PARTNERSHIP IN CRITICAL CARE DEPT. WAIKATO

CRITICAL CARE WAIKATO HOSPITAL

WORKBOOK

NAME: _____

Te Hanga Whaioranga Mō Te Iwi
Building Healthy Communities

Waikato District Health Board

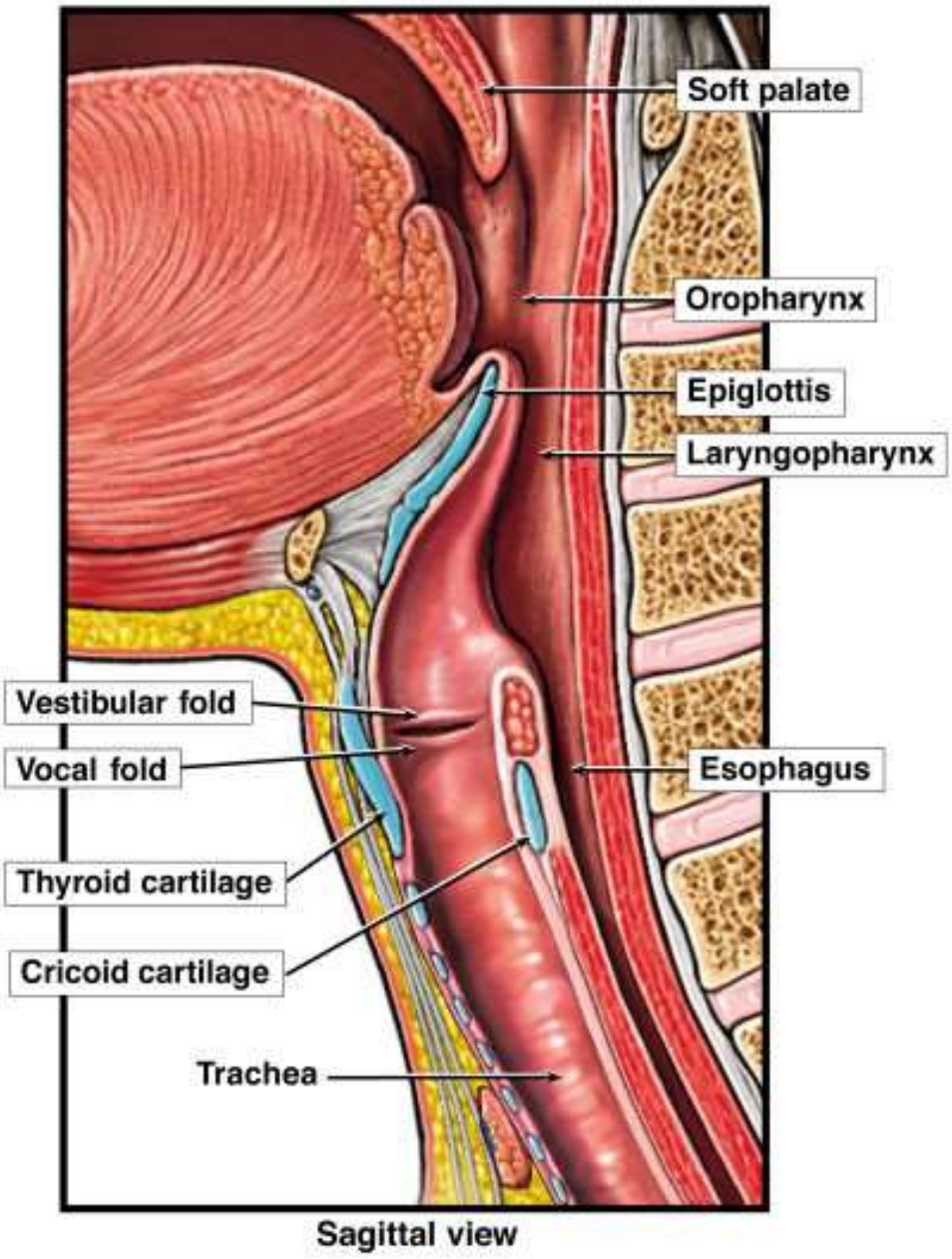
Section 1

Anatomy and physiology of the respiratory system

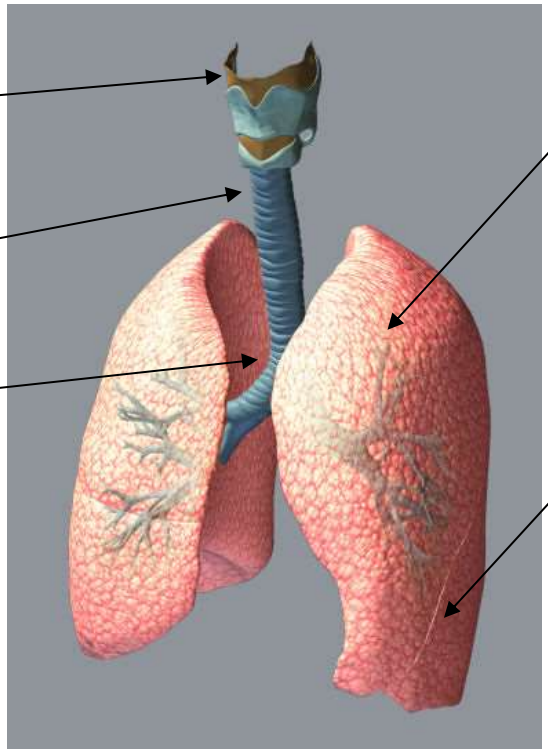
The Upper and Lower Airway

In terms of function, the respiratory system can be divided into two parts: the conducting airways (through which the air moves between the atmosphere and the lungs), and the respiratory tissue of the lungs (where gas exchange takes place).

The conducting airways consist of the nasal passages, mouth, pharynx, larynx, trachea, bronchi and bronchioles.



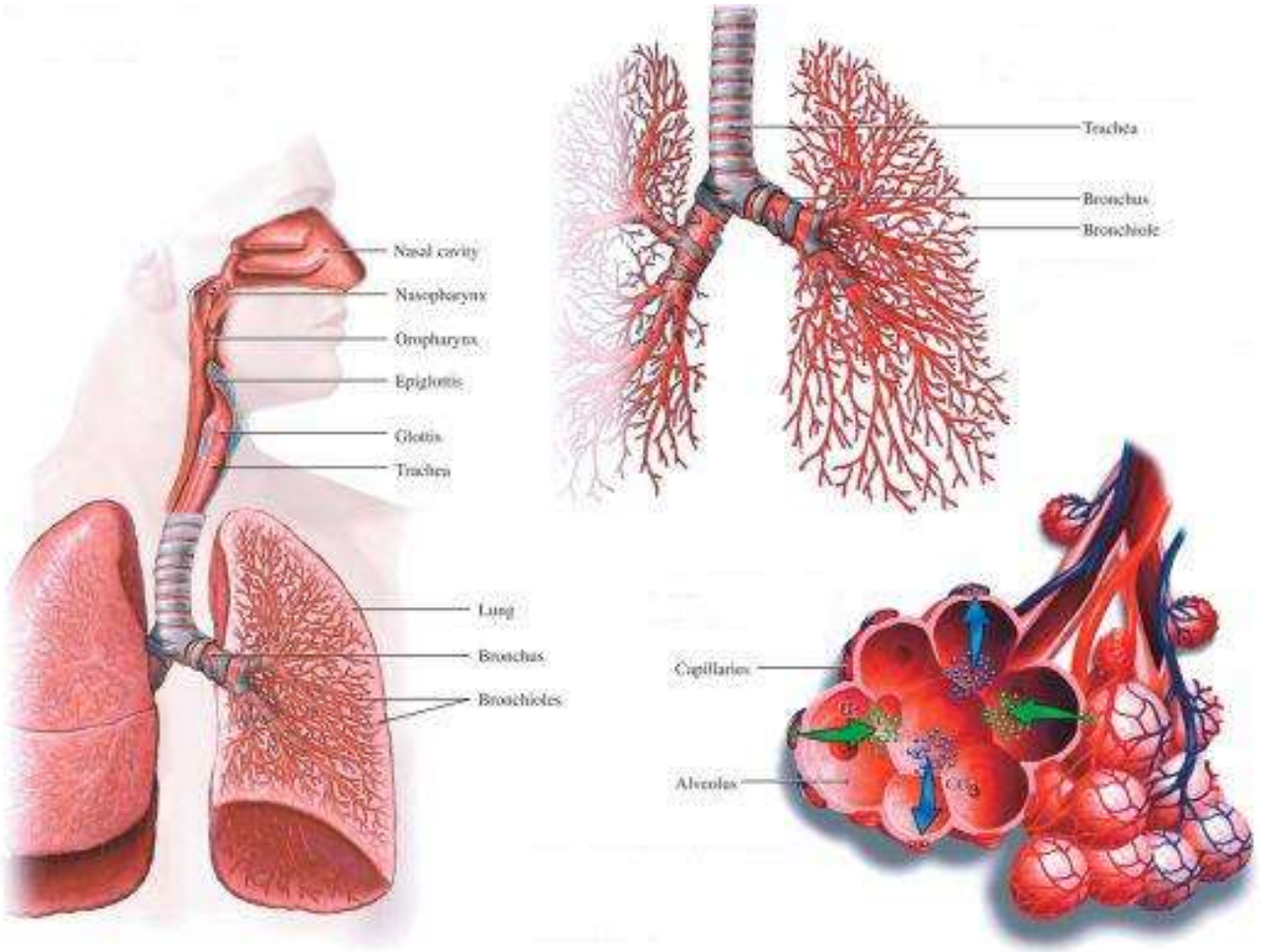
Conducting airways not only deliver air to the alveoli, they also serve to warm, filter and moisten the air as it passes through. The mucous produced by the epithelial cells in the conducting airways forms a layer called the mucociliary blanket. It protects the respiratory system by trapping dust, bacteria or other particles entering the airway. Cilia move the blanket towards the oropharynx, where it is expectorated or swallowed. The performance of cilia is impaired in conditions where oxygen levels are higher or lower than normal, in drying conditions, and with cigarette smoking.



The tracheobronchial tree

The trachea runs from the larynx to the major bronchi. The walls are supported by C shaped cartilages which prevent it from collapsing from the negative pressure in the chest. The open part of the 'C' is where the trachea intersects with the oesophagus.

The trachea divides into the right and left main bronchi at the carina, then continues to divide into secondary bronchi and from there into smaller bronchi and finally into terminal bronchioles. The structure of these bronchi changes as they become smaller. As branching progresses, the cartilage decreases and smooth muscle and elastic tissue increase until, as bronchioles, the cartilage has disappeared and the walls are mainly smooth muscle.

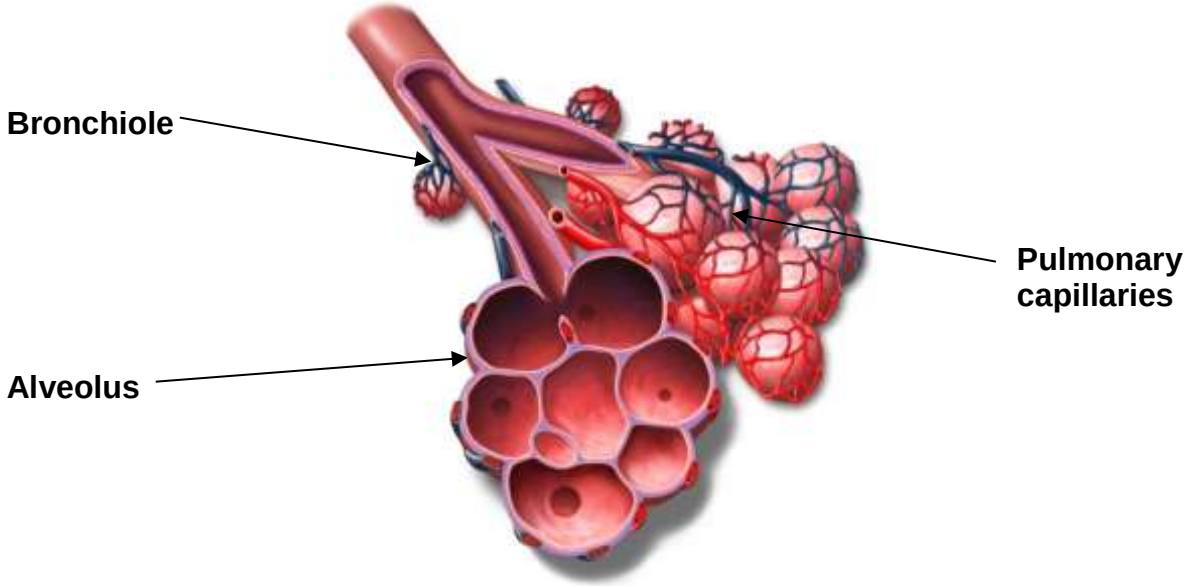


The lungs

The lungs are divided into lobes with three on the right and two on the left.
The lungs serve more than one purpose. In addition to gas exchange, they also convert angiotensin I to angiotensin II, serve as a reservoir for blood, and inactivate some vasoactive substances. There are a number of heparin-producing cells in the capillaries of the lung, where the heparin helps to prevent the formation of small clots. The lungs have a dual blood supply. The bronchial circulation supplies the structures and airways, whilst the pulmonary circulation provides for gas exchange in the lungs.

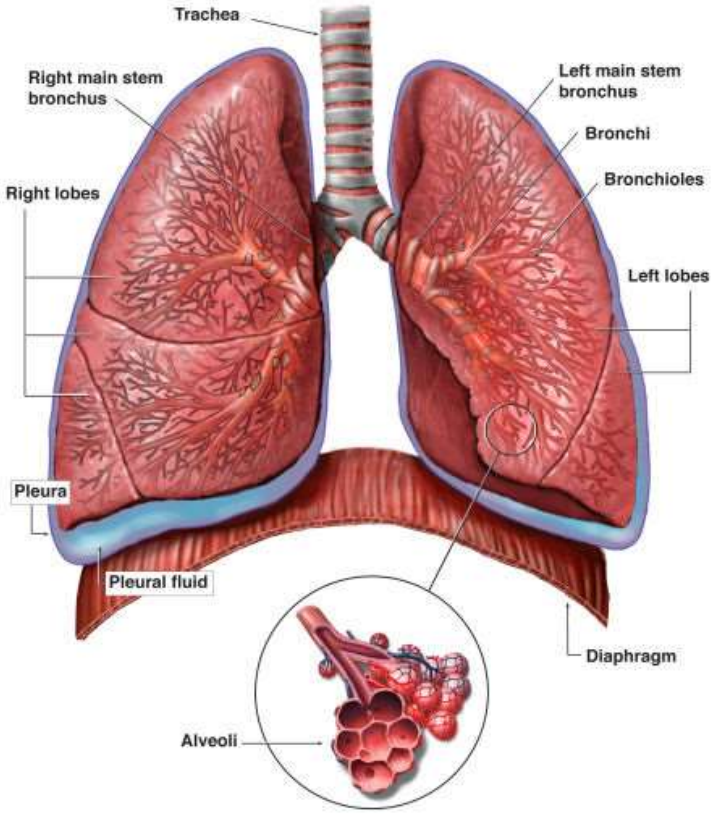
Compliance refers to how easily a lung can be distended by a given pressure. In other words, a non-compliant (or stiff) lung will need more pressure to be inflated by a given volume than a stretchy, compliant lung, which distends easily. Compliance is determined by the elasticity of the fibres of the lung (elastin is more stretchy than collagen), the water content of the lung, and the amount of surfactant. It can also be affected by the anatomy of the chest.

Gas exchange takes place in the terminal lobules. The terminal lobule is the anatomical and functional unit of the lung, including a respiratory bronchiole, alveoli and pulmonary capillaries.



Pleura

The pleura consist of a double layer of membrane, one of which lines the thoracic cavity, while the other encases the lung. There is a thin layer of serous fluid between the two pleura, enabling the layers to glide over each other. In health, there is no separation between the two layers.



Normal breathing

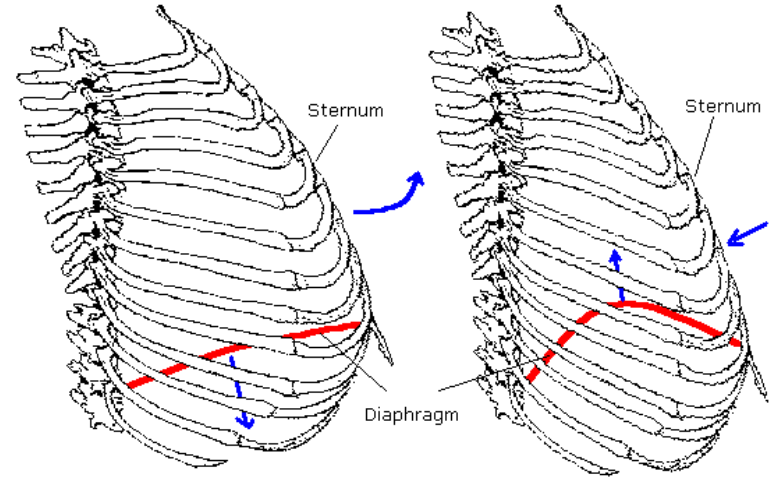
Ventilation is the movement of gases in and out of the lungs. Movement of the gases is caused by changes in pressure as the size of the chest cavity is increased or decreased by the respiratory muscles. Gas movement also depends on the airway remaining open.

The main muscle involved in breathing is the diaphragm, but the external intercostals are also involved. The main accessory muscles of respiration are the scalene and sternocleidomastoid.

On inspiration, the size of the chest cavity is increased as the diaphragm contracts downwards and the intercostals draw outwards and upwards. This increase in the volume of the chest cavity causes a reduction in pressure, because pressure is inversely related to volume (Boyle's Law). As a result, air is drawn into the lungs because the pressure in the chest is lower than the pressure outside the chest cavity. Note that inspiration is due to the *negative* pressure generated inside the chest cavity – this is where normal breathing differs from mechanical ventilation, which depends on *positive* pressure.

On expiration, the respiratory muscles relax. As a result, the volume of the chest cavity decreases, resulting in increased pressure. Air then exits the lungs following this pressure gradient. The ratio of inspiration to expiration in normal breathing is 1:2, i.e. expiration lasts twice as long as inspiration.

Breathing in and out



Breathing in:

Sternum rotated upwards as intercostals contract. Diaphragm descends

Breathing out:

Sternum rotates downwards as intercostals relax. Diaphragm ascends.

Image source: Wikibooks.org File Gcsebiolbreathe2.gif

Pleural Pressure

The pressure in the pleural cavity is always negative. This negative pressure causes the two pleura to stick to each other, preventing the lungs from collapsing as the chest expands.

Ventilation and perfusion

For gas exchange to take place, ventilation must be associated with perfusion, and vice versa. Optimal gas exchange would happen if each alveolus was well ventilated and was surrounded by a capillary with good perfusion. In this case, ventilation (V) would be equal to perfusion (Q). There are several factors that can interfere with this:

1. Atelectasis (alveolar collapse) or consolidation (the alveoli contain liquid instead of gas) will mean that blood in the pulmonary capillaries passes the affected unventilated alveolus and returns to the heart without taking part in gas exchange. This is referred to as ‘shunting’.

2. In conditions such as asthma or bronchitis the alveolus is perfused but only partially ventilated, because narrow inflamed airways reduce the amount of air reaching the alveoli.

3. In pulmonary vasoconstriction the alveolus is ventilated but only partially perfused. One cause of pulmonary vasoconstriction is hypoxia – if the pulmonary vessels detect a PaO_2 of less than 8kPa then they will constrict and limit blood supply to those areas.

4. In pulmonary embolism an area of lung will have ventilation but no perfusion as the flow of blood is obstructed by the embolus.

Ventilation

In a normal healthy person (standing up), gravity and the lung's weight act on ventilation by increasing pressure at the base (making it less negative) and thus reducing the alveolar volume ('squashing' of the alveoli). These 'squashed' alveoli are more compliant because they contain relatively small amounts of air before inspiration and can therefore stretch to a much larger volume on inspiration. Therefore a larger volume of gas enters and leaves the alveolus with each breath compared to alveoli at the top (apex) of the lung.

Alveoli at the apex of the lung (the top) are more distended because the pleural pressure is lower. Because they are already quite distended (stretched), even before inspiration, these alveoli increase in size only a relatively small amount on inspiration, and so smaller volumes of gas enter and leave these alveoli on inspiration and expiration.

Perfusion

In a normal healthy person (standing up), there is greater blood flow to the base of the lung compared with the higher parts of the lung because of gravity. As a result, there is better perfusion to the lower regions of the lung (which, as mentioned above, have better ventilation) and reduced perfusion to the higher regions of the lung (which have poorer ventilation). In normal health, this results in a good balance between ventilation and perfusion in the different regions of the lung.

Work of breathing

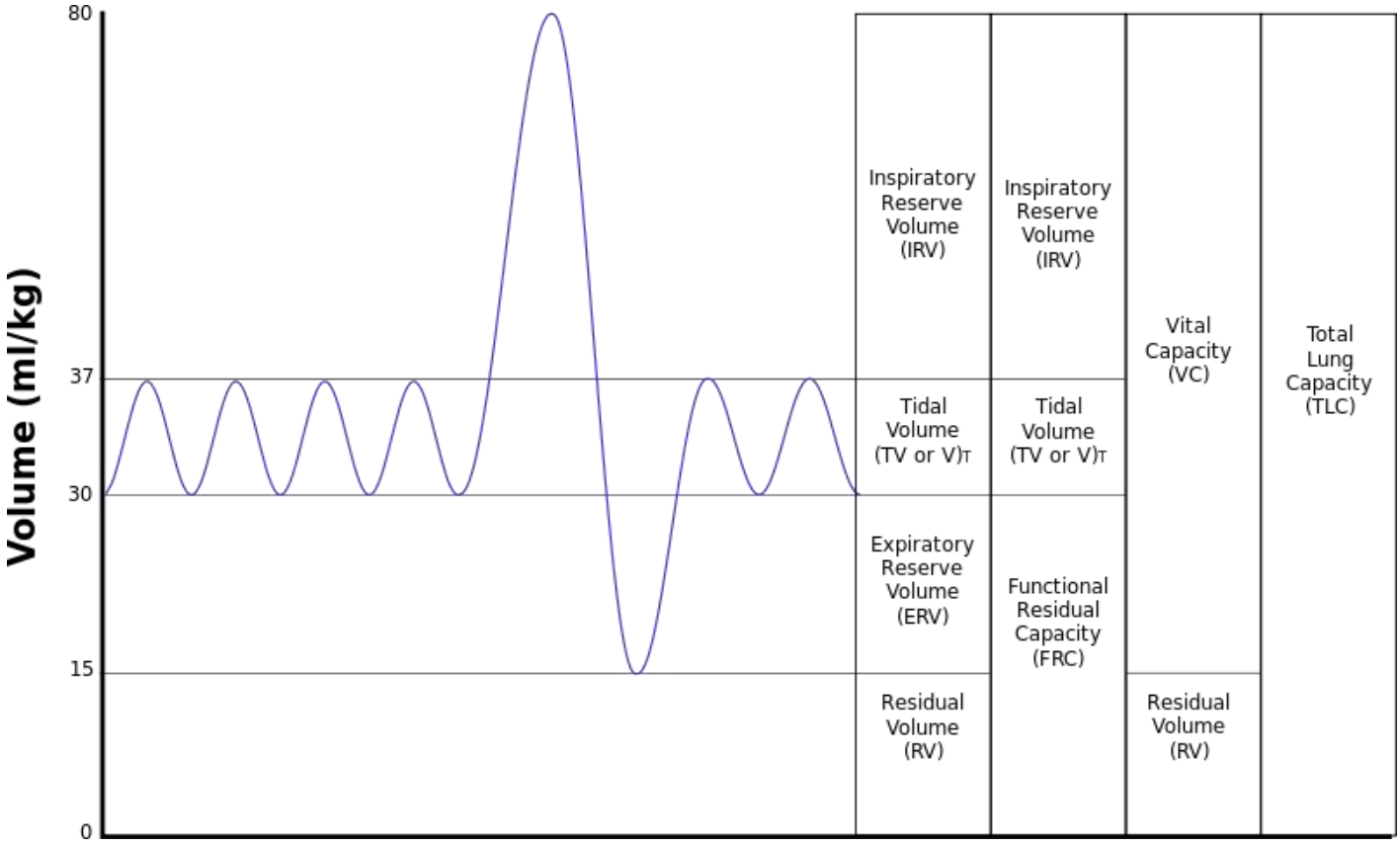
In good health, breathing requires work only during inspiration, using around 3-5% of the body's energy. Factors such as increased minute volume (e.g. during exercise), decreased compliance, or increased airway resistance will mean that the work of breathing is increased and therefore more energy and oxygen are used.

Lung volumes

Volume measurements show the amount of air in the lung at various parts of the respiratory cycle. Normal volumes are as follows:

Tidal volume (volume inhaled and exhaled with each breath)	500 ml
Inspiratory reserve volume (Maximum air that can be inhaled after normal inhalation)	3100 ml
Expiratory reserve volume (Maximum air that can forcibly be exhaled after normal exhalation)	1200 ml
Residual volume (Volume of air remaining after maximum exhalation)	1200ml
Inspiratory capacity (Inspiratory reserve + Tidal volume)	3600ml

Functional residual capacity (Expiratory reserve + Residual volume)	2400ml
Total lung capacity (Inspiratory capacity +Functional residual capacity)	6000ml



Gas exchange and transport

The air in the alveolus is composed of a mixture of gases, along with water vapour. The pressure of these gases will determine diffusion across the alveolar wall and into the bloodstream as the gases will pass from areas of high pressure to areas of low pressure. Therefore oxygen will move out of the alveolus and into the bloodstream, whilst CO_2 will diffuse out of the blood and into the alveolus to be exhaled (CO_2 diffuses more readily than O_2).

The rate at which gas moves through a membrane depends on the pressure difference across the membrane, the surface area of the membrane, and the permeability of the membrane. The surface area of the alveolus is very large and the membrane is very thin so gases diffuse efficiently.

One way of referring to the transfer of gases across the alveolar membrane is the 'Alveolar - arterial gradient'. This value reflects the difference between the oxygen pressure in the alveolus (PAO_2) and the oxygen pressure in the arteries (PaO_2). In a healthy person breathing room air there is only a small difference between alveolar and arterial oxygen pressure (0.5–1.5kPa), whereas a patient with severe pneumonia, on high oxygen (e.g. 100%) with a low PaO_2 (e.g. 8kPa) will have a very large Alveolar arterial – gradient (around 80kPa).

There are many factors which influence the Alveolar-arterial gradient (e.g. the four pathologies listed on page 8). In addition to these, inflammation or infection can cause thickening of the alveolar wall and this will slow the rate of diffusion of oxygen from the alveolus to the bloodstream, increasing the A-a gradient.

It is not easy to correct hypoxaemia in a patient with V-Q mismatch. Turning up their oxygen percentage will often have only a small effect. The easiest way to understand this is to think of an imaginary example: Let us imagine that we have a patient: half of his alveoli are working perfectly and the other half of his alveoli have a severe V-Q mismatch, so that they have perfusion but no ventilation. If you were to put this patient on 80% oxygen then all the good alveoli would oxygenate well, so that the capillaries leaving these alveoli would contain blood that is 100% saturated. But this blood will then mix with the other half of the blood coming from the alveoli that have not been ventilated (with an oxygen saturation of about 70%). When the well oxygenated blood (100% saturated) mixes with the poorly oxygenated blood (70% saturated) the result will be that this blood returns to the left side of the heart with an overall saturation of around 85%. This will then be pumped around the body and the pulse oximeter will read 85%.

Now, in this example, if you turn up the oxygen to 100% it will make no difference at all. This is because the capillaries of unventilated alveoli will not pick up any more oxygen because no oxygen or air is getting into these alveoli (e.g. they are collapsed or consolidated), and the capillaries of the well-ventilated alveoli cannot pick up any more oxygen because they were *already* 100% saturated even before you turned the oxygen up from 80 to 100%. You cannot saturate blood above 100%, and so the 'good' alveoli cannot compensate for the 'bad' alveoli.

However, this is not the case with CO_2. In this case, the patient's 'good' alveoli can compensate for the 'bad' alveoli if they work harder. Take the same example as above. If a patient has half 'good' and half 'bad' alveoli, then you would expect them to have a raised CO_2 because only half of their alveoli are eliminating CO_2. This is true, they would have a higher CO_2, but, as long as the patient is able to take bigger breaths and/or breathe more frequently (i.e. increase their minute volume), then they will be able to reduce their CO_2 because by breathing harder they expose the blood in their pulmonary capillaries to increased volumes of fresh inspired air, and the more they increase their breathing (minute volume), the more CO_2 is able to escape by diffusing out of the patient's bloodstream. For this reason, a patient in the early stages of respiratory distress or failure will often have a normal or low PCO_2, but then as they become increasingly fatigued their breathing will become shallower and their minute volume will decrease, and their PCO_2 will then increase to above normal.

Respiratory failure with a low PaO_2 and a normal (or low) PCO_2 is called 'Type 1 Respiratory Failure'. Respiratory failure with a low PaO_2 and a raised PCO_2 is called 'Type 2 Respiratory Failure'.

Once oxygen has diffused into the blood it is transported in two ways: dissolved in the plasma and bound to haemoglobin. Less than 3% of oxygen is carried dissolved in the plasma, and this is what we measure as the PaO_2. 97% of the oxygen in the blood is carried bound to haemoglobin and this is measured as the SaO_2 (or SpO_2). It is called 'SaO_2' when we measure this value using an arterial blood gas sample and 'SpO_2' when we measure the same thing using a pulse oximeter.

There are various factors affecting the affinity of haemoglobin for oxygen. Haemoglobin binds more readily with oxygen if the patient is alkalotic, CO_2 is lower, the body temperature is lower, or there is less 2,3-DPG. If these conditions are present there will be a higher oxygen saturation for any given PaO_2 and less oxygen is released to the tissues. The opposite will occur if the patient is acidotic, febrile, has a high CO_2 or higher levels of 2,3-DPG. The affinity of haemoglobin for oxygen is represented as the oxyhaemoglobin dissociation curve.

Methaemoglobin is an abnormal form of the Hb molecule where the iron form of the haem cannot bind with O_2. This can be associated with nitric poisoning and is monitored in the ICU when a person is receiving Nitric Oxide therapy.

Control of ventilation

Breathing is controlled by both the brainstem and by chemical regulation.
Brain: groups of neurons in the pons and medulla control the rate and rhythm of breathing through impulses sent to the respiratory muscles.
Chemical: Chemoreceptors monitor changes in oxygen and carbon dioxide levels. These are located in the medulla (carbon dioxide) and aortic arch plus carotid arteries (oxygen).

The cerebral cortex also allows the person to have some conscious control over rate of breathing. However, ultimately the physiological effects of increased CO_2 and decreased O_2. will override conscious control.