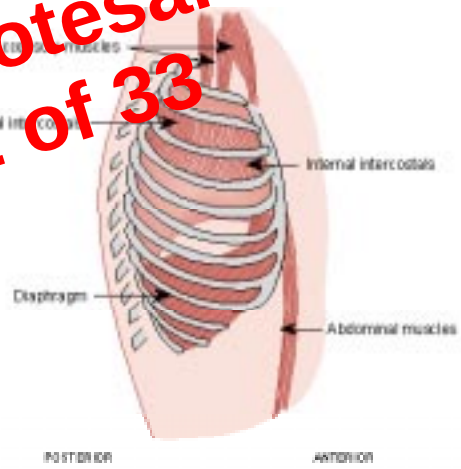
1. MECHANICS OF BREATHING:

INSPIRATION: Inspiration is the active part of the breathing process, which is initiated by the respiratory control centre in medulla oblongata (Brain stem). Activation of medulla causes a contraction of the diaphragm and intercostal muscles leading to an expansion of thoracic cavity and a decrease in the pleural space pressure. The diaphragm is a dome-shaped structure that separates the thoracic and abdominal cavities and is the most important muscle of inspiration. When it contracts, it moves downward and because it is attached to the lower ribs it also rotates the ribs toward the horizontal plane, and thereby further expands the chest cavity. In normal quite breathing the diaphragm moves downward about 1 cm but on forced inspiration/expiration total movement could be up to 10 cm. When it is paralysed it moves to the opposite direction (upwards) with inspiration, *paradoxical movement*. The external intercostal muscles connect adjacent ribs. When they contract the ribs are pulled upward and forward causing further increase in the volume of the thoracic cavity. As a result fresh air flows along the branching airways into the alveoli until *the alveolar pressure equals to the pressure at the airway opening.*



EXPIRATION: Expiration is a passive event due to elastic recoil of the lungs. However, when a great deal of air has to be removed quickly, as in exercise, or when the airways narrow excessively during expiration, as in asthma, the internal intercostal muscles and the anterior abdominal muscles contract and accelerate expiration by raising pleural pressure.

COUPLING OF THE LUNGS AND THE CHEST WALL: The lungs are not directly attached to the chest wall but they change their volume and shape according to the changes in shape and volume of the thoracic cavity. Pleura covering the surfaces of the lungs (visceral) or the thoracic cavity (parietal) together with a thin (20 µm) layer of liquid between them create a liquid coupling.

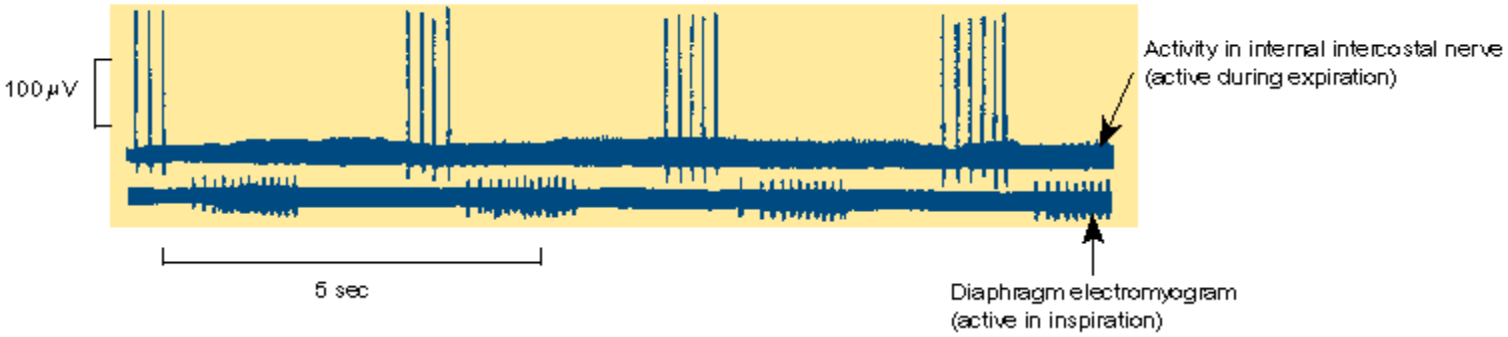
cross communication between them. As a consequence they behave in synchrony and the respiratory movements are symmetric.

2.Apneustic Centre: It is located in the lower pons. Exact role of this centre in the normal breathing is not known. Lesions covering this area in the pons cause a pathologic respiratory rhythm with increased apnoea frequency. What is known is nerve impulses from the apneustic centre stimulate the inspiratory centre and without constant influence of this centre respiration becomes shallow and irregular.

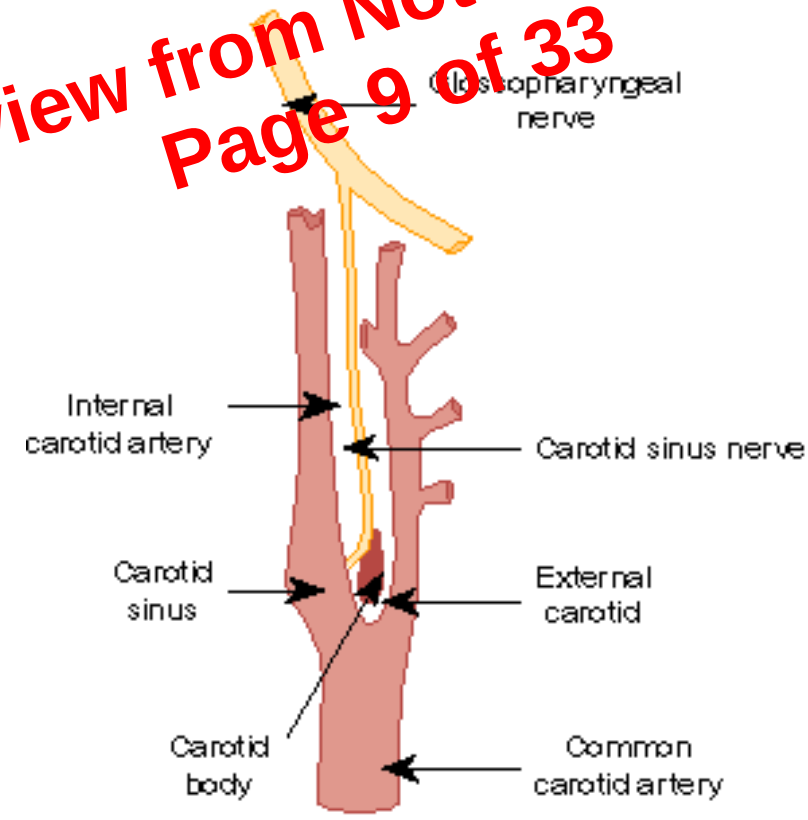
3.Pneumotaxic centre: It is located in the upper pons. This centre is a group of neurones that have an inhibitory effect on the both inspiratory and apneustic centres. It is probably responsible for the termination of inspiration by inhibiting the activity of the dorsal medullar neurones. It primarily regulates the volume and secondarily the rate of the respiration. Because in the lesions of this area normal respiration is protected it is generally believed that upper pons is responsible for the fine-tuning of the respiratory rhythm. Hypoactivation of this centre causes prolonged deep inspirations and brief, limited expirations by allowing the inspiration centre remain active longer than normal. Hyperactivation of this centre on the other hand results in shallow inspirations. The apneustic and pneumotaxic centres function in co-ordination in order to provide a rhythmic respiratory cycle: Activation of the inspiratory centre stimulates the muscles of inspiration and also the pneumotaxic centre. Then the pneumotaxic centre inhibits both the apneustic and the inspiratory centres resulting in initiation of expiration. Spontaneous activity of the neurones in the inspiratory centre starts another similar cycle again.

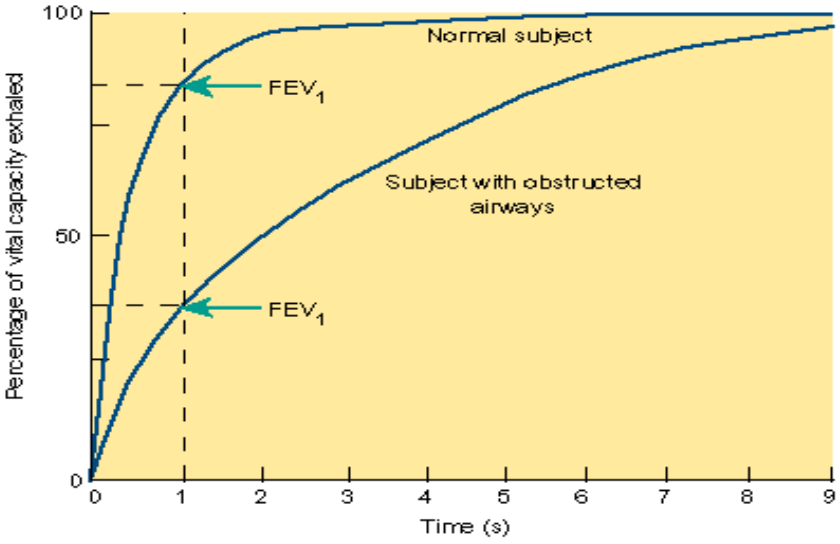
Breathing in some extent is also controlled consciously from higher brain centres (e.g. cerebral cortex). This control is required when we talk, cough and vomit. It is also possible voluntarily change the rate of the breathing. Hyperventilation can decrease blood partial carbon dioxide pressure (PCO_2) due to loss of CO_2 resulting in peripheral vasodilatation and decrease in blood pressure. One can also stop breathing voluntarily. That results in an increase in arterial partial oxygen pressure (PO_2), which produces an urge to breathe. When eventually PCO_2 reaches the high enough level it overrides the conscious influences from the cortex and stimulates the inspiratory system. If one holds his breath long enough to decrease PO_2 to a very low level one may loose his consciousness. In an unconscious person automatic control of the respiration takes over and the normal breathing resumes.

Other parts of the brain (limbic system, hypothalamus) can also alter the breathing pattern e.g. affective states, strong emotions such as rage and fear. In addition, stimulation of touch, thermal and pain receptors can also stimulate the respiratory system.



RESPIRATORY MUSCLES: Diaphragm, intercostal muscles and the other accessory respiratory muscles work in co-ordination for normal breathing under central controller. There is evidence suggesting that in premature new-born babies this co-ordination is not mature enough and this could be responsible for the sudden infant death syndrome.





Forced Expiratory Flow (FEF_{25-75}): This measurement represents the expiratory flow rate over the middle half of the FVC (between 25 – 75 %). It is obtained by identifying the 25 % and 75 % volume points of FVC, measuring the time between these points and calculating the flow rate.

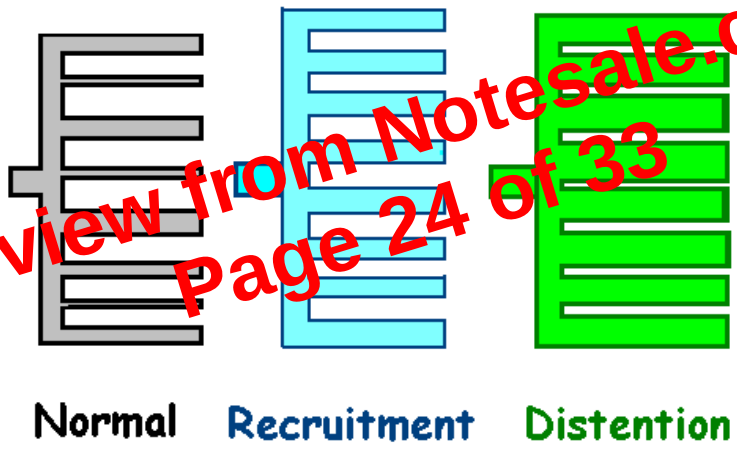
Interpretation of tests of forced expiration: On the basis of the knowledge obtained from these functional tests, lung diseases can be classified as ***restrictive*** or ***obstructive***. In restrictive lung diseases (such as pulmonary fibrosis), the vital capacity is reduced to below normal levels. However, the rate at which the vital capacity is forcefully exhaled is normal. In obstructive lung disease (such as asthma, emphysema, bronchitis) the vital capacity is normal because lung tissue is not damage and its compliance is unchanged. In asthma the small airways (bronchioles) constrict, bronchoconstriction increases the resistance to airflow. Although the vital capacity is normal, the increased airway resistance makes expiration more difficult and takes longer time. Obstructive disorders are therefore diagnosed by tests that measure the rate of forced expiration, such as the FEV_1 and FEF_{25-75}**.** A significant decrease in these values suggests an obstructive lung disease.

5. DIFFUSION. *How do gasses get across the blood-gas barrier?*

BLOOD-GAS EXCHANGE: Oxygen and carbon dioxide move between air and blood by simple diffusion: from an area of high to low partial pressure, as simple as water runs downhill. It is a passive process which means requires no energy. ***Fick's law of diffusion*** determines the amount of gas moves across the tissue is proportional to the area of the tissue but inversely proportional to its thickness. Because the blood-gas barrier in the lung is extremely thin and has a very large area (50-100 m^2), it is well suited to its function.

whole of the cardiac output at all times. Keeping the pulmonary pressure as low as possible allows the right heart answer this demand with a minimum work.

3. Unlike the systemic capillaries, which are organised as tubular network with some interconnections, the pulmonary capillaries mesh together in the alveolar wall so the blood flows as a thin sheet (capillary bed).
4. Another unique property of the pulmonary circulation is its ability to decrease resistance as cardiac output increases. Two mechanisms are responsible for this function.
    1. **Capillary recruitment:** opening of initially closed capillaries when cardiac output increases.
    2. **Capillary distension**: The decrease in pulmonary pressure with increased cardiac output has several beneficial effects: It (1) minimise the load on the right heart, (2) prevents pulmonary oedema, (3) maintains the adequate flow rate of the blood in the capillary and (4) increases the capillary surface area.



7. GAS TRANSPORT TO THE PERIPHERY *(How do gases move to the peripheral tissues?)*

OXYGEN:

Oxygen is carried in the blood in two forms, dissolved and combined with haemoglobin (Hb).

Dissolved Oxygen: The amount of oxygen dissolved in the blood is proportional to its partial pressure (Henry's Law). 100 ml of arterial blood with normal oxygen partial pressure (100 mm Hg) contains 0.3 ml oxygen. By this way amount of oxygen delivered to the tissues is only about 90 ml/min. Taking