INTRODUCTION

The primary functions of the respiratory system are: *Ventilation*; the movement of air into and out of the lungs, *Gas exchange*; the transfer of oxygen into the blood and the removal of carbon dioxide. General anesthesia has a number of effects on both of these key functions. The passage of gas into the lungs may be impaired by obstruction of the airway; the drive to ventilation may be reduced or cease altogether; the distribution of gas within the lungs may change and the transfer of oxygen (and anesthetic gases) into the blood may be impaired. Most of these adverse effects can be seen during anesthesia and in many patients these extend into the post-operative period ***(Rachel & Daniel, 2010).***

All guidelines for monitoring respiratory function have in common the following protocols: a) Oxygenation must be monitored continuously by pulse oximetry when providing moderate sedation, deep sedation, or general anesthesia. b) Ventilation must be monitored continually (periodically) during moderate sedation and continuously (uninterrupted) during deep sedation and general anesthesia ***(Quoted from Guyton & Halln, 2006).***

Monitoring ventilation can be difficult. During mild and moderate sedation using nitrous oxide, deflation and subsequent inflation of the reservoir bag is useful in assessing ventilation. Observation of chest movement and auditory clues

are also helpful. An increase in sonorous breath sounds may be a sign of increasing airway obstruction. Nasal flaring, discordant chest wall motion, and retraction at the suprasternal area (tracheal tug) with respiratory effort are all physical signs of airway obstruction ***(Fu et al., 2004).***

The purest measure of adequate ventilation is by assessing carbon dioxide tension. Obviously, continuous sampling of arterial blood gases is impractical. However, the tension of carbon dioxide in expired gas, particularly at the end of a tidal expiration, closely approximates the tension in arterial blood. While normal $PaCO_2$ is ~40 mm Hg, so-called end-tidal CO_2 ($ETCO_2$) is ~35–38 mm Hg. This measurement is the basis of capnometry ***(Fu et al., 2004).***

Patients are at risk for several types of lung injury in the perioperative period. These injuries include atelectasis, pneumonia, pneumothorax, bronchopleural fistula, acute lung injury and acute respiratory distress syndrome (ALI/ARDS). Anesthetic management can cause, exacerbate or ameliorate most of these injuries. Lung-protective ventilation strategies using more physiologic tidal volumes and appropriate levels of positive end-expiratory pressure (PEEP) can decrease the extent of this injury ***(Slinger& Kilpatrick, 2012).***

In controlled ventilation using modern anesthetic monitoring devices, the peak inspiratory pressure (PIP) and plateau pressure (P_{plat}) can be measured by a monitor, and the

respiratory dynamics can be monitored and evaluated indirectly. When the amount of gas that expands the lungs is constant, the increase in PIP is considered to be the state when the airway resistance (R_{aw}) increases, when pulmonary compliance decreases, or when both occur simultaneously ***(Kim, 2009).***

Aim of the Work

The aim of essay is to assess the monitoring of different respiratory parameters during anesthesia ventilation and the effect of anesthesia on this parameters.

Chapter 1

PHYSIOLOGY OF RESPIRATION

INTRODUCTION

The main function of the lungs is to provide continuous gas exchange between inspired air and the blood in the pulmonary circulation, supplying oxygen and removing carbon dioxide, which is then cleared from the lungs by subsequent expiration. Survival is dependent upon this process being reliable, sustained and efficient, even when challenged by disease or an unfavorable environment. Evolutionary development has produced many complex mechanisms to achieve this, several of which are compromised by anesthesia. A good understanding of respiratory physiology is essential to ensure patient safety during anesthesia ***(Galvin et al., 2007).***

MECHANISM OF BREATHING

A pressure gradient is required to generate airflow. In spontaneous respiration, inspiratory flow is achieved by creating a sub-atmospheric pressure in the alveoli (of the order of-5cmH2O during quiet breathing) by increasing the volume of the thoracic cavity under the action of the inspiratory muscles. During expiration the intra-alveolar pressure becomes slightly higher than atmospheric pressure and gas flow to the mouth results ***(Galvin et al., 2007).***

The respiratory muscles

Inspiratory muscles

- Diaphragm-very powerful, has the ability to contact 10cm in forced inspiration
- External intercostals-pull the ribs up and forwards
- Accessory inspiratory muscles-scalene muscles (elevate first 2 ribs) and sternomastoids (raise the sternum)
- Muscles of neck and head (seen in small babies in respiratory distress)

Expiratory muscles

Expiration is usually passive and relies on the elastic recoil of the lungs and the chest wall. Under anesthesia or extreme exercise expiration may become active due to the activation of abdominal muscles. Muscles have their use in forced expiration.

- Abdominal wall muscles-rectus abdominus, internal and external oblique.
- Internal intercostal muscles-pull ribs down and inwards ***(Nadine & Sarah, 2009).***

Control of the Breathing

Respiratory center receive input from the chemoreceptors, cortex, hypothalamus, pharyngeal mechanoreceptors, vagus nerve

and other afferents. The respiratory centres are located in the pons and the medulla. These contain many different types of inspiratory and expiratory neurons that fire during the three phases of the respiratory cycle:

Inspiratory phase: A sudden onset is followed by a ramp increase in discharge to the inspiratory muscles and the dilator muscles of the pharynx.

Post inspiratory phase: A gradual decline of discharge to the inspiratory muscle leads to a gradual reduction in tone which modulates expiratory flow.

Expiratory phase: Both expiratory and inspiratory muscles are silent unless forced expiration or high minute ventilation (>40 min) is required. The medulla contains cells that discharge rhythmically, including the dorsal and ventral groups of respiratory neurons. The dorsal area lies close to the the tractus solitaries and mainly discharges on inspiration. The ventral area also contains some inspiratory neurons, i.e. the nucleus paraambigus (controls force of inspiration) (figure1).However, most cells are expiratory, including the expiratory Botzinger complex and the nucleus retroambigualis. The pons has a lessre role, adjusting the fine control of respiratory rhythm, including setting the volume at which inspiration is terminated. Upper motor neurons from the respiratory centers pass via the ventrolateral part of the cord to the anterior horn cells. Their effect is combined with voluntary

inputs that pass via the ventrolateral and dorsolateral cord, as well as involuntary inputs such as coughing and swallowing. Tension in the respiratory muscles is adjusted by the muscle spindles which are present in small number in the diaphragm ***(Gray, 2001).***

Chemoreceptors

Peripheral and central chemoreceptors provide inputs to the respiratory centers. The peripheral chemoreceptors lie in the carotid and aortic bodies. The carotid bodies are more important in stimulating ventilation, while the aortic bodies are also capable of responding to hypotension. The peripheral chemoreceptors in both sites respond to hypoxemia (unlike the central receptors), hypercapnia and hydrogen ion concentration. The carotid body receives a very high blood flow, enabling it to respond rapidly to changes in partial pressure. The bodies consist of structural type II cells and chemoreceptor type I or glomus cells, which contain many neurotransmitters. They appear to be inhibited by exogenous dopamine and α2 adrenergic agonist, but are stimulated by nicotine, atropine, doxapram and almitrine. The central chemoreceptors lie close to the origins of the glossopharyngeal and vagus nerves on the anterolateral surface of the medulla. They are within the blood brain barrier and bathed in CSF. This slows the response of the central chemoreceptors relative to peripheral sites. Carbon dioxide diffuses across the blood brain barrier into the CSF, which is less buffered than the plasma. This causes a fall in

CSF pH which stimulates the central chemoreceptors, if PaCO2 is maintained at abnormal values for several days. CSF pH is restored to normal by changes in CSF bicarbonate ***(Gray, 2001).***

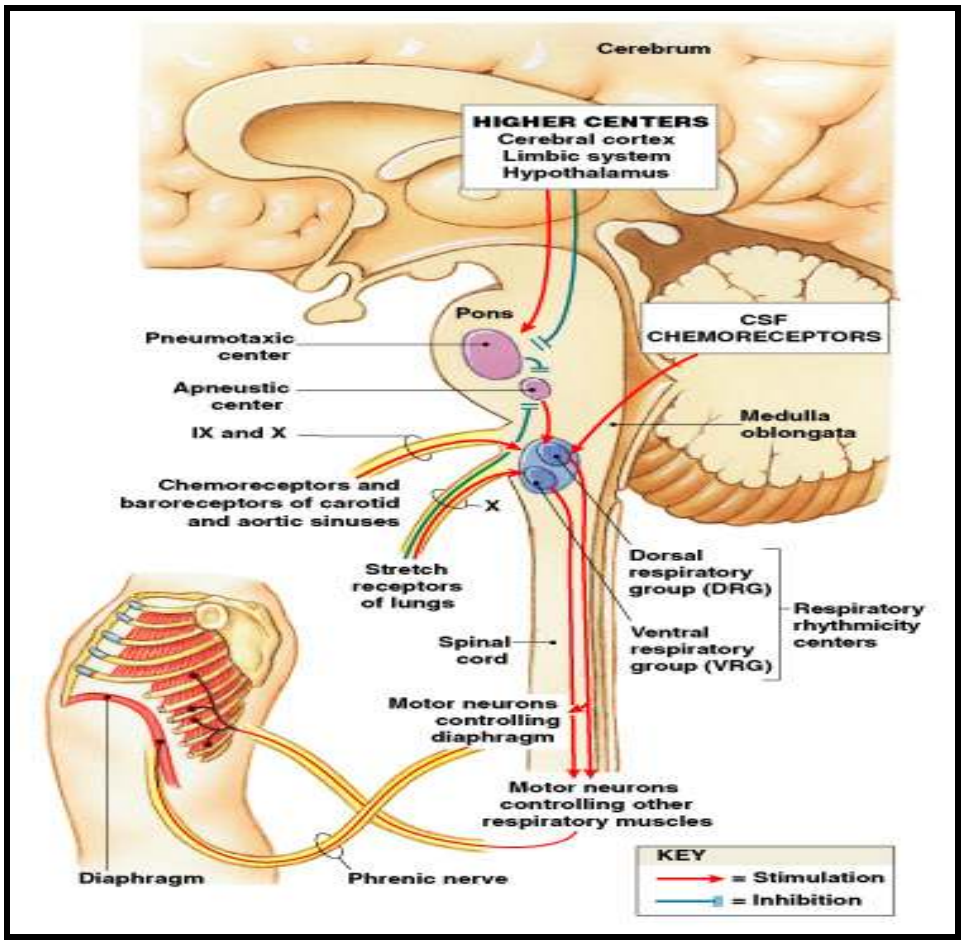

Figure (1): Control of breathing ***(Gray, 2001).***

LUNG VOLUMES

Normal requirements of the body can be easily met by normal tidal ventilation which is approximately 4-8 ml/kg.

Body has kept mechanism to provide extra ventilation in the form of inspiratory reserve volume and expiratory reserve volume whenever required (e.g., exercise). When an individual, after tidal expiration, takes full inspiratory breath followed by expiration to reserve volume, it is called as vital capacity breath and is 4–5 L in an average 70kg individual. There is always some amount of air remaining in the alveoli which prevents it from collapsing. The volume remaining in the lungs after vital capacity breath is called as residual volume. Residual volume with expiratory reserve volume is called as functional residual capacity (FRC). FRC is basically the amount of air in the lungs after a normal expiration. Gases remaining in the lungs at the end of expiration not only prevent alveolar collapse but also it continues to oxygenate the pulmonary blood flowing through the capillaries during this time period. Reported FRC values vary with various reports but on average it is between 2.8 and 3.1L in standing position. FRC varies with change of position, anesthesia and body weight. FRC is the reserve which prolongs non-hypoxic apnea time. The portion of the minute ventilation which reaches alveoli and takes part in the gas exchange is called as alveolar ventilation. Normal value of alveolar ventilation is approximately 5 L/min which is similar to the volume of blood flowing through the lung (cardiac output 5 L/min). This makes alveolar ventilation to perfusion ratio approximately one (figure2) ***(Apeksh & Amit, 2009).***

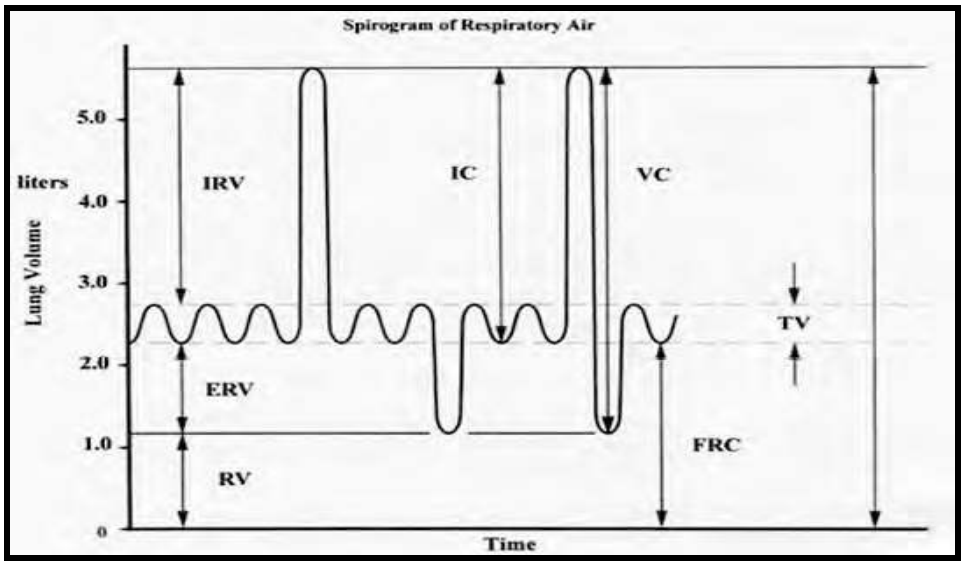

Figure (2): Basic spirometry trace of lung volumes and capacities the actual requirements of the body ***(Apeksh & Amit, 2009).***

Oxygenation

Tissue Oxygenation describes the process by which oxygen from the atmosphere is supplied to the tissues. The phases in this process are either convective or diffusive:

1. **The convective or "bulk flow" phases** are alveolar ventilation and transport in the blood from the pulmonary to the systemic microcirculation: these are energy requiring stages that rely on work performed by the respiratory and cardiac "pumps".

2. **The diffusive phases** are the movement of oxygen from alveolus to pulmonary capillary and from systemic capillary to cell: these stages are passive and depend on the gradient of oxygen partial pressures, the tissue capillary density

(which determines diffusion distance), and the ability of the cell to take up and use oxygen. (figure3) ***(Leach & Treacher, 2002).***

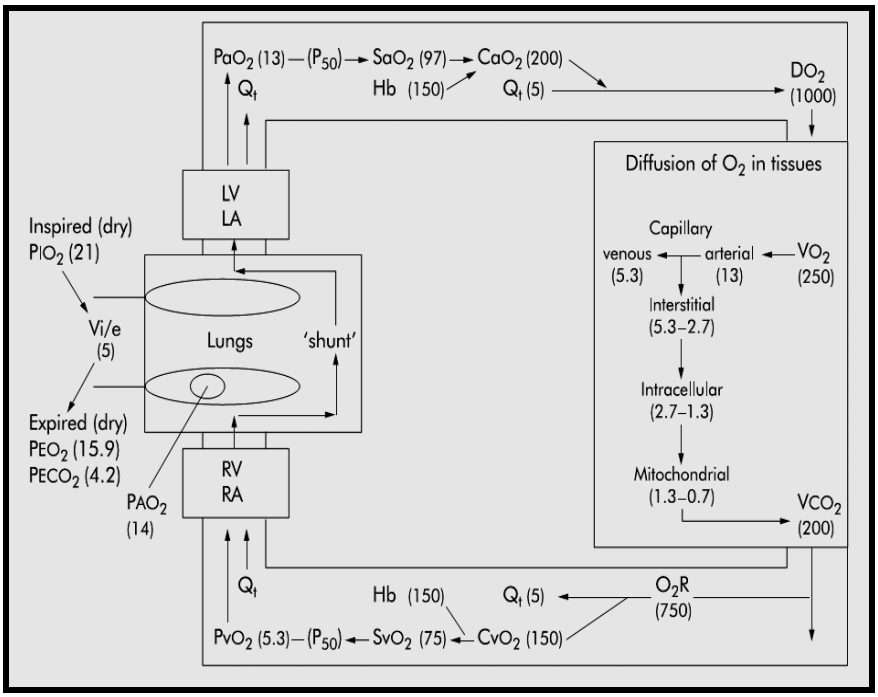

Figure (3): Oxygen transport from atmosphere to mitochondria, Values in parentheses for a normal 75kg individual (BSA 1.7m^2) breathing air (**FIO_2** 0.21) at standard atmospheric pressure (101 kPa). Partial pressures of O_2 and CO_2 (PO_2, PCO_2) in kPa; saturation in %; contents (CaO_2, CvO_2) in ml/l; Hb in g/l; blood/gas flows (Q_t, Vi/e) in l/min. P_{50} = position of oxygen hemoglobin dissociation curve; it is PO_2 at which 50% of hemoglobin is saturated (normally 3.5 kPa). DO_2 = oxygen delivery; VO_2 = oxygen consumption, VCO_2 = carbon dioxide production; PIO_2, PEO_2 = inspired and mixed expired PO_2; $PECO_2$ = mixed expired PCO_2; PAO_2 = alveolar PO_2 ***(Leach & Treacher, 2002).***

Oxygen uptake in the lungs

- **Inspired oxygen concentration and barometric pressure:**

However by the time the inspired air reaches the trachea it has been warmed and humidified by the upper respiratory tract. The humidity is formed by water vapour which is a gas, so exerts a pressure. At 37°C the water vapour pressure in the trachea is 6.3kPa. Taking the water vapour pressure into account, the PO2 in the trachea when breathing air is (101-6.3) x 21/100 = 19.9kPa. By the time the oxygen has reached the alveoli the PO2 has fallen to about 13.4kPa. This is because the PO2 of the gas in the alveoli (PAO2) is further reduced by dilution with carbon dioxide entering the alveoli from the pulmonary capillaries ***(Rob& Henry, 1999).***

- **Diffusion from alveoli to pulmonary capillaries:**

Gas diffusion from alveolus to capillary is driven by the gradient in the partial pressure of oxygen (PO_2) between alveolus (PAO_2) and the surrounding capillaries. It is proportional to the total area of the alveolar-capillary membrane available for diffusion in the lung and inversely proportional to the diffusion distance across the membrane ***(Schober & Schwarte, 2011).***

Blood returning to the heart from the tissues has a low PO_2 (4.3kPa) and travels to the lungs via the pulmonary arteries. The pulmonary arteries form pulmonary capillaries,

which surround alveoli. Oxygen diffuses (moves through the membrane separating the air and the blood) from the high partial pressure in the alveoli (13kPa) to the area of lower partial pressure - the blood in the pulmonary capillaries (4.3kPa) ***(Rob & Henry, 1999).***

- **Ventilation and perfusion**(V/Q)

An ideal lung would have a uniform distribution of V/Q with values (Expressed in L/m) close to 0.8. In reality this does not happen and a spectrum of V/Q exists from zero to infinity. In normal subjects, the main factor that affects V/Q is gravity resulting, in the upright posture, in a gradient from high values at the lung apices to low values at the lung bases ***(Christopher et al., 2008).***

Carrying capacity of blood

In blood, oxygen is mainly bound to hemoglobin and only a small amount is dissolved in plasma. One gram of hemoglobin carries about 1.34–1.39 mL of oxygen and the dissolved amount of oxygen is proportional to its partial pressure, with the solubility coefficient for oxygen in plasma (0.0031 when paO_2 is expressed in mmHg) being the coefficient of proportionality. These properties allow calculation of arterial blood oxygen content (CaO2):

$$C_aO_2 = S_aO_2 \cdot [Hb] \cdot 1.34 + 0.0031 \cdot p_aO_2$$

Since the quantity of dissolved oxygen is normally negligible, arterial oxygen saturation (SaO_2, expressed as a ratio of 1) and hemoglobin concentration ([Hb], in g/dL) are the two major determinants of arterial oxygen content. The relationship between oxygen saturation and paO_2 is described by the oxygen-hemoglobin-dissociation curve. Assuming physiologic values of [Hb] and SO_2, normal oxygen content of arterial blood is about 180–200 mL oxygen per L blood ***(Nichols & Nielsen, 2010).***

- **Oxyhemoglobin Dissociation Curve**

An oxyhaemoglobin dissociation curve (ODC) quantifies the most important function of red blood cells and that is the affinity for oxygen and its delivery to the tissues. Oxygen affinity for haemoglobin plays a critical role in the delivery of oxygen to the tissues and is changed by shifting to the left or right (figure4) ***(Richard et al., 2007).***