is often used in patients in intensive care (ICU) for the prevention and treatment of pulmonary complications such as sputum retention and atelectasis (Mackenzie et al 1980, Pontpiddan 1985). Ntoumenopoulos et al (2002) demonstrated in critically ill patients that physiotherapy treatment was associated with a reduction in nosocomial pneumonia. In addition, various components have been shown to improve lung and thorax compliance, reduce the volume of retained secretions and improve intrapulmonary shunt (Hodgson et al 2000, Jones et al 1992, Mackenzie et al 1980, Mackenzie and Shin 1985, Ntoumenopoulos and Greenwood 1996). In particular, the short term effects of manual hyperinflation (MHI) using a manual resuscitation bag on pulmonary compliance, resolution of atelectasis and sputum production have been well documented (Hodgson et al 2000, Jones et al 1992, Rothen et al 1993, Stiller et al 1990, 1996 and 2000). A recent study demonstrated that hyperinflation can be performed with equal benefit using a manual resuscitation bag or a ventilator (Berney and Denehy 2002).

As with many other routine ICU interventions, however, this treatment has been associated with an increase in metabolic and cardiovascular demand (Cohen et al 1996, Gormezano and Branthwaite 1972, Harding et al 1993 and 1994, Klein et al 1988, Mathews and Weissman 1990, Swinamer et al 1987, Weissman et al 1984, Weissman and Kemper 1993). In critically ill patients, cellular oxygen consumption (VO₂) is already increased and peripheral oxygen (O₂) extraction capabilities may be impaired (Clarke 1997, Silance et al 1994). Therefore any reduction in delivery or increase in O₂ demand, as may occur with physiotherapy treatment, may predispose the patient to adverse events (Trehacher and Leach 1994).

Previous studies examining the effect of physiotherapy treatment on VO₂ have failed to differentiate the effects of this treatment from patient positioning alone, a procedure that reportedly increases VO₂ by 31% (Swinamer et al 1987). In addition, the aim of these studies was to investigate the administration of medication to suppress cardiovascular and metabolic responses using physiotherapy treatment as a physiological stress. There is, however, no supporting evidence from controlled trials that physiotherapy treatment is physiologically stressful. The purpose of this study was to determine the effect of physiotherapy treatment on VO₂, MAP and CI in stable intubated and ventilated patients. [Berney S and Denehy L (2003): The effect of physiotherapy treatment on oxygen consumption and haemodynamics in patients who are critically ill. Australian Journal of Physiotherapy 49: 99-105]
Methods

Study population This study was approved by The Austin Hospital Ethics and Human Research Committee, and informed consent was obtained from next of kin and from the treating intensive care physician. Using the results from Harding et al 1993 (difference in the means VO₂ = 115 mL/min, mean standard deviation = 78 mL/min) with alpha = 0.05, two tailed paired t-test, for a power > 0.80 the sample size required was 10 subjects. Ten sequential patients who met the inclusion criteria were studied. Patients were included if they a) were intubated and ventilated; b) had a pulmonary artery and arterial line in situ; c) were cardiovascularly stable; and d) would receive physiotherapy as a part of routine care. Patients were considered cardiovascularly stable if they had a MAP > 75mmHg, with a fluctuation of MAP < 15mmHg with position change and a CI > 2.3 L/min/m². Patients were excluded from the trial if a) they had a fraction of inspired oxygen (FiO₂) > 0.6; b) a positive end expiratory pressure > 10cmH₂O; c) pulmonary pathology where lung hyperinflation was contra-indicated; or d) were suffering from neurological conditions which necessitated a resting head up position.

Metabolic computer Oxygen consumption was measured using the Engstrom metabolic computer (EMC) which is a microprocessor based instrument attached to the Engstrom Elvira ventilator⁴. The EMC consists of a microprocessor and oxygen-sensing fuel cell that measures both the inspired and expired fractions of oxygen (FeO₂). The VO₂ is calculated as the difference between the product of the inspiratory volume and FiO₂ and the expiratory volume and FeO₂. Calibration and service of the EMC was performed by the Biomedical Engineering Department at the Austin and Repatriation Medical Centre according to manufacturers’ guidelines for routine service. Prior to each occasion of use, the barometric pressure was checked with the Bureau of Meteorology and programmed into the ventilator. Calibration of the oxygen sensors of the EMC to FiO₂ = 1 and 0.4 gas mixture was also performed. The computer was considered calibrated when the difference between the O₂ sensing fuel cells and the ventilator’s FiO₂ was within 0.3%. The validity and accuracy of measurement of VO₂ by the EMC has been previously established in a laboratory setting against a system developed by Nunn et al (1989) for the absolute calibration of indirect calorimeters, under the condition of mechanical ventilation and increased O₂ concentrations. This same system was utilised to test the reliability of the EMC against two other commercially available systems and demonstrated the lowest relative error in measurement of 1.4% (Makita et al 1990).

Procedure All subjects were randomly allocated to a treatment sequence (Figure 1). The randomisation process involved picking a letter from an otherwise sealed envelope denoting treatment sequence. Treatment A involved physiotherapy treatment which included gravity assisted drainage with the foot of the bed elevated to 35 to 45 degrees from the horizontal plane with the patient lying on the side, ventilator hyperinflation (VHI), and suctioning. The entire duration of physiotherapy treatment was 20 minutes and contained no more than three suction passes.

Baseline 1 = 10 minutes steady state VO₂
Baseline 2 = within 5% VO₂ of Baseline 1
Baseline 3 = within 5% VO₂ of Baseline 2

Figure 1. Flow chart of the experimental procedure.
Oxygen saturation was continuously monitored during treatment using pulse oximetry. Ventilator hyperinflation involved reducing the ventilator's respiratory rate to six, decreasing the inspiratory flow to 20 L/min, incorporating a square wave form and increasing the patient's tidal volume by 100 mL increments until a peak inspiratory pressure of 40 cmH₂O was achieved. Six mechanical breaths were delivered to this pressure and the patient was then returned to the original ventilatory parameters and allowed to rest for a period of 30 seconds when the procedure was repeated. In total, the treatment consisted of six sets of six VHI breaths which were all performed by the same investigator. Pre-oxygenation to FiO₂ = 1 was not performed prior to suctioning, as the measurement of VO₂ becomes unreliable with variation in FiO₂.

Endotracheal suction was performed using size 12 Baxter catheters. Treatment B involved turning the patient onto the side and leaving him or her undisturbed for 20 minutes. Patients were positioned onto the same side for both Treatment A and Treatment B. The position was decided upon by consultation between the radiologist, intensive care physician and treating physiotherapist. If bibasal opacities were present, patients were turned to the side to which they were next due in accordance with their pressure care routine.

**Measurement** Once the treatment sequence was decided, the patients were started on the Elvira ventilator with ventilatory parameters remaining unchanged. The patient was placed in the supine position and allowed to stabilise on the ventilator for a period of one hour prior to the commencement of the data collection. Initial measurement involved recording of minute-by-minute VO₂ whilst the patient lay undisturbed for a period of 10 minutes. This was called Baseline 1. There were no restrictions regarding administration of sedative, analgesic or vasoactive drugs or the provision of essential care throughout this period. Measures of CI were made using the pulmonary artery catheter. Three measures of cardiac output were taken, the first was discarded and the average of the second and third measure was divided by the body surface area of the patient and recorded as the CI. These measures of CI were performed immediately on completion of Baseline 1 by the attending nurse. The patient was then positioned into side lying and another set of CI measures was performed prior to the commencement of the pre-determined phase of intervention. During treatment, minute-by-minute measurements of VO₂ and MAP from an indwelling arterial line were recorded and on completion of treatment, CI was re-measured with the patient in the same side lying position. Patients were then returned to the supine position and lay undisturbed until they recovered to within 5% of the Baseline 1 VO₂, which was called Baseline 2. Once this was achieved, CI was re-measured and subjects were turned onto the same side. Measurement followed the same sequence as previously described and the alternate treatment was performed. When the second phase of the intervention was complete, patients were returned to the
supine position and the length of time required for the patients to return to within 5% of Baseline 2 was recorded, which was called Baseline 3. The same physiotherapist performed each treatment whilst an assistant recorded the data.

**Statistical analysis** The values for VO₂ were averaged for each phase of the study to give a VO₂mean for both physiotherapy treatment and side lying. The VO₂mean was used as it reflects the average metabolic cost of each intervention and has been utilised extensively in previous literature (Cohen et al 1996, Harding et al 1993 and 1994, Klein et al 1988, Swinamer et al 1987, Weissman et al 1984, Weissman and Kemper 1993). Peaks in VO₂ were also measured in order to determine whether any specific components of intervention involved greater metabolic demand and these were presented as a percentage rise in VO₂ compared with corresponding previous baseline measures. The variables VO₂mean, MAP and CI were analysed using two way analysis of variance with two repeated measures. Time to recovery of baseline VO₂ was analysed using a one way analysis of variance. The level of significance was set at \( p < 0.05 \). Data are expressed as mean difference (95% confidence limits for the difference).

**Results**

Nine males and one female fulfilled the study inclusion criteria. The mean age of the patients was 68 years with a range of 54 to 75 years. The mean FiO₂ delivered was 0.47 with a range 0.4 to 0.6. The mean Acute Physiological and Chronic Health Evaluation score (APACHE II) was 17.8 with a range of 10 to 24. The descriptive data for patients are presented in Table 1.

No patients required an increase in inotropic support during the course of the study. Only one patient required a 1 mg bolus dose of Midazolam throughout the testing procedure. This was administered following the turn into side lying, prior to physiotherapy treatment.

There was no significant difference in the increase in VO₂ mean during 20 minutes of undisturbed side lying compared with 20 minutes of physiotherapy treatment from baseline (\( F_{(1,9)} = 2.19, p = 0.17 \)). The mean difference in VO₂ between physiotherapy treatment and side lying was 14.8 (-8.8 to 38.4) mL/min.

During the study, increases in VO₂ sustained for less than one minute were observed in all patients and were classified as transient. In 50% of cases, these occurred as a result of turning from supine into side lying and involved a mean transient increase of 39% in VO₂ (Table 2). In four patients, suction induced a mean 50% transient increase in VO₂, whilst in one patient, physiotherapy treatment produced a 61% increase in VO₂.

Time to recovery to within 5% of baseline VO₂ measured with the patient in the supine position was achieved within seven minutes for all subjects. There was no significant difference in recovery time between side lying or physiotherapy treatment (\( F_{(1,9)} = 0.248, p = 0.63 \)). The mean difference was -0.6 (-1.8 to 3.0) minutes.

There were no significant differences in the cardiovascular parameters CI (\( F_{(1,9)} = 0.65, p = 0.44 \)) or MAP (\( F_{(1,9)} = 0.004, p = 0.95 \)) during physiotherapy treatment compared with undisturbed side lying. The mean difference in CI was 0.20 (-0.4 to 0.4) L/min/m² and the mean difference for MAP was 1 (-2.1 to 4.1) mmHg.

**Discussion**

The benefits of physiotherapy treatment for intubated and ventilated patients have been previously documented (Ciesla 1996, Hodgson et al 2000, Jones et al 1992, Mackenzie et al 1980, Mackenzie and Shin 1985, Mackenzie 1989, Ntoumenopoulos and Greenwood 1996, Stiller et al 1990 and 1996), however there have also been reports of concerning metabolic side effects (Cohen et al 1996, Harding et al 1994, Klein et al 1988, Weissman and Kemper 1993). Because of methodological and statistical inconsistencies, these earlier studies failed to demonstrate conclusively that physiotherapy treatment increases metabolic demand as measured by VO₂ in the ICU. The findings of this study suggest that physiotherapy treatment does not increase the metabolic or cardiovascular demand of the patient population studied in this trial.

The resting VO₂ of the current patient group appeared somewhat higher than that reported in previous literature. It is possible that the current patients were a more severely ill sample. However APACHE II scores were not routinely recorded in earlier studies to allow comparisons (Cohen et al 1994, Harding et al 1993 and 1994, Klein et al 1988, Weissman and Kemper 1993). Patients in this study received relatively low doses of sedative drugs which should not have masked any induced metabolic or cardiovascular responses (Harding et al 1993 and 1994, Klein et al 1988).

The physiotherapy treatment performed in this study lasted

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**Table 2. Activities associated with greatest overall % rise in VO₂ throughout the experimental procedure.**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Activity</th>
<th>%VO₂ rise</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>turning</td>
<td>37</td>
</tr>
<tr>
<td>2</td>
<td>turning</td>
<td>40</td>
</tr>
<tr>
<td>3</td>
<td>turning</td>
<td>63</td>
</tr>
<tr>
<td>4</td>
<td>suction</td>
<td>75</td>
</tr>
<tr>
<td>5</td>
<td>suction</td>
<td>47</td>
</tr>
<tr>
<td>6</td>
<td>turning</td>
<td>18</td>
</tr>
<tr>
<td>7</td>
<td>treatment</td>
<td>61</td>
</tr>
<tr>
<td>8</td>
<td>turning</td>
<td>36</td>
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<td>9</td>
<td>suction</td>
<td>41</td>
</tr>
<tr>
<td>10</td>
<td>suction</td>
<td>40</td>
</tr>
</tbody>
</table>
substantially longer than that reported in previous literature (Cohen et al 1994, Harding et al 1993 and 1994, Klein et al 1988, Weissman and Kemper 1993). One of the key methodological differences between this study and earlier research was that VO2 was allowed to return to baseline levels before proceeding with the next intervention. In allowing this recovery time it was hoped to isolate the effects of turning the patient from that of active physiotherapy treatment and reduce possible carry-over effects. Earlier research reported increases in VO2 of between 31% and 47% (Horiuchi et al 1997, Swinamer et al 1987) associated with repositioning the patient. The current study concurs with these findings, with an increase in VO2 of 39% observed in 50% of the patients upon positioning into side lying. These peaks in VO2 were, however, not sustained and there was no significant increase observed in VO2mean. As previous investigators (Cohen et al 1994, Harding et al 1993 and 1994, Klein et al 1988, Weissman and Kemper 1993) did not allow a recovery period and reported an increase in VO2mean of up to 52% (Weissman and Kemper 1993) it is possible that the multiple patient turns in rapid succession may have contributed to a cumulative rise in VO2mean rather than being an outcome due to physiotherapy treatment per se.

The findings of the current study therefore suggest that the metabolic and cardiovascular effects of turning a patient into a side lying position were greater than the physiological stress incurred by the addition of physiotherapy treatment. These conclusions support the work of Horiuchi et al (1997) who found that the rise in VO2 associated with physiotherapy treatment could be explained by turning the patient into a side lying position.

Endotracheal suctioning produced peaks in VO2 which amounted to a mean 56% increase from baseline. In addition, during physiotherapy treatment six of the 10 patients recorded the highest VO2 during endotracheal suctioning. This was not a surprising result, as the stressful effects of this procedure are well documented (Paratz 1992, Young 1984). It must also be remembered that the patients were suctioned in the gravity-assisted drainage position. This may have further increased the metabolic stress and may have required an increased effort by the patient to cough, given the altered diaphragmatic and abdominal muscle position. Previous studies have failed to separate the effects of physiotherapy treatment and endotracheal suctioning (Cohen et al 1994, Harding et al 1993 and 1994, Klein et al 1988, Weissman and Kemper 1993). Based upon the findings of this study, this may have influenced their conclusions regarding the metabolic demands of physiotherapy treatment. The observed peaks in VO2 associated with suctioning were not sustained. However, repetitive suctioning, which is often required in the clinical situation, may alter this finding. In critically ill patients with copious pulmonary secretions, pre-oxygenation and recovery time between suction passes may be important strategies in minimising the possible metabolic side effects of this procedure. However it must be remembered that, although included in a physiotherapy treatment session, suctioning is part of the routine nursing care of the patient.

The metabolic effects of suctioning should not be attributed to physiotherapy treatment alone.

The metabolic effects of VHI and MHI have not previously been reported. A peak increase in VO2 was observed in one patient during physiotherapy treatment that included VHI. One of the aims of hyperinflation is the mobilisation of pulmonary secretions (Singer et al 1994, Tweed et al 1993, Webber and Pryor 1993). The effect of mobilising secretions often results in the patient coughing at times during the procedure and it was therefore expected that there may be transient increases in VO2 associated with this treatment technique. In addition, even when the patient is at rest in side lying, transitory increases in VO2 occur. This may be a response to anxiety, agitation, anticipation or voices at the bedside (Weissman et al 1984). For these reasons, the clinical relevance of an isolated VO2 peak could be questioned.

There appeared to be no significant adverse cardiovascular effects of the physiotherapy treatment as measured by CI and MAP when applied in this patient population. Neither the CI nor the MAP of the patients studied altered significantly from baseline during either physiotherapy treatment or positioning in side lying. This was of interest as previous literature has reported variable effects of hyperinflation on cardiovascular stability with some investigators reporting a 50% reduction in cardiac output with MHI (Laws and McIntyre 1969) whilst others report an increase in cardiac output (Preussler et al 1988, Stone et al 1989 and 1991). The stability of cardiovascular status observed in this study may have been the result of the inherent differences between VHI and MHI, such as slower incremental rises in tidal volume.

The time to recovery to within 5% of baseline VO2 occurred within seven minutes of completion with either intervention. This time was not significantly different between the side lying phase or the physiotherapy treatment component. This generally supports the findings of previous literature (Cohen et al 1994, Harding et al 1993 and 1994, Klein et al 1988, Weissman and Kemper 1993). Although these authors stated no time frame for this recovery, their findings, and those of the current study, provide evidence that any increase in VO2 induced by physiotherapy treatment (or turning into side lying) is not sustained and is reversible in this patient population.

In this study, both the mean difference in VO2 (14.8 mL/min) and the upper confidence limit (38.4 mL/min) were small. Therefore we are confident that the increase in VO2mean associated with physiotherapy treatment is not clinically relevant (Goodman and Berlin 1994). The results are in contrast with previous research (Cohen et al 1994, Harding et al 1993 and 1994, Klein et al 1988, Mathews and Weissman 1990, Weissman and Kemper 1993), although few of these studies adequately investigated the effects of physiotherapy treatment in isolation. It would also have been preferable to employ MHI rather than VHI, as physiotherapists generally utilise MHI in clinical practice in Australia. However, VHI has been shown to be
as effective in improving short term physiological outcomes as MHI (Berney and Denehy 2002). The use of VHI due to technical reasons in this study is therefore supported.

**Conclusions**

Physiotherapy treatment per se does not appear to have adverse effects on VO₂, CI and MAP in stable critically ill patients. Associated activities such as positional changes or suctioning may be more important, although transient, causes of such changes. These activities are part of routine care and occur independently of the physiotherapy treatment. Knowledge of the aetiology and severity of such changes may be useful in guiding practitioners with the selection of appropriate interventions in the critically ill.

**Footnotes**

(a) Gambro Engstrom AB, Bromma, Sweden.
(b) Hewlett Packard monitoring systems M1046-9001b, HP GmbH, Boedhingen, Germany.
(c) Baxter Health Care Corporation, Edwards Critical Care Division, Irvine, Ca 92714-5686 USA.
(d) Surgicare, Argon, Athens, Texas 75751 USA.

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