

# Physiological responses to the early mobilisation of the intubated, ventilated abdominal surgery patient

Bill Zafiropoulos<sup>1</sup>, Jennifer A Alison<sup>2</sup> and Bredge McCarren<sup>2</sup>

<sup>1</sup>General Intensive Care Unit, Concord Repatriation General Hospital <sup>2</sup>School of Physiotherapy, University of Sydney

The aim of this study was to investigate the effects of mobilisation on respiratory and haemodynamic variables in the intubated, ventilated abdominal surgical patient. Mobilisation was defined as the progression of activity from supine, to sitting over the edge of the bed, standing, walking on the spot for one minute, sitting out of bed initially, and sitting out of bed for 20 minutes. Seventeen patients with age (mean  $\pm$  SD)  $71.4 \pm 7.1$  years satisfied inclusion criteria. Respiratory and haemodynamic parameters were measured in each of the above positions and compared with supine. In the 15 subjects who completed the protocol, standing resulted in significant increases in minute ventilation ( $V_E$ ) from  $15.1 \pm 3.1$  l/min in supine to  $21.3 \pm 3.6$  l/min in standing ( $p < 0.001$ ). The increase in  $V_E$  in standing was achieved by significant increases in tidal volume ( $V_T$ ) from  $712.7 \pm 172.8$  ml to  $883.4 \pm 196.3$  ml ( $p = 0.008$ ) and in respiratory rate ( $f_R$ ) from  $21.4 \pm 5.0$  breaths/min to  $24.9 \pm 4.5$  breaths/min ( $p = 0.03$ ). No further increases were observed in these parameters beyond standing when activity was progressed to walking on the spot for one minute. When supine values were compared with walking on the spot for one minute, inspiratory flow rates ( $V_T/T_I$ ) increased significantly from  $683 \pm 131.8$  ml/sec to  $985.1 \pm 162.3$  ml/sec ( $p = 0.001$ ) with significant increases in rib cage displacement ( $p = 0.001$ ) and no significant increase in abdominal displacement ( $p = 0.23$ ). Arterial blood gases displayed no improvements following mobilisation. Changes in  $V_T$ ,  $f_R$ , and  $V_E$  were largely due to positional changes when moving from supine to standing. [Zafiropoulos B, Alison JA and McCarren B (2004): Physiological responses to the early mobilisation of the intubated, ventilated abdominal surgery patient. *Australian Journal of Physiotherapy* 50: 95–100]

**Key words:** Abdomen, Surgery, Intubation, Mechanical Ventilators, Physiotherapy

## Introduction

Mobilisation in postoperative patients may be defined as low intensity exercise that aims to elicit cardiopulmonary responses which enhance oxygen transport and assist in the reduction of postoperative pulmonary complications (Dean 2002). It is the belief among physiotherapists that mobilisation results in increased tidal volumes which may assist the reversal of atelectasis leading to improved gas exchange (Dean 2002). However the evidence surrounding this remains inconclusive. Scheidegger et al (1976) investigated the effects of early mobilisation in non-intubated patients following a wide range of surgical procedures requiring general anaesthesia. Early mobilisation was shown to improve forced vital capacity, maximum voluntary ventilation, and arterial oxygenation more than breathing exercises alone. These researchers suggested that the changes were the result of increased tidal volumes with mobilisation compared with breathing exercises. This has been challenged by Orfanos et al (1999) who observed that mobilisation resulted in smaller increases in tidal volumes when compared with breathing exercises in non-intubated abdominal surgical patients. Orfanos et al (1999) suggested that patients should be encouraged to perform deep breathing exercises during mobilisation to enable re-expansion of collapsed alveolar lung units. Hence the precise mechanisms by which mobilisation affects lung function are presently theoretical and require further research (Dean 1994, Dean and Ross 1989, Stiller 2000).

Frequently the results from literature examining the use of mobilisation in non-intubated surgical subjects (Chumillas et al 1998, Dull and Dull 1983, Fagevik Olsen et al 1997, Hallbook et al 1984, Jenkins et al 1989, Roukema et al 1998,

Stiller et al 1994) are extrapolated to the intubated patient (Dean 2002, Stiller 2000). However no studies have examined the responses to early mobilisation in intubated patients. Therefore, the aim of this study was to investigate the respiratory and haemodynamic responses to early mobilisation in the intubated, ventilated abdominal surgery patient.

## Method

This observational study with repeated measures was conducted in the general intensive care unit (ICU) at Concord Repatriation General Hospital, Sydney, Australia. Ethical approval was obtained from the Human Ethics Committees of Central Sydney Area Health Service and The University of Sydney.

**Subjects** A convenience sample of subjects requiring a period of intubation and mechanical ventilation following abdominal surgery was recruited for this study during the year 2000. These subjects satisfied a number of inclusion criteria (Table 1). Subjects with planned admissions to the ICU following elective major abdominal surgery were interviewed preoperatively and written consent was obtained. For unplanned emergency admissions consent was obtained postoperatively from the next of kin.

**Procedures and equipment** Data collection occurred in the morning between days 1 and 6 postoperatively (Table 1). Subjects were free from procedures, interventions, ventilatory and cardiovascular support amendments for one hour before execution of the protocol, and had not participated in mobility tasks immediately before the study period. Respirace bands<sup>(a)</sup> were applied to the patient's rib cage and abdomen and calibrated. Rib cage bands were placed 2 cm below the level

of the axilla and the abdominal band was placed below the thoracic cage overlying the umbilicus. Subjects were left in the supine position for 20 minutes on initial mechanical ventilator settings before collection of arterial blood gas samples and collection of all other measures.

The subject's endotracheal tube was then connected to a heated linear pneumotachograph<sup>(b)</sup> which was attached to a standard sterilized Ayres bagging circuit<sup>(c)</sup> with a continuous supply of 100% oxygen at 15 l/min. This was in accordance with the hospital protocol for mobilisation. The pneumotachograph was calibrated using a 3 l syringe prior to each study. The following measurements were taken over a one minute period: a) rib cage and abdomen displacement via the Resptrace; b) tidal volumes ( $V_T$ ), frequency of respiration ( $f_R$ ), minute ventilation ( $V_E$ ), inspiratory time ( $T_I$ ), and expiratory time ( $T_E$ ) via the pneumotachograph; c) heart rate, invasive blood pressure including systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, percutaneously measured oxygen saturation ( $S_pO_2\%$ ) recorded from the subject's monitor<sup>(d)</sup> by an independent observer unaware of the aims of the study. Pneumotachograph measurements were recorded for every breath made during each one minute measurement cycle with haemodynamic measurements recorded every 10 seconds within each minute cycle.

Measurements were collected in the following standardised positions as the patient progressed through mobilisation whilst breathing spontaneously on the Ayres circuit: supine, sitting over the side of the bed (by rolling onto the side and pushing up into sitting), standing, walking on the spot for one minute as able (the number of steps taken was recorded), initially after sitting out of bed in a chair. The order of these positions were not randomised as the intention of the study was to record the cumulative changes occurring with the progression of mobilisation. Subjects were provided with some assistance as required to maintain safety. They were left in each of the above positions for 30 seconds before the one minute data collection periods to eliminate artifact associated with positional change. Arterial lines were re-zeroed in each position before recording haemodynamic data.

Following completion of mobilisation, the patient was reattached to the ventilator with initial settings and fraction of inspired oxygen ( $F_{IO_2}$ ) and remained in sitting for 20 minutes in a chair after which a second arterial blood gas sample was taken. The patient was then reattached to the Ayres circuit and left in this position for a 30 second period before a one minute data collection period in which respiratory and haemodynamic parameters were measured. Finally, the patient was reattached to the ventilator and this concluded the measurement period. Termination of the study occurred if the following were observed: mean arterial blood pressure < 60 or mean arterial blood pressure > 120mmHg, ST segment depression on electrocardiogram (ECG) with anginal chest pain,  $f_R > 35$  breaths per minute,  $S_pO_2\% < 90\%$ .

**Statistical analysis** Data were analysed using Statistica version 6 for Windows 1998. Parametric data that were normally distributed (analysed via Shapiro-Wilks W test) were summarised as means and standard deviations (SD). Non-normally distributed data were summarised as medians and interquartile ranges. A significance level of  $p < 0.05$  was chosen. Mobilisation data were analysed with a one way repeated measures analysis of variance (ANOVA). Statistically significant changes were further analysed with

**Table 1.** Preoperative and postoperative selection criteria.

Preoperative subject exclusion criteria	
•	Evidence of neurological compromise e.g. stroke or hemiplegia
•	Unstable coronary artery disease, recent angina
•	Not independently mobile (with or without walking aids) before the surgical procedure
•	Evidence of severe respiratory compromise (e.g. severe end stage COPD with $FEV_1 < 40\%$ of predicted, interstitial pulmonary fibrosis, bronchiectasis), or previous lung surgery such as pneumonectomy, lobectomy
Postoperative subject inclusion criteria	
•	Intubated and ventilated after major abdominal surgery
•	Able to understand and follow commands appropriately
•	Less than or equal to 12.5 cmH <sub>2</sub> O of CPAP (as per ICU protocol for disconnection from mechanical ventilation)
•	Haemodynamically stable postoperatively not requiring inotropes
•	No postoperative evidence of recent angina pectoris or recent myocardial infarct (no ST or T wave changes on ECG)
•	No evidence of a motor block from an epidural that would affect the subject's ability to mobilise (i.e. subjects had to be able to actively lift each lower limb off the bed whilst in supine)
•	One hour must have elapsed after the administration of a bolus of local anaesthetic for pain relief with no evidence of haemodynamic compromise
•	No physiotherapy intervention for one hour before data collection

COPD = chronic obstructive pulmonary disease,  $FEV_1$  = forced expiratory volume in 1 second, CPAP = continuous positive airway pressure, ICU = intensive care unit, ECG = electrocardiograph.

post hoc Scheffé tests. Changes in arterial blood gases and arterial oxygen saturation were analysed for significance with dependent samples t tests.

## Results

Twenty-one subjects were invited to participate in this study. Two potential subjects declined the invitation to participate and a further two subjects failed to meet the inclusion criteria by returning to the ICU extubated. Seventeen subjects (8 males and 9 females) with age  $71.4 \pm 7.1$  years entered the study. Subjects had undergone a range of major abdominal surgical procedures including open abdominal aortic aneurysm repair, aortobifemoral bypass, hemicolectomy, Hartmann's procedure, gastrectomy, and major hernia repair. Twelve of the 17 subjects had a previous history of chronic obstructive pulmonary disease. One subject presented with a previous history of obstructive sleep apnoea, while another subject had a previous history of mild asthma. Three subjects had no previous respiratory history.

All subjects required intubation and mechanical ventilation after surgery either routinely or due to the development of respiratory or medical complications. Subjects were on a combination of pressure support (ranging from 0–10 cmH<sub>2</sub>O) and continuous positive airway pressure (ranging from 5–12.5 cmH<sub>2</sub>O) with an  $F_{IO_2}$  of 0.3–0.4. The mean number of days of

**Table 2.** The effect of mobilisation on respiratory parameters (n = 15). Data are means (SDs).

	Supine	Sit	Stand	WOS	SOOBI	SOOB20
$V_T$ (ml)	712.7 (172.8)	826.8 (196.3)	883.4** (196.3)	904.3 (221.6)	873.1 (164.3)	710.0+ (202.0)
$f_R$ (breaths/min)	21.4 (5.0)	24.3 (4.5)	24.9* (4.5)	26.8 (5.1)	26.1 (5.3)	20.3** (4.5)
$V_E$ (l/min)	15.1 (3.3)	19.6 (3.1)	21.3** (3.6)	22.8 (4.5)	22.2 (5.0)	13.8** (3.5)
$T_I/T_{TOT}$	0.36 (0.04)	0.38 (0.04)	0.38 (0.05)	0.39 (0.03)	0.38 (0.03)	0.35 (0.04)
$T_I$ (s)	1.06 (0.27)	0.98 (0.20)	0.97 (0.18)	0.92 (0.16)	0.92 (0.18)	1.08 (0.25)
$T_E$ (s)	1.90 (0.65)	1.61 (0.38)	1.55 (0.30)	1.45 (0.29)	1.52 (0.36)	2.03 (0.57)
$T_{TOT}$ (s)	2.97 (0.86)	2.60 (0.52)	2.54 (0.44)	2.37 (0.43)	2.44 (0.52)	3.08 (0.72)
$V_T/T_I$ (ml/s)	683.0 (131.3)	836.3** (111.4)	919.8** (145.7)	985.1 (162.3)	953.9 (165.3)	668.6** (199.5)
RC disp (cm)	0.42 (0.18)	0.56 (0.28)	0.62* (0.29)	0.65 (0.36)	0.56 (0.28)	0.48*** (0.28)
Abd disp (cm)	0.46 (0.22)	0.49 (0.27)	0.55 (0.27)	0.62 (0.30)	0.52 (0.27)	0.35 (0.21)
RC+Abd disp (cm)	0.85 (0.25)	1.02 (0.42)	1.10** (0.42)	1.17 (0.46)	1.01 (0.44)	0.79** (0.34)

\* $p < 0.05$ , \*\* $p < 0.01$  when compared with supine, + $p < 0.05$ , \*\* $p < 0.01$  when compared with SOOBI, \*\*\* $p < 0.05$  when compared with WOS, SD = standard deviation, WOS = walking on the spot for 1 minute, SOOBI = sitting out of bed initially, SOOB20 = sitting out of bed for 20 minutes,  $V_T$  = tidal volume, ml = millilitres,  $f_R$  = respiratory rate, breaths/min = breaths per minute,  $V_E$  = minute ventilation, l/min = litres per minute,  $T_I/T_{TOT}$  = ratio of inspiratory time versus total inspiratory and expiratory time, s = seconds,  $T_I$  = inspiratory time,  $T_E$  = expiratory time,  $T_{TOT}$  = total inspiratory expiratory time,  $V_T/T_I$  = inspiratory flow rate, RC disp = rib cage displacement, Abd disp = abdominal displacement, RC + Abd disp = total rib cage plus abdominal displacement.

intubation was  $1.9 \pm 1.6$  days, with a median size endotracheal tube of 7.5. Fifteen subjects displayed bilateral postoperative chest X-ray changes of basal atelectasis and consolidation, and two subjects displayed unilateral basal consolidation as documented by the ICU radiologist who was blinded to the aims of the study. Fifteen of the 17 subjects completed the study protocol for mobilisation. Two subjects were excluded based on termination criteria due to the development of haemodynamic instability prior to sitting over the edge of the bed.

**Effect of mobilisation on respiratory parameters** Data were analysed to compare changes from supine values during mobilisation. Mobilisation resulted in a significant increase in  $V_T$ ,  $f_R$  and  $V_E$  when compared with supine in the 15 subjects analysed ( $F_{(5,70)} = 8.75$   $p < 0.001$ ,  $F_{(5,70)} = 14.33$   $p < 0.001$ ,  $F_{(5,70)} = 42.83$   $p < 0.001$  respectively) (Table 2). Standing produced significant increases in  $V_T$  ( $170.7 \pm 161.9$  ml  $p = 0.008$ ),  $f_R$  ( $3.5 \pm 4.1$  breaths per minute  $p = 0.03$ ), and  $V_E$  ( $6.2 \pm 2.9$  l/min  $p < 0.001$ ) when compared with supine. There were no further significant increases in these respiratory parameters with progression of activity to walking on the spot for one minute, or sitting out of bed initially. The number of steps taken whilst subjects walked on the spot for a one minute period ranged from 15–60 steps with a mean of  $30.8 \pm 14.9$  steps.

Mobilisation produced a significant increase in inspiratory flow rates ( $V_T/T_I$ ) when compared with supine ( $F_{(5,70)} = 35.48$   $p < 0.001$ ) (Table 2). Inspiratory flow rates increased by  $153.3$

$\pm 83.7$  ml/sec (24.4%) when subjects sat over the edge of the bed ( $p = 0.001$ ) with further increases in  $V_T/T_I$  of  $148.8$  ml/sec (17.8%) when walking on the spot for one minute was compared with sitting over the edge of the bed ( $p = 0.002$ ). Twenty minutes after sitting out of bed,  $V_T$ ,  $f_R$ ,  $V_E$  and  $V_T/T_I$  decreased significantly to initial baseline compared with sitting out of bed initially ( $p = 0.01$ ,  $p < 0.001$ ,  $p < 0.001$ ,  $p < 0.001$  respectively) (Table 2). Mobilisation did not cause any significant changes in  $S_pO_2\%$  ( $F_{(5,70)} = 1.78$   $p = 0.13$ ).

#### Effect of mobilisation on rib cage and abdominal movement

Mobilisation resulted in a significant increase in rib cage displacement when compared with supine baseline values ( $F_{(5,70)} = 6.03$   $p < 0.001$ ) (Table 2). Rib cage displacement increased by  $0.20 \pm 0.19$  cm (an increase of 63.8%) when the standing position was compared with supine values ( $p = 0.01$ ). No further increases in rib cage displacement occurred with walking on the spot for one minute or sitting out of bed initially. After the subject sat out of bed for 20 minutes, rib cage values decreased significantly when compared with walking on the spot for one minute ( $p = 0.04$ ) (Table 2). There was no significant change in abdominal displacement with mobilisation ( $p = 0.23$ ) (Table 2).

**Effect of mobilisation on arterial blood gases** There were no significant changes in any arterial blood gas parameters 20 minutes after sitting out of bed following mobilisation, compared to the initial resting values. A slight decrease in partial pressure of arterial oxygen ( $P_aO_2$ ) by a mean of  $4.6 \pm 2.7$  mmHg (a mean decrease of 4.0%) was observed

**Table 3.** The effect of mobilisation on arterial blood gas variables (n = 15). Data are means (SDs).

	Before mobilisation	After mobilisation	p value
pH	7.40 (0.06)	7.39 (0.07)	p = 0.11
P <sub>a</sub> O <sub>2</sub> (mmHg)	94.7 (19.8)	90.2 (17.2)	p = 0.11
P <sub>a</sub> CO <sub>2</sub> (mmHg)	37.1 (4.7)	36.9 (5.1)	p = 0.58
Base excess	-1.6 (4.9)	-2.0 (4.8)	p = 0.18
S <sub>a</sub> O <sub>2</sub> %	97.5 (1.1)	97.1 (1.5)	p = 0.27

P<sub>a</sub>O<sub>2</sub> = partial pressure of arterial oxygen, P<sub>a</sub>CO<sub>2</sub> = partial pressure of arterial carbon dioxide, S<sub>a</sub>O<sub>2</sub>% = arterial saturation of oxygen.

**Table 4.** The effect of mobilisation on haemodynamic parameters (n = 15). Data are means (SDs).

	Supine	Sit	Stand	WOS	SOOBI	SOOB20
HR (bpm)	88.6 (12.1)	94.7 (14.8)	98.9** (13.9)	102.1 (14.6)	100.7 (15.1)	91.2** (12.4)
SBP (mmHg)	132.6 (14.8)	151.5** (15.8)	151.1 (18.5)	153.5 (19.8)	154.6 (17.7)	134.1** (14.9)
DBP (mmHg)	66.0 (9.6)	74.8** (8.3)	76.3 (8.4)	73.8 (12.2)	72.6 (9.7)	66.1** (10.1)
MAP (mmHg)	85.9 (12.4)	100.2** (8.5)	101.1 (9.3)	100.3 (12.9)	99.8 (10.7)	88.3** (11.2)

\*p < 0.05 \*\*p < 0.01 when compared with supine, \*\*p < 0.01 when compared with SOOBI, WOS = walking on the spot for 1 minute, SOOBI = sitting out of bed initially, SOOB20 = sitting out of bed for 20 minutes, HR = heart rate, bpm = beats per minute, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure.

compared with initial values, but this failed to reach significance ( $t_{(14)}=1.71$  p = 0.11) (Table 3).

#### **Effect of mobilisation on haemodynamic parameters**

Mobilisation resulted in a significant increase in heart rate and mean arterial blood pressure when compared with supine ( $F_{(5,70)} = 18.53$  p < 0.001,  $F_{(5,70)} = 12.70$  p < 0.001 respectively) (Table 4). Specifically, when compared with supine, heart rate increased by  $10.3 \pm 5.5$  beats per minute when the standing position was reached (p < 0.001) (Table 4), whereas mean arterial blood pressure increased by a mean of  $14.3 \pm 8.8$  mmHg when the sitting position was reached (p < 0.001) (Table 4). The increase in mean arterial blood pressure was achieved by increases in both systolic blood pressure and diastolic blood pressure when the subjects sat over the edge of the bed (p = 0.006 and p = 0.007 respectively). No further significant increases occurred in mean arterial blood pressure beyond these points with further progression of activity. Heart rate and mean arterial blood pressure decreased significantly to baseline supine values when the subjects sat out of bed for 20 minutes when compared with sitting out of bed initially (p < 0.001, p = 0.006 respectively) (Table 4).

#### **Discussion**

Mobilisation, as defined in this study, resulted in significant increases in  $V_T$ ,  $f_R$ , and  $V_E$  when patients moved from supine to standing. No further statistically significant increases were observed in these parameters with walking on the spot for one minute. The progression of activity to walking on the spot for one minute, in this group of patients, favoured an upper chest pattern of breathing with significant increases in  $V_T/T_I$ . Haemodynamic changes were mainly observed as increases in blood pressure when the patients initially sat over the edge

of the bed. No statistically significant changes were observed in arterial blood gas parameters after mobilisation.

#### **Changes in respiratory parameters with mobilisation**

Increases in  $V_T$ ,  $f_R$ ,  $V_E$ , and heart rate appear to be related to positional change as walking on the spot for one minute did not further stimulate increases in these parameters beyond standing. Orfanos et al (1999) found similar results in a non-intubated upper abdominal surgical sample when walking along a corridor was compared with positional change from supine to standing. Self-paced walking on the spot for one minute and corridor walking are short duration activities that offer a low level of exercise intensity which may not be of sufficient magnitude to further stimulate ventilation beyond standing. Furthermore, it may not be possible to set the pace of walking on the spot for one minute to achieve further increases in  $V_T$ ,  $f_R$ , and heart rate in patients who are intubated and ventilated post major abdominal surgery for a number of reasons. Diaphragmatic inhibition as a consequence of upper abdominal surgery (Chuter et al 1991, Ford et al 1993) may limit further increases in  $V_T$  with exercise. Although pain was not rated or controlled during mobilisation in this study, pain may have limited the pace and intensity at which patients could walk on the spot with a resultant effect on ventilatory responses. Other factors such as impairments in preoperative and postoperative nutritional state, and poor exercise tolerance prior to surgery may affect energy levels and strength, therefore limiting the patient's ability to mobilise (Brooks-Brunn 1995, Catley 1984). Cardiac medications, intraoperative anaesthesia and postoperative sedation may also affect patient alertness as well as inhibit heart rate and blood pressure responses which may further limit patients' ability to walk on the spot (Christensen and Kehlet 1993, Vatner 1978).

In our intubated patient group the increase in  $V_E$  with walking on the spot for one minute was achieved by increases in both  $V_T$  and  $f_R$  (26.9% and 24.9% respectively) when compared with baseline values. Orfanos et al (1999) observed in non-intubated patients that walking along a corridor resulted in an increase in  $V_E$  that was achieved by a 33.0% increase in  $V_T$  and a 14.8% increase in  $f_R$  when compared with baseline. The greater increases in  $f_R$  observed with walking on the spot for one minute in our intubated sample, compared with corridor walking in a non-intubated sample (Orfanos et al 1999), may be for several reasons. First, the greater increases in  $f_R$  may be due to the patients' attempts to compensate for the increased work of spontaneous breathing through an endotracheal tube without ventilatory support (Straus et al 1998, Weissman et al 1986) whilst attempting to exercise. Second, patients with a compromised respiratory state may increase  $f_R$  to maintain and increase  $V_E$  during ambulation as this may be more easily achieved than increases in  $V_T$  (Askanazi et al 1979, Okinaka 1966). Changes in lung and chest wall compliance following upper abdominal surgery mean that greater pressures are required to open collapsed lung units in the basal regions of the lung (Grimby 1974). An upper chest pattern of breathing with greater increases in  $f_R$  than tidal volumes during ambulation may be the most efficient means for the respiratory system to overcome decreases in lung compliance during tidal breathing (Mead 1960). Third, increases in  $f_R$  may be favoured over  $V_T$  (Okinaka 1966), as this may generate less pain over the incision site.

The increases in  $V_E$ ,  $f_R$ ,  $V_T$ , and  $V_T/T_I$  generated by mobilisation in the intubated abdominal surgical patient were not maintained after 20 minutes of sitting out of bed. This finding was also supported by Orfanos et al (1999) who demonstrated no carryover effects in  $V_E$ ,  $f_R$ , or  $V_T$  30 minutes after ambulation along a corridor in a non-intubated upper abdominal surgical group. Our study demonstrated no significant changes in arterial blood gases 20 minutes after sitting out of bed, although there was a minor trend towards a decrease in  $P_aO_2$  which may be within the normal limits of acceptable variation.

**Possible effects of mobilisation on the distribution of ventilation** Mobilisation, as conducted in this study, most likely encouraged a distribution of ventilation to the non-dependent regions of the lungs. Grimby (1974) suggested that  $V_T/T_I$  beyond 200 ml/sec resulted in a progressive increase in the relative ventilation of the non-dependent regions in the upright lung. Our study demonstrated significant increases in rib cage displacement with no significant increases in abdominal displacement, suggesting an upper chest pattern of breathing. The Resptrace, as used in our study, only provided data on rib cage and abdominal displacement independently and did not examine the relationship between rib cage and abdominal movement. Chuter et al (1991) postulated that observable decreases in abdominal compartmental contribution to ventilation after abdominal surgery may be due to pain, abdominal cavity stiffness, and reflex diaphragm inhibition postoperatively which impede diaphragm movement, contributing to basal atelectasis. Therefore, the upper chest pattern of breathing combined with increased  $V_T/T_I$  during mobilisation may result in an increased distribution of ventilation to the non-dependent regions of the upright lung (Roussos et al 1977).

**The effect of mobilisation on haemodynamic parameters** Our study demonstrated significant increases in systolic blood pressure, diastolic blood pressure, and mean arterial

blood pressure when the subjects sat over the side of the bed, with significant increases in heart rate observed with standing. Our results are in contrast to previous studies which have demonstrated slight decreases in blood pressure with increases in heart rate associated with orthostatic challenge (upright positioning) in healthy young and elderly subjects, subjects with cardiac disease (Winslow et al 1995), and non-intubated subjects following lower abdominal surgery (Gelle et al 1971). The increases observed in haemodynamic parameters, in our patient group, may be the result of increased pain experienced during mobilisation. This may further stimulate the postoperative sympathetic stress response observed as a consequence of surgical trauma (Dorman et al 1997). In addition, a portion of the increase in haemodynamic parameters may be attributable to the stress and increased work of breathing associated with the removal of ventilatory support and breathing via an endotracheal tube (Frazier et al 2000, Pinsky 2000) during mobilisation. Two patients in our sample demonstrated mean arterial blood pressure of greater than 120 mmHg before the sitting position was reached, requiring termination of mobilisation. Constant monitoring of the patient is required throughout mobilisation to observe signs of cardiac stress and increases in blood pressure which may affect the surgical anastomoses.

### Clinical implications

Changes in  $V_T$  and  $f_R$  during mobilisation of the intubated, ventilated abdominal surgery patient are largely due to positional change from supine to standing. No further increases were observed in these parameters with walking on the spot for one minute. Mobilisation also produced high  $V_T/T_I$  with an upper chest pattern of breathing which may favour regional ventilation to the non-dependent regions of the upright lung and may not assist the reversal of basal atelectasis. This does not mean that walking on the spot for one minute is not a useful intervention in this patient group. Walking on the spot for one minute may minimise lower limb muscle deconditioning, prevent muscle contractures, or improve orthostatic intolerance (Imle and Klemic 1989).

An appropriate level of exercise intensity in this patient group may be one that achieves increases in  $V_T$  and  $f_R$ , beyond those achieved by positional change and of sufficient magnitude to assist opening of collapsed alveolar lung units. It may be useful to use the modified Borg rate of perceived exertion scale (Asakuma et al 1999) or dyspnoea scale (Mahler 1992) as a means of establishing the intensity of physical exertion for the purpose of exercise prescription in these patients. However it may not be possible to achieve a higher level of exercise intensity than demonstrated in this study for the reasons previously outlined.

This study also demonstrated that increases in haemodynamic parameters tended to be greatest initially when the patient sat over the side of the bed. Mobilisation in this patient population needs to be balanced against any adverse haemodynamic changes. This study demonstrated that mobilisation can be performed safely provided that haemodynamic parameters are monitored continuously for adverse effects.

Further studies are required to investigate the effects of mobilisation on regional ventilation and gas exchange with the patient attached to mechanical ventilation. In addition, the effects of early mobilisation on duration of intubation and ventilation, length of stay in the ICU, and incidence of

postoperative pulmonary complications warrant further investigation.

**Footnotes** <sup>(a)</sup>Respirtrace Systems, Ambulatory Monitoring Inc., Ardsley New York <sup>(b)</sup>Model 3813 Hans Rudolph Inc., Kansas, USA <sup>(c)</sup>Model SRAN 2107-2, Rusch anaesthetic bag, Anaesthetic Supplies Pty Ltd. <sup>(d)</sup>Spacelabs Inc., 90600A Series Monitor Washington USA

**Correspondence** Mr B. Zafiropoulos, Physiotherapy Department, Royal Prince Alfred Hospital, Missenden Rd, Camperdown, NSW, 2050. Email: bzaf@optusnet.com.au

## References

- Asakuma S, Fujiwara M, Ohyanagi M and Iwasaki T (1999): A simple, reliable method of assessing exercise capacity in patients with chronic heart failure. *Medicine and Science in Sports and Exercise* 31: 52–56.
- Askanazi J, Silverberg PA, Hyman AI, Rosenbaum SH, Foster R and Kinney JM (1979): Patterns of ventilation in postoperative and acutely ill patients. *Critical Care Medicine* 7: 41–46.
- Brooks-Brunn JA (1995): Postoperative atelectasis and pneumonia: risk factors. *American Journal of Critical Care* 4: 340–349.
- Catley DM (1984): Postoperative analgesia and respiratory control. *International Anesthesiology Clinics* 22: 95–111.
- Christensen T and Kehlet H (1993): Postoperative fatigue. *World Journal of Surgery* 17: 220–225.
- Chumillas S, Ponce JL, Delango F, Viciano V and Mateu M (1998): Prevention of postoperative pulmonary complications through respiratory rehabilitation: A controlled clinical study. *Archives of Physical Medicine and Rehabilitation* 79: 5–9.
- Chuter TAM, Weissman C and Starker PM (1991): Respiratory patterns after cholecystectomy – effect of posture and CO<sub>2</sub> stimulation. *Chest* 100: 23–27.
- Dean E (1994): Oxygen transport: A physiologically-based conceptual framework for the practice of cardiopulmonary physiotherapy. *Physiotherapy* 80: 347–355.
- Dean E (2002): The effects of positioning and mobilisation on oxygen transport. In Pryor JA and Prasad SA (Eds): *Physiotherapy for Respiratory and Cardiac Problems: Adults and Pediatrics* (3rd ed.) Edinburgh: Churchill Livingstone, pp. 121–136.
- Dean E and Ross J (1989): Integrating physiological principles into the comprehensive management of cardiopulmonary dysfunction. *Physical Therapy* 69: 255–259.
- Dorman T, Clarkson K, Rosenfeld BA, Shanholtz C, Lipsett PA and Breslow MJ (1997): Effect of clonidine on prolonged postoperative sympathetic response. *Critical Care Medicine* 25: 1147–1152.
- Dull JL and Dull WL (1983): Are maximal breathing exercises or incentive spirometry better than early mobilisation after cardiopulmonary bypass? *Physical Therapy* 63: 655–659.
- Fagevik Olsen M, Hahn I, Nordgren S, Lonroth H and Lundholm K (1997): Randomised controlled trial of prophylactic chest physiotherapy in major abdominal surgery. *British Journal of Surgery* 84: 1535–1538.
- Ford GT, Rosenthal TW, Clergue F, and Whitelaw WA (1993): Respiratory physiology in upper abdominal surgery. *Clinics in Chest Medicine* 14: 237–252.
- Frazier SK, Stone KS, Schertel ER, Moser DK and Pratt JW (2000): A comparison of hemodynamic changes during the transition from mechanical ventilation to T-piece, pressure support, and continuous positive airway pressure in canines. *Biological Research for Nursing* 1: 253–264.
- Gelle P, Crepin G and Theillier L (1971): Dihydroergotamine in the prevention and treatment of post-operative orthostatic hypotension in gynaecology and obstetrics. *Lille Medical* 16: 631–637.
- Grimby G (1974): Aspects of lung expansion in relation to pulmonary physiotherapy. *American Review of Respiratory Disease* 110: 145–153.
- Hallbook T, Lindblad B, Lindroth B and Wolff T (1984): Prophylaxis against pulmonary complications in patients undergoing gall bladder surgery: a comparison between early mobilisation, physiotherapy with and without bronchodilatation. *Annales Chirurgiae et Gynaecologiae* 73: 55–58.
- Imle PC and Klemic N (1989): Changes with immobility and methods of mobilisation. In Mackenzie CF, Imle PC and Ciesla N (Eds): *Chest Physiotherapy in the Intensive Care Unit* (2nd Ed.) London: Williams and Wilkins, pp. 188–214.
- Jenkins SC, Soutar SA, Loukota JM, Johnson LC and Moxham J (1989): Physiotherapy after coronary artery surgery: Are breathing exercises necessary? *Thorax* 44: 634–639.
- Mahler DA (1992): The measurement of dyspnoea during exercise in patients with lung disease. *Chest* 101: 242S–247S.
- Mead J (1960): Control of respiratory frequency. *Journal of Applied Physiology* 15: 325–336.
- Okinaka AJ (1966): Postoperative pattern of breathing and compliance. *Archives of Surgery* 92: 887–891.
- Orfanos P, Ellis E and Johnston C (1999): Effects of deep breathing exercises and ambulation on pattern of ventilation in post-operative patients. *Australian Journal of Physiotherapy* 45: 173–182.
- Pinsky MR (2000): Breathing as exercise: The cardiovascular response to weaning from mechanical ventilation. *Intensive Care Medicine* 26: 1164–1166.
- Roukema JA, Carol EJ and Prins JG (1988): The prevention of pulmonary complications after upper abdominal surgery in patients with noncompromised pulmonary status. *Archives of Surgery* 123: 30–34.
- Roussos CS, Fixley M, Genest J, Cosio M, Kelly S, Martin RR and Engel LA (1977): Voluntary factors influencing the distribution of inspired gas. *American Review of Respiratory Disease* 116: 457–467.
- Scheidegger D, Bentz L, Piolino G, Pusterla C and Gigon JP (1976): Influence of early mobilisation on pulmonary function in surgical patients. *European Journal of Intensive Care Medicine* 2: 35–40.
- Stiller K (2000): Physiotherapy in intensive care: Towards an evidence based practice. *Chest* 118: 1801–1813.
- Stiller K, Montarello J, Wallace M, Daff M, Grant R, Jenkins S, Hall B and Yates H (1994): Efficacy of breathing and coughing exercises in the prevention of pulmonary complications after coronary artery surgery. *Chest* 105: 741–747.
- Straus C, Louis B, Isabey D, Lemaire F, Harf A and Brochard L (1998): Contribution of the endotracheal tube and the upper airway to breathing workload. *American Journal of Respiratory and Critical Care Medicine* 157: 23–30.
- Vatner SF (1978): Effects of anesthesia on cardiovascular control mechanisms. *Environmental Health Perspectives* 26: 193–206.
- Weissman C, Askanazi J, Rosenbaum SH, Damask MC, Hyman AI and Kinney JM (1986): Response to tubular airway resistance in normal subjects and postoperative patients. *Anesthesiology* 64: 353–358.
- Winslow EH, Lane LD and Woods RJ (1995): Dangling: A review of relevant physiology, research and practice. *Heart and Lung* 24: 263–272.