EVOLUTIONARY DEVELOPMENT AND ANATOMY OF THE LUNGS

Early fishes probably swallowed air at the surface of the water which was used to fill a blind pouch on the ventral surface of their oesophagus. This served as a swim bladder enabling fish to float without the need to swim to maintain their position (Fig. 1). Later this pouch evolved to become the lungs which enabled primitive amphibians to survive out of water.

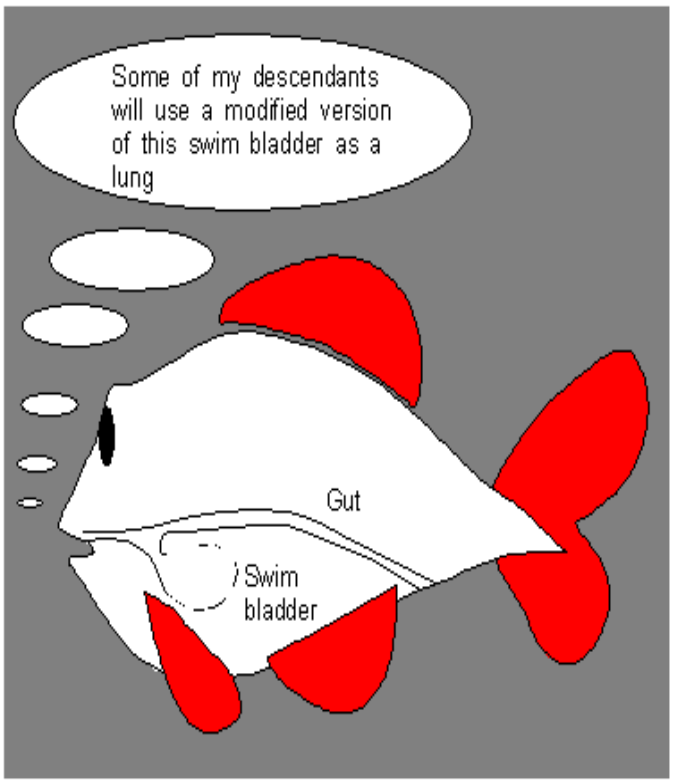
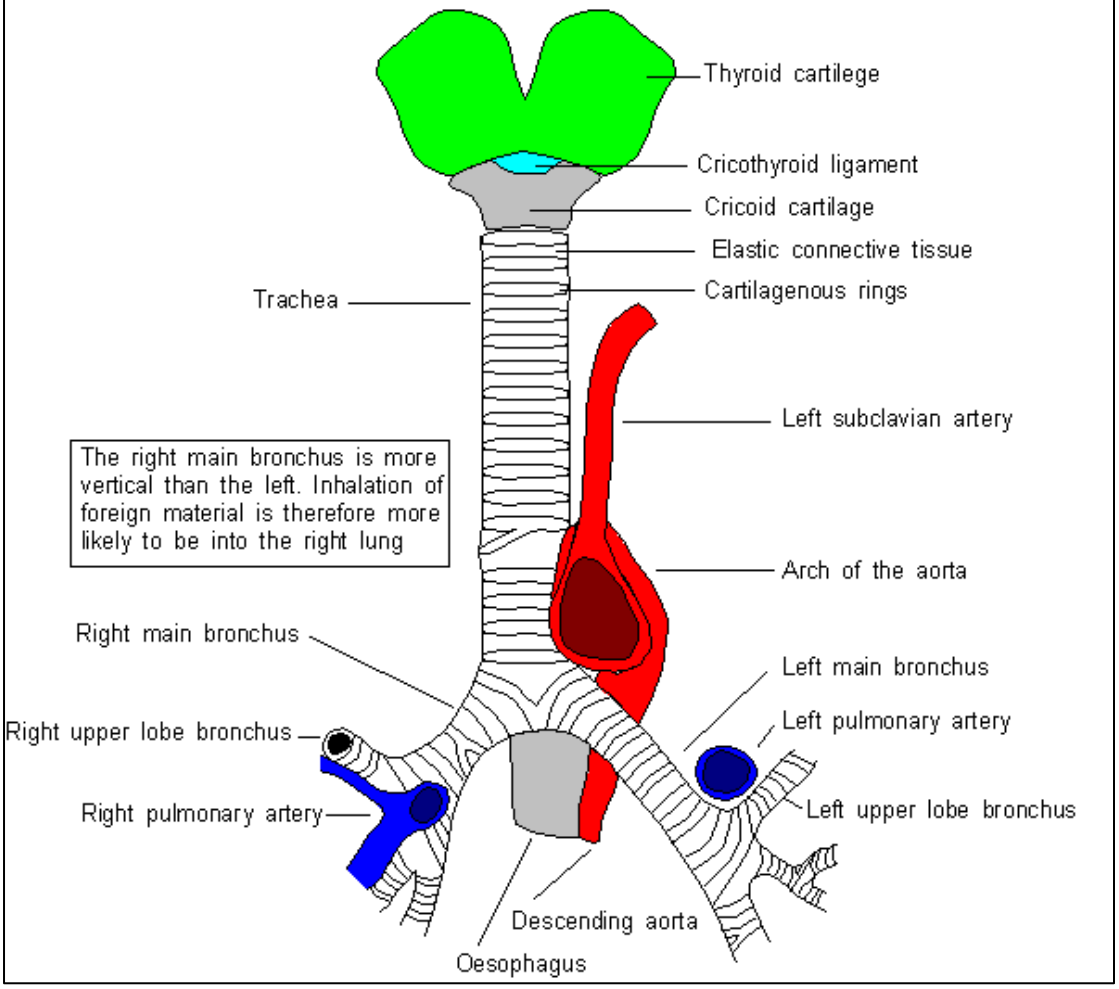


Figure 1. The swim bladder of fish. The amount of air in the bladder determines bouyancy.

In human embryos the lung buds develop from the gut tube and press out laterally into the pulmonary balloons the lining of which envelopes to form two layers, the inner (visceral) pleura and the outer (parietal) pleura which later allow lung expansion and contraction by sliding over each other.

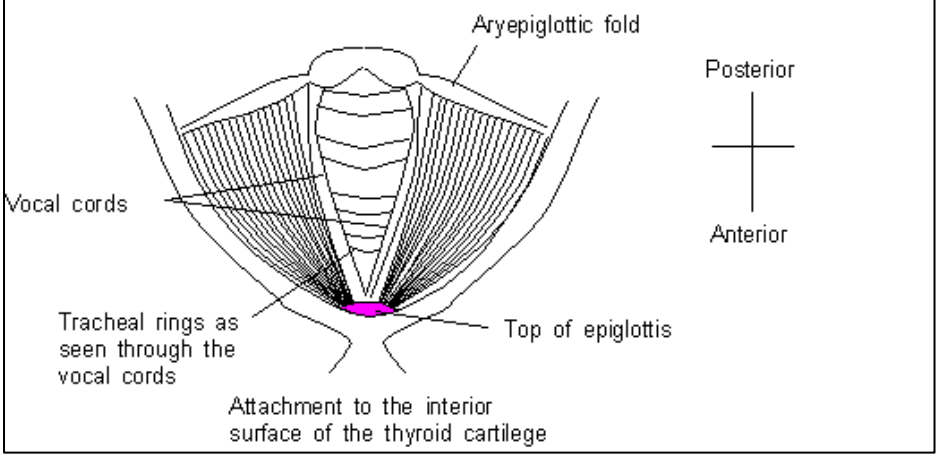
The lungs remain connected to the initial gut tube by the trachea. The trachea is supported by rings of cartilage (to prevent it collapsing) connected together by elastic connective tissue (which allows flexibility as the neck is turned or flexed) (Fig. 2).

Figure 2. The structures around the trachea

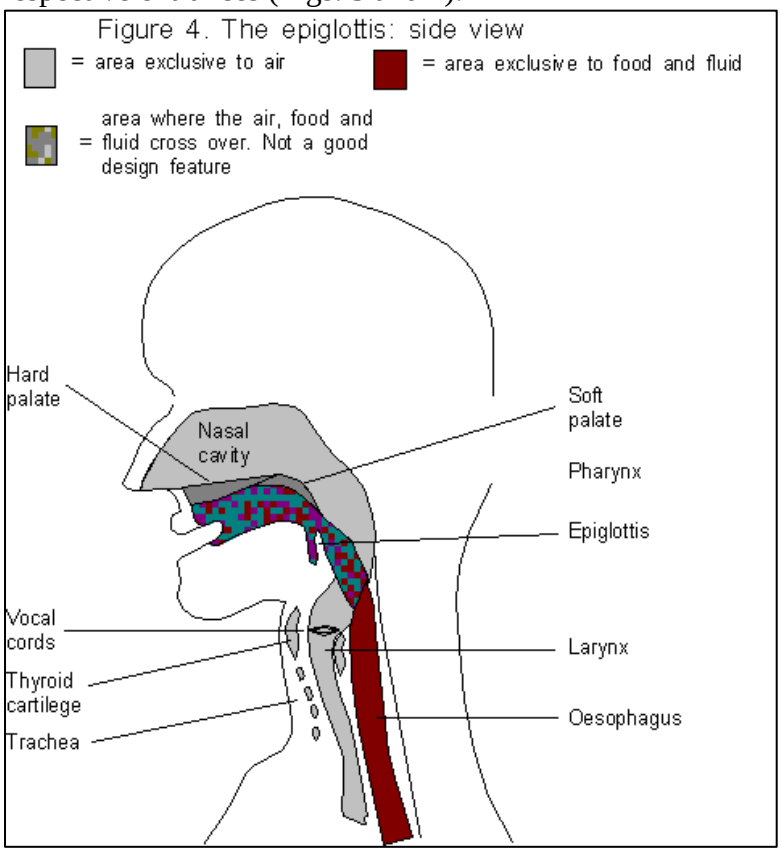


The larynx, which was initially a valve that protected the primitive lungs from dehydration is the dividing line between the upper and lower airways. In the larynx there are two inner folds, the vocal cords (Fig. 3) which vibrate to give speech.

Figure 3. The vocal cords and the larynx as seen when looking down the throat with a mirror at the back of the tongue



The epiglottis is a cartilaginous valve-like flap which guides air and food to the respective entrances (Figs. 3 and 4).



The anatomy of the ribs is shown in Figure 5, the chest wall and lungs in Figure 6 and Figure 7 shows the anatomy of gas exchange.

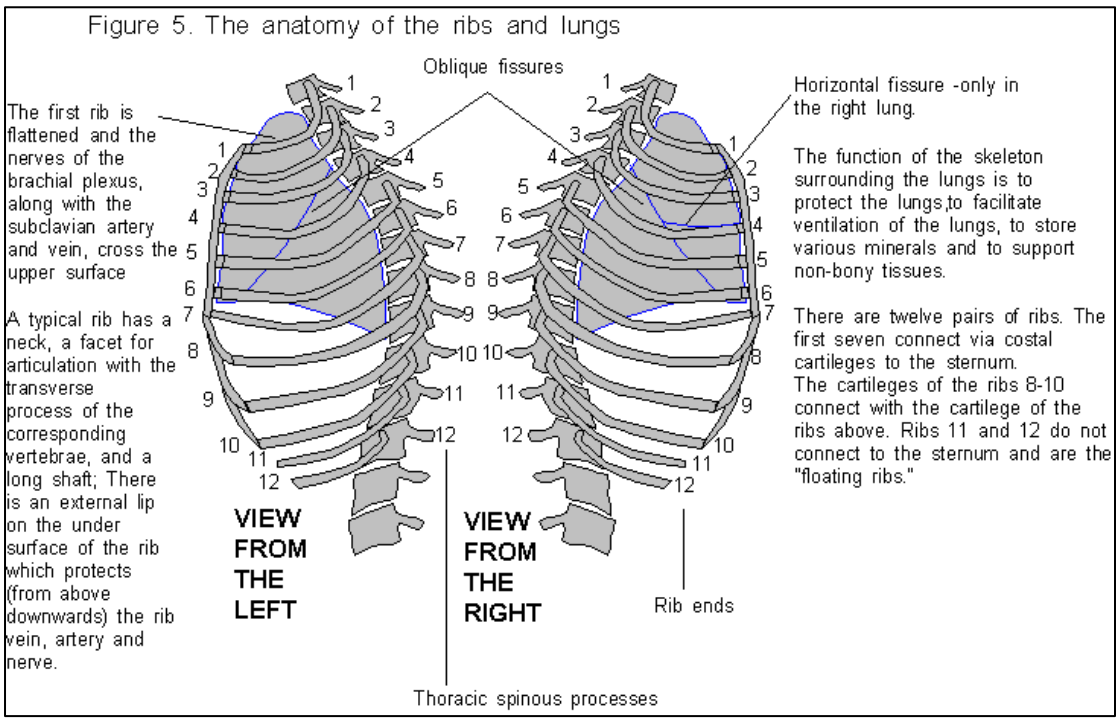
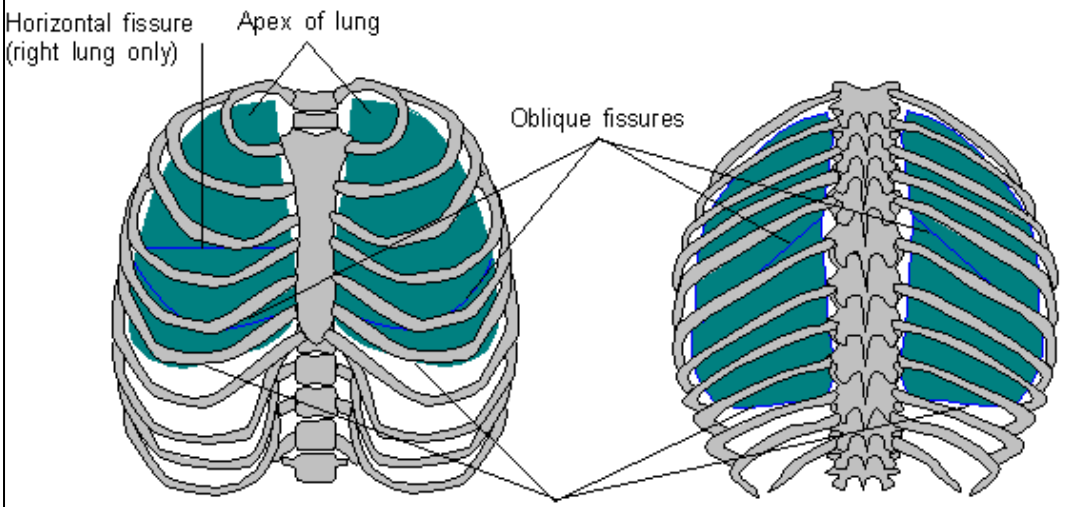


Figure 6a and 6b. Surface markings of the lungs

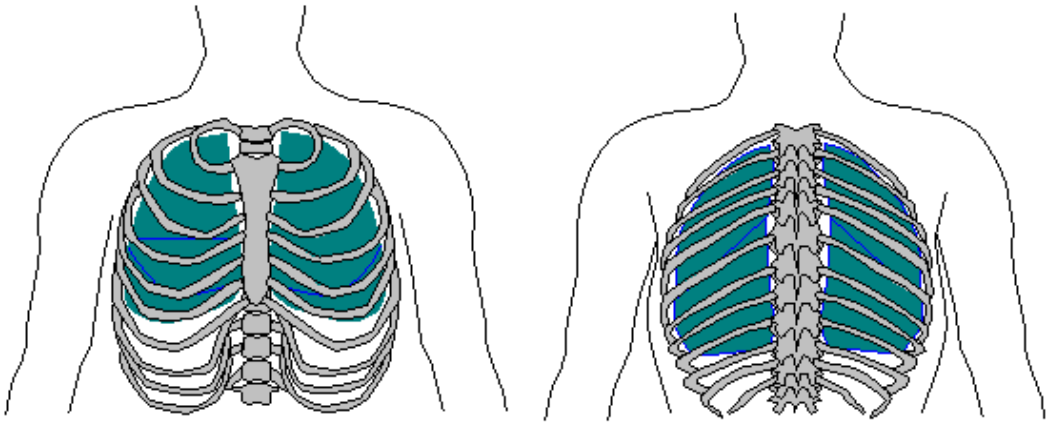
Figure 6a



The oblique fissure is approximately parallel to the medial border of the scapula when the arm is raised above thehorizontal

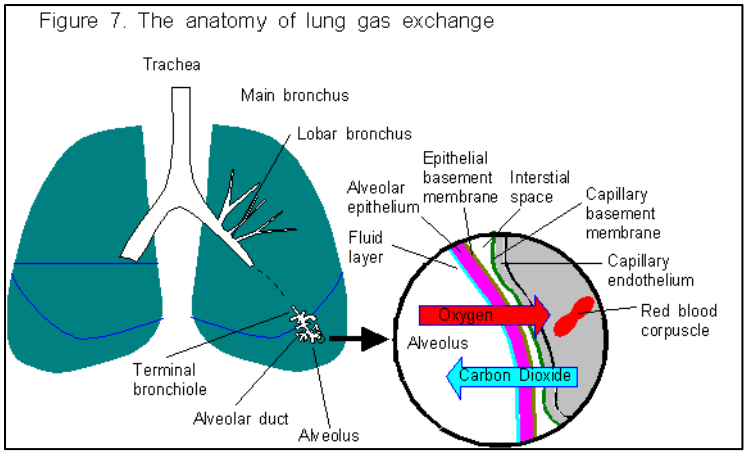
The lungs do not reach down to the lower border of the lowermost ribs - there is a (potential) pleural cavity

Figure 6b



The lungs are much higher up in the chest and are smaller than most people think!

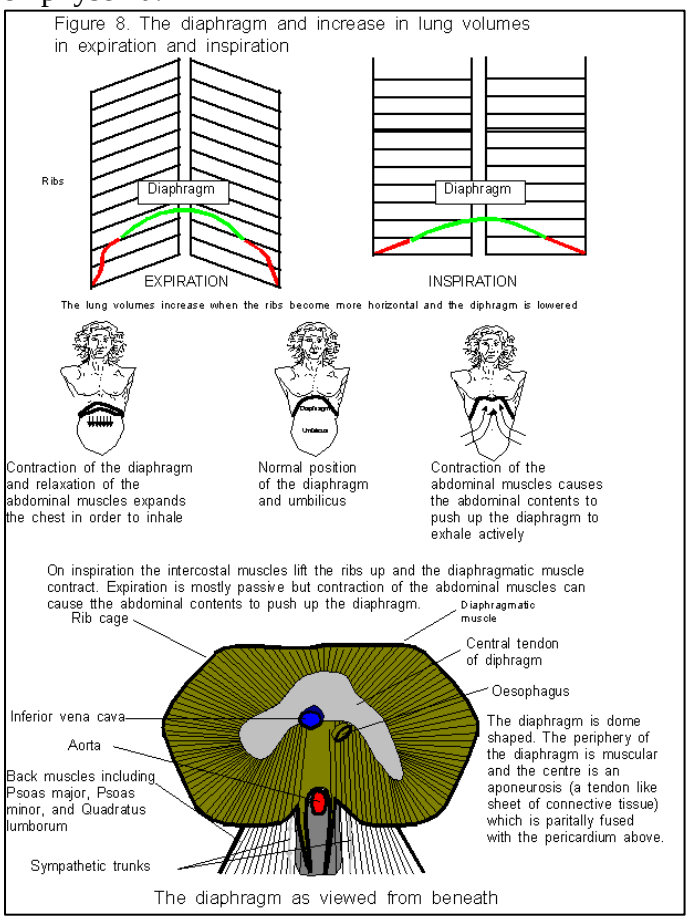
The right lung is slightly larger than the left, and has three lobes (whereas the left lung only has two lobes. The intralobular fissures often limited spread of diffusely invasive bacterial infections - hence lobar pneumonia.



PHYSIOLOGICAL PRINCIPLES

For adequate oxygenation of the tissues it is necessary that the lungs be ventilated, oxygen is able to diffuse into the blood passing through the lungs, and there is an adequate circulatory system.

Inspiration is an active process achieved by increasing the intrathoracic volume by contraction of the diaphragm and intercostal muscles (Fig. 8). Expiration, in contrast, is a passive process which relies upon the natural elastic recoil of the lung. If a more substantial expiration is required the abdominal muscles can contract and push up the diaphragm. The elasticity of the lung is reduced by processes such as fibrosis or emphysema.



Various measurements of ventilatory function (Fig. 9) can be made by getting patients to blow into various measuring devices.

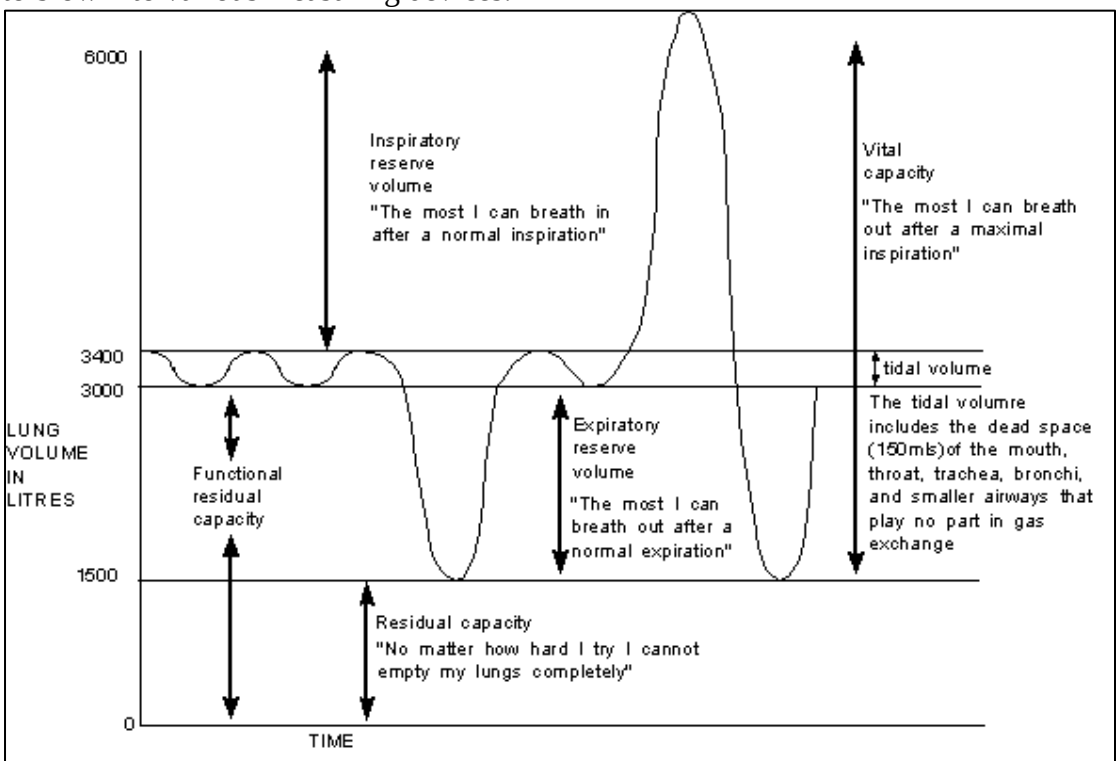
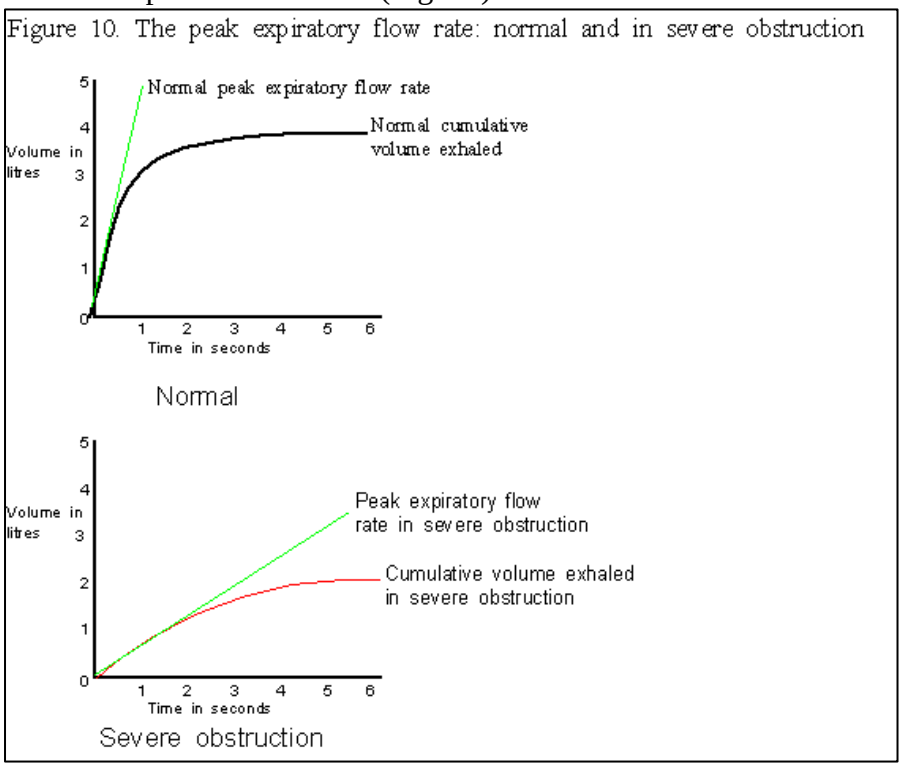


Figure 9. Respiratory volumes in an average adult

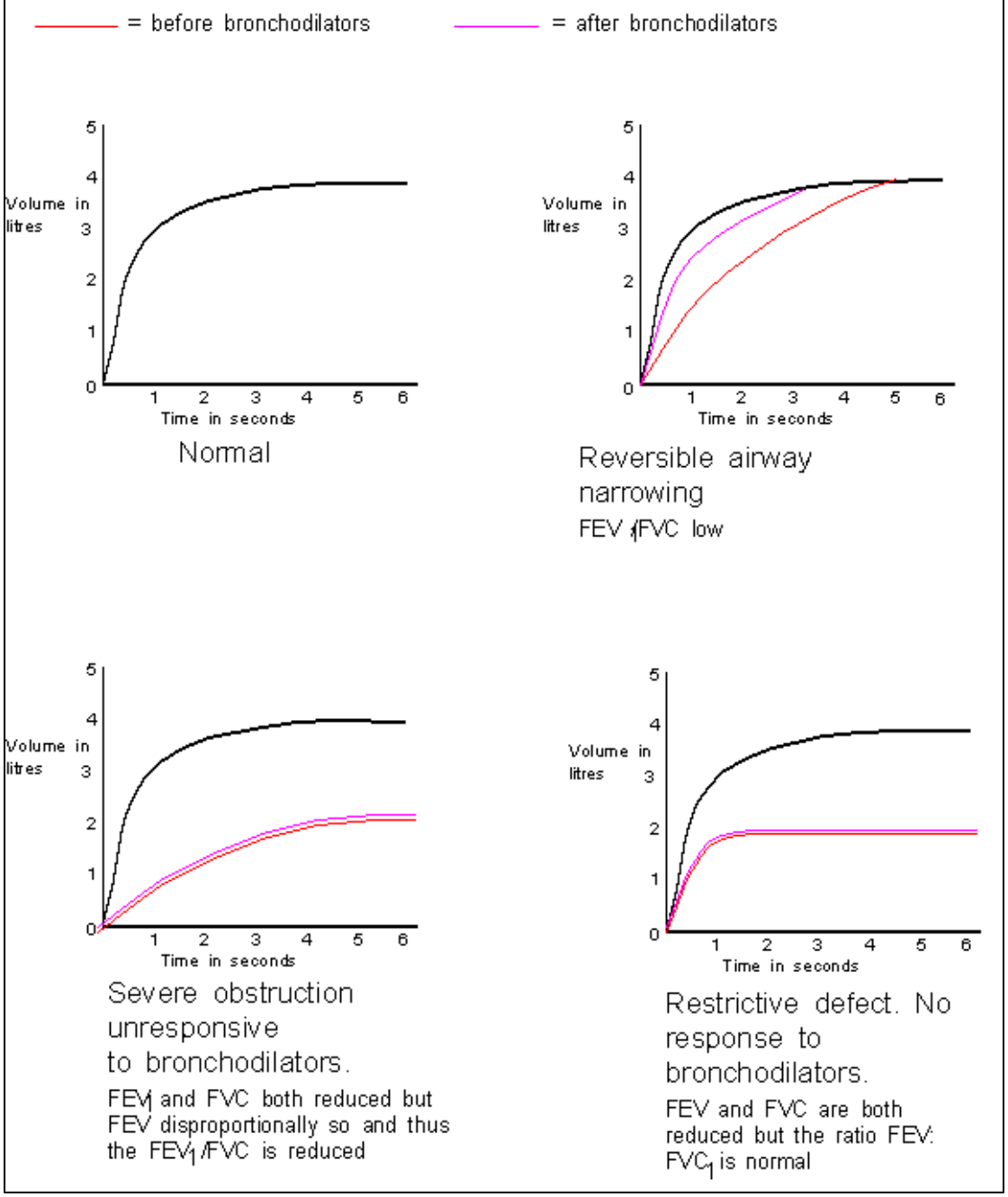
The peak expiratory flow (PEF) is the peak flow after the largest possible breath in and fastest possible blow out (Fig. 10).



It is reduced by airway narrowing or expiratory muscle weakness. The forced expiratory volume in the first second (FEV_1) and forced vital capacity (FVC) are

obtained by getting the patient to take in the largest possible breath (to total lung capacity) and then blow out as hard, fast and completely as possible (Fig. 11).

Figure 11. The forced expiratory volume in the first second and forced vital capacity in various states



Normally the forced expiratory volume in the first second is 70-80 percent of the forced vital capacity. If the airflow is reduced, for example by bronchial narrowing, then the forced expiratory volume in the first second is reduced *proportionally more* than the forced vital capacity and the ratio of FEV_1 to FVC is reduced. If the lungs are stiff, a restrictive defect, or the ventilatory muscles are weak then the forced expiratory volume in the first second and the forced expiratory volume are both decreased *in the same proportion* and the FEV_1 to FVC ration remains normal. In general the FEV_1, although less easy to measure, gives a more useful assessment of ventilatory flow obstruction than the PEF.

With exercise there is increased extraction of oxygen by skeletal muscles which is met by increases in the ventilatory rate and tidal volume.

The main business of the lungs is to oxygenate red blood corpuscles (RBCs) in the blood and to blow off excess carbon dioxide (the major by-product of energy production). To do this the average adult breaths about 400 mls of air (the tidal volume) in and out about 15-20 times per minute. Air comprises three gases (oxygen, carbon dioxide, nitrogen) and water vapour. The pressure (p) depends on the concentration of the gas present in the substance being tested. Gases pass from areas with a high pressure to areas with a low pressure. Another function of the lungs is to regulate weight: different individuals have different abilities to excrete carbon dioxide (on average 161 out of the 183 grams of carbon excreted daily – for a fuller explanation see Carbon Consideration are more important than Calories in Weight Regulation. Welsby PD *Int. J. of Design, Nature and Ecodynamics* 2013:**8**;388-395

At rest 250 mls of oxygen per minute are absorbed from the air and 200 mls of carbon dioxide per minute are exhaled as well as some water vapour. At sea level air is 21 percent oxygen with an arterial blood oxygen level (pO_2) of 160mms mercury with minimal carbon dioxide (Fig. 12).

Figure 12. Constituents of air, alveolar air and tissues.
In air the percentages and partial pressures are in direct proportion.

	DRY AIR	ALVEOLAR AIR	TISSUES
% OXYGEN	21	14	
pO_2 mm Hg (kPa)	160(20.8kPa)	100(13.3kPa)	40
% CARBON DIOXIDE	0	6	
pCO_2 mm Hg (kPa)	0	40 (5.3 kPa)	46
% NITROGEN	79	80	
pN_2 mm Hg (kPa)	600	573	
	Total p = 760 = atmospheric pressure		

The tissues have a pO_2 of 40 and an arterial blood carbon dioxide pressure (pCO_2) of 46 mms mercury. Thus there is a diffusion gradient across alveolar walls such that oxygen diffuses into the body and carbon dioxide diffuses out (Fig. 7). As expected alveolar air is intermediate between inspired air and tissue levels.

Measurement of the systemic arterial pO_2 gives a guide as to how well the lungs have oxygenated the blood that leaves the lungs. The percentage of oxygen saturation of

the blood in the peripheries can be measured by devices which colourmetrically measure the percentage oxygen saturation of the haemoglobin that is present (usually measured on an earlobe or at a fingertip) and this reflect tissue oxygenation. If the haemoglobin saturation with oxygen is low but the arterial pO_2 is normal then the tissues are desperate for oxygen. The saturations are not usually affected by mild anaemia but if anaemia is severe the tissues are oxygen starved and extract what oxygen there is in the haemoglobin and the saturation will then be low. Thus if the peripheral saturations are normal there is usually no need to measure the arterial pO_2.

Oxygen moves from areas of high partial pressure to areas of low partial pressure (Figs 12 and 13).

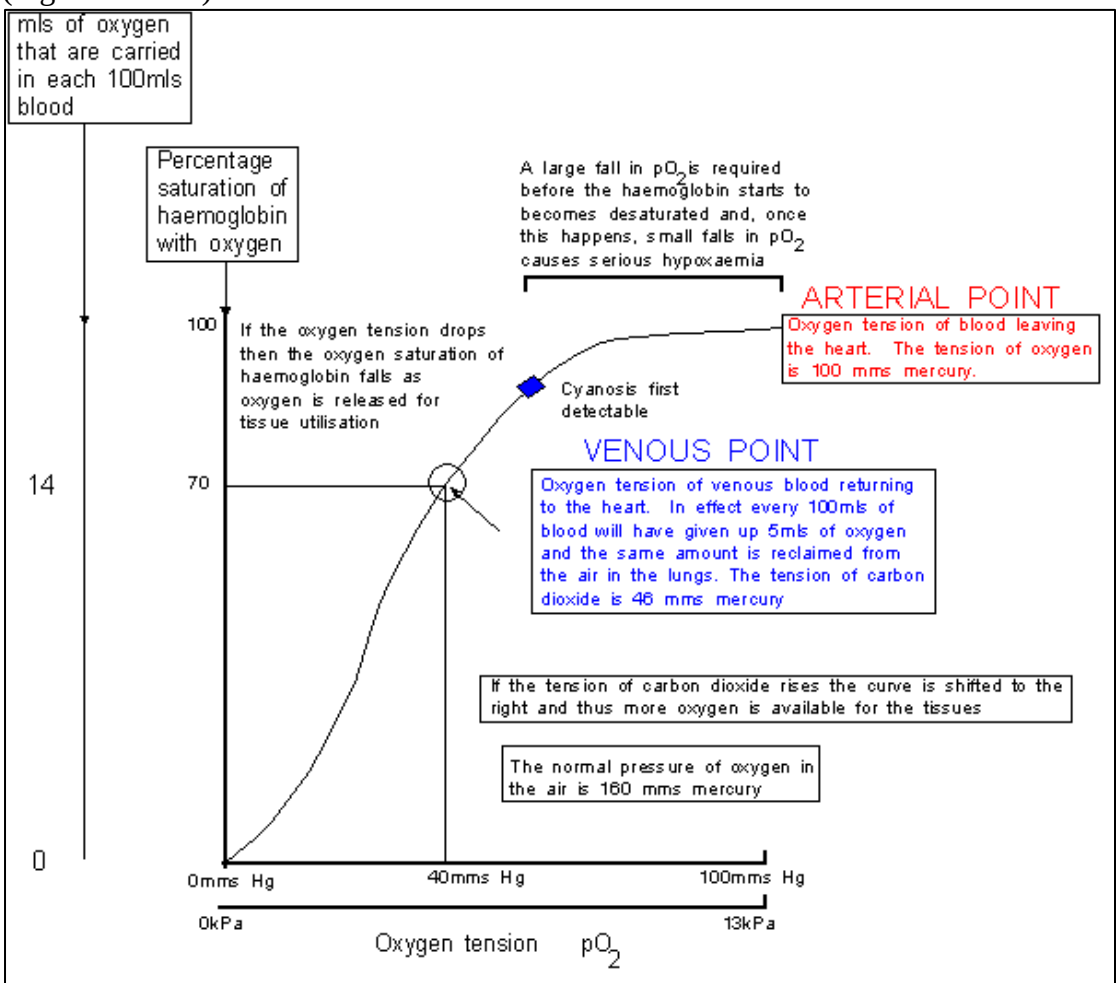


Figure 13.. The oxygen dissociation curve (this assumes that the red blood cells have a normal concentration of haemoglobin)

At a pO_2 of 100 mms mercury only 0.3 mls of oxygen is dissolved in every 100mls of blood and this is inadequate to sustain aerobic metabolism and an oxygen storage medium is required. This is provided by the haemoglobin in red blood corpuscles. With a normal haemoglobin level, 20 mls of oxygen is contained in every 100 mls of blood. The oxygen-haemoglobin dissociation curve, shows the relevant pressures and the volumes of oxygen carried by haemoglobin. Once the oxygen carrying power of the plasma and haemoglobin is saturated hyperventilation of the lungs or giving higher concentrations of oxygen will not be of significant benefit - the haemoglobin is saturated anyway and the amount or oxygen carried in the plasma will not alter

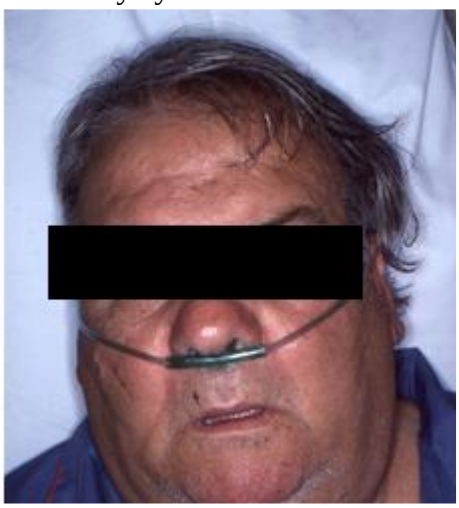
significantly (unless the pO_2 is increased by providing oxygen under increased pressure - hyperbaric oxygen).

Carbon dioxide can diffuse into and out of blood far more readily than can oxygen. The quantity of carbon dioxide in the blood can be directly affected by ventilation (*hyper*ventilation causes carbon dioxide levels to fall whereas *hypo*ventilation causes carbon dioxide levels to rise).
In normal individuals the ventilatory drive is provided by the plasma acidity which usually depends on the pCO_2 - the higher the pCO_2 the greater the ventilatory drive.

Why does carbon dioxide and not oxygen normally provide the ventilatory drive? Probably because there is a need to keep the tissue oxygenation at a near normal level for as long as possible, and this is achieved because the oxygen-haemoglobin dissociation curve allows haemoglobin to maintain tissue oxygen levels at normal levels within a wide range of tissue requirements until "the system" breaks down (this maintenance of oxygen provision is the main reason why the oxygen-haemoglobin dissociation curve is as it is). In contrast tissue and blood levels of carbon dioxide rise almost linearly (Fig. 14) and these can be used as the early warning system to cause increased ventilation before tissue oxygen levels fall.

Aspects of ventilation

If carbon dioxide levels are chronically high the respiratory centre in the brain may downgrade its sensitivity to carbon dioxide and instead rely on a low oxygen concentration (hypoxia) as the major ventilatory drive. Patients may become chronically cyanosed – "blue bloaters (Fig. 15)."



Giving a high concentration of oxygen to such patients may cause them to stop breathing until the oxygen returns to a low level, perhaps to a level dangerously lower than before the start of additional oxygen.

Oxygen can be given in high concentration to previously well patients with acute onset respiratory disease and a low pO_2 (low oxygen levels and not a high carbon dioxide levels that kill people). In such circumstances there should be gradual increments in the administered oxygen concentration which will increase pO_2 levels whilst the arterial blood CO_2 is being monitored. If the arterial pCO2 goes up then

too high a concentration of oxygen is being given and the patient's ventilation will need to be increased by drug therapy or mechanical ventilation.

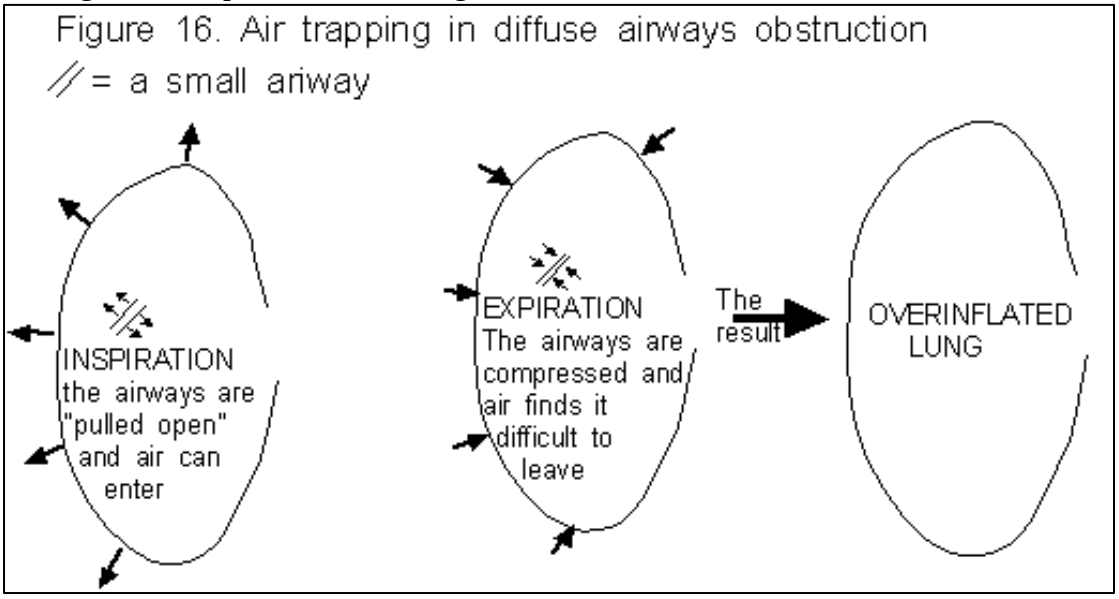
A normal or low carbon dioxide level, when combined with a low oxygen level, suggests that blood is not being exposed to oxygen in the lung, either because a ventilated part of the lung is not receiving blood (a ventilation-perfusion mismatch) or because the alveolar transmission of oxygen is impaired. The carbon dioxide in such situations does not rise as the oxygen falls for two main reasons. *Firstly*, carbon dioxide diffuses much more easily than oxygen. *Secondly*, the areas of the lung that are receiving blood are being hyperventilated because of the low pO_2 and thus more carbon dioxide than normal is washed out. The abnormally low carbon dioxide blood returning from the underperfused part(s) of the lungs results in a normal or low carbon dioxide content when mixed with the high carbon dioxide blood returning from the hyperventilated and perfused lung. *Oxygen does not have this facility* because a mixture of low oxygen haemoglobin and fully oxygenated haemoglobin can only result in low oxygen levels because haemoglobin cannot be supersaturated with oxygen. Carbon dioxide does not have a carrier system analogous to haemoglobin although some carbon dioxide is combined with protein or carried as bicarbonate.

Diffuse airway obstruction is caused by increased bronchial smooth muscle tone (which is normally at a low level) with increasing tone narrowing the airways. Diffuse narrowing also occurs if:

- there is left sided heart failure with airway narrowing being produced by swollen airway walls
- the bronchial smooth muscle is overdeveloped
- there are copious bronchial secretions.

In diffuse airway narrowing the major difficulty is with expiration - it is easy to suck air into the chest (an active muscular process), but on attempted expiration (usually a passive process) the airways are compressed (Fig. 16), and expiration is impaired, leading to overexpansion of the lungs.

Figure 16. Air trapping in diffuse airways obstruction



In medical terms oxygen lack is termed anoxia of which there are four types:

1) ***Anoxic*** *anoxia.* There is a low pO2 in arterial blood brought about by:

a) Hypoventilation or
b) Breathing low oxygen concentrations or
c) Breathing oxygen at low pressures such as at high altitudes or
d) Defective transalveolar transport of oxygen or
e) Shunting of blood away from normally ventilated alveoli (ventilation perfusion defects) or
f) An inadequate circulation in shock states.

2) ***Anaemic*** *anoxia*. If a person is anaemic (having a decrease in the number of red blood corpuscles and/or a less than normal haemoglobin content) or has an abnormal haemoglobin with reduced oxygen transporting ability then the pO2 will be normal initially - the red blood corpuscles can give up oxygen but there is a reduced total quantity to give up, but once the depoleted oxygen carrying power is exhausted then anoxia results. Carbon monoxide poisoning has the same effect as it binds more tightly to haemoglobin than does oxygen and thus oxygen is displaced.

3) ***Stagnant*** *anoxia*. This occurs when the circulation of blood is so slow that the tissues are starved of oxygen.

4) ***Cell damage*** *anoxia*. Tissue cells are damaged such that they cannot utilise the available oxygen.

Respiratory failure

Respiratory failure exists when, if a patient is at sea level, awake and breathing air, there is a lung dysfunction such that 1) arterial pO_2 fall below normal or 2) when pCO_2 rises above normal There are two types of respiratory failure.

- ***Type I*** respiratory failure occurs when there is a low pO_2 with a normal or low pCO_2. Causes include conditions in which the increased carbon dioxide diffusabilty or mixing of high and low carbon dioxide blood is able to keep the pCO_2 at a normal or even low level. Such conditions include *acute* asthma or pneumonia. The normal ventilatory drive driven by a (high) carbon dioxide levels is replaced by a low oxygen drive caused by the pathology.
- ***Type II*** respiratory failure occurs when there is a low pO_2 with a high pCO_2. Type II failure is thus a ventilatory failure.

Patients with *chronic* respiratory disease who maintain their pO_2 and, unlike blue bloaters, use their pCO_2 induced ventilatory drive to prevent their carbon dioxide from rising by hyperventilating are often termed “pink puffers” whereas patients who do not hyperventilate and rely upon their low pO_2 to provide ventilatory drive gradually accept their low pO_2 levels and consequent become cyanosed. Such patients are often obese and are thus termed “blue bloaters” (Fig. 15). If blue bloaters are given too much oxygen they will stop breathing and their carbon dioxide rises even further. There are other metabolic effects of respiratory problems.

Figure 17 details the common causes of cough, Figure 18 the common causes of sputum production and Figure 19 the common causes of shortness of breath.

Figure 17. Common causes of cough

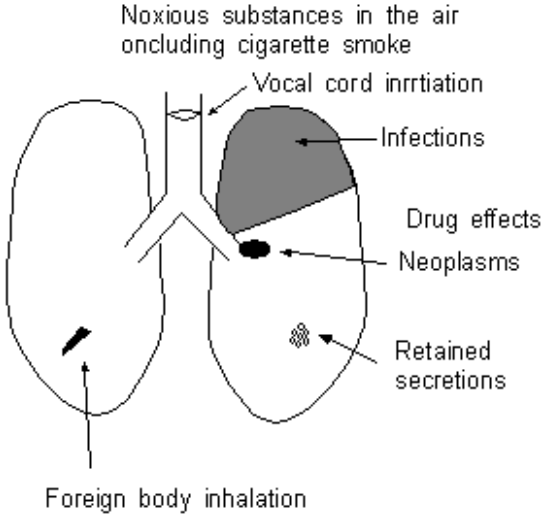


Figure 18. Common causes of sputum production

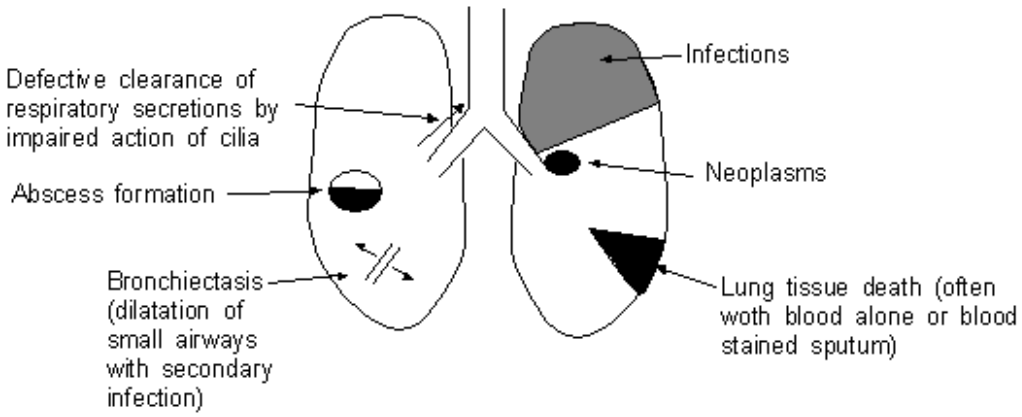
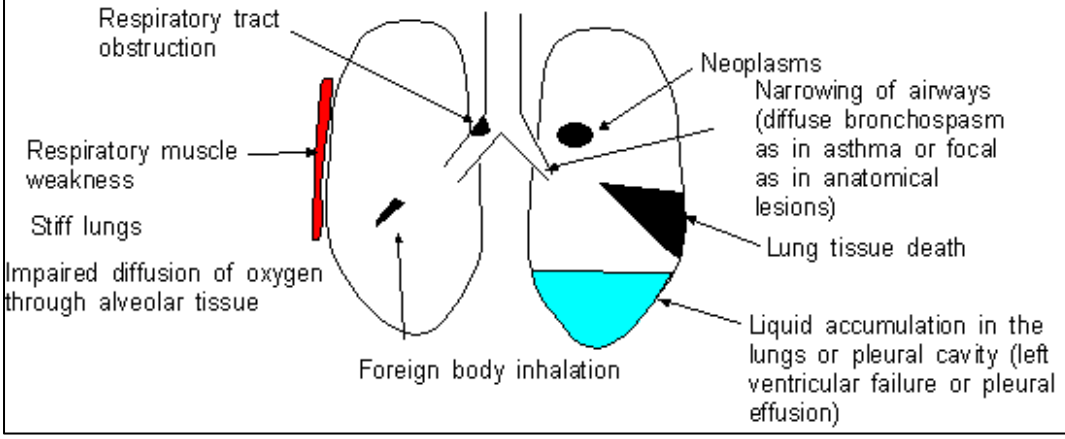


Figure 19. Some common causes of respiratory shortness of breath



Some specific conditions

Asthma is diffuse reversible airways obstruction in which there is airway inflammation with eosinophils and bronchial hyperreactiveness. Treatments include avoidance of provoking factors, use of drugs to reverse respiratory smooth muscle contraction and to reduce vagal bronchoconstrictor tone, to reduce inflammation and to reduce secretions. *Emphysema* is an abnormal and permanent enlargement of air spaces distal to the terminal bronchiole, accompanied by destruction of their walls and without obvious fibrosis. In effect there is honeycombing of the lungs. *Pneumonia* is

an accumulation of secretions and inflammatory cells in the alveolar spaces of the lung, usually caused by infection. *Bronchiectasis* is persistent chronic dilatation of the bronchi.