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Review Article

Management of Respiratory Failure in the Emergency Department

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Abstract

Introduction: A commonly encountered complication in the emergency department (ED) is that of acute respiratory failure. The long term effects of this can be influenced by early treatment.The improved outcome of acute exacerbations of chronic obstructive lung disease and congestive heart failure can be attained by non-invasive ventilation which is commonly used in patients with respiratory failure.It is imperative to understand the underlying causes of the condition so as to correctly identify the underlying pathology.

The Aim of Work: The review aimed to summarize the pathophysiology of management options and rescue therapies, including airway pressure release ventilation, continuous neuromuscular blockade, inhaled nitric oxide, and extracorporeal membrane oxygenation of acute respiratory failure.

Methodology: The review is comprehensive research of PUBMED from the year 1980 to 2015.

Conclusion: Even though the occurrence of acute respiratory failure is common in the ED, the correct management of it can be complicated and is of utmost importance. The outcome of treatment relies on the ability to assess ventilation, oxygenation, lunch function, airway resistance, and airflow. Newer techniques of ventilation like noninvasive ventilation have been developed over the last few years. Inappropriate initial treatment can lead to increased mortality amongst the older group of patients.

Keywords: *Respiratory Failure; Therapy; Emergency Management; The Timing of Surgery; Initial Management*

Introduction

A commonly encountered condition in critically ill patients in the emergency department (ED) is that of acute respiratory failure. Exacerbations of chronic obstructive pulmonary diseases account for about 1.5 million visits to the emergency department, asthma exacerbations account for about 2 million, cardiogenic pulmonary edema accounts for over 1 million hospitalizations and acute lung injury or acute respiratory distress syndrome accounts for about 200,000 admissions [1-3]. A common observation is that of patients who receive suboptimal mechanical ventilation in the ED, which can be harmful to the patient [3].

The inability of the respiratory system to exchange gases and to oxygenate the blood adequately is what is called Acute Respiratory Failure (ARF). Two of the mechanisms which can be distinguished on the basis of ARF are

- Failure in pulmonary ventilation (pump failure);
- Failure in gas exchanges (lung failure).

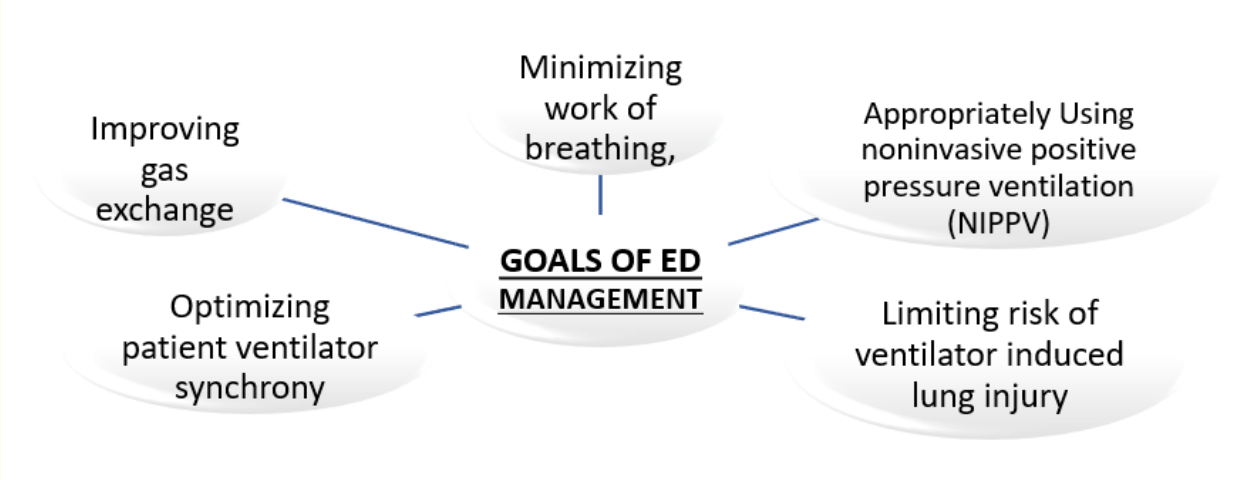

Figure 1

Two of the main manifestations of pulmonary diseases, which are arterial hypercapnia and hypoxemia, are the clinical sign and symptoms of ARF [4].

The pathophysiology of hypercapnia could be due to

1. Increase in CO_2 production
2. Deteriorated gaseous exchanges as the increase in alveolar death space ventilation
3. Deterioration in respiratory mechanics
4. Alteration in the mechanism of control of the ventilation

And that of hypoxemia could be because of

1. Hypoventilation
2. Alteration in gas diffusion
3. Mismatch in ventilation/perfusion ratio
4. Pulmonary shunt.

The part of non-invasive ventilation, the timing of intubation, pharmacologic and nonpharmacological rescue therapies and their role in the ED, and the role of extracorporeal membrane oxygenation (ECMO) still remains a matter of controversy. A state of art treatment of acute respiratory failure and treatment therapies can be considered for patients rather than deferring them to the ICU for management [3].

Non-invasive positive pressure ventilation (NIPPV)

To allow the movement of air and to enable gaseous exchange, the respiratory muscles have to overcome resistive and elastic forces. Resistive work will help overcome the resistance of airflow, and elastic work will help to overcome the functional residual capacity in which the lung remains. It is important to understand the relationships that contribute to breathing because then only a better understanding of why certain conditions lead to respiratory failure can be gained. Usually, patients in shock have increased work of breath-

ing which can be reduced by mechanical ventilation. There is also a reduction in the high oxygen consumption; hence it is a preferred treatment for shock. In conditions like chronic obstructive pulmonary disease (COPD) due to the increase in airways resistance, there is increased work of breathing. The dynamic hyperinflation causes increased elastance. The development of auto–positive end-expiratory pressure (PEEP) is seen due to the inequality of volumes. With the use of Non-Invasive Positive Pressure Ventilation (NIPPV), there seems to be a reduction of the elastic work of breathing [5,6].

The usage of NIPPV for respiratory failure is increasing due to the benefits that can be seen in improved breathing and respiratory support [7,8]. The outcomes of NIPPV can be used effectively in the treatment of COPD exacerbations, decompensated congestive heart failure, and immune compromised patients but it doesn't give the greatest benefits in acute respiratory distress syndrome and pneumonia [9]. NIPPV is also commonly used in mixed respiratory failure. NIPPV reduces the need for intubation in respiratory failure but intubation after failed NIPPV results in increased mortality. Higher rates of mortality are associated with patients with chronic obstructive pulmonary disease who are intubated primarily [7]. NIPPV has a high failure rate in hypoxemic respiratory failure. It is still uncertain what among initial patient selection, disease progression, or intubation-related complications accounts for the NIPPV mortality [8].

Ventricular failure

When minute ventilation is no longer sufficient to remove carbon dioxide from the circulation, there is an increase in the arterial CO_2 [$PaCO_2$] and a decrease in Ph and is called ventricular failure. This could be due to hypercapnic respiratory failure (type 2) or an increase in carbon dioxide production. Dead space is that portion of breath that does not participate in gaseous exchange. If there is a volume increase in conducting airways or a decreased blood supply to alveoli, an increase in the dead space will result. This, in turn, will decrease the alveolar.

Abnormal range of alveolar ventilation causes metabolic acidosis which requires respiratory compensation which cannot be met by increasing the alveolar ventilation alone [3].

Recommendations for emergency department management

Patients with COPD or asthma can have ventilator failure which can be treated with NIPPV in the absence of complications (which include the inability to protect the airway, vomiting, hemodynamic instability, excessive secretions, or inability to tolerate accidental removal of NIPPV mask). Improved work of breathing, improving the symptoms reducing the mortality and decreases the need for intubation can be expected with NIPPV as opposed to oxygenation alone [11]. The use of NIPPV is less commonly recommended in asthmatic patients because the regional hyperinflation that can be caused by mucus plugs and flow restriction due to bronchospasm can increase the susceptibility to pneumothorax [3]. The blood gas analysis should be monitored for a while the patient receives NIPPV (Every two hours).A venous blood gas can be used instead of arterial blood gas in patients with COPD without hypoxemia [3].

In COPD and asthma, invasive mechanical ventilation can be done with a pressure of volume targeted mode. The easiest way is using a volume-targeted mode (i.e. assist control or pressure-regulated volume control) with a low respiratory rate (10 to 12 breaths/min) along with a neuromuscular blocker. Until the expiratory flow waveform comes back to baseline before the next breath, the respiratory rate should be decreased. In the absence of air trapping, an inspiratory pause should be performed to elevate the plateau pressure. A portable radiograph or ultrasound must be done if the plateau pressure is above 30cm H_2O [12].

Manipulating the respiratory rate has become the primary means of adjusting ventilation because of the latest focus on lung-protective ventilation with low tidal volumes. If there is an increase in the respiratory rate, there seems to be a decrease in the time spent in exhalation. This could increase the risk of dynamic hyperinflation because more air is entering during inspiration than what leaves during expiration. This will, in turn, lead to increased intrathoracic pressure and a higher inspiratory threshold pressure, which is required to trigger the ventilator, which could eventually impair the venous return to the heart. In non-paralyzed patients with severe cases of meta-

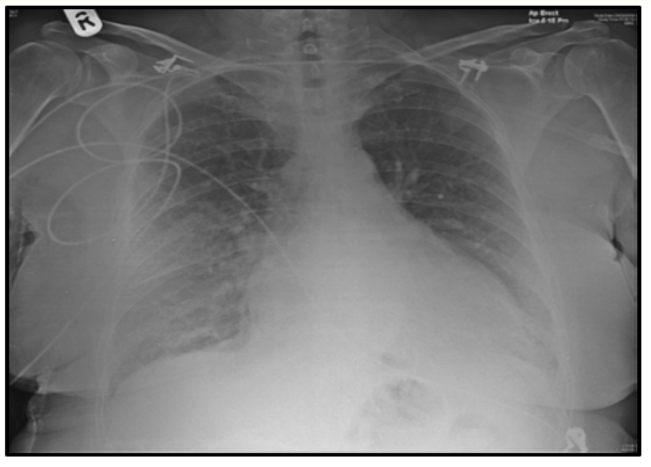

Figure 2: *Portable anteroposterior chest radiograph showing cardiomegaly and overinflated lungs [13].*

bolic acidosis (e.g. diabetic ketoacidosis, salicylate toxicity pressure support (or "spontaneous") mode ventilation with adequate pressure support could be useful [12].

Oxygenation failure

Ventilation-perfusion (VQ) mismatch and shunt are the most common clinical causes of hypoxemic respiratory failure. The increased partial pressure of carbon dioxide ($PC0_2$) in the alveolar space can displace oxygen which leads to hypoxemia caused by hypoventilation [14].

An under perfused or under-ventilated alveoli can result from VQ mismatch, disruption of the optimal ratio of alveolar ventilation to alveolar perfusion. Dead space ventilation commonly seen in COPD can result due to a high VQ ratio that occurs with under perfused alveoli relative to ventilation. When the perfused alveolar units do not participate in gas exchange, a low VQ ratio, or shunt physiology (anatomic or physiologic) occurs [15].

Direct or indirect lung injury can cause serious life-threatening acute respiratory distress syndrome which could result in hypoxemic respiratory failure. This represents a severe form of VQ mismatch which is usually seen in the ED, as exudative alveolar filling leading to shunting physiology [16].

Acute respiratory distress syndrome –rescue oxygenation

The use of airway pressure release ventilation is beneficial as it can maintain spontaneous breathing and improve alveolar recruitment apart from improving oxygenation in patients with severe airspace disease. It can maintain a high airway pressure of about 30 cm H_2O for 3 to 4 seconds to intermittently releasing the pressure for setting the PEEP for a short duration (e.g. 10 cm H_2O for 0.5 to 1 second) to allow for ventilation. However, the risk of injurious tidal volumes because of the change in lung compliance persists. There could be an increase in the tidal volume, which exceeds the lung-protective volumes which can result in volutrauma if the compliance improves with the resolving disease. If there is a worsening of compliance, the tidal volumes will decrease and result in ineffective ventilation, which is why it is important to monitor tidal volumes in airway pressure release ventilation. This method has been beneficial for patients who suffer from respiratory distress due to H1N1 of those who are at risk of developing respiratory distress due to high-risk trauma [17-19].

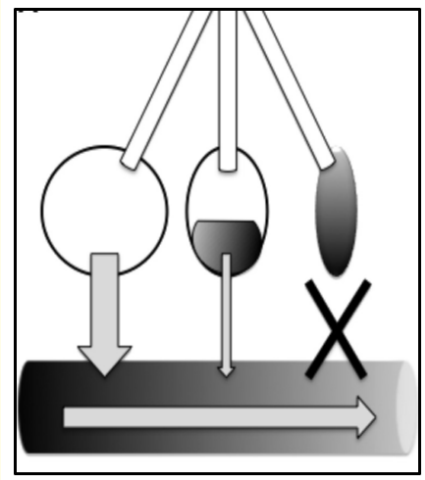

Figure 3: *VQ mismatch showing wide, narrow or prohibited gaseous exchange [3].*

As an adjunct therapy to improve patient-ventilator synchrony and chest wall compliance, the continuous neuromuscular blockade can be used. However, the risk of developing critical illness myopathy and high sedative requirements, leading to delirium and long-term cognitive abnormalities are seen [20].

Delivery of inhaled nitric oxide to well-ventilated portions of the lung can dilate the surrounding vasculature that improves VQ matching and oxygenation. However, there is no evidence that it can improve mortality in patients but does improve oxygenation and reduce oxidative stress. Non-pharmacological method of keeping the patient in a prone position can help to reduce respiratory distress. There is an improvement in the VQ match. Mechanically supporting systemic oxygenation or cardiac output by removing venous blood from a large cannula in a central vein, oxygenating and removing carbon dioxide with a membrane lung, and returning that oxygenated blood to the circulation.is known as ECMO (extracorporeal membrane oxygenation).Since cannulation is common in ECMO, a systemic inflammatory response due to cytokine release can warrant the use of anticoagulation [3].

Recommendations for emergency department management

NIPPV is useful in the management of oxygenation failure since it can improve the work of breathing, reduces symptoms, mortality, and need for intubation and mechanical ventilation in patients with cardiogenic pulmonary edema. PEEP can help to improve the left ventricular performance which can benefit in cases with cardiogenic pulmonary edema. In the case of high work of breathing with inspiration, inspiratory pressure support is desired. Post-end-expiratory pressure (PEEP) 8 to 10 cm H_2O should be the initial NIPPV setting and inspiratory pressure support of 12 to 15 cm H_2O. PEEP can be increased up to 15 cm H_2O if there is persistent hypoxemia or tachypnea and then intubation can be considered if no improvement is seen. If NIPPV is being used for hypoxemic respiratory failure due to pneumonia or acute respiratory distress syndrome, extreme caution should be taken because of the high risk of failure and poor outcomes in patients in whom treatment fails. A volume targeted mode (assist control, synchronized intermittent mandatory ventilation, or pressure regulated volume control) is recommended during invasive mechanical ventilation for hypoxemic respiratory failure [3].

Managing patients with dyspnoea

- Dyspnoea, cyanosis, tachypnea, accessory muscle use, paradoxical breathing, and tachycardia are the signs of respiratory distress patients with ARF usually presents with. Firstly Acute Upper Airway Obstruction (AUAO) must be excluded after which the following can be done [4].

- Administration of high flows of oxygen, measure the BP, record the heart rate, respiratory rate, oxygen saturation, to make an electrocardiogram. Evaluation of CK, CKMB, T-troponin, LDH, AST, ALT, and hemochrome must be done.
- The neurological score (Kelly score) should be evaluated. If it is more than 3 resuscitators must be asked for
- Arterial blood gas (ABG) evaluation can be done if the neurological state has not been compromised.
- The clinical evaluation must be supported with appropriate radiological evaluation and continuous ABG evaluations.in the case of a deterioration of clinical conditions, endotracheal intubation must be done [4].

Managing patients with acute upper airway obstruction (AUAO)

The condition should be differentiated as intrathoracic or extrathoracic airway obstruction. Clinical signs like stridor are commonly associated with cyanosis, loss of consciousness, nape's hyperextension, trismus, and dysphagia should be looked out for. Blood pressure and oxygen saturation, and control of ABG should be done in those patients who are stable with no cardiovascular variations. The use of anaphylaxis is recommended in patients with the glottis.

Edema before complete obstruction of the airways [4].

Managing patients with ARF due to neuromuscular diseases or chest wall deformities

Hypoventilation is the reason for ARF in patients with chest wall diseases. The evaluation of ABG after 1 hour to control clinical conditions must be done if the arterial blood sample shows an oxygen saturation > 90% and a $PaCO_2$ < 45 mmHg. A chest radiograph can help the physician with differential diagnosis. In case of unstable vital functions like a cardio-circulatory arrest, a team to follow the ALS algorithm followed by immediate admission of the patient to the Intensive Care Unit (ICU) [4].

Managing patients with hypoxemic ARF

Pulmonary oedema

It can be cardiogenic and non-cardiogenic (commonly known as Acute Respiratory Distress Syndrome). The use of oxygen, nitrates, diuretics, and, if coexists cardiogenic shock, inotropes can be used in the treatment of acute pulmonary edema. The first line of treatment would be administering furosemide and nitrates (10 microns/min) along with morphine (if the arterial systolic blood pressure > 100 mmHg). Dopamine (5 - 15 microg/kg/min) can be administered in case of signs of cardiogenic shock which will help increase the contractility and improve the arterial blood pressure. Administration of oxygen should be done with face masks with Venturi system. The use of CPAP along with NIV is gaining popularity for patients with cardiogenic pulmonary edema [4].

Pneumonia

Oxygen should be administered to obtain an increase in saturation major that 90% if the oxygen blood partial pressure is less than 70 mmHg. NIV can also be used instead of endotracheal intubation if the patient becomes hypercapnic. The most commonly used antibiotic is with beta-lactam alone (amoxicillin ± clavulanic acid or second and third generation intravenous cephalosporin). Treatment with *Mycoplasma* and/or *Chlamydia pneumonia* may be involved and so treatment with doxycycline/macrolide or fluoroquinolone should be initiated when the earlier antibiotic therapy fails. Within 8 hours of admission of the patient, the empirical therapy should begin. In the case of aspiration pneumonia treatment against anaerobic bacteria should be begun. It is better to use high and frequent dosage as opposed to extending the treatment as there is a possibility to develop resistance [4].

Managing patients with ARF due to COPD

Once the confirmatory clinical evaluation, diagnostic tests like chest radiography, spirometry, and serological assays like sputum samples etc. are used to confirm the condition, ECG can confirm the presence of cor pulmonale. If a pulmonary embolism is suspected, a CT scan can be done. Bronchodilators are the central management of the clinical symptoms of the condition. Vagal stimulation of the bronchial tree can be inhibited with anticholinergic drugs, which helps to reduce the tone of the smooth muscles. A new long-acting an-

ticholinergic bronchodilator that must be inhaled once daily has been reported with tiotropium. The combined use of beta-agonists and anticholinergics are more effective and better tolerated than higher doses of using either of the agents alone [21].

Theophylline, a non-selective inhibitor of phosphodiesterase, can increase the central respiratory drive, respiratory muscle endurance, mucociliary clearance, cardiac output and dilation of pulmonary arteries apart from bronchodilation. However, they have a narrow therapeutic index. Short course oral glucocorticoids (0.5 mg of prednisone or prednisolone/kg body weight) have been used recently. In patients with severe COPS, inhaled glucocorticoids are recommended. Long term inhaled glucocorticoid therapy can be used in patients with acute bronchodilator reversibility [4].

Conclusion

Two forms (type 1/hypoxemic respiratory failure or type 2/hypercapnic respiratory failure) are the commonly encountered acute respiratory failure in the emergency department. The two primary goals of respiration are oxygenation and ventilation, both of which can be accomplished by energy. Respiratory rate, resistance to airflow, and the elastance of the respiratory system determine the work of breathing. During acute exacerbations of chronic obstructive pulmonary disease and congestive heart failure, NIPPV has shown improved outcomes; however it must be used carefully for managing asthma, pneumonia, and acute respiratory distress syndrome.

In the ED, initiation of invasive mechanical with a volume control mode must be done. Even in the absence of acute respiratory distress syndrome, Lung-protective tidal volumes should be provided. The goal of invasive mechanical ventilation is to control the mean airway pressure, surge the expiratory time, augment set PEEP to improve work of breathing in ventilatory failure caused by asthma or COPD. Rescue therapies such as airway pressure release ventilation, neuromuscular blockade, prone positioning, inhaled nitric oxide, or ECMO should be considered for refractory hypoxemia. A critical and common aspect of emergency medicine is the management of acute respiratory failure is. It is important to know the underlying pathophysiology, which can help the emergency physician to manage the acute respiratory failure appropriately.

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