International
Journal of Medical and Exercise Science
(Multidisciplinary, Peer Reviewed and Indexed Journal)

ORIGINAL ARTICLE

RESPIRATORY REHABILITATION OF MECHANICALY VENTILATED PATIENTS WITH ORGANOPHOSPOROUS POISIONING

Search engine:
www.ijmaes.org

Dr.Gitanjali Sikka[1] and Dr.Suman Mehra[2]

Corresponding Author: [1]Assistant Professor, College of Physiotherapy, Pt.B.D.S, University of Health Sciences, Rohtak, Haryana,India. Email: gitanjali.sikka@gmail.com

Co-Author: [2]Assistant Professor, College of Physiotherapy, Pt.B.D.S, University of Health Sciences, Rohtak, Haryana,India.

Abstract

Background and objectives: *Organophosphates (OP) are used as insecticides in agricultural and domestic settings throughout the world. Acute organophosporous (OP) pesticide self- poisoning is a major global problem. Early recognition of respiratory failure, prompt endotracheal intubation and mechanical ventilation are life- saving measures in severe OP poisoning. Patients with OP poisoning may have respiratory failure for many reasons, including aspiration of gastric contents, excessive secretions, pneumonia and septicemia complicating adult respiratory distress syndrome and thereby physical therapy may be indicated for patients in the intensive care setting.* ***Methods:*** *A total of twenty patients ranging in age from 25-45 years, mechanically ventilated for respiratory muscle paralysis, due to organophosporous poisoning, recruited from various ICU's were included in the study. Effects of physiotherapy treatment were studied on static lung compliance (C_{ST}), oxygenation ratio (PaO_2:FiO_2 ratio). Measurements of dependent variables were recorded (PRE) before commencement of treatment, 30 minutes and 60 minutes after treatment.* ***Results:*** *Analysis of variance showed that there was highly significant improvement in C_{ST} mean values ($p<0.01$). Comparing mean values with critical difference, significant critical difference was observed between mean values at PRE and Post-30, and between PRE and Post-60 time intervals ($p<0.05$) for C_{ST}, PaO_2:FiO_2 respectively.* ***Conclusion:*** *Respiratory rehabilitation of patients with organophosporous poisoning can be effectively achieved with employment of various physiotherapeutic techniques including manual hyperinflation and bronchial hygiene therapy.*

Keywords: *Manual hyperinflation, organophosphorous poisoning, static lung compliance, mechanical ventilation.*

Received on 09th Oct 2016, Revised 10th Nov 2016, Accepted on November 26th 2016

INTRODUCTION

Acute organophosporous (OP) pesticide self-poisoning is a major global problem[1]. Organophosphates (OP) are used as insecticides in agricultural and domestic settings throughout the world[2]. Most of the OP pesticide poisoning and subsequent deaths occur in developing countries following a deliberate self ingestion particularly in young, productive age group, as highly toxic pesticides are readily available at the moments of stress[3]. As nerve agents, they have also been used in warfare and terrorist attacks [2]. The mortality rate of OP poisoning is high: fatal issue is often related to a delay in diagnosis or an improper management[4] , deaths occur within hours of pesticide ingestion during the acute cholinergic crisis, either before or soon after reaching medical care[1]. Most result from acute respiratory failure due to central respiratory depression, respiratory muscle weakness, and /or direct pulmonary effects (bronchospasm and bronchorrhoea)[5].

Respiratory failure being major reason for mortality, careful monitoring, and appropriate management and early recognition of this complication may decrease the mortality rate among these patients. Patients with OP poisoning may have respiratory failure for many reasons, including aspiration of gastric contents, excessive secretions, pneumonia and septicemia complicating adult respiratory distress syndrome. Early recognition of respiratory failure, prompt endotracheal intubation and mechanical ventilation are life-saving measures in severe OP poisoning[4].

We present our experience with physical therapy management of patients in various Intensive Care Units (ICU's) of the hospital with severe organophosphorous poisoning and associated respiratory failure.

Materials And Methods

A total of twenty patients ranging in age from 25-45 years, who required, mechanical ventilation for respiratory muscle paralysis, secondary to organophosporous poisoning, seen during five months period, were recruited from various ICU's in the institute and were included in the study. All the patients included were mechanically ventilated on Hamilton Evita ventilator, on volume control (CMV) mode with PEEP<10 cmH2O and had stable hemodynamics with heart rate = 60-100 beats/min; MABP = 70-110 mm Hg. Patients with mild respiratory distress and not requiring ventilatory support were excluded from the study. Patients suffering with acute cardiac dysarrthymias/ added cardiac pathology, undrained pneumothorax, obstructive lung disease, haemoptysis of unknown cause, severe bronchospasm and any associated head injuries, chest wall deformity, history of smoking and impaired lung mechanics were excluded from the study.

Detailed history and physical examination, including the onset and nature of symptoms, general and systemic manifestations, laboratory investigations performed at admission included arterial blood gas (ABG) analysis, electrocardiogram (ECG), chest radiograph, serum biochemistry and haemogram were recorded for each patient.

All the patients were ventilated initially in volume controlled mode. With the improvement of respiratory paralysis, weaning was implemented through SIMV-CPAP modes. When successfully tolerated, they were put on T-piece trial. Extubation was done after clinical assessment and monitoring of arterial blood gas parameters by the medical personnel's. All patients received chest physiotherapy with maintenance of sepsis, and care of ETT.

The patients were observed for evidence of side effects such as ventilator associated pneumonia, septicemia or barotrauma.

Procedure

All the patients who met the inclusion criteria were evaluated thoroughly using an evaluation performa. Patients received chest physiotherapy intervention twice in a day. Effects of physiotherapy treatment were studied on dependent variables including static lung compliance (CST) and oxygenation (PaO_2:FiO_2). During the entire procedure, all precautions were undertaken to prevent infection to the patients. Heart rate (HR), electrocardiogram, mean arterial blood pressure (MABP), arterial blood oxygen saturation (SpO_2) and temperature were monitored using bedside monitor (Philips Intelli Vue MP 40, Philips International B.V., The Netherlands).

Patients were placed in the indicated postural drainage position for a time period of ten minutes, in accordance with the reporting of chest X-ray's. Bronchial hygiene therapy was given to all patients followed by endotracheal (ET) suctioning, approximately 30 minutes before physical therapy intervention, in order to remove excess secretions. Fifteen minutes before the intervention, FiO_2 of the ventilator was set as 1.0. After 15 minutes of being ventilated at FiO_2 1.0, a pre intervention (i.e. pre-hyperinflation) measure (PRE) of dependent variables was performed. After recording the pre-intervention (baseline) measurements of dependent variables, the patients were then given recruitment maneuver in form of manual hyperinflation.

Manual hyperinflation

Manual hyperinflation was performed using Mapleson-"C" circuit and a two liter reservoir bag (Intersurgical Mapleson C circuit with two liter bag) connected to 100% wall oxygen at 15 L per minute. The waveform consisted of an inspiration of three seconds, sustained inspiration for 2 seconds, and a fast release of the valve to ensure a short expiration, during which bag was held compressed. Expiration was passive and unobstructed to facilitate expiratory flow with no PEEP applied. The I: E ratio was 2:1. A manometer (Medisys pressure manometer with T –piece connector) was included in the circuit and patients were manually hyperinflated to a maximum peak airway pressure of 40cm H_2O.With a two - handed technique, six sets of six MHI breaths were delivered to the patient. Each MHI set was followed by six tidal breaths to a peak airway pressure of 20cm H_2O. The patients were suctioned three times throughout the procedure following every second set of hyperinflation breaths and treatment was of 20 minutes duration.

Measurements

Post intervention measures of the dependent variables were recorded at 30 minutes (POST-30) and 60 minutes (POST-60) after the intervention. Static lung compliance (CST) readings were recorded from the display on the ventilator. An average of three readings of static pulmonary compliance was taken. Arterial blood gas analysis samples were taken to monitor oxygenation (PaO_2:FiO_2). Heart rate (HR) and Mean arterial blood pressure (MABP) were read directly from the monitoring system and recorded before intervention(for Manual Hyperinflation intervention recording was done after disconnection from the ventilator); 1-minute during intervention; and 1, 5, and 20 minutes following intervention.

Data analysis

The data collected for different variables at different time intervals was subjected to statistical analysis using SAS software (version 6.0, Chicago, IL).Mean and standard deviation

of demographic variables were calculated for all patients. Measurements of different variables (CST , PaO_2/FiO_2 ratio, HR and MABP) before and after intervention were compared using One way Analysis of Variance, to test the effect of time interval on different variables and the time interval means were compared using critical difference (C.D.) values ($p<0.05$).

RESULTS

Twenty victims, following organophosporous poisoning were admitted to various ICU's of this Institute during the study period and were given chest physiotherapy management (Table 1). All the patients had history of consumption of different organophosporous compounds (Table 2). Analysis of variance showed that there was highly significant improvement in CST mean values ($p<0.01$) (Table 3). However, PaO_2:FiO_2 ratio could not reach statistical significance. CST mean values showed highly significant improvement at Post-30 and Post-60 time intervals after intervention ($p<0.01$). Comparing CST mean values in terms of percentage improvement, there was significant percentage increase in CST mean values at Post-30 (16.28%) and Post-60 (8.71%) time intervals. In addition, post-hoc analysis revealed that means had significant critical difference between each other ($p<0.05$).

For PaO_2:FiO_2 ratio mean values, the percentage increase in means at Post-30 and Post-60 time interval was by 41.28% and 16.47%., respectively. Also, there was significant critical difference between mean values observed at PRE and Post-30, and between PRE and Post-60 time intervals ($p<0.05$).

There were no adverse changes in blood pressure, heart rate, or heart rhythm.

Patient Demographics	
Mean age ± SD in years	32.75 ±6.89
Tidal volume (ml/Kg)	6.73 ± 0.44
PEEP (cm H_2O)	5.75 ± 0.97
FiO_2 (%)	0.38 ± 0.06
Details of OP exposure	
Suicidal n, (%)	19, (95%)
Mean Quantity of OP Consumed ± SD	95.18 ± 87.32
Occupation	
Farmer n, (%)	02, (10%)
Household n, (%)	08, (40%)
Student n, (%)	05, (25%)
Labour n, (%)	05, (25%)

Table 1: Demographics of the OP poisoning patients

OP Compound	**Frequency (%)**
Unknown	09(45%)
Dimethoate	05 (25%)
Methyl-Parathion	02 (10%)
Chlorpyrifos	02 (10%)
Quinolphos	02 (10%)

Table 2: Different OP compounds consumed

Variable	PRE (Before Intervention)	POST-30 (30 minutes after Intervention)	POST-60 (60 minutes after Intervention)	P value	F value	LSD
	(Mean ± S.D.)					
C_{stat} (mL/ cm H_2O)	26.84 ± 4.72 [c]	31.21 ± 4.81 [a] (16.28%)	29.18 ±4.79 [b] (8.71%)	0.0001***	9.76***	2.18
PaO_2:FiO_2 Ratio	229.49 ± 109.59 [b]	324.24 ± 120.29 [a] (41.28%)	270.81 ± 69.78 [ab] (16.47%)	0.096	2.63	114.39

Table 3: Measurements of Dependent Variables before and after Physiotherapy Treatment at various intervals of time

C_{stat}= static lung compliance; PaO_2:FiO_2 = oxygenation; S.D. = standard deviation.

*Values in parenthesis are mean percentage changes from the baseline measurements.

**Measurements are statistically significant at P<0.05;

*** Highly Significant at P<0.01.

Means with same superscripts in a column are not significantly different (P<0.05), LSD = Least Significant Difference.

DISCUSSION

Organophosphate (OP) insecticides inhibit both acetyl cholinesterase and pseudo cholinesterase activities. The inhibition, leads to accumulation of acetylcholine at synapses which causes overstimulation and disruption of neurotransmission in both central and peripheral nervous systems, causing exaggerated manifestations of nicotinic and muscarinic receptors[6]. Nicotinic manifestations are increasing or decreasing muscle power and fasciculation. Muscarinic manifestations are excessive salivation, meiosis and diarrhea. The most frequent signs are meiosis, vomiting, hypersalivation, respiratory distress, loss of consciousness and abdominal pain and muscle fasciculation[3, 4, 6]. Some of these effects such as miosis are minor and will resolve over time. Other effects such as increased pulmonary secretions escalate post exposure and have the potential to cause morbidity or even mortality if not adequately treated. Still other effects such as central apnea are life threatening if not treated immediately. Many of the clinical effects of acute OP poisoning have the potential to cause respiratory dysfunction, and respiratory failure post OP exposure is most likely multi-factorial. Interstitial edema from OP exposure may contribute to decreased

pulmonary compliance and ventilation perfusion mismatch[7]. Clinically, patients with respiratory failure post-OP exposure demonstrate significantly impaired gas exchange despite anti-cholinergic medication and aggressive mechanical ventilation. Increased pulmonary secretions are a prominent feature of acute OP exposure [4].

The clinical course of OP poisoning may be quite severe and may need intensive care management. The major reason for mortality is respiratory failure, hence careful monitoring, appropriate management and early recognition of this complication is required. Cholinergic circuits are integral part of the central control of respiration[6]. OP agents could also increase the work of breathing through an increase in static and dynamic lung compliance, constriction of the airways or airway outlet obstruction. Alternatively, OP agents could cause a central respiratory depression or central apnea.[8,9] Thus OP poisoning patients are very susceptible to respiratory associated problems especially respiratory muscle paralysis[6].

Respiratory failure being major reason for mortality, careful monitoring, and appropriate management and early recognition of this complication may decrease the mortality rate among these patients. Patients with OP poisoning may have respiratory failure for many reasons, including aspiration of gastric contents, excessive secretions, pneumonia and septicemia complicating adult respiratory distress syndrome[4]. Aspiration pneumonia is another troublesome complication, and careful monitoring during transport and early recognition of an absent gag reflex may reduce the incidence of aspiration pneumonia. Patients with OP poisoning may be followed with oxygen support without intubation and mechanical ventilation, but hypoxia and signs of respiratory failure such as tachypnea, paradoxical respiration and vigorous use of accessory respiratory muscles should be followed with assessment of the patient for endotracheal intubation and mechanical ventilation. Such patient generally needs ventilation support which generally has high chances of getting VAP. Early recognition of respiratory failure, prompt endotracheal intubation and mechanical ventilation are life-saving measures in severe OP poisoning [4].

Mechanically ventilated patients are at risk for retained secretions due to endotracheal intubation disrupting mucociliary escalator, relative immobility of mechanically patient confined to bed can lead to postoperative atelectasis, impaired cough, and retained secretions[10]. Physical therapy may be indicated for patients in the intensive care setting when they have retained secretions and radiological evidence of atelectasis or infiltrate, or as prophylaxis in conditions such as acute head injury and smoke inhalation. Physical therapy interventions include postural drainage, breathing exercises, percussion, vibration, manual hyperinflation, coughing, huffing, and suction[11, 18].

C_{ST} is considered as important clinical outcome measure, and it may be used to predict mortality in patients with respiratory failure[12]. In the present study, there was a highly significant improvement in static lung compliance, 30 and 60 minutes after intervention. The application of manual hyperinflation with a larger than normal tidal volume breath coupled with an inspiratory pause adopted in this study, may have facilitated collateral ventilation and effective recruitment of alveoli, thereby improving the time-dependent elastic behavior of the lung. There was also a possibility that the manual hyperinflation technique was effective in mobilization of pulmonary secretions from peripheral to central airways, which were subsequently removed with suctioning, thereby leading to further recruitment of more

functional alveolar units[13]. In addition, the immediate improvement in lung compliance with MHI could be attributed to the use of Mapleson-C circuit which produces significantly larger inspiratory pressures and tidal volumes than other circuits during manual hyperinflation [14, 15].

In addition, PaO_2:FiO_2 ratio mean values showed percentage improvement at Post-30 and Post-60 time intervals and also there was a significant critical difference between the mean values. The probable reason is that with increase in the recruitment of functional alveolar units after intervention, there may have been an improvement in the ventilation-perfusion ratio; decreased shunting of blood in lungs and improved oxygen transport in the blood. [16] However, the ratio could not reach statistical significance. This could be explained on the basis of hypoxic effects of endotracheal suctioning after manual hyperinflation[17].

CONCLUSION

Recruitment maneuvers such as manual hyperinflation accompanied with various physiotherapeutic techniques including manual hyperinflation, postural drainage, percussion, vibration and chest expansion exercises are effective in respiratory rehabilitation of patients with organophosporous poisoning.

REFERENCES

1. Eddleston, M., Mohamed, F., Davies, J.O..J, Eyer, P, Worek, F., Sheriff, M. H. R., Buckley N. A. (2006).Respiratory failure in acute organophosphorous pesticide self-poisoning, Q J Med; 99:513-522.
2. Aardema H, Meertens J.h.J.M., Ligtenberg O.m., Polman, P, Tulleken J. E., Zijlstra J.G.(2008).Organophosporous pesticide poisoning: Cases and developments : Netherlands The Journal of Medicine, 66(4):149-153.
3. Paudyal BP: (2008).Organophosphorous poisoning: J Nepal Med Asssoc: 47(172): 251-8.
4. Sungur M, Muhammed, G. (2001). Intensive care management of organophosphate insecticide poisoning: Critical Care:; 5: 211-215.
5. Ballantyne B, Marrs, T.C. (1992). Overview of the biological and clinical aspects of organophosphates and carbamates. In: clinical and experimental toxicology of organophoshates and carbamates. Butterworth Heinemann, Oxford, 3-14.
6. Raphael M, Karimzad, H.S., Agarwal, J., Bhandakar, A.A, Thunga, G., Shreedhar, N. et al. (2015). Prevalence of ventilator acquired pneumonia in organophosphorous poisoning patients in tertiary care hospital : Int J Drug Dev & Res; 7(4): 5-8.
7. Delaunois, A., P. Gustin, and M. Ansay (1992). Altered capillary filtration coefficient in parathion- and paraoxon-induced edema in isolated and perfused rabbit lungs. Toxicol Appl Pharmacol. 116(2): p. 161-9.
8. Fleming, N.W., T.R. Henderson, and K.L. Dretchen (1991). Mechanisms of respiratory failure produced by neostigmine and diisopropyl fluorophosphate. Eur J Pharmacol 195(1): p. 85-91.
9. Rickett, D. L., J. F. Glenn, and E. T. Beers (1986). Central respiratory effects versus neuromuscular actions of nerve agents. Neurotoxicology; 7(1): p. 225-36.
10. Branson, R, D. (2007). Respir Care. 52(10): 1328-42.
11. Wai Pong Wong (2000). Physical Therapy for a Patient in Acute Respiratory Failure. Phys Ther. 80: 662-670.
12. The Acute Respiratory Distress Syndrome Network (2000). Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 342:1301–1308.
13. Choi, J. S, Jones, A.Y. (2005). Effects of manual hyperinflation and suctioning in respiratory mechanics in mechanically ventilated patients with ventilator-associated pneumonia. Aust J Physiother. 51(1):25-30.

14. Hodgson C, Ntoumenopoulos G, Dawson H, Paratz J. (2007). The Mapleson C Circuit clears more secretions than the Laerdal circuit during manual hyperinflation in mechanically-ventilated patients: a randomized cross-over trial. Aust J. Physiother. 53(1): 33-8.
15. Savian, C., Paratz, J., Davies, A. (2006). Comparison of the effectiveness of manual and ventilator hyperinflation at different levels of positive end- expiratory pressure in artificially ventilated and intubated intensive care patients. Heart Lung. 35(5):334-41.
16. Ahmed, F., Shafeeq, A. M., Moiz, J. A., Geelani M. A. (2010). Comparison of effects manual versus ventilator hyperinflation on respiratory compliance and arterial blood gases in patients undergoing mitral valve replacement. Heart Lung. 39(5):437-43.
17. Ciesla, N. D.(1996). Chest physical therapy for patients in the intensive care unit. Phys Ther. 76:609-25.
18. Rakesh, O. R. and Jibi Paul (2016). The effectiveness of post isometric relaxation technique in relation with pulmonary function and the chest expansion of post thoracic surgery patients. International Journal of Medical and Exercise Science; 2(3): 205-216.

Citation:
Dr.Gitanjali Sikka and Dr.Suman Mehra. Respiratory rehabilitation of mechanicaly ventilated patients with organophosporous poisioning. *IJMAES, 2016; 2 (4), 231-238.*