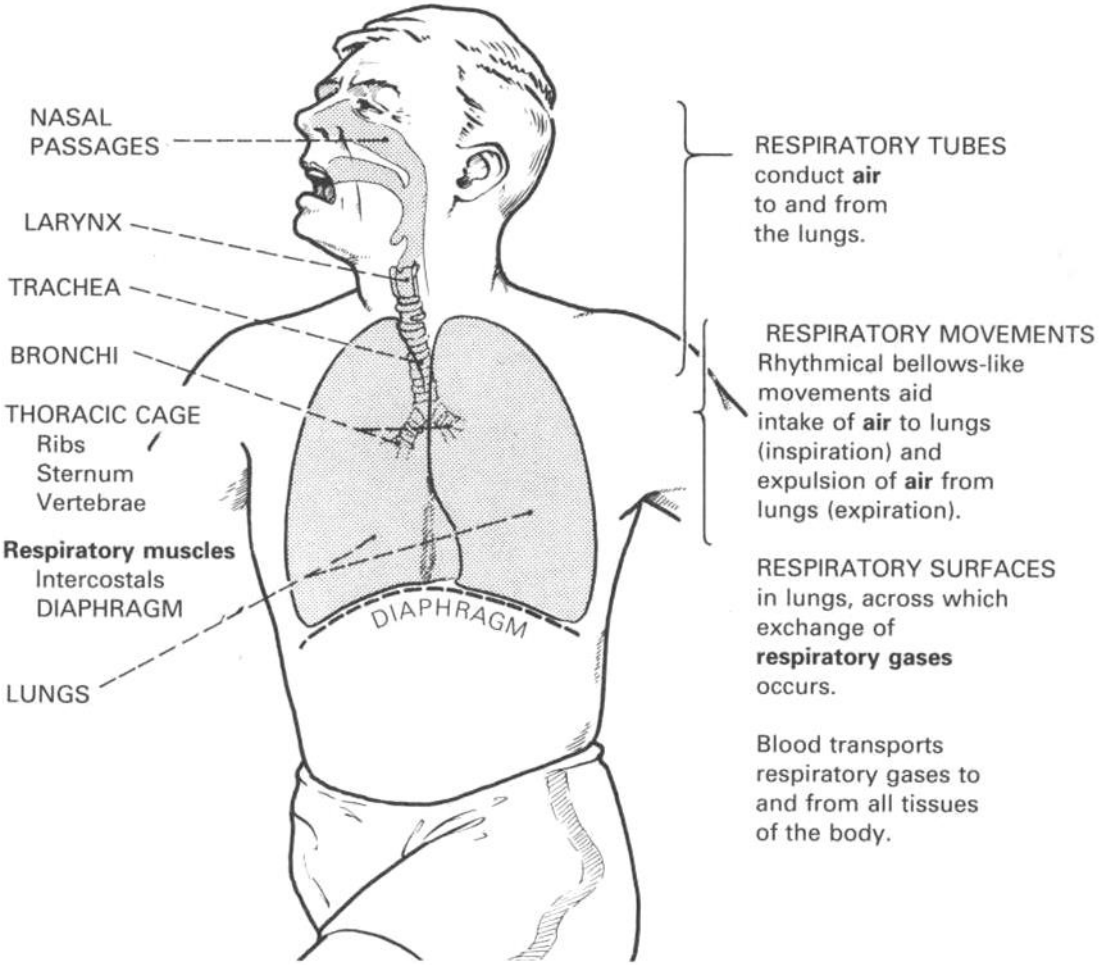
The Respiratory System

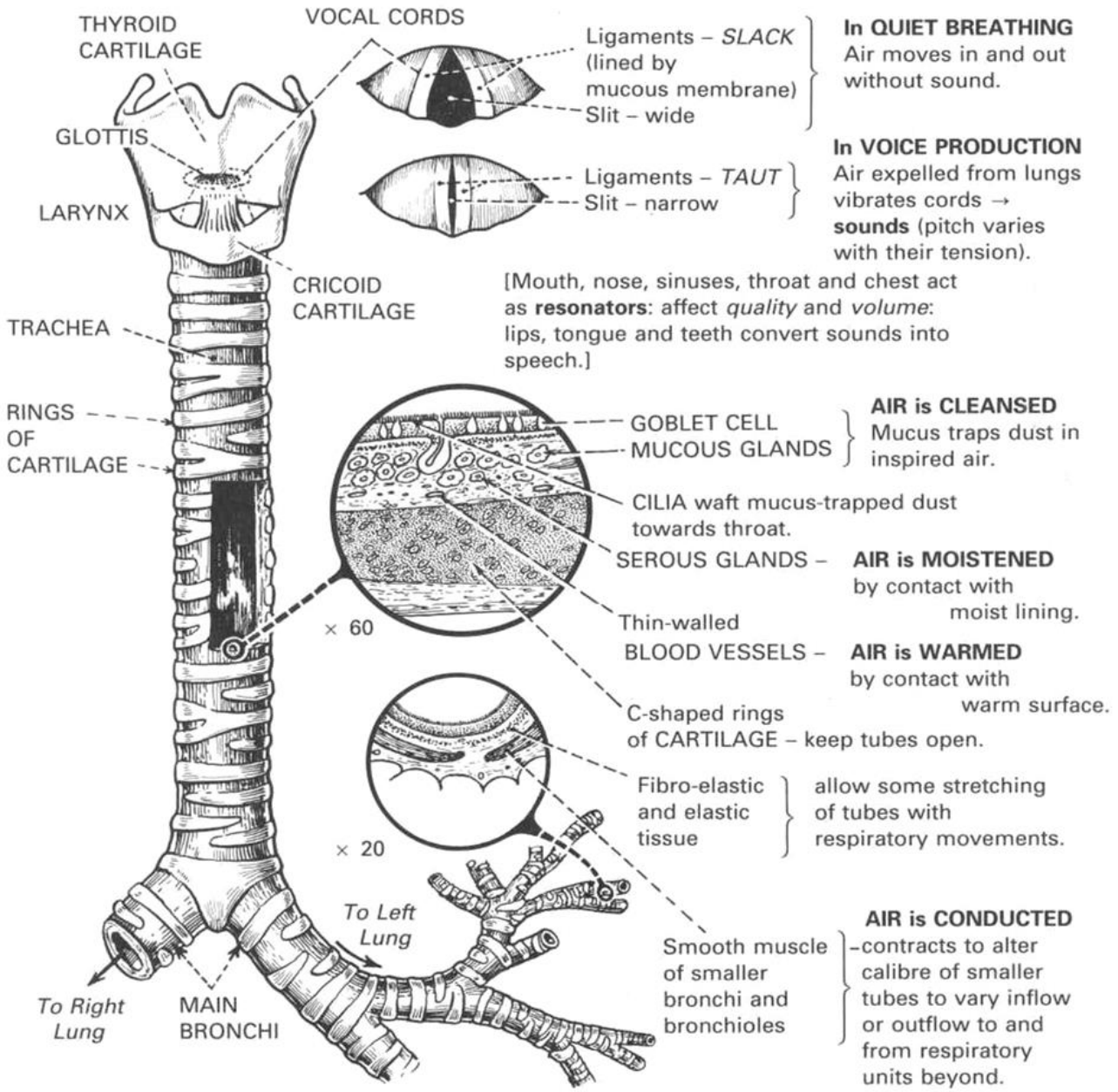
All living cells require to get **oxygen** from the fluid around them and to get rid of **carbon dioxide** to it.

Internal respiration is the exchange of these gases between tissue cells and their fluid environment.

External respiration is the exchange of these gases (oxygen and carbon dioxide) between the body and the external environment.

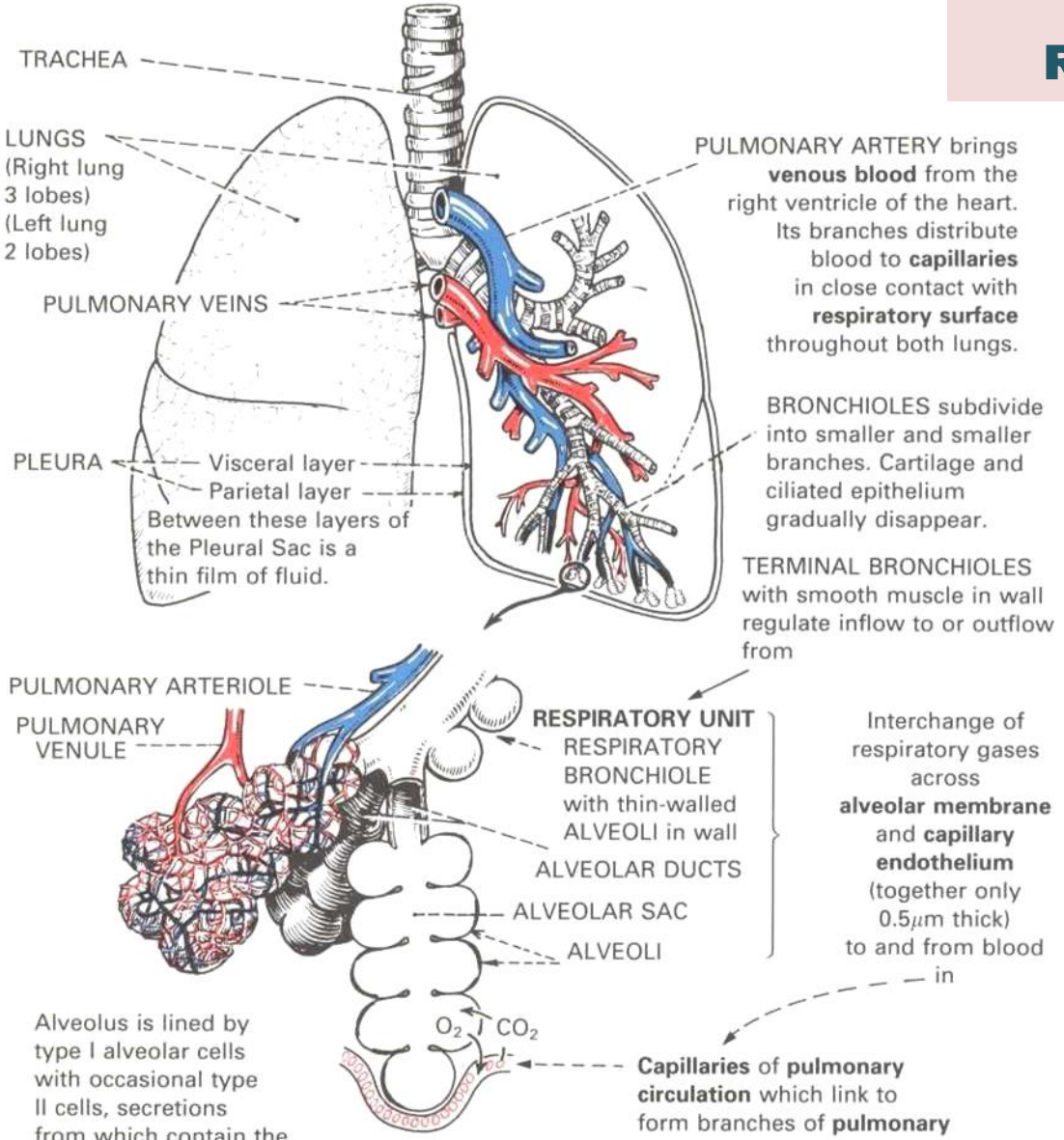


Air Conducting Passages



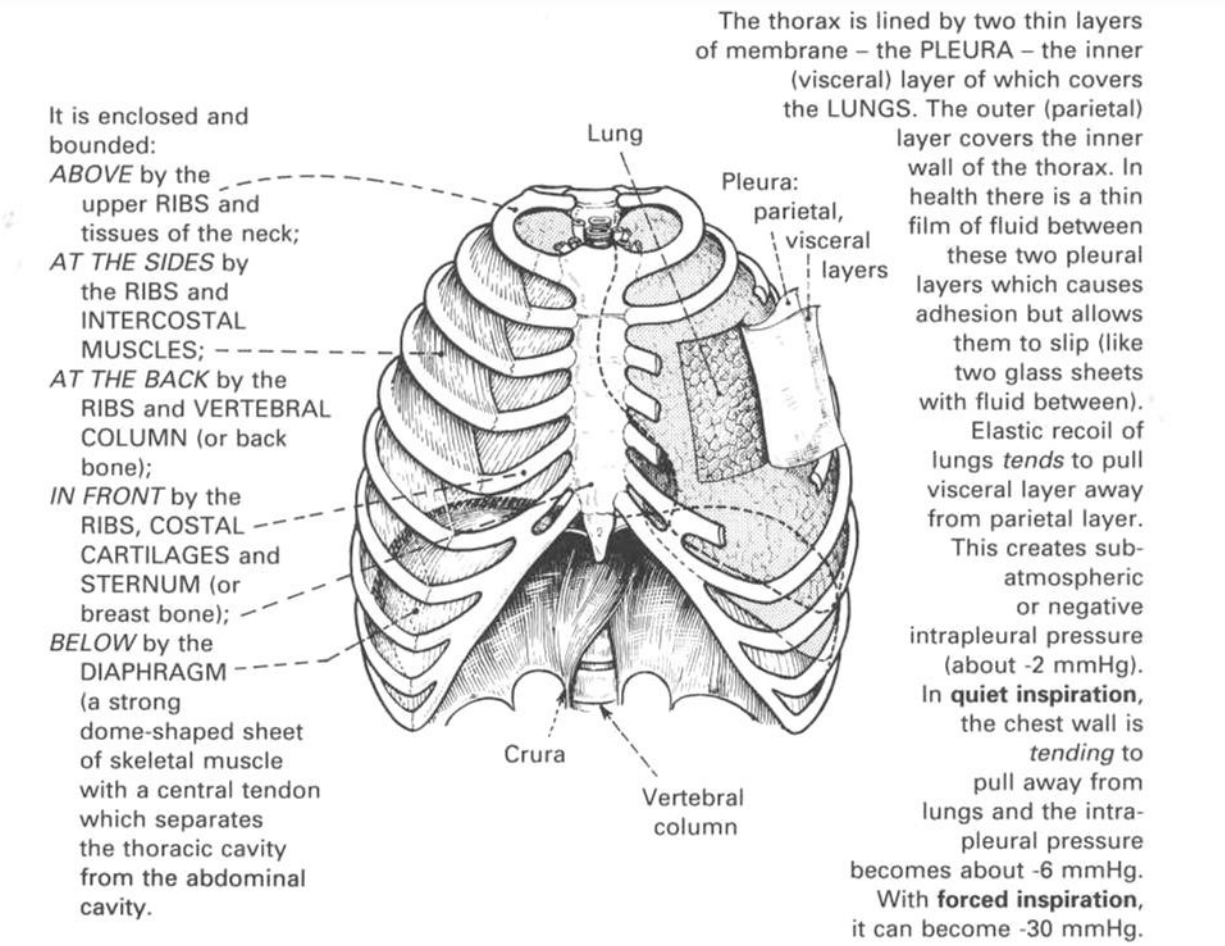
Lungs: Respiratory Surfaces

The trachea and the bronchial 'tree' conduct air down to the **respiratory surfaces**. There is no exchange of gases in these tubes.



Thorax

The thorax is the closed cavity which contains the lung, heart, and great vessels.



NB: A negative pressure is a pressure *below* atmospheric pressure (approx. 760 mmHg). A positive pressure is *above* atmospheric pressure.

Capacity of thoracic cage and the **pressure** between pleural surfaces change rhythmically about 12–14 times a minute with the **movements** of **respiration** – air movement in and out of the lungs follows the dimension changes.

The rhythmical changes in the capacity of the thorax are brought about by the action of skeletal muscles. The changes in the lung volume which intake or expulsion of air, discussed here-

In NORMAL QUIET BREATHING

INSPIRATION

external intercostal muscles actively contract
- ribs and sternum move upwards and outwards because first rib is fixed
- width of chest increases from side to side and depth from front to back increases.

diaphragm contracts
- descends
- length of chest increases.

capacity of **thorax** is *increased*

↓

pressure between **pleural surfaces** (already negative) becomes more negative: from -2 to -6 mmHg (i.e. an increased 'suction pull' is exerted on **lung tissue**)

↓

elastic tissue of lungs is *stretched*

↓

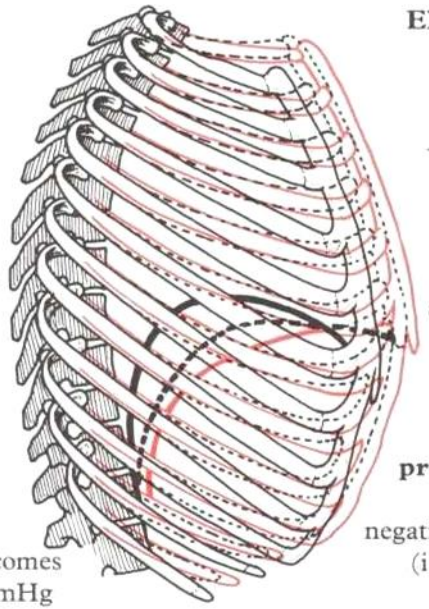
lungs *expand* to fill **thoracic cavity**

↓

air pressure in alveoli is now -1.5 mmHg, i.e. *less* than atmospheric pressure

↓

air is sucked into **alveoli** from atmosphere because of pressure difference.



EXPIRATION

external intercostal muscles relax
- ribs and sternum move downwards and inwards
- width and depth of chest diminishes.

diaphragm relaxes – ascends – length of chest diminishes.

capacity of **thorax** is *decreased*

↓

pressure between **pleural** surfaces becomes less negative: from -6 to -2 mmHg (i.e. less pull is exerted on **lung tissue**)

↓

elastic tissue of lungs *recoils*

↓

air pressure in alveoli is now +1.5 mmHg.
i.e. *greater* than atmospheric pressure

↓

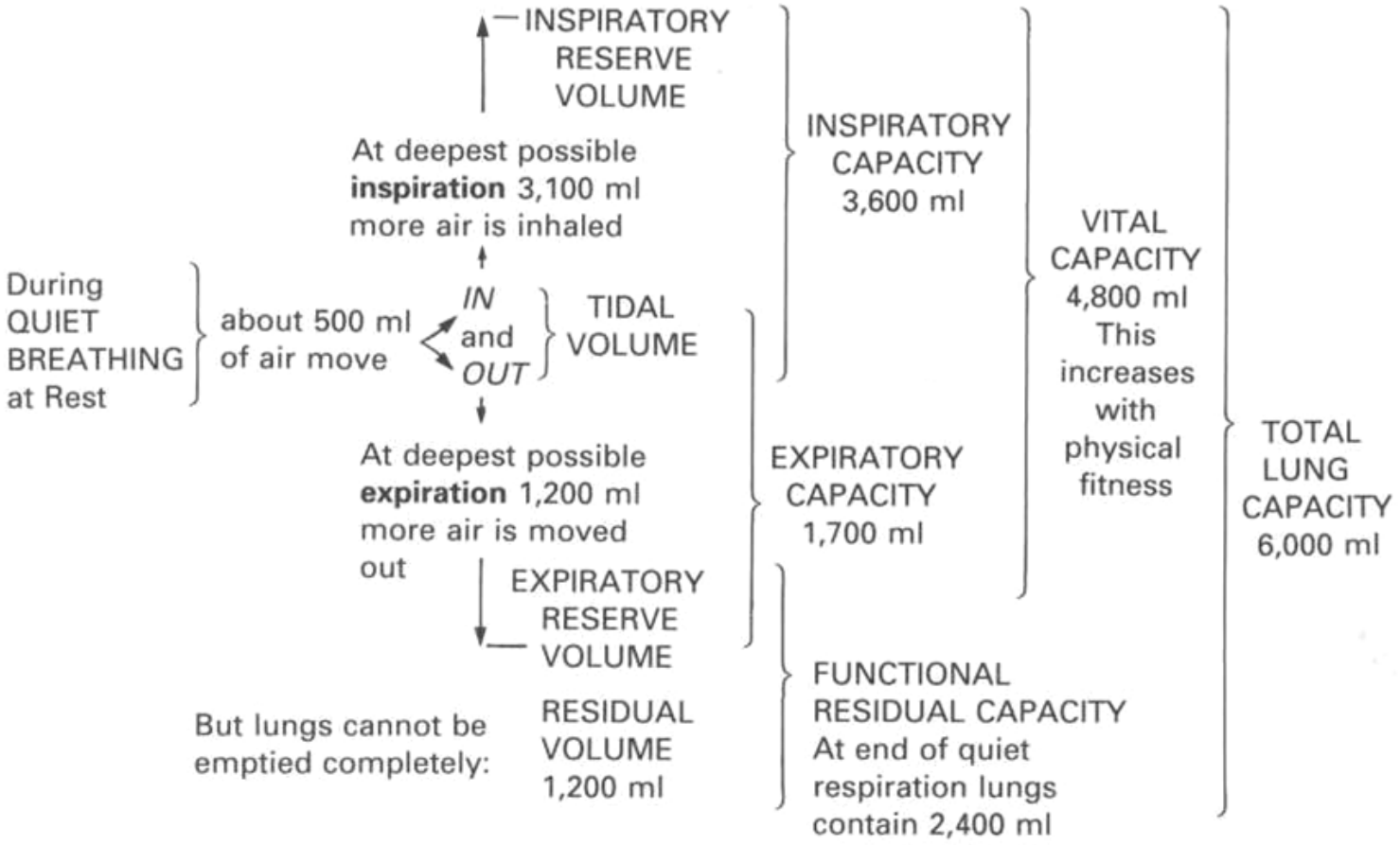
air is forced out of **alveoli** to atmosphere

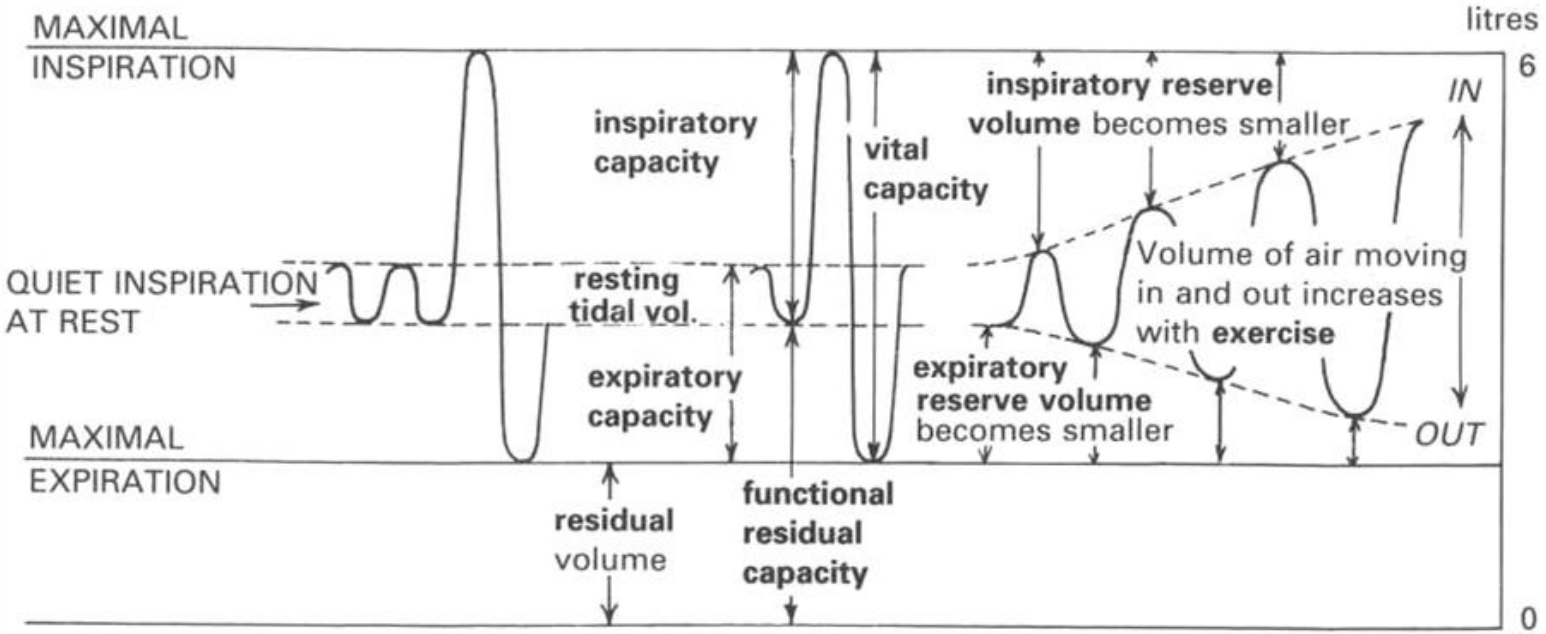
In FORCED BREATHING

Muscles of nostrils and round glottis may contract to aid entrance of air to lungs.
Extensors of vertebral column may aid inspiration.
Muscles of neck contract – move 1st rib upwards (and sternum upwards and forwards).

Internal intercostal may contract – move ribs downwards more actively.
Abdominal muscles contract – actively aid ascent of diaphragm.

Lung volumes and capacities





(after Pappenheimer, J.R., et al (1950) Fed. Proc., **9**,602). *Not to scale*

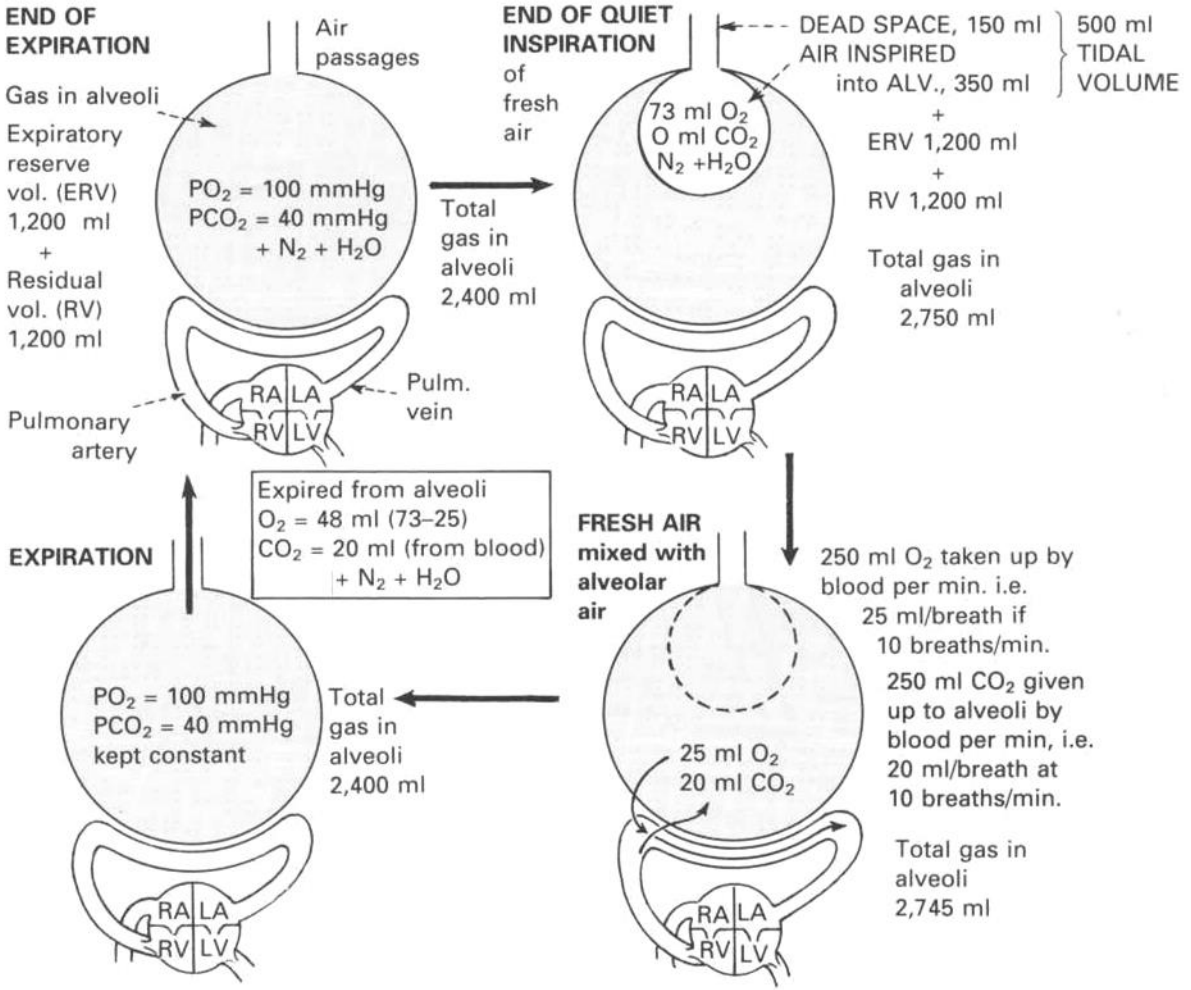
Values for volumes and capacities are typical values but will vary with the subject's size and weight. Values are usually about 25% less in women.

At rest a normal male adult breathes in and out about 12 times per minute. The amount of air breathed in per minute is therefore 500 ml × 12 i.e. 6000 ml or 6 litres – this is the **respiratory minute volume** or **pulmonary ventilation**. In exercise it may go up to as much as 200 litres.

In deep breathing the volume of **atmospheric air inspired** with each inspiration and the amount which reaches the **alveoli** increase.

Alveolar ventilation and Dead Space

At rest, with each breath, we breath in about 500 ml of fresh atmospheric air (the tidal volume). Of this volume 350 ml mix with air already in the lung alveoli and 150 ml occupy the air passages (anatomical dead space) and do not take part in exchange with gases in the blood.



Although shown in stages, the process is continuous.
In this case, **dead space ventilation** = 150 × 10 = 1,500 ml/minute.
Alveolar ventilation = 350 × 10 = 3,500 ml/minute.
Total ventilation = 500 × 10 = 5,000 ml/minute.
For simplicity, the CO_2 in 350 ml of *atmospheric air* which would be 0.14 ml has been called 0 ml and the N_2 which would be approximately 276 ml has not been quantified, nor has the water output.

Composition of respired air

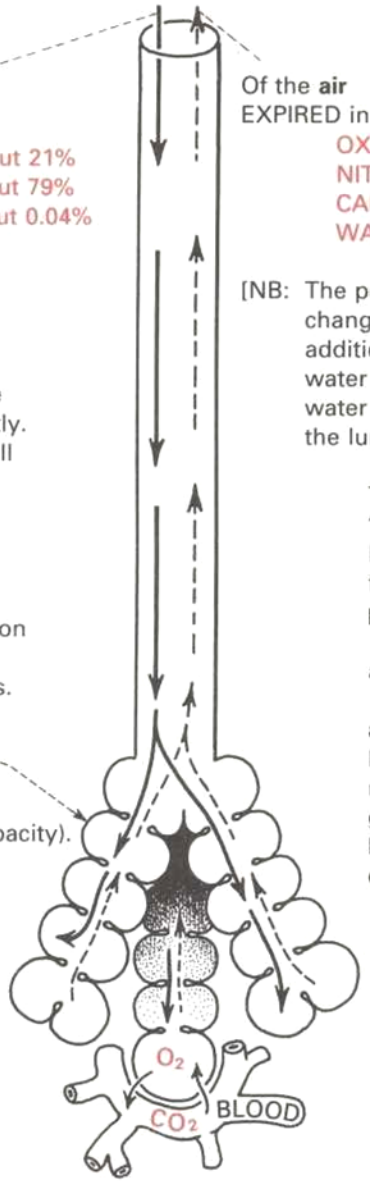
In QUIET BREATHING
Of the
500 ml **atmospheric air**
INSPIRED in a single inspiration

OXYGEN makes up about 21%
NITROGEN about 79%
CARBON DIOXIDE about 0.04%

In most climates, water vapour in air will reduce these percentages slightly. There are also very small amounts of inert gases.

150 ml occupy the **conducting passages** – **'dead space' air**. This remains unchanged in composition since it is not in contact with respiratory surfaces.

350 ml reach the **respiratory units** and mix with 2.4 litres alveolar air (Functional Residual Capacity). Alveolar air is saturated with WATER VAPOUR. It constantly gives up OXYGEN *to* the blood, and constantly takes up CARBON DIOXIDE *from* the blood.



Of the **air**
EXPIRED in a single expiration

OXYGEN makes up 15.7%
NITROGEN 74.5%
CARBON DIOXIDE 3.6%
WATER VAPOUR 6.2%

[NB: The percentage of nitrogen is changed because it is diluted by the addition of other gases, especially water vapour. Air is saturated with water vapour by the time it reaches the lungs.]

This represents a mixture of:
'dead space' air – air which has moved out *unchanged* from the **conducting passages**

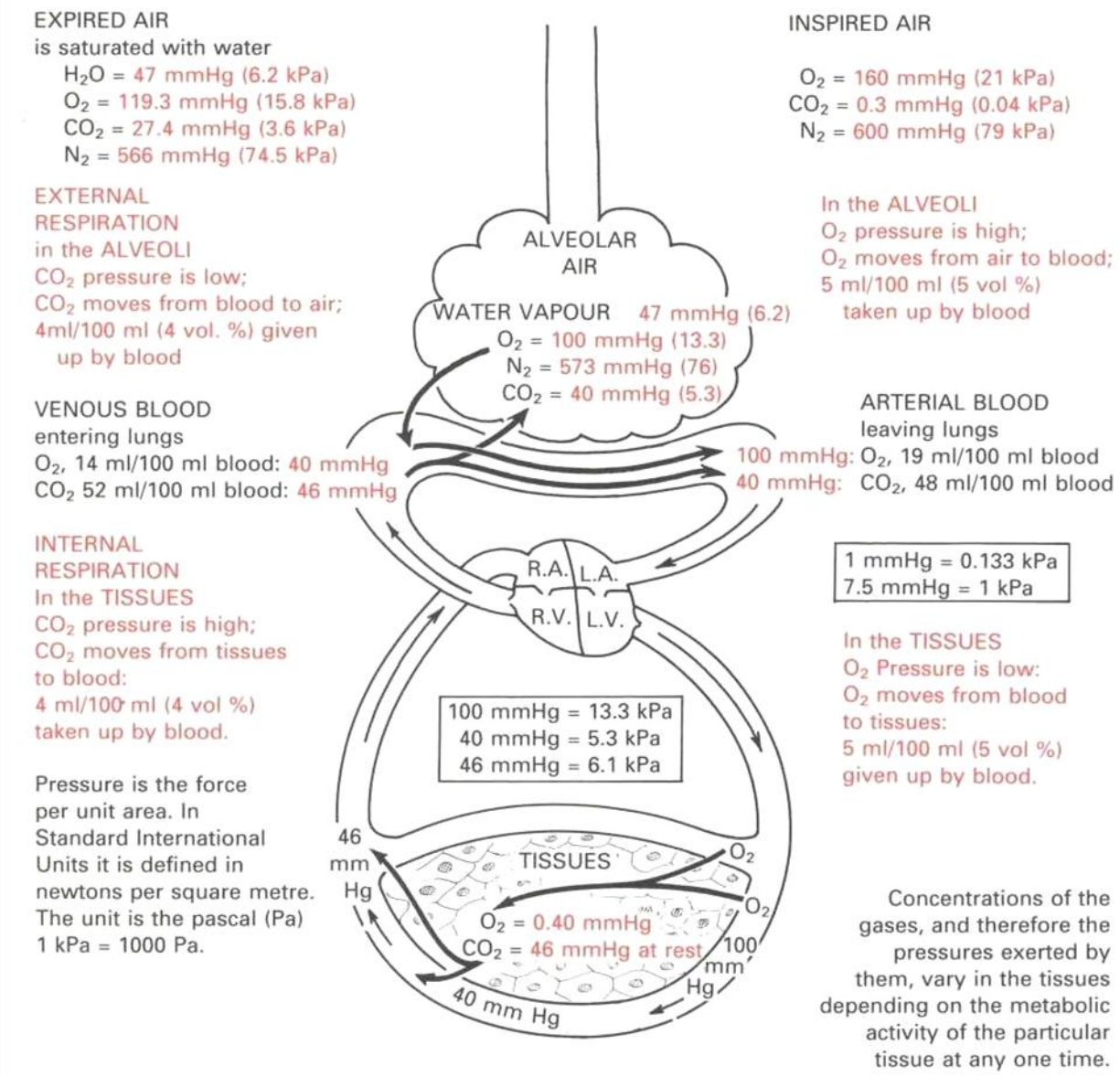
and

alveolar air – air which has been in contact with respiratory surfaces and has given up some **oxygen** *to* the blood and taken up **carbon dioxide** *from* it.

OXYGEN 13.6%
NITROGEN 74.9%
CARBON DIOXIDE 5.3%
WATER VAPOUR 6.2%

In **VOLUNTARY DEEP BREATHING** at rest (hyperventilating) more new air exchanges with the alveolar air. Thus O_2 content of alveolar air will increase and the CO_2 content will decrease.

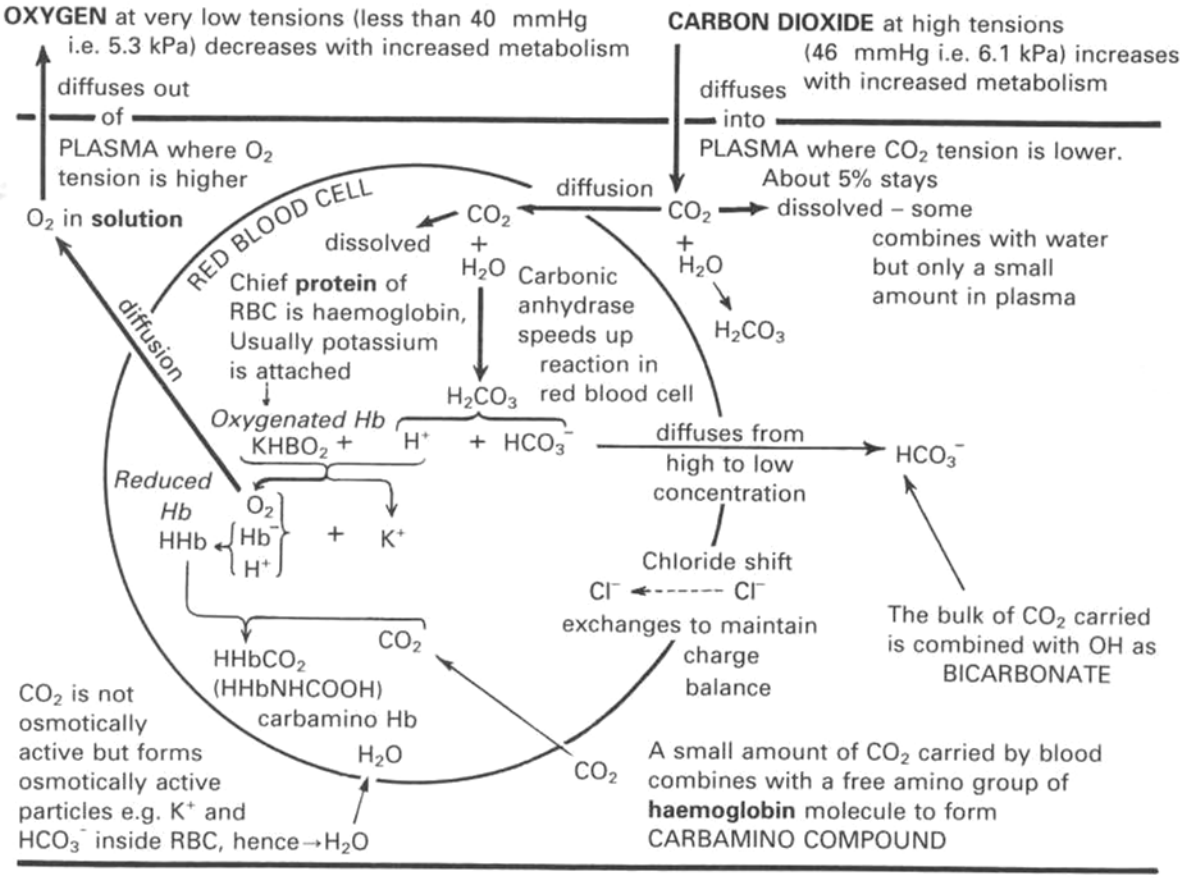
Movement of respiratory gases



A gas moves from an area where it is present at higher pressure to an area where it is present at lower pressure. The movement of gas molecules continues till the pressure exerted by them is the same throughout both areas. Dry atmospheric air has a pressure of 1 atmosphere = 760 mmHg = 101.3 kilopascals (kPa)

Carriage and transfer of O_2 and CO_2

When arterial blood is delivered by systemic capillaries to the tissues it is exposed to-



During its passage through the tissues, each 100 ml of blood gives up about 5 ml of oxygen, i.e. its Hb is still up to 70% O_2 saturated.

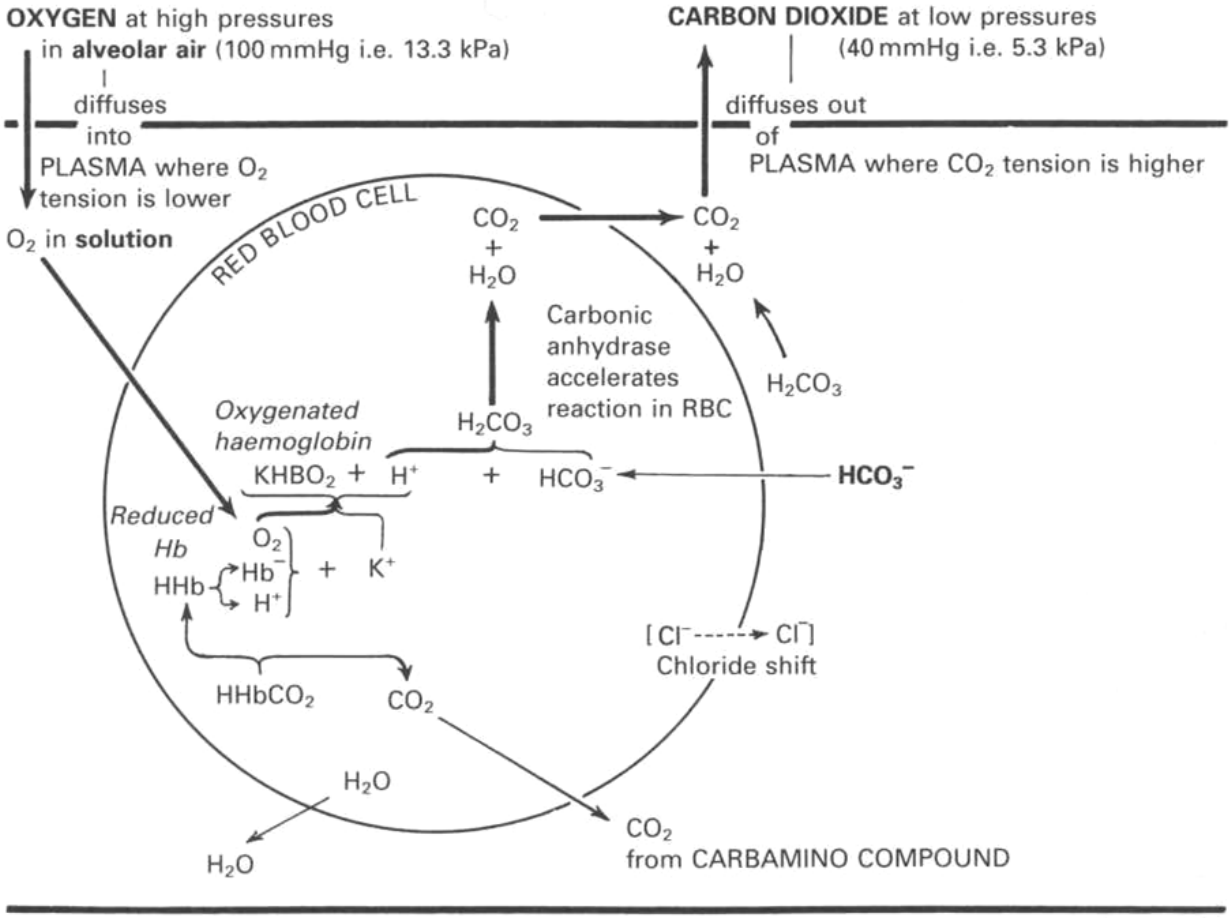
The release of O_2 to tissues is speeded up by an increase in temperature, acidity or DPG such as occurs when tissues are active.

During its passage through the tissues, each 100 ml of blood takes up about 4 ml CO_2.

CO_2 is carried, 5% in solution, 5% as carbamino compounds and 90% as HCO_3^-.

Carbonic anhydrase causes rapid formation of HCO_3^- inside the RBC and it then diffuses down a concentration gradient into the plasma.

When venous blood flows through the pulmonary capillaries it is exposed to-



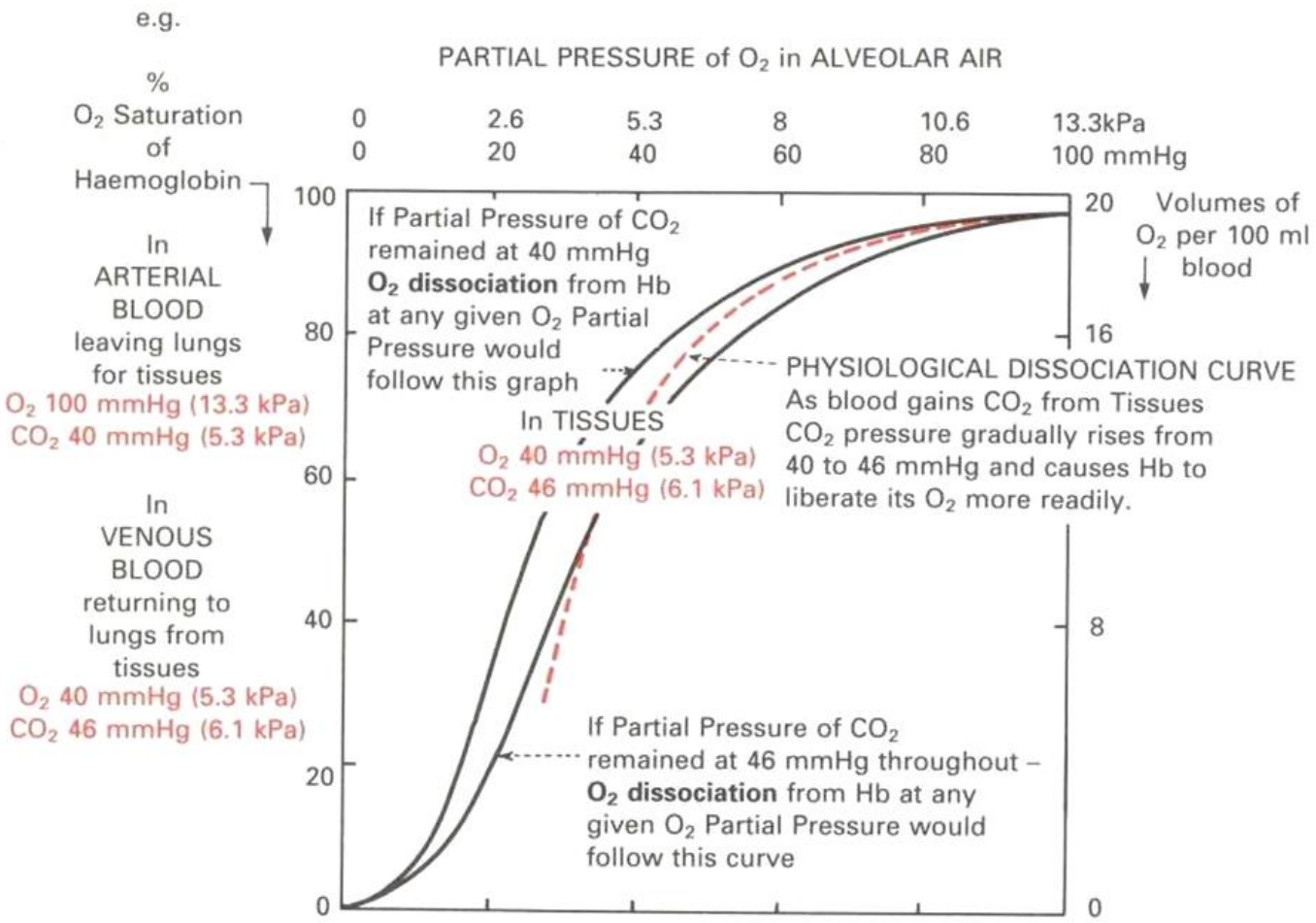
As blood passes through capillaries of lungs, 100 ml take up approximately 5 ml of **oxygen**. O_2 combines with haemoglobin (Hb) molecule. It becomes about 95–97% saturated with oxygen.

As blood passes through capillaries of lungs, each 100 ml blood gives up approximately 4 ml of carbon dioxide. A small amount is released from combination with the free amino group in haemoglobin molecule – carbamino compound. Most comes from bicarbonate in RBC and plasma by processes indicated in diagram.

Dissociation of Oxygen from haemoglobin

The amount of O_2 taken up by **haemoglobin** in the **lungs** or given up by **oxyhaemoglobin** in the **tissues** depends on the **partial pressure** of the O_2 in the immediate environment.

It is also influenced by the **partial pressure** of CO_2, by **temperature**, by **acidity** and by the concentration of 2,3-diphosphoglycerate (DPG) [or 2,3-biphosphoglycerate (BPG)].



This effect of CO_2 partial pressure on dissociation of O_2 from Hb (the Böhr effect) is advantageous, e.g. an increase in CO_2 partial pressure locally during tissue activity causes Hb to part more readily with its O_2 to the active tissues.

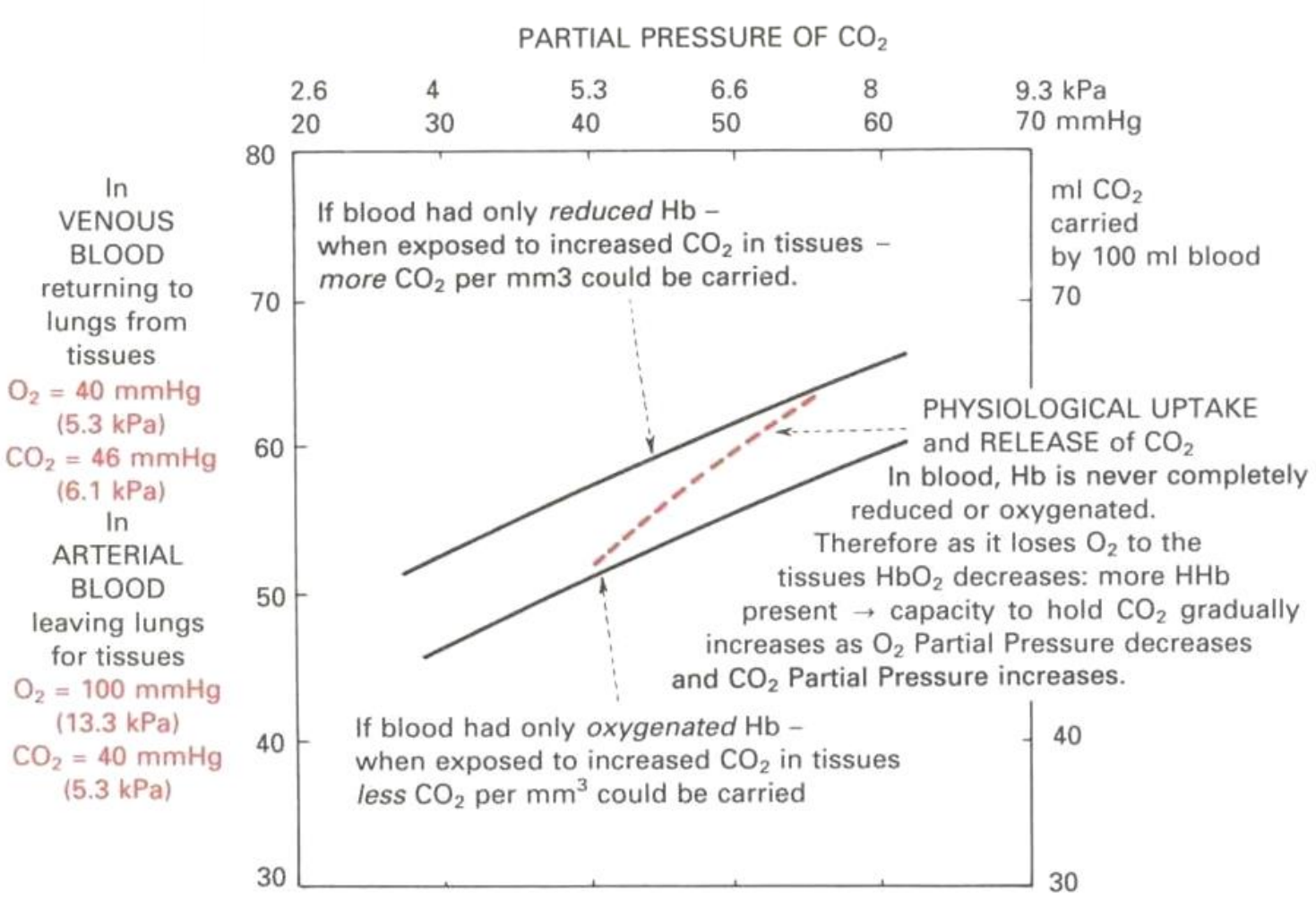
Similarly, an increase in temperature, H^+ and DPG move the curve to the right. DPG is formed when glucose is broken down for energy (glycolysis) in RBCs. Its presence favours the dissociation of oxygen from HbO_2. Thyroxine, human growth hormone and testosterone increase DPG formation. It is higher also in people living at high altitude. Fetal haemoglobin has a higher affinity for O_2 than maternal haemoglobin because it binds DPG less strongly.

Uptake and release of carbon dioxide

CO_2 is carried by the blood in 3 forms: (a) about 90% is carried as **bicarbonate formed** chiefly in the RBCs and **carried** largely by plasma, (b) about 5% is carried **dissolved** in blood water, and (c) about 5% is carried combined to the terminal amino groups of blood proteins as carbamino compounds. Especially important is the globin of haemoglobin.

The Haldane effect

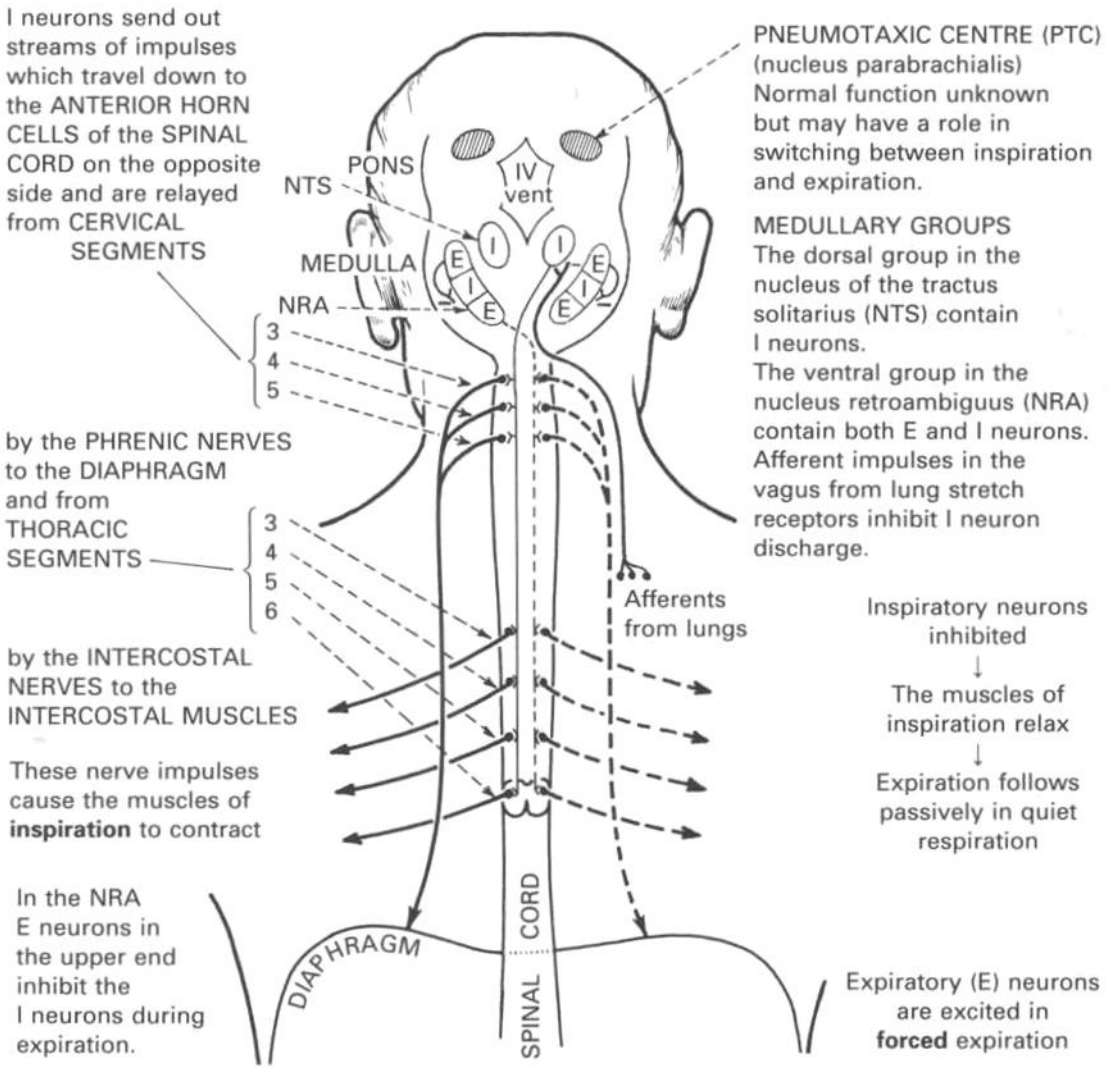
The presence of reduced Hb in the peripheral blood helps with the loading of CO_2 into the blood from the tissues. The oxygenation which occurs in the pulmonary capillaries helps with the off-loading of CO_2 from the blood into the alveoli. The fact that the deoxygenation of the blood increases its ability to carry CO_2 is known as the Haldane effect. The explanation for this is that reduced Hb has a better ability to mop up H^+ produced when carbonic acid dissociates in the reaction $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$, hence driving the reaction to the right. In addition, reduced Hb can bind much more CO_2 than HbO_2.



i.e the more **oxygen** the blood holds the less **CO_2** it can hold and vice versa. This facilitates uptake of CO_2 in tissues and release of CO_2 in the lungs.

Nervous control of respiratory movements

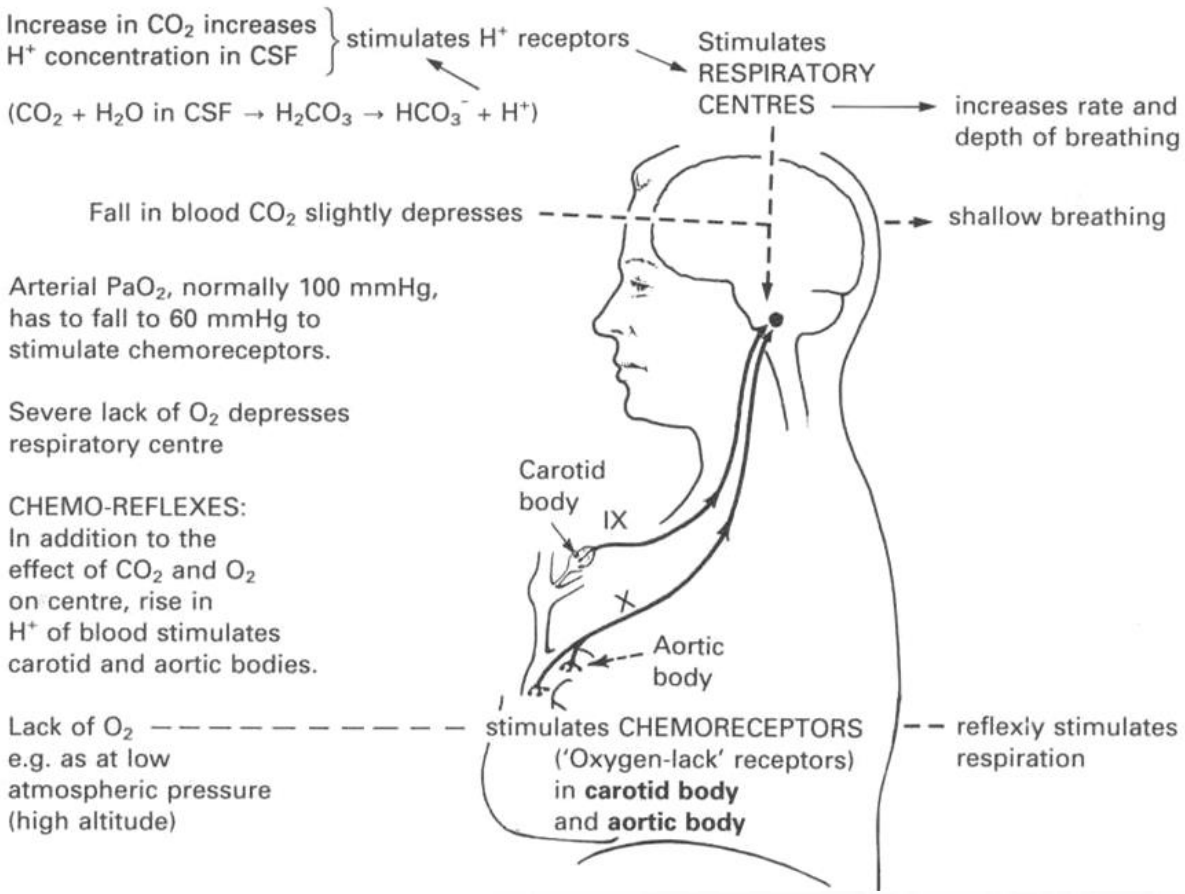
Normal respiratory movements are involuntary. They are carried out automatically (i.e. without conscious control) through the rhythmical discharge of nerve impulses from **controlling centres** in the medulla oblongata and pons. Respiratory neurons in the brainstem are of two types: **I neurons** discharge during **inspiration**; **E neurons** discharge during **expiration**.



Despite intensive research, the mechanism responsible for rhythmic respiratory discharge remains unsettled. The main components are in the medulla where there may be a group of pacemaker neurons situated.

Chemical regulation of respiration

The activity of the respiratory centres is regulated by the O_2, CO_2 and H^+ content of the blood. **Carbon dioxide** and **H^+** are the most important. CO_2 dissolves in cerebrospinal fluid (CSF) which bathes receptors sensitive to H^+ on the ventral aspect of the medulla. Stimulation of these receptors is responsible for about 70% of the increase in the rate and depth of respiration in response to increased CO_2. Carotid and aortic bodies are responsible for the other 30% of the response to raised CO_2. They also increase ventilation in response to a rise in H^+ or a large drop in PaO_2 (to below 60 mmHg).



Note:– These reflexes are usually powerful enough to override the direct depressant action of lack of O_2 on respiratory centres themselves

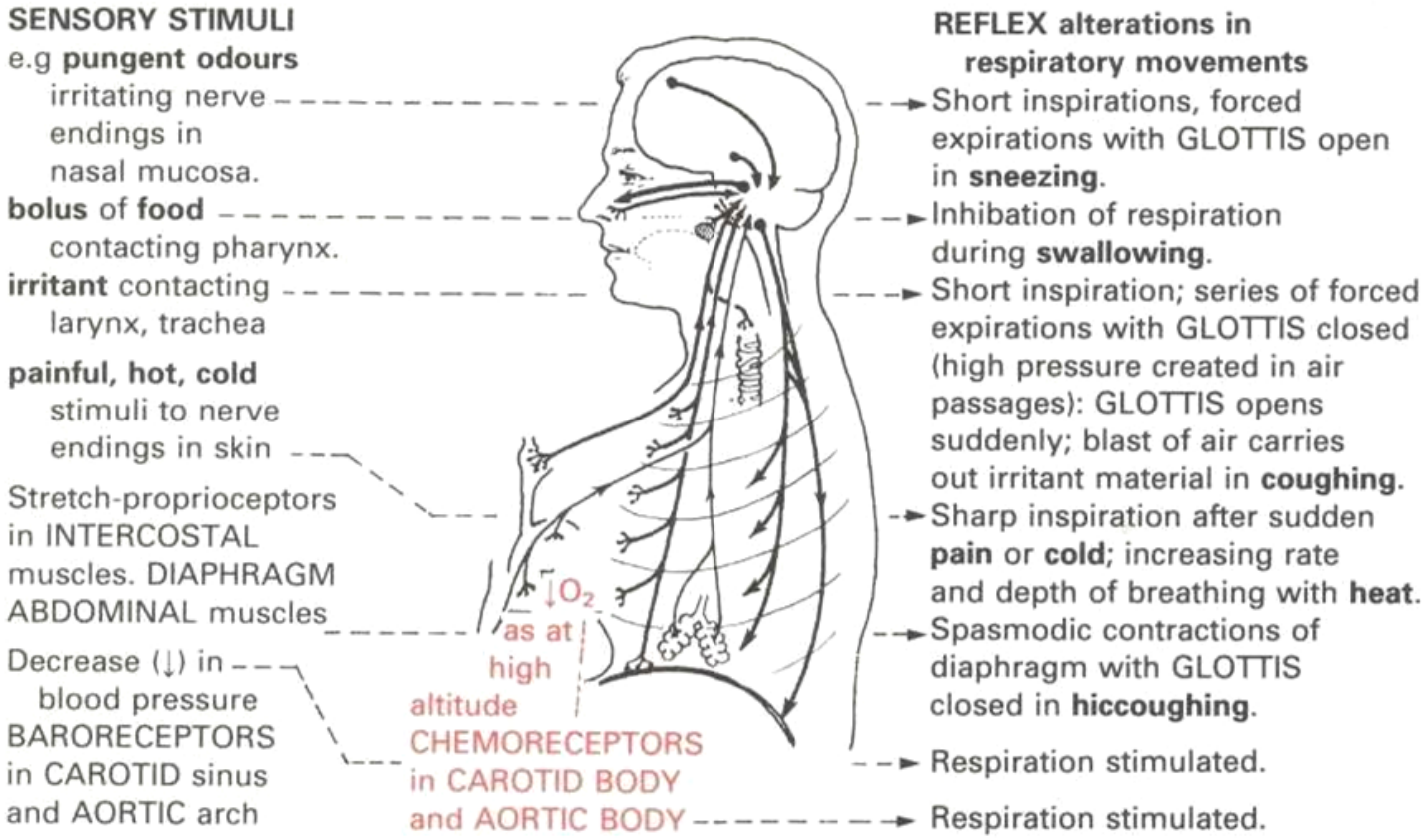
The **chemical** and **nervous** means of regulating the activity of **respiratory centres** act together to adjust rate and depth of breathing to keep the $PaCO_2$ close to 40 mmHg. This automatically sets the PaO_2 to an appropriate value depending on the partial pressure of O_2. For example, exercise causes increased requirement for O_2 and the production of more CO_2. Ventilation is increased to get rid of the extra CO_2 and keep the alveolar $PaCO_2$ at 40 mmHg. More oxygen is used by the tissues. The alveolar PO_2 and PCO_2 both remain constant

Voluntary and reflex factors in the regulation of respiration

Although fundamentally automatic and regulated by chemical factors in the blood there is a separate voluntary system for the regulation of ventilation. It originates in the cerebral cortex and sends impulses to the nerves of the respiratory muscles via the corticospinal tracts. In addition, ingoing impulses from many parts of the body modify the activity of the **respiratory centres** and consequently alter the outgoing impulses to the respiratory muscles to coordinate **rhythm, rate** or **depth** of breathing with other activities of the body.

Impulses from HIGHER CENTRES – PSYCHIC and EMOTIONAL INFLUENCES

- Voluntary alterations in breathing.
- Interruptions of expiration in **speech** and **singing**.
- Deep inspiration then short spasmodic expirations in **laughter** and **weeping**.
- Prolonged expiration in **sighing**.
- Deep inspiration with mouth open in **yawning**.
- Slow shallow breathing in **suspense** and **concentration**.
- Rapid breathing in **fear** and **excitement**.



Proprioceptors stimulated during muscle movements send impulses to respiratory centre → ↑ rate and depth of breathing. (NB: This occurs with active or passive movements of limbs.)

In normal breathing respiratory rate and rhythm are thought to be influenced rhythmically by the **Hering-Breuer reflex**.

Distension of alveoli at end of inspiration → stimulates stretch receptors in bronchioles → Stream of ingoing impulses passes along vagus nerves to inhibit inspiratory centres → Withdrawal of outgoing impulses to respiratory muscles → expiration