Introduction

Noninvasive positive-pressure ventilation (NPPV) is the delivery of mechanical ventilation to patients with respiratory failure without the requirement of an artificial airway ***(Oscar et al., 2007).***

The concept of positive airway pressure (PAP) started in the ancient Middle East when midwives used bellows with modified nasal adaptors to resuscitate newborns. However, the introduction of positive-pressure ventilation into modern medicine did not occur until the disastrous polio epidemic in the middle of last century ***(Andreea and Sairam, 2010)***.

Interfaces are devices that connect the ventilator tubing to the patient's face and facilitate the entry of pressurized gas into the upper airway. The choice of interface is a crucial issue in noninvasive ventilation. Currently available interfaces include nasal, oronasal and facial masks, mouthpieces and helmets. Studies have not shown a clear superiority of one interface over the others. For treatment of acute respiratory failure, facial masks are most commonly used (70% of cases), followed by nasal masks (25%) and nasal pillows (5%) ***(Oscar et al., 2007).***

Although NPPV has been in use in patients with chronic respiratory failure for decades, its use in patients with acute respiratory failure is relatively more recent. The most widely utilized and familiar form of NPPV is mask continuous positive airway pressure (CPAP).

Duke and Bersten describe five common modes of NPPV:

1. Continuous positive airway pressure (CPAP).
2. Pressure support ventilation (PSV).
3. Bi-level positive airway pressure (BiPAP).
4. Other combinations of mask CPAP and PSV.
5. Controlled and assisted modes of mask intermittent positive pressure ventilation (IPPV).

(Craig, 2002).

There are some recent indications of noninvasive ventilation (NIV) such as respiratory problems associated with neuromuscular diseases that damage the respiratory muscles or their supplying nerves resulting in reduced lung volumes and may cause chronic respiratory failure that is characterised by respiratory distress and altered arterial blood gas levels (in particular raised levels of arterial carbon dioxide) ***(Trevor, 2005).***

It is becoming clearer that NIV in motor neuron disease, amyotrophic lateral sclerosis and duchenne muscular dystrophy can prolong survival as well as improve quality of life in some patients ***(Simonds, 2003).***

Non-invasive ventilation is increasingly being used by patients at home. They receive support from supplying centres to maintain this treatment, although they may also seek help from nurses working in the community. There are three main conditions that benefit from domiciliary NIV:

1. Obstructive sleep apnea.
2. Respiratory problems associated with neuromuscular disease and chest wall deformities.
3. Chronic obstructive pulmonary disease (COPD).

(Trevor, 2005).

Noninvasive ventilation is postulated to improve abnormal physiologic effects of various diseases by reducing the work of breathing, improving oxygenation and alveolar ventilation and thereby reducing arterial carbon dioxide tension. These effects in turn can reduce the need for endotracheal mechanical ventilation as well as the rates of its associated complications ***(Andrew, 2011).***

There are many Complications associated with the application of NIV include cases of decubitus skin necrosis (10%), aspiration pneumonia (5%), hypotension (5%), gastric distension (3%) and dry eyes and mouth. However, orotracheal intubation (OTI) is associated with more adverse effects and potential complications, including: loss of verbal communication, impaired oral and pharyngeal flora, impaired mucociliary clearance, increased airway resistance, problems with weaning after prolonged OTI time, sedation and muscle relaxants, nosocomial pneumonia, laryngotracheal stenosis and the need for tracheostomy ***(Fernando et al., 2009).***

Aim of the Work

To evaluate the non-invasive ventilation and its role in management and outcome of the critically ill patient suffering acute respiratory failure.

Chapter (1)
Pathophysiology of Respiratory Failure

Physiology of respiration:

From our first breath at birth, the rate and depth of our respiration is unconsciously matched to our activities, whether studying, sleeping, talking, eating or exercising. We can voluntarily stop breathing, but within a few seconds we must breathe again. Breathing is so characteristic of life that, along with the pulse, it's one of the first things we check for to determine if an unconscious person is alive. Breathing is necessary because all living cells of the body require oxygen and produce carbon dioxide. The respiratory system allows exchange of these gases between the air and the blood, and the cardiovascular system transports them between the lungs and the cells of the body. The capacity to carry out normal activity is reduced without healthy respiratory and cardiovascular systems ***(Seeley et al., 2004).***

There are four major functions of respiration (1) pulmonary ventilation, which means the inflow and outflow of air between the atmosphere and the lung alveoli; (2) diffusion of oxygen and carbon dioxide between the alveoli and the blood; (3) transport of oxygen and carbon dioxide in the blood and body fluids to and from the body's tissue cells; and (4) regulation of ventilation ***(Guyton and Hall, 2011).***

The term ventilation is defined as the process that exchanges gases between the external environment and the alveoli. It is the mechanism by which oxygen is carried from the atmosphere to the alveoli and by which carbon dioxide (delivered to the lungs in mixed venous blood) is carried from the alveoli to the atmosphere ***(Terry, 2002).***

Pulmonary ventilation can be studied by recording the volume of movement of air into and out of the lungs. For ease in describing the events of pulmonary ventilation, the air in the lungs has been subdivided into four volumes and four capacities, which are the average for a young adult man;

I Pulmonary Volumes: four pulmonary lung volumes (Fig.1) that, when added together, equal the maximum volume to which the lungs can be expanded as:

1. The tidal volume (VT) is the volume of air inspired or expired with each normal breath (The average VT is 500 ml).
2. The inspiratory reserve volume (IRV) is the extra volume of air that can be inspired over and above the normal tidal volume when the person inspires with full force (the average IRV is 3000 ml).
3. The expiratory reserve volume (ERV) is the maximum extra volume of air that can be expired by forceful expiration after the end of a normal tidal expiration (the average ERV is 1000 ml).
4. The residual volume (RV) is the volume of air remaining in the lungs after the most forceful expiration (the average RV is 1200 ml) ***(Guyton and Hall, 2011).***

II Pulmonary Capacities: in describing events in respiratory cycle, it is sometimes desirable to consider two or more of the volumes together. Such combinations are called pulmonary capacities (Fig.1).

1. The inspiratory capacity (IC) equals the tidal volume plus the inspiratory reserve volume (equal VT +IRV = 3500 ml).
2. The functional residual capacity (FRC) equals the expiratory reserve volume plus the residual volume (about 2200 ml).
3. The vital capacity (VC) equals the inspiratory reserve volume plus the tidal volume plus the expiratory reserve volume (about 4500 ml).
4. The total lung capacity (TLC) is the maximum volume to which the lungs can be expanded with the greatest possible effort; it is equal to the vital capacity plus the residual volume (about 5700 ml) ***(Guyton and Hall, 2011).***

The ultimate importance of pulmonary ventilation is to continually renew the air in the gas exchange areas of the lungs, where air is in proximity to the pulmonary blood. These areas include the alveoli, alveolar sacs, alveolar ducts, and respiratory bronchioles. The rate at which new air reaches these areas is called alveolar ventilation which is one of the major factors determining the concentrations of oxygen and carbon dioxide in alveoli**.** The alveolar ventilation equal tidal volume (VT) minus dead space which is about 350 ml per each breath, and resting alveolar ventilation = 350×12 = 4200 ml per minute ***(Guyton and Hall, 2011).***

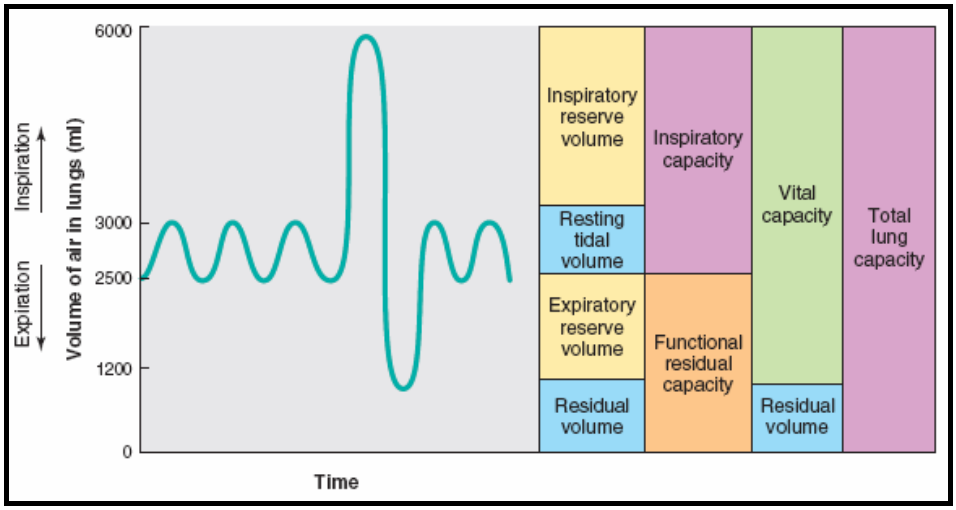

Figure (1): Lung volumes and capacities recorded on spirometer ***(Eric et al., 2003).***

The process of diffusion is the passive movement of gas molecules from an area of high partial pressure to an area of low partial pressure until both areas are equal in pressure and in the healthy resting individual, venous blood entering the alveolar-capillary system has an average oxygen tension (PvO_2) of 40 mm Hg, and an average carbon dioxide tension ($PvCO_2$) of 46 mm Hg. As blood passes through the capillary, the average alveolar oxygen tension (PAO_2) is about 100 mm Hg, and the average alveolar carbon dioxide tension ($PACO_2$)is about 40 mm Hg (fig. 2) ***(Terry, 2002).***

The exchange of gases between the air in the alveoli and the blood in the pulmonary capillaries is called external respiration and internal respiration is the exchange of gases between the blood in the systemic capillaries and the tissue fluid (cells) of the body.Within the body, a gas will diffuse from

an area of greater concentration to an area of lesser concentration.The concentration of each gas in a particular site (alveolar air, pulmonary blood, and so on) is expressed in a value called partial pressure (table1) ***(Valerie and Tina, 2007).***

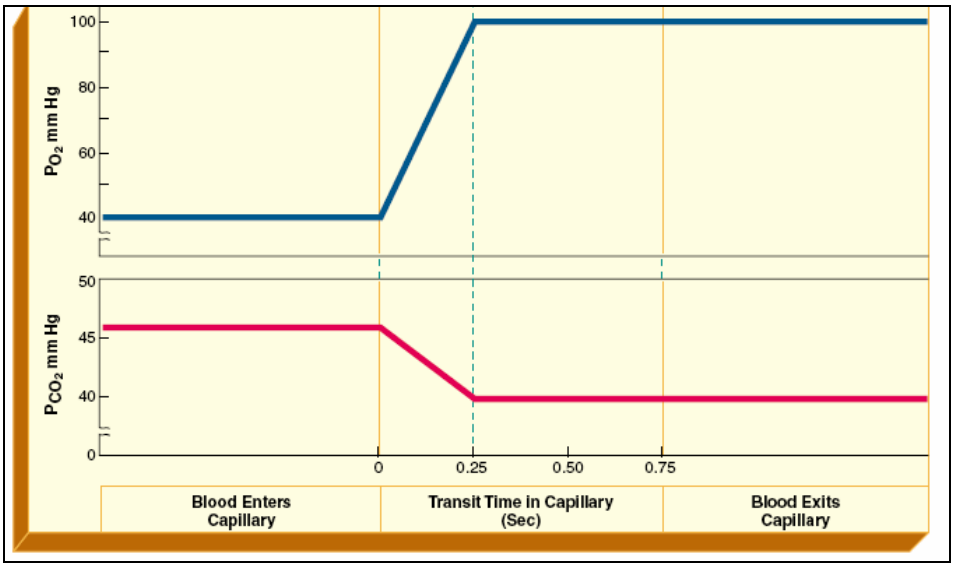

Figure (2): Gases pressures under normal resting condition through the alveolar-capillary membrane ***(Terry, 2002).***

The PO_2 of alveolar air is normally 100 mm Hg and the PO_2 of the blood entering the pulmonary capillaries is 40 mm Hg. The diffusing capacity for O_2 is about 25 mL/min/mm Hg, and the PO_2 of blood is raised to 97 mm Hg, a value just under the alveolar PO_2. This falls to 95 mm Hg in the aorta because of the physiologic shunt and on the other hand the PCO_2 of venous blood is 46 mm Hg, whereas that of alveolar air is 40 mm Hg, and CO_2 diffuses from the blood into the alveoli along this gradient. The PCO_2 of blood leaving the lungs is 40 mm Hg. CO_2 passes through all biological membranes with ease, and the

diffusing capacity of the lung for CO_2 is much greater than the capacity for O_2. It is for this reason that CO_2 retention is rarely a problem in patients with alveolar fibrosis even when the reduction in diffusing capacity for O_2 is severe (table 1) ***(Ganong, 2005).***

I. Definition of respiratory failure:

Respiratory failure results when the pump function of the respiratory muscles can no longer sustain a threshold level of alveolar gas exchange to meet the metabolic demands of cellular respiration. The magnitude of the metabolic "load"on the respiratory system is dependent on several factors, including the levels of tissue O_2 consumption and CO_2 production. The precise onset of respiratory failure is a dynamic process, due to the net balance of physiological variables that favor and impede inherent compensatory response ***(Elliot, 2005).***

Table (1): Partial pressures and oxygen saturation ***(Valerie and Tina, 2007).***

Site	PO_2	PCO_2	Hemoglobin Saturation (SaO_2)
Atmosphere	160	0.15	--
Alveolar air	104	40	--
Systemic venous blood	40	45	70 – 75 %
Systemic arterial blood	100	40	95 – 100 %
Tissue Fluid	40	50	--
Partial pressure is calculated as follow: % of the gas in the mixture x Total pressure = PGas Example: O2 in the atmosphere 21% x 760 mmHg = 160 mmHg (PO2)			

Also respiratory failure can be defined as arterial carbon dioxide tension ($PaCO_2$) greater than 50 mm Hg or arterial oxygen tension (PaO_2) less than 50 to 60 mm Hg ***(Wilson et al., 2006).***

Oxygenation and elimination of carbon dioxide are the primary gase exchange functions of the lung, and a failure of either one results in acute respiratory failure, so, acute respiratory failure is secondry either to failure of oxygenation (hypoxic respiratory failure) or to a failure of elimination of carbon dioxide (hypercapnic respiratory failure) ***(Frederic et al., 2008).***

II. Classification of respiratory failure:

One classification of respiratory failure separates disorders that affect the lungs (airways, alveolar spaces, interstitium, and pulmonary circulation) from those that affect primarily the nonlung components of the respiratory system.

A. Type I respiratory failure from diseases that directly affect the lungs almost always has hypoxemia, but these patients may or may not have hypercapnia depending on the type of disease and its severity such as pneumonia, aspiration of gastric contents, acute respiratory distress syndrome (ARDS), pulmonary embolism, asthma, and interstitial lung diseases (table 2) ***(Frederic et al., 2008).***

Table (2): Causes of acute hypoxemic respiratory failure ***(Hudson and Arthur, 2004).***

Diffuse Pulmonary Abnormalities
Cardiogenic Pulmonary Edema (CPE)
Adult Respiratory Distress Syndrome (ARDS)
Diffuse infectious pneumonitis
Alveolar hemorrhage
Pulmonary alveolar proteinosis
Focal Pulmonary Lesions
Lobar pneumonia
Atelectasis
Pulmonary contusion
Alveolar and pulmonary hemorrhage
Reperfusion pulmonary edema
Reexpansion pulmonary edema

B. Type II respiratory failure from disorders of the nonpulmonary respiratory system usually cause hypercapnia plus hypoxemia such as diseases that cause weakness of the respiratory muscles, CNS diseases that disrupt ventilatory control, and conditions that affect chest wall shape or size (table 3) ***(Frederic et al., 2008).***

C. Type III respiratory failure typically occurs in the perioperative period when factors that reduce functional residual capacity combine with causes of increased closing volume to produce progressive atelectasis ***(Lawrence, 2005).***

D. Type IV respiratory failure ensues when the circulation fails and resolves when shock is corrected ***(Lawrence, 2005).***

Table (3): Causes of hypercapnic respiratory failure ***(Hudson and Arthur, 2004).***

Abnormal Respiratory Capacity (Normal Respiratory Workloads):
Acute depression of central nervous system.
Chronic central hypoventilation syndromes.
Obesity-hypoventilation syndrome.
Sleep apnea syndrome.
Hypothyroidism.
Shy-Drager syndrome (multisystem atrophy syndrome).
Acute toxic paralysis syndromes.
Botulism, Tetanus, Toxic ingestion or bites and Organo-phosphate poisoning.
Neuromuscular disorders (acute and chronic).
Muscular dystrophies.
Myasthenia gravis, Guillain-Barre syndrome.
Amyotrophic lateral sclerosis.
Polymyositis.
Spinal cord injury and Traumatic phrenic nerve paralysis.
Abnormal Pulmonary Workloads:
COPD.
Asthma and acute bronchial hyperactivity syndromes.
Upper airway obstruction.
Interstitial lung diseases.
Abnormal Extrapulmonary Workloads:
Chronic thoracic cage disorders.
Severe kyphoscoliosis.
After thoracoplasty or thoracic cage injury.
Acute thoracic cage trauma and burns.
Pneumothorax.
Pleural fibrosis and effusions.

A. Hypoxic respiratory failure:

Oxygen stores in human lungs are extremely poor, being limited to the small amount present in the functional residual capacity (FRC), which functions as a sort of oxygen reservoir. In the case of apnea, such as after loss of consciousness, the amount of oxygen present at FRC continues to diffuse into the blood and is sufficient to maintain arterial oxygen saturation for no more than 1 min. Pathological changes of FRC, such as those that occur in restrictive or obstructive diseases, may also explain why patients can develop hypoxemia slowly (obstructive, with increased FRC) or quickly (restrictive, with decreased FRC) upon interruption of oxygen therapy ***(Ceriana et al., 2006).***

Pathogenesis of hypoxic respiratory failure (table 4)

1. Hypoventilation:

Arterial pressure of carbon dioxide ($PaCO_2$) increases with decrease in minute ventilation. An increase in $PaCO_2$ decrease alveolar partial pressure of oxygen (PAO_2) because the carbon dioxide displace oxygen in the alveoli. Narcotics, anesthetics,and other medications that induce respiratory depression are the usual causes of primary hypoventilation (table 4) ***(Lakshimpathi, 2005).***

Hypoventilation is a rare cause of hypoxemia in ICU patients. Hypoventilation should be suspected as the cause of hypoxemia in patients with an elevated $PaCO_2$. Oversedation or hypercapnic respiratory failure are common causes of this

condition. Hypoventilation and a low FIO_2 may be separated from the other causes of hypoxemia in that they are the only ones associated with a normal alveolar-arterial (A-a) oxygen gradient. The alveolar-arterial (A-a) gradient is the difference between PAO_2 and PaO_2.

A-a gradient= (FIO_2 (PB - PH_2O) - $PaCO_2$/R) –PaO_2 Where FIO_2 means the fraction of inspired oxygen, PB means the barometric pressure, PH_2Omeans the partial pressure of water, $PaCO_2$ means the partial pressure of arterial carbondioxide and R means the respiratory quotient (which about 0.8) ***(Michael, 2001).***

Table (4): Mechanism, site and possible causes of hypoxia ***(Ceriana et al., 2006).***

			$P_{A\text{-}a,O_2}$	
Mechanism	Site	Possible causes	Room air	100% O_2
Hypoventilation	Extrapulmonary	Respiratory centre depression	Normal	Normal
Impaired alveolar oxygen diffusion	Pulmonary	Pulmonary fibrosis, pulmonary resection	Normal at rest, reduced under effort	Normal
Ventilation/perfusion mismatch	Pulmonary	Emphysema, pulmonary embolism	Increased	Normal
Shunt of systemic venous blood	Pulmonary, extrapulmonary (cardiac)	Pneumonia, cardiac septal defect	Increased	Increased
Inhalation of hypoxic gas mixture	Extrapulmonary	Toxic fume inhalation, high altitude	Normal	Normal
Abnormal desaturation of systemic venous blood	Extrapulmonary	Fever, low cardiac output	Normal	Normal

The only mechanism not responsive to supplemental oxygen is shunt. $P_{A\text{–}a,O_2}$: alveolar–arterial oxygen tension difference.

2. Impaired diffusion:

In the presence of diffusion defects, full equilibration of alveolar gas with pulmonary capillary blood is prevented; factors that can be an obstacle to oxygen diffusion are an increased distance between alveolus and erythrocyte, a decreased oxygen gradient for diffusion and a shortened transit