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Respiratory Muscle Weakness In Mechanically Ventilated Patients

Essay

Submitted for partial fulfillment of Master Degree In ***Intensive Care***

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2012

Acknowledgement

Thanks first and last to **ALLAH** *as we owe him for his great care, support and guidance in every step in our life.*

I want to thank ***Prof. Dr. Mohsen Abd El-Ghany Bassiony,*** *Professor of Anesthesiology and Intensive Care, Faculty of Medicine, Ain Shams University, who guided this work and helped whenever I was in need.*

Special thanks are due to ***Prof. Dr. Hazem Abd El-Rahman Fawezi,*** *assistant profesor of Anesthesiology and Intensive Care, Faculty of Medicine, Ain Shams University, for dedicating so much of his precious time and effort to complete this work.*

I owe my deep thanks and gratitude to ***Dr. Walied Hamed Nofal,*** *Lecturer of Anesthesiology and Intensive Care, Faculty of Medicine, Ain Shams University, for his great support, patience and fruitful comments without which this work have never been accomplished.*

Lastly but not least, I would like to thank all members in my family and all members of the intensive care unit in EL-Helal Hospital-Sohag specially for pushing me forward all the time.

Beshoy Mina Tanagho

Abbreviations

ARDS	Acute respiratory distress syndrome
Akt	Protein kinase B
ALS	Amyotrophic lateral sclerosis
ANCA	Anti-neutrophil cytoplasmic antibody
AP	Adductor Pollicis
CIP/CIM	Critical illness polyneuropathy / critical illness myopathy
CIPNM	Critical illness polyneuromyopathy
CK	Creatine kinase
CNS	Central nervous system
CO2	Carbon dioxide
CMV	Controlled mechanical ventilation
COPD	Chronic obstructive pulmonary disease
CSA	Cross-sectional area
CSF	Cerebrospinal fluid
CT	Computerized tomography
DEC	Dynamic effective compliance
DLO_2	Diffusing capacity for oxygen
EMG	Electromyography
ERV	The expiratory reserve volume
FRC	The functional residual capacity
HIV	Human immunodeficiency
IC	The inspiratory capacity
ICU-AP	Intensive care unit acquired paresis
ICU	Intensive care unit

ICU-AW	Intensive care unit acquired weakness
IIT	Intensive insulin therapy
IL	Interleukin
L/min	Liter per minute
L/sec	Liter per second
MB	Muscle biopsy
MEP	Maximal expiratory pressure
MIP	Maximal inspiratory pressure
MODS	Multiple organ dysfunction syndromes
MP	Methylprednisolone
MV	Mechanical ventilation
MVV	Maximum voluntary ventilation
NIPPV	Noninvasive positive pressure ventilation
NMB	Neuromuscular blockers
NMBDs	Neuromuscular blocking drugs
O2	Oxygen
PaCO2	Arterial carbon dioxide pressure
PaO2	Arterial oxygen pressure
PCF	Peak cough flow
PEEP	Positive end expiratory pressure
PFS	Pulmonary function score
PTP	Pressure-time product
RARS	Rapidly acting irritant receptors
REM	Rapid eye movement sleep
ROS	Reactive oxygen species

Abbreviations

RV	The residual volume
SARS	Slowly adapting pulmonary stretch receptors
SIRS	Systemic inflammatory response syndrome
TLC	The total lung capacity
TNF	Tumor necrosis factor
TwPdi	Trans-diaphragmatic pressure after bilateral
BAMPS	anterior magnetic phrenic nerve stimulation
Twpdi	Supramaximal twitch stimulation
UPS	Ubiquitin-proteasome system
V_A/Q	Ventilation perfusion ratio
VC	Vital capacity
VIDD	Ventilator-induced diaphragmatic dysfunction
VT	The tidal volume
WOB	Work of Breathing

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Introduction

Acquired respiratory muscle weakness is a huge clinical problem in intensive care unit (ICU). An increasing number of patients are being discharged from the intensive care unit to chronic care facilities because they cannot be weaned from mechanical ventilation. ***(Callahan, 2009).***

Weaning from mechanical ventilation is an important and time-consuming process in critically ill patients as it comprises approximately 40% of the time spent on the ventilator. There are several factors that may contribute to delayed weaning, a major determinant appears to be respiratory muscle weakness. ***(Hermans et al., 2010)***

Although mechanical ventilation can be a life saving measure, it is also associated with major complications such as infection, barotrauma, cardiovascular compromise, tracheal injuries, oxygen toxicity and ventilator-induced lung injury. In addition to the above well-known complications of ventilatory support, a rapidly accumulating body of evidence suggests that mechanical ventilation, with its attendant diaphragm muscle inactivity and unloading, is an important cause of diaphragmatic dysfunction. ***(Theodoros et al., 2004).***

Indeed, numerous well-controlled animal studies have demonstrated that prolonged mechanical ventilation results in diaphragmatic weakness due to both atrophy and contractile dysfunction. Importantly, a recent clinical investigation has confirmed that prolonged mechanical ventilation results in atrophy of the human diaphragm. This mechanical ventilation-

induced diaphragmatic weakness is important because the most frequent cause of weaning difficulty is respiratory muscle failure due to inspiratory muscle weakness and/or a decline in inspiratory muscle endurance. Therefore, developing methods to protect against mechanical ventilation-induced diaphragmatic weakness is important. ***(Powers et al., 2008).***

A large body of evidence from animal models, and more limited data from humans, indicates that mechanical ventilation can cause muscle fiber injury and atrophy within the diaphragm. So there is increasing recognition of a condition termed ventilator-induced diaphragmatic dysfunction (VIDD). ***(Petrof et al., 2010).***

Acquired weakness syndromes in critically ill patients have been shown to be a major cause of mortality and long-term morbidity. ***(Herridge et al., 2008 & Khan et al., 2008).***

A key component of these syndromes is the development of respiratory muscle weakness, which leads to prolonged duration of mechanical ventilation, difficulty weaning from the ventilator, and recurrence of respiratory failure after extubation.

Clinical studies have identified sepsis and hyperglycemia as the two major risk factors for development of intensive care unit acquired weakness, including respiratory muscle weakness. In addition to these factors, an extensive literature has recently emerged revealing that mechanical ventilation per se also produces deleterious effects on the diaphragm. A number of studies using animal models showing that relatively short durations of controlled mechanical ventilation rapidly produce diaphragm weakness and atrophy***. (Callahan et al., 2009).***

Recently, there has been a great expansion in our knowledge of how mechanical ventilation can adversely affect diaphragmatic structure and function. Future studies need to better define the evolution and mechanistic basis for ventilator-induced diaphragmatic dysfunction in humans, in order to allow the development of mechanical ventilation strategies and pharmacologic agents that will decrease the incidence of ventilator-induced diaphragmatic dysfunction (VIDD). ***(Petrof et al., 2010).***

Aim of the work

The aim of the work is to study the causes and pathogenesis of respiratory muscle weakness in mechanically ventilated patients and updates in management.

The ability to conduct gas exchange depends basically on the "respiratory pump" which moves air in and out of the lung. The respiratory muscles, an integral and vital component of the respiratory process, serve as a vital link between the different components of the pump, which consists of the respiratory centers, the conducting nerves, and the lung itself. All these links have to be coordinated to perform in an efficient fashion. ***(Flaminiano & Bartolome, 2001)***

Anatomy of the Respiratory System

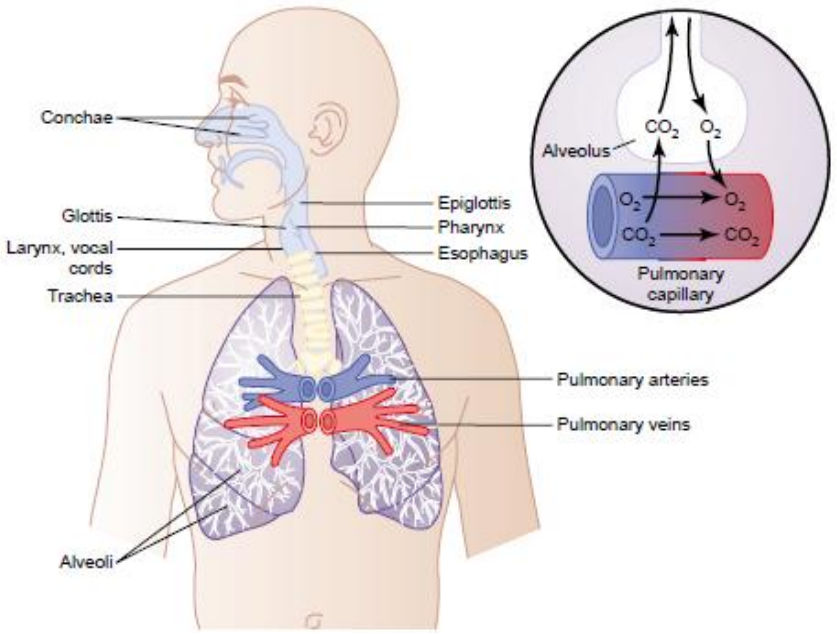

Figure 1: Respiratory passages ***(Gyton and Hall, 2006).***

1- The upper airway:

The upper airway consists of the nasal passages, the Paranasal sinuses, the pharynx, the epiglottis, and the larynx. Its functions are to conduct air to the lower airway, to protect the lower airway

from foreign matter, and to warm, filter, and humidify the inspired air. ***(Thibodeau et al., 2003)***

2-The lower airway:

The lower airway consists of a series of tubes that divide like branches of a tree, becoming narrower, shorter, and more numerous as they penetrate deeper into the lung. Its functions are to conduct air, provide mucociliary defense, and, most important, perform external gas exchange.

After penetrating the lung, the main stem bronchi divide into lobar bronchi which then bifurcate and trifurcate into segmental bronchioles or terminal bronchioles that supply the lung segments on the left and right. The bronchioles lack cartilages and are made of connective tissue that contains elastic fibers and limited smooth muscles and are held open by radial traction from the elastic recoil forces of the lung tissue. With the lack of supporting cartilage, these airways are susceptible to bronchospasm ***(Des Jardins, 2002).***

The terminal respiratory unit, or acinus, is that portion of the lung arising from a single terminal bronchiole. The acinus is the primary gas-exchanging unit of the lung, consisting of the respiratory bronchiole, alveolar ducts, alveolar sacs, and the alveoli. ***(Thibodeau et al., 2003)***

3- Lung lobes and segments (Figure 2):

Each lung is divided into lobes surrounded by pleura. There are two lobes on the left: the upper and lower, separated by the major (oblique) fissure; and three on the right: the upper, middle and lower lobes separated by the major (oblique) and minor