Invasive Mechanical Ventilation Versus Non Invasive In Management of Cardiogenic and Non Cardiogenic Pulmonary Oedema Patients

Essay

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Dedication

A special thanks and appreciation to **my lovely parents** and **my husband** for their support, advice, help, encouragement, standing beside me and love which gave me the power to complete this work.

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List of Abbreviations

Abb.	Meaning
ABG	Arterial blood gas
AC	Assist control
AECOPD	Acute exacerbation of chronic obstructive pulmonary disease
AMI	Acute myocardial infarction
APRV	Airway pressure release ventilation
ARDS	Acute respiratory distress syndrome
ARF	Acute respiratory failure
ASV	Adaptive support ventilation
ATC	Automatic tube compansation
BiPAP	Bilevel positive airway pressure
BNP	B-type natriuretic peptide
BPAP	Bilevel positive airway pressure
CDC	Centers for disease control
CHF	Congestive heart failure
CMV	Continuous mandatory ventilation
CMV	Continuous mandatory ventilation
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CPE	Cardiogenic pulmonary edema
CPIS	Clinical pulmonary infection score
ED	Emergency department
EPAP	Expiratory positive airway pressure

List of Abbreviations

Abb.	Meaning
ET	Endotracheal
F	Frequency
HFJV	High frequency jet ventilation
HFOV	High frequency oscillatory ventilation
HFV	High frequency ventilation
HFV-A	High frequency ventilation active
HFV-P	High frequency ventilation passive
HHME	Hygroscopic heat moisture exchanger
ICU	Intensive care unit
IMV	Intermittent mandatory ventilation
IMV	Invasive mechanical ventilation
IPAP	Inspiratory positive airway pressure
LMA	Laryngeal mask airway
LV	Left ventricle
LV	Liquid ventilation
MI	Myocardial infarction
MMV	Mandatory minute ventilation
NAVA	Neurally adjusted ventilatory assist
NCPE	Non cardiogenic pulmonary edema
NIMV	Non invasive mechanical ventilation
NIV	Non invasive ventilation
NNIS	National nosocomial infection surveillance
NNT	Numbers needed to treat
NPPV	Noninvasive positive pressure ventilation

List of Abbreviations

Abb.	Meaning
P_{ALV}	Pressure in alveoli
P_{AO}	Pressure at airway opening
Pao2	Pressure of oxygen in arterial blood
PAV	Proportional assist ventilation
PC	Pressure control
PEEP	Positive end expiratory pressure
PLV	Partial liquid ventilation
PRVC	Pressure regulated volume control
PSB	Protected specimen brush
PSV	Pressure support ventilation
PTA	Transairway pressure
SBTs	Spontaneous breathing trials
SIMV	Synchronized i intermittent mandatory ventilation
SIRS	Systemic inflammatory response symdrome
T_{low}	Exhalation time
TLV	Total liquid ventilation
TV	Tidal volume
V/Q	Ventilation perfusion ratio
VAP	Ventilator associated pneumonia
VC	Volume control
VE	Minute ventilation
VSV	Volume support ventilation
WBCs	White blood cells

Introduction

Pulmonary edema is differentiated into two categories cardiogenic and noncardiogenic. Both result from acute fluid accumulation in the alveoli, with resultant varying degrees of oxygen desaturation and respiratory distress. Cardiogenic shock primarily results from increased pulmonary hydrostatic pressure, which causes plasma ultrafiltrate to cross the pulmonary capillary membrane into the interstitium **(Abraham et al., 2000).**

In contrast, noncardiogenic pulmonary edema most often results from permeability changes in the pulmonary capillary membrane itself. Understanding the differences between cardiogenic and noncardiogenic pulmonary edema is essential for effective therapeutic intervention to occur **(Bernard et al., 1994).**

Cardiogenic pulmonary edema (CPE) is a common and potentially deadly condition frequently encountered in emergency medicine **(Fromm et al., 1995).** Many conditions exist that directly or indirectly lead to the development of pulmonary edema. Regardless of the underlying cause of CPE, all patients who develop CPE must be diagnosed and managed expeditiously **(Mattu et al., 2002).**

Patients who have developed CPE can quickly develop respiratory failure if delays occur in recognition or management of the condition. Patients who develop CPE, in fact, have an in-hospital mortality of 15% to 20%, and mortality may be even higher when the condition is associated with acute myocardial infarction (AMI) or acute valvular dysfunction. Acute care providers should maintain a high level of vigilance for this condition and initiate management strategies promptly **(Mattu et al., 2002).**

The chance of COPD patient with acute respiratory failure having a second episode of acute respiratory failure after an initial (first 48 hours) successful response to NIMV is about 20%. This event is more likely to occur in patients with more severe functional and clinical disease who have more complications at the time of admission to the ICU. These patients have a very poor in hospital prognosis, especially if NIMV is continued rather than prompt initiation of Invasive ventilation **(Ambrosino et al., 1995).**

The severity of the episode of acute respiratory failure as assessed by clinical and functional compromise, and the level of acidosis and hypercapnia during an initial trial of non-invasive mechanical ventilation, have an influence on the likelihood for success with non-invasive mechanical ventilation

and may prove to be useful in deciding whether to continue with this treatment **(Colice et al., 1993).**

In recent years non-invasive mechanical ventilation (NIMV), delivered through a facial or nose mask, has been successfully used in selected populations as an effective treatment for acute respiratory failure and as technique for weaning intubated patients **(Celikel et al., 1998).**

Despite NIMV having also been used in patients affected by "pure" hypoxic respiratory failure, most studies have concentrated on patients with chronic obstructive pulmonary disease (COPD). The rate of failure of NIMV in these patients ranges from 5% to 40% **(Nava et al., 1998).**

Recognition of this subset of patients is very important both from a clinical and ethical point of view since prolonged application of NIMV may unduly delay the time of intubation**.** Failure of NIMV has usually been defined as: (a) need for intubation because of lack of improvement in arterial blood gas tensions and clinical parameters after a few hours of ventilation (usually 1–3 hours); (b) clinical deterioration and subsequent intubation during hospital stay, and (c) death **(Briasoulis and Pavlides, 2001).**

Studies specifically designed to assess the best predictors of NIMV outcomes agree that changes in PH in the first hour of

ventilation and the clinical condition of the patients before ventilation are the most powerful factors linked to success or failure. It has also been shown that, despite an initial improvement in blood gas tensions and clinical condition, a subset of patients go on to die or need to be intubated days after the successful application of NIMV **(Lam et al., 2011).**

NIMV resulted in an immediate and sustained improvement in blood gas tensions in pulmonary oedema patients enrolled after an episode of acute respiratory failure, requiring more than one day of ventilation. The arterial blood gases continued to improve in most of the responders so that weaning and discharge from the hospital became possible **(Cohen et al., 2006).**

Aim of the Work

The following essay compare the use of non invasive and invasive ventilation as a mechanical ventilation in pulmonary oedema patients with respect to its indications, limitations, related ventilators and ventilator- patient interfaces. Also, complications of NIV and IV were discussed in details.