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ETIOLOGY AND MANAGEMENT OF SHOCK LUNG

ESSAY

SUBMITTED IN PARTIAL FULFILMENT FOR THE DEGREE OF MS. (GENERAL SURGERY)

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INTRODUCTION

INTRODUCTION.

During the last decade, considerable attension has been directed toward what appears to be a new and important medical disorder called shock lung syndrome.

Although patients with similar findings were occassionally observed during the first and second world wars, recently there has been a remarkable increase in the numbers of patients encountered with shock lung; this has been attributed to improvements in medical and surgical care that allow patients to survive long enough after a catastrophic illness, from which they previously would have died, to aquire the delayed respiratory complications.

It is clear that shock lung syndrome is an important disorder because of both the number of patients afficted and the associated high mortality rate. The mortality rate ranges between 50 % to 75 % (Montgomery et al, 1985).

There is no satisfactory definition of shock lung syndrome. Patients are said to have the disorder when they manifest certain characteristic clinical, radiological, and physiological abnormalities (Petty and Ashbaugh, 1971).

There is a tetrade of clinical features characterizes shock lung syndrome:

Progressive respiratory distress; decreased lung compliance and increased work of breathing; acute hypoxemia not releived by oxygen breathing; and diffuse radiographic opacification of the lung. Functional disturbances accompanying shock lung also include a decrease in functional residual capacity, an increase in the ratio of dead space ventilation to tidal volume, and increase in pulmonary artery pressure and pulmonary vascular resistance (Lawler, 1986).

The shock lung syndrome goes by many other names including Adult respiratory distress syndrome, post-traumatic pulmonary insufficiency, wet lung, adult hyaline membrane disease, hemorrhagic lung syndrome, congestive atelectasis, stiff lung, respirator lung, post-transfusion lung, or da Nang lung.

The mechanism of acute lung injury in shock lung remains unknown, but speculation, has focused on the following possibilities:

- 1) Hemodynamic factors, especially hypoperfusion of the lungs due to shock or hemorrhage.
- 2) Vasoactive factors, particularly platlet activation within the lungs or the release of inflamatory mediators within the pulmonary vessels.
- 3) Pulmonary ischemia due to venoarterial shunting.
- 4) Toxic factors initiated by the primary trauma.

- 5) Surfactant abnormalities .
- 6) Exogenous influences, including overtransfusion of crystalloids and colloids. and
- 7) Leukocyte aggregation in pulmonary capillaries due to inappropriate activation of complement (Divertie and petty, 1979).

Shock lung syndrome can result from a variety of causes e.g., shock, sepsis, post-traumatic, Oxygen toxicity, fat emboli, amniotic fluid emboli, aspiration of gastric contents, acute pancreatitis etc.

ANATOMY

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EMBRYOLOGY:

The principal respiratory organs develop as a medial longitudinal groove in the ventral wall of the pharynx (Arey's, 1974). This groove gradually changes into a tube separate from the pharynx and is lined by endoderm from which the respiratory epithelium develops, the cephalic part of the tube becomes the larynx, followed by the trachea, and from its caudal end, two lateral outgrowths arise forming the right and left lung buds. From these buds, the bronchi and lungs develop. Initially the right and la left lung buds are symmetrical. However, as development: proceed they appear asymmetrical, with three lobes on the right lung and two on the left. This is due to compression of the left lung by the heart. During the course of deved lopment, the lung migrate caudally so that at birth the bifurcation of the trachea lies at the level of the fourth thoracic vertebra. The lungs grow and project into the part of the coelom that ultimately forms the pleural cavities.

The pulmonary arteries arise from the sixth arch. The pulmonary artery is closely related to the main bronchus of its side. The artery provides an arterial partner for each new bronchial ramification. The veins develop and run in the connective tissue plains that separate adjacent

lobules, Interlobular veins combine to form intersegmental veins which, near the pulmonary hilus combine into superior and inferior pulmonary veins.

ANATOMY OF THE RESPIRATORY SYSTEM:

The respiratory system consists of the nose, nasal passages, nasopharynx, larynx, trachea, bronchi and lungs. The respiratory tree functions proximate to other structures i.e. ribs, intercostal muscles, pleura and mediastinum. A disease affecting any of these associated structures has a reflection on the respiratory system. One should be familiar with these anatomic relationships and the topographhic anatomy of the thorax.

TRACHEA:

The enterance of the trachea is gaurded by the larynx. It functions to prevent aspiration, also, the Larynx plays an important role in cough and as an organ of phonation. The mucous membrane of the larynx is lined with ciliated epithelial cells and few goblet cells. The epithelial surfaces in contact with food and pressure are covered by stratified squamous epithelium.

The trachea is a fibromuscular tube commencing at the lower border of the cricoid cartilage at the level of the sixth cervical vertebra. Its length is 10 to 12 cm and varying from 13 to 22 mm in width. It ends at the sternal

angle(lower border of the fourth thoracic vertebra) Sabiston(1986). However, last (1986) mentioned that this is
true only in the recumbent cadever. In the living erect
person the bifurcation descends two inches lower than this
in full inspiration. Last(1986) gave a length of 6 inches
(15 cm) for the trachea.

The patency of the trachea is maintained by about 20 C- shaped hyaline cartilages. The gapes posteriorly are closed by a sheet of unstipped muscle called the trachea-lis. The contraction of this muscle diminishes the calibre of the trachea, likewise it prevents overdistension of the tube when the pressure is raised e.g. in abdominal straining...ect. The dimensions of the trachea are constantly changing with movements of the head and neck.

The mucous membrane of the trachea rests on elastic lamina propria beneath which is the submucosa. The submucosa varies in thickness with the thickest portion on the muscular wall. In addition to blood vessels, nerves, and lymphatics, this layer contains the secretory portions of the mucous and serous glands. The trachea is lined with pseudostratified columnar ciliated epithelium containing qoblet cells.

The trachea is supplied by the inferior thyroid artery, its veins drain to the brachio-cephalic veins. Its lymphatics run to nodes along the trachea and to the postero-

inferior group of deep cervical nodes. The nerve supply is derived from the autonomic system, the vagus and the sympathetic. The vagus is sensory to the mucous membrane and motor to the trachealis muscle, also it is secreto-motor to the glands. The sympathetic fibres are vasoconstrictor.

BRONCHI:

At its termination, the trachea divides into the right and left principal bronchi. The right main bronchus is wider than the left and it deviates less from the axis of the trachea than does the left bronchus. This explains why foreign objects entering the trachea more often lodge in the right bronchus.

Within a primary lobe, the secondary bronchus soon divides into tertiary branches. The segment of a lobe, areated by a tertiary bronchus, is usually well delineated from adjoining segments by nearly complete planes of connective tissue. Knowledge of the segmental anatomy is of great practical importance in radiology, and pulmonary surgery.

The lung segments are ten in each lung. These segments are pyramidal in shape, their apices toward the hilum and the bases lying on the surface of the lung(Last, 1986).

The structure of the bronchial wall is very similar to that of the trachea except that cartilage rings are $\operatorname{repl}_{\pi}$

aced by irregular plates that completely surround the bronchus. These prevent collapse of the bronchi, but nevertheless, as in the trachea some alternation in size, with respiration, does occur. 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 (Walter and Israel,1984). The authors added that contraction of the bronchial muscles on expiration helps to reduce the anatomical dead space to a minimum. Sabiston(1986) mentioned that smaller bronchi are distinguished by rich venous plexuses between the muscular and cartilagenous fibrous layers. The effect of bronchial muscle contraction on the venous and lymphatic channels probably plays an important role in propelling the vascular fluids toward the hilus of the lung. Also, these rich venous networks are thought to be an important factor in warming of air entering the pulmonary parenchyma.

The mucous membrane of the bronchial mucosa is made up of an epithelium, a basement membrane, and richly vascular and fibrous tunica propria.

The bronchial epithelium, like that of the trachea, consists of pseudostratified ciliated and non ciliated cells, including goblet cells (Williams and Warwick, 1980). The peribronchial connective tissue extends from the pulmonary hilus to the primary bronchioles. The peribronchium

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is continuous with the connective tissue investment of the arterial partners of the bronchi and the connective tissue sheath of the large veins.

Sabiston(1986) mentioned that these connections form the bases for understanding the location and spread of certain types of edema and inflamation and the paths followed by air in and about the lung in interstitial emphysema. Interestingly, the peribronchium occupies a space in which subatmospheric pressure prevails. Von Hayek(1960) felt that this subatmospheric pressure plays an important role in the flow of venous blood, lymph, and alveolar fluid as well as in migration of inhaled particulate matter.

CELL OF THE RESPIRATORY EPITHELIUM: CILIATED CELLS:

Each cell approximately bears 270 cilia(Sabiston, 1986).

Although the cilia are born by separate cells, these many thousands of cilia beat in an organized, coordinated manner.

They beat in a whiplike fashion, the cycle of activity being divided into a rapid forward propulsive stroke and a slower rescovery stroke(Kilburn 1968, Krahl 1964, and Spack 1967). This propulsion is effective in moving a superimposed carpet of mucous along with a variable number of trapapped particles and cells upwards towards the larynx.

The rate at which particulate matter is propelled by