Prone Position in Artificially Ventilated Chronic Obstructive Pulmonary Disease Patients Assessment of Lung Mechanics

Thesis Submitted by

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ABSTRACT

Prone Position in Artificially Ventilated chronic obstructive pulmonary disease Patients Assessment of Lung Mechanics

Key words: mechanical ventilation, COPD, prone position, lung mechanics, gas exchange

The purpose of this study was to compare the effect of prone position on the lung mechanics and blood gases exchange in mechanically ventilated chronic obstructive pulmonary disease (COPD) patients relative to the supine position.

METHODS: Thirty mechanically ventilated patients with acute respiratory failure type Π on top of chronic obstructive pulmonary disease on a volume controlled mode were put in prone position for one hour then turned back to supine position . Arterial samples for blood gases analysis "ABG" and Lung mechanics parameters were taken before turning, 30and 60 minutes after turning the patient to prone position and 30 minutes after returning the patient to supine position.

RESULTS : After one hour of pronation the PIP increased from 33.23±6.8 to 36.4±6.5, the Pmean increased from 10.1±2.3 to 11.74±2.8, Cdyn decreased from22.47±3.9 to 20.77±3.2, the Pplate decreased from 24.08±4.1 to 21.8±3.9, the Cstat increased from 33.36±5.7 to 42.04±8.9, the PaO2 increased from 94.83±38.8 to 105±39.4 and the PaCO2 decreased from 55.1±12.02 to 50.6±11.08.

conclusion: Prone position showed significant decrease in plateau pressure and increase in static compliance, it also improves oxygenation and CO_2 wash relative to supine position. The peak inspiratory pressure, mean airway pressure, and intrinsic positive end expiratory pressure increase in prone position. The effect of prone position on oxygenation and CO_2 wash lasts for at least on hour after turning the patients to the supine position.

Key Words : CoPD – Mechanical Ventilation – Prone Position – Lung Mechanics .

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List of abbreviations

PP	:	Prone Positioning
Paw	:	Mean Airway Pressure
ALI	:	Acute Lung Injury
ARDS	:	Adult Respiratory Distress Syndrome
ATS	:	American Thoracic Society
BIPAP	:	Bi-Level Positive Airway Pressure
C_{dyn}	:	Dynamic Compliance
COPD	:	Chronic Obstructive Pulmonary Disease
CPR	:	Cardiopulmonary Resuscitation
C_{stat}	:	Static Compliance
CT	:	Computed Tomography
ECMO	:	Extracorporeal Membrane Oxygenation
ERS	:	European Respiratory Society
Ers	:	Elastance Of The Respiratory System
FEV1	:	Forced Expiratory Pressure In First Second
FRC	:	Functional Residual Capacity
GOLD	:	Global Initiative For Chronic Obstructive Lung Disease
LTOT	:	Long-Term Oxygen Therapy
MEFV	:	Maximum Expiratory Flow Volume Loop
NHLBI	:	US National Heart, Lung, And Blood Institute

NIV	:	Noninvasive Mechanical Ventilation.
$PaCO_2$	:	Carbon Dioxide Pressure In Arterial Blood In Millimeter Mercury
PaO_2	:	Oxygen Partial Pressure In Arterial Blood In Millimeter Mercury
PAV	:	Proportional-Assist Ventilation
PCV	:	Pressure-Controlled Ventilation
$PEEP_I$	:	Intrinsic Positive End Expiratory Pressure
Pel	:	Elastic Component Opposed By The Thoracopulmonary System
PIP	:	Peak Inspiratory Pressure
Pmus	:	Pressure Generated By The Respiratory Muscles
P_{peak}	:	Peak Airway Pressure
P_{plat}	:	Plateau Pressure
Pres	:	Resistive Component Of The Conducting Airways
PSV	:	Pressure-Support Ventilation
R_{aw}	:	Airway Resistance
RV	:	Residual Volume
TLC	:	Total Lung Capacity
V/Q	:	Ventilation/Perfusion Ratio
VC	:	Vital Capacity
WHO	:	World Health Organization

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Introduction

Chronic obstructive pulmonary disease (COPD) is now recognized as an inflammatory disease of the airways. The new American Thoracic Society (ATS)/ European Respiratory Society (ERS) definition reflects these scientific advances: "Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking". The diagnosis of COPD is suggested by findings on history and/or physical examination and is confirmed by laboratory tests, usually with a supportive risk factor (eg, of familial COPD and/or of cigarette exposure). Spirometry is indispensable in establishing the diagnosis because it is a standardized and reproducible test that objectively confirms the presence of airflow obstruction (1)**.**

In the presence of high resistance to expiratory flows and short expiratory times, the respiratory system is unable to return to its resting volume at the end of exhalation. The positive pressure within regions of hyper inflated lung raises the mean intrathoracic pressure and causes the inspiratory muscles to operate at a higher than resting lung volume. Thus, dynamic hyperinflation places the respiratory muscles at a considerable mechanical disadvantage (2) and further impairs respiratory muscle function. Intrinsic positional end expiratory pressure (PEEPi) also imposes a substantial inspiratory threshold load.

Several factors contribute to development of gas exchange abnormalities in patients with obstructive lung disease. Airway obstruction produces regional hypoventilation that produces ventilation/perfusion mismatch and hypoxemia. Loss of the capillary bed due to emphysema or compression of pulmonary capillaries by over inflated alveoli also tends to increase dead space, and this wasted ventilation further compromises the ability of the respiratory muscles to provide adequate ventilation (3).

Turning and positioning of critically ill patients are well-accepted nursing activities, with the primary purpose being to relieve pressure, improve patients' comfort and aid pulmonary secretions drainage (4). However, body positioning of critically ill patients may have a profound effect on arterial oxygenation, which is reflected by the oxygen saturation level in the blood (5). Optimal oxygenation depends on the match of ventilation (V) to perfusion (Q) ratio, with optimal oxygenation, occurring when the best ventilated areas are best perfused. Therefore, proper positioning of patients may promote Oxygenation in a less traumatic, less invasive and less expensive manner than high-tech treatment (6).

The use of the prone position (PP) was first advocated over two decades ago as a strategy for improving oxygenation in patients with acute bilateral lung injury disease, pneumonia and adult respiratory distress disease (ARDS) (7, 8, 9**)**. Despite the numerous studies demonstrating a significant improvement in oxygenation, prone positioning is still underused (10)**.**

This reluctance may be due to the logistical difficulties in turning critically ill patients onto their stomachs, as well as the unpredictability of the prone position.

Altering the patient's position to prone improve oxygenation by reducing the ventilation/perfusion (V/Q) mismatch and decreases the shunt (11, 12). The Prone position also maintains perfusion, as it does not decrease blood flow to the recruited regions. In other words, the PP improves the V/Q match and decreases the shunt (11). Changes in pleural pressures favoring alveolar recruitment in the dorsal areas in the prone position may also contribute to the increased oxygenation; however, this is not yet fully understood. It is also suggested that the prone position aids pulmonary secretions to be mobilized and drained aiding ventilation**.**

Decreased atelectasis and more uniform inflation should result in more homogenous and increased average alveolar septal tension the latter is transmitted to airway walls via connective tissue cables (13), resulting in outward wall traction and airway caliber increase (14). If parenchymal elastic recoil is ‘‘maintained’’ (e.g. chronic bronchitis) (15) pronation might enhance ‘‘parenchyma-induced bronchodilation’’.

Aim of The Work

In this study we tried to compare the effect of prone position on the lung mechanics and blood gases exchange in mechanically ventilated chronic obstructive pulmonary disease (COPD) patients relative to the supine position.

Chapter I

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is a major public health problem. It is the fourth leading cause of chronic morbidity and mortality in the United States (16) and is projected to rank fifth in 2020 as a worldwide burden of disease according to a study published by the World Bank/World Health Organization (17).

Definition:

Internationally accepted opinion, including the 1995 American Thoracic Society (ATS) statement, had defined COPD as a disease state characterized by chronic airflow limitation due to chronic bronchitis and emphysema. Chronic bronchitis has been defined in clinical terms: the presence of chronic productive cough for at least 3 consecutive months in 2 consecutive years. Other causes of chronic productive cough must be ruled out. Emphysema, on the other hand, has been defined by its pathologic description: an abnormal enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of their walls and without obvious fibrosis.

The new American Thoracic Society (ATS)/ European Respiratory Society (ERS) guidelines, like the GOLD guidelines, have parted from this traditional description of COPD. Similar to the changes in the definition of asthma by the National Heart, Lung, and Blood Institute(NHLBI) more than a decade ago, the definition of COPD has undergone major revision (18).

In 2001, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) was published. This was a collaborative effort, between the US NHLBI and the World Health Organization (WHO), to increase awareness of COPD around the globe. This evidence-based report was offered as a guideline for the detection and management of COPD. Revisions were made in 2003, 2004 and 2006 (1).

According to the GOLD document, COPD is defined as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases (19).

The chronic airflow limitation characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Chronic inflammation causes structural changes and narrowing of the small airways.

Destruction of the lung parenchyma, also by inflammatory processes, leads to the loss of alveolar attachments to the small airways and decreases

lung elastic recoil; in turn, these changes diminish the ability of the airways to remain open during expiration. Airflow limitation is best measured by spirometry, as this is the most widely available, reproducible test of lung function.

Pathology:

Pathological changes characteristic of COPD are found in the central airways, peripheral airways, lung parenchyma, and pulmonary vasculature. In the central airways—the trachea, bronchi, and bronchioles greater than 2 to 4 mm in internal diameter— inflammatory cells infiltrate the surface epithelium (20). Enlarged mucus-secreting glands and an increase in the number of goblet cells are associated with mucus hypersecretion.

In the peripheral airways — small bronchi and bronchioles that have an internal diameter of less than 2 mm — chronic inflammation leads to repeated cycles of injury and repair of the airway wall (21). The repair process results in a structural remodeling of the airway wall, with increasing collagen content and scar tissue formation, which narrows the lumen and produces fixed airways obstruction (22).

Destruction of the lung parenchyma in patients with COPD typically occurs as centrilobular emphysema. This involves dilatation and destruction of the respiratory bronchioles (23).