Hemodynamic and ventilatory effects of manual respiratory physiotherapy techniques of chest clapping, vibration, and shaking in an animal model

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1Department of Physiotherapy, Singapore General Hospital, Singapore 169608; 2Department of Physiotherapy, University of Queensland, Brisbane 4072; 3Physiotherapy Department, Alfred Hospital, Prahran 3004; 4School of Physiotherapy, La Trobe University, Victoria 3086; and 5Cardiac Research Theatre, The Prince Charles Hospital, Queensland 4032, Australia

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Wong, W. P., J. D. Paratz, K. Wilson, and Y. R. Burns. Hemodynamic and ventilatory effects of manual respiratory physiotherapy techniques of chest clapping, vibration, and shaking in an animal model. J Appl Physiol 95: 991–998, 2003. First published May 16, 2003; 10.1152/japplphysiol.00249.2003.—Chest clapping, vibration, and shaking were studied in 10 physiotherapists who applied these techniques on an anesthetized animal model. Hemodynamic variables (such as heart rate, blood pressure, pulmonary artery pressure, and right atrial pressure) were measured during the application of these techniques to verify claims of adverse events. In addition, expired tidal volume and peak expiratory flow rate were measured to ascertain effects of these techniques. Physiotherapists in this study applied chest clapping at a rate of 6.2 ± 0.9 Hz, vibration at 10.5 ± 2.3 Hz, and shaking at 6.2 ± 2.3 Hz. With the use of these rates, esophageal pressure swings of 8.8 ± 5.0, 0.7 ± 0.3, and 1.4 ± 0.7 mmHg resulted from clapping, vibration, and shaking respectively. Variability in rates and “forces” generated by these techniques was <20% in average coefficients of variation. In addition, clinical experience accounted for 76% of the variance in vibration rate (P = 0.001). Cardiopulmonary physiotherapy experience and layers of towel used explained ~79% of the variance in clapping force (P = 0.004), whereas age and clinical experience explained >80% of variance in shaking force (P = 0.003). Application of these techniques by physiotherapists was found to have no significant effects on hemodynamic and most ventilatory variables in this study. From this study, we conclude that chest clapping, vibration, and shaking 1) can be consistently performed by physiotherapists; 2) are significantly related to physiotherapists’ characteristics, particularly clinical experience; and 3) caused no significant hemodynamic effects.

The mucus from bronchial walls, whereas vibration and shaking increase the expiratory flow rate in the peripheral and central airways. All three techniques essentially produce mechanical energy transmitted across the chest wall into the airways with the possibility of generating mist flow great enough to detach mucus from the airway wall (28). Such enhancement of the gas-liquid interactions that create a cephalad bias should therefore produce the desirable effects, that is, moving excessive secretions and improving tracheobronchial secretion clearance. However, this has not been consistently demonstrated in clinical studies (32), with those showing no significant treatment effects (14, 29, 33) after addition of chest clapping or vibration.

Other studies have documented hemodynamic changes after these techniques were applied. Cardiac arrhythmias were reported after chest clapping (9). Physiotherapy, comprising 2 min of chest clapping and vibration in alternate side lying followed by airway suctioning, was shown to significantly increase heart rate (HR), systolic and mean arterial blood pressures (BPs), cardiac output (CO), and rate-pressure product with the extent of these effects markedly attenuated by anesthetic agents, muscle relaxants, sedatives, and pressure support ventilation (4, 10–13). However, hemodynamic stability was reported in other acute care settings (19). All of these studies used a combination of techniques, e.g., a combination of postural drainage, chest clapping, and suctioning. The techniques were also not described, e.g., vibration rate, clapping force, etc. Differences in findings may also have been due to patient variability.

Ventilatory variables such as peak expiratory flow rate (PEFR) and tidal volume (VT) have not been studied, although evidence of increased expiratory flow rates during chest vibration was reported in intubated patients (20). Because the effects of chest clapping and,

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in particular, vibration and shaking are aimed to create airflow changes in the airways, it will be important to verify their effects on ventilatory variables of PEFR and Vt.

Different personnel performing chest clapping may apply different amount of forces (6). A recent study has shown interoperator variability in force [mean coefficient of variation (CV) was 15%] for chest clapping when 35 physiotherapists were studied (1). However, whether the same rate was maintained throughout the application of the technique was unknown. Because these techniques require repeated compressive force application, it is reasonable to hypothesize that the rate and force of these techniques may be important determinants of the measured effects on the hemodynamics and ventilation. Furthermore, chest vibration and shaking have not been evaluated. Knowledge of rates and forces generated by these manual techniques and how they influence hemodynamic or ventilatory variables, or how they relate to characteristics of the personnel applying them are important to understand the different clinical outcomes when these techniques are evaluated.

In the present study, physiotherapists performed chest clapping, vibration, and shaking in anesthetized, ventilated healthy sheep with the aim of describing the rates and forces produced by the physiotherapists applying these techniques. A second aim of the study was to establish whether the rates and forces of these techniques could be correlated with characteristics of the personnel who performed them. Finally, the study was to determine whether the rates and forces of these techniques have a significant effect on hemodynamic and ventilatory variables. An animal model was chosen to minimize confounding variables from patient variability as well as allow invasive hemodynamic monitoring.

MATERIALS AND METHODS

All personnel applying the techniques were practicing clinical physiotherapists recruited both from the facility where the study took place and the research team. The physiotherapists completed a questionnaire about age, years of experience, and usage frequency of the techniques.

Two Merino sheep (37.9 and 38.4 kg) were instrumented for the study. They were induced with thiopentone sodium (15–20 ml/kg, Sigma Chemical, St. Louis, MO) through a 20-gauge intravenous catheter inserted into the animal’s right jugular vein, and anesthesia was effected and maintained in the experiment with 1.5% halothane/oxygen and boluses of thiopentone sodium (10 ml i.v bolus). Orotracheal intubation was secured with a cuffed endotracheal tube with a 10-mm inner diameter. The animals were ventilated with a constant-volume anesthetic ventilator (Campbell Respirator, ULCO Engineering Pty, Marrickville, Australia) at 10 ml/kg, 10–14 breaths/min, inspiration-to-expiration ratio of 1:2, and inspired oxygen fraction of 0.21. The animals were positioned in right lateral decubitus and reverse Trendelenburg to prevent regurgitation and aspiration.

For hemodynamic monitoring, the left internal carotid artery was cannulated with a 18-gauge catheter and the left internal jugular vein with the pulmonary artery (PA) catheter (model 131F, Abbott Critical Care). These catheters were connected to pressure transducers (Baxter Edwards), which were in turn connected to a VR-16 Simultrace Recorder (Honeywell, Electronics for Medicine, Pleasantville, NY) with electrocardiographic/DC (model V1207) and pressure (model V2206) amplifier plug-ins. These amplifiers were interfaced with an analog chart recorder (Thermal Arrayrecorder WR 800, Graphite, Tokyo) that provided real-time recordings on the chart running at 10 mm/s. The indwelling arterial line measured the systolic and diastolic BP, while the PA catheter, inserted into the main PA, monitored the PA and right atrial pressures (Pra) via the distal and proximal lumens respectively. Electrocardiography and hence HR were also monitored.

The respiratory variables of PEFR and expired Vt were captured by the VenTrak Respiratory Mechanics monitoring system model 1550 (Novametrix Medical Systems), which was connected by a pneumotach to the ventilatory circuit of the ventilator.

An esophageal balloon (C.R. Bard, Murray Hill, NJ) was inserted to the level of the lower lobes of the lungs, as detected by an electronic stethoscope (Meteerec). The balloon was attached to a pressure transducer (Baxter Edwards), which was similarly connected to the VR-16 simultrace recorder and analog chart recorder. The esophageal balloon pressure was calibrated to 0 mmHg, and any pressure swing (APes) during chest clapping, vibration, and shaking would reflect changes in the intrathoracic pressure. These pressure changes would in turn reflect the intensity of the force applied by these techniques. The area over which the esophageal balloon was positioned was sheared of fleece and marked “X” on the chest wall.

The physiotherapists, instructed to perform the manual respiratory physiotherapy techniques as they normally do with adult patients, applied chest clapping for 1 min, followed by six vibrations and six shakings over the area marked X. Vibrations and shakings were applied during the expiratory phase of the respiratory cycle. Use of towels over the animal’s chest wall during clapping was determined by the physiotherapists, and the layers of the towels were recorded. In clinical practice, towels are utilized during the technique of clapping to ensure patient comfort and to avoid unnecessary sensory stimulation of the skin. It is standard clinical assessment for physiotherapists to compress the chest to gauge how much force to apply. Each technique was randomly performed with a period of 10 min between each technique. The animal’s hemodynamic status was observed to be stable and within safe limits before the next physiotherapist began. Approximately 10–15 min was required between physiotherapists.

The physiotherapists traced their hand shapes on an A4-sized paper provided. The shape of the hand was closed at the level of the A4-sized paper. The tracing was scanned into JPEG format and then exported to computer-aided drafting software (AutoCAD 2002 package, Autodesk Architectural Desktop 3.3, Autodesk). The tracing was outlined by an architect who was blind to the nature of the study. The AutoCAD software then computed the area of the hand size by referencing each hand shape to the standard area of the A4-sized paper.

The procedures involving the animals were approved by the Animal Experimentation Ethics Committee, The University of Queensland, which complied with the Australian Code of Practice for Care and Use of Animals for Scientific Purposes by the NH&MRC/CSIRO/AAC (21). These guidelines are consistent with those from the American Physiological Society.
Society’s “Guiding Principles in the Care and Use of Animals.”

Data and statistical management. The rates (or frequencies) of the three techniques were measured in Hertz (number of compressive forces/s). The force of any of the techniques was the ΔPes measured in mmHg. Clapping rates and forces were calculated off the strip chart recordings for every 10 s during the 1 min of application. Vibration, shaking rates, and forces were calculated for each time the technique was applied. Variability of these two variables was determined by CVs calculated by the ratio of standard deviation (SD) over means for each subject. The means, SDs, and 95% confidence intervals of the mean were calculated for each CV.

Pearson’s product moment correlation coefficients were calculated for rates and forces of the techniques, subjects’ ages, body mass indexes (BMI), hand sizes, and years of experience [since graduation, total and cardiopulmonary physiotherapy (CP) practice], and number of layers of towels used by the physiotherapists during clapping. Spearman’s rho correlation coefficient was used to calculate reported frequencies of usage of chest clapping, vibration, and shaking with the rates and forces of the techniques. Where there were significant correlations, regression analyses were performed to determine the extent these independent variables were related to the rates and forces of the techniques.

Ventilatory and hemodynamic data were tested to have homogeneity of variance and normality. Effects of these variables on the rates and forces produced by the three respiratory physiotherapy techniques were then determined by repeated-measures analysis of variance. Significance level was set at \( P < 0.05 \). SPSS 11.0 (SPSS, Chicago, IL) software was used to analyze the data.

RESULTS

The subjects consisted of 10 physiotherapists, (9 females, 1 male), aged 31.1 ± 7.1 (mean ± SD) yr, with a BMI of 20.5 ± 2.4. The median total clinical experience was 8.3 yr (range 0.4–21.0 yr), of which 5.0 yr (median; range of 0.4–20.0 yr) had been practiced in the cardiopulmonary specialty. The two sheep received treatments from two and eight physiotherapists, respectively.

Typical strip chart traces of the application of the techniques by the physiotherapists are shown in Fig. 1. Table 1 shows the proportions of physiotherapists reporting their usage frequencies. Only the frequency of clapping use was correlated with the clapping rate (Spearman’s rho = –0.646, \( P = 0.044 \)). Values of means ± SD for clapping, vibration, and shaking rates were 6.2 ± 0.9, 10.5 ± 2.3, and 6.2 ± 2.3 Hz, respectively. Clapping, vibration, and shaking forces were 8.8 ± 5.0 mmHg (12.0 ± 6.8 cmH2O), 0.7 ± 0.3 mmHg (1.0 ± 0.4 cm H2O), and 1.4 ± 0.7 mmHg (1.9 ± 1.0 cm H2O), respectively.

Fig. 1. Strip chart traces of chest wall clapping (a), vibration (b), and shaking (c) during two breaths. a: Notice the effect of hand dominance on chest clapping pattern. The esophageal pressure swing (ΔPes) measures the “force” of the chest clapping. The small “negative” ΔPes with each strike was due to the upward deceleration force caused by tissue recoil as the hand was lifted off the chest wall. The heavier the chest clapping strike, the greater that deceleration force seemed. b and c: Rate of application was greater in vibration than in shaking. In some traces, the physiotherapist appeared to have applied heavier force in the first compression, as shown in c.
Subjects were able to maintain within 20% variability in the rates and forces of these techniques, although 95% confidence interval shows CV can be as high as 18% (Fig. 2). Of these, clapping rates were the least variable, with a mean CV of 5%. There was no correlation between BMI, hand size, and the rate or force applied in either technique. Age and years of CP experience were negatively correlated with vibration rate \( (r = -0.839, P = 0.002; \text{and} \ r = -0.746, P = 0.013, \text{respectively}) \), but positively correlated with clapping force \( (r = 0.635, P = 0.049; \text{and} \ r = 0.651, P = 0.042, \text{respectively}) \) and shaking force \( (r = 0.731, P = 0.016; \text{and} \ r = 0.727, P = 0.017, \text{respectively}) \). Number of layers of towel used during clapping was negatively correlated with the clapping force \( (r = -0.697, P = 0.025) \). Years of total clinical experience were also negatively correlated with vibration rate \( (r = -0.872, P = 0.001) \) but positively correlated with shaking force \( (r = 0.797, P = 0.006) \).

By regression analysis \[ \text{predicted vibration rate} = 12.921 - 0.261 \times \text{years of total clinical experience} \], years of total clinical experience was significantly related to vibration rate and accounted for 76% of its variance \( r^2 = 0.760, \text{adjusted } r^2 = 0.731, F(1,8) = 25.402, P = 0.001 \). Age and CP experience did not predict significantly over and above years of total clinical experience as a predictor of vibration rate \[ r^2 \text{ change} = 0.103, F(2,6) = 2.272, P = 0.184 \].

Clapping force was significantly related to layers of towel used and CP experience \[ \text{predicted clapping force} = 8.111 - 2.541\times \text{layers of towel} + 0.426\times \text{CP experience} \]. This regression model explained >79% of the variance in clapping force \[ r^2 \text{ change} = 0.791, F(2,7) = 13.221, P = 0.004 \]. The addition of the variables of age and years of total clinical experience did not predict significantly over and above the model \[ r^2 \text{ change} = 0.012, F(2,5) = 0.154, P = 0.861 \].

### Table 1. Percentage of physiotherapists reporting their usage frequencies

<table>
<thead>
<tr>
<th>Percentage of Physiotherapists Reporting</th>
<th>25% of the time</th>
<th>50% of the time</th>
<th>75% of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clapping</td>
<td>30%</td>
<td>70%</td>
<td>0%</td>
</tr>
<tr>
<td>Vibration</td>
<td>10%</td>
<td>20%</td>
<td>70%</td>
</tr>
<tr>
<td>Shaking</td>
<td>60%</td>
<td>10%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Fig. 2. Variability of the rate (top) and force (bottom) of the 3 techniques. The individual rates and forces by each subject \( (n = 10) \) are plotted. Rates measured the number of compressive forces applied by each technique per second. Forces were reflected by the largest \( \Delta\text{Pes} \), in mmHg, made during the 10 s of clapping and during each vibration or shake. In addition, the means \( \pm \text{SD} \) (95% confidence interval) for coefficients of variations of clapping, vibration, and shaking rates were 4.5 \( \pm \text{SD} \) (3.8–5.1), 12.0 \( \pm \text{SD} \) (6.2–17.6), and 10.6 \( \pm \text{SD} \) (6.7–14.6), respectively. Coefficients of variations for forces were 11.9 \( \pm \text{SD} \) (6.2–17.6), 8.5 \( \pm \text{SD} \) (0–18.5), and 11.7 \( \pm \text{SD} \) (5.1–18.4), respectively.
On the contrary, age and years of total clinical experience, but not CP experience, were significantly related to shaking force (predicted shaking force = 7.559 + 0.358(years of total clinical experience) - 0.304(age); $r^2 = 0.802$, adjusted $r^2 = 0.746$, $F(2,7) = 14.215$, $P = 0.003$. Between the two variables, years of total clinical experience explained $\geq 63\%$ of the variance ($r^2$ change = 0.635, $F(1,8) = 13.914$, $P = 0.006$), whereas age explained a further $17\%$ ($r^2$ change = 0.167, $F(1,7) = 5.934$, $P = 0.045$).

Of the ventilatory variables, vibration significantly increased the expired VT in the anesthetized sheep (Table 2). Abnormally low PEFR of 9.9 ± 5.7 l/min and expired VT of 9.5 ± 1.1 ml during chest clapping were recorded; these were likely to be artifacts and were not subjected to statistical analysis.

Chest clapping, vibration, and shaking had no significant effects on any of the hemodynamic variables (Table 3). Apart from artifacts from changes in intrathoracic pressure during clapping, no arrhythmias were seen during any of the techniques.

**DISCUSSION**

The main aims of this study were to describe the rates and forces generated by chest clapping, vibration, and shaking, as well as technique variability among physiotherapists, to determine what characteristics of physiotherapists were related to the rates and forces, and to study their effects on hemodynamic and ventilatory variables.

The techniques were evaluated in healthy animals. By using an animal model, variability in the rates and intrathoracic pressure changes arising from the application of these techniques can be attributed to inherent characteristics of the physiotherapists instead of to possible confounding effects of a patient’s age, tolerance level of techniques, disease processes, mechanical ventilation modes, and respiratory system and rib compliance. An important finding from this study was the significant relationships between the physiotherapist’s age and clinical experience on vibration rate, clapping, and shaking forces. The study also demonstrated no adverse hemodynamic or ventilatory effects after the application of the techniques, except for an unexpected significant increase in expired VT after vibration.

The present study on mostly Australian graduate physiotherapists has found a clapping rate of 6.2 Hz, which was consistent with that from another study involving mostly Australian graduates (1). This contrasted with the range of clapping rates defined by several clinical studies from Europe, viz., 3 (31), 4 (7), 5 (30), and up to 8.0 Hz (6). Our study has also demonstrated an acceptable variability, i.e., within a CV of 20%, in technique performance. Of significant note was consistency of chest clapping rate (CV = 4.5 ± 0.9%), possibly reflecting the emphasis in undergraduate training on maintaining consistent clapping rate.

The esophageal balloon model offers an estimate of the change in intrathoracic pressures produced by the manual techniques (Fig. 1). The intrathoracic pressure changes of 8.8 mmHg (or 12.0 cmH2O) during chest clapping by our 10 subjects are consistent with those reported by other researchers (6, 37). Repetitive changes in intrathoracic pressures may lead to “squeezing” secretions from small airways (32), and, therefore, one might hypothesize that as the force

Table 2. *Effects of clapping, vibration, and shaking on ventilatory variables*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Units</th>
<th>At Rest</th>
<th>During Clapping</th>
<th>During Vibration</th>
<th>During Shaking</th>
<th>$F^\dagger$</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEFR</td>
<td>l/min</td>
<td>44.7 ± 2.3</td>
<td>*</td>
<td>43.6 ± 3.9</td>
<td>43.9 ± 5.4</td>
<td>(2,27) = 0.186</td>
<td>0.831</td>
</tr>
<tr>
<td>Expired VT</td>
<td>ml</td>
<td>445.4 ± 50.6</td>
<td>*</td>
<td>506.1 ± 30.8</td>
<td>453.8 ± 66.8</td>
<td>(2,27) = 4.067</td>
<td>0.029‡</td>
</tr>
<tr>
<td>$f$</td>
<td>breaths/min</td>
<td>10.4 ± 1.3</td>
<td>10.4 ± 1.3</td>
<td>10.4 ± 1.3</td>
<td>11.5 ± 1.3</td>
<td>(3,36) = 1.887</td>
<td>0.149</td>
</tr>
</tbody>
</table>

Values are means ± SD. PEFR, peak expiratory flow rate; $f$, respiratory rate; VT, tidal volume. *Data probably contained artifacts and were not analyzed. †Repeated-measure analysis of variance statistics were calculated. ‡Significant at $p < 0.05$. Post hoc Bonferroni test for multiple comparisons showed significant difference between the pair, at rest vs. after vibration, with a mean difference of 60.6 ml, 95% confidence interval of 1.8 (at rest) and 119.5 (after vibration), $P = 0.042$.

Table 3. *Clapping, vibration, and shaking had no effects on hemodynamic variables in anesthetized sheep*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Units</th>
<th>At Rest</th>
<th>After Chest Clapping</th>
<th>After Vibration</th>
<th>After Shaking</th>
<th>$F(3,36)^* \dagger$</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>beats/min</td>
<td>88.3 ± 6.2</td>
<td>89.8 ± 5.5</td>
<td>92.5 ± 4.6</td>
<td>90.9 ± 5.5</td>
<td>1.054</td>
<td>0.381</td>
</tr>
<tr>
<td>SBP</td>
<td>mmHg</td>
<td>104.0 ± 9.1</td>
<td>102.3 ± 8.4</td>
<td>102.3 ± 8.4</td>
<td>102.7 ± 8.7</td>
<td>0.087</td>
<td>0.967</td>
</tr>
<tr>
<td>DBP</td>
<td>mmHg</td>
<td>86.2 ± 8.7</td>
<td>83.6 ± 10.4</td>
<td>84.1 ± 10.0</td>
<td>85 ± 9.1</td>
<td>0.143</td>
<td>0.934</td>
</tr>
<tr>
<td>MAP</td>
<td>mmHg</td>
<td>92.2 ± 8.6</td>
<td>90.0 ± 9.8</td>
<td>90.3 ± 9.5</td>
<td>91.0 ± 8.8</td>
<td>0.114</td>
<td>0.952</td>
</tr>
<tr>
<td>PAS</td>
<td>mmHg</td>
<td>13.0 ± 0.8</td>
<td>12.9 ± 0.3</td>
<td>12.6 ± 1.0</td>
<td>12.5 ± 1.0</td>
<td>0.857</td>
<td>0.472</td>
</tr>
<tr>
<td>PAD</td>
<td>mmHg</td>
<td>6.5 ± 1.3</td>
<td>6.8 ± 1.3</td>
<td>6.2 ± 1.2</td>
<td>6.7 ± 0.9</td>
<td>0.338</td>
<td>0.798</td>
</tr>
<tr>
<td>PP</td>
<td>mmHg</td>
<td>17.8 ± 3.6</td>
<td>18.7 ± 3.5</td>
<td>18.2 ± 3.3</td>
<td>17.7 ± 2.3</td>
<td>0.204</td>
<td>0.883</td>
</tr>
<tr>
<td>Pra</td>
<td>mmHg</td>
<td>0.6 ± 0.5</td>
<td>0.8 ± 0.4</td>
<td>0.3 ± 0.5</td>
<td>0.5 ± 0.5</td>
<td>1.814</td>
<td>0.162</td>
</tr>
</tbody>
</table>

Values are means ± SD. HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial blood pressure; PAS, systolic pulmonary artery pressure; PAD, end-diastolic pulmonary artery pressure; PP, pulse pressure (SBP - DBP); Pra, right atrial pressure. *Repeated-measure analysis of variance statistics were calculated. For significance, $P < 0.05$ was used. None of the variables showed significant differences among the 4 conditions.
increases, so would the intrathoracic $\Delta$Pes and squeezing actions. However, how intrathoracic pressure changes actually relate to mucus transport remains to be investigated (24). CV of clapping force, as indicated by change in intrathoracic pressure in the present study, was 11.9 ± 8.0%, similar to that in another study that measured forces in Newton (1). Because the effects are to induce intrathoracic pressure changes, the measurement of $\Delta$Pes was chosen in the present study instead of direct force impact (in Newton) used in other studies (1, 6). At high rates or frequencies, the change in oscillatory intrathoracic pressures may not be transmitted across the chest wall (37). Contact forces may be attenuated by overlying subcutaneous tissue and clothing. The extent of decrease in respiratory system compliance as a result of anesthesia and mechanical ventilation (23) was unknown in our animal model, and, therefore, so was the effect on the force generated by chest clapping. Nevertheless, in our study, physiotherapists spent some time to gauge compliance and judge the amount of force to apply. The confounding variable of varying respiratory system compliance was controlled with an animal model. Therefore, any variability should be due to inherent inconsistency alone. Future studies may examine the effect of respiratory system compliance on force of chest clapping.

Our study is the first to report on the rates and intrathoracic pressure changes generated by manual chest vibration and shaking performed by physiotherapists. Our finding of a mean of 10.5 Hz for vibration and 6.2 Hz for shaking differed from the stated rates of 12–16 and 2 Hz, respectively, by some authors (30). The highest average vibration rate in this study was 14 Hz. Intrathoracic $\Delta$Pes generated by these techniques was $<$1.5 mmHg (or 2 cmH$_2$O).

The effect of chest vibration, and to a certain extent clapping, on mucus transport seems to be rate rather than pressure dependent (2, 8, 17, 18, 26). In a series of experiments on high-frequency oscillation applied to dog chest wall, mechanical vibration at rates of 11–15 Hz has been shown to enhance both tracheal and peripheral mucus transport rates in dogs, reaching three times the control group’s mucus transport rate at 13 Hz (8, 17, 26). Enhanced tracheal mucus transport rate at 5 and 8 Hz, but not at 3 Hz, was also noted, although not as remarkable as at higher rates (17). Evidence exists to suggest that the mechanism of enhanced mucus transport at these vibratory rates was an increased PEFR, resulting in an expiratory-flow bias in air-mucus interactions and a subsequent cephalad movement of mucus (2, 18). Shaking at 2 Hz, as suggested by some authors (30), would not have been effective in enhancing mucus clearance through this mechanism. Nevertheless, other possible mechanisms through which these techniques might affect mucociliary clearance include vibration-induced alteration in mucus cross-linking, vagal stimulation by direct chest wall vibration, and enhanced ciliary action through mechanical frequency resonance with inherent ciliary beat frequencies in the airways (17). Ciliary beat frequencies, in the range of the clapping, vibration, and shaking rates reported in the present study, have been reported for distal airways (3, 27) and during respiratory tract infection (36).

Another main finding of the present study is how physiotherapists’ reported frequency of usage, age, years of clinical experience (including CP experience), and the layers of towels used for clapping were related to the vibration rate, as well as clapping and shaking forces. To our knowledge, this is the first report of significant relationships between physiotherapists’ characteristics and technique performance. Our regression analyses have also demonstrated a strong relationship between clinical experience and technique performance.

Greater years of clinical experience appeared to predict a lower vibration rate in this study. An explanation could be better adherence to reported higher vibration rates (12–16 Hz) by recently graduated physiotherapists. Through experience, physiotherapists could have modified their vibration rates to suit their patients. Clinical experience, whether total or exclusive in cardiopulmonary-specific exposure, might have given confidence to physiotherapists, resulting in greater force applied during chest clapping and shaking. Other health professionals also found similar relationships, e.g., age and years of postgraduate training among surgeons significantly and positively influenced the performance of laparoscopic surgery time (25).

Layers of towel used and CP experience are related to clapping force in this study. As expected, the relationship between force and towel layers was negative. It further supports why measurements of $\Delta$Pes to reflect force generated by clapping are preferred to directly impact forces due to attenuation of clapping effects with overlying tissues or towel.

Frequency of usage of chest clapping is shown to be moderately but negatively correlated with clapping force. This finding is in contrast with that reported by other authors (1). Fatigue from performing too much clapping (70% reported performing clapping 50% of the time; see Table 1) could be a plausible explanation and possible area for future study. Consistent with a previous study (1), BMI and hand size were not correlated with any aspects of the techniques in this study.

The present study did not find an effect on PEFR during vibration, unlike the increase in maximal expiratory flow rates with chest compression reported in other studies (20). PEFRs should increase if expiratory flow bias in two-phase interactions, as discussed before, were to occur. Instead, expired $V_t$ was increased during this maneuver. In contrast to our finding, Nishino et al. (22) found a reflex-mediated increase in respiratory rate and a small decrease in $V_t$ in anesthetized subjects, which was thought to be due to mechanical restriction from chest compressions. The difference could be due to rhythmic compressions (of $<$1.5 mmHg) in our study, rather than sustained compressive loads ($\geq$25 mmHg) reported by Nishino et al. (22). In such a situation, chest wall mechanoreceptor could have been stimulated, and an increase in inspiratory force, hence
minute ventilation, may have occurred (23). Why this did not occur with chest clapping or shaking cannot be answered by this study. The fact that artifacts were possibly recorded during chest clapping should also caution over-interpretation of the increased \( V_t \) during vibration.

Although chest clapping effect on intrathoracic pressure changes in the present study, no significant effect was found on hemodynamic variables of HR, BP, PA pressure, and Pra. The fact that we maintained optimum anesthesia throughout the experiments could have explained the lack of effects in our study, compared with those in other studies (4, 9–13, 34). Indeed, the increase in Pra, CO, and/or PA wedge pressure (Ppaw) in some of these studies were attenuated by anesthetic, paralytic, and sedative agents (10, 11, 13). Deleterious effects found in previous studies may have been due to awareness of subjects receiving these techniques. Another possible difference in the outcome was that our study examined chest clapping for only 1 min, as well as only a set of six vibrations and shakings each to the anesthetized animals. In the other studies, physiotherapy consisted of 2 min of clapping and vibration in alternate side-lying positions and then followed by airway suctioning (4, 10, 12). Whether clapping and vibration, positioning, or suctioning were responsible for the hemodynamic changes in those studies was uncertain, but we have shown that chest clapping in half the duration together with vibration and shaking resulted in no hemodynamic changes. The greater oxygen demand during the act of positioning from side to side, the endotracheal suctioning or the combination of techniques could have also led to increased venous return, and thus higher preload and CO to the right and left ventricles (higher Pra and Ppaw, respectively). In such a state, HR and systolic BP, mediated by catecholamines, would be increased as shown in these same studies (4, 10–13, 34). However, this did not occur in our anesthetized animals. Similar lack of change in systolic BP was also observed in other studies involving healthy human subjects (5, 35).

A limitation of the present study was that we did not measure Ppaw and CO, which would have enabled an evaluation of the effect of intrathoracic pressure changes on CO. How intrathoracic pressure changes during chest clapping could influence CO and other intracardiac pressures could be investigated in future studies.

Evidence of a significant relationship between some physiotherapist’s characteristics and performance parameters of chest clapping, vibration, and shaking in the present study provides implications in teaching and research. Of relevance to clinical practice is the demonstration of fairly consistent technique application as well as lack of significant effects on hemodynamic and ventilatory variables in the animal model. In clinical practice, measurement of ΔPes to indicate intrathoracic pressure change during chest clapping, vibration, and shaking would normally be impossible or difficult. Given that these techniques are commonly used in treating pulmonary disorders in intensive care units (15), knowledge provided by the present study on how these techniques were performed, the influence of an operator’s characteristics, and the effects of hemodynamic and ventilatory variables is important.

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DISCLOSURES

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