MANAGEMENT OF PSYCHOLOGICAL PROBLEMS IN MECHANICALLY VENTILATED PATIENTS

Essay Submitted for Partial Fulfillment of
Master Degree in Anesthesiology
2014

Submitted by:

Omar Sameh Mahmoud Abdel-Hafez
M.B.; B.Ch.

Supervised by:

Prof. Dr. AYMAN MOKHTAR KAMALY

Prof. of Anesthesiology, Intensive Care & Algology,
Faculty of Medicine, Ain Shams University

Dr. MAHMOUD AHMED ABDEL-HAKEEM

Lecturer of Anesthesiology, Intensive Care & Algology,
Faculty of Medicine, Ain Shams University

Dr. ABDEL-AZIZ ABDALLAH ABDEL-AZIZ

Lecturer of Anesthesiology, Intensive Care & Algology,
Faculty of Medicine, Ain Shams University

علاج المشاكل النفسية التي تصيب مرضى التنفس الصناعي

٢٠١٤

مقدمة من:

ط./ عمر سامح محمود عبد الحافظ

بكالوريوس الطب و الجراحة

أشراف

أ.د./ أيمن مختار كمالي

أستاذ التخدير و الرعاية المركزة وعلاج الألم - كلية الطب - جامعة عين شمس

د./ محمود أحمد عبد الحكيم

مدرس التخدير و الرعاية المركزة وعلاج الألم - كلية الطب - جامعة عين شمس

د./ عبد العزيز عبد الله عبد العزيز

مدرس التخدير و الرعاية المركزة وعلاج الألم - كلية الطب - جامعة عين شمس

Contents:

List of Tables:

List of Figures:

Acknowledgements

*First of all, all gratitude is due to **ALLAH**, the almighty guided and aided me in bringing this work to light which would have never been crowned by success without blessing of **ALLAH,** to whom my faithful loyalty will remain however beyond my compromises.*

*It's a pleasure to express my deepest appreciation, gratitude and sincere thanks to my supervisor **Prof. Dr. Ayman Mokhtar Kamaly**, Professor of Anesthesiology, Intensive Care & Algology, Faculty of Medicine, Ain Shams University, for his constructive suggestions, excellent guidance and eminent supervision which aided me over difficulties I met with throughout this work.*

*It's a pleasure to express my deepest appreciation, gratitude and sincere thanks to my supervisor **Dr. Mahmoud Ahmed Abdel-Hakeem**, Lecturer of Anesthesiology, Intensive Care & Algology, Faculty of Medicine, Ain Shams University, for his constructive suggestions, excellent guidance and eminent supervision which aided me over difficulties I met with throughout this work.*

*I would like to express my profound gratitude to **Dr. Abdel-Aziz Abdallah Abdel-Aziz**, Lecturer of Anesthesiology, Intensive Care & Algology, Faculty of Medicine, Ain Shams University, for his excellent guidance, continuous encouragement and valuable help.*

Aim of the Work

Reviewing the latest literatures updates about prevention, diagnosis and state of the art treatment(s) of psychological problems affecting mechanically ventilated patients, to prevent their adverse effects on morbidity and mortality.

Introduction

Critical Care Medicine is continuously being improved with better outcomes and improved mortality and morbidity, multidisciplinary approach for management of cases has also improved the outcomes. But it was noticed that the diseases and the treatment modalities themselves can cause adverse psychological outcomes on the short and long terms; one of these modalities is intubation and mechanical ventilation, especially of conscious patients. Delirium in critically ill patients is now recognized as a major public health problem, affecting up to 80% of mechanically ventilated adult ICU patients **(McNicoll *et al.*, 2003)**.

Endotracheal intubation achieves four main goals: 1) Airway protection, 2) provides upper airway patency, 3) pulmonary hygiene and 4) allows mechanical positive pressure ventilation **(Irwin RS *et al.*, 2010)**.

Traditionally, the goals of ICU analgesia and sedation have been to facilitate mechanical ventilation, to prevent patient and caregiver injury, and to avoid the psychological and physiologic consequences of inadequate treatment of pain, anxiety, agitation, and delirium. Avoiding complications of over-sedation, such as ventilator dependency, delirium, muscle atrophy and weakness, pneumonia, thrombo-embolic disease, nerve compression and pressure sores are also important **(Schweickert *et al.*, 2004)**.

Risk factors of delirium include a history of hypertension and alcoholism, higher APACHE II score, and with clinical effects of sedative and analgesic drugs. Several reports have shown sedative-induced coma to be an independent risk factor for delirium in ICU patients **(Ouimet *et al.*, 2007)**

Indications for Intubation and Mechanical Ventilation

Endotracheal intubation achieves four main goals:

(1) airway protection,
(2) provides upper airway patency,
(3) pulmonary hygiene and
(4) allows mechanical positive pressure ventilation **(Irwin, 2010)**.

The specific indications for endotracheal intubation are difficult to define. Although a seasoned practitioner could easily identify a patient who requires intubation, it is challenging to explain the exact parameters used for making such a decision. To date, there have been no significant studies evaluating the specific indications or guidelines for endotracheal intubation. These indications are increasingly more complicated in an era of advanced technology in oxygen delivery systems and noninvasive forms of ventilation **(Brainard, 2010)**.

Currently accepted indications can be divided into three basic groups:
(I) hypoxic respiratory failure,
(II) hypercarbic ventilatory failure (including cardiac arrest), and
(III) impaired consciousness and airway protection.
These general indications are all based on accepted practice, with few or no data available to support specific guidelines. Perhaps Marino stated it best when he commented that, ". . .the indication for intubation and mechanical ventilation is thinking of it." **(Marino, 2007)**.

(I) Hypoxic Respiratory Failure.

Acute hypoxic respiratory failure results from inadequate exchange of oxygen across the pulmonary alveolo-capillary membrane. This impairment leads to a decrease in arterial oxygen tension (hypoxemia) and insufficient delivery of oxygen to tissues and cells (hypoxia). In medical literature, this type of failure is often described as type-I failure, that is, hypoxemia without hypercarbia. Oxygen delivery is the product of arterial oxygen content and cardiac output. Therefore, hypoxia can also occur secondary to decreased cardiac output, anemia, or abnormal oxygen-hemoglobin binding affinity **(Nielsen, 2008)**.

The diagnosis of hypoxemia requires obtaining an arterial blood gas and is commonly defined as a PaO_2 of less than 60 mmHg. Pulse oximetry is commonly used for assessing hypoxemia. However, this modality measures the saturation of hemoglobin and not PaO_2, reflecting oxygen dissolved in the blood or oxygen content, which includes both bound and unbound O_2. Thus, a patient with severe anemia may have a normal PaO_2 but a low O_2 content. Low pulse oximetry values coincide with significant hypoxemia, but normal oxygen saturation does not exclude hypoxemia, especially in patients receiving a high FIO_2. Normal PaO_2 levels are 80 to 100 mmHg in a healthy patient breathing room air and can exceed 500 mm Hg in a patient breathing 100% oxygen. Pulse oximetry values may remain normal until PaO2 decreases to less than 60 mm Hg. For this reason, the alveolar-arterial oxygen gradient should be evaluated in patients receiving a high FIO_2. A widening alveolar-arterial oxygen gradient is a sign of worsening hypoxemia. Pulse oximetry may be unreliable in cases of severe anemia, carbon monoxide poisoning, methemoglobinemia, or peripheral vasoconstriction **(Marik, 2011)**.

The symptoms and signs of hypoxia are nonspecific and are noted in Table 1.1. Tachypnea and dyspnea may or may not be present depending on the etiology of the hypoxia. Many disease processes can lead to hypoxemia, and the most common causes of hypoxemia respiratory failure and their pathophysiologies are described in Table 1.2 **(Brainard, 2010)**.

Table 1.1: Symptoms and Signs of Hypoxia

SYMPTOMS	SIGNS
Headache	Agitation
Irritability	Lethargy
Confusion	Somnolence
Exhaustion	Coma
	Central cyanosis
	Seizures

(Brainard, 2010)

Table 1.2. Causes of Hypoxemic Respiratory Failure

INTRINSIC LUNG DISEASE	Atelectasis Pneumonia Lung consolidation Noncardiogenic pulmonary edema Transfusion-related acute lung injury (TRALI) ARDS
CARDIAC DISORDERS	Cardiogenic pulmonary edema
VASCULAR DISORDERS	Pulmonary embolism
TOXINS	Carbon monoxide

(Brainard, 2010)

The initial treatment of all causes of hypoxemia is the same: ensure a patent airway and adequate ventilation, and provide supplemental oxygen. A PaO_2 value of 50 to 60 mmHg or an arterial oxygen saturation of 88% to 90% is often suggested as a minimal accepted value, although specific patients (i.e., patients with myocardial ischemia and those in shock) may warrant other cutoff values for escalation of therapy. Except in patients with severe shunt, hypoxemia will improve with delivery of high FIO_2. Initial treatment starts with low-flow nasal cannula and escalates to a 100% non-rebreather mask or high-flow O2 therapy. If hypoxemia fails to reverse with supplemental oxygen and the patient has symptoms, noninvasive assisted ventilation with 100% O2 may be attempted. Certain specific contraindications, described elsewhere, preclude this approach. If a patient is unable to maintain minimal oxygen saturation while ventilating with 100% FIO_2, endotracheal intubation and mechanical ventilation will be required to improve this value **(Schmidt, 2009)**.

(II) Hyperbaric Ventilatory Failure.

Acute ventilatory failure results from inadequate removal of gas from distal alveoli. This alveolar hypoventilation results in subsequent hypercarbia and respiratory acidosis. Mild ventilatory failure can exist alone or, when impairment is more severe, may be associated with hypoxemia. Ventilatory failure can result from a primary lung process or can occur secondary to disorders in the cardiac, neurologic, metabolic, or other systems. When associated with hypoxemia, this type of failure may be described in the literature as type II respiratory failure **(Nielsen, 2008)**.

The diagnosis of hypercarbia is best made by obtaining an arterial blood gas. Hypercarbia is commonly defined as a $PaCO_2$ of more than 45. Unlike pulse oximetry for detecting hypoxemia, bedside monitors for detecting hypercarbia are not routinely available. End-tidal CO_2 monitoring, now standard in intraoperative care, is not currently available at many institutions. This lack of bedside monitoring is particularly significant because the most common form of respiratory monitoring is normal pulse oximetry. Normal oxygen saturation can be found in the presence of significant hypoventilation, providing false confidence. It also is important to follow $PaCO_2$ values over time because changes in this parameter may provide information that is more important than the absolute value **(Marik, 2011)**.

The signs and symptoms of hypercarbia depend on the patient's baseline $PaCO_2$, the absolute value of $PaCO_2$, and the rate of change. Chronic hypercapnia may be well tolerated. Eliciting a history of chronic CO_2 retention and performing careful serial evaluations of arterial pH are essential because hypercarbia with a near-normal pH is a sign of chronic compensation and often does not reflect an acute disorder. The symptoms and signs of hypercarbia, like those seen in patients suffering from hypoxia, are nonspecific and are noted in Table 1.3. These all may indicate respiratory fatigue and suggest that the patient soon may be unable to achieve the minute ventilation required to maintain normocarbia. As stated previously, the etiology of hypercarbic ventilatory failure can be a primary lung process or result from a nonpulmonary process. For the purposes of this chapter, respiratory and cardiac arrests are included as ventilator failure. The most common causes of ventilatory failure are listed in Table 1.4 **(Brainard, 2010)**.

Table 1.3: Symptoms and Signs of Hypercarbia

SYMPTOMS	SIGNS
• Dyspnea	• Increased work of breathing
• Headache	• Accessory respiratory muscle use
• Confusion	• Tachypnea
• Exhaustion	• Shallow or small tidal volume breathing
	• Lethargy
	• Somnolence
	• Coma
	• Flapping tremor
	• Seizures
	• Cardiovascular collapse

(Brainard, 2010)

As in hypoxic respiratory failure, the initial treatment of hypercarbic ventilatory failure is to ensure a patent airway and provide supplemental oxygen to treat associated hypoxemia. However, although the treatment for all causes of hypoxemic respiratory failure is to increase the oxygen content in the blood, the approach to hypercarbic ventilatory failure depends on etiology. In cases in which ventilatory failure is not the primary disorder, support of ventilation may be indicated, but definitive therapy should be directed at the underlying cause. For example, a narcotic overdose is treated with reversal agents, whereas ventilatory failure secondary to cardiogenic shock can be treated with inotropic agents **(Schmidt, 2009)**.

Table 1.4: Causes of Hypercarbic Ventilatory Failure

INTRINSIC LUNG DISEASES	Chronic obstructive pulmonary disease Asthma
ANATOMIC DISORDERS	Sleep apnea Airway obstruction
NEUROLOGIC DISORDERS	Brainstem or medullary stroke Opiate or sedative overdose Obesity-hypoventilation syndrome Myasthenia gravis, Guillain-Barre´ syndrome Critical illness myopathy or polyneuropathy
CARDIAC DISORDERS	Cardiac arrest Cardiogenic shock Heart failure
VASCULAR DISORDERS	Pulmonary embolism
METABOLIC DISORDERS	Hypomagnesemia Hypophosphatemia

(Brainard, 2010)

When specific medical therapies are not available or not successful in increasing ventilation, or when ventilatory failure is the primary problem, treatment is concentrated on providing a means to increase minute ventilation. This most often is provided through noninvasive positive-pressure ventilation or endotracheal intubation and mechanical ventilation. Therapy is often initiated when hypercapnia is