

Cardiorespiratory Response to Chest Physiotherapy in Intensive Respiratory Care Unit

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ABSTRACT

Background: Physiotherapy intervention is regarded as an important component in the management of patient in intensive care unit. However, there are fears that chest physiotherapy results in adverse physiological changes with clinically significant alterations in hemodynamic and respiratory parameters. With contrasting studies regarding its threat to the hemodynamic stability in patients in intensive care unit, the study aims to evaluate the effect of chest physiotherapy on the cardiorespiratory variables in patients admitted in intensive respiratory care unit.

Methodology: 40 patients admitted in intensive respiratory care unit were included in this study. Pulse rate, respiratory rate, blood pressure, oxygen saturation was noted prior and immediately, at 5 minutes, 15 minutes and 30 minutes post chest physiotherapy. The patients were divided into two groups those with ventilatory support and those who were spontaneously breathing.

Results: Repeated measures ANOVA was applied using SPSS16 with a level of significance at $p < 0.05$. A statistical significant rise was found in systolic blood pressure and heart rate in both groups immediately after chest physiotherapy. The values returned to baseline level within 15 minutes of therapy. No adverse changes were observed during or post 30 minutes of intervention. Rise in heart rate and blood pressure were well within the physiological range in both the groups. Oxygen saturation increased significantly in both the groups till 30 minutes after chest physiotherapy. A significant difference was obtained only in systolic blood pressure values between the two groups.

Conclusion: Chest physiotherapy can be safely administered to patients with acute pulmonary conditions in the intensive care unit with or without mechanical ventilation. The change in cardiorespiratory parameters is well within the physiological limit. Oxygen saturation remains significantly improved for 30 minutes post chest physiotherapy.

Key words: Haemodynamic, respiratory, chest physiotherapy, intensive care unit

INTRODUCTION

With thrust towards evidence based practice and health care, there are major implications for chest physiotherapy in intensive care unit. ^[1] Critical care therapy and respiratory care section of National Institute of Health (NIH) defines chest physiotherapy as an aspect of bronchial hygiene that aims at moving bronchial secretions to the central airways via gravity, external manipulation of the chest and

eliminate secretions by cough or by suctioning. ^[2] The intensive care unit (ICU) is perceived as a daunting environment. The patients present with a complexity of problems which may be multisystem in origin and a continuous use of an updated and advanced technology for treatment and monitoring. ^[3] The physical therapist plays a pivotal role in providing care to minimize complications, reduce the ventilator load

and mobilize the patients at earliest in the ICU. [4]

Physiotherapy is often considered to be a threat to hemodynamic stability. Hammom et al in his study claims, that in intensive care, physiotherapy intervention results in adverse physiological changes i.e. clinically significant alterations in hemodynamic, respiratory or intracranial parameters necessitating remedial intervention. [5] They have also found occurrence of arrhythmias after intervention. In contrast, other studies have found beneficial or minimal adverse effects during physiotherapy intervention (Berney and Denhey 2003). With contrasting views on the hemodynamic stability, this study aims at evaluating the immediate effect of chest physiotherapy on cardiorespiratory parameters in patients admitted for acute respiratory impairment, with and without ventilatory support.

METHODOLOGY

It was a prospective interventional observational study carried out in Intensive Respiratory Care Unit (IRCU) of tertiary care hospital. The study was approved by Ethics Committee for Research on Human Subjects. Forty subjects admitted in IRCU with and without ventilator support referred for chest physiotherapy were selected for this study. Subjects with neurological involvement like GBS, stroke, Head injuries, and with multiple organ failure and in whom chest physiotherapy was absolutely contraindicated were excluded. A written informed consent was taken from the patient and the care giver. The patients were assessed as per the need of Chest physiotherapy (CPT) based on auscultation findings and x-ray. Baseline parameters which included heart rate, Blood pressure, respiratory rate and oxygen saturation were recorded. Bronchodilator Nebulization was not administered before the study intervention as it may affect the haemodynamic changes.

Precautions were taken before and during the treatment intervention to avoid

any disconnection or kinking of central lines and intravenous lines if present. The patients were connected to a cardio scope so that parameters could be continuously monitored. The treatment time was well spaced from the meal times and nasogastric feeds. Care was taken to ensure patients comfort for positioning. Treatment procedure was explained to conscious and oriented subjects.

Chest physiotherapy was provided as per evaluation findings based on auscultation, X-ray and the area of lung involved. A modified postural drainage position with a 15 degree of head down tilt was employed, along with percussions and vibrations. Suctioning and or Forced Expiratory techniques were used for facilitating secretion removal. Suctioning with strict aseptic measures was carried out in intubated patients and those with ineffective cough reflex. Care was taken to limit suctioning to 15 seconds at a time. It was repeated if required. Pre oxygenation was ensured to avoid any adverse events. Huffing and coughing was taught to the subjects with effective cough reflex. The sequence of the treatment was kept constant and end points of the treatment pre-defined. In case of bilateral lung involvement, the patient was taken to the alternate position and the above procedure was repeated. The treatment was discontinued if the subjects reached the predefined end points i.e. signs of improved ventilation on auscultation and reduced or no secretions.

The chest physiotherapy session was terminated if the patient complained of fatigue or in case of adverse events. Adverse events were defined as: a. Alteration in blood pressure > or < 20% of resting values which necessitates stopping intervention or requires remedial intervention (e.g. inotropes). b. Alteration in heart rate > or < 20% of resting values which necessitates stopping intervention or requires remedial intervention. c. New arrhythmia (e.g. atrial fibrillation, increased number of ectopic beats per minute, ST depression or elevation, increased magnitude. of ST

depression, bigeminy, trigeminy, ventricular tachycardia, ventricular fibrillation, asystole).

d. Desaturation of oxyhaemoglobin >10% of baseline levels or a figure which necessitates stopping intervention or requires remedial intervention. e. Pulmonary artery pressure (systolic) over 60mmHg. f. Pneumothorax detected immediately following intervention. g. Agitation resulting in detachment of equipment or lines or requiring increased sedation. Episode related to incorrect procedure (e.g. incorrect connection of equipment, level of inspired oxygen too high).

Heart rate, blood pressure, respiratory rate, oxygen saturation were recorded at four time intervals (immediate, post 5mins, 15mins & 30 mins) after the intervention along with continuous monitoring of ECG in both the groups on ventilator support and spontaneously breathing patients and the change was compared between the two groups.

STATISTICAL ANALYSIS:

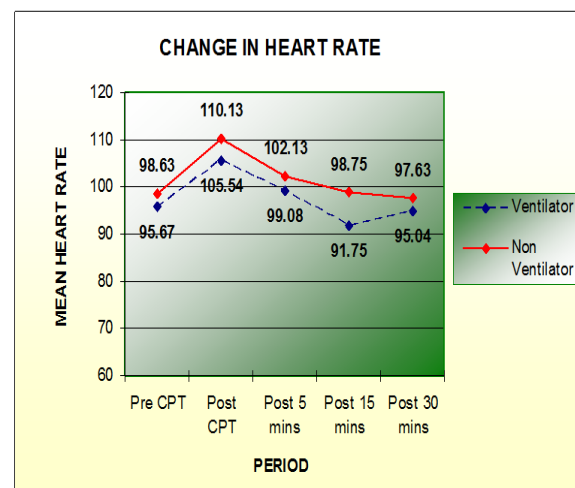
Statistical analysis was done using SPSS version 16. Repeated measures of ANOVA was used to test the changes in cardiorespiratory parameters (heart rate, systolic and diastolic blood pressure, respiratory rate and oxygen saturation) within the group at all-time intervals. Between the group comparison of ventilator support (VS) and spontaneously breathing (SB) patients was done by calculating the mean difference and unpaired test was applied. The level of significance was kept at $p < 0.05$ at 95% confidence interval

RESULTS

Out of total 40 patients 17 patients were diagnosed as obstructive disease, 11 as restrictive disorder and 12 were mixed. Associated co morbidities were as cor pulmonale (n=2), cardiac (n=17), Hypertension (n=7), Ischemic heart disease (n=7) and diabetes mellitus (n=12). All patients were on bronchodilators and antibiotics. 24 were on ventilator support

and 16 were spontaneously breathing. The mean age of the patients in VS was 52.08 +/- 18.7 and that of SB group was 49.06 +/- 22.83 years. There were equal number of males and females in VS group (n=12 each) and 9 males and 7 females in SB group. Out of the 24 patients in VS, 14 patients were on synchronized intermittent mandatory ventilation, 7 were on continuous positive airway pressure and 3 were on noninvasive positive pressure ventilation. The patients on VS required a mean fraction of inspired oxygen (FiO₂) of 0.10 +/- 0.48 in the range of 0.35 to 0.70 and patients who were SB needed mean Fi O₂ of 0.11 +/- 0.38 with a range of 0.24 to 0.60. The mean treatment duration was 30.4 +/- 11.02 minutes. The groups were comparable at baseline level for all variables. A significant increase in heart rate, systolic blood pressure and respiratory rate was observed immediately post chest physiotherapy. All the above parameters did not show significant change from 5 minutes to 30 minutes post chest physiotherapy.

HEART RATE: A significant increase ($p < 0.05$) in heart rate was observed immediately post chest physiotherapy in both the ventilated and spontaneously breathing patients (Graph 1). The change in heart rate post 5 minutes to 30 minutes of chest physiotherapy was not significant in both the groups. The change in heart rate when compared between the two groups was not statistically significant.

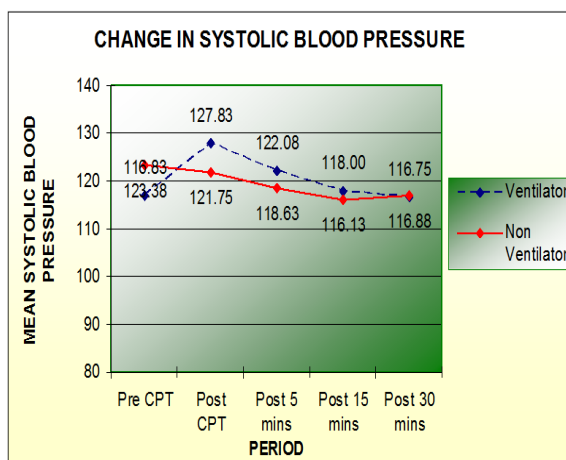


Graph 1: Heart rate changes with chest physiotherapy

BLOOD PRESSURE:

A significant increase ($p < 0.05$) in systolic blood pressure was seen immediately post and 5 mins post chest physiotherapy in VS group which returned to baseline value in 30 minutes after the intervention. However, the increase in systolic blood pressure immediately post chest physiotherapy in SB patients was not statistically significant ($p > 0.05$) (Graph2). A significant fall in systolic blood pressure was observed after 15 minutes in this group and it was maintained till 30 minutes after the intervention.

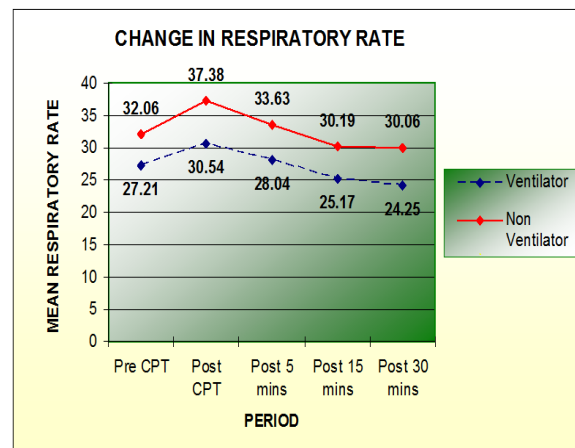
When comparing the change in systolic blood pressure between the two groups it was statistically significant immediately post chest physiotherapy and 5 minutes post. No statistically significant change was observed in diastolic blood pressure in VS and SB group at any of the time intervals after chest physiotherapy. The change when compared between the two groups was also not statistically significant



Graph2: Systolic Blood Pressure changes with chest physiotherapy

RESPIRATORY RATE:

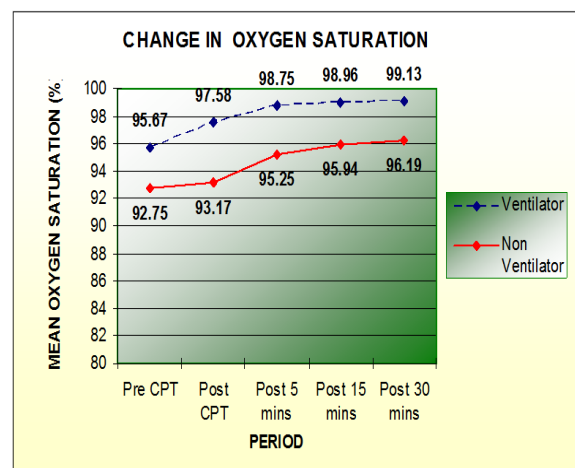
No statistically significant change was observed in respiratory rate in VS and SB group at any of the time intervals after chest physiotherapy (Graph 3). The change when compared between the two groups was also not statistically significant.



Graph 3: Respiratory rate changes with chest Physiotherapy.

OXYGEN SATURATION:

A significant increase ($p < 0.05$) in oxygen saturation was observed from post 5 minutes to post 30 minutes of chest physiotherapy compared to baseline values (Graph 4). Intra group comparison of VS and SB patients showed no significant difference ($p > 0.05$) in heart rate and respiratory rate post physiotherapy between the two groups. There was no significant difference in the amount of rise in oxygen saturation compared between the two groups



Graph 4: Changes in oxygen saturation with Chest Physiotherapy.

ADVERSE EVENTS:

As seen in Table 1 patients did not show any of the adverse events during and up to 30 minutes post chest physiotherapy. There were occasional ventricular premature contractions seen in 7.5% of patients during the treatment.

Table 1:

Adverse events	No. of patients
SBP < or > 20%	nil
Heart rate < or > 20%	nil
Fall in Oxygen saturation > 10%	nil
Arrhythmias	Occasional VPC (n=3, 7.5%)
Respiratory distress	nil

DISCUSSION

Our study examined the cardio respiratory changes to chest physiotherapy maneuvers in patients admitted with acute exacerbations of pulmonary conditions and compared the changes in patients on VS and SB. The outcome measures used were heart rate, blood pressure, respiratory rate and oxygen saturation. When studying the effect on heart rate, there was a significant rise in heart rate in both VS and SB groups immediately post chest physiotherapy, which returned to baseline value post 30 minutes of intervention. The change in heart rate was not statistically significant when compared between the groups at any time interval post chest physiotherapy. Chest physiotherapy (CPT) is a stressful procedure. Stimulation of sympathetic nerves during CPT releases the neural hormones which act to accelerate the depolarization of the Sino atrial node and cause heart to beat faster along with increased myocardial contractility due to release of catecholamine's. [6] The increase in metabolic demand during chest physiotherapy is the result of increased muscular activity as evidenced by the suppression of maximal oxygen consumption (VO₂) following the administration of the muscle relaxant. The increase in physiologic activity produced by chest physiotherapy is secondary to both exercise-like and stress-like responses. [7]

Katie Dallison et al (1998) reports a significant increase in heart rate after percussions in normal individuals due to stimulatory effect of percussions on ventilation leading to increase work of breathing and VO₂ to meet oxygen demands. [8] CPT causes greater increases above resting levels of VO₂, heart rate, and rate pressure product than does any other routine daily intensive care procedure. [9,10] Patti et

al have found that heart rate, systolic and mean blood pressures, and cardiac output were all increased during the CPT sessions. Mean increases of approximately 50% in cardiac output, 25% in mean and systolic blood pressure, and 15% in heart rate were observed. Major hemodynamic and metabolic stress caused by CPT can be attenuated but not abolished by prior administration of moderate IV dose of fentanyl. [11] This is generally done in patients with high intracranial pressures.

There was a significant increase in systolic blood pressure in ventilated patients immediately after chest physiotherapy and returned to baseline levels after 15 minutes of the intervention. The SB group did not show a significant change immediately post chest physiotherapy but post 5 minutes there was a significant drop in systolic blood pressure which was maintained 30 minutes post intervention. When compared between the two groups the change in systolic blood pressure was significant immediately post chest physiotherapy. Even though the changes in systolic blood pressure were statistically significant, the changes were within the physiological limit and were not clinically significant.

The decrease in cardiac output due to positive pressure is offset by a sympathetic compensatory response from the arterial baroreceptors. [12] Also, positive pressure results in increase in HF index of the baroreflex gain resulting in increase in blood pressure. [13] This combined with the effects of postural drainage such as increase in oxygen consumption, carbon dioxide production, and right atrial pressure and preload may be the cause for the rise in systolic blood pressure. With chest physiotherapy the stresses and muscular activity increases resulting in rise in stroke volume and cardiac output to meet the increased oxygen demands. Suctioning stimulates sympho-excitatory receptors localized in larger airways whose afferent fibres course within the vagus resulting in increased sympathetic activity which induces peripheral vasoconstriction and

elevates mean arterial blood pressure. No significant change was observed in diastolic blood pressure when observed in all the patients and both the groups individually.

On analyzing respiratory rate in this study, a significant increase was noted immediately post CPT, post 5, 15 and 30 minutes of intervention. The inter group comparison showed no statistically significant difference. Airway clearance techniques like postural drainage, percussions and vibrations causes increase in muscular activity resulting in rise in respiratory rate. Also the alteration imposed by changed in mechanics with positioning and altered load on respiratory muscles may have increased demand on breathing. This could be offset by the clearance of secretions. As the air flow improves the work of breathing is reduced, accessory muscle usage is eliminated and dyspnea is reduced. Hence the respiratory rate is maintained close to the baseline values. The non-significant change in respiratory rate could also be attributed to the incorporation of breathing exercises which would control expiratory and inspiratory flow and hence the rate.

A significant rise in oxygen saturation was seen throughout the time frame of intervention in both the groups. Postural drainage enhances peripheral lung clearance, increases functional residual capacity, and accelerates mucus clearance. Postural drainage in conjunction with mechanical ventilation and PEEP is thought to increase trans-pulmonary pressure, improve ventilation-perfusion ratios, increase lung/thorax compliance of the nondependent hemithorax, and reduce collateral airway resistance.

The responses obtained are a result of complex interactions because of positioning, percussions, vibrations and coughing. We did not study the change in cardiorespiratory parameters with an individual intervention however we monitored the patient responses up to 30 minutes of recovery after chest physiotherapy. During chest physiotherapy

and immediately post 5 mins there was a statistically significant rise in all parameters which gradually returned to baseline levels by 30 minutes however oxygen saturation continued to improve up to 30 minutes.

CONCLUSION

Chest Physiotherapy though causes an immediate increase in heart rate and systolic blood pressure; they are well within the physiologic limit. Parameters return to baseline within 15 minutes of therapy. There is improved Oxygen saturation which is maintained after 30 minutes of the therapy in both VS and SB patients. Occasional ventricular premature contractions were observed in 7.5% of patients during therapy. No other adverse events were noted during and 30 minutes after it. Multimodality Chest physiotherapy can be safely given to patients with acute exacerbations of pulmonary conditions with or without ventilator support in the intensive care unit with appropriate monitoring of cardio respiratory parameters.

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