Hemodynamic Effects of Manual Hyperinflation in Critically Ill Mechanically Ventilated Patients*

Mervyn Singer, MD; Julia Vermaat; Gareth Hall, RGN; George Latter, MSc; and Manesh Patel, MB, BS

Subject objective: To assess the hemodynamic effects of manual lung hyperinflation in mechanically ventilated patients and to measure the different inspiratory pressures and tidal volumes generated by different operators.

Design: Measurements of aortic blood flow (by esophageal Doppler ultrasonography), systemic blood pressure, tidal volumes (by respirometry), and inspiratory pressures in the ventilator circuit were measured on the ventilator, during six intended manual hyperinflations (tidal volume > 150% that delivered by ventilator) using a 2-L rebreathing bag, and at 1, 5, 10, and 15 min after reconnection to the ventilator.

Setting: Intensive care unit.

Patients: Eighteen mechanically ventilated patients with normovolemia and stable circulatory status were assessed on a total of 20 occasions.

Interventions: Patients were disconnected from the ventilator to enable six manual hyperinflations to be given. Measurements were made before and at 5-min intervals until no further hemodynamic changes were seen.

Measurements and results: Hyperinflation (50% increase in tidal volume) was achieved only in 10 of 20 studies. Large variations were seen in percentage change in peak inspiratory pressure (−30% to +250%) and tidal volume (−33% to +127%) generated. Falls in cardiac output correlated to the increase in tidal volume but not to the increase in peak inspiratory pressure and took up to 15 min to recover to baseline values. Changes in cardiac output were independent of lung compliance and concurrent vasoactive drug support. No consistent change was noted in either blood pressure or heart rate.

Conclusions: Lung hyperinflation is frequently not achieved by the manual technique. Significant changes in cardiac output can occur and appear to be related to the tidal volume rather than pressure generated.

(Chest 1994; 106:1182-87)

**Key words:** cardiac output; hemodynamics; hyperinflation; mechanical ventilation; physical therapy

---

The chest physical therapy procedures of percussion, vibration, and postural drainage, hyperinflation, and endotracheal suction are regularly administered to the mechanically ventilated patient. However, despite routine use, little conclusive evidence of benefit exists. Much more data have been published regarding hazards and drawbacks, including arterial oxygen desaturation, hemodynamic changes, and barotrauma.

Hyperinflation, i.e., manually inflating the patient’s lungs with tidal volumes 50% greater than those delivered by the ventilator, is advocated to mobilize secretions in peripheral bronchi, to prevent and treat atelectasis, and to improve oxygenation. This technique was first described by Clement and Hubsch in 1968, yet to our knowledge, no scientific validation of its effectiveness has been reported other than prevention of postsuctioning hypoxemia. The potential dangers of manual hyperinflation such as hemodynamic instability, raised intracranial pressure, and barotrauma are well recognized. However, pressure blow-off valves are rarely used and no measurement of actual tidal volume delivered is made. The manual hyperinflation (MHI) technique currently advocated by most physical therapists in the United Kingdom consists of three phases: a slow inspiration, an inspiratory hold phase of 1 to 2 s, and then rapid release of the bag to simulate a “huff”. Yet this recommendation, though theoretically sound, also lacks clinical confirmation of benefit.

Likewise, changes in cardiac output occurring during MHI are unreported except in a few anecdotal cases. As a prelude to an investigation formally assessing hyperinflation techniques, we decided to...
Table 1—Patient Details

<table>
<thead>
<tr>
<th>Patient No./Sex/</th>
<th>Age, yr</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/25</td>
<td></td>
<td>Heroin overdose</td>
</tr>
<tr>
<td>2/F/81</td>
<td></td>
<td>Obstructed, perforated hernia; cardiogenic shock</td>
</tr>
<tr>
<td>3/M/51</td>
<td></td>
<td>Sepsis</td>
</tr>
<tr>
<td>4/F/58</td>
<td></td>
<td>Hypothermia, pneumonia</td>
</tr>
<tr>
<td>5/M/64</td>
<td></td>
<td>Cardiogenic shock</td>
</tr>
<tr>
<td>5/M/64</td>
<td></td>
<td>Re-admission with sepsis</td>
</tr>
<tr>
<td>6/M/19</td>
<td></td>
<td>Pneumonia</td>
</tr>
<tr>
<td>7/F/54</td>
<td></td>
<td>Pneumonia, cardiogenic shock</td>
</tr>
<tr>
<td>8/M/87</td>
<td></td>
<td>Ruptured femoral aneurysm, pneumonia</td>
</tr>
<tr>
<td>9/M/42</td>
<td></td>
<td>Rhabdomyolysis, pneumonia</td>
</tr>
<tr>
<td>10/M/62</td>
<td></td>
<td>Adult respiratory distress syndrome</td>
</tr>
<tr>
<td>11/M/38</td>
<td></td>
<td>Multiple trauma</td>
</tr>
<tr>
<td>12/M/52</td>
<td></td>
<td>Multiple trauma, pneumonia</td>
</tr>
<tr>
<td>13/M/67</td>
<td></td>
<td>Adult respiratory distress syndrome postsepsis</td>
</tr>
<tr>
<td>14/M/59</td>
<td></td>
<td>Pneumonia</td>
</tr>
<tr>
<td>14/M/59</td>
<td></td>
<td>Pneumonia</td>
</tr>
<tr>
<td>15/F/65</td>
<td></td>
<td>Wegener’s granulomatosis</td>
</tr>
<tr>
<td>16/F/27</td>
<td></td>
<td>Adult respiratory distress syndrome posttrauma</td>
</tr>
<tr>
<td>17/F/88</td>
<td></td>
<td>Cardiovascular accident, aortic neureoma</td>
</tr>
<tr>
<td>18/M/45</td>
<td></td>
<td>Subarachnoid hemorrhage, cardiac arrest</td>
</tr>
</tbody>
</table>

perform a pilot study recording tidal volumes, changes in airway pressure, and cardiac output (by continuous esophageal Doppler measurement of aortic blood flow) to assess whether tidal volume and/or airway pressure influenced cardiac output. Furthermore, recording was also made of airway pressure waveforms produced by nursing staff to see how closely these followed the recommended technique.

METHODS AND MATERIALS

Patients requiring mechanical ventilation on the intensive care unit (ICU) were enrolled into the study. Cardiorespiratory stability for a period of at least 2 h was mandatory for inclusion. Prior approval had been sought from the District Medical Ethics Committee and informed assent was obtained from the patients’ relatives.

The patients were positioned supine and, if not already in situ, an esophageal Doppler probe was inserted. This Doppler system (ODM II, Abbott Laboratories, Maidenhead, UK) consists of a 5-mm-diameter transducer emitting 4-MHz continuous wave Doppler ultrasound placed orally to a depth of 35 to 40 cm and oriented toward the descending thoracic aorta. Doppler frequency shifts reflected off moving blood corpuscles undergo spectral analysis and characteristic blood flow velocity waveforms are displayed on a color monitor. The probe is fixed in position to the catheter mount of the endotracheal tube with paper tape. Integral on-line software continuously measures the velocity waveform area that represents the stroke distance, i.e., the distance a column of blood travels down the descending aorta with each left ventricular stroke. A nomogram incorporating age, height, and weight converts the stroke distance into an estimate of total left ventricular stroke volume. This estimate is accurate to 85 to 90% compared with thermodilution. Furthermore, changes in cardiac output are reliably and accurately followed by this method with the coefficient of variation being half that of thermodilution. The machine was set to average stroke volume over every five beats to allow for respiratory variations in stroke volume.

Adequate intravascular volume status was confirmed by 200-mL colloid challenges producing no increase in stroke volume. For at least 30 min thereafter, no procedures were performed, and no alterations were made in the level of sedation. For consistency of measurement (as the patients were mechanically ventilated by different machines), a calibrated pressure transducer (MX860,
Medex, Haslingden, UK, connected to a 7834C Monitor, Hewlett Packard, Bracknell, UK) and a respirometer (Wright, Medishield, High Wycombe, UK) were placed in the ventilator circuit to measure changes in airway pressure and inspired tidal volume, respectively. Tidal volumes were checked against those delivered by the ventilator (either Servo 900C or 900B, Siemens, Sunbury-on-Thames, UK or Evita, Draeger, Hemel Hempstead, UK). All patients were receiving either controlled or synchronized intermittent mandatory ventilation with tidal volumes of approximately 10 mL/kg. All patients receiving synchronized intermittent mandatory ventilation had mandatory respiratory rates of at least 12 breaths/min. Pressure waveforms were recorded onto a precalibrated chart recorder.

Two sets of control measurements were taken at least 5 min apart to confirm cardiorespiratory stability. These consisted of (1) esophageal Doppler measurement of heart rate and cardiac output, (2) blood pressure directly transduced from an indwelling 20-G radial arterial catheter, and (3) arterial oxygen saturation measured by pulse oximetry (HP Monitor 7834C, Hewlett Packard, Bracknell, Herts). Peak airway pressure and tidal volume were also measured as previously described.

Nurses or physical therapists then performed manual hyperinflation in their usual fashion but were kept blinded to the additional measurements being recorded simultaneously. A 2-L circuit manual rebreathing bag (Mapleson-C) attached to an oxygen supply delivering 15 L of O2 per minute was connected to the catheter mount with the pressure transducer and respirometer placed in between. No positive end-expiratory pressure (PEEP) valve was added to the circuit. The operator delivered three to four breaths during which the lung compliance was subjectively assessed and the expiratory pressure relief valve adjusted accordingly, and then delivered six manual hyperinflations. The Doppler-measured cardiac output was recorded over the last hyperinflation before the patient was reconnected to the ventilator. Sets of cardiorespiratory measurements were then repeated at 1 min, 5 min, and every 5 min thereafter until no further change occurred. No other procedure or change in drug dosage was performed on the patient during this period. In particular, endotracheal suction was not performed during the study period unless clinically indicated, in which case that particular study was invalidated.

Dynamic lung compliance was calculated as the ratio between tidal volume and (peak inspiratory-end expiratory pressure) and expressed as mL/cmH2O.

Statistical analysis was performed using repeated measures analysis of variance with post-hoc Fisher PLSD tests, and the \( \chi^2 \) test (Statview software for Apple Macintosh).

RESULTS

Eighteen mechanically ventilated patients were studied on a total of 20 occasions. Patient details are shown in Table 1 and ventilator settings are shown in Table 2. Only two patients (cases 10 and 16) had ventilator-set inspiratory-expiratory ratios of 1:1 or greater and only three patients were receiving paralyzing agents.

The tidal volumes and peak inspiratory pressures generated by each manual lung inflation were consistent for each individual patient, both varying by no more than 15%. However, hyperinflation, ie, an increase greater than 50% in ventilator-set tidal volume, was achieved only in 10 of the 20 studies. The percentage change in tidal volumes generated ranged from −33% to +127% with the highest tidal volume recorded being in excess of 2 L. The peak inspiratory pressure generated by MHI varied from −30% to 250% of ventilator-delivered peak inspiratory pressure, the highest pressure recorded being 60 mm Hg. An example of the different pressure waveforms generated by the ventilator and MHI is shown in Figure 1.

Falls in mean blood pressure ≥10% occurred in three studies while increases ≥10% were seen in three. Both were associated with significant falls in cardiac output (mean 15.8% and 10%, respectively). Four of the 12 patients not demonstrating any major change in blood pressure also had falls in cardiac output exceeding 10%. The change in cardiac output was not related to either changes in tidal volume or peak inspiratory pressure (Fig 2). Heart rate was generally unaffected by MHI with only one patient showing an increase of 14% during MHI and two having falls of 10% and 12%, the latter (patient 14/1) also having the largest falls in blood pressure (−32%) and cardiac output (−26%).

A correlation (\( r^2=0.53 \)) was seen between percentage changes in peak inspiratory pressure and tidal volume (Fig 2). Whereas a similar though negative correlation was seen between percentage changes in cardiac output and tidal volume, no relationship could be shown between percentage changes in peak inspiratory pressure and cardiac output.

Figure 3 shows the changes in mean blood pressure, heart rate, and cardiac output in the ten patients in whom hyperinflation was achieved and the ten in whom tidal volumes did not exceed a 50% increase. No significant difference existed between baseline levels of blood pressure, heart rate, and cardiac output in the two groups. No significant overall change was noted in blood pressure or heart rate during hyperinflation; however, the cardiac output fell signif-
icantly. This fall was more pronounced and pro-
longed in the hyperinflation group, taking up to 15
min to recover. Of the five studies in patients
receiving vasoactive drug support, only one showed
a fall in cardiac output exceeding 10%.

In addition, changes in cardiac output were in-
dependent of lung compliance. In those patients in
whom the cardiac output fell >10%, the compliance
was 31.5 ± 5.9 mL/cm H2O (median ± SE) compared
with 30.5 ± 3.8 mL/cm H2O in those where the car-
diac output remained unchanged (p=NS). Four of
the eight patients requiring a fractional inspired ox-
gen concentration ≥0.70 showed a fall in cardiac
output with MHI as opposed to 7 of 12 of those re-
quiring a FiO2 <0.70 (p=NS).

Physical therapists performed MHI in two studies
(14/1 and 15), generating percentage increases in
tidal volume of 119% and 57% and peak inspiratory
pressure of 28% and 11%, respectively. Patient 14/1
had the greatest degree of cardiovascular instability
while patient 15 also had a significant fall in cardiac
output (18%) during MHI. Apart from the two

![Graphs showing hemodynamic changes with hyperinflation](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21701/)

**Figure 2.** Relationship between percentage change during hyperinflation from baseline values in tidal volume (TV), peak inspiratory pressure (PIP), blood pressure (BP), and cardiac output (CO).

**Figure 3.** Hemodynamic changes with hyperinflation. White circles=hyperinflation achieved; black
squares=hyperinflation not achieved.
physiotherapist-performed studies, the recommended MHI technique (slow inspiration, plateau, rapid expiration) was only used in 3 of the remaining 18 studies. The remainder mainly consisted of rapid inspiratory and expiratory phases with no intervening plateau.

**DISCUSSION**

Manual hyperinflation of critically ill patients produced significant reductions in cardiac output, taking up to 15 min to recover to baseline values. These falls in output were directly related to the degree of hyperinflation produced rather than the increase in peak inspiratory pressure generated. Large increases in intrathoracic volume impede venous return, resulting in falls in venous preload and, often, cardiac output. Our unit policy of maintaining a well-filled intravascular compartment may thus attenuate but not prevent significant falls in cardiac output with MHI. However, the duration of depression of cardiac output following MHI and the fall, albeit more transient, in cardiac output in the group in whom hyperinflation was not achieved suggests other mechanisms are important. Ventricular afterload is increased by the procedure and this may result in a decrease in ventricular compliance. An increase both in frequency of ventilation and in tidal volume during MHI may generate a high yet unrecognized level of auto-PEEP. Indeed, a follow-up investigation currently in progress using esophageal balloon measurements of pleural pressure has shown significant levels of auto-PEEP in a number of the patients studied.

The patients studied are representative of a mixed ICU population requiring continuous or synchronized intermittent mandatory ventilation and their sedation/analgesia paralysis regimens reflect current opinion with a lesser emphasis on paralysis. Whether similar hemodynamic changes would be seen with MHI in patients who are more alert and taking more spontaneous breaths requires further investigation. All patients investigated were well sedated during their participation in this study—no obvious differences were seen between those receiving and not receiving paralysis, analgesia, and/or vasoactive drug support.

Although no relationship was seen between hemodynamic changes and peak inspiratory pressures generated, these pressures were measured in the ventilator circuit rather than within the chest itself. How much of the inspiratory pressure generated during MHI is actually transmitted to the lung remains open to question. This gradient may be considerable; a recent article found that the pressure measured at the ventilator during synchronized intermittent mandatory ventilation was considerably higher than at a point just distal to the endotracheal tube (47 ± 10.4 cm H2O compared with 34.6 ± 12.2 cm H2O).12 We are currently performing a follow-up study measuring changes in intrathoracic pressure during MHI with an esophageal pressure transducer.

Changes in blood pressure and heart rate were variable, although few gross effects were seen and no overall trend could be detected. Similar findings were reported by Goodnough6 in patients following cardiothoracic surgery, only one of whom demonstrated a severe fall in blood pressure with an accompanying bradycardia. The esophageal Doppler technique used in our study, however, demonstrated that significant falls in cardiac output occurred despite stability of both systemic blood pressure and heart rate. This underlines the relative insensitivity of these hemodynamic variables on which decisions on whether or not to "bag" are often taken.7

The reliability of the esophageal Doppler technique is a necessary assumption. The ability to view the blood flow velocity waveforms on a continuous beat-by-beat basis on the monitor enables verification of a satisfactory Doppler signal; a sharply defined velocity waveform with minimal spectral dispersion is indicative of a laminar midstream plug flow profile in the descending thoracic aorta. These signals were not seen to be affected by manual hyperinflation and one of the investigators tethered the esophageal probe and endotracheal tube during the procedure to prevent movement artifact or probe displacement. The rapidity of change in stroke volume during MHI precludes confirmation by any other measurement technique; however, validation of esophageal Doppler measurement of cardiac output against reference techniques such as Fick10 and thermodilution has been shown in a variety of circumstances such as alterations in PEEP13 and left ventricular failure.14 The study involving changes in PEEP13 suggests reliability of this technique at varying levels of intrathoracic pressure and volume.

We found that our ICU nurses, who perform the majority of such treatments, were generally unaware of the "recommended" MHI technique as reflected by only 3 of the 18 nurse-performed studies producing the desired airway pressure waveform. In some cases, large inspiratory pressures (with lesser changes in tidal volume) were produced by their efforts, although this did not appear to compromise cardiovascular status as much as when hyperinflation was properly performed, such as was seen when undertaken by the physical therapists. The question as to whether MHI is indeed beneficial needs to be answered, especially as true hyperinflations will depress cardiac output for up to 15 min and, in patients requiring high levels of PEEP, disconnection from the ventilator may also compromise arterial oxygenation.
This is especially pertinent when even the target of a 50% increase in tidal volume is not reached, as was seen in half of our studies and, as in three instances, the manually delivered tidal volume was actually lower. This variability in tidal volume delivery underlines the deficiencies of subjective assessment and implies the need for incorporation of a respirometer into the circuit. Whether alternative delivery systems such as an Ambu bag would be more reliable requires further investigation.

A further question arising from this study is what should be monitored during MHI. The tidal volume generated seems to be more crucial than airway pressure in compromising hemodynamic stability and if MHI is to be performed then at least a true hyperinflation should be achieved.

In conclusion, this study shows that significant hemodynamic changes can occur with manual hyperinflation and that these appear to be related to tidal volume rather than peak inspiratory pressure, although other factors such as generation of high levels of intrinsic PEEP may also be responsible. There also appears to be a wide discrepancy in tidal volumes and pressures generated and in technique used by the nursing staff. Further investigation is warranted to elucidate whether hyperinflation is indeed beneficial and, if so, which is the best technique to apply.

REFERENCES
1 Adlkofe RM, Povaser MM. The effect of endotracheal suctioning on arterial blood gases in patients after cardiac surgery. Heart Lung 1978; 7:1011-17
6 Goodnough SKC. The effects of oxygen and hyperinflation on arterial oxygen tension after endotracheal suctioning. Heart Lung 1985; 14:11-7
7 King D, Morrell A. A survey on manual hyperinflation as a physiotherapy technique in intensive care units. Physiotherapy 1992; 78:747-50
13 Singer M, Bennett ED. Optimisation of positive end-expiratory pressure for maximal delivery of oxygen to tissues by using oesophageal Doppler ultrasonography. BMJ 1989; 298:1350-53