

Original Papers

An Investigation of the Early Effects of Manual Lung Hyperinflation in Critically Ill Patients

C. HODGSON*, L. DENEHY†, G. NTOUMENOPOULOS‡, J. SANTAMARIAS§, S. CARROLL**

Intensive Care Units, St Vincent's Hospital and Heidelberg Repatriation Hospital, Melbourne, Victoria

SUMMARY

This prospective within-group multicentre study was designed to assess the safety and short-term effectiveness of manual lung hyperinflation in mechanically ventilated patients. Eighteen patients from the intensive care units of two tertiary institutions were included and acted as their own control. Manual lung hyperinflation treatment involved patient positioning (side-lying), suctioning and manual lung hyperinflation. Side-lying treatment involved patient positioning and suctioning alone. Patients received both treatments on the day of data collection. Results demonstrated significant improvement for static respiratory system compliance ($P=0.001$) with manual lung hyperinflation treatment compared to side-lying treatment. Manual lung hyperinflation treatment also cleared a significantly greater wet weight of sputum ($P=0.039$). There were no differences between manual lung hyperinflation and side-lying treatment for gas exchange (P_{aO_2}/F_{iO_2} and P_aCO_2), mean arterial pressure or heart rate. In conclusion, total static respiratory system compliance and sputum clearance were improved by the addition of manual hyperinflation to a physiotherapy treatment of positioning and suctioning in mechanically ventilated patients without compromise to cardiovascular stability or gas exchange.

Key Words: VENTILATION: hyperinflation, physical therapy, compliance, sputum

Manual hyperinflation (MHI) is a technique which provides a greater than baseline tidal volume to the lungs¹. It is frequently used by physiotherapists in the management of intubated patients. It was first described in 1968 as "bag squeezing"² and consists of a series of larger than normal tidal volumes combined with an inspiratory pause and a rapid release of the resuscitation bag to simulate a cough. Evidence supports the use of MHI for the re-expansion of acute atelectasis^{3,4} and for improving static respiratory system compliance⁵ and oxygenation^{6,7}.

Adverse short-term changes in haemodynamic function associated with MHI have been reported^{8,9}.

The mechanisms for these changes remain controversial. Mean arterial pressure (MAP) may decrease as a result of increased intrathoracic pressure and decreased venous return causing a fall in the cardiac output⁹. Conversely, it may increase as a result of reduced left ventricular afterload, which leads to an increase in cardiac output⁸.

There is also controversy regarding the most appropriate method of application of MHI. The use of a pressure manometer within the bagging circuit to monitor peak airway pressure has been advocated¹⁰. In 1993 Rothen et al⁴ found that inflation to an airway pressure of 40 cmH₂O eliminated all areas of atelectasis in otherwise healthy subjects undergoing general anaesthesia as assessed by computed X-ray tomography.

Consequently, variability of the MHI technique, inclusion of diverse patient diagnoses and the use of concurrent hyperoxygenation confound any examination of the effects of MHI^{3,5,8}. Furthermore, no studies examining the efficacy of MHI in secretion clearance have been reported. The aim of this study was to investigate the acute effects of MHI without hyperoxygenation on MAP, heart rate (HR), total static lung compliance (Cr_s), gas exchange and sputum wet

*B.App.Sc.(Phy), PostGrad.Dip.Physio.(Cardio.), M. Physio., M.A.P.A., Senior Clinician Physiotherapist, Alfred Hospital, Prahran, Victoria.

†B.App.Sc.(Phy), Grad.Dip.Physio.(Cardio.), M.A.P.A., Lecturer, School of Physiotherapy, The University of Melbourne, Victoria.

‡B.Sc., B.App.Sc.(Physio.), Ph.D., Senior Clinician Physiotherapist, The Alfred Hospital, Prahran, Victoria.

§M.D.B.S., F.R.A.C.P., Director of Intensive Care, St Vincent's Hospital, Melbourne, Victoria.

**B. App.Sc.(Physio.), M.Sc., M.A.P.A., Lecturer, School of Physiotherapy, The University of Melbourne, Melbourne, Victoria.

Address for Reprints: Ms C. Hodgson, 63 Eglinton Street, Kew, Vic. 3101.

Accepted for publication on February 21, 2000.

weight in critically ill patients who were mechanically ventilated.

MATERIALS AND METHODS

Patients admitted to the intensive care units (ICU) at St Vincent's and Heidelberg Repatriation Hospitals were enrolled in the study if they were mechanically ventilated had an arterial line in situ, chest X-ray (CXR) changes of lung collapse and/or consolidation and $P_aO_2/F_iO_2 < 350^{11}$. The use of CXR signs of lung collapse/consolidation as entry criteria were selected as it is a common clinical indication for physiotherapy. A staff radiologist reported the CXR findings each morning. The intensivist and treating physiotherapist decided upon inclusion of patients following the morning ward round. Patients were withdrawn from the study if their MAP fell below 60 mmHg during treatment.

Exclusion criteria included acute respiratory distress syndrome, acute pulmonary oedema, acute head injury, MAP < 60 mmHg, peak inspiratory airway pressure > 40 cmH₂O (as recorded from the ventilator), acute bronchospasm, subcutaneous emphysema or presence of an intercostal catheter with a visible air leak. The study was approved by the hospitals' ethics committees. Informed consent from patients or their surrogates was not required as the study procedures were not considered to be different from routine management of these patients by the ethics committees.

Patients were randomly allocated, using cards in unmarked envelopes, to receive either MHI treatment or side-lying (SL) treatment first so that the order of treatment did not confound the results. In general, the patient was positioned so that the more affected lung, as seen on CXR, was upper-most. All patients received two physiotherapy treatments on the day of measurement, one in the morning and one in the afternoon with a minimum of three hours between treatments. Baseline measurements of P_aCO_2 , P_aO_2/F_iO_2 and C_{rs} were performed in the supine position prior to turning the patient¹².

MHI treatment consisted of repositioning the patient into side-lying (bed flat) combined with suctioning of the endotracheal tube (ETT) to clear airway secretions immediately followed by 10 minutes of side-lying alone to allow the patient to settle without interruption. At the end of the 10 minutes, six sets of six MHI breaths were delivered to the patient using a two-litre circuit manual rebreathing bag (Ohmeda Inc, Liberty Corner, NJ, U.S.A.) connected to a flow of gas of 15 l/min at the same F_iO_2 as provided by the mechanical ventilator (calibrated with an oxygen

analyser). A blender (Bird Products Corporation, Palm Springs, U.S.A.) was used in the MHI circuit to provide the same F_iO_2 as the patient's ventilator. A manometer was included in the circuit and patients were manually hyperinflated to a peak airway pressure of 40 cmH₂O with a breath hold of no less than two seconds maintained at the end of the inspiratory phase. Expiration was passive and unobstructed to facilitate expiratory flow with no positive end expiratory pressure applied. Exhaled or inhaled tidal volume and rate of MHI were not measured. Airway suctioning was also performed at the end of the MHI treatment. A closed suction system was used for all patients (Steri-cath, Smiths Industries, Keene NH, U.S.A.) and sputum was collected in a sputum trap (Sherwood Medical, St Louis MO, U.S.A.) connected to the suction catheter. The total duration of the treatment was 20 minutes. Side-lying treatment involved positioning the patient in SL (bed flat) for 20 minutes, the airways were suctioned as described above (immediately after the turn and at the end of treatment) but the treatment did not include MHI or any other manual physiotherapy technique. Hyperoxygenation to 100% was not used in any patient.

Measurements

Total static respiratory system compliance was calculated^{13,14}. In order to measure compliance, mandatory breaths at 8 ml/kg (AC, SIMV) were delivered to patients in the supine position. Measurement of compliance requires zero flow, therefore the C_{rs} was measured using plateau pressure by including an inspiratory pause of two seconds duration into the mandatory breath. Spontaneously breathing patients were changed temporarily to SIMV (8 bpm and tidal volume at 8 ml/kg) for measurement of C_{rs} . There were two different types of ventilators used in the intensive care units throughout the study (Bird 8400ST, Bird Products Corporation, Palm Springs, CA, U.S.A. and Evita 2, Dräger, Lubeck, Germany). The intra-rater reliability of the physiotherapist to determine pressure and volume changes from the mechanical ventilator was assessed prior to commencement of this study. The treating physiotherapist viewed, in random order, 15 identical pairs of video segments showing tidal volume and the corresponding plateau inspiratory pressures. After viewing each separate segment, the therapist recorded the plateau inspiratory pressures. The intra-rater reliability of the 15 pairs of readings of plateau pressure and exhaled tidal volume was assessed using the Intraclass Correlation Coefficient (ICC). Results indicated good therapist intra-rater

reliability, ICC (3,1)=0.91 and minimal variability $Sd_{diff}=1.6 \text{ ml/cmH}_2\text{O}$.

The $P_a\text{O}_2/F_i\text{O}_2$, $P_a\text{CO}_2$ and Crs were measured in the supine position before treatment, immediately after treatment and 20 minutes after treatment. Each patient was treated by the same physiotherapist on both occasions and a second therapist recorded all measurements. The $P_a\text{O}_2/F_i\text{O}_2$ and $P_a\text{CO}_2$ were calculated from arterial blood samples (ABG) taken immediately prior to measurement of Crs with the patient in supine and analysed (CIBA-Corning, Medfield MA, U.S.A.).

For each treatment, sputum was collected in a pre-weighed sputum trap attached to the suction catheter of the closed suction system. On completion of the suction passes, 2 ml of sterile saline were flushed through the suction tubing into the trap to clear any secretions in the catheter. The sputum trap containing the saline and sputum was then taken for measurement. The wet weight of sputum was calculated by subtracting the weight of the trap from the total weight of the trap and secretions obtained.

MAP and HR were read directly from the monitoring equipment (HP Component Monitoring System, Hewlett Packard, Andover MA, U.S.A.) at one minute intervals during each 20 minutes of treatment for both MHI and SL. The arterial transducer was maintained at the level of the zero reference point after the patient was positioned in side-lying to allow accurate monitoring of the MAP. The HR was read from the five lead ECG. The MAP and HR were compared between MHI and SL treatments by taking the mean of the first 10 minutes in side-lying and comparing it to the mean of the second 10 minutes in side-lying, which for MHI treatment included manual hyperinflation.

Data Management

Data are summarized as mean and 95% CI for normally distributed variables and as median and range for non-normally distributed. Sputum wet weight was analysed using t-tests and Wilcoxon to compare groups. Measurements of Crs, gas exchange, HR and MAP were analysed with a two-way, two repeated measures analysis of variance (ANOVA)¹⁵. Significant interactions were then analysed using a paired t-test. The results are expressed as *P* values. The level of significance was $P<0.05$ for all tests.

RESULTS

A convenient sample of 19 mechanically ventilated patients (eight from Heidelberg Repatriation

Hospital and 10 from St Vincent's Hospital) were studied between August 1994 and October 1995. One patient was withdrawn from the study as he was unable to maintain MAP greater than 60 mmHg while positioned in side-lying. The demographic details for the 18 remaining patients are given in Table 1. The mean age of the patients was 64.5 years and the patient diagnoses were diverse. In one unit (Repatriation Hospital) seven of the eight patients were ventilated using pressure support ventilation, whilst in the other unit (St Vincent's Hospital) seven of the 10 patients were ventilated with synchronized intermittent mandatory ventilation (SIMV) or assist-control ventilation (AC). During the period of data collection, St Vincent's Hospital had a sixteen-bed combined general and cardiothoracic ICU while Heidelberg Repatriation Hospital had a six-bed general ICU.

Sputum Wet Weight

A significant difference in the sputum wet weight was found between MHI and SL treatments ($P=0.039$). The mean and (95% CI) for sputum wet weight was 5.5 g (2.6,8.5) after MHI and 3.5 g (2.4,4.6) after SL treatment. Upon further examination of sputum data it was found that data was skewed by one very large sputum producer (patient 10). As a result sputum wet weight was analysed again using non-parametric statistics (Wilcoxon signed-rank): results were still found to be significant ($P=0.007$) as shown in Figure 1. The treatment with MHI yielded a mean increase in sputum wet weight of 58.6% compared to side-lying positioning alone. A significant difference in the sputum wet weight was also found between

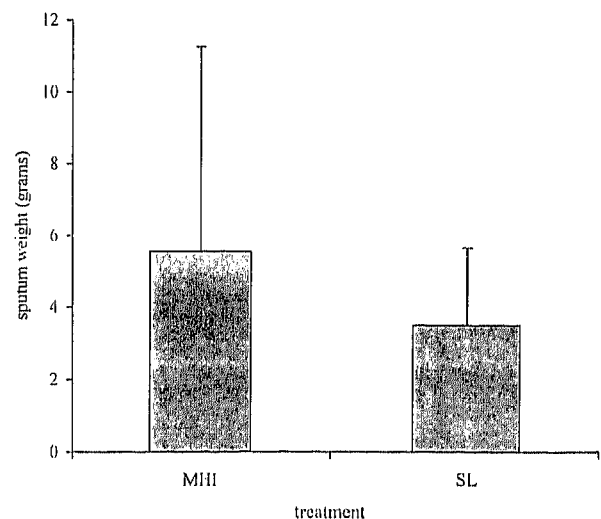


FIGURE 1. Sputum wet weight (mean±SE) comparing MHI and SL treatments.

TABLE 1
Patient details

	Age	Sex	Diagnoses	CXR	Mode of ventilation	Mechanical Rate	Initial TV (ml)	PSV (cmH ₂ O)	PEEP (cmH ₂ O)	F _I O ₂ (%)
1	79	F	Cholecystectomy	(L) LL consol	PSV	0	N/a	10	5	0.3
2	73	M	AAA	Bilat consol	PSV	0	N/a	10	5	0.3
3	72	M	Laparotomy	(R) LL consol	PSV	0	N/a	7	5	0.4
4	78	F	Sepsis	(L) LL coll	PSV	0	N/a	15	5	0.5
5	72	M	Whipples	(L) LL coll	SIMV	6	950	10	5	0.6
6	72	M	AAA	Bilat LL coll	PSV	0	N/a	10	5	0.5
7	72	M	Splenectomy	(L) LL coll	PSV	0	N/a	5	5	0.3
8	60	F	Pancreatitis	(L) LL	PSV	0	N/a	10	5	0.4
9	76	M	MND	Bilat consol	SIMV	10	540	5	5	0.4
10	50	M	Pneumonia	(L) LL	PSV	0	N/a	15	5	0.4
11	48	M	CVA	(R) LL consol	AC	14	630	0	7.5	0.4
12	57	M	Pneumonia	(R) LL consol	PC	20	550	0	5	0.7
13	49	F	AAA	Bilat	SIMV	16	540	10	7	0.4
14	41	M	VATS	(L) LL	SIMV	10	470	0	5	0.4
15	67	M	AAA	(L) LL coll	BIPAP	15	650	20	5	0.4
16	59	M	Pneumonia	Bilat consol	AC	14	600	0	5	0.6
17	62	M	Pneumonia	(R) LL consol	AC	15	500	0	5	0.4
18	75	F	MCA	(R) LL coll	SIMV	10	425	15	5	0.5

F=female; M=male; AAA=abdominal aortic aneurysm; MND=motor neurone disease; CVA=cerebrovascular accident; MCA=motor car accident; (L)=left; (R)=right; LL=lower lobe of the lung; consol=consolidation; coll=collapse; Bilat=bilateral; coll/consol=collapse and consolidation; PSV=pressure support; SIMV=synchronised intermittent mandatory ventilation; AC=assist control ventilation; PC=pressure controlled ventilation; BIPAP=biphasic positive pressure ventilation; TV=tidal volume; N/a=not available; BPM=breaths per minute; cmH₂O=centimetres of water pressure; %=per cent.

MHI and SL treatments ($P=0.009$) by discarding the data of the patient 10 which provided a normal distribution.

Total Static Respiratory System Compliance

There was a significant difference in Crs between the two interventions ($P=0.015$), across time ($P=0.009$) as well as a significant interaction between treatment and time ($P=0.001$) as shown in Figure 2. For up to 20 minutes following treatment with MHI,

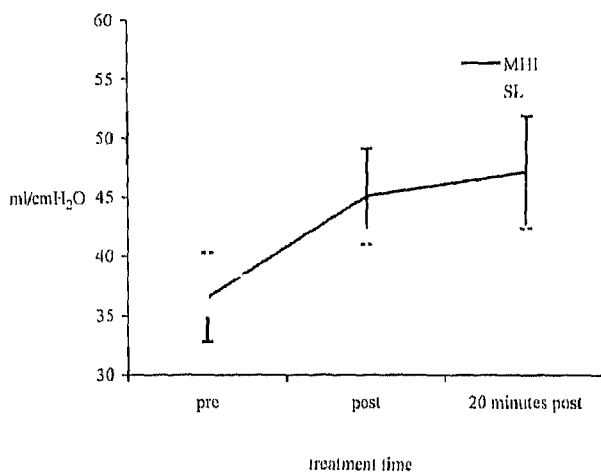


FIGURE 2. Static respiratory system compliance for MHI and SL treatments, measured in supine immediately before treatment, immediately after treatment and twenty minutes after treatment.

Crs was significantly improved compared with positioning in SL. Using the difference between means pre and 20 minutes post MHI treatment the 95% CI were calculated. The mean 95% CI for Crs after MHI was 8.5 ml/cmH₂O (1.4 to 19.6) and after SL treatment was 0.2 ml/cmH₂O (-6.9 to 7.9). Clinically, this improvement in Crs represents a 30% increase in the Crs with treatment that includes manual hyperinflation.

Gas Exchange

There were no significant differences in P_aO₂/F_IO₂ for the two treatments ($P=0.155$). When the P_aO₂/F_IO₂ was analysed to determine if there was a difference between subjects with unilateral or bilateral lung disease on CXR, no significant result was found ($P=0.275$). In addition, the P_aCO₂ was not significantly different between treatments ($P=0.741$).

Haemodynamic Variables

There were no main effects or interactions for haemodynamic variables measured. The mean values of MAP and HR were not significantly different between the two treatment occasions. The median and range of values were also similar, indicating that haemodynamic stability was maintained during the measurement period for all patients.

Table 2 summarizes the mean data for all variables.

TABLE 2
Summary of mean (SE) data for all variables

Variable	MHI			SL		
	pre	post	20 min post	pre	post	20 min post
Crs	36.5 (3.7)	45.0 (4.1)	47.0 (4.7)	38.3 (3.3)	38.6 (3.6)	38.8 (3.6)
P _a O ₂ /F _I O ₂	215.8 (14.8)	198.7 (12.9)	216.0 (21.5)	214.9 (16.5)	216.7 (14.2)	226.4 (18.4)
P _a CO ₂	45.1 (1.7)	46.6 (2.2)	45.4 (2.1)	44.8 (1.6)	45.9 (1.9)	45.4 (2.1)
MAP	88.0 (2.9)	87.9 (3.8)		86.9 (2.3)	82.1 (2.0)	
HR	99.0 (3.9)	98.6 (3.9)		99.0 (3.2)	98.7 (3.2)	
Sputum		5.5 (3.5)			3.5 (2.1)	

Crs=total static respiratory compliance; HR=heart rate; MAP=mean arterial pressure; MHI>manual hyperinflation treatment; P_aCO₂=partial pressure of carbon dioxide; P_aO₂/F_IO₂=ratio of partial pressure of arterial oxygen to fraction of inspired oxygen; SL=side-lying treatment; sputum wet weight.

DISCUSSION

Manual hyperinflation to 40 cmH₂O with an inspiratory pause of two seconds in side-lying in critically ill patients produced significant improvements in Crs and removal of secretions. The improvement in Crs was significant immediately after treatment and persisted for up to 20 minutes. There were no changes in the cardiovascular parameters of MAP and HR or gas exchange measured using P_aO₂/F_IO₂.

The patients studied were representative of a mixed, but mainly surgical ICU population. Ventilatory parameters used reflect current practice in the units. No patient was paralysed. The parameters measured may have responded differently to treatment in a paralysed patient population⁵.

In comparison to other recently published studies on MHI^{3,5}, this study did not incorporate other treatment techniques which may have confounded the results. This is the first study to examine the effect of MHI alone on clearance of secretions. The role of MHI in the clearance of secretions was described in papers as early as 1968², however to date there has been no research to support these early claims. The improved clearance of secretions during MHI and side-lying may be due to the increased expiratory flow rates achieved, causing secretions to be swept proximally in the airways to the carina where they can be removed with suctioning¹⁶. Another explanation may be that by restoring lung volume using MHI, expiratory flow rate may be greater due to a stronger passive elastic recoil of the lungs¹⁷. There may also be potential additional benefits in lowering the head of the bed to the flat position. Additional research may also identify whether MHI in a gravity assisted drainage position improves secretion clearance.

The transport of sputum in the bronchial tree has been described since the 1970s and may be, in part, attributed to two-phase gas-liquid flow^{18,19}, which suggests that the greater the difference in flow between

expiration and inspiration, the better the mechanism of clearing sputum. In this non-ciliary-dependent phasic flow, energy is transmitted from the moving air to the static liquid, shearing and moving the liquid in the direction of flow¹⁸. Therefore, if expiratory flow rate is greater than inspiratory flow rate, sputum may be moved proximally by either mist or annular flow in the airways¹⁷. Further research involving measurement of inspiratory and expiratory flows during MHI are warranted to investigate whether this theory may be used to support the improvement in secretion clearance during treatment with MHI.

Previously MHI has been used in conjunction with chest vibrations to remove secretions from the lungs^{3,5,10}. This study has demonstrated that manual hyperinflation alone is an effective treatment technique to clear lung secretions when used in the side-lying position. Chest wall vibrations have been previously demonstrated to enhance expiratory flow rates in addition to MHI²⁰ and may therefore further improve secretion clearance. Chest wall vibrations and MHI may be even more effective in a head-down position.

Static respiratory system compliance is considered an important clinical outcome measure in ventilated patients and may be useful in predicting mortality in patients with severe respiratory failure¹⁴. Ideally, measurement of static respiratory system compliance is more accurately performed using an oesophageal balloon measures of intrathoracic pressure. However, this is a more invasive, complex and costly procedure²¹. Whilst the Crs measurement in this study may have limitations, it was compared between treatments in the same patient with the same equipment and procedures, reducing error and consequently allowing valid comparisons.

The improvement in Crs found in this study supports previous work by Jones et al⁵ who compared the effects of manual hyperinflation in paralysed

mechanically ventilated patients using a similar within-group study design. Patients in this present study were not paralysed, but lightly sedated, and improvement in Crs was similar to that previously reported⁵. The increase in total static respiratory system compliance with MHI treatment was still present 20 minutes after treatment in the current study and persisted for up to 120 minutes in the study by Jones et al⁵.

The improvement in Crs found in this study may be explained by the recruitment of atelectatic areas of the lung⁴ and the increased removal of secretions from the lungs with MHI. This is the first study to document an improvement in Crs with manual hyperinflation in patients who are ventilated but not paralysed. Clinically, the results of this study represent a 30% increase in Crs with a treatment that includes manual hyperinflation. However, it is important to note that all patients were on PEEP before being detached for MHI, therefore unless PEEP was optimal in terms of the inflection point of the pressure-volume curve at the time of measurement, it is uncertain whether the change in Crs represents a change in the inflection point or a change in the slope of the pressure-volume curve above the inflection point. Also further research to investigate the potential long-term benefits of improved Crs associated with MHI in mechanically ventilated patients is required.

It is important to acknowledge that the main limitation of this study is the small sample size used, and therefore the lack of statistical power to potentially determine any adverse or beneficial effects on gas exchange or haemodynamic function despite the within-subject design.

Most of the previous research has described MHI using hyperoxygenation^{5,8,9}, and therefore the changes in oxygenation due to the technique of MHI alone have not been evaluated. However, Tweed et al⁶ found that MHI with $F_{I}O_2$ set at 0.5 improved oxygenation in anaesthetized surgical patients receiving mechanical ventilation. In this study, oxygenation was measured using alveolar-arterial oxygen difference. The parameter used to measure oxygenation in the current study was $P_aO_2/F_{I}O_2$ and there was no significant difference between the treatments for $P_aO_2/F_{I}O_2$. It may have been expected that $P_aO_2/F_{I}O_2$ would improve with the improvement in Crs. To minimize the impairment in gas exchange associated with acute lung collapse or consolidation, hypoxic pulmonary vasoconstriction diverts blood flow from areas with low partial pressures of oxygen to better ventilated areas. With the re-expansion of collapsed

lung after physiotherapy, increased deadspace may occur initially¹³. Improved perfusion to re-expanded lung areas and therefore improved gas exchange may not have occurred within the 20 minute post-treatment monitoring period in this study. A longer observation period may have detected improved $P_aO_2/F_{I}O_2$ ratios. Several other factors including the loss of PEEP while patients were disconnected from the ventilator circuit and the increase in oxygen consumption during physiotherapy treatment²² may have prevented the improvements in gas exchange. Ventilation-perfusion mismatch associated with patient repositioning for physiotherapy may have occurred especially in patients with bilateral lung disease¹³.

In addition, McCarren and Chow¹ report that patients may be underventilated during MHI as a result of a slow inflation rate reducing minute ventilation (MV). Although MV was not measured in the current study, the P_aCO_2 was measured and values did not alter significantly after either treatment, indicating that minute ventilation remained adequate.

Data for MAP and HR were recorded at one-minute intervals in the side-lying position. Prior to analysis, this data was graphed visually to assess the stability of the cardiovascular responses for each patient. No patient demonstrated large changes in MAP and HR responses. This result is consistent with Singer and co-workers who found no change in MAP and HR with the use of MHI in 18 stable, ventilated patients⁹.

Impaired mucociliary transport occurs frequently in the intubated and mechanically ventilated patient and may be associated with the development of pneumonia²³. Mechanical defences are impaired with intubation and mechanical ventilation, and contribute to the onset of pneumonia (atelectasis, impaired mucociliary clearance). Chest physiotherapy may therefore assist in the management of pneumonia²⁴. Ntoumenopoulos et al²⁴ compared chest physiotherapy (manual lung hyperinflation, postural drainage) to no physiotherapy in a group of intubated and mechanically ventilated trauma patients. Approximately twice as many patients developed ventilator-associated pneumonia in the control group, demonstrating clinically important differences, but these results failed to reach statistical significance.

The physiotherapy treatment administered in this study included MHI to an inspiratory pressure of 40 cmH₂O combined with an inspiratory pause of two seconds. This protocol was based on the work of Haake et al²⁵ and Rothen et al¹ where an inspiratory pressure of 40 cmH₂O was considered both safe and effective in recruiting lung atelectasis in healthy lungs

during anaesthesia. However these authors included a much longer inspiratory pause at the end of inspiration, which may be more deleterious in the acutely ill patient due to the potential effect on the cardiovascular system.

The use of MHI in the treatment of mechanically ventilated patients needs to be further investigated to determine the long-term effects of the technique on gas exchange and Crs. The most effective type of MHI circuit, dosage of treatment and the importance of maintaining PEEP need to be determined. Perhaps most importantly, ventilated patients need to be classified into those who may benefit from MHI and those who may potentially be made worse with the use of this technique. Because MHI involves increasing peak inspiratory pressure, plateau pressure and lung volumes, care in instituting this technique is vital and close monitoring of all parameters is essential.

In conclusion, significant improvements in Crs and removal of secretions occurred in mechanically ventilated patients with the use of MHI without an increase in the $F_{I}O_2$ delivered. There were no adverse effects on parameters of cardiovascular stability or gas exchange when MHI was used to a pressure of 40 cm H_2O with an inspiratory pause of two seconds. Further investigation is required to determine the long-term effects of MHI on mechanically ventilated patients, including the prophylactic use of this modality (prevention of atelectasis especially with low TV ventilation) and the use of MHI with specific respiratory complications, e.g., lung collapse and or consolidation, secretion retention and impaired mucociliary clearance²¹.

ACKNOWLEDGEMENTS

This study could not have been completed without the assistance of the physiotherapy, nursing and medical staff of St Vincent's and Heidelberg Repatriation Hospitals and of Dr Ian Story from the University of Melbourne. Their assistance and support is gratefully acknowledged.

REFERENCES

1. McCarren B, Chow C. Manual hyperinflation: a description of the technique. *Aust J Physiother* 1996; 42:203-208.
2. Clement AJ, Hubsch SK. Chest physiotherapy by the "Bag Squeezing" method. *Physiotherapy* 1968; 54:355-359.
3. Stiller K, Geake T, Taylor J, Grant R, Hall B. Acute lobar atelectasis: A comparison of two chest physiotherapy regimens. *Chest* 1990; 98:1336-1340.
4. Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G. Re-expansion of atelectasis during general anaesthesia: A computed tomography study. *Br J Anaesth* 1993; 71:788-795.
5. Jones AYM, Hutchinson RC, Oh TE. Effects of bagging and percussion on total static compliance of the respiratory system. *Physiotherapy* 1992; 78:661-666.
6. Tweed WA, Phua WT, Chong KY, et al. Tidal volume, lung hyperinflation and arterial oxygenation during general anaesthesia. *Anaesth Intensive Care* 1993; 21:806-810S.
7. Stiller K, Jenkins S, Grant R, Geake T, Taylor J, Hall B. Acute lobar atelectasis: a comparison of five physiotherapy regimens. *Physiother Theory Practice* 1996; 12:197-209.
8. Stone KS, Vorst EC, Lanham B et al. Effects of lung hyperinflation on mean arterial pressure and postsuctioning hypoxemia. *Heart Lung* 1989; 18:377-385.
9. Singer M, Vermaat J, Hall G, et al. Hemodynamic effects of manual hyperinflation in critically ill mechanically ventilated patients. *Chest* 1994; 106:1182-1187.
10. King D, Morrell A. A survey on manual hyperinflation as a physiotherapy technique in intensive care units. *Physiotherapy* 1992; 78:747-750.
11. Gallagher J, Civetta J. Goal-directed therapy of acute respiratory failure. *Anesth Analg* 1980; 59:831-834.
12. Weissman C, Kemper M, Damask MC, et al. Effect of routine intensive care interactions on metabolic rate. *Chest* 1984; 86:1815-188.
13. Mackenzie CF. Physiological changes following chest physiotherapy. In: *Chest Physiotherapy in the Intensive Care Unit*, 2nd Ed. Mackenzie CF, Imle PC, Ciesla N, eds. Williams and Wilkins 1989; 216-242.
14. Mancebo J, Benito S, Martin M, Net A. Value of static pulmonary compliance in predicting mortality in patients with acute respiratory failure. *Intens Care Med* 1988; 14:110-114.
15. Winer BJ. *Statistical principals in experimental design*, 2nd Ed. New York, McGraw-Hill 1971.
16. Jones AYM, Jones RD, Bacon-Shone J. A comparison of expiratory flow rates in two breathing circuits used for manual hyperinflation of the lungs. *Physiotherapy* 1991; 77:593-597.
17. Maxwell L, Ellis E. Secretion clearance by manual hyperinflation: Possible mechanisms. *Physiother Theory Practice* 1998; 14:189-197.
18. Kim CS, Rodriguez CR, Eldridge MA, Sackner MA. Criteria for mucus transport in the airways by two-phase gas-liquid flow mechanism. *J Appl Physiol* 1986; 60:901-907.
19. King M, Gilboa A, Meyer FA, Silberberg A. On the transport of mucus and its rheologic simulants in ciliated systems. *Am Rev Resp Dis* 1992; 110:740-745.
20. MacLean D, Drummond G, Macpherson C, McLaren G, Prescott R. Maximum expiratory airflow during chest physiotherapy on ventilated patients before and after the application of an abdominal binder. *Intens Care Med* 1989; 15:396-399.
21. Georgopoulos D, Mitrouska I, Webster K, Bshouty Z, Younes M. Effects of inspiratory muscle unloading on the response of respiratory motor output to CO_2 . *Am J Respir Crit Care Med* 1997; 155:2000-2009.
22. Weissman C, Kemper M. The oxygen uptake-oxygen delivery relationship during ICU interventions. *Chest* 1991; 99:430-435.
23. Konrad F, Schreiber T, Brecht-Kraus D, Georgieff M. Mucociliary transport in ICU patients. *Chest* 1994; 105:237-241.
24. Ntounenopoulos G, Gild A, Cooper DJ. The effect of manual lung hyperinflation and postural drainage on pulmonary complications in mechanically ventilated trauma patients. *Anaesth Intensive Care* 1998; 26:492-496.
25. Haake R, Schlichtig R, Ulstad D, Henschen R. Barotrauma: Pathophysiology, risk factors and prevention. *Chest* 1987; 91:608-613.