Summary

The mouth is made up of the vestibule and the mouth cavity, the former communicating with the latter through the aperture of the mouth.

The vestibule is formed by the lips and cheeks without and by the gums and teeth within. An important feature is the opening of the parotid duct on a small papilla opposite the 2nd upper molar tooth Normally the walls of the vestibule are kept together by the tone of the facial muscles; a characteristic feature of a facial nerve paralysis is that the cheek falls away from the teeth and gums, enabling food and drink to collect in, and dribble out of, the now patulous vestibule.

The function or respiration is to ensure the needs of the tissues for oxygen and for removal of CO_2. The Process of respiration can be divided into four major mechanistic events:

Pulmonary ventilation: Which means the inflow and outflow of air between the atmosphere and lung alveoli.

Diffusion: Diffusion of oxygen and carbon dioxide between the alveoli and the blood.

Pulmonary perfusion: The volume of blood Pumped by the right ventricle to the lungs is 5L/min.

Gas transport function of the blood: The transport of oxygen and carbon dioxide by blood is mainly in chemical combination. The Process of pulmonary ventilation is regulated by respiratory centers present in the brain stem according to cellular needs. (Zwart, et al. 1984).

Compliance refers to the ease with which the lungs and thoracic wall can be expanded. High compliance means that the lungs and thoracic wall expanded easily. Low compliance means that they resist expansion. Compliance is related to two principal factors: elasticity and surface tension. Presence of elastic fibers in lung tissue results in high compliance. If surface tension within lung tissue were high, the tissues would resist expansion, but surfactant lowers surface tension and thus

increases compliance. Compliance is a ratio between change in volume and change in distending pressure $\frac{\Delta V}{\Delta P}$ (volume change per unit pressure change) because a lung may be considered as an elastic sac, its volume depends on the pressure difference between its inside and outside surface, the transmural pressure. Transmural pressure = Distending pressure.
= pressure inside–pressure outside or =Alveolar pressure–pleural pressure
Compliance is a measure of expandability (Dispensability) and not elasticity. (Young, et al. 1970).

In normal range (expanding pressure about -2 to -10 Cm water) the lung is remarkably dispensable or compliant.

The compliance is 0.22 liters per Cm H_2O pressure.

Layers of respiratory membrane, the gas has to cross: A thin layer of fluid lining the alveoli, Alveolar epithelium. (type I and II cells), Basement membrane (alveolar & capillary endothelium), Interstitial space, and Capillary endothelium. (Iravani and Van, 1972).

The thickness of membrane is about 0.6 microns and its surface area is about 70 square meters in both lungs.

$$\mathbf{D \alpha \frac{pressure\ gradient \times Surface\ area \times Solubility}{thickness\ of\ membrane \times \sqrt{MW.}}}$$

Pulmonary circulation starts by the right ventricle and ends by the left atrium. Blood enters the lungs through two sources: The pulmonary artery bringing venous blood and the bronchial arteries carrying arterial blood. The pulmonary artery has the same cross-sectional area as the aorta, but it is more elastic and more distensable. Through the pulmonary artery venous blood of the right ventricle goes to the lung for oxygenation, it also carries nutrition to pulmonary tissues.

The lungs as a Blood Reservoir: Under different physiological and pathological conditions, the quantity of blood in the lungs can vary from

as little as 50% of normal up to as high as 200% of normal. Loss of blood from the systemic circulation by hemorrhage can be partly compensated for, by autonomic shift of blood from the lungs in to the systemic vessels. Failure of the left side of the heart, or increased resistance to blood flow through the mitral valve, causes blood to accumulate in the pulmonary circulation increasing the pulmonary blood volume as much as 100 %, also causes corresponding increase in the pulmonary pressures. On the other hand, exactly the opposite effects take place when the right side of the heart fails. (Vincent, et al. 1970).

In normal lung some of bronchial artery blood is collected by pulmonary veins after it has perfused the bronchi (depleted O_2),and small amount of coronary venous blood which drains directly into the cavity of left ventricle through the besian veins. The effect of addition of this poorly oxygenated blood is to depress the arterial Po_2.

The heart is irregularly conical in shape, and it is placed obliquely in the middle mediastinum. The right border is formed entirely by the right atrium, the left border partly by the auricular appendage of the left atrium but mainly by the left ventricle, and the inferior border chiefly by the right ventricle but also by the lower part of the right atrium and the apex of the left ventricle.

The right atrium receives the superior vena cava in its upper and posterior part, the inferior vena cava and coronary sinus in its lower part, and the anterior cardiac vein anteriorly. Running more or less vertically downwards between the venae cavae is a distinct ridge, the crista terminalis. This ridge separates the smooth-walled posterior part of the atrium, derived from the sinus venosus, from the rough-walled anterior portion which is prolonged into the auricular appendage and which is derived from the fetal atrium. (Mercat, et al. 1997).

The arterial supply **to the cardiac musculature is derived from the right and left coronary arteries.**

The right coronary artery arises from the anterior aortic sinus and passes forwards between the pulmonary trunk and the right atrium to descend in the right part of the atrio-ventricular groove. At the inferior border of the heart it continues along the atrio-ventricular groove to anastomose with the left coronary at the inferior interventricular groove. It gives off a marginal branch along the lower border of the heart and an interventricular branch which runs forward in the inferior interventricular groove to anastomose near the apex of the heart with the corresponding branch of the left coronary artery.

The nerve supply of the heart is derived from the vagus and the cervical and upper thoracic sympathetic ganglia by way of the superficial and deep cardiac plexuses.

Surface markings:

The outline of the heart can be represented on the surface by the irregular quadrangle bounded by the following four points:

The 2nd left costal cartilage 1.25 cm from the edge of the sternum, the 3rd right costal cartilage 1.25 cm from the sternal edge, the 6th right costal cartilage 1.25 cm from the sternum and the 5th left intercostal space 9cm from the mid-line. The left border of the heart and is formed almost entirely by the left ventricle, the lower border corresponds to the right ventricle and the apical part of the left ventricle; the fight border is formed by the right atrium.

The cardiac output (co) is quantity of blood pumped by pumped by each ventricle into the circulation (greater or lesser) per minute.

The co is responsible for the transport of substances to and from the tissues. (Bulter, et al. 1975).

At complete physical rest the cardiac output is minimal, but the healthy body responds to any physical and emotional demands by increasing the cardiac output, raising the arterial blood pressure and augmenting the circulation of the blood.

Variations in cardiac output can be produced by changes in heart rate or stroke volume. The heart rate is controlled by the cardiac Innervation sympathetic stimulation increasing the rate and parasympathetic stimulation decreasing it. The stroke volume is affected by. Neural Inputs:

The greater the heart is filled during diastole, the greater will be the quantity of blood pumped into the aorta, As a result, the heart pumps all the blood that comes to it without allowing excessive accumulation of blood in the veins. The heart can pump either a small amount of blood or a large amount depending on the amount that flows into from the veins. Local Tissue Metabolism: Each tissue in the body controls its own blood flow by dilating or constricting its local blood vessels. If some tissues need extra blood flow and their local blood vessels dilate, the venous return increases automatically, and the cardiac output increases by an equivalent amount. If all the tissues throughout the body require increased blood flow at the same time, the venous return becomes very great and the cardiac output increases accordingly, Therefore, we can state that, under normal conditions, venous return and cardiac output are determined by the degree of dilatation of local blood vessels in the tissues through out the body i.e. total peripheral resistance. This is as long as the arterial pressure is maintained constant by its regulatory mechanisms, otherwise. the produced vasodilatation reduces the arterial pressure, mean systemic filling pressure and cardiac output. (Johnson and hedly, 1972).

Age: The blood pressure rises with age. At birth the ABP is very low 50/30 mm. Hg. Thereafter, it rapidly rises during the first few weeks to 90/60 mm. Hg. The pressure increases gradually with growth until the adult level is reached. It often rises with advancing age ; the average at 80 years is 170/90 mm. Hg. A considerable number of individuals maintain the healthy elastic stage of their arteries, and do not show a rise in their ABP with advancing age.

It has been given that each tissues can control its own blood flow by simply dilating or constricting its local arterioles for this mechanism to occur it is necessary that the arterial pressure remains constant or nearly constant. (Vanlith, et al. 1967).

The cardiovascular response to peripheral chemoreceptor stimulation by hypoxia and hypercapnia is vasoconstriction and bradycardia, therefore the peripheral and central actions tend to cancel each other. However hypoxia produces also increased catecholamine secretion from the adrenal medulla and increased respiratory rate, both of which produce tachycardia and increase the cardiac output.

- The total recoil tendency of the lungs can be measured by the amount or negative pressure in the pleural space required to prevent collapse of the lungs. It is normally about -3 mmHg, at the end of normal expiration -3 mmHg, at the end or normal inspiration -6 mmHg and at the end of forced inspiration -12 mmHg.

Trans-airway pressure gradient = (10-20 cm H_2O): It is a pressure difference between the atmosphere and alveoli. This is the pressure gradient that will result in movement of gas into or out of the pulmonary system it is also called driving pressure or inflation pressure. The expiratory driving pressure is the result of the elastic recoil of the lung tending to return the lung to its resting state producing a transairway pressure the rate of expiratory flow will be affected by airway resistance

lung compliance and chest wall compliance. Elastic forces create an increased pressure within alveoli and alveolar pressures become positive during expiration. At end of expiration there is a period of absent flow (expiratory pause). (Mans, 1969).

Transpulmonary pressure gradient: It is the pressure difference between the atmospheric pressure and intro thoracic pressure Trans pulmonary pressure gradient = 10 cm H_2O at the end of inspiration 5 cm at the end of expiration and 3 cm H_2O at the residual volume.

Alveolar distending pressure is the pressure difference between the alveoli and intra pleural space. Alveolar distending pressure (transmural pressures gradient) = 14.4 cm. H_2O Alveolar pressure are affected by the changing pleural pressures and become subatmospheric. This creates trans airway pressure gradient which leads to gas flow from upper airway to alveoli. By the end of inspiration the airway and alveolar pressure are equal and so transpulmonary pressure equals zero. The primary factor determining the flow of gas from upper airway to the alveoli is the airway resistance.

Positive pressure ventilators apply a positive pressure to the airway inlet. The intrapleural pressure becomes less negative and even positive. Alveolar pressures are initially atmospheric, a positive transpulmonary pressure gradient is created and inflation of the lungs occur.

Continuous mandatory ventilation "CMV" previously termed intermittent positive pressure ventilation (IPPV), controlled ventilation is defined as the patient taking no role in the ventilatory cycle the machine initiates inspiration, determine rate of ventilatoion and tidal volume.

Augmented ventilation is defined as both the patient and machine being involved. Patients maintained on augmented mode have a better survival rate than those maintained on control mode because patients who are maintained on augmented ventilation have reasonable cardio-vascular

reserves while most patients requiring CMV are severely suffering from unstable cardio-vascular system. (Takishima, et al. 1972).

This mode of mechanical ventilation allows the patient to breathe spontaneously through the ventilator circuit, it is used as a weaning technique, and as a maintenance mode IMV is more efficient, and safer, the ventilator can be adjusted to the patient without any sedation or even muscle relaxants, other advantages of IMV are the reduced risk of incidents caused by disconnection and the fact that the combination of ventilation and spontaneous breathing improves auto-regulation of the acid-base balance. IMV trains the inspiratory muscles and decrease severity of disorders of breathing co-ordination, IMV reduces the mean intrathoracic pressure that leads to positive effect on circulatory system.

Three forms of HFV are available. High–frequency positive–pressure ventilation involves delivering small "conventional" tidal volume at a rate of 60 – 120 breaths/min. High–frequency jet ventilation (HFJV) utilizes a small cannula at or in the airway through which gas is injected 80-300 times /min; gas entrainment (Bernoulli effect) may augment tidal volume. (Springer and Stevens, 1979).

Expiration retard: Aims at slowing down exhalation, This tends to hold collapsing alveoli and airways open for longer, In most cases of severe obstruction, expiratory flow will be markedly improved when retard is applied, it can be useful in asthma.

As originally formulated by Starling, the major determinants of filtration include the net hydrostatic pressure, the difference between the hydrostatic pressure of the intravascular and surrounding interstitial compartments; the net protein osmotic pressure, the difference between the oncotic pressure of the intravascular and interstitial compartments; and the permeability of those small vessels in the lung called the microcirculation, through which filtration occurs. The term permeability

describes not only the leakiness of the microcirculation but also the total vascular surface area available for filtration. in contrast to the multiple factors that govern filtration, there is a single major pathway for clearance of liquid from the lung, lymphatic removal. Abrief discussion of each of theses factors is necessary to clarify peeps diverse effects on pulmonary edema.

Positive end-expiratory pressure (PEEP) maintains airway pressure above atmospheric at the end of expiration. Application of PEEP to the respiratory system has multiple organ effects. the magnitude of which is determined and the state of the airway and lungs. If the lung complaince is transmitted to other intrathoracic structures and the cardiovascular consequences are minimized.

The decrease in cardiac output produced by PEEP is due to interference with venous return and a leftward displacement of the cardiac ventricular septum that restricts filling of the left ventricle. It is conceivable that improvements in PO_2 produced by PEEP could be offset by decreases in cardiac output.