

Coastal Critical Care Patients: Effect of Manual Chest Compression to Decrease Lung Disability Among Vulnerable Patients on Mechanical Ventilator-

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Abstract

Background: Patients with invasive mechanical ventilation are susceptible to a number of respiratory issues, with secretory retention representing one of the most common. Clinically, secretion buildup may result in bronchial blockage and deteriorating gas exchange, and in some extreme circumstances, may impair ventilator support, extending the time required for mechanical ventilation and increasing death. Re-expansion methods like the Manual Chest Compression Maneuver can be employed to stop this, This technique supports the re-expansion of collapsed alveoli. **Methods:** total of 15 patient receiving Mechanical Ventilator support in an intensive care unit were included in this study with signs of hypoventilation, hypo expansion, or atelectasis were eligible to receive the Manual Chest Compression maneuver. Manual Chest Compression maneuver were performed for 10 minutes, after the maneuver the outcomes such as Tidal Volume, Minute ventilation, Fraction of inspired oxygen are recorded. **Result:** Manual Chest Compression Maneuver applied for the patients had positive effect on improving the patients Tidal Volume, Minute ventilation, Fraction of inspired oxygen. Based on the statistical analysis of pre and post interventions of the Manual Chest Compression Maneuver the statistical difference is $P < 0.05$. **Conclusion:** In patients admitted in a coastal hospital, , Manual Chest Compression Maneuver had positive effect on patients' Tidal Volume, Minute ventilation, Fraction of inspired oxygen. This effect may help the patient in early weaning off from the mechanical ventilator.

1. Introduction:

Coastal patients with invasive mechanical ventilation are susceptible to a number of respiratory issues, with retention of secretion, being the most common. Clinically, secretory build up may result in bronchial obstruction, deterioration of gas exchange, and, in certain serious cases, the heart's ability to pump blood. The blood pressure may drop or your heart rate may increase if your heart is not functioning properly. Although if a lot of oxygen is flowing into your lungs, these changes might also imply that less oxygen gets to your blood (decreased perfusion). Changes in

intrathoracic pressure and lung volume caused by spontaneous and mechanical ventilation have independent effects on the key determinants of cardiovascular performance— preload, afterload, heart rate, and myocardial contractility. Intracardiac pressure fluctuations affect a variety of intrathoracic structures, notably the heart, pericardium, and key arteries and veins. A negative pleural pressure is created during spontaneous inspiration, and the right atrium is the recipient of the decreased intrathoracic pressure. In contrast, intermittent positive pressure ventilation (IPPV) induces right atrial pressure (PRA) and intrathoracic pressure to go up during the inspiratory

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timing, and both of these pressures keep rising throughout the duration of breathing if positive end expiratory pressure (PEEP) is added. Intrathoracic pressure and lung volume changes can have a significant influence on individuals with congenital heart disease, or a pulmonary pathology, or both. These modifications may have an impact on ventilator support performance, which leads to a prolonged period of mechanical ventilation (MV) and increased mortality. Acute renal failure, the degree of gas exchange impairment, and poor cardiac outputs are risk factors for patients on a mechanical ventilator in the ICU². Mechanical ventilation might cause a reduction in urination. This may also be connected to increases in plasma antidiuretic hormone and decreases in atrial natriuretic peptide that happen with mechanical breathing. This is largely related to reduce renal perfusion caused by lower cardiac output. Due to reduced urine production, excessive IV fluid administration, and the elimination of insensible water loss from the respiratory tract as a result of humidifying the inspired gas, fluid overload is a common occurrence during mechanical ventilation². Gastric distention (meteorism) can occur in patients receiving mechanical ventilation. Patients who are mechanically ventilated might potentially develop stress ulcers and experience gastrointestinal bleeding, hence stress ulcer prevention should be given². Critical sickness and weakness are more likely to occur in people who are mechanically ventilated (polyneuropathy and polymyopathy). Diaphragm dysfunction brought on by the ventilator may happen if the respiratory muscles are not recruited during mechanical ventilation (paralysis)². Those who use mechanical ventilation might not have typical sleep cycles. Delirium, patient-ventilator asynchrony, and sedation-induced ventilator reliance can all be results of sleep deprivation. Typically, endotracheal or tracheostomy tubes are used to mechanically ventilate critically sick patients. These patients are currently at risk for all consequences related with artificial airways, including laryngeal edema, tracheal mucous trauma, lower respiratory tract infection, loss of the upper airway's moisturising function, and other communication issues. To treat respiratory insufficiency and lessen the effort required to breathe, a mechanical ventilator is crucial. It typically occurs in conjunction with sedation, or neuromuscular blockade (NMB), which results in atelectasis, diaphragm atrophy, and decreased functional residual capacity. Hypoventilation (commonly in dependent regions of

the lung) is also caused by this condition. Using sedatives, NMB. Corticosteroids, infections, and the patient's prolonged immobility are all hazards for acquiring "intensive care unit acquired weakness" (ICUAW), that is linked to respiratory muscle weakness¹. This illness causes diaphragmatic atrophy and hypoventilation, necessitating prolonged mechanical ventilation. The re-expansion process, which starts with hypoventilation and atelectasis, can be stopped using techniques like the manual chest compression manoeuvre. The manual chest compression technique promotes the re-expansion of collapsed alveolar units via collateral channels, the alveolar interdependence mechanism, and alveolar surfactant renewal.^{1,3,4}. Acute renal failure, the degree of gas exchange impairment, and poor cardiac outputs are risk factors for patients on a mechanical ventilator in the ICU². In the ICU environment, additional chest physiotherapy procedures like chest percussion are often employed, however manual chest compression has not been utilized as frequently as the other chest physiotherapy approaches. The manual chest compression maneuver makes the respiratory system's elastic elasticity stronger, which raises negative pleural pressure and, in turn, the Trans pulmonary pressure¹. The manual chest compression maneuver also activates the atelectatic alveolus. Other significant benefits of the manual chest compression maneuver include improved ventilator perfusion ratio and oxygenation, reduced shunts and respiratory effort, and the avoidance of barotrauma and disruption of the alveolocapillary barrier without increasing airway pressure¹.

The purpose of the present investigation is to determine the impact of manual chest compression on lung function in individuals who receive a mechanical ventilator.

2. Methodology:

The study was a single group pre and post experimental study involving consecutive sampling and was conducted at our institute. Informed consent was obtained from the patient's attender. Total number of 15 subjects were involved in this study, The subjects that we included in this study were both male and female who were aged between 30 and 70 and subjects who were under the support of mechanical ventilator (SIMV mode). Exclusion criteria for this current research were patients with Rib fracture, osteoporotic, subjects who underwent any cardiac surgeries and

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pulmonary surgeries, subjects with ICT, pneumothorax and hemodynamic instability were excluded from this study. The outcome data recorded are tidal volume, minute ventilation, Fraction of inspired Oxygen. The pre and post interventions data are mentioned below.

After being included, the patients are placed in 30° semi fowlers, supine position and the baseline data was recorded from the ventilator monitor. Moreover, the manual chest compression maneuver was constantly carried out for 10 minutes. After 10 minutes the post intervention data such as tidal volume, minute ventilation, Fraction of inspired Oxygen. from the ventilator monitor was recorded. This technique was performed for 4 sessions per day and for each sessions the tidal volume, minute ventilation, Fraction of inspired Oxygen data was collected.

MANUAL CHEST COMPRESSION:

When using the manual chest compression technique, the ribcage is compressed during the expiratory phase and held in place until the first third of the inspiratory phase, at which point the compression is suddenly released. The subjects will be held supine at a 30° semi-fowlers angle. Bilaterally, manual pressure is given

over the thorax's lower third. In order to match the manoeuvre pace with the subject's breathing frequency, the therapist squeezes the rib cage with both hands while the patient is exhaling⁹. The air is displaced when the chest pressure in the manoeuvre application area changes, expanding the chest.^{1,3,4,5}.

3. Result:

Total of 15 number of patients who were under the support of mechanical ventilator were included in this study. The mean time of the ICU stay of the patient included for this study was 4 days and their support on mechanical ventilation (SIMV) was also 4 days.

Data were analyzed by paired t test to compare the pre and post intervention data of the group. The results are presented as mean and standard deviation, and statistical significance was considered when $p < 0.05$. The data analysis are mentioned in Table:1.1, 1.2, 1.3, 1.4. And comparison between the pre and post intervention datas are mentioned in Graph.1.1, 1.2, 1.3, 1.4.

Table: 1.1. Inspiratory Tidal Volume

S. NO	OUTCOME	MEAN	STANDARD DEVIATION	t VALUE	SIGNIFICANCE
1.	Pre	310.6	6.79	4.02	0.001
2.	post	321.4	7.10		

Table: 1.2. Expiratory Tidal Volume

S. NO	OUTCOME	MEAN	STANDARD DEVIATION	t VALUE	SIGNIFICANCE
1.	Pre	306.6	7.73	3.7	0.002
2.	post	318.6	9.51		

Table: 1.3. Fraction of Inspired Oxygen (FiO₂)

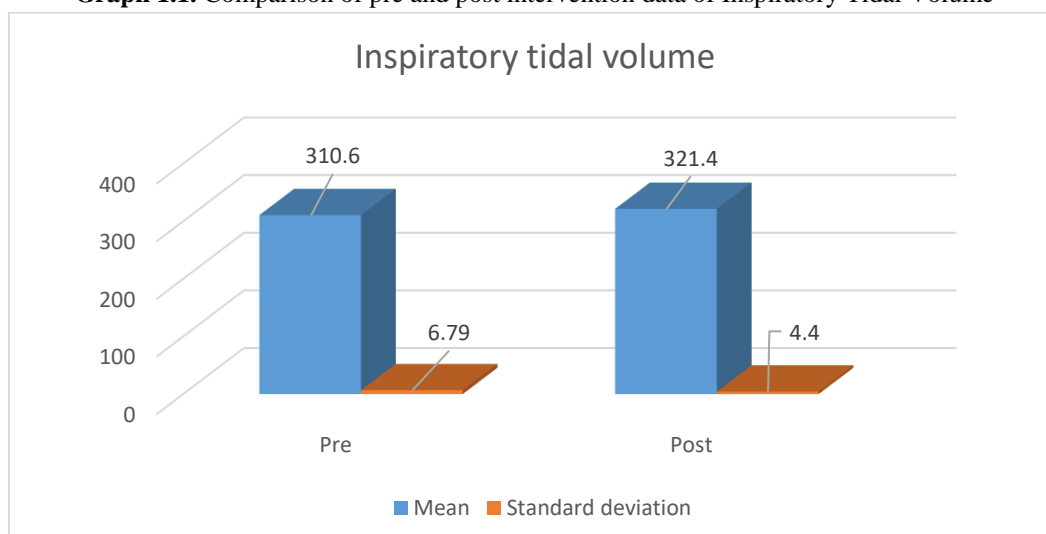
S. NO	OUTCOME	MEAN	STANDARD DEVIATION	t VALUE	SIGNIFICANCE
1.	Pre	18.8	4.84	3.4	0.003
2.	post	23.8	2.26		

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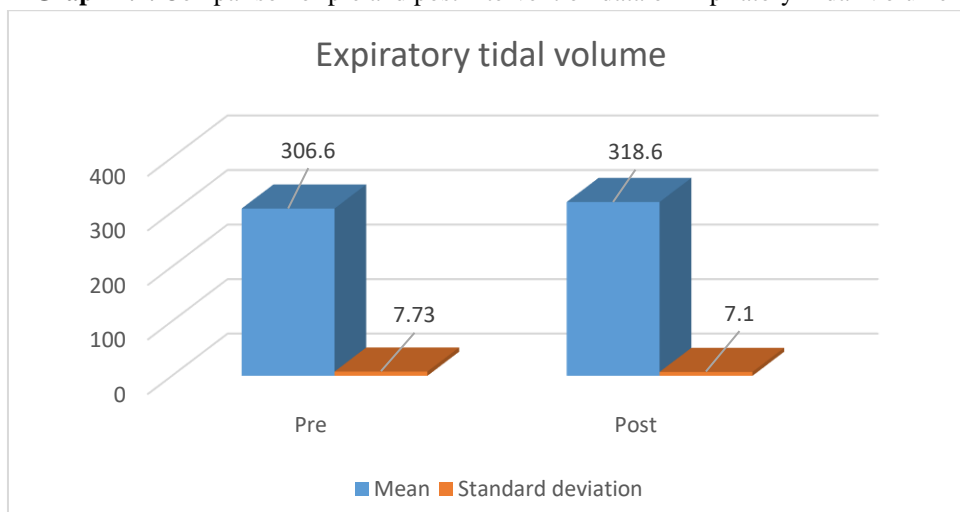
Table: 1.4. Minute Ventilation

S. NO	OUTCOME	MEAN	STANDARD DEVIATION	t VALUE	SIGNIFICANCE
1.	Pre	7.72	0.53	6.2	0.0001
2.	post	8.16	0.5		

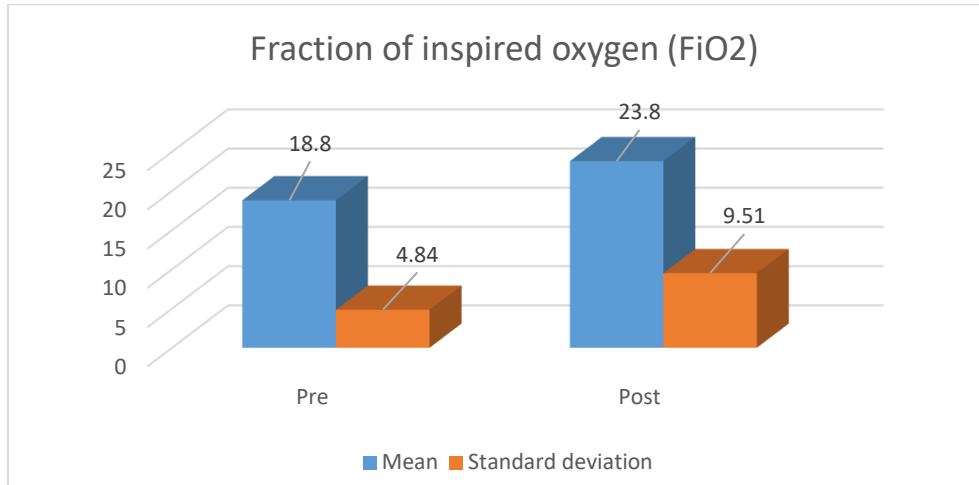
Graph 1.1. Comparison of pre and post intervention data of Inspiratory Tidal Volume



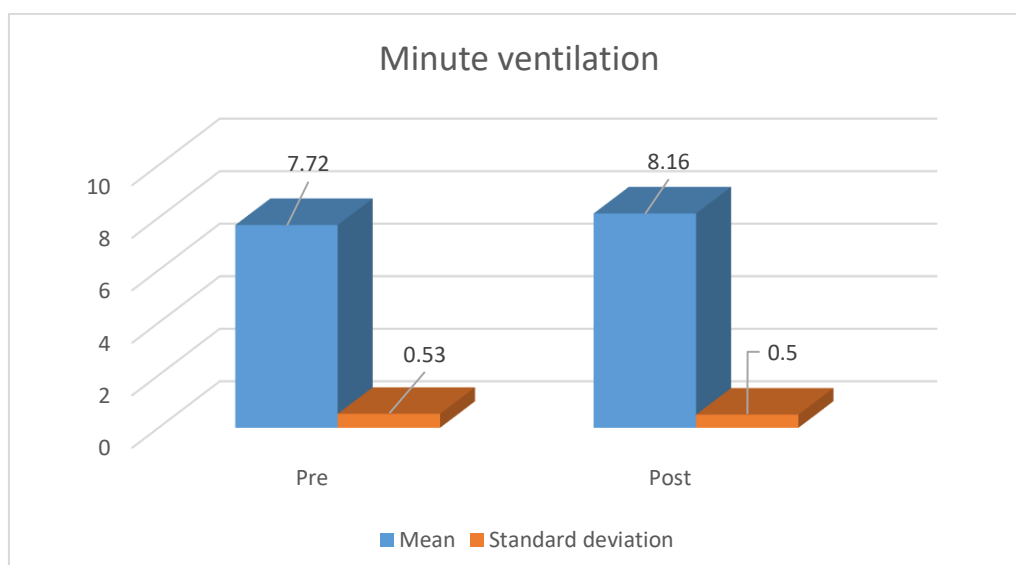
Graph 1.2. Comparison of pre and post intervention data of Expiratory Tidal Volume



Graph 1.3. Comparison of pre and post intervention data of Fraction of Inspired Oxygen (FiO₂)



Graph 1.4. Comparison of pre and post intervention data of Minute Ventilation



Based on the statistical analysis of the data, the result of the intervention which was performed for a duration of 10 minutes showed that there was an increase in the lung function such as inspiratory tidal volume, expiratory tidal volume, fraction of inspired oxygen (FiO₂) and minute ventilation.

4. Discussion:

The present study shows that manual chest compression increases the inspiratory tidal volume, expiratory tidal volume, fraction of inspired oxygen and minute ventilation during the application of the maneuver. In this study this maneuver had no effect on respiratory rate (RR), SpO₂, systolic blood pressure

(SBP) or heart rate (HR), due to the recoiling nature of the lung and thoracic cage while applying this maneuver it increases the elastic strength of the respiratory system. This in turn caused increased negative pleural pressure, resulting in the alveoli to recruit more ventilation inside the atelectasis areas resulted in improving the lung functions for the weaning off. In this study the manual chest compression was applied continuously for four times a day with an interval of 2 hours in between each interventions for four consecutive days. There was an increase in lung function while comparing the pre intervention data with the post intervention data of fourth day.

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Sedation, respiratory comorbidity, or obesity had no impact on the effectiveness of manual chest compression and decompression, and the effect of manual chest compression and decompression was not sustained after 10 minutes (Biarzi et al., 2022). The inspiratory and the expiratory tidal volumes, along with dynamic compliance were all enhanced by manual chest compression and decompression.

In a previous study, Bousarri et al (2014) The ongoing rib cage compression up to 25 minutes after suctioning is revealed, and this could be a positive indicator for an improvement in vital signs. Rib cage compression thereafter suctioning compared to 25 minutes after that showed no significant change.³

Patients on mechanical ventilation frequently develop atelectasis (Maa et al. 2005; Via et al 2012) and are at risk of lung infection¹⁸. Hypoventilation is a major cause of atelectasis since it reduces inspiratory tidal volume; hence, physical chest compression and decompression play a significant role in re expanding the afflicted portions of the lung^{(Costa 1999).11}.

Manual chest compression and decompression maneuver (MCCD) increased the lung volume in patient under the support of mechanical ventilation (Della Via et al 2012) and its effect was observed for 40 minutes after the maneuver²⁷.

Evaluating with the other respiratory variables like peak pressure and SpO₂ there was no change, but there was increase in lung compliance while applying this manual chest compression and decompression (MCCD), the increase in lung compliance was due to the increase in tidal volume, these changes are caused due to the pressure exerted during the intervention of this maneuver, which also increases the trans pulmonary pressure (Sarmiento et al., 2006)²².

Manual chest Compression is often administered either gradually and continuously during the expiratory phase or suddenly and quickly just at the beginning of expiration (A Carolina. et al 2018). We opted for the latter in the current investigation because, in comparison to the former, studies using mechanical models and animal models have shown it to be hemodynamically safe and more successful in terms of an increase in PEF and secretion clearance³¹.

Intervention of this maneuver on younger samples may have effective lung outcomes, but due to the dialyzing

protocols of the subjects involved in this study, the technique was not able to be applied continuously for four days, so the samples for this study were limited. Also, the age-related problems on these samples caused a larger number of samples to be excluded from this study. To get the desired effect of this technique, this maneuver can be used on larger sample groups. Because of the critical condition of the subjects, obtaining informed consent from their relatives made the study use limited samples.

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5. Conclusion:

As the technique of manual chest compression maneuver increases the inspiratory tidal volume, expiratory tidal volume, fraction of inspired oxygen (FiO₂), minute ventilation, this technique may be expected to be helpful in the early weaning off from the ventilator. Based on the results obtained from this study the manual chest compression maneuver is helpful in increasing the lung function. If the patient is osteoporotic then there may be the possible complication of rib fracture may occur.

CONFLICT OF INTEREST: Nil

FUNDING: NIL

Ethical issues – approval yes

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