CHAPTER 2

Cardio-respiratory exercise physiology

OBJECTIVES

By the end of this chapter students should be able to:

- introduce the concept of homeostasis
- describe the structure and function of the ventilatory system
- consider the relative importance of static and dynamic lung volumes
- explain the processes of gas exchange and transport
- state the structure and function of blood cells
- describe the structures and functions of the cardiovascular system
- detail the main responses of the ventilatory and cardiovascular systems during exercise, including how training affects these responses
- discuss the importance of blood pressure and redistribution of blood flow
- introduce the functional importance of maximal oxygen uptake (\( \dot{V}O_2 \text{max} \))
- identify some of the factors that contribute to differences in \( \dot{V}O_2 \text{max} \).

Introduction

“Even when all is known, the care of a man is not yet complete, because eating alone will not keep a man well; he must also take exercise. For food and exercise, while possessing opposite qualities, yet work together to produce health.”

Hippocrates

Physiology is the study of how the human body functions and has intrigued mankind for centuries. Advances in technology have allowed us to explore in ever greater detail, progressing our knowledge and understanding to reinforce quite how remarkable the human body is. Exercise presents an excellent model to demonstrate just how effectively the systems within the body are regulated and interact with each other. Importantly, the same processes apply to everyone across a very wide spectrum, from elderly people trying to complete activities of daily living, to a highly trained athlete competing at the very top levels of sport.
A central theory of human physiology is homeostasis, defined as maintenance of a constant internal environment. The underpinning theory in its current format was detailed in Walter Cannon’s seminal book The Wisdom of the Body (1932). It is based on the stability of several key variables, achieved by changes in a number of physiological systems. It has since become apparent that many systems within the body are continuously working in a highly coordinated manner to keep a large number of variables at, or as close as possible to, resting levels. Exercise presents a number of challenges to the homeostasis of the body and successful completion of exercise requires the systems within the body to function together, tightly regulating the conditions of the internal tissues.

The cardiovascular and ventilatory systems are examples of such systems that work together to regulate variables such as the oxygen content of arterial blood, acid-base status and core body temperature, to name a few. The transport of oxygen is an excellent illustration of the body’s systems in action during exercise and this will present a common theme while studying the cardiovascular and ventilatory responses to exercise in this chapter. Indeed, the rate at which oxygen is taken into the body and used (known as oxygen uptake, $\dot{V}O_2$) is an excellent indicator of how well these systems are working together.

Figure 2.1 shows how oxygen is transported from the atmosphere to exercising muscles for use in the aerobic energy systems. Note the representation of the systems as cogs that are required to fit together and work dependent on one
another. Note also the importance of oxygen and carbon dioxide transfer between the systems, known as gas exchange (taking place in the shaded areas where the cogs meet).

In healthy humans these cogs work well at rest, irrespective of age. During exercise, when more oxygen is required by the active muscles, we can establish how well the systems are integrating by directly analysing the breathing responses and air content to calculate $\text{VO}_2$. This makes $\text{VO}_2$ a valuable marker of physiological function and an exercise physiologist will measure this during exercise to evaluate health and fitness. The relevance applies to a patient with some form of respiratory or cardiovascular disease where exercise capacity is seriously compromised, as well as to the highly trained endurance athlete where adaptations to training have greatly increased exercise capacity.

**KEY POINT**

The ventilatory and cardiovascular systems work together in a highly coordinated manner to increase oxygen delivery during exercise. This is part of the body continuously trying to maintain a constant internal environment (homeostasis).

**TO THINK ABOUT**

The exercise test

Tests of physiological responses to exercise have changed considerably. Early experiments were interested in learning what happens to try and further understand the interaction of systems. Most testing was confined to research projects in specialist labs, using early forms of heart monitoring and gas exchange equipment on people who performed controlled exercise attached to considerable amounts of equipment.

Nowadays, exercise tests are routinely conducted in a very wide range of settings: physical screening of employees to confirm fitness to do a job (e.g. emergency services or military); health testing in hospitals to determine causes or impact of illness, as well as responses to treatment; monitoring of athletes to evaluate levels of fitness and responses to training.

Advances in technology mean that physiological responses to exercise can now be measured using a variety of online systems that are often portable and wireless. This removes many of the constraints on what can easily be achieved, further enabling the study of exercise physiology in action. The demands of many forms of exercise have now been well characterized, such that sport and exercise scientists can develop various methods of optimizing performance through interventions such as training, nutrition and cooling.
Ventilatory system

The first stage in the oxygen transport system is the breathing in of oxygen-rich air through the mouth or nose and into the lungs. The action of breathing is mostly an involuntary process, although we can control it by choice to an extent, e.g. holding your breath when under water or trying to blow up a balloon.

The basis of air movement by breathing during rest and exercise is a principle of physics. A substance, air in this case, will flow from an area of higher pressure to an area of lower pressure. Therefore, for inhalation (breathing in) to occur the air pressure in the lungs needs to be lower than in the atmosphere. At rest this is almost entirely caused by contraction of the muscular diaphragm (Figure 2.2) at the base of the chest cavity (thorax). The diaphragm pulls downwards and, because of a vacuum between the lungs, chest walls and diaphragm, this increases the volume of the lungs. This increase in lung volume reduces the pressure in the lungs causing air to flow from the atmosphere into the lungs to balance the pressure gradient.

At rest, the exhalation (breathing out) process is passive (no energy required) as the diaphragm relaxes and therefore recoils back to its original position without any conscious muscular work. This recoil naturally reduces the volume of the lungs, increasing the pressure to greater than that of the atmosphere, causing air to flow back out again. The cycle then repeats and during exercise the principle remains the same.

However, during exercise when more oxygen is needed by the active muscles and more carbon dioxide is being produced by the muscles, more air needs to be inhaled and exhaled at a faster rate. To achieve this, some additional muscles in the chest wall (external intercostal muscles), abdomen, and even the shoulders, can assist with increasing the lung volume during inhalation. Furthermore, contraction of these muscles during exhalation will also compress the lungs faster and more forcefully than the natural recoil. This is therefore an active process, requiring energy to fuel the muscles of the chest and abdomen.

The inhaled air initially passes through the conducting airways (the nasal and oral passageways, and the larger airways such as the trachea and bronchi) and although no gas exchange takes place here, the air is warmed, moistened and filtered by the lining of the airways.

The airways continuously branch into smaller bronchioles and eventually end in small air sacs, each one known as an alveolus. This is where gas exchange takes place, with oxygen and carbon dioxide moving across the very thin barrier that separates the alveoli from the passing blood for further transport.

The lungs are ideally designed for gas exchange as they cover a very large surface area (millions of alveoli make a total of approximately 50–100 m², equivalent to around half a tennis court), have a good blood supply, and have a very thin total distance between the alveoli and blood (0.4 μm).

**KEY POINT**

The movement of air in and out of the lungs is achieved by repeated contraction and relaxation of muscles in the base of the chest cavity (diaphragm) and chest wall to alternately increase and decrease the volume of, and therefore pressure in, the lungs.
What do lung volumes tell us about fitness?

The above trace shows the change in volume during some resting breaths and some forced breaths [where the person breathes in and out as much as possible]. From the above trace, define the following terms:

- **Vital capacity**
- **Residual volume**
- **Tidal volume**
- **Total lung capacity**
- **Inspiratory and expiratory reserve volumes**

Interestingly, when we compare trained and untrained people of similar size, it becomes clear that these lung volumes are not something that can be trained and are not related to aerobic fitness. They are mostly determined by natural body size and age/health status.

In contrast, the rate at which the air can be exhaled is a very sensitive marker of lung function. For example, the maximum volume that can be breathed out in one second [Forced Expiratory Volume in one second, FEV₁] is often used as a test of dynamic lung volume.

An interesting area for research at present is whether training the respiratory muscles can assist performance. In contrast to original ideas in this field, there is some suggestion that training these muscles with special resisted breathing devices can improve long duration endurance performance by making breathing easier without any changes in lung volume.

**Gas exchange**

Gas exchange in the lungs, as well as in other body tissues, takes place according to another passive process known as diffusion. This is another basic principle of physics. Gas will move along a gradient from an area of higher partial pressure to lower partial pressure. Partial pressure is similar to concentration, but represents the pressure exerted by a single gas (e.g., oxygen) within a mixture (e.g., air, blood or tissue fluid).

In the lungs, the air breathed in is high in oxygen and low in carbon dioxide. The blood being pumped to the lungs from the active tissues via the heart is lower in oxygen and higher in carbon dioxide. Therefore, oxygen will diffuse from the alveolus into the blood, and carbon dioxide will diffuse from the blood into the alveolus (Figure 2.3). The blood leaving the lungs, now high in oxygen and low in carbon dioxide, will be pumped to the tissues via the heart. At the
tissues where oxygen is being used up and carbon dioxide produced, the pressure gradients will drive oxygen from blood into tissues and carbon dioxide from tissues into blood.

During exercise, the pressure gradient at the tissues and lungs becomes greater as more oxygen is being used up and more carbon dioxide is being produced (Figure 2.3). The challenge for the lungs is to maintain resting partial pressures in the alveoli. This is achieved by breathing out the air with less oxygen and more carbon dioxide and then breathing in fresh air to maintain the pressure gradients for diffusion to occur. Otherwise the exercise could not be sustained for long.

**Ventilation during exercise**

The minute ventilation ($V_e$) describes the volume of air being exhaled per minute (and inhaled as we don’t store air).

$V_e$ is determined as the product of the size of each breath ($V_t = \text{tidal volume}$) multiplied by the number of breaths per minute ($B_f = \text{breathing frequency}$).

$$V_e (L.min^{-1}) = V_t (L.breath^{-1}) \times B_f (breaths.min^{-1})$$

**TO DO**

Complete the table below that presents some data collected during an exercise test.

| EXERCISE INTENSITY DURING RUNNING AT PROGRESSIVELY FASTER SPEEDS |
|---------------------------------|----------------|----------------|----------------|----------------|----------------|----------------|
|                                 | Rest 8 km.h^{-1} | 10 km.h^{-1} | 12 km.h^{-1} | 14 km.h^{-1} | 16 km.h^{-1} | 18 km.h^{-1} |
| $V_t$ [L.br^{-1}]              | 0.67            | 2             | 3.3           | 3.6           | 4             |
| $B_f$ [br.min^{-1}]           | 12              | 22.3          | 24.2          | 30            | 38            |
| $V_e$ [L.min^{-1}]             | 8               | 40            | 58            | 98            | 115           |

**Table 2.1**: Comparison of $V_e$, $V_t$, and $B_f$ values at rest and during incremental exercise

As exercise intensity increases, how is the increased ventilation achieved?
During exercise $\dot{V}_E$ typically increases by increasing both $V_t$ and $B_f$ (Table 2.1) to maintain resting gas partial pressures in the lungs and arterial blood supply to the active tissues. As the exercise becomes harder the ventilation increases further, such that in healthy individuals the homeostasis of arterial partial pressure of oxygen is preserved, even at maximal exercise intensities (with the exception of some highly trained athletes). This very tight regulation raises the question of how the ventilation response to exercise is controlled.

Despite a very large amount of research conducted on this topic, there is no single factor in the body that regulates ventilation alone. Instead, researchers have identified a number of factors that can stimulate or inhibit ventilation according to the conditions, e.g. gas partial pressures, acidity, temperature, hormones. The relative contribution of these factors during exercise depends on the characteristics of the exercise, such as intensity, duration and environmental conditions. For example, we know that when exercising at altitude, where the atmospheric partial pressure of oxygen is reduced, receptors that are sensitive to the oxygen content of the blood stimulate increased ventilation. However, at sea level, in the majority of individuals, arterial oxygen content is kept constant. So, although oxygen can be a contributing factor, it would appear that during exercise the ventilation response is actually more sensitive to carbon dioxide increases, particularly during high intensity exercise.

**KEY POINT**

Ventilation increases in response to the increasing intensity of exercise to maintain resting oxygen and carbon dioxide levels in the arterial blood supplying exercising muscles. This is achieved by increasing breathing depth and rate. The control of ventilation is very complex and no single factor is responsible, although carbon dioxide plays an important role.

**TO THINK ABOUT**

Examples of the importance of CO$_2$

The term hyperventilation refers to an increase in ventilation above what is actually required to meet the oxygen demand of the exercise. Such a response means that we exhale more carbon dioxide than necessary and arterial blood levels of carbon dioxide fall. Such a drop in carbon dioxide will reduce our drive to breathe and there are two very different ways in which we can demonstrate this.

**Rebreathing**

In the past, some people recommended breathing into a paper bag when someone has had a ‘panic attack’. The theory behind this advice was that when a person suffers from an acute period of heightened anxiety he or she will hyperventilate. This causes a drop in arterial carbon dioxide which reduces the stimulus to breathe, which in turn can cause the sensation of further panic as the person becomes confused and further hyperventilates making the situation worse.

This is not caused by an excess of oxygen being detected by the brain. By breathing in and out of a paper bag the person breathes some of the carbon dioxide back in and this builds up over a few breathing cycles to restore arterial carbon dioxide levels. Provided the person is able to reduce their state of anxiety their breathing should return to normal.

**NOTE:** This explanation and practice only applies when there is no further pathological cause for the hyperventilation, otherwise rebreathing could actually cause further problems and place the person at increased risk. This is why current first aid guidance advises against using such rebreathing techniques.
Cardiovascular system

Blood

During exercise the primary function of blood is transport to and from various tissues, whether it be transport of gases, nutrients, waste products, hormones or even heat. The total volume of blood in the body is around 5 litres for a 70 kilogram male. Approximately 55% of this blood is fluid known as plasma which contains some dissolved substances, and the remainder is blood cells and platelets.

The primary role of platelets (< 1% of blood volume) is to assist in the process of repair following injury, but the blood cells have a variety of roles. White blood cells (< 1% of blood volume), known as leucocytes, are primarily involved in immune function, protecting the body from infection. Trillions of red blood cells, known as erythrocytes, make up around 40–45% of the blood volume which is known as the hematocrit, although the exact value is dependent on factors such as how well trained an individual is and gender.

The increased volume of carbon dioxide produced during exercise is transported from the muscles to the lungs for exhalation, partly dissolved in blood but mostly in the temporary form of bicarbonate. Oxygen is less soluble in the plasma; only a few per cent of the total oxygen delivered to the active muscles is transported this way. Instead, oxygen temporarily attaches to an iron-rich pigment in the blood called hemoglobin. In the lungs where there is high partial pressure, oxygen easily binds to the hemoglobin. In the active muscle where partial pressure is lower, oxygen detaches and diffuses from the blood into the active tissues. The deoxygenated red blood cells then return to the lungs (via the heart) where more oxygen can bind.

If hemoglobin concentration can be increased by manipulating the hormone erythropoietin (EPO) responsible for stimulating red blood cell production, then more oxygen can be transported and aerobic exercise performance will improve. This is the reason behind many endurance athletes often living and/or training at altitude, where less oxygen availability naturally stimulates more hemoglobin production so that when athletes return to sea level they can perform better.

Holding breath

The world record for breath-holding (19 min 21 s) belongs to an athlete who takes part in the sport of freediving, i.e. diving as deep into water as possible without supplementary air. There is a lot of training and technique to extended breath-holding and it should not be attempted unsupervised as it can leave the person feeling dizzy and could result in drowning or falling once back on land.

Before the breath-hold the diver uses a range of techniques, including meditation and breathing high concentrations of oxygen. However, one important technique is a controlled hyperventilation for a number of minutes. This reduces the arterial carbon dioxide levels and delays the stimulus to breathe considerably, enabling the freediver to stay under water for much longer. As stated, this can be dangerous and should not be attempted unsupervised. The long-term effects of repeated breath-holding are currently unknown.
Unfortunately, there are also illegal methods that are abused in sport to achieve the same goal. For example, blood doping involves removing some blood from an athlete weeks before a competition and storing it while the athlete’s hemoglobin is restored naturally by EPO stimulation. Then just before the competition the stored blood is reintroduced so that hemoglobin concentration is higher than normal, more oxygen can be transported and exercise performance is better. Or athletes can be injected with synthetic EPO to achieve the same goal without even removing blood. The detection of synthetic EPO or blood doping abuse remains a very significant challenge for the World Anti-Doping Agency (WADA), as significant immediate benefits can be achieved for athletes (see chapter 9).

Circulation

Blood is transported around the body through an extensive network of blood vessels. These include the following:

- **Arteries** These are vessels which are relatively large in diameter. They have thick muscular walls as there is considerable pressure exerted from the oxygen-rich blood in these vessels. They are responsible for transport away from the heart to tissues. (Tip: remember that arteries take blood away from the heart.) Arteries then branch into narrower arterioles.

- **Capillaries** Supplied by the arterioles, these are very narrow vessels with very thin walls. They form an extensive branching network through tissues and are the sites of exchange between blood and tissues.

- **Veins** The capillaries link to larger vessels called venules and then larger veins which are the vessels that deliver mostly deoxygenated blood back towards the heart. They are less muscular and fibrous than arteries as pressure is lower, so they are flexible and contain valves to prevent back-flow.

The pump at the center of the cardiovascular system is the heart, which is a sequence of chambers enclosed by walls of specialist muscle fibers called cardiac muscle fibers. The heart is the link between two distinct loops of circulation (look at the central cog in Figure 2.1).

- The pulmonary circulation delivers deoxygenated blood from the right side of the heart to the lungs for oxygenation and then back to the left side of the heart.

- The systemic circulation delivers this oxygenated blood from the left side of the heart to the other tissues of the body where oxygen is used up, and then delivers deoxygenated blood back to the right side of the heart for the cycle to continue. This includes the heart itself which consists of specialized muscle tissue and therefore needs to be supplied with essential blood.
through the coronary arteries. Any disruption to the coronary arteries will result in a heart attack as the cardiac muscle is starved of oxygen and therefore cannot function correctly.

The cardiac cycle

The heart can be considered a four-chamber double-pump system. The left and right sides of the heart work in parallel simultaneously. Each has an atrium that first receives blood from a vein and then pushes it into a larger and thicker-walled ventricle. The ventricle then pushes blood out of the heart into an artery for transport away from the heart.

There are a series of valves between chambers that close and open by force in response to a highly coordinated sequence of muscle contractions. The valves ensure that the system operates in one direction and enable heart muscle contractions to increase pressure in the chambers for ejection of blood (either from an atrium into a ventricle, or from a ventricle into an artery and away from the heart).

Unlike other muscular contractions in the body, the cardiac cycle does not require a nerve stimulation to make the heart muscle contract. This means that the heart makes itself contract and this is called a myogenic contraction. The sequential contractions of the chambers are initiated from a specialist group of cells called the pacemaker (or sinoatrial node, SAN), found in the wall of the right atrium. However, before detailing the stages of the cardiac cycle it is important to point out that the pacemaker firing rate is heavily influenced by many factors.

As well as direct hormonal stimulation (e.g. adrenaline) or manipulation by drugs, the autonomic nervous system (involuntary) can speed up or slow down the pacemaker firing rate by adjusting the relative contributions from the sympathetic and parasympathetic branches respectively. This involuntary control is an example of systems responding to a range of stimuli to try and maintain homeostasis, by increasing or decreasing the release of specific chemicals called neurotransmitters. For example, when exercise begins the parasympathetic stimulation is reduced (this normally keeps the heart rate low) and the sympathetic stimulation is increased, resulting in increased heart rate.
The pacemaker sends an impulse through the walls of the atria (left and right sides) to a second group of specialist cells called the atrioventricular (AV) node (Figure 2.6). This rapid conduction of the impulse causes the muscles in the walls of the atria to contract simultaneously, increasing the pressure in the atria and forcing blood from the atria, through the AV valves, into the ventricles. The AV valves then close.

Following a very brief delay, the impulse is then conducted rapidly via a bundle of specialist cells called the Bundle of His. These cells rapidly conduct the impulse along the very fast conducting Purkinje fibers that spread the impulse along the ventricle walls. This impulse now causes the fibers in the ventricle walls to contract simultaneously, increasing the pressure in the ventricles and forcing blood up and out through the main arteries leaving the heart.

The semi-lunar valves at the openings into the main arteries now close, and while the ventricles relax the cycle has already started again with the atria filling with blood returning to the heart ahead of the pacemaker firing.

**Blood pressure**

In order for blood to flow around the body we have so far recognized the importance of the heart pumping the blood. We have mentioned the contraction and relaxation of the chamber walls to cause changes in the pressure exerted on the blood to drive it through and out of the heart and around the body. Accordingly, if we directly measure the pressure in the blood vessels leaving the heart (arteries), it fluctuates according to the different phases of the cardiac cycle, between very high peaks as the ventricle contracts and forces blood out, to troughs where the ventricle is relaxing and no blood is being pumped out (semi-lunar valves are closed). These represent what are known as the systolic (contracting) and diastolic (relaxing) pressures respectively.

In a resting, healthy adult the typical values would be in the range of 120 mmHg (systolic) to 80 mmHg (diastolic), described as “120 over 80 mmHg”. This is the ideal balance to permit efficient emptying and filling of the heart, but with enough pressure in the system to maintain blood flow to the tissues of the body. The pressure (and extent of the fluctuations) lessens as the blood goes from the arteries to arterioles and then capillaries. The pressure in the venules and veins is comparatively low and consistent, but it is the arterial pressure that is most important and this is what is routinely measured by physicians.
Interpret blood pressure readings

Blood pressure can be measured manually using an adjustable pressure cuff (that can be inflated or deflated gradually to restrict blood flow) attached to a pressure measuring device (a sphygmomanometer) and a stethoscope (to listen to an artery for what are known as the Korotkoff sounds). Alternatively, there are many commercially available automatic blood pressure measuring devices, but the principle is exactly the same.

The cuff is inflated to a pressure higher than systolic pressure (around 150–180 mmHg in a healthy young person) to stop blood flow through the artery as it is compressed (there will be no sound in the artery below the cuff). The cuff is then very slowly allowed to deflate. As the systolic pressure is reached a tapping sound can be heard as blood is intermittently able to get through the blocked artery immediately after each heart contraction only—the systolic pressure is noted at this point. With further deflation the sounds will disappear as full blood flow is restored—at this point the diastolic pressure is noted.

<table>
<thead>
<tr>
<th>ACTIVITY</th>
<th>DIASTOLIC PRESSURE (mmHg)</th>
<th>SYSTOLIC PRESSURE (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>80 kg healthy male</td>
<td>Rest 75</td>
<td>116</td>
</tr>
<tr>
<td></td>
<td>Running 80</td>
<td>180</td>
</tr>
<tr>
<td></td>
<td>Lifting 150</td>
<td>240</td>
</tr>
<tr>
<td>100 kg unhealthy male</td>
<td>Rest 95</td>
<td>150</td>
</tr>
</tbody>
</table>

The table above presents data for a healthy trained 80 kg male at rest and performing two different actions (running fast, a dynamic activity; trying to lift a very heavy object, static but very high forces), as well as resting data for another untrained and unhealthy individual. Answer the following questions.

1. What effect does dynamic exercise have on blood pressure?
2. What effect does static exercise have on blood pressure?
3. Why is one higher than the other?
4. What difference is there between the two participants at rest?
5. Elevated blood pressure is known as hypertension. Why would a higher blood pressure present a challenge that may result in health complications?

Blood flow distribution

During exercise, and even at rest, the diameter of the arteries, arterioles and opening/closing of capillaries needs to be carefully regulated to keep blood pressure at a sufficient level to ensure cardiovascular function. This is achieved by involuntary control of the smooth muscle which lines the walls of the arteries and arterioles, and also around tiny sphincters throughout the network of capillaries. If all of this smooth muscle relaxed then there would not be sufficient pressure to return blood to the heart and the cardiac cycle could not function. Therefore, the nervous system and cardiovascular system interact carefully so that there is sufficient relaxation of some vessel walls and contraction of others to ensure that enough blood is passing through all organs requiring exchange, yet blood pressure is maintained.

During exercise the muscles that are being used become the main demand on blood flow, as more oxygen and nutrients are required and more waste products and heat need to be removed. Therefore, in addition to the increases
in cardiac output (below), more blood is directed towards the active muscles by dilating the arterioles supplying the muscles and opening more of the capillary network within the muscles. However, to prevent a resulting catastrophic drop in blood pressure throughout the whole system, the vessels supplying other organs in the body constrict and many of the capillaries are closed so that blood flow is reduced to these organs.

As shown in Figure 2.7, some essential organs such as the brain and heart are protected so that they still have sufficient supply (life could be compromised if either had insufficient blood flow), but active muscles can demand as high as 90% of the total blood flow during exercise compared to only 20% at rest.

**Figure 2.7**

### Acute cardiovascular responses to exercise

With ventilatory responses deemed to be sufficient to maintain efficient gas exchange in healthy individuals, even during maximal exercise, the cardiovascular system is crucial to maintain function and attempt to maintain homeostasis in the face of the exercise challenge. Previous sections have highlighted various ways in which this is achieved to an extent, but it is the responses of the heart to dynamic exercise that can truly be considered “central”. Accordingly, the responses of the heart are very accurately regulated according to the demands of the exercise.

This is nicely illustrated by exploring the increase of blood flow out of the heart during exercise and how this is achieved. Cardiac output is defined as the amount of blood ejected from the left side of the heart (and therefore supplying the whole body except the lungs) in litres per minute. Cardiac output is determined by how quickly the heart is beating (heart rate in beats per minute) and the amount of blood being ejected with each contraction (stroke volume in millilitres per beat), according to the following equation:

\[
\text{Cardiac output} = (\text{Heart rate} \times \text{Stroke volume}) / 1000
\]

In order to achieve the increases in cardiac output required during exercise the heart beats faster (increased heart rate) and the heart fills and empties more during each contraction (increased stroke volume). As exercise becomes progressively harder, the heart rate and stroke volume both increase until their respective maximum rate and volume are achieved. As maximum cardiac output is reached so too is exhaustion and exercise cannot continue at this intensity (see section below).

In contrast, during prolonged sub-maximal exercise at a fixed intensity (endurance activities), the cardiac output is maintained at the same level.
throughout as the demand stays constant with stroke volume and heart rate at values higher than rest. Interestingly, however, while cardiac output stays constant eventually the heart rate starts to increase slightly and progressively. This is known as cardiovascular drift and reflects a decline in stroke volume, primarily due to changes in thermoregulation.

TO DO
The table below shows the cardiovascular responses during dynamic whole-body exercise for 2 adult males of similar age (20 years old) and size (1.8 m, 70 kg), but one is sedentary and the other is a well-trained endurance athlete. Data reflect 3 levels of exercise intensity: rest; sub-maximal exercise (Sub-max.) walking at the same speed; and maximal exercise (Max.) at the point of exhaustion.

<table>
<thead>
<tr>
<th>INTENSITY</th>
<th>UNTRAINED ADULT MALE</th>
<th>TRAINED ADULT MALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats.min⁻¹)</td>
<td>Rest 75</td>
<td>50</td>
</tr>
<tr>
<td>Sub-max. 110</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>Max. 197</td>
<td>195</td>
<td></td>
</tr>
<tr>
<td>Stroke volume (ml.beat⁻¹)</td>
<td>Rest 60</td>
<td>90</td>
</tr>
<tr>
<td>Sub-max. 85</td>
<td>112</td>
<td></td>
</tr>
<tr>
<td>Max. 120</td>
<td>190</td>
<td></td>
</tr>
<tr>
<td>Cardiac output (L.min⁻¹)</td>
<td>Rest 4.6</td>
<td>4.5</td>
</tr>
<tr>
<td>Sub-max. 9.4</td>
<td>9.0</td>
<td></td>
</tr>
<tr>
<td>Max. 19.7</td>
<td>32.2</td>
<td></td>
</tr>
</tbody>
</table>

1 Evaluate the effect of training on the cardiovascular responses to sub-maximal and maximal dynamic exercise.
2 Aside from any differences in training status, predict any differences that you would expect if the data in the above table were compared to an adult female.

TO THINK ABOUT
Sub-maximal cardiovascular responses are different in children and adults. Both boys and girls have a lower cardiac output than adults at a given absolute sub-maximal rate of work. This lower cardiac output is attributable to a lower stroke volume, which is partially compensated for by a higher heart rate. The table below shows the data from a study comparing cardiovascular responses to cycling and treadmill running in 7–9 year old children versus 18–26 year old adults.

<table>
<thead>
<tr>
<th>CARDIAC OUTPUT L.min⁻¹</th>
<th>STROKE VOLUME ml</th>
<th>HEART RATE beats.min⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHILD</td>
<td>ADULT</td>
<td>CHILD</td>
</tr>
<tr>
<td>Cycle 60 W</td>
<td>9.4</td>
<td>12.4</td>
</tr>
<tr>
<td>Run 3 mph</td>
<td>6.7</td>
<td>12.3</td>
</tr>
</tbody>
</table>

Differences in sub-maximal cardiovascular responses between children and adults are related to the smaller hearts and a smaller amount of muscle doing a given rate of work in the children.
Functional capacity of the cardiorespiratory systems

It is clear that the response of the human body to exercise requires the successful integration and regulation of a number of systems in the body. There are limits to how hard these systems can be pushed and this is reflected in the varied durations and intensities of exercise that people can tolerate. The most commonly used marker of an individual’s aerobic fitness brings us back to the importance of \( \dot{\text{VO}}_2 \) discussed at the start of this chapter.

Maximal oxygen uptake (\( \dot{\text{VO}}_2 \max \)) quantifies the maximum rate that an individual can take in and use oxygen. The \( \dot{\text{VO}}_2 \) is directly assessed by measuring the gas concentration and volume of air being breathed out at progressively increasing intensities of exercise. As the oxygen demand increases so too does the \( \dot{\text{VO}}_2 \), until the person approaches their limit, that is their \( \dot{\text{VO}}_2 \max \). At this time, even if intensity (and therefore oxygen demand) is further increased, the \( \dot{\text{VO}}_2 \) cannot increase any further and the person will stop exercising as they can no longer continue.

For this reason, the \( \dot{\text{VO}}_2 \max \) is sometimes known as aerobic capacity and this is why it is a parameter of interest for physiologists working with both severely limited patients and elite endurance athletes. The patients will have a very low \( \dot{\text{VO}}_2 \max \) and therefore cannot cope with what may seem relatively easy exercise to us. Whereas the elite endurance athletes will have very high \( \dot{\text{VO}}_2 \max \) values and are therefore capable of the impressive endurance performances that we see in high performance sport.

Fick equation

In maximum exercise the Fick equation summarises the important relationship between maximum cardiac output, maximum arterio-venous oxygen difference, and \( \dot{\text{VO}}_2 \ max \):

\[
\dot{\text{VO}}_2 \max = \text{Maximum cardiac output} \times \text{Maximum arterio-venous oxygen difference}
\]

As well as a larger cardiac output, other positive endurance training adaptations include both a more effective blood “shunting” (redistribution) and increases in skeletal muscle microcirculation (ratio of capillaries to muscle fiber). This helps to increase tissue oxygen extraction during intense exercise for both children and adults. Higher heart rates in children compared to adults during sub-maximal exercise do not fully compensate for the smaller stroke volume of the children. Children have a smaller cardiac output relative to adults at a given sub-maximal exercise oxygen consumption. As a result, the arterio-venous oxygen difference increases to meet the oxygen requirements of the children.

During sub-maximal exercise a higher arterial-mixed venous \( \text{O}_2 \) difference \([a-v]\)\( \text{O}_2 \) in children also helps compensate for their lower cardiac output compared to adults to achieve a similar \( \dot{\text{VO}}_2 \). The table below shows the data from a study comparing responses to cycling and treadmill running in 7-9 year old children versus 18-26 year old adults.

<table>
<thead>
<tr>
<th></th>
<th>CARDIAC OUTPUT L.min(^{-1})</th>
<th>(A-V)O(_2) ml per 100 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CHILD</td>
<td>ADULT</td>
</tr>
<tr>
<td><strong>Cycle 60 W</strong></td>
<td>9.4</td>
<td>12.4</td>
</tr>
<tr>
<td><strong>Run 3 mph</strong></td>
<td>6.7</td>
<td>12.3</td>
</tr>
</tbody>
</table>

**TO DO**

From the data provided in the table, use the Fick equation to calculate the \( \dot{\text{VO}}_2 \max \) of children and adults when cycling at 60 W.
When comparing VO₂ max values between different populations it is crucial to recognise that the values can be expressed in two formats:

- **Absolute VO₂ max** is reported in L.min⁻¹.
- **Relative VO₂ max** is the same value but normalised according to body mass in ml.kg⁻¹.min⁻¹.

For activities that are considered weight-bearing it is more appropriate to use the relative VO₂ max values as this makes an attempt to account for individual differences in size and mass. This is important as differences in size and mass explain the majority of the variability in absolute VO₂ max values between individuals, due to factors such as active muscle mass, heart size, blood volume etc.

For example, an untrained healthy adult with a body mass of 70 kg may have an absolute VO₂ max of 3.0 L.min⁻¹, which means a relative VO₂ max of 42.9 ml.kg⁻¹.min⁻¹ (3.0 × 1000 to convert L to ml, then divide by 70kg).

In contrast a 58 kg female hockey player may also have an absolute VO₂ max of 3.0 L.min⁻¹, yet her relative VO₂ max of 51.7 ml.kg⁻¹.min⁻¹ reflects her training adaptations that mean she will be able to run at faster speeds and for longer than the untrained male. Highest values of VO₂ max have been recorded in cross-country skiers (over 90 ml.kg⁻¹.min⁻¹ in males and over 75 ml.kg⁻¹.min⁻¹ in females), however in those who are seriously ill the values can be considerably lower than even 20 ml.kg⁻¹.min⁻¹. The general pattern is therefore for relative VO₂ max to reflect cardio-respiratory fitness, although it must be recognised that there is still a lot of individual variation and these are population averages. For 20-year old males, an untrained healthy relative VO₂ max may be 40–45 ml.kg⁻¹.min⁻¹, with moderately trained in the range 45–55 ml.kg⁻¹.min⁻¹, professional team sport athletes 50–60 ml.kg⁻¹.min⁻¹ and top endurance athletes higher than 65 ml.kg⁻¹.min⁻¹. Changes in size between these groups mean that the training effects on absolute VO₂ max will be less obvious.

**Gender**

Regardless of training status gender also has an effect. Absolute VO₂ max values are considerably lower in age-matched females, primarily due to the size differences. However, even when expressed in relative terms active, healthy adult females typically have lower VO₂ max values than males. For 20-year old females an untrained healthy range may be 35–40 ml.kg⁻¹.min⁻¹, moderately trained 40–50 ml.kg⁻¹.min⁻¹, professional team sport athletes 45–55 ml.kg⁻¹.min⁻¹ and endurance athletes higher than 55–60 ml.kg⁻¹.min⁻¹. The primary factors that contribute to these gender differences are related to body composition (dividing by body mass does not account for the naturally higher percentage of non-oxygen-using body fat in females) and hemoglobin concentration to a lesser extent (males have slightly more hemoglobin than females).

**Age**

A further important factor that influences VO₂ max is age. During childhood and adolescence absolute VO₂ max increases according to patterns of growth and maturation, peaking in the early 20s for males and mid-teens for females. So children typically have much lower absolute VO₂ max values than adults due to their size. However, when the values are normalized to body mass male children and adolescents have very similar values to healthy adults, i.e. relative VO₂ max is very similar in male adults and children. Does this mean that a trained boy should be able to run a marathon in the same time as trained man? Children of course could not achieve such fast times and this nicely illustrates that normalising VO₂ max to body mass cannot fully explain differences in
actual endurance performance, for a variety of reasons. In females the patterns of growth and maturation are clearly different (typically peaking earlier by a few years and with more accumulation of body fat and less muscle mass during puberty) and the effects on VO$_{2}\text{max}$ reflect this. In girls absolute VO$_{2}\text{max}$ increases with growth, peaking in the mid-teens. However, relative VO$_{2}\text{max}$ actually tends to decrease from early-teens in girls, partly due to the changes in body composition, potentially in addition to changes in physical activity patterns.

From adulthood, in males and females, the relative VO$_{2}\text{max}$ typically declines by approximately 1% each year on average. This reflects a gradual decline in the maximum heart rate that can be achieved, although again changes in physical activity patterns may contribute. So, for a healthy untrained 20-year-old with a VO$_{2}\text{max}$ of 45 ml.kg$^{-1}$.min$^{-1}$ natural ageing would mean that at 45 and 70 years old the VO$_{2}\text{max}$ would be expected to have declined to 35.0 and 27.2 ml.kg$^{-1}$.min$^{-1}$ respectively. This implies a steady decline in endurance capacity as we get older and any illness or injury will speed-up this decline. However, very importantly this does not mean that all elderly people have very low VO$_{2}\text{max}$ values and limited endurance capacity. Although the size of training response gets lower as we get older, physical exercise can still induce significant improvements in VO$_{2}\text{max}$ in the elderly such that a trained 65-year-old may well have a higher VO$_{2}\text{max}$ than an untrained and over-weight 30-year-old. Indeed Masters athletes compete all over the world until the end of their lives. A remarkable example is that of Fauja Singh, a 100-year-old Briton who completed the Toronto Waterfront marathon in 2011.

**Type of exercise**

A final factor that can further influence recorded values for VO$_{2}\text{max}$ is the type of exercise that is being performed. Within the same individual the highest rate of oxygen uptake that is recorded will be different dependent on whether the person is running or cycling for example. As more muscle mass is being
used during running (compared to cycling the upper body and postural muscles are being used more as this is a weight-bearing activity) it would be expected that a higher VO2max would be recorded compared to cycling. This is the main reason underpinning why the highest observed values are in cross-country skiers compared to runners – as cross-country skiing places more oxygen demand on the upper body, in addition to the lower body and postural muscles that are working hard in both types of exercise.

What limits VO2max?

Although we can see from Figure 2.1 that a limitation could occur anywhere in the oxygen transport system when VO2max is reached, it is widely believed that in the majority of healthy individuals the primary limitation is the capacity of the cardiovascular system to deliver oxygen. There are exceptions to this rule, including illness and extremely high aerobic fitness levels, but in most cases it is believed that the ventilation system and oxygen use at the muscle do not cause someone to reach VO2max.

How does training increase VO2max?

The training responses that can be seen following a period of aerobic training support the idea that training can increase VO2max. The increases in VO2max that are observed are underpinned, at least in part, by training-induced changes in the heart and cardiovascular system (central adaptations), as well as being helped by some changes within the muscle (peripheral adaptations). Centrally, the main training response is an increase in stroke volume at sub-maximal and maximal values. In contrast, the heart rate response becomes lower at sub-maximal intensities and the maximum heart rate is actually unchanged with training, it just isn’t reached until the person is working harder than before training (their capacity has improved).

The mechanism responsible for the increased stroke volume is mainly an increase in the volume of the left ventricle, meaning that more blood can fill the ventricle ahead of each contraction. As well as some changes in the blood, the muscles also develop more capillaries so that more blood can supply oxygen to the exercising muscles. In terms of oxygen use, there are also some adaptations that occur within the muscle itself to increase the amount of oxygen being extracted from the blood as it passes. Collectively, these central and peripheral adaptations permit an individual to exercise harder as their VO2max has increased.

TO DO

‘To become an Olympic champion you must choose the right parents!’
Said by a very famous Swedish exercise physiologist Per-Olof Astrand. Evidence from training studies suggests that at least some of the variation in VO2max between individuals can be explained by genetic variation. However, there is little doubt that training can have a positive impact on VO2max. This raises a classic question of fitness—is it due to nature (genetics) or nurture (training)?

Some very insightful research has compared the training adaptations of identical twins with non-identical twins. Interestingly, both improved but the responses were more similar in the identical than non-identical twins, despite identical training programmes and similar initial fitness levels.

→ Why does such research imply that genetics must at least play some part?
→ Why did using twins in both groups help to answer the nature versus nurture question?

A good way to think of this is that each person may have a ‘ceiling’ VO2max that is determined by their genes, but this will only become limiting once that person has trained and increased their VO2max as much as possible. Therefore, people with a low untrained VO2max cannot blame their genes as they have not reached their genetic ceiling.

KEY POINT

VO2max is the maximal rate of oxygen uptake and represents someone’s maximal aerobic capacity. It is affected by factors such as training status, age and gender.

KEY POINT

Aerobic training can increase VO2max. The main mechanism is an increased stroke volume, although other adaptations in the cardiovascular and muscular systems also contribute.
SUMMARY

- Homeostasis is the maintenance of a constant internal environment.
- The ventilatory and cardiovascular systems function together to maintain homeostasis during exercise.
- Ventilation functions to ensure that blood leaving the lungs is oxygenated and low in carbon dioxide. Breathing occurs by repeated contraction and relaxation of muscles around the chest cavity.
- No single factor controls the ventilation response, although carbon dioxide plays an important role.
- Static lung volumes are more related to size than health or fitness, although dynamic volumes are more functional and sensitive to illness.
- Gas exchange in the lungs and tissues occurs by diffusion from higher partial pressure to lower partial pressure, through thin capillary, alveoli and cell walls.
- During exercise ventilation is increased by increasing the depth and frequency of breathing.
- Blood consists of fluid (plasma) and cells with various functions including transport. Oxygen attaches to hemoglobin in red blood cells for transport.
- The circulation system is made up of a pump (the heart) and a series of blood vessels, whose diameter and opening can be controlled by smooth muscle in the walls and sphincters.
- The heart contains four chambers (two atria and two ventricles), with the left side supplying blood to the systemic circulation and the right side supplying the pulmonary circulation.
- A heartbeat consists of a series of carefully coordinated contractions of the heart muscle tissue to eject blood from chamber to chamber and out of the heart. The initial impulse is generated within the heart itself, but the rate of firing can be controlled by other factors.
- Blood pressure must be maintained to ensure blood flow is high enough. This is achieved through constriction and relaxation of vessel walls and sphincters. During exercise this ensures that more blood is diverted away from other organs towards the muscle.
- Total blood flow (cardiac output) increases in proportion to the intensity of exercise up to a maximum value. This is achieved by increasing heart rate and stroke volume.
- $\text{VO}_2\text{max}$ is the maximal rate of oxygen uptake achieved during maximal aerobic exercise.
- Training, age and gender all affect someone’s $\text{VO}_2\text{max}$. Training increases $\text{VO}_2\text{max}$ primarily through increases in maximal stroke volume, although other peripheral adaptations can also contribute.

Self-study questions

1. Why might a doctor be interested in assessing $\text{VO}_2$ in an exercise test?
2. Describe the process of breathing and comment on how exercise affects this process.
3. Insert higher or lower and from or to into the correct places in the following statements.
   - The partial pressure of oxygen is ............... in arterial blood supplying exercising muscles than in the muscle tissue. Therefore, oxygen will diffuse ........... the blood ........... the muscle.
   - The partial pressure of carbon dioxide is ............... in the lungs (alveoli) than in the blood returning from exercising muscles. Therefore, carbon dioxide will diffuse ........... the lungs ........... the blood.
4 What is the name of the pigment that binds oxygen for transport in the blood? Which type of blood cell is it found in?

5 Draw and label the four chambers of the heart, including the valves.

6 List the names of the specialist cells found in the heart that generate and then relay the electrical signal that causes the heart to contract.

7 Explain what will happen to the smooth muscles in the arterioles and capillaries within active muscle, compared to within the kidney during exercise.

8 Describe the responses of heart rate, stroke volume and hence cardiac output during exercise.

9 Define VO2max and discuss why it is considered to be of functional importance from a health and sports perspective.

10 How does aerobic training increase VO2max?

**DATA BASED QUESTION**

Thirteen children (9–10 years old) completed two tests to determine their VO2 peak (litre/min). The “ramp” test involved cycling for 3 min at 10 W and then increasing the workload by 10 W per min. The “supra-maximal” test commenced with 2 min cycling at 10 W and then involved cycling at 105% of the peak power achieved during the “ramp” test. Both tests stopped when the children had a drop in cadence below 60 rpm for five consecutive seconds, despite encouragement to maintain the required workload. Their physiological responses are shown in the table below.

Peak physiological responses during the ramp and supra-maximal tests

<table>
<thead>
<tr>
<th>Variable</th>
<th>Ramp test</th>
<th>Supra-maximal test</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2 peak (L.min^{-1})</td>
<td>1.690 [0.284]</td>
<td>1.615 [0.307]</td>
</tr>
<tr>
<td>Heart rate peak (beats.min^{-1})</td>
<td>202 [7]</td>
<td>196 [8]</td>
</tr>
<tr>
<td>RER peak</td>
<td>1.11 [0.06]</td>
<td>1.07 [0.13]</td>
</tr>
<tr>
<td>Cardiac output peak (L.min^{-1})</td>
<td>15.10 [4.82]</td>
<td>14.64 [4.51]</td>
</tr>
<tr>
<td>Oxygen extraction peak (ml.min^{-1} per 100 ml)</td>
<td>12.61 [2.57]</td>
<td>12.28 [2.50]</td>
</tr>
</tbody>
</table>

Data are reported as mean (SD).


1 State which test resulted in:
   i) the highest VO2 peak
   ii) the highest heart rate
   iii) the lowest cardiac output.

2 Distinguish between the standard deviations for:
   i) RER peak
   ii) oxygen extraction peak.

3 Suggest reasons for the different physiological responses to the two tests.