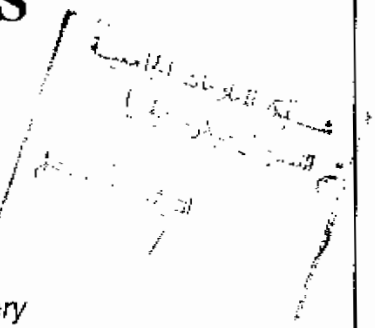
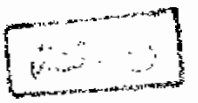
POST-OPERATIVE PULMONARY COMPLICATIONS



AN ESSAY

Submitted in Partial Fulfilment of
the Master Degree in General Surgery

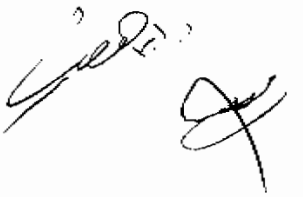


BY
ADEL FAROUK MOURSEY
(M.B., B.Ch.)

SUPERVISED BY

DR. RAFIK RAMSIS MORCOS
Assistant Professor of
General Surgery
Faculty of Medicine
Ain Shams University

Dr. MAHER MOUSA
Consultant of
Cardiothoracic Surgery
National Heart Institute



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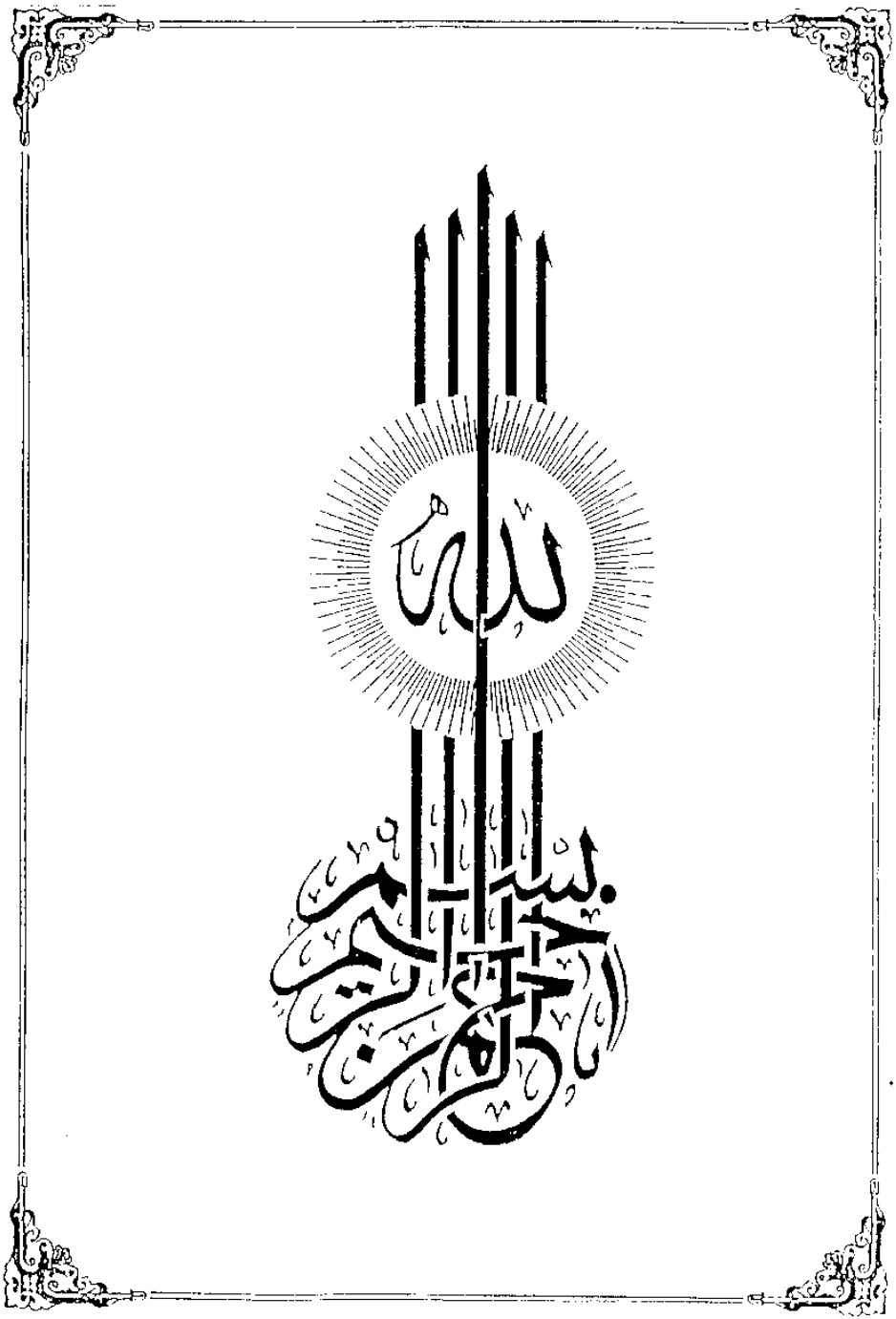

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Introduction

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Introduction

Post-operative pulmonary complications is the commonest cause of morbidity and mortality of surgical patients. Patients with preoperative pulmonary diseases are most susceptible to these complications.

Obesity, old age, smoking and diabetes mellitus are risk factors for their occurrence. So, reduction of weight, stopping smoking and eradication of chest infection and physiotherapy are very important to prevent or to reduce the incidence of post-operative pulmonary complications.

Post-operative pain and inability to cough are the most serious predisposing factors.

Lack of mobility and dehydration predisposes to venous stasis and thrombosis. Also, abdominal distention due to paralytic ileus is a predisposing factor.

Post-operative pulmonary complications can be largely prevented by adequate pre-operative and post-operative care.

The various post-operative pulmonary complications are atelectasis, Pneumonia, lung abscess, aspiration lung injury, pulmonary embolism, pneumothorax and ARDS.

It should be realised that one may merge into another, or neglect of one frequently leads to the production of more serious conditions.

At the conclusion of an operation the tracheobronchial tree should be aspirated if any secretions are suspected. Subsequently coughing and breathing exercises with regular rolling of the patient from side to side are desirable. Pain should be controlled by repeated small doses of analgesics of which pethidine is the most useful, as it does not depress respiration.

Thus, here in our essay, we concern with detection of aetiology, diagnosis, prevention and management of these complications.

The mechanical ventilation is used in treatment of respiratory failure and various ventilator modes with its benefits is very important to be understood.

Complications of mechanical ventilations include airway obstruction, barotrauma, ventilator-associated pneumonia, cardiac effects, renal effects and its effect on cerebral blood flow.

The shorter the time of mechanical ventilation, the easier the weaning from it.

CHAPTER II

Applied Anatomy And Physiology Of The Respiratory System

CHPATER II

Applied Anatomy And Physiology Of The Respiratory System

The trachea is lined by a pseudostratified columnar ciliated epithelium containing in addition numerous goblet cells. Argentaffin cells of the diffuse endocrine system are also present. Regulation of secretion of goblet cells is not known, but local "irritation", e.g. trauma and chemicals, leads to an increased discharge of mucus. Opening on to the surface are the ducts of submucosal compound mucous and seromucous glands. Vagal stimulation produces secretion from these glands, an action blocked by atropine. The wall of the trachea contains hoops of cartilage, incomplete pasteriorly; this allows considerable change in size of

the lumen. Thus, on inspiration there is an increase, and on expiration a decrease in size.

Some alteration in size of bronchi does occur with respiration. It follows therefore that a mass nearly occluding a bronchus will not prevent air entering the affected lung, but will effectively block its escape on expiration. The lung, therefore, becomes overdistended. Contraction of the bronchial muscle on expiration helps to reduce the anatomical dead-space to a minimum.

There are channels, described by Lambert, connecting bronchioles directly with alveoli. It therefore follows that the only complete separation of lung tissue occurs at the anatomically distinct septa, and it is for this reason that collapse due to bronchial obstruction is frequently lobar in distribution. Blockage of smaller bronchi does not always produce collapse.

The interstitium of the lung contains elastic fibres and reticulin (type-III collagen) and several types of cells-smooth muscle, mast cells, lymphocytes, pericytes, and most numerous of all interstitial cells, or alveolar histiocytes. These cells can become phagocytic and pass into the alveolar spaces to form alveolar macrophages. They can also function as fibroblasts to form collagen. An additional suggested function is contraction, because both actin and myosin are present their cytoplasm. Thus their contraction in the walls of

any hypoxic alveolus can decrease alveolar size and ventilation **(Walter and Israel, 1987).**

Anatomy Of The Pleura

Each lung is invested by and enclosed within the pleura. The pleura is divided into two parts: the parietal pleura, which retains its original relationship to the thoracic wall, and the visceral pleura, which is invaginated by the growing lung bud. The visceral pleura invests the lung intimately, cannot be dissected from it, and follows all the fissures and indentations of the lobes.

The parietal pleura lines the thoracic cavity and is divided into four areas: (1) the costal pleura, which lies against the ribs and intercostal muscles; (2) the diaphragmatic pleura, which covers the thoracic surface of the diaphragm; (3) the mediastinal pleura, which lies against the mediastinum; and (4) the cevical pleura, which covers the superior aspect of the pleural space in the plane of the first rib. These terms designate regional parts of the contiguous sac that extends beyond the lung and provides space for maximal expansion of the lung during forced ventilation. Beyond the lung margins, parts of the parietal pleura are in contact until they are separated by excursions of the lung with deep inspiration. Such potential spaces are prominent inferiorly, where the diaphragmatic and costal pleurae are in contact at the sides of the diaphragm. This space is called the costodiaphragmatic sinus.

A similar potential cleft behind the sternum, where the costal and mediastinal pleurae are in contact, is called the costomediastinal sinus. These clefts make it easy for the surgeon to enter the pleural cavity inadvertently when operating in these areas. Compared with the parietal pleura, the visceral pleura is insensitive to contact becasue it recieves no nerves of general sensation.

Diseases that affect the pleural space are essentially mechanical in pathophysiology and consist of spontaneous pneumothorax, spontaneous hemothorax, chylothorax, pleural effusions, empyema, bronchopleural fistula, fibrothorax, and iatrogenic space problems. Diseases that affect the pleura are essentially cellular in pathophysiology and include tuberculosis, pleural plaques, and pleural tumors **(De Meester and Lafontaine, 1990)**.

The Control Of Breathing

The rate, depth, and rhythm of breathing are determined by nerve cells in the reticular system. The respiratory centre has an intrinsic rhythmicity of its own, but is influenced by a variety of extramedullary function:

The cortex: Impulses from the higher centres are probably an important factor in the causation of the increased ventilation of voluntary exercise.

Proprioceptive impulses from the muscles of respiration. The tension developed in the inspiratory muscles is integrated with the movement produced. Any decrease in the volume of inspired air produced by an additional load, e.g. airways obstruction, therefore produces an abnormal sensation. This stimulates an increase in inspiratory effort to overcome the obstruction **(Campbell and Howell, 1962)**.

Blood PCo_2 and PH. The volume of respiration is finely regulated by arterial PCo_2, an increase stimulating and a decrease depressing it. A fall in blood PH also stimulates ventilation.

Blood Po_2. Hypoxia stimulates breathing via the aortic and carotid - body receptors. However, this response is less sensitive than that to changes in PCo_2.

Some patients with diseases of the lungs or of the central nervous system may chronically underventilate since their chemical control has **gas exchange in the lungs** become adjusted to a higher PCo_2 level **(Park, 1965)**.

The Exchange Of Gas Depends Upon Three Factors

I- Ventilation. This includes both the volume and the distribution of the inspired air which ventilates the alveoli. Ventilation involves two phases: inspiration and expiration.

Inspiration is carried out by the contraction of the diaphragm and intercostal muscles. The result is an increase in the negative intrathoracic pressure, which is about - 5cm H_2O (about -4 mmHg or -0.5 KPa). The inspiratory muscles are opposed by two forces.

A- The elastic force, which has two components:

1. The elastic properties of the lung substance.

2. The surface tension of the fluids lining the airways.

B- The non elastic or resistive load: This is chiefly due to the frictional resistance of the respiratory passages to airflow.

The compliance of the lung is a measure of its ability to increase in volume in response to increase in pressure within the alveoli. The force responsible for the increase in volume is the transmural pressure gradient.

$$\text{Compliance} = \frac{\text{change in lung volume}}{\text{change in transmural pressure gradient}}$$

It is normally 0.12 to 0.25 litres per cm H_2O. An alternative concept is elastance of the lung. It is reciprocal of the compliance **(D.C. White, 1983).**

Expiration is achieved by the elastic recoil of the lungs, during which the inspiratory muscles relax. During forced expiration