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Role of non invasive ventilation in decreasing length of postextubation ICU stay

Thesis submitted in partial fulfillment of the requirements of the doctorate degree in

Chest diseases

BY

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Master Degree in chest diseases, Cairo University, 2011

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2015

جامعة القاهرة
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دور التنفس الصناعي الغير نافذ في تقليل مدة البقاء بوحدة العناية المركزة عقب الفطام من جهاز التنفس الصناعي

رسالة مقدمة تمهيداً للحصول على درجة الدكتوراه في الأمراض الصدرية
من الطبيب

ياسر محرز الصارم
ماجستير الأمراض الصدرية – طب القاهرة – 2011

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INTRODUCTION

Acute respiratory failure (ARF) in COPD generally marks a serious change in clinical state and is a frequent cause of admissions to emergency and/or intensive care units (ICU). Even more, ARF is also associated with excess mortality both during the hospital stay and in the months following discharge from the hospital **(Connors et al., 1996).** The long-term prognosis of patients with COPD and ARF particularly worsens if their clinical state calls for a ventilatory support, irrespective of whether this is applied invasively **(Connors et al., 1996)** or non-invasively **(Chu et al., 2004).**

Non-invasive ventilation (NIV) is a broad term for any ventilation therapy applied in a non-invasive way, e.g. via a mask, nasal prongs or a helmet. Therefore, NIV, or NPPV (Non-invasive Positive Pressure Ventilation), is also very often referred to as "mask ventilation". This is in contrast to "invasive ventilation", where an endotracheal tube or a tracheal canula serves as an invasive interface between the patient and the ventilator. NIV can be used either before intubation or following extubation. The classical use of NIV therapy in a hospital setting is following extubation, in particular in cases where spontaneous breathing is not sufficient for adequate gas exchange. Increasingly, the early use of NIV on the ICU now serves as a first line interventional tool to prevent intubation. Ideally, the entire spectrum of ventilation therapy (prevent – stabilize – wean – recover) can be adequately covered using NIV **(Ram, 2005).**

NIV may be somewhat more labor-intensive than conventional invasive ventilation, but benefits such as reduced length of stay on the ICU**, (Lellouche, 2006)** shorter ventilation times and the lower incidence of nosocomial pneumonia and their subsequent positive effects in terms of both cost and outcome can make NIV well worth the effort **(Hunter, 2006).**

All patients should be closely monitored following extubation. In many patients, early aggressive management with oxygenation and airway clearance can prevent reintubation. This may include suctioning, bronchodilator therapy, diuresis, or noninvasive positive pressure ventilation (NPPV) **(Tiruvoipati et al., 2010).**

Patients most likely to benefit from the early application of NPPV following extubation include those with chronic obstructive pulmonary disease (COPD), especially those who have compensated hypercapnia during their pre-extubation spontaneous breathing trial. In addition, a trial of NPPV is reasonable in patients if impending acute hypercapnic respiratory failure develops soon after extubation. Patients should be promptly intubated if they either fail the trial of NPPV or they develop definitive acute respiratory failure before the trial of NPPV can be initiated **(Burns et al., 2013).**

AIM OF THE WORK

To assess whether early application of non invasive ventilation, immediately after extubation, is effective in decreasing length of postextubation intensive care unit stay in patients with respiratory failure type II.

Acknowledgments

*First of all thanks to gracious **ALLAH** who guide me to the frank clear way and offers me great assistance of honest professors and lecturers.*

*It is my pride to record my gratitude to **Prof. Hoda Ali Abou Youssef**, Professor of Chest Diseases, Faculty of Medicine, Cairo University, for her supervision, precision, constructive instructions and guidance from the very early stage of this research. I was extraordinarily fortunate in having her as my professor, and what she has done throughout this work.*

*I am extremely grateful to **Prof. Amany Atef Elkarim Abou Zeid**, Professor of Chest Diseases, Faculty of Medicine, Cairo University for her continuous guidance, support, his precise scientific opinions ,his encouragement during the progress of this work and valuable suggestions from the very early stage of this research.*

*I would also like to thank **Dr. Raef Hosny Emam**, Assistant Professor of Chest Diseases, Faculty of Medicine, Cairo University, without his participation and input; the validation survey could not have been successfully conducted. I do not have enough words to thank him.*

*My sincere thanks and appreciation to **Dr. Hebatallah Hany assal**, Lecturer of Chest Diseases, Faculty of Medicine, Cairo University, the person behind all efforts and good things in this work and the source for moral and scientific support, for her great help and advice.*

Yasser Mehrez

Abstract

Rationale: Respiratory failure after extubation and reintubation is associated with increased ICU stay, morbidity and mortality.

Objectives: To assess whether early application of non invasive ventilation, immediately after extubation, is effective in decreasing length of postextubation intensive care unit stay in patients with respiratory failure type II.

Methods: A prospective randomized controlled trial was conducted in 56 mechanically ventilated patients who tolerated a spontaneous breathing trial after recovery from the acute episode. Patients were randomly allocated after extubation to receive noninvasive ventilation for 24 h (n = 26), or standard medical treatment with oxygen therapy (control group, n = 26).

Measurements and Main Results: Outcome variables in this study were, Trial duration was significantly shorter in NIV group than SMT group, and ICU stay was shorter in NIV group than SMT group but did not reach the level of significance**.** Respiratory failure and reintubation were less frequent in NIV group than SMT group but did not reach the level of significance (3 NIV versus 8 SMT, P =0.205). The time from extubation to respiratory failure and reintubation was longer in NIV group than SMT group and did not affect the mortality which was more frequent in SMT group than NIV group but did not reach the level of significance so this supports the use of NIV early after extubation in all patients regardless of risk for respiratory failure.

Conclusions: it was concluded that early use of non-invasive ventilation after extubation decreased ICU stay, diminished risk of respiratory failure after extubation and reduced mortality in patients with respiratory failure type Π.

Chronic Obstructive Pulmonary Disease

Definition:

According to **GOLD 2014**, COPD is a common preventable and treatable disease is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases, exacerbations and co-morbidities contribute to the overall severity in individual patients. COPD is a leading cause of morbidity and mortality worldwide and results in an economic and social burden that is both substantial and increasing **(Lopez et al., 2006).**

Chronic obstructive pulmonary disease (COPD), also known as chronic obstructive lung disease (COLD), and chronic obstructive airway disease (COAD), among others, is a type of obstructive lung disease characterized by chronically poor airflow. It typically worsens over time. The main symptoms include shortness of breath, cough, and sputum production **(Vestbo and Jørgen, 2013)**. Most people with chronic bronchitis have COPD **(Reilly et al., 2011).**

Signs and symptoms:

The most common symptoms of COPD are sputum production, shortness of breath and a productive cough **(Vestbo and Jørgen, 2013).**These symptoms are present for a prolonged period of time **(Reilly et al., 2011)** and typically worsen over time **(Rabe et al., 2007)**. It is unclear if different types of COPD exist **(Decramer et al., 2012)**. While previously divided into emphysema and chronic bronchitis, emphysema is only a description of lung changes rather than a disease itself, and chronic bronchitis is simply a descriptor of symptoms that may or may not occur with COPD **(Vestbo and Jørgen, 2013)**.

Cough

A chronic cough is usually the first symptom to occur. When it exists for more than three months a year for more than two years, in combination with sputum production and without another explanation, there is by definition chronic bronchitis. This condition can occur before COPD fully develops. The amount of sputum produced can change over hours to days. In some cases the cough may not be present or only occurs occasionally and may not be productive. Some people with COPD attribute the symptoms to a "smoker's cough". Sputum may be swallowed or spat out, depending often on social and cultural factors. Vigorous coughing may lead to rib fractures or a brief loss of consciousness. Those with COPD often have a history of "common colds" that last a long time **(Vestbo and Jørgen, 2013)**.

Shortness of breath

Shortness of breath is often the symptom that bothers people the most **(National Institute for Health and Clinical Excellence**, **2010).** It is commonly described as: "my breathing requires effort," "I feel out of breath," or "I can't get enough air in" **(Mahler, 2006)**. Different terms, however, may be used in different cultures **(Vestbo and Jørgen, 2013).**

Typically the shortness of breath is worse on exertion, of a prolonged duration, and worsens over time **(Vestbo and Jørgen, 2013).** In the advanced stages it occurs during rest and may be always present **(National Heart, Lung, and Blood Institute**, **2013)**. It is a source of both anxiety and a poor quality of life in those with COPD **(Vestbo and Jørgen, 2013)**. Many people with more advanced COPD breathe through pursed lips and this action can improve shortness of breath in some **(Morrison et al., 2013).**

Other features

In COPD, it may take longer to breathe out than to breathe in **(Gruber et al., 2008).** Chest tightness may occur **(Vestbo and Jørgen, 2013)** but is not common and may be caused by another problem **(National Institute for Health and Clinical Excellence**, **2010)**.

Those with obstructed airflow may have wheezing or decreased sounds with air entry on examination of the chest with a stethoscope **(Gruber et al., 2008)**. A barrel chest is a characteristic sign of COPD, but is relatively uncommon **(Gruber et al., 2008).** Tripod positioning may occur as the disease worsens **(Reilly et al., 2011).** Advanced COPD leads to high pressure on the lung arteries, which strains the right ventricle of the heart **(Weitzenblum and Chaouat, 2009).**

This situation is referred to as corpulmonale, and leads to symptoms of leg swelling **(Vestbo and Jørgen, 2013)** and bulging neck veins **(Rabe et al., 2007)**. COPD is more common than any other lung disease as a cause of corpulmonale **(Weitzenblum and Chaouat, 2009)**. Corpulmonale has become less common since the use of supplemental oxygen **(Reilly et al., 2011)**.

COPD often occurs along with a number of other conditions, due in part to shared risk factors **(Decramer et al., 2012).** These conditions include: ischemic heart disease, high blood pressure, diabetes mellitus, muscle wasting, osteoporosis, lung cancer, anxiety disorder and depression **(Decramer et al., 2012)**. In those with severe disease a feeling of always being tired is common **(Vestbo and Jørgen, 2013).**

Fingernail clubbing is not specific to COPD and should prompt investigations for an underlying lung cancer **(Mandell et al., 2009).**

Exacerbation

An acute exacerbation of COPD is defined as increased shortness of breath, increased sputum production, a change in the colour of the sputum from clear to green or yellow, or an increase in cough in someone with COPD **(Gruber et al., 2008).** This may present with signs of increased work of breathing such as fast breathing, a fast heart rate, sweating, active use of muscles in the neck, a bluish tinge to the skin, and confusion or combative behavior in very severe exacerbations **(Brulotte and Lang, 2012)**. Crackles may also be heard over the lungs on examination with a stethoscope (**Spiro and Stephen, 2012)**.

Causes:

The primary cause of COPD is tobacco smoke, with occupational exposure and pollution from indoor fires being significant causes in some countries **(Vestbo and Jørgen, 2013)**. Typically these exposures must occur over several decades before symptoms develop **(Vestbo and Jørgen, 2013).** A person's genetic makeup also affects the risk **(Vestbo and Jørgen, 2013)**.

Smoking

Percentage of males smoking tobacco as of the late 1990s early 2000s. Note the scales used for females and males differ **(Who, 2008)**.The primary risk factor for COPD globally is tobacco smoking **(Vestbo and Jørgen, 2013).** Of those who smoke about 20% will get COPD **(Ward and Helen, 2012)**, and of those who are lifelong smokers about half will get COPD **(Laniado, 2009)**.

In the United States and United Kingdom, of those with COPD, 80-95% are either current smokers or previously smoked **(Rennard and Stephen, 2013).** The likelihood of developing COPD increases with the total smoke exposure **(Goldman and Lee, 2012).** Additionally, women are more susceptible to the harmful effects of smoke than men **(Anita Sharma et al., 2010).** In non-

smokers, secondhand smoke is the cause of about 20% of cases **(Rennard and Stephen, 2013).** Other types of smoke, such as marijuana, cigar, and water pipe smoke, also confer a risk **(Vestbo and Jørgen, 2013).** Women who smoke during pregnancy may increase the risk of COPD in their child **(Vestbo and Jørgen, 2013).**

Air pollution

Poorly ventilated cooking fires, often fueled by coal or biomass fuels such as wood and animal dung, lead to indoor air pollution and are one of the most common causes of COPD in developing countries **(Kennedy et al., 2007).** These fires are a method of cooking and heating for nearly 3 billion people with their health effects being greater among women due to more exposure **(Vestbo and Jørgen, 2013)**.

They are used as the main source of energy in 80% of homes in India, China and sub-Saharan Africa **(Pirozzi et al., 2012).** People who live in large cities have a higher rate of COPD compared to people who live in rural areas **(Halbert et al., 2006)**. While urban air pollution is a contributing factor in exacerbations, its overall role as a cause of COPD is unclear **(Vestbo and Jørgen, 2013)**. Areas with poor outdoor air quality, including that from exhaust gas, generally have higher rates of COPD **(Pirozzi et al., 2012).** The overall effect in relation to smoking, however, is believed to be small **(Vestbo and Jørgen, 2013)**.

Occupational exposures

Intense and prolonged exposure to workplace dusts, chemicals and fumes increase the risk of COPD in both smokers and nonsmokers **(Devereux and Graham, 2006).** Workplace exposures are believed to be the cause in 10-20% of cases **(Laine and Christine, 2009)**.

In the United States they are believed to be related to more than 30% of cases among those who have never smoked and probably represent a greater risk in countries without sufficient regulations **(Vestbo and Jørgen, 2013).**

A number of industries and sources have been implicated, including **(Pirozzi et al., 2012)** high levels of dust in coal mining, gold mining, and the cotton textile industry, occupations involving cadmium and isocyanides, and fumes from welding **(Devereux and Graham, 2006).**

Working in agriculture is also a risk **(Pirozzi et al., 2012).** In some professions the risks have been estimated as equivalent to that of half to two packs of cigarettes a day **(Laine and Christine, 2009)**. Silica dust exposure can also lead to COPD, with the risk unrelated to that for silicosis **(Rushton and Lesley, 2007)**.

The negative effects of dust exposure and cigarette smoke exposure appear to be additive or possibly more than additive **(Barnes et al., 2009).**

Genetics

Genetics play a role in the development of COPD **(Vestbo and Jørgen, 2013)**. It is more common among relatives of those with COPD who smoke than unrelated smokers **(Vestbo and Jørgen, 2013).** Currently, the only clearly inherited risk factor is alpha 1-antitrypsin deficiency (AAT**) (Foreman et al., 2012).**

This risk is particularly high if someone deficient in alpha 1-antitrypsin also smokes **(Foreman et al., 2012)**. It is responsible for about 1-5% of cases **(Foreman et al., 2012)** and the condition is present in about 3-4 in 10,000 people **(Reilly et al., 2011).** Other genetic factors are being investigated, **(Foreman et al., 2012)** of which there are likely to be many **(Pirozzi et al., 2012).**

Other

A number of other factors are less closely linked to COPD. The risk is greater in those who are poor, although it is not clear if this is due to poverty itself or other risk factors associated with poverty, such as air pollution and malnutrition **(Vestbo and Jørgen, 2013)**.

There is tentative evidence that those with asthma and airway hyper reactivity are at increased risk of COPD **(Vestbo and Jørgen, 2013)**. Birth factors such as low birth weight may also play a role as do a number of infectious diseases including HIV/AIDS and tuberculosis **(Vestbo and Jørgen, 2013)**. Respiratory infections such as pneumonia do not appear to increase the risk of COPD, at least in adults **(Reilly et al., 2011).**

Exacerbations

An acute exacerbation (a sudden worsening of symptoms) **(Vestbo and Jørgen, 2013)** is commonly triggered by infection or environmental pollutants, or sometimes by other factors such as improper use of medications **(Dhar and Raja, 2011).** Infections appear to be the cause of 50 to 75% of cases, **(Palange and Paolo, 2013)** with bacteria in 25%, viruses in 25%, and both in 25 % **(Lötvall et al., 2011)**.

Environmental pollutants include both poor indoor and outdoor air quality **(Dhar and Raja, 2011).** Exposure to personal smoke and secondhand smoke increases the risk **(Pirozzi et al., 2012)**. Cold temperature may also play a role, with exacerbations occurring more commonly in winter **(Barnes et al., 2009).** Those with more severe underlying disease have more frequent exacerbations: in mild disease 1.8 per year, moderate 2 to 3 per year, and severe 3.4 per year **(Hanania and Nicola, 2010).**

Those with many exacerbations have a faster rate of deterioration of their lung function **(Beasley et al., 2012)**. Pulmonary emboli can worsen symptoms in those with pre-existing COPD **(Decramer et al., 2012)**.

Pathophysiology:

COPD is a type of obstructive lung disease in which chronic incompletely reversible poor airflow (airflow limitation) and inability to breathe out fully (air trapping) exist **(Decramer et al., 2012)**. The poor airflow is the result of breakdown of lung tissue (known as emphysema) and small airways disease known as obstructive bronchiolitis. The relative contributions of these two factors vary between people **(Vestbo and Jørgen, 2013)**.

Severe destruction of small airways can lead to the formation of large air pockets—known as bullae—that replace lung tissue. This form of disease is called bullous emphysema **(Murphy and Fishman, 2008).**

COPD develops as a significant and chronic inflammatory response to inhaled irritants **(Vestbo and Jørgen, 2013)**. Chronic bacterial infections may also add to this inflammatory state **(Beasley et al., 2012)**. The inflammatory cells involved include neutrophil, granulocytes and macrophages, two types of white blood cell. Those who smoke additionally have Tc1 lymphocyte involvement and some people with COPD have eosinophil involvement similar to that in asthma. Part of this cell response is brought on by inflammatory mediators such as chemotactic factors. Other processes involved with lung damage include oxidative stress produced by high concentrations of free radicals in tobacco smoke and released by inflammatory cells, and breakdown of the connective tissue of the lungs by proteases that are insufficiently inhibited by protease inhibitors **(Vestbo and Jørgen, 2013)**.