

ASSESSMENT
OF
CHESTY PATIENT

ESSAY

Submitted for Partial Fulfilment of
Master Degree in Anesthesia.

BY

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1987

***ACKNOWLEDGEMENT . .**

My Deepest thanks to God for his countless gifts..

I am heartily grateful to Professor Dr. NABIL ABDEL MOOTY Prof. of Anesthesiology, Ain Shams University, who planned, revised and completed this study. I am very grateful to him for generous help, he has given me much of his time and effort.

Many great thanks to Dr. MOHAMED SALLAM , for his kind guidance and continued supervision at all steps of work.

To all professors in the Faculty of Medicine, Ain Shams University, who all over the years taught me Anesthesiology, I should pay much gratitude and respect.

ESAM HAMZA **



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INTRODUCTION *

INTRODUCTION

Respiratory diseases in surgical patients present real hazards and enhance the risk of complications and fatalities. The specific chest problem should be identified, the extent of interference with ventilation or gas exchange assessed by function studies and attempts made to control the disease process or to correct the pathology.

Patients with chronic lung disease such as bronchiectasis, active tuberculosis and other conditions associated with increased secretions must be submitted to an "improvement regimen" postural drainage, aerosol therapy to liquify secretions and antibiotics are necessary.

Asthmatic patients should be assessed carefully and attempts to decrease bronchiolar constriction. It is equally important to decrease mucosal oedema. Patient should not be exposed to offending allergens. Bronchodilators and bronchorrhea therapy should be instituted. Concerning the latter, expectorants are helpful and potassium iodide is especially effective.

A frequent and serious complicating pulmonary problem encountered in surgical patients is that of emphysema. These patients have ineffective alveolar ventilation. It is usually due to both an interference with ventilation and secondarily to poor alveolar gas mixing with impaired alveolar-capillar diffusion. Consequent to this situation, carbon dioxide

retention occurs and respiratory acidosis ensues. In addition, the increased resistance to blood flow through the lungs produces cor-pulmonale and often right-sided heart failure.

The extent of the various functional deficiencies in emphysema must be assessed. The pulmonary reserve must be evaluated in terms of tests. Not only should the pathology be corrected, but a serious attempts made to improve function.

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RESPIRATORY PHYSIOLOGY\*

## RESPIRATORY PHYSIOLOGY

Understanding normal respiratory physiology is prerequisite to understanding disordered processes and mechanisms of gas exchange during anaesthesia and surgery. (Jonathan Benumof, 1984).

### NERVOUS CONTROL OF BREATHING

There are nerve cells in the brain stem which under normal circumstances generate rhythm of breathing (Karczewski, 1974). Classically various respiratory centers have been described.

#### 1. Inspiratory center.

This lies in the dorsal part of the medulla on each side and thought to generate the basic rhythm of respiration.

#### 2. Pneumotaxic center.

Situated in the pons, it transmits impulses to the inspiratory area limiting inspiration.

#### 3. Apneustic center.

This center lies in the lower pons and is normally over ridden by pneumotaxic center.

#### 4. Expiratory center.

This is situated ventrally on each side of the medulla and when stimulated causes expiratory muscle activity.

Expiration is normally a passive process during quite breathing but becomes active during exercise.

There is as much uncertainty concerning the origin of respiratory rhythmicity as there is concerning the respiratory center. It is generally thought that there is an inherent rhythmicity in group

of neurons in the brain stem which is modified by their afferent input. Even this idea has been questioned recently and it may be that there is no inherent rhythmicity, the respiratory center merely transducing the afferent input into a rhythmic output but in absence of sufficient efferent input remaining silent. (Sullivan et al., 1978).

**\* Other centers affecting respiratory center.**

Many of higher centers exert some influence on respiration for example; the acts of swallowing, speaking and coughing require a careful integration of the mechanical systems. These changes are due to impulses arising in the cerebral cortex. Similar impulses from cortical and thalamic areas influence the respiratory pattern during crying and laughing. (Wyllie-Davidson, 1984).

### **CHEMICAL CONTROL OF BREATHING**

Ventilation is normally controlled to:

1. Provide adequate oxygenation of blood passing through alveolar capillaries.
2. Excrete CO<sub>2</sub> so as to maintain brain PH within narrow limits.

These are achieved through central and peripheral respiratory chemoreceptors.

**1. Central chemoreceptor.**

In 1963, Mitchell and his colleagues located a bilateral superficial chemoreceptors on the ventro-lateral surface of the

medulla in the region of IX<sup>th</sup> and X<sup>th</sup> cranial nerves, extending towards the mid-line. These areas are termed medullary H<sup>+</sup> chemoreceptors (Mitchell et al., 1963).

They do not respond to an increase of hydrogen ion concentration (H<sup>+</sup>) of arterial blood as a result of addition of fixed acid because the hydrogen ion can not pass the blood brain barrier but carbon dioxide can diffuse through the blood brain barrier from blood to cerebro-spinal fluid. So, if the carbon dioxide tension in the arterial blood (PaCO<sub>2</sub>) is increased, it diffuses to cerebro-spinal fluid where it is converted to H<sup>+</sup> ions. The hydrogen ions stimulate the medullary H<sup>+</sup> chemoreceptors which stimulate the neurones of respiratory centers. On the other hand, hyperventilation lowers carbon dioxide tension in the alveolar air and consequently in the arterial blood. This leads to a decrease in carbon dioxide tension in the cerebro-spinal fluid, so hydrogen ion concentration is decreased and this leads to inhibition of respiratory centers.

## 2. Peripheral chemoreceptors.

They are present in carotid and aortic bodies. The carotid body lies at the bifurcation of common carotid artery and the aortic body in the aortic arch. They are sensitive to decreased oxygen tension of arterial blood and to a lesser extent to an increase in H<sup>+</sup> ion concentration and to increased carbon dioxide tension of arterial blood. The fall in arterial oxygen tension causes a rise in pulmonary ventilation.

### GAS EXCHANGE IN THE LUNG

In order to see whether the blood gas barrier in the lung is suitable for gas exchange or not we have to take a look on the various factors affecting gas transfer across tissue as mentioned in Ficks law which states that the rate of transfer of a gas through a sheet of tissue is proportional to the tissue area and the difference in gas partial pressure between the two sides, and inversely proportional to the tissue thickness. (West, 1979).

As regard the tissue area in the lung, it is very big measuring about 50 - 100 meters. the thickness of blood-gas barrier is less than half a micron.

The difference in gas partial pressure between the two sides is about 60 mmHg for O<sub>2</sub> to pass from alveoli to the venous blood and from the tissue to the arterial blood and from the venous blood to the alveoli. (Guyton, 1977).

So that the dimensions of the barrier are ideal for diffusion. Also, the rate of transfer is proportional to a diffusion constant which depends on the properties of the tissue and the particular gas.

There are the solubility of the gas in the tissue and the increase of the square root of the molecular weight of the gas itself. Because the molecular weight of O<sub>2</sub> and Co<sub>2</sub> are not very different from each other and Co<sub>2</sub> diffuses about 20 times more rapidly than O<sub>2</sub> through tissue sheets. (Nunn, 1977).

### GAS TRANSPORT TO THE PERIPHERY

#### \* Oxygen transport.

Both elements of blood \_plasma and red cells\_ shares in the carriage of oxygen.

In the plasma oxygen is carried in simple physical solution 0.3 ml/100 ml of blood, while within the red cells it is carried in chemical combination with haemoglobin.

#### ~ Oxygen flux:

It is the amount of oxygen (in chemical combination with haemoglobin) leaving the left ventricle per minute in arterial blood (Nunn,1969)

{oxygen flux = cardiac output  $\times$  arterial saturation  $\times$  haemoglobin concentration  $\times$  1.39 }

(where 1.39 is the volume of oxygen in ml which joins one gram of haemoglobin )

$$= 5000 \text{ ml/min} \times \frac{97}{100} \times \frac{5}{100} \text{ gm/100 ml} \times 1.39 \text{ ml/gm}$$

$$= 1000 \text{ ml/min.}$$

(Gregory,1974)

#### ~ Oxygen haemoglobin dissociation curve:

This curve relates the percentage saturation of the oxygen carrying power of haemoglobin to the  $P_{O_2}$ . This curve has a characteristic sigmoid shape because combination of the first haem in the haemoglobin molecule with oxygen increases the affinity of the second haem for oxygen, and so on , so that the affinity of haemoglobin for the fourth oxygen molecule is many times that for the first. At rest, 250 ml of oxygen are given from the blood to the tissue (Finch,1972)

~ Factors affecting the affinity of haemoglobin for oxygen:

The followings change the affinity of haemoglobin and cause shift of the oxygen haemoglobin dissociation curve :-

- pH
- PCO<sub>2</sub>
- Temperature

The affinity of haemoglobin for oxygen is decreased and the curve is **shifted** to the right if :

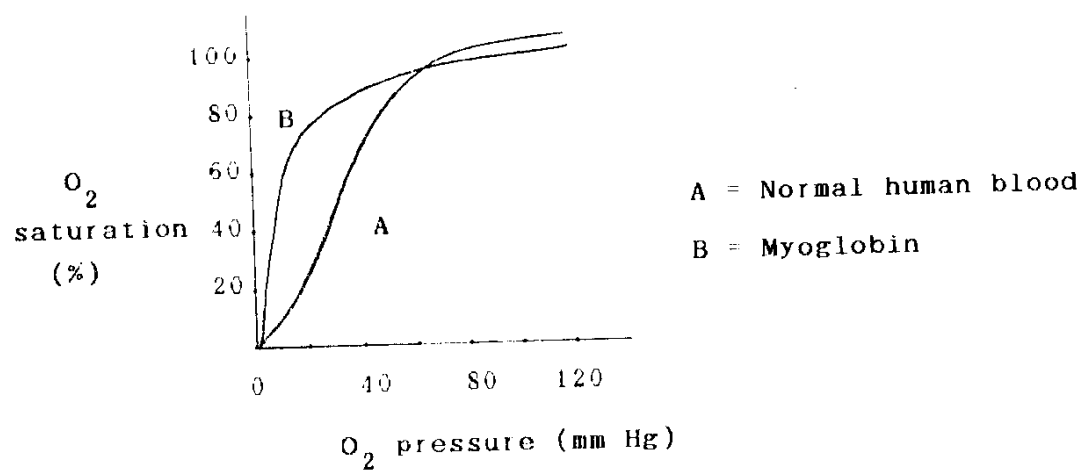
- the pH of the blood falls "Bohr effect"
- rise in PCO<sub>2</sub>
- rise in temperature

These factors are easily remembered by knowing that the exercising muscle is **acidic, hot, hypercarbic** and it benefits from increased unloading of oxygen from the blood.

2,3 diphosphoglycerate (2,3 DPG) formed within the red cells as a result of glycolysis. It binds to the beta chain of deoxygenated haemoglobin but not those of the oxyhaemoglobin. so, reducing their oxygen affinity (Arnone,1972)

~ Myoglobin:

It is an iron containing pigment found in skeletal muscles. Its concentration is highest in muscles specialized for sustained contractions. It resembles haemoglobin, but it binds only one oxygen molecule. Its dissociation curve is **rectangular hyperbola** because this curve lies to the left of haemoglobin. It takes up its oxygen from the blood. It releases its oxygen only at low P<sub>O2</sub> values. Myoglobin provides the skeletal muscles with oxygen when their blood supply is compressed during contractions (Stainsby and Barclay,1972).



Dissociation curves of Oxyhaemoglobin  
and Myoglobin at 38°C and pH 7.4

Because the curve of myoglobin is to  
the left of that of oxyhaemoglobin, it  
takes up O<sub>2</sub> from haemoglobin in the  
blood.