CHAPTER 4: Effects of exercise – responses and adaptations of the body systems – cardio-vascular and respiratory systems

Introduction to the structure of the heart

STUDENT NOTE
Prior knowledge of the structure and function of the cardio-vascular system is assumed, so we include elements of this here.

Heart structure
The heart (figure 4.1) is a muscular pump lying deep within the chest cavity and slightly to the left of the sternum.

Heart layers
The heart consists of three layers:
- The outer layer, known as the pericardium, is a double layered bag surrounding the heart. The fluid between the two layers reduces friction between the heart itself and the surrounding tissue as the heart moves (beats). This layer also maintains the heart’s shape.
- The second layer is called the myocardium or striped cardiac muscle tissue consisting of united fibres (united because they are all in one mass) joined by intercalated discs. This muscle tissue is activated by the ‘all-or-none law’. The cardiac impulse is transmitted throughout the entire myocardium at the same point in time, and hence this muscle tissue is all activated at once. When there is no cardiac impulse, none of the heart muscle can be activated. Since the heart generates its own impulse it is said to be myogenic. The septum consists of myocardial tissue (muscle) and divides the heart into two sections, each of which acts as a pump.
- The third layer is an inner glistening membrane called the endocardium. Its function is to prevent friction between heart muscle and flowing blood.

Heart chambers
The heart consists of four chambers:
- Two are at the top (atria). Both the right and left atria have thin walls.
- Two are at the bottom (ventricles). Both ventricles have thicker walls than the atria. The left ventricle wall is the thickest, since this ventricle pumps blood to the main body mass, whereas the right ventricle pumps blood to the lungs only.

Heart valves
Heart valves prevent back-flow of blood, with the (cuspid) mitral or bicuspid valve sited between the left atrium and the left ventricle, and the tricuspid valve sited between the right atrium and the right ventricle. The semi-lunar valves prevent back-flow of blood into the heart from the pulmonary artery and aorta, and only allow blood to flow in one direction through the heart. This means that when the heart muscle contracts, it only pumps the blood out to the lungs (pulmonary artery) or body (aorta), and not back the wrong way.

Blood vessels
Blood vessels attached to the heart are the venae cavae and the pulmonary artery on the right side, and the pulmonary veins and the aorta on the left side.

Coronary blood supply
The coronary blood supply consists of arteries (within the cardiac muscle itself) which supply glucose and oxygen (O\textsubscript{2}) to myocardial tissue, and coronary veins, which transport carbon dioxide (CO\textsubscript{2}) and other wastes from the heart muscle.
Cardiac responses to physical activity

How the heart works

The cardiac impulse

- The dynamic action of the heart (figure 4.2) is that of a dual-action pump in that both sides of the heart contract simultaneously, even though the functions of the two sides are different.
- Cardiac contractions are initiated by an electrical impulse (the cardiac impulse) that originates from the pacemaker or sinoatrial node (SA node). Because the heart generates its own impulses it is said to be myogenic.
- The electrical impulse travels down the atrial mycardium until it reaches the atrioventricular node (AV node) situated in the wall of the atrial septum. This is followed by the atrial walls contracting (atrial systole).
- The AV node conducts the impulse through the bundle of His to the branched network of Purkinje fibres located within the septum and the ventricular walls (both the bundle of His and the Purkinje fibres are modified cardiac muscle), causing both ventricles to contract (ventricular systole).

The heart’s conducting system regulates the sequence of events that make up the cardiac cycle.

The cardiac cycle

The cardiac cycle (figure 4.3) is a sequence of events that make up one heart beat and lasts for about 0.8 seconds, thus occurring about 75 times per minute.

The cardiac cycle consists of a period of relaxation of the heart muscle, known as diastole (0.5 seconds), followed by a period of contraction of the heart muscle, known as systole (0.3 seconds). During systole the electrical impulse is initiated in a set-timed sequence.

Cardiac diastole

During diastole (0.5 seconds), the relaxed heart muscle allows the chambers to fill with blood. This occurs with the cuspid valves open, and the semi-lunar valves closed.

Cardiac systole

During atrial systole (0.3 seconds), the SA node impulse causes a wave-like contraction over the atria forcing blood past the cuspid valves into the ventricles. The semi-lunar valves remain closed.

In ventricular systole, the impulse reaches the AV node, the cuspid valves close because the fluid pressure (of blood) in the ventricles is greater than in the atria, and rises further as the ventricles contract. The semi-lunar valves open (since now the fluid pressure in the ventricles is greater than in the main arteries) and blood is pushed out into the pulmonary artery (towards the lungs) and the aorta (around the body).

The pulse is a wave of pressure produced by the contraction of the left ventricle. This pressure wave transmits itself around the arterial system of the rest of the body. The frequency of the waves represents the number of beats per minute (heart rate).
Short-term cardiac responses to physical activity

See figure 4.4 for a summary of cardiac factors in short-term responses to exercise.

- **Heart rate** (HR) is defined as ‘the number of beats of the heart per minute (bpm)’.
- The average resting HR for males is 70 bpm, and for females 72 bpm.
- At rest, the HR for a trained athlete = 60 bpm or lower (heart rates of less than 60 is bradycardia), and the HR for an untrained person = 70-90 bpm (average 80).
- **Maximum heart rate** can be calculated using the formula: $HR_{\text{max}} = 220 - \text{age}$.
  So, for a 20 year old, $HR_{\text{max}} = 220 - 20 = 200$ bpm.
  Usually, maximum heart rates for untrained people are slightly less than for highly trained people, an example would be 190 (untrained) to 200 (trained) in the calculation outlined below.
- **Stroke volume** (SV) is ‘the volume of blood pumped by the left ventricle of the heart per beat’ and is determined by venous return and elasticity and contractility of the myocardium.
- For example, the SV for a trained athlete = 110 ml at rest, and the SV for an untrained person = 70 ml at rest.
  The SV for a trained endurance athlete during maximal exercise intensity = 190 ml, and for an untrained person, during his / her maximum exercise intensity SV = 110 ml.
- **Cardiac output** ($Q$) is ‘the volume of blood pumped by the left ventricle of the heart in one minute’, and is the product of stroke volume and heart rate: $Q = SV \times HR$
  Example figures are:
  - For an untrained person at rest, $Q = 70 \times 80 = 5.60 \text{ l/min (or dm}^3 \text{ min}^{-1})$.
  - For an untrained person during maximal exercise, $Q = 110 \times 190 = 20.90 \text{ l/min (or dm}^3 \text{ min}^{-1})$.
  - For an endurance athlete at rest, $Q = 110 \times 51 = 5.61 \text{ l/min (or dm}^3 \text{ min}^{-1})$.
  - For an endurance athlete during maximal exercise, $Q = 190 \times 200 = 38 \text{ l/min (or dm}^3 \text{ min}^{-1})$.

Short-term changes in heart rate, stroke volume and cardiac output during different intensities of physical activity

Heart rate response to exercise

Referring to the graph in figure 4.5:

- **a** = Anticipatory rise due to the hormonal action of adrenaline and noradrenaline. This happens because the person tends to get excited before the exercise starts, and hence heart rate rises slightly.
- **b** = Sharp rise during anaerobic work due to proprioceptor sensory stimulation, and also due to continued release of hormones and action of the skeletal muscle pump (see page 55).
- **c** = Steady state and some recovery of $O_2$ debt (aerobic).
- **d** = Continued high HR due to maximal workloads which continue to stress anaerobic systems, producing lactic acid + $CO_2 + K^+$, which stimulate chemoreceptors. Additionally, intrinsic factors are also stimulated at maximal level (refer to page 52).
- **e** = Rapid recovery due to cessation of proprioceptive stimuli, the skeletal muscle pump, and the withdrawal of hormones.
- **f** = Slow recovery, clearance of metabolites such as lactic acid, as systems return to normal resting values.
Stroke volume response to exercise

Referring to the graph in figure 4.6:

a = An increase in stroke volume, from a resting value of 60 ml beat\(^{-1}\) to 85 ml beat\(^{-1}\) prior to the start of the exercise period, and is due to the release of hormones such as adrenaline and noradrenaline. This effect is known as the anticipatory rise.

b = An increase in stroke volume as exercise commences. This is primarily due to an increased venous return and increased myocardial contraction during ventricular systole (Starling’s Law of the Heart) which causes the heart muscle to contract more forcefully, from 85 ml beat\(^{-1}\) to more than 110 ml beat\(^{-1}\) during submaximal work.

c = As work intensity increases during maximal exercise, there is a slight decline in stroke volume. At this point heart rate will rise rapidly to sustain the continued increase in cardiac output to meet exercise demands.

Cardiac output response to exercise

Since cardiac output is the product of stroke volume and heart rate values \(Q = SV \times HR\), it will increase directly in line with exercise intensity.

Cardio-vascular drift

- With prolonged aerobic exercise, at a constant exercise intensity, such as marathon racing or aerobic exercising in a hot environment, stroke volume gradually decreases and heart rate increases, and hence cardiac output remains approximately constant. During this process arterial blood pressure declines.
- These responses are due to the need to transfer excess heat produced by active tissues from deep in the body (known as the core) to the skin where it has access to the outside environment.
- This heat is moved by the blood during vasodilation of blood vessels directly underneath the skin. Evaporation is the primary route for heat dissipation and so as fluid or sweat evaporates heat is lost. Loss of fluid results in a reduced plasma volume and subsequent decreased venous return and stroke volume.
- A reduced stroke volume initiates a compensatory heart rate increase to maintain a nearly constant cardiac output as exercise progresses. All these circulatory responses are collectively referred to as the cardio-vascular drift. See figure 4.7.
- It is important for athletes to rehydrate with sports drinks (water containing a little sodium and glucose) during prolonged exercise periods or whilst performing aerobic exercise in a hot environment. This will minimise the loss of fluids and thus reduce the effects of the cardio-vascular drift.
Regulation of heart rate

The cardiac control centre (in the medulla oblongata in the brain) regulates feedback that results in changes to heart rate from important neural, hormonal and intrinsic factors (figure 4.8).

**Neural control factors**

Neural control factors are the key controlling regulators and consist of:

- **Chemoreceptor reflexes** which involve receptors located in blood vessels such as the aortic arch and carotid sinuses. These reflexes detect chemical changes in blood $O_2$, $CO_2$, $H^+$ concentrations, and pH levels. Decrease in $O_2$ and pH levels, and increase in $CO_2$ and $H^+$ concentrations, all stimulate changes in heart rate via the cardiac accelerator nerve.

- **Proprioceptor reflexes** found in muscle spindles and Golgi tendons which respond to mechanical stimuli such as compression, bending or stretching of cells, detecting changes in movement. Increase in tension within cell structures will increase heart rate via the cardiac accelerator nerve.

- **Baroreceptor reflexes** which involve receptors located in blood vessels (such as the aortic arch and carotid sinuses). Their role is to detect changes in blood pressure. When blood pressure is too high the parasympathetic nerve releases acetylcholine, which decreases heart rate.

**Hormonal factors**

Hormones are released by the body in response to various stimuli. Those that affect heart rate are:

- **Noradrenaline** and **adrenaline** (the key hormonal regulators) which act to accelerate heart rate (tachycardia) and increase the strength of ventricular contraction which increases stroke volume.

- **Acetylcholine** which slows the heart (bradycardia) as described below.

- **Thyroid** hormone and **glucagon** which increase HR.

- **Increased glucagon** levels which assist in the breakdown of **glycogen to release glucose** into the circulatory system to fuel muscular contractions.

**Intrinsic factors**

Intrinsic factors account for changes in venous return:

- **Venous return** is *the volume of blood returning to the heart during each cardiac cycle*. This changes as a result of the actions of the skeletal muscle and respiratory pumps, and the electrolyte balance (Na$^+$, K$^+$) in muscular tissue.

- **Myocardial temperature** also affects venous return, in that the speed of nerve impulse transmission increases with temperature, and this will increase heart rate.

- **Starling’s Law of the Heart** states that cardiac output is equal to venous return. An increase in venous return stretches the ventricular walls more and results in an increased strength of contraction and therefore an increase in stroke volume.
Neural control

- **Neural impulses** (resulting from feedback from neural, hormonal and intrinsic control) override the inherent rhythm of the heartbeat. Signals originate in the cardiac control centre (CCC) in the medulla and travel via the antagonistic actions of the sympathetic and parasympathetic nervous systems, to the pacemaker or SA node.

**Sympathetic influence**

- The sympathetic nervous system, the SNS (via the cardiac accelerator nerve), releases the neurotransmitters adrenaline and noradrenaline onto the SA node to speed up heart rate.

**Parasympathetic influence**

- The parasympathetic nervous system, PNS (via the vagus nerve), releases the neurotransmitter acetylcholine onto the SA node to slow down heart rate.

Long-term adaptations of the heart and its control system due to aerobic exercise

**Long-term responses of the heart (figure 4.9)**

- Regular aerobic training results in hypertrophy of the cardiac muscle, meaning that the muscle becomes larger and stronger. This means that the heart pumps a larger volume of blood out per beat, hence the stroke volume is larger. This is termed bradycardia and has the consequence of producing a resting HR below 60 bpm. This in turn affects cardiac output, as illustrated in the equations above.

- At rest a bigger and stronger heart pumps more blood out per beat, even though the body’s requirement for oxygenated blood would be approximately the same as for an untrained person. Hence resting heart rate decreases, with the net effect of an unchanged cardiac output. Highly trained sportspeople tend to have resting heart rates of well below 60 bpm.

- During maximum exercise, an increase in heart rate, coupled with an increase in stroke volume, results in an increase in cardiac output. As expected, cardiac output for the endurance athlete is more than double that of the untrained person due to cardiac muscle hypertrophy.

- During the recovery period following maximal exercise, heart rate will decrease more rapidly, and so will return to its resting level much more quickly for the endurance athlete when compared with an untrained person.

- Hence heart rate recovery is used as an index of cardio-respiratory fitness.

**Long-term responses of the nervous control system**

- Endurance training creates an imbalance between the two sets of nerves (the PNS and the SNS) in favour of parasympathetic dominance. This type of training is also known to decrease intrinsic firing rate of the SA node.

- These adaptations account for the significant bradycardia observed amongst highly conditioned endurance athletes.

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**figure 4.9 – long-term adaptations of the heart**

[Diagram showing cardiac muscle hypertrophy, bradycardia, parasympathetic dominance, and equations for cardiac output at rest and during maximal exercise]
Vascular responses to physical activity

The blood circulation systems
There are two systems circulating blood from the heart as in figure 4.10.

The systemic circulatory system
This system consists of all the vessels which carry oxygenated blood away from the heart via the aorta, the arteries and arterioles and on to the capillaries embedded in the working tissues of the body. Then after giving up the oxygen (to the working tissues), the deoxygenated blood returns to the heart via venules, veins and the venae cavae.

The pulmonary circulatory system
This system consists of the pulmonary arteries that carry this deoxygenated blood from the right atrium of the heart to the lungs, where the blood is re-oxygenated from the air breathed into the lungs. Oxygenated blood is then returned to the heart via the pulmonary veins.

Blood vessel structure
Blood vessels (see table 4.1) have properties that help circulation and allow blood to perform many of its functions (see summary in figure 4.11).

Except for single-walled capillaries and venules, all blood vessels have 3 layers. The thickness and composition of the layers vary with blood vessel type and diameter. Smooth involuntary muscle, within the middle layer of blood vessel walls, regulates the diameter of blood vessels via vasomotor and venomotor control (described on page 56).
### Table 4.1 – Blood vessel structure and function

<table>
<thead>
<tr>
<th>Type of Blood Vessel</th>
<th>Vessel Structure</th>
<th>Vessel Function / Structure</th>
<th>Vessel Function</th>
<th>Blood Pressure in Vessels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elastic Arteries (aorta)</td>
<td>Are thin-walled with large diameters</td>
<td>Middle layer (tunica media) contains a high proportion of elastic fibres and little smooth muscle</td>
<td>During ventricular systole, these arteries extend with a rise in left ventricular pressure and recoil (contract) during ventricular diastole</td>
<td>Transport blood at high pressure away from the heart</td>
</tr>
<tr>
<td>Muscular Arteries</td>
<td>Thick-walled vessels with small diameters</td>
<td>Middle layer (tunica media) consists of some elastic fibres and lots of smooth muscle</td>
<td>Smooth muscle controls the shape of the central space or lumen via vasoconstriction and vasodilation</td>
<td>Transport blood at high pressure</td>
</tr>
<tr>
<td>Arterioles</td>
<td>Reduce in size and muscular content as they get closer to the capillary bed</td>
<td>Smooth muscle (in the tunica media)</td>
<td>Smooth muscle contracts (to reduce blood inflow) and relaxes (to increase blood inflow) to control inflow to their own capillary bed</td>
<td>Blood pressure reduces as vessel diameter reduces but total CSA of all vessels increases</td>
</tr>
<tr>
<td>Pre-capillary Sphincters (contained within arterioles)</td>
<td>Placed before capillary bed (within muscle or other tissue)</td>
<td>Contract (to reduce blood inflow) and relax (to increase blood inflow) to control inflow to their own capillary bed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Capillaries</td>
<td>Tiny blood vessels whose walls are one cell thick, have semi-permeable walls or small spaces in the walls (tunica intima)</td>
<td>Walls allow fluids rich in nutrients (O₂ &amp; glucose) to be delivered to tissue cells, nutrients travel through the capillary walls into the tissue cells</td>
<td>Waste products (CO₂ and urea / lactate) are removed by travelling through the capillary walls from the tissue cells into the blood fluids this is the opposite direction to the nutrients</td>
<td>Very low blood pressure as total vessel area reaches a maximum</td>
</tr>
<tr>
<td>Venules</td>
<td>Walls consist of an inner wall (tunica intima), surrounded by a few smooth muscle cells</td>
<td>Positioned where several capillaries unite to collect outflow from a capillary bed at low pressure</td>
<td>As venules approach the veins they develop a thin middle layer coat (tunica media)</td>
<td>Blood pressure still very low as blood is transported towards the heart</td>
</tr>
<tr>
<td>Muscular Veins</td>
<td>Thin walled vessels contain less smooth muscle and fewer elastic fibres than arteries of same size</td>
<td>Have non-return valves, called pocket valves positioned within the central space (or lumen) of these vessels</td>
<td>Sympathetic nerves causing vasoconstriction activate the tunica media. The outer wall (tunica externa) is supported by collagen</td>
<td>Low blood pressure</td>
</tr>
<tr>
<td>Veins</td>
<td>Thin middle layer supported by smooth muscle cells and collagen and elastic fibres</td>
<td>Blood flows in the veins because of muscular action in the surrounding skeletal muscle - skeletal muscle pump</td>
<td>Contracting muscle squashes veins forcing blood forwards towards the heart since blood cannot flow back away from the heart due to the pocket valves within each vein</td>
<td>Low pressure blood reservoirs moving stored blood into general circulation during exercise</td>
</tr>
<tr>
<td>Venae Cavae</td>
<td>Are valveless and contain more smooth muscle in the middle wall</td>
<td>Smooth muscle acts to constrict or dilate the vessel (venomotor control)</td>
<td>Deliver blood to the right atrium of the heart</td>
<td>Low blood pressure</td>
</tr>
</tbody>
</table>

**Student Note**

CSA means cross sectional area, and represents the internal area of a blood vessel as viewed end-on. This approximately circular space is the space into which blood will flow. The further away from the heart, the bigger the total cross sectional area (CSA) of all the blood vessels carrying blood, hence the flow is slower (as the blood flows into a bigger space), and the blood pressure is lower (see figure 4.15 on page 58).
Venous return mechanism

The venous return mechanism (see figure 4.12) is the process by which blood returns to the right side of the heart. It depends on:

- **Gravity** that assists the flow of blood from body parts above the heart.
- The **skeletal muscle pump** in which contracting skeletal muscle squashes veins forcing blood forwards towards the heart (since blood cannot flow back away from the heart due to the pocket valves within each vein), as described above in table 4.1.
- The **respiratory pump** which relies upon the changes in pressure that occurs in the thoracic and abdominal cavities during inspiration and expiration. These pressure changes compress nearby veins and so assist blood flow back to the heart.
- **Valves (pocket valves)** which ensure that blood can only flow in one direction back towards the heart.
- **Venomotor control** which describes the limited capacity of veins to change their shape and therefore slightly increase venous return, due to **venoconstriction**. For a fuller description of this concept see page 55.

Hence the mechanism by which the bulk of blood returns to the heart during exercise is via the skeletal muscle pump, with the respiratory and cardiac pumps also helping.

How is blood flow controlled?

Changes in blood vessel diameter depend upon the metabolic needs of body tissues. The vasomotor centre, located in the medulla oblongata of the brain, controls blood pressure and blood flow. This is an example of **negative feedback control**, in which an increase of blood pressure as sensed by baroreceptors causes a decrease in the blood pressure by changing blood vessel diameter.

As cardiac output increases, sensory receptors such as **baroreceptors** (responding to changes in blood pressure) and **chemoreceptors** (responding to changes in chemical composition of the blood) are stimulated.

**Vasomotor control**

Vasomotor control is concerned with the ability of muscular **arteries** and **arterioles** to change their shape. During exercise, the sensory receptors, baroreceptors and chemoreceptors, are stimulated. The vasomotor centre receives this sensory information. From here sympathetic nerves carry impulses to the smooth muscle walls of arteries and arterioles.

**Non-active tissue**

Within non-active tissues, these impulses cause vasoconstriction (tightening or narrowing) in the arteries, arterioles, and to the pre-capillary sphincters located at the openings of capillaries to the inactive tissue. The effect of this constriction is to restrict blood flow into the capillary bed of the non-active tissue.

**Active tissue**

In contrast, within active tissue, sympathetic stimulation to the smooth walls of arteries and arterioles and pre-capillary sphincters is reduced, and the muscles in the arterial walls and pre-capillary sphincters relax. Therefore these vessels dilate or open wider (known as vasodilation) and the pre-capillary sphincters open up, resulting in additional blood flow into active muscles.
Hence, as exercise begins, as a result of vasomotor control, blood flow is diverted into active skeletal muscle where it is needed. This redirection of blood flow is called the vascular shunt mechanism and is illustrated in figures 4.13 (on page 56) and 4.14.

The vasomotor centre works in conjunction with the cardiac centre in maintaining blood pressure.

**Vascular shunt mechanism**

Table 4.2 illustrates the redistribution of blood flow as exercise begins, away from the major organs of the body towards working muscle.

Table 4.2 – comparison of the distribution of cardiac output at rest and during exercise

<table>
<thead>
<tr>
<th>tissue</th>
<th>rest</th>
<th>maximal</th>
<th>exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>ml min⁻¹</td>
<td>%</td>
</tr>
<tr>
<td>liver</td>
<td>27</td>
<td>1350</td>
<td>1</td>
</tr>
<tr>
<td>kidneys</td>
<td>22</td>
<td>1100</td>
<td>1</td>
</tr>
<tr>
<td>brain</td>
<td>14</td>
<td>700</td>
<td>3</td>
</tr>
<tr>
<td>heart</td>
<td>4</td>
<td>200</td>
<td>4</td>
</tr>
<tr>
<td>muscle</td>
<td>20</td>
<td>1000</td>
<td>88</td>
</tr>
<tr>
<td>skin</td>
<td>6</td>
<td>300</td>
<td>2</td>
</tr>
<tr>
<td>other</td>
<td>7</td>
<td>350</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>100</td>
<td>5000</td>
<td>100</td>
</tr>
</tbody>
</table>

**Venomotor control**

Venomotor control describes the limited capacity of veins to change their shape. This is the result of venomotor tone, whereby the veins’ muscular coat receives stimulation from the sympathetic nervous system. The effect of limited vеноconstriction of veins causes a small increase in blood velocity and hence an increase in venous return.

**Transport of oxygen and carbon dioxide by the vascular system**

Blood consists of 55% plasma (which transports dissolved nutrients, hormones and waste) and 45% corpuscles.

- 97% of the oxygen carried by the blood is transported via *haemoglobin* in the red corpuscles, since haemoglobin readily attaches itself to O₂ when exposed to it in the alveoli within lung tissue. The remaining 3% of the oxygen carried is dissolved in the blood plasma.

- Exercise causes a small increase in pulmonary blood pressure, which distorts red blood corpuscles within the alveolar capillary system, and this enables 10 times as much oxygen to be picked up as at rest.

- The formula for the oxygenation of haemoglobin (Hb) is: \( \text{Hb} + 4\text{O}_2 \rightarrow \text{Hb(O}_2\text{)}_4 \), where one molecule of Hb combines with 4 molecules of O₂ to form a molecule of oxyhaemoglobin Hb(O₂)_4.

- The amount of oxygen transported by the blood is a function of cardiac output and the oxygen content of the blood.

- At rest, we use about 25% of available oxygen. This leaves an unextracted 75% of the available oxygen in blood returned to the heart via venous return. This is called the oxygen reserve, which is immediately available for exercise when it begins.

- The remaining corpuscles within the blood are the white blood cells. These cells produce antibodies and regulate the body’s immune system, and platelets responsible for blood clotting.

**Carbon Dioxide** (CO₂) is produced by the respiration process in tissue cells, the oxidation of fuels in oxygen to produce energy, which in muscle cells enables the person to move / run / jump. CO₂ is transported in venous blood as:

- Carbonic acid (most of which dissociates into H⁺ and HCO₃⁻) (70%).
- Carbaminohaemoglobin (the combination of CO₂ with haemoglobin in a similar way that O₂ combines with Hb to form oxyhaemoglobin) (23%).
- CO₂ dissolved in blood plasma (7%).
Carbon dioxide is excreted from the lungs during expiration. This CO₂ has to be removed from the tissue cells since if it stays it forms carbonic acid (dissociated into H⁺ and HCO₃⁻ as mentioned above), which in effect acts as a poison and will reduce a muscle cell to complete inactivity within a few seconds. Further notes on gas transport and exchange are to be found on page 66.

Hence it is very important to maintain an efficient blood transport system (carrying oxygen into, and carbon dioxide away from muscle) if a person is to be able to exercise and live healthily.

**Blood pressure**

**Blood pressure** is defined as ‘the force per unit area exerted by the blood on the inside walls of blood vessels’ and so represents the driving force that moves blood through the circulatory system. It is the combination of cardiac output and peripheral resistance of blood vessels and is measured using a sphygmomanometer around the upper arm.

- **Systolic** pressure (the highest pressure) is generated by left ventricular contraction (systole) as blood is ejected into the aorta and main arteries.
- **Diastolic** pressure (the lowest pressure) is reached when the heart relaxes (diastole) and the aortic valves close due to pressure changes.
- Hence blood pressure is measured using these two pressures:

  - systolic pressure mmHg
  - diastolic pressure

**Blood velocity**

- You will notice from figure 4.15 that as blood flows through the network of blood vessels the blood velocity falls. This is because it encounters vessels which branch repeatedly, with a bigger space to flow into.
- This means that the vessels have a bigger total combined cross sectional area (CSA – see solid green line in figure 4.15) and hence the blood slows down (blood velocity falls – see the solid red line in figure 4.15). When blood reaches the capillaries, the CSA is a maximum (many tiny vessels) and therefore the blood flows very slowly.
- This process reverses as the blood flows back towards the heart. CSA reduces (see the green line in figure 4.15), and blood velocity increases until it almost matches the speed of blood leaving the heart.
- You have to note that the same volume of blood will return to the heart as leaves it (in any given period of time) – this is **Starling’s Law of the Heart**.

**Blood pressure**

- Blood pressure forces blood through arteries and arterioles, and as the CSA increases, the individual diameters of blood vessels reduce. This increases the peripheral resistance (the resistance - fluid friction drag - to flow of a fluid through a tube), and reduces the rate of flow through these vessels.
- Therefore, in comparison with the pressure forcing the blood into the system (by the pumping heart), the pressure forcing the blood along the tubular system of blood vessels is less at any particular point, and lowest near the end of the system – which is the capillary bed. Hence the blood pressure (represented by the black line in figure 4.15) falls as the blood travels from the heart to the capillary system. Therefore once the blood has flowed through the capillary beds the pressure forcing the flow onwards is very low.
- This means that unless action is taken, the blood will remain in the capillary beds at the furthest points from the heart, and before the veins and venules are reached. This is called **blood pooling** and is the main reason why an active cool-down is essential after intense exercise.
- Therefore in veins, venous return is forced by the action of the skeletal muscle and respiratory pumps, and the cardiac pump, which is the action of the heart itself beating as it forces blood out into the aorta, and draws blood in from the venae cavae. This follows from Starling’s Law of the Heart as mentioned above.
During vigorous rhythmic exercise, the skeletal muscle and respiratory pumps are much bigger, therefore venous return is bigger and blood flow is higher. Also, as blood flow is increased and venous return is higher, the heart is stimulated to pump harder and more frequently. Hence the systolic blood pressure is increased - which in turn forces greater blood flow into the arteries at greater blood velocity. Note that diastolic pressure remains relatively unchanged in dynamic exercise as quoted in the values in table 4.3.

<table>
<thead>
<tr>
<th>Systolic</th>
<th>Dynamic Exercise</th>
<th>Static Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>120</td>
<td>170</td>
<td>200</td>
</tr>
<tr>
<td>Diastolic</td>
<td>80</td>
<td>88</td>
</tr>
</tbody>
</table>

Table 4.3 – blood pressure

Long-term responses to the cardio-vascular system due to exercise and the consequences for long-term health

The heart

The long-term adaptations to the heart produced by exercise are outlined on page 53 above, illustrated in figure 4.16, and include:

- Increase in maximum heart rate, as the myocardium (heart muscle) becomes fitter and able to contract more often per minute.
- Bradycardia or increase in size and strength of the heart muscle which causes:
  - Increase in stroke volume (SV).
  - A consequent decrease in resting heart rate.
  - Increase in blood supply to the myocardium, therefore the heart is more efficient.

These adaptations enable more blood (and hence oxygen) to be pumped around the body during exercise.

The vascular system

The long-term adaptations to the vascular system would be:

- Greater tolerance to blood lactate levels. The onset of blood lactate accumulation (OBLA) occurs at higher exercise intensity.
- Increase in blood volume and haemoglobin (Hb) count.
- Improved capillarisation around lung alveoli and skeletal muscle tissue.
- Therefore increase in $\dot{V}O_{2max}$.
- Increase in blood flow to working muscle tissue.
- Therefore increase in $a-VO_{2sat}$ (see page 67 for details of this concept).
- Elasticity of blood vessels (the muscular walls) increases, reducing blood pressure.
- Blood lactate is less acidic due to a more efficient aerobic system.

The consequences for long-term health are that oxygenated blood is able to reach the body’s extremities more easily, and therefore service the limbs and reduce circulatory problems such as thrombosis. Cardiac functioning is more efficient reducing the risk of CHD.
Respiratory responses to physical activity

Introduction to respiratory structures

Prior knowledge of the structure and function of the respiratory system is assumed, so we include elements of this here.

Respiratory structures

From figure 4.17 you will see that the air pathway as the air is breathed in follows the route: nasal cavity to pharynx to larynx to trachea to bronchi to bronchioles to respiratory bronchioles (the smaller tubes which branch out from the bronchioles) to alveolar ducts (the tubes connecting the respiratory bronchioles to the alveoli) to alveoli.

The trachea consists of an incomplete ring of cartilage that keeps the airway open and allows swallowing. The nasal cavity, pharynx, larynx, trachea and bronchi have ciliated linings and mucous glands to provide a cleaning and filtering mechanism for incoming air.

Lung structure

The pulmonary pleura is a self-enclosed serous membrane covering the lungs. It lines the thoracic cavity, middle wall of the thorax and diaphragm. This membrane secretes pleural fluid into the pleural cavity thereby reducing friction between lung tissue and ribs, aiding inspiration as pleural pressure reduces, and expiration as pleural pressure increases.

Alveoli (see figure 4.17) are elastic, moist, and permeable (as single layered epithelium cells) and are surrounded by a network of capillaries. These are adapted for gaseous exchange, as oxygen travels through the capillary walls from the lung space into the blood within the capillaries, and carbon dioxide travels in the opposite direction through the capillary walls.

Pulmonary ventilation is ‘the process by which we move air into and out of the lungs.’
**Mechanics of breathing**

The actual mechanism of breathing is brought about by changes in air pressure (intrapulmonary pressure) in the lungs relative to atmospheric pressure, and as a result of the muscular actions of the 11 pairs of intercostal muscles and the diaphragm.

---

**Table 4.4 – inspiration and expiration at rest and during exercise (see figures 4.18 and 4.19)**

<table>
<thead>
<tr>
<th>Inspiration at Rest</th>
<th>Expiration at Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>external intercostal muscles contract</strong></td>
<td><strong>external intercostal muscles relax</strong> - a passive process</td>
</tr>
<tr>
<td><strong>diaphragm contracts – becomes flatter</strong></td>
<td><strong>diaphragm relaxes – domes upward into chest cavity – a passive process</strong></td>
</tr>
<tr>
<td><strong>internal intercostal muscles relax</strong></td>
<td><strong>ribs and sternum move downwards and inwards</strong></td>
</tr>
<tr>
<td><strong>ribs and sternum move upwards and outwards</strong></td>
<td><strong>increase in chest cavity volume</strong></td>
</tr>
<tr>
<td><strong>pressure between pleural membranes is reduced</strong></td>
<td><strong>decrease in chest cavity volume</strong></td>
</tr>
<tr>
<td><strong>allows elastic pulmonary tissue to expand</strong></td>
<td><strong>compresses elastic pulmonary tissue</strong></td>
</tr>
<tr>
<td><strong>lung volume increases</strong></td>
<td><strong>lung volume decreases</strong></td>
</tr>
<tr>
<td><strong>pulmonary air pressure falls below atmospheric pressure (outside the body)</strong></td>
<td><strong>pulmonary air pressure is driven above atmospheric pressure (outside the body)</strong></td>
</tr>
<tr>
<td><strong>hence atmospheric air is forced into the lungs</strong></td>
<td><strong>hence atmospheric air is forced out of the lungs via the respiratory passages</strong></td>
</tr>
<tr>
<td><strong>until lung pressure equals the pressure outside again</strong></td>
<td><strong>until lung pressure equals the pressure outside again</strong></td>
</tr>
</tbody>
</table>

---

**During exercise**

**additional muscles in the chest and torso contract** (scalenes, sternocleidomastoid, pectoralis major / minor)  
**internal intercostal muscles and abdominal muscles contract powerfully, acting on ribs and body cavity**

**chest cavity volume further increased**  
**chest cavity volume is further reduced**

**more air forced into the lungs**  
**more pulmonary air is forced out of the lungs**

---

**STUDENT NOTE**

See page 37 above for an introductory description of the location of the various muscles listed above. More detail can be found in specialist books or charts on human anatomy or the skeleto-muscular system.
Lung volumes and capacities

Interpretations from spirometer readings

A spirometer is a device that is used to measure pulmonary volumes. Figure 4.20 presents a typical lung volume trace resulting from a person breathing into a calibrated spirometer, at rest and during exercise. Note that during the exercise period tidal volume increases because of the encroachment on inspiratory reserve volume (IRV) and expiratory reserve volume (ERV), but more noticeably on the IRV.

Definitions for pulmonary volumes and average values for male and females are shown in table 4.5 below.

Lung volumes vary with age, gender, body size and stature, and are defined and explained in table 4.5.

Table 4.5 – lung volumes and definitions

<table>
<thead>
<tr>
<th>Lung volumes</th>
<th>Definitions</th>
<th>Average values (ml)</th>
<th>Change during exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>TLC</td>
<td>Total lung capacity</td>
<td>6000</td>
<td>4200</td>
</tr>
<tr>
<td>VC</td>
<td>Vital capacity</td>
<td>4800</td>
<td>3200</td>
</tr>
<tr>
<td>TV</td>
<td>Tidal volume</td>
<td>600</td>
<td>500</td>
</tr>
<tr>
<td>IRV</td>
<td>Inspiratory reserve volume</td>
<td>3000</td>
<td>1900</td>
</tr>
<tr>
<td>ERV</td>
<td>Expiratory reserve volume</td>
<td>1200</td>
<td>800</td>
</tr>
<tr>
<td>RV</td>
<td>Residual volume</td>
<td>1200</td>
<td>1000</td>
</tr>
<tr>
<td>VE = TV x f</td>
<td>Minute ventilation</td>
<td>7200</td>
<td>6000</td>
</tr>
</tbody>
</table>

Lung capacities are made up of combinations of lung volumes. The following list uses as examples the average male values from table 4.5 above.

- **Inspiratory capacity (IC)** = TV + IRV (3600 ml)
- **Expiratory capacity (EC)** = TV + ERV (1800 ml)
- **Vital capacity (VC)** = TV + IRV + ERV (4800 ml)
- **Functional residual capacity (FRC)** = RV + ERV (2400 ml)
- **Total lung capacity (TLC)** = VC + RV (6000 ml)
Minute ventilation

Minute ventilation (\(\dot{V}E\)) is defined as ‘the volume of air that is inspired or expired in one minute’. Minute ventilation can be calculated by multiplying tidal volume (TV) by the number of breaths (f) taken in one minute (see the last row of table 4). Below are examples of minute ventilation values you would expect at rest and during differing intensities of exercise. A normal male resting breathing frequency is about 12 breaths per minute, and this is the value of f in the first row of the list below. This would increase to about 25 (breaths per minute) for submaximal exercise, and rapid breathing of about 55 breaths per minute during maximal exercise.

\[
\text{(dm}^3) \quad \dot{V}E = TV \times f
\]

| at rest | 7.2 = 0.6 \times 12 = 7.2 litres per minute or 7200 ml per minute – since 1 dm\(^3\) is 1 litre or 1000 ml. |
| sub-max | 60 = 2.4 \times 25 = 60 litres per minute or 60000 ml per minute. |
| max     | 121 = 2.2 \times 55 = 121 litres per minute or 121000 ml per minute. |

Hence from sub-maximal to maximal exercise breathing rate or respiratory frequency doubles. This dramatic increase often corresponds with the onset of anaerobic metabolism or the onset of blood lactate accumulation or OBLA at the expense of a decreasing tidal volume.

What is actually happening is a regulation of minute ventilation in response to increased carbon dioxide production and the need to get rid of carbon dioxide during expiration. The tidal volume decreases slightly because it is not physically possible to inspire the maximum possible volume of air during maximal exercise at a high breathing rate. This regulatory response is discussed further on page 64 below.

Figure 4.21 compares the changes in minute ventilation with time during low intensity and high intensity exercise.

Ventilation – short-term response to exercise

During the short period before exercise begins, during the exercise period, and during the recovery period immediately after exercise (see graph in figure 4.21) the following describes the reasons for the changes in rate of ventilation or minute ventilation.

- **Anticipatory rise** in \(\dot{V}E\) is due to the hormonal action of adrenaline and noradrenaline on the respiratory centre in the brain. This rise is caused by the excitement in anticipation of exercise beginning.

- **Rapid rise** of \(\dot{V}E\) on exercise beginning is due to proprioceptor sensory stimulation, and also due to continued release of hormones. During this period, exercise is anaerobic in nature and does not require oxygen from the respiratory system. However, an oxygen debt is building up, and this will need to be dealt with later.

- **During sub-maximal exercise**, a **levelling off** of \(\dot{V}E\) occurs as a steady state is developed between oxygen required and provided by the respiratory system. Some recovery of \(O_2\) debt (aerobic) occurs.

- **During maximal workloads** there is a continued **slower increase** in \(\dot{V}E\) as anaerobic systems continue to be stressed. This produces lactic acid + \(CO_2\) + \(K^+\), which stimulate chemoreceptors at maximal level. The main stimulant for increased rates of ventilation is the presence of carbon dioxide in the blood flowing past chemoreceptors. See section on page 64 below for details of the location and function of these receptors which stimulate the respiratory centre in the brain.

- **As exercise ends**, there is a **rapid decline** in \(\dot{V}E\) due to cessation of proprioceptive stimuli and the withdrawal of hormones, then a levelling out to pre-exercise values.

- **Later, after maximal work**, there is a **much slower decrease** in \(\dot{V}E\) due to the clearance of metabolites such as lactic acid and carbon dioxide as systems return to normal resting values.
Neural regulation of pulmonary ventilation (breathing)

The respiratory control centre (RCC, see figure 4.22) is located within the medulla oblongata of the brain and regulates pulmonary ventilation. Rate of breathing (also called the frequency of breathing \( f \)) and defined as ‘the number of breaths taken in one minute’) and depth of breathing (known as tidal volume (TV) and defined as ‘the volume of air inspired or expired in one breath’) are controlled by neurones within the medulla. Although the medullary neurones establish a basic rhythm of breathing, their activities can be influenced by input from other parts of the brain and by input from peripherally located receptors discussed in figure 4.23.

The RCC consists of two parts: the inspiratory and expiratory centres:

- The inspiratory centre is responsible for the basic rhythm of ventilation. At rest impulses are sent via the phrenic and intercostal nerves to the external intercostal muscles and diaphragm causing these muscles to contract to bring about inspiration. When stimulation ceases these muscles relax causing expiration.

- The expiratory centre is inactive during quiet breathing. However, during forceful breathing such as during exercise, the expiratory centre actively sends impulses to stimulate the muscles of expiration (sternocleidomastoid, scalenes, pectoralis major and minor) to increase the rate of breathing (refer to table 4.4 on page 61 to remind yourself of the mechanics of breathing).

Two additional brain centres aid the control of breathing:

- The apneustic centre controls the intensity of breathing. It does this by prolonging the firing of the inspiratory neurones, thereby increasing TV.

- The pneumotaxic centre antagonises the apneustic centre, resulting in the fine-tuning of the breathing rate (\( f \)).

Factors and neural control influencing breathing

The body exquisitely regulates the rate and depth of breathing in response to metabolic needs. Figure 4.23 lists the primary factors involved in ventilatory control. This is another example of negative feedback control, where, for example, an increase of carbon dioxide in venous blood tends to increase breathing rate which helps extract the carbon dioxide from pulmonary blood and reduce carbon dioxide in blood arriving back at the heart from the lungs.

Chemical control

The chemical state of the blood largely regulates pulmonary ventilation at rest.

- Central chemoreceptors (located in the medulla) are major regulators, and respond to increased concentration of carbon dioxide in the blood. The partial pressure of \( \text{CO}_2 \) is termed \( \text{pCO}_2 \), and this regulation process tries to keep \( \text{pCO}_2 \) to below 5.3 kPa as well as controlling increased acidity (or decreased pH due to \( H^+ \) ions from carbonic acid formed in the blood plasma).

- Peripheral chemoreceptors (in the aortic and carotid bodies) provide an early warning system as they sense the constituents of blood as it passes them.

- Both central and peripheral chemoreceptors respond to increased \( \text{pCO}_2 \) and decreased pH and \( \text{pO}_2 \) (oxygen concentration in the blood).
These receptors send messages to the inspiratory centre which then stimulates respiratory muscles to increase rate (f) and depth of breathing (TV) as described above. For example, lack of oxygen at high altitude stimulates respiration which has nothing to do with exercise, but indicates how these receptors work. This chemical control, via the pneumotoxic and apneustic centres of the brain, adjusts ventilation to maintain arterial blood chemistry within narrow limits. This means that these brain centres attempt to keep blood oxygen to a maximum, and blood carbon dioxide to a minimum by causing the person to adjust breathing rate and depth.

**Proprioceptors in joints and muscles**

Proprioceptors (such as working muscle spindles) send signals to the RCC about the tension within and state of contraction of a muscle, and hence when a muscle is being used intensely. During physical activity increased stimulation will increase rate and depth of breathing via the inspiratory centre as described above.

**Lung stretch receptors**

A type of proprioceptor, these lung receptors are located within the walls of bronchi and bronchioles. When stimulated these receptors relay information, via the vagus nerves, to the RCC to inhibit the inspiratory centre, resulting in expiration via the expiratory centre. As expiration proceeds, the stretch receptors are no longer stimulated and the decreased inhibitory effect on the inhibitory centre allows the inspiratory centre to become active again, known as the Hering-Breuer Reflex. Its overriding effect is to prevent over-inflation of the lungs.

**Temperature**

Thermoreceptors (located in the hypothalamus region of the brain) respond to increases in body / blood temperatures. These receptors directly excite the neurones of the RCC and help control ventilation during prolonged exercise.

**Irritant receptors**

The activation of touch, thermal and pain receptors can also stimulate the RCC.

**Higher centres of the brain**

Through the cerebral cortex it is possible consciously to increase or decrease rate and depth of breathing. Swimmers and sports divers hyperventilate and breath-hold to improve physical performance. At the start of a swimming race athletes hyperventilate on the starting blocks to prolong breath-hold time during the swim. In short course racing, the breath-hold time can be the whole of the racing time. Snorkel divers hyperventilate to extend breath-hold time. During breath-hold time the pO\textsubscript{2} content of the blood can fall to critically low values before arterial pCO\textsubscript{2} increases to stimulate breathing.

**Emotions** acting through the limbic system can also affect the RCC.

**The process of transport of respiratory gases**

Several factors affect the rate at which the gases taking part in the respiration process are exchanged (figure 4.24).

**Diffusion**

The exchange of gases between lungs and blood and their movement at tissue level takes place passively by diffusion.

This is the movement of molecules through space by random collision with other molecules. This process would eventually result in random mixing of all the molecules present in a space. Molecules move using this process through gases and liquids, and can migrate through membranes (like tissue boundaries such as cell walls).
A diffusion gradient is a situation where the concentration of molecules of a particular substance (say oxygen for example) is greater on one side of a space than on the other side of the same space. Hence a diffusion gradient will cause molecules to move across a space (or membrane) by random mixing or random molecular collision with membrane walls. Steep diffusion gradients are maintained by the factors shown in figure 4.24, and help move substances from higher concentrations to lower concentrations where they take part in the respiration process.

Gases diffuse from high to low pressure, and so the rate of exchange (either at lungs or tissue site) depends on the partial pressure of each gas (in blood or tissue site or alveolar air), gas solubility (in blood or tissue cell fluids), and temperature.

Partial pressure

Partial pressure (p) is defined as 'the pressure a gas exerts within a mixture of gases', so pO₂ and pCO₂ are the partial pressures exerted by oxygen and carbon dioxide respectively within a mixture of these and other gases (for example nitrogen) present in the air or the tissues. The partial pressure of a gas is directly related to the concentration (number of molecules per cubic metre) of gas molecules in a space. At a given temperature, the bigger the p, the more molecules of gas are present.

Gaseous exchange

Gaseous exchange is the process whereby oxygen from the air in the lungs is transferred by diffusion to the blood flowing through the alveoli (see figure 4.25). At the same time, carbon dioxide is transferred from the blood arriving at the lungs, into the air in the lungs, which is subsequently breathed out. The gases travel through the capillary/alveolar walls, with oxygen diffusing into the blood, and carbon dioxide diffusing out of the blood.

The reverse process happens at the tissue site (for example, active muscle tissue – see figure 4.26). Here, oxygen is carried by blood into the tissues and there it diffuses into tissue cells. At the same time, carbon dioxide diffuses out of tissue cells into the blood (which then flows back through the venous system and the heart and back to the lungs).

How gaseous exchange is achieved in the alveoli

The first step in oxygen transport involves the diffusion of oxygen from the alveoli into the blood.

In venous blood (arriving at the lungs from tissues) the partial pressure of oxygen (pO₂) = 5.3 kPa. The partial pressure of oxygen in alveolar air is 13.3 kPa, so the oxygen travels through the alveolar and capillary walls from the lung space into the blood where it combines with haemoglobin to form oxyhaemoglobin (Hb(O₂)₄) as follows:

$$\text{Hb} + 4\text{O}_2 \rightarrow \text{Hb(O}_2)_4$$

One of the short-term effects of physical activity is to cause a small increase in pulmonary blood pressure, which distorts red blood corpuscles within the alveolar capillary system, and this enables 10 times as much oxygen to be picked up as at rest.

The oxyhaemoglobin dissociation curve (see figure 4.27) describes the percentage of haemoglobin saturated with oxygen at a given pO₂. At 13.3 kPa pressure, oxygen will combine with Hb at 98% of the maximum possible (see the red vertical line labelled C).
A on figure 4.27, this is at 13.3 kPa and intersects the graph line at almost 100%. This means that Hb leaving the lungs is almost completely saturated with $O_2$. (Note that 3% of oxygen dissolves in blood plasma).

Blood carrying this $O_2$ then travels out of the lungs to the heart via the pulmonary vein, then out to the body through the aorta and main arteries. At altitude, the pO$_2$ is less, which means that haemoglobin cannot carry as much oxygen as at sea level, therefore reducing the ability to perform physical work. This is called **hypoxia** (lowered pO$_2$).

At the same time, carbon dioxide is transferred in the opposite direction, from the blood into the alveolar air. The concentration of CO$_2$ in atmospheric air is about 0.049% (very small), and therefore pCO$_2$ in venous blood arriving (via the heart) from the body tissues is higher than in the alveoli air (breathed into the lungs). Therefore CO$_2$ diffuses through the alveolar membrane (from blood to air in lung) and is expired. Between 3% and 6% of air breathed out is CO$_2$ as shown in table 4.5.

<table>
<thead>
<tr>
<th>Table 4.5 – differences between inhaled and exhaled air</th>
</tr>
</thead>
<tbody>
<tr>
<td>differences between inhaled and exhaled air</td>
</tr>
<tr>
<td>inhaled(%)</td>
</tr>
<tr>
<td>O$_2$</td>
</tr>
<tr>
<td>CO$_2$</td>
</tr>
</tbody>
</table>

**How gaseous exchange is achieved at the tissue cell site**

The second step in oxygen transport involves the transfer of oxygen from the blood into tissue cells.

**The role of myoglobin**

Myoglobin is a substance somewhat similar to haemoglobin in that it attracts and binds to molecular oxygen. Myoglobin has a greater affinity for oxygen than haemoglobin and is located within cells, where its role is to enable oxygen to be carried across a cell to the **mitochondria** where the oxygen is consumed and energy transfer takes place (which, for example, enables muscle tissue to contract). Arriving (arterial) blood has an oxygen partial pressure (pO$_2$ = 13.3 kPa). This is greater than tissue pO$_2$ since the oxygen is being used up in the cells during the energy creating process. Because myoglobin in the tissue cells has a greater affinity for oxygen than does haemoglobin, oxygen diffuses through the capillary and cell walls from the blood into the tissue cells. Myoglobin then facilitates oxygen transfer to the mitochondria, notably at the start of exercise and during intense exercise when cellular pO$_2$ decreases considerably.

**Oxygen transfer at rest**

At a pO$_2$ of 5.5 kPa, which is the normal pO$_2$ in resting tissue capillaries, haemoglobin is about 70% saturated (this corresponds to red vertical line B in figure 4.27). This means that approximately 30% of the oxygen bound to haemoglobin is released into the blood and can diffuse into the tissue spaces.

**Oxygen transfer during vigorous exercise**

During vigorous exercise the pO$_2$ in tissue spaces may decline to levels as low as 2.5 kPa. Therefore, looking at line C in figure 4.27, only about 25% of the haemoglobin remains saturated, and 75% of the oxygen bound to haemoglobin is released into the blood and can diffuse through the capillary walls into the active tissue spaces. The absorption and utilisation of oxygen from the blood leads to a difference in the oxygen content of arterial and venous blood. This difference is known as the **arterio-venous oxygen difference** or $a-V_O_{2diff}$.

**Differences in oxygen and carbon dioxide – arterio-venous oxygen difference ($a-V_O_{2diff}$)**

At rest, as blood moves from arteries to veins, its oxygen content reduces from 20 ml of oxygen per 100 ml of arterial blood to 15 ml of oxygen per 100 ml of venous blood. As you can see in figure 4.28 overleaf on page 68, the difference between these two values (20 ml − 15 ml) = 5 ml. This value of 5 ml represents the **arterio-venous oxygen difference** ($a-V_O_{2diff}$).

**STUDENT NOTE**

Note that the bar over the v in $a-V_O_{2diff}$ refers to an average based on calculations for mixed venous return.
The value of a-\(\Delta\)O\(_2\) represents the extent to which oxygen has been removed from the blood as it passes through the body. This means that, at rest, about 75% of the blood’s original oxygen load (15 ml per 100 ml of blood out of the original 20 ml) remains bonded to the haemoglobin. This is called the oxygen reserve, which is immediately available for exercise when it begins.

During exercise the a-\(\Delta\)O\(_2\) value triples leaving only 25% of the blood’s original oxygen load (now it is 5 ml per 100 ml of blood out of the original 20 ml) bonded to the haemoglobin as illustrated in figure 4.29.

A long-term effect of aerobic training is to increase the a-\(\Delta\)O\(_2\) because trained athletes can extract more oxygen from the blood. This is due to trained people having more myoglobin, mitochondria and oxidative enzymes in their muscle cells than untrained people. This increase in a-\(\Delta\)O\(_2\) is also attributed to a more effective distribution of arterial blood from inactive tissue to the active tissue (more effective vascular shunt) and increased capillarisation and utilisation of capillaries around and through active tissues.

The effect of pH, pCO\(_2\) and temperature on oxygen release

Other factors influence the degree to which oxygen binds to haemoglobin. During exercise, tissue cell and blood temperature increases, pCO\(_2\) increases due to the greater need for energy, and pH decreases due to the greater presence of H\(^+\) ions from dissociating carbonic acid by the released CO\(_2\). All these conditions cause reduction in the affinity of haemoglobin for oxygen. This means that more O\(_2\) is released (than would be the case if no exercise were being taken), and hence more O\(_2\) is then available to active tissue sites which are working harder. So the harder the tissue is working, the more O\(_2\) is released.

The effect of increases in acidity, pCO\(_2\) and temperature is to cause the oxyhaemoglobin dissociation curve to shift downward and to the right (enhanced unloading). This phenomenon is called the ‘Bohr effect’.

Carbon dioxide transport (figure 4.30)

CO\(_2\) is produced in the cells as an end product of tissue cell respiration (production of energy from combination of fuel with oxygen). Hence, the fluid within muscle tissue cells has a higher pCO\(_2\) than in the blood. Therefore CO\(_2\) diffuses back through cell and capillary walls in the opposite direction (from tissue to departing blood).

CO\(_2\) is transported in venous blood as:

- Carbonic acid mostly dissociated into H\(^+\) and HCO\(_3^-\) (70%).
- Carbaminohaemoglobin (23%).
- CO\(_2\) dissolved in blood plasma (7%).

In the lung capillaries carbon dioxide is released, then it diffuses from the blood into the alveoli and is expired out of the lungs.
The impact of exercise on the respiratory system and the consequences for long-term health

Aerobic exercise

Intense aerobic exercise (see figure 4.31) has the effect of forcing the person to breathe more deeply and more often (the vital capacity of the lung is fully utilised, and the breathing frequency (f) increases).

Therefore, as a result of long-term exercise, the following adaptations take place within the body which tend to make more efficient the transfer (from air breathed in) of oxygen to working muscle.

- Long-term exercise has the effect of exercising the respiratory muscular system – namely, the diaphragm and intercostal muscles. If exercise is continued at least two to three times per week, these muscles will get fitter and stronger and more capable of working without cramps and conditions like stitches.
- The efficiency of the respiratory system will depend on the utilisation and capacity of the alveoli to take oxygen from air breathed in and transmit it to blood flowing through the alveolar capillary bed. Long-term physical activity increases blood flow to the upper lobes of the lungs to increase utilisation of lung alveoli, hence increases gaseous exchange and therefore VO$_{2\text{max}}$ at high intensity aerobic workloads.
- At submaximal workloads VO$_2$ will be less because of greater efficiency of oxygen uptake, and general improvements in lung function will occur such as increase in tidal volume (TV) and vital capacity (VC) at the expense of residual volume (RV).
- Increased efficiencies of the respiratory system will improve recovery from exercise and reduce oxygen debt during exercise.
- At submaximal workloads there is a slight decrease in the breathing rate (f – the frequency of breaths).
- During maximal workloads there is a big increase in breathing rate (f), hence much bigger values in minute ventilation are achieved.

Practice questions

1) Figure 4.32 shows a diagrammatic picture of the cardiac impulse. Using the information in this diagram, describe the flow of blood during the specific stages of the cardiac cycle, in relation to the cardiac impulse. In your answer explain how the heart valves help control the direction of blood flow. 8 marks

2) \[ Q = SV \times HR \]. Explain the meaning of this equation and give typical resting values that you would expect in an endurance-based athlete. 6 marks

3) A fit 18 year old female student performs a 400m time trial in one minute.
   a) Sketch and label a graph to show a typical heart rate response from a point 5 minutes before the start of the run, during the time trial, and over the 20 minute recovery period. 4 marks
   b) Explain why heart rate takes some time to return to its resting value following the exercise period. 2 marks
   c) Identify a hormone that is responsible for heart rate increases prior to and during an exercise period. 1 mark
   d) Heart rate is regulated by neural, hormonal and intrinsic factors. How does the nervous system detect and respond to changes in heart rate during an exercise period? 4 marks
4) Table 4.6 shows the rate of blood flow (in cm$^3$ per minute) to different parts of the body in a trained male athlete, at rest and while exercising at maximum effort on a cycle ergometer. Study the data carefully before answering the following questions.

a) The rate of blood flow to the ‘entire body’ increases significantly during exercise. Explain briefly how the heart achieves this. 3 marks
b) What percentage of the total blood flow is directed to the skeletal muscle (show your calculations) at rest and during maximum effort? 2 marks
c) How is blood flow to various regions of the body controlled? 4 marks

5) a) Explain venous return and describe how it is aided during physical activity when a person is exercising in an upright position. 3 marks
b) Explain the importance of the skeletal muscle pump mechanism. 2 marks
c) What effect does enhanced venous return have upon cardiac output and stroke volume? 4 marks

6) a) How is oxygen transported by the blood? 2 marks
b) Identify the main method whereby carbon dioxide is transported in venous blood. 1 mark
c) Explain how increased levels of carbon dioxide affect performance during physical activity. 3 marks

7) A simple calculation for the value of blood pressure can be written as:
   \[ \text{Blood Pressure} = \text{Cardiac Output} \times \text{Resistance to blood flow}. \]
   a) Identify one factor which affects resistance to the flow of blood within systemic blood vessels. 1 mark
   b) Blood pressure is quoted as two numbers. An example would be resting values of 120/80 mmHg. Explain what each of these numbers refers to. 2 marks
   c) How would these blood pressure values change during a game of football and a rugby scrum lasting 6 seconds? Give a reason for each of your answers. 3 marks

8) Endurance training results in aerobic physiological adaptations, some of which are illustrated in table 4.7 below.

<table>
<thead>
<tr>
<th>organ or system</th>
<th>estimated blood flow in cm$^3$ min$^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>at rest</td>
</tr>
<tr>
<td>skeletal muscle</td>
<td>1000</td>
</tr>
<tr>
<td>coronary vessels</td>
<td>250</td>
</tr>
<tr>
<td>skin</td>
<td>500</td>
</tr>
<tr>
<td>kidneys</td>
<td>1000</td>
</tr>
<tr>
<td>liver &amp; gut</td>
<td>1250</td>
</tr>
<tr>
<td>other organs</td>
<td>1000</td>
</tr>
</tbody>
</table>

Table 4.6 – estimated blood flow at rest and during maximum effort

8) Endurance training results in aerobic physiological adaptations, some of which are illustrated in table 4.7 below.

<table>
<thead>
<tr>
<th></th>
<th>trained elite endurance athlete</th>
<th>untrained subject</th>
</tr>
</thead>
<tbody>
<tr>
<td>resting heart rate (bpm)</td>
<td>50</td>
<td>70</td>
</tr>
<tr>
<td>maximum heart rate (bpm)</td>
<td>210</td>
<td>190</td>
</tr>
<tr>
<td>resting stroke volume of left ventricle (ml)</td>
<td>110</td>
<td>70</td>
</tr>
<tr>
<td>maximum stroke volume of left ventricle (ml)</td>
<td>190</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 4.7 – heart rate and stroke volumes for trained and untrained people

Compare and contrast the heart of the elite endurance-trained athlete with the untrained subject. Give reasons for the differences in stroke volume at rest. 6 marks

9) a) Using practical examples from the cardio-vascular systems explain the difference between a short-term response and a long-term adaptation to exercise. 4 marks
b) Identify the long-term adaptations that you would expect to occur to the structure and the function of the cardio-vascular system, as a result of aerobic exercise. 8 marks

10) a) A hockey player has a match in one hour’s time. Describe how inspiration occurs during this resting period. 4 marks
b) During the hockey match, the player must increase the volume of gas exchanged in the lungs and muscles. Explain the changes in the mechanics of breathing (inspiration and expiration) which facilitate this increase. 6 marks
11) a) The diagram in figure 4.33 represents the lung volume changes based on a number of spirometer readings during various breathing actions. With reference to the trace, briefly explain resting tidal volume (TV), expiratory reserve volume (ERV), vital capacity (VC), and residual volume (RV). 4 marks

b) Using the information in the spirometer trace (figure 4.33), state what happens to the following volumes during the exercise period: residual volume (RV), inspiratory reserve volume (IRV), and expiratory reserve volume (ERV). 3 marks
c) Why does tidal volume change by only a small amount during the exercise period? 3 marks
d) Identify two effects of regular aerobic training on lung volumes and capacities. 2 marks
e) A student measured the volume of air that she ventilated at rest and during sub-maximal exercise. The results are shown in table 4.8 below.

Table 4.8 – ventilation at different levels of exercise

<table>
<thead>
<tr>
<th>activity level</th>
<th>inhalation volume (TV)</th>
<th>breathing rate (f)</th>
<th>minute ventilation volume (V E)</th>
</tr>
</thead>
<tbody>
<tr>
<td>at rest</td>
<td>500 ml</td>
<td>one every 6 seconds</td>
<td>A</td>
</tr>
<tr>
<td>sub maximal exercise</td>
<td>800 ml</td>
<td>one every 2 seconds</td>
<td>B</td>
</tr>
</tbody>
</table>

Define what is meant by the term ‘minute ventilation volume’ and calculate the values for A and B, clearly showing the method used. 4 marks

12) a) Describe how pulmonary ventilation is regulated during quiet breathing. 6 marks
b) Describe the chemical stimuli that control the rate and depth of breathing. How do these chemical stimuli control respiration during exercise? 6 marks

13) The breathing characteristics of individuals vary during physical activity. Table 4.9 shows the proportion of oxygen and carbon dioxide breathed during exercise compared with resting values.

Table 4.9 – proportion of O₂ and CO₂ breathed during exercise, compared to at rest.

<table>
<thead>
<tr>
<th></th>
<th>inhaled air</th>
<th>exhaled air at rest</th>
<th>exhaled air during exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>%O₂</td>
<td>21</td>
<td>17</td>
<td>15</td>
</tr>
<tr>
<td>%CO₂</td>
<td>0.049</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

a) Use the information in table 4.9 to describe the effects of exercise on gaseous exchange in the lungs. Explain why these changes occur. 4 marks
b) Explain how oxygen is exchanged between the blood and active muscle tissues. 3 marks
c) Identify the three ways CO₂ is transported by the blood. 3 marks
d) How does increased CO₂ production stimulate further release of O₂ for tissue cell respiration? 2 marks
14) The binding of oxygen to haemoglobin depends on pO$_2$ in the blood and the affinity of haemoglobin with oxygen. The curves in figure 4.31 show how different concentrations of carbon dioxide affect the saturation of haemoglobin at varying partial pressures of oxygen.

a) Explain what is meant by partial pressure of oxygen (pO$_2$).  
1 mark

b) What are the values of percentage saturation of haemoglobin on the three curves when the partial pressure of oxygen is 5.0 kPa?  
3 marks

c) What are the implications of the carbon dioxide values for curves B and C for an athlete?  
2 marks

d) Why is the partial pressure of oxygen (pO$_2$) important to the process of gaseous exchange?  
3 marks

15) There is a decline in lung function with age. How might regular resistance training and aerobic exercise affect this decline?  
6 marks