Respiratory and Hemodynamic Effects of Manual Hyperinflation in Mechanically Ventilated Critically Ill Patients

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Citation

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Abstract

<B>Objectives:</B> To evaluate retrospectively the acute effects of manual hyperinflation (MH) on the respiratory and hemodynamic variables in mechanically ventilated critically ill patients. <B>Methods:</B> MH was delivered in 34 medically stable, mechanically ventilated patients with a Mapleson F circuit using a peak inspiratory pressure of 35 cmH2O with an inspiratory pause of 2 - 3 seconds. Baseline, 5 and 30 minutes after MH; heart rate and mean arterial blood pressure as hemodynamic parameters, static pulmonary compliance (Crs), PaO2/FiO2, SpO2, PaCO2 and airway pressures as respiratory parameters were recorded routinely before and after MH.<B>Results:</B> After 5 minutes there were significant improvements in Crs and PaO₂/FiO₂ with values remaining above baseline measures at the 30 minutes post-intervention. PaCO₂ displayed a significant decrease at the 30 minutes post- intervention ($p<0.05$). There were no significant differences in the hemodynamic parameters before and after MH ($p>0.05$).<B>Conclusion:</B> MH performed in the stable ventilated patients significantly increased the Crs and PaO₂/FiO₂ and decreased PaCO₂ without hemodynamic compromise, but the clinical significance of these improvements on patient outcome is unclear.

INTRODUCTION

Ventilation strategies within the intensive care unit (ICU) are now directed at optimal recruitment of the injured, non-compliant lungs and protecting it from the shear stresses, and thus further damage, induced by continual alveolar collapse and reinflation episodes [1]. Manual hyperinflation (MH) is one of a number of techniques which provides a greater than baseline tidal volume to the lungs. It is frequently used by physiotherapists in the treatment of intubated mechanically ventilated patients with the aim of increasing alveolar oxygenation, recruiting atelectasis or mobilizing pulmonary secretions [123456789].

Despite the widespread use of the technique, there is insufficient research on the effects of MH in cardiorespiratory parameters and a standardized regimen for delivery of MH has not been defined in terms of duration of application, number of repetitions or standardized description of the technique [10]. As some studies have reported detrimental effects of MH, such as barotraumas or hemodynamic instability, it is important to investigate the effects of MH and its ability to achieve desired objectives [11].

Therefore, the aim of this study was to evaluate retrospectively the acute effects of MH on the respiratory and hemodynamic variables.

METHODS

SUBJECTS

After approval by the Medical Faculty Clinical Research Ethics Committee, the charts of the 34 medically stable, mechanically ventilated and MH delivered patients at Dokuz Eylül University School of Medicine Research Hospital from June 2000 to May 2002 were reviewed retrospectively. Patients had to meet the following criteria for the treatment of MH: a) over 18 years of age , b) intubated and ventilated mechanically (via an oral endotracheal tube) on pressure controlled (PC) or pressure regulated volume control (PRVC) ventilation via a Siemens Servo Elema 300 ventilator, c) adequately sedated and paralysed on a combination of benzodiazepines and neuromuscular blockers, d) hemodynamically stable, with a mean arterial blood pressure (MABP) >65 mmHg and no acute cardiac

dysrhythmias. Patients were not treated with MH if they: a) required a FiO_2 ≥0.6, b) had a positive end expiratory pressure (PEEP) ≥10 cmH_2O, c) presence of a pneumothorax, d) had an arterial oxygen saturation (SaO_2) ≤90%.

MH was performed in a standardized manner by an experienced physiotherapist as follows: a) Patients were maintained in supine position with the bed flat throughout the intervention period, b) MH was delivered via a Mapleson C circuit. The oxygen flow rate to the Mapleson F circuit was set at 12 L/min. A manometer was incorporated into the MH circuitry to ensure peak airway pressure did not exceed 35 cmH_2O during MH, c) MH was performed with an inspiratory pause of approximately 2-3 seconds and inspiratory: expiratory ratio of approximately 1/2 and at a rate of 10-12 breaths/min for a period 5 min.

The baseline, 5 and 30 minutes post-intervention following parameters that were recorded routinely taken from the charts:

The ratio of partial pressure of arterial oxygen to fraction of inspired oxygen (PaO_2/FiO_2) and arterial partial pressure of carbon dioxide ($PaCO_2$) were recorded. PaO_2/FiO_2 was derived from arterial blood gas (ABG) analysis and FiO2. A calibrated blood gas analyzer (Nova Biomedical, Stat Profile M, U.S.A.) was used for ABG analysis and the FiO_2 was read from the ve$_n$tilator digital display (Servo 300, Siemens, Germany).

Static pulmonary compliance (Crs), peak and mean airway pressures were recorded from th$_e$ Siemens ventilator digital display.

Heart rate (HR), mean arterial blood pressure (MABP) and peripheral oxygen saturation (SpO_2) were recorded from the patient monitoring equipment (Draeger Medical Systems Inc, U.S.A.).

STATISTICAL ANALYSIS

Data were analyzed using SPSS 11.0 for Windows. Interval data from the different time periods were compared using repeated measures analysis variance test. When a significant time effect was found, paired-samples t tests with Bonferroni correction was used to identify which time periods were significantly different. Data are expressed as mean ± Standard Deviation (mean ± SD). A p value less than 0.05 was considered statistically significant.

RESULTS

Seventeen man and seventeen women who met the inclusion criteria were studied. Table 1 and 2 shows demographic data and ventilation parameters for 34 patients.

Figure 1

Table 1: Demographic data of patients.

Sex *(n-%)*	*Female-Male*	17 (50) – 17 (50)
Primary diagnose *(n-%)*	*Surgical-Trauma-Medical*	18 (52.9) – 11 (32.4) – 5 (14.7)
Comorbid disorders *(n-%)*	*Cardiovascular* *Diabetes* *Neurologic*	5 (14.7) 5 (14.7) 2 (5.8)
Age (years) *(mean ±SD)*		49.97 ± 20.75
Days in study (days) *(mean ±SD)*		3.29 ± 3.91

Figure 2

Table 2: Ventilation parameters of patients.

Mode of ventilation *(n-%)*	*PRVC-PC*	32 (94.1) – 2 (5.9)	
I/E *(n-%)*	*1/2-1/1-1/1.5*	28 (82.4) – 2 (5.9) – 4 (11.8)	
		mean ± SD	***Minimum - Maximum***
Mechanical rate *(breath/min)*		13.97 ± 2.16	10 - 20
PEEP *(cmH_2O)*		4.85 ± 0.98	3 - 7
FiO_2 *(%)*		0.40 ± 0.05	0.3 -0.5
Initial TV *(mL)*		577.57 ± 75.44	446 - 720
VE *(mL/min)*		7.85 ± 1.95	4.1 – 12.9

%=per cent, PRVC=pressure regulated volume control ventilation, PC=pressure controlled ventilation, I/E= ratio of inspiration to expiration, SD=standard deviation, min= minutes, PEEP=positive end expiration pressure, cmH_2O=centimeters of water pressure, FiO_2=fraction of inspired oxygen, TV=tidal volume, VE= minute volume, ml=milliliter.

PaO_2/FiO_2 ratio showed a significant difference over time (p=0.001) with increases observed at the 5 minutes following MH (p<0.001). The mean improvement in PaO_2/FiO_2 ratio was approximately 27 mmHg (7%) at the 5 minutes post-intervention and remained above baseline at the 30 minutes post-intervention (p<0.001). A similar trend was seen with SpO_2 value which improved by a mean of approximately 0.4% at the 5 minutes post-intervention (p=0.003), with improvement did not maintain at the 30 minutes post-intervention (p=0.03; Table 3).

Compared to baseline, $PaCO_2$ value displayed a significant decrease at the 30 minutes post- intervention (p=0.009). The mean decrease in $PaCO_2$ value was approximately 7%. Static compliance displayed a significant difference over time (p<0.001). Compared to baseline, a mean improvement of approximately 10.2 ml/cmH_2O (24%) occurred at the 5 minutes post-intervention (p<0.001) and remained above

baseline at the 30 minutes post-intervention (p<0.001). Peak airway pressure (PAP) decreased significantly 5 minutes after treatment (p=0.001). The mean decrease in PAP was approximately 0.8 cmH_2O (4%) with the change was not significant at the 30 minutes post-intervention (p=0.08). No significance was demonstrated in the mean airway pressure (MAP) over time (p=0.512; Table 3).

HR did not change significantly over time (p=0.378). MABP decreased at the 5 minutes after MH and at the 30 minutes post-intervention but it was not significant. (p=0.06, p=0.02 respectively; Table 3).

Figure 3

Table 3: Descriptive data for the dependent variables for the patients (mean ± SD)

		Time post-intervention	
	Baseline	**5 Minutes**	**30 Minutes**
HR *(bpm)*	95.38 ± 17.39	95.44 ± 17.73	95.2 ± 17.43
MABP *(mmHg)*	96.29 ± 17.46	93.84 ± 16.96	91.68 ± 16.73
Crs *(ml/cmH_2O)*	42.67 ± 12.19	52.88 ± 15.11*	50.52 ± 13.96*£
PAP *(cmH_2O)*	19.41 ± 4.67	18.55 ± 4.32*	18.82 ± 4.84
MAP *(cmH_2O)*	9.67 ± 2.07	9.47 ± 1.94	9.61 ± 2.21
$PaCO_2$ *(mmHg)*	36.22 ± 4.98	34.6 ± 6.01	34.8 ± 5.02*
SpO_2 *(%)*	99.17 ± 1.48	99.55 ± 0.95*	99.55 ± 1.02
PaO_2/FiO_2 *(mmHg)*	375.96 ± 115.94	403.63 ± 125.18*	404.88 ± 124.23*

HR=heart rate, bpm=beats per minute, MABP= mean arterial blood pressure, mmHg= millimeters of mercury, Crs= total static lung compliance, ml= milliliters, cmH_2O= centimeters of water, PAP= peak airway pressure, MAP= mean airway pressure, $PaCO_2$= arterial partial pressure of carbon dioxide, SpO_2= peripheral oxygen saturation, %= per cent, PaO_2/FiO_2= the arterial oxygen to fraction of inspired oxygen ratio.

** Statistically different from baseline value p<0.01.*

£ Statistically different from 5 minutes after intervention value p<0.01.

DISCUSSION

Results of this retrospective study indicated that manual hyperinflation to 35 cmH_2O with an inspiratory pause of two-three seconds in critically ill patients produced significant improvements in Crs and gas exchange measured using PaO_2/FiO_2. The improvement in Crs and PaO_2/FiO_2 were significant 5 minutes after MH and persisted for up to 30 minutes. There were no changes in the heart rate and mean arterial blood pressure after MH.

In the literature, there is no consensus or guidelines as to the appropriate dose of MH (standardized description of the technique, in terms of number of repetitions or length of time of application) that required achieving improvements in alveolar oxygenation, reverse atelectasis or mobilize pulmonary secretions [12]. The physiotherapy treatment administered in our institute included MH to an inspiratory pressure of 35 cmH_2O combined with an inspiratory pause of two-three seconds. This protocol was based on the work of Haake et al. [13] and Rothen et al. [14] where an inspiratory pressure of 40 cmH_2O was considered both safe and effective in recruiting lung atelectasis in the healthy lungs during anesthesia. However these authors included 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 [12]. Five minutes was reflected the clinical application of MH by physiotherapist in our ICU.

The patients studied were representative of a mixed ICU population requiring continuous mandatory ventilation. All patients investigated were well sedated/paralyzed during their participation in this study were not making any respiratory efforts of their own during the data collection period.

Consistent with previous findings, Crs improved after MH [151617]. Jones et al [15] and Patman et al [16] reported that compared to baseline a maximum improvement of 16 % (6.5 ml/cmH_2O) and a mean improvement of approximately 15 % (6 ml/cmH_2O) with improvements remained over baseline at 60 minutes post-intervention in Crs after MH respectively. The mean improvement in the Crs of approximately 24 % (10 ml/cmH_2O) seen in this study immediately after MH and remained above baseline at 30 minutes post-intervention is comparable to the increases of 23 % (8.5 ml/cmH_2O) for up to 20 minutes post-intervention reported by Hodgson et al. [12] following a treatment regimen which included MH in the treatment subjects with respiratory failure. This improvement may have been secondary to recruitment of more functioning alveolar units and volume restoration. The application of MH may utilize collateral ventilation within the lungs, to facilitate the mobilization of secretions and the recruitment of atelectatic lung units [18].

The parameters used to measure oxygenation in the current study were PaO_2/FiO_2 and SpO_2. Significant increase was detected in these parameters. The increase in PaO_2/FiO_2 ratio with MH treatment was still present 30 minutes after treatment in the present study. Three other studies found that MH, as part of a treatment regimen, had no significant effect on PaO_2 or PaO_2/FiO_2 in post-cardiac surgery patients [192021]. In studies investigating the effects of MH on oxygenation for non-cardiac surgery populations, Jones et al. [15] reported a significant increase in SpO_2, of approximately 2%, immediately following MH in subjects with respiratory

failure. Similarly, Patman et al [16] reported that mean improvement in PaO_2/FiO_2 ratio was 56 mmHg (17%) immediately post-intervention with improvements over baseline measures being maintained at 60 minutes in patients with respiratory failure. In contrast to the above, Barker and Adams 2002, Hodgson et al [12] and Patman et al [22] reported no significance difference in PaO_2/FiO_2 after MH treatment.

McCarren and Chow [23] report that patients may be under ventilated during MH as a result of slow inflation rate reducing minute ventilation. Although minute ventilation was not measured in the current study, hypoventilation during MH was improbable as $PaCO_2$ at the 5 minutes post-intervention was not increased but decreased insignificantly. $PaCO_2$ value displayed a significant decrease at the 30 minutes post- intervention which was consistent with the improvements seen in Crs. The failure to measure minute ventilation during MH and tidal volumes post-interventionally is the limitations of our study.

Changes in HR and MAP were variable. Hodgson et al. [12] and Singer et al. [24] found that HR was generally unaffected by MH. Patman et al. [22], who found a significant decrease in HR 5 and 20 min after MH compared to baseline. MH has previously been associated with alterations in cardiac output and arterial blood pressure [2425]. Singer et al. [24] suggested that the increased respiratory rate used by physiotherapists while manually hyperinflating, led to the development of gas trapping which reduced cardiac output and falls in MAP. Barker and Adams [26] reported that heart rate increased 10 min after MH. MAP displayed a significant difference over time with an initial fall, followed by an increase to about baseline values by the 60 min after MH. Hodgson et al. [12] and Patman et al [22] reported that MAP was no significant affected by MH. Our findings showed that while heart rate did not change during study period, there was insignificant decrease in MAP 5 minutes after MH and 30 minutes post-intervention. Hemodynamic stability was also maintained during the measurement period in spite of this decrease in MH. However, there might be significant cardiovascular deterioration in hemodynamically unstable, hypovolemic and low cardiac output patients.

It is concluded that MH performed in the hemodynamically stable ventilated patient significantly increased Crs and PaO_2/FiO_2 and decreased $PaCO_2$ without hemodynamic compromise, but the clinical significance of these improvements on patient outcome unclear. Further investigations are required to validate the results of this study as well as to determine the therapeutic value of MH on patient outcome.

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