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Acute Effects of Manual Hyperinflation on Oxygenation and Hemodynamic Stability in Mechanically Ventilated Patients with Stroke-Associated Pneumonia

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ABSTRACT

Background: Stroke-associated pneumonia (SAP) is a prevalent and serious consequence of acute stroke, often necessitating intubation and mechanical ventilation (MV). This condition significantly increases the hospital stay period and the risk of mortality.

Objective: To explore the acute impacts of manual hyperinflation on oxygenation and hemodynamic variables in MV patients with SAP.

Subjects and Methods: Forty mechanically ventilated patients (aged 50–70 years) diagnosed with acute SAP were recruited from the Stroke Intensive Care Unit (ICU) at El Kasr El Ainy Hospital. Patients were randomized and assigned to either a study group or a control group (n = 20 each). The study group underwent a single session of conventional chest physiotherapy (CPT) combined with manual hyperinflation, while the control group received conventional CPT alone. Arterial blood gases (PaO_2, $PaCO_2$), SpO_2, and Horowitz index, and hemodynamic variables (heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP)) were measured instantly pre- and post-intervention to assess acute physiological responses.

Results: A statistically significant improvement in oxygen saturation (SpO_2) was indicated in the two groups following treatment ($p < 0.05$). No significant variations were observed in the residual respiratory parameters (PaO_2, $PaCO_2$, Horowitz index) in both groups. No significant variations were indicated in SBP or MAP in either group. However, the control group demonstrated a significant increase in HR and DBP. Between-group comparisons indicated no statistically significant differences across all measured variables.

Conclusion: Manual hyperinflation appears to be a safe and well-tolerated intervention in mechanically ventilated patients with SAP, with no immediate adverse effects on oxygenation or hemodynamic stability.

Keyword: Stroke associated pneumonia, Manual hyperinflation, Mechanical ventilation.

INTRODUCTION

Stroke-associated pneumonia (SAP) is a prevalent and risky problem of acute cerebrovascular events, typically defined as pneumonia manifesting within the initial seven days post-stroke [1]. Epidemiological data suggest that approximately 14% of people who have a stroke develop SAP [2].

Several neurological risk factors significantly elevate susceptibility to SAP, including impaired consciousness, severe neurological impairment, elevated National Institutes of Health Stroke Scale (NIHSS) scores, large strokes, dysarthria, and aphasia [3]. In patients with severe stroke, mechanical ventilation (MV) is often required due to pulmonary or neurological compromise and is associated with elevated mortality rates [4].

The primary goals of MV in this population are to maintain adequate oxygenation and regulate arterial carbon dioxide tension ($PaCO_2$), thereby minimizing the risk of ventilator-induced lung injury [5].

Respiratory physiotherapy has a critical role in treating MV patients, particularly in reducing the incidence of pulmonary infections [4].

One primary objective of ICU physiotherapy for the MV patients is to enhance the discharge of retained or excessive airway excretions. The therapy aims to mitigate airway resistance, augment pulmonary compliance, and reduce the patient's breathing effort through a combination of various respiratory maneuvers, manual techniques, and mechanical devices [6]. Manual hyperinflation (MHI) is a physical therapy procedure that increases the tidal volume above the standard volume, creating a turbulent flow that helps the MV patients mobilize secretions, improve static compliance, increase oxygenation, and recruit collapsed lung zones [7].

In patients who are hemodynamically unstable, MHI should be used cautiously since, despite its benefits, it has certain hazards, such as alterations in the mean arterial pressure and barotrauma [8].

Cardiovascular instability is particularly concerning in stroke patients, as it may complicate MV management and contribute to higher morbidity. At three months, tachycardia was linked to worse outcomes for stroke patients, including increased in-hospital and post-discharge mortality [9]. Similarly, hypertension is related to worse prognoses, greater hematoma expansion, and neurological deterioration in stroke patients [10].

Although MHI has demonstrated potential respiratory benefits, its immediate physiological effects in stroke patients with SAP remain inadequately explored. Therefore, this research aimed to determine the acute effect of MHI on oxygenation and hemodynamic variables in MV patients with SAP.

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SUBJECTS AND METHODS

This randomized controlled trial was conducted at the Stroke Intensive Care Unit, El Kasr El Ainy Hospital, Cairo University, between June 2023 and March 2025.

Ethical considerations

The Ethical Committee of the Faculty of Physical Therapy, Cairo University, approved the study (P.T.REC/012/003763). Prior to enrollment, informed written consent was provided by a legally authorized family member after a thorough clarification of the study's aims, procedures, potential benefits, and data confidentiality. The study adhered to the Helsinki Declaration throughout its execution.

Participants

Forty MV patients (aged 50–70 years) of both sexes diagnosed with acute SAP participated in the study.

The diagnosis of SAP was performed by a pulmonologist based on a comprehensive clinical assessment and the Centers for Disease Control and Prevention criteria. Diagnosis required the presence of at least two of the following: abnormal chest X-ray, fever, leukocytosis, tachypnea, tachycardia, oxygen desaturation, altered arterial blood gases, or the presence of sputum or crackles on auscultation [11].

Patients were eligible for inclusion if they were intubated and on MV for at least 48 hours, had a Glasgow Coma Scale score of eight or higher, and were receiving a fraction of inspired oxygen (FiO_2) not exceeding 0.8. Patients with a history of pneumonia on admission, who presented with arrhythmia or hemodynamic instability, had an undrained pneumothorax or unstable blood pressure, or required a positive end-expiratory pressure (PEEP) greater than 10 cm H_2O, weren't included in the study.

Randomization

Eligible patients were randomized and assigned to two equal groups using simple randomization with sealed, unmarked envelopes. The control group (Group A) underwent a single session of conventional chest physiotherapy (CPT), while the study group (Group B) underwent a single session of MHI plus conventional CPT. Conventional CPT included postural drainage, mechanical vibration, percussion, and suction (Figure 1).

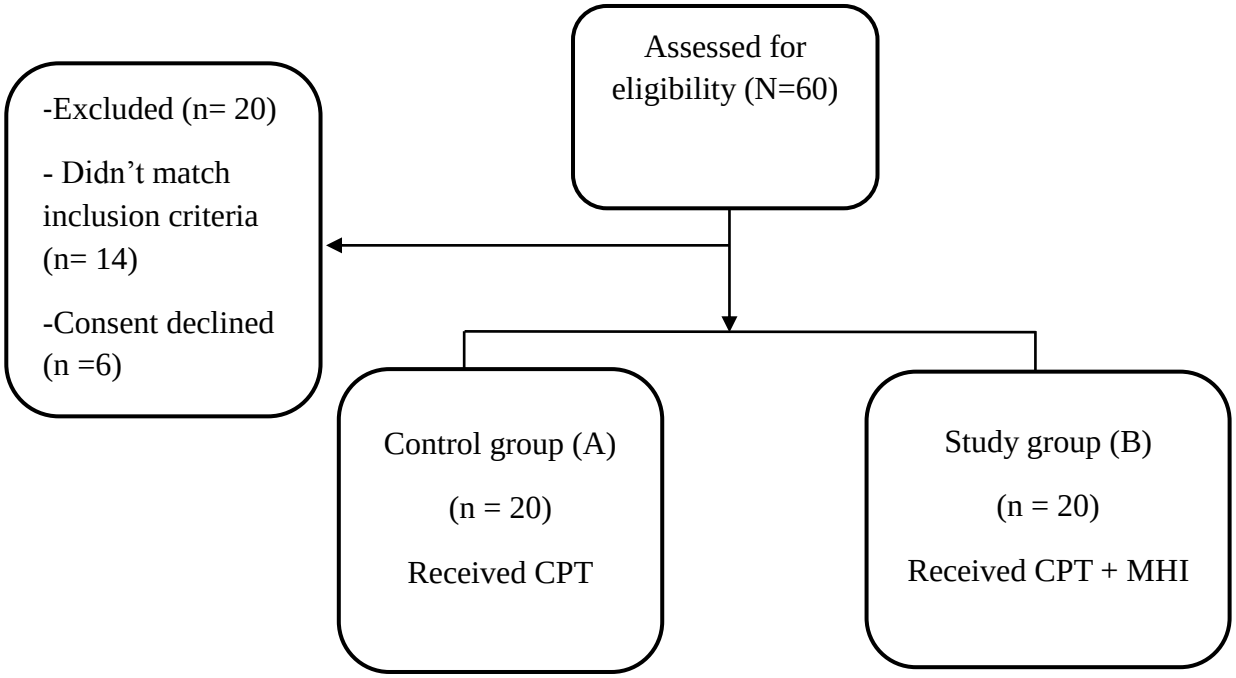

Figure (1): Flow chart of study participants.

Interventions

Manual hyperinflation

Manual hyperinflation was applied using a resuscitation circuit composed of a 2 L reservoir bag, a pressure manometer, a bacterial filter, and an oxygen supply, which was raised to 15 L/min. It was used with patients in a relaxed half-supine position with the head supported. After the patient was disconnected from the ventilator, the resuscitation circuit was attached to the patient's endotracheal tube and heat and moisture exchanger (HME) filter.

Slow, deep inspirations aiming to deliver 1.5 times the tidal volume (TV) of the ventilator were given using the two-hand technique while observing the manometer to ensure that pressure didn't exceed 40 mm H_2O. After holding the maximum inflation and pressure for two to three seconds, a rapid release was carried out. Two sets were given, each containing four MHI breaths followed by tidal volume breaths, for a total of eight MHI breaths per session. Patients were then unplugged from the resuscitation circuit and reconnected with the ventilator [8].

Conventional CPT techniques

Postural drainage: Patients were placed in various positions, allowing gravity to have maximal effect, facilitating movement of secretions from the affected lung segments [12].

Vibration: A mechanical vibration device was applied to the chest wall during expiration to produce oscillatory energy waves transmitted to the airways, thereby enhancing expiratory flow and secretions mobilization [13].

Percussion: Using cupped hands over the affected area produces an energy wave, generating mechanical force through the chest wall, which is transferred to the lungs and airways to manipulate intrathoracic pressure and fluctuating airflow in the bronchial tree, aiming to loosen thick, sticky, or retained secretions from the chest wall [14].

Suction: A suction catheter was positioned into the endotracheal tube after disconnecting the patient from the ventilator circuit and turning it on to 80–250 mmHg to remove excessive lower respiratory tract secretions [15].

Outcome measures

Primary outcomes

The primary outcomes were hemodynamic parameters, including HR, SBP, DBP, and MAP, obtained using a patient monitor (GE Carescape B650).

MAP was computed utilizing the standard formula: MAP= (SBP+2DBP)/3 [16].

Secondary outcomes

The secondary outcomes were arterial blood gas (ABG) parameters, such as partial pressure of oxygen (PaO_2), partial pressure of carbon dioxide ($PaCO_2$), oxygen saturation (SpO_2), and the Horowitz index (PaO_2/FiO_2).

Arterial blood samples were obtained using a heparinized syringe inserted at a 30° angle to the skin, with the patient's arm positioned palm-up on a flat surface and the wrist dorsiflexed at approximately 45°. Samples were collected directly from the radial artery and analyzed using a blood gas analyzer (GEM Premier 3000).

All outcome measures were obtained 5 minutes before the start of the session and 1 minute after its end.

Statistical analysis

All statistical analyses were performed using IBM SPSS version 25 for Windows. An unpaired t-test was used to compare participant characteristics between groups. The X^2-test was used to compare the distribution of sexes between groups. The Shapiro-Wilk test was used to ensure that the data followed a normal distribution. The group homogeneity was evaluated using Levene's test for variance homogeneity. Mixed MANOVA was used to analyze how treatment affected HR, BP, MAP, SaO_2, PaO_2, $PaCO_2$, and the Horowitz index. Post-hoc analyses were performed using the Bonferroni correction for subsequent multiple comparisons. Statistical tests were considered significant at $p < 0.05$.

RESULTS

- *Participant characteristics*:

Table 1 shows the participant characteristics of both groups. No significant changes were detected across groups in age, GCS, FiO_2, PEEP, and sex distribution ($p > 0.05$).

Table (1): Comparison of participant characteristics across both groups:

	Group A	Group B			
	Mean ±SD	**Mean ±SD**	**Mean difference**	**t- value**	**p-value**
Age (years)	63.70 ± 5.49	62.80 ± 7.37	0.9	0.44	0.66
GCS	10.45 ± 1.67	11.05 ± 1.67	-0.6	-1.14	0.26
FiO_2 (%)	48.70 ± 12.55	46.75 ± 10.85	1.95	0.52	0.60
PEEP (cm H_2O)	5.15 ± 0.81	5.20 ± 1.06	-0.05	-0.17	0.87
Females	7 (35%)	5 (25%)	χ^2 = 0.48		0.49
Males	13 (65%)	15 (75%)			

SD, Standard deviation; χ^2, Chi squared value; p value, Probability value

Effect of treatment on HR, BP, MAP, SaO_2, PaO_2, $PaCO_2$, and Horowitz index:

Mixed MANOVA indicated a non-significant interaction effect of treatment and time (F = 1.19, p = 0.34). There was a significant main effect of time (F = 3.88, p = 0.003). There was a non-significant main effect of treatment (F = 0.73, p = 0.66).

Within-group comparison

A significant increase in HR and DBP in group A was detected post-intervention compared to pre-intervention (p = 0.03 and p = 0.001, respectively); however, no significant changes were noted in group B (p > 0.05). Furthermore, a significant increase in SaO_2 was noted in both groups post-intervention compared to pre-intervention (p < 0.01). However, changes in systolic BP, MAP, PaO_2, $PaCO_2$, and the Horowitz index were not statistically significant in either group (p > 0.05).

Between-group comparison

No significant difference was indicated across groups pre-intervention (p > 0.05). Comparing both groups post-intervention indicated no significant difference in HR, BP, MAP, SaO_2, PaO_2, $PaCO_2$, and the Horowitz index (p > 0.05) (Tables 2-3).

Table (2): Mean HR, BP and MAP pre- and post-intervention of group A and B:

	Pre treatment	Post treatment			
	Mean ±SD	**Mean ±SD**	**Mean difference**	**% of change**	**p value**
HR (beats/min)					
Group A	83.05 ± 18.82	87.95 ± 21.39	-4.90	5.90	0.03
Group B	85.10 ± 15.44	86.65 ± 20.07	-1.55	1.82	0.49
MD	-2.05	1.3			
	p = 0.71	***p = 0.84***			
Systolic BP (mm Hg)					
Group A	129.50 ± 18.77	127.50 ± 13.72	2.00	1.54	0.46
Group B	127.50 ± 15.17	128.50 ± 15.23	-1.00	0.78	0.71
MD	2	-1			
	p = 0.71	***p = 0.83***			
Diastolic BP (mm Hg)					
Group A	73.00 ± 9.23	77.50 ± 7.86	-4.50	6.16	0.001
Group B	75.90 ± 6.91	75.40 ± 6.96	0.50	0.66	0.69
MD	-2.9	2.1			
	p = 0.27	***p = 0.38***			
MAP (mm Hg)					
Group A	92.17 ± 10.33	94.50 ± 9.75	-2.33	2.53	0.08
Group B	91.38 ± 10.71	90.80 ± 9.26	0.58	0.63	0.65
MD	0.79	3.7			
	p = 0.82	***p = 0.23***			

SD, Standard deviation; p value, Probability value

Table (3): Mean SaO_2, PaO2, PaCO2 and Horowitz Index pre- and post-intervention of both groups:

	Pre treatment	Post treatment			
	Mean ±SD	**Mean ±SD**	**Mean difference**	**% of change**	**p value**
SaO_2 (%)					
Group A	95.35 ± 4.42	97.86 ± 2.05	-2.51	2.63	0.002
Group B	96.25 ± 2.97	98.85 ± 1.53	-2.60	2.70	0.001
MD	-0.9	-0.99			
	p = 0.45	***p = 0.09***			
PaO_2 (mm Hg)					
Group A	113.09 ± 43.58	122.28 ± 41.65	-9.19	8.13	0.33
Group B	119.39 ± 38.37	124.84 ± 37.82	-5.45	4.56	0.56
MD	-6.3	-2.56			
	p = 0.63	***p = 0.84***			
$PaCO_2$ (mm Hg)					
Group A	31.93 ± 4.96	30.91 ± 5.27	1.02	3.19	0.29
Group B	32.85 ± 5.85	31.62 ± 5.80	1.23	3.74	0.21
MD	-0.92	-0.71			
	p = 0.59	***p = 0.68***			
Horowitz index					
Group A	250.80 ± 122.75	267.58 ± 115.11	-16.78	6.69	0.42
Group B	265.26 ± 96.62	283.65 ± 113.80	-18.39	6.93	0.38
MD	-14.46	-16.07			
	p = 0.68	***p = 0.66***			

SD, Standard deviation; p value, Probability value

DISCUSSION

This randomized controlled study evaluated the immediate impacts and safety of manual hyperinflation (MHI) in mechanically ventilated (MV) patients with SAP. While MHI has been investigated in other critically ill populations, such as post-cardiac surgery patients, those with ventilator-associated pneumonia, and traumatic brain injury, to the author's knowledge, this research is the first one to determine its efficacy specifically on MV patients with SAP.

The current findings demonstrated no significant changes across groups post-intervention ($P > 0.05$), which could be explained by the immediacy of evaluation following only a single session.

Regarding hemodynamic response, both groups showed no significant variations in SBP or MAP. However, the control group exhibited a significant increase in HR and DBP, whereas no significant differences were indicated in these parameters in the study group. Despite these variations, all values remained within physiological limits in both groups, suggesting that MHI does not compromise cardiovascular safety in this population. These findings align with a previous study of sedated septic shock patients undergoing MHI with rib cage compression, which reported no significant changes in HR, SBP, DBP, or MAP. Additionally, research conducted on pediatric pneumonia patients comparing MHI with and without suction found no hemodynamic disturbances in both groups when peak inspiratory pressure remained below 40 cm H_2O [17,18].

Conversely, CPT appears to induce more pronounced cardiovascular responses. A prior study indicated significantly higher HR in the CPT group in comparison to the MHI group, concluding that MHI during postural drainage was better tolerated and produced less cardiovascular stress in chronic obstructive pulmonary disease (COPD) patients, which supports the present study results [19]. Another study noted that CPT caused transient increases in HR and BP in ventilated patients, although these values returned to baseline within 15 minutes [20].

In contrast to the current findings, a study involving MV patients following valve replacement surgery reported a significant increase in HR along

with a reduction in both SBP and DBP after MHI. Variations in patient characteristics, particularly the presence of cardiac pathology, may account for these differences [7]. Additionally, another study conducted in a general ICU setting observed significant elevations in HR, SBP, and DBP when MHI was applied in combination with thoracic compression and suction. This discrepancy could be explained by differences in intervention protocols, including multiple follow-up assessments, unlike the immediate evaluation used in the present study [21].

In terms of oxygenation, both groups demonstrated a significant increase in oxygen saturation (SpO_2), with percentage increases of 2.63% in the control group and 2.7% in the study group. These findings are corroborated by earlier research, which demonstrated that a single session of MHI integrated with expiratory rib compression can effectively enhance SpO_2 in MV, sedated patients with septic shock [17] and in general ICU patients receiving MHI alongside thoracic compression and suction [21]. In contrast, a study involving MV patients following cardiothoracic surgery reported no significant change in SpO_2 when MHI was applied in isolation [7].

In addition to oxygen saturation, the study evaluated more direct measures of gas exchange. No significant changes in arterial oxygen pressure (PaO_2) were observed in either group after a single session of conventional physiotherapy or MHI. The results could be explained by the use of endotracheal suctioning (ETS) immediately following MHI, a common clinical practice. As ETS is known to transiently induce hypoxemia through mechanisms such as alveolar de-recruitment or interruption of ventilation, any immediate improvements in PaO_2 by MHI may have been weakened or masked by the subsequent suctioning, potentially impeding the observation of a statistically significant increase in PaO_2 [22,23].

In the present study, the two groups showed no statistically significant variation in the Horowitz index, although a greater increase was noted in the study group. This suggests a probable benefit of MHI that did not reach statistical significance after a single intervention. These results are aligned with an earlier study that reported an increase in the Horowitz index following MHI and ventilator hyperinflation (VHI) at different levels of PEEP in MV general ICU patients, though the change wasn't statistically significant [24]. Conversely, a study involving MV critically ill patients recovering from septic shock and presenting with acute intrapulmonary lung injury reported a significant decrease in the Horowitz index following MHI, indicating potential variability in response depending on patient condition and lung pathology [25].

Similarly, the present study did not demonstrate any significant changes in arterial carbon dioxide pressure ($PaCO_2$) in either group. This finding is consistent with a previous study that compared the impacts of MHI and VHI in paralyzed MV patients during the initial postoperative phase following mitral valve replacement, which also reported no significant changes in $PaCO_2$ in both groups [22].

However, this finding contrasts with a study on patients with acute lung injury, where all intervention groups, including suction alone, positioning with suction, and positioning with MHI and suction, showed significant increases in $PaCO_2$ [26]. These differences may be due to variations in intervention protocols, patient severity, or timing of measurements.

The current study indicated that MHI was proven safe and didn't alter hemodynamic parameters, making it a valuable option for this vulnerable patient population. Its efficacy in improving respiratory function without the risk of cardiovascular instability highlights its potential to be a standard, low-risk component of care that can contribute to better patient outcomes.

Several limitations were noted in this study: the small sample size, which may have made it more difficult to identify minor differences, and the absence of long-term follow-up. Additional research is necessary to assess the impacts of repeated MHI interventions on long-term outcomes for patients with SAP, particularly regarding time to extubation and length of hospital stay.

CONCLUSION

Applying manual hyperinflation in addition to conventional chest physiotherapy is an effective and safe technique and has no adverse effects on SAP patients. Additionally, it is effective in significantly increasing oxygen saturation and improving oxygenation, although this increase was not statistically significant.

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