Learning objectives

After reading, considering and discussing with a study partner the material in this chapter you should be able to:

➤ describe the structure and function of nervous tissue
➤ distinguish between the divisions within the nervous system
➤ explain the sodium-potassium pump and its role in nerve impulse conduction
➤ teach others about how a nerve impulse or action potential is conducted along a neuron
➤ identify the differences between the central and peripheral nervous systems
➤ list the key endocrine organs, highlighting those that have another role within the body in addition to their endocrine function
➤ explain the essential components of endocrine messaging
➤ summarise the function of a number of key hormones
➤ describe the relationship and interaction between the nervous and endocrine systems

Human cells are the smallest living units within the body and work in specialised groups to form our tissues and organs. Under the control of the nervous and endocrine systems, the body’s other organ systems carry out specialised functions to maintain homeostasis, that is, to provide a stable internal environment for optimal physiological functioning. The nervous system is the main controller of the body. Through electrical and chemical signals, the nervous system is the immediate response mechanism of the body. It collects and interprets information from the senses and effects the appropriate response within milliseconds. Through interaction with the nervous system, the endocrine system plays a role in the control of metabolic activity through the release of hormones (chemical messengers) into the blood and lymph. Compared to the nervous system, the endocrine system is slower to initiate a response. However, the response is sustained longer than those initiated by the nervous system. The combined work of the nervous and endocrine systems allows the internal environment of the body to remain within a narrow range required for cell and organism survival.

The first section of this chapter includes an overview of the organisation, structure and functioning of the nervous system, with the endocrine organs and hormonal functions discussed in section two. The responses of these two control systems to acute exercise are considered in the final section.
4.1 The nervous system

The nervous system is comprised of the brain, spinal cord, ganglia, nerves and sensory receptors. Despite its importance to the body, the nervous system comprises less than 5% of total body mass, with a mass of around 2 kg. The nervous system plays multiple roles during sports performance, including the initiation of muscular contractions that bring about movement. It is through the nervous system that we are able to respond to the ever-changing environment of the sports field, for example, a defender coming in to make a tackle, a tennis ball lobbed over your head, maintaining balance on a gymnastics beam. In a movement response, such as the start of a 100 m sprint race, nerve impulses from the brain or spinal cord link with the effector organ (muscle fibres) at neuromuscular junctions where the nervous stimulation is passed to the muscle fibre to initiate contraction of that fibre. This movement response is the result of three overlapping functions of the nervous system:

1. Sensory input – the information collected via sensory receptors which allows the nervous system to monitor changes both within, and outside, the body (e.g. the BANG of the starter’s gun).
2. Integration – the method of processing and interpreting the sensory input and deciding on the appropriate course of action.
3. Motor output – the response initiated by the nervous system by activation of the effector organs (muscles and glands) (e.g. the sudden, explosive push off the starter blocks).

In addition to their roles in sports performance, these three overlapping functions of the nervous system enable the maintenance of homeostasis, and hence, survival.

Divisions of the nervous system

The nervous system is divided into two main divisions, the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS includes the brain and spinal cord and is responsible for interpreting the sensory input and dictating the motor response. The PNS is the part of the nervous system outside the CNS and consists of peripheral nerves (12 pairs of cranial nerves, 31 pairs of spinal nerves), sensory receptors and ganglia. These nerves serve as the communication network between all parts of the body and the CNS. The PNS is further subdivided into the afferent (sensory) and efferent (motor) divisions. All divisions of the nervous system are illustrated in Figure 4.1.

Key point

The nervous system comprises:

➤ the central nervous system (the brain and spinal cord)
➤ the peripheral nervous system (all nerves).

The 43 pairs of nerves within the body are formed from bundles of neurons.

Each neuron is comprised of dendrites (for receiving information or stimuli), a cell body and a single axon that propagates nerve impulses.

The sensory or afferent division of the PNS comprises sensory (afferent) neurones which, as the name suggests, detect internal and external (to the body) signals and relay them to the central nervous system. There are five main types of sensory receptors within the body:

- Mechanoreceptors are responsive to stretching and pressure and are associated with the senses of touch, pressure, sound and balance. Proprioception, the awareness of where the body is in space, is a vital sense for many sports and is brought about through specific mechanoreceptors that are also referred to as proprioceptors.
- Photoreceptors are sensitive to light and are the basis for the sense of vision.
- Chemoreceptors detect chemicals that bind with their cell membranes and are responsible for the senses of smell and taste as well as detecting chemical changes within the body. The detection of increased CO₂ in the blood vessels, which is a trigger for an increase in heart and respiration rates, is brought about by chemoreceptors.
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- **Thermoreceptors** detect changes in temperature and relay this information to the brain and spinal cord.
- **Nociceptors** detect pain and respond to mechanical, thermal and chemical sources which can be from internal or external sources.

Once sensory information has been relayed to and processed by the CNS, the nerves of the motor or efferent division transmit a response to the relevant tissues, organs or systems. We have conscious control over some motor responses, such as the movement of our limbs. However, others, such as the change in heart rate that occurs when playing sport, we have little or no control over. The somatic nervous system describes the parts of the nervous system which give us control of our skeletal muscles. The autonomic nervous system (ANS) refers to those parts over which the brain and spinal cord have control without us consciously having to think, such as breathing and the movement of food along the GI tract. The final division of the nervous system is within the autonomic nervous system which can be further separated into the sympathetic and parasympathetic divisions. The sympathetic division is responsible for speeding up the body systems in what is sometimes called the ‘fight or flight’ response. In contrast, the parasympathetic division is responsible for returning the body to normal functioning and can be thought of as the ‘rest and digest’ response.

**Knowledge integration question**

Explain the fight or flight response.

**Nervous tissue**

At a cellular level the nervous tissue is made up of two principal types of cell, **neurons** and **neuroglia** (or **glial cells**). Neurons (nerve cells) are excitable cells that process and transmit information by electrical and chemical signalling. In contrast, the much smaller neuroglia, are non-excitable cells that provide the neurons with structural and metabolic support. Neurons and neuroglia are formed during development from neuroblasts, the nervous system equivalent of the osteoblasts found in the skeletal system.

**Neurons**

Neurons and the nerves they comprise, in common with muscle tissue, are **excitable**, meaning that they can conduct an electrical impulse. This **nerve impulse**, or **action potential**, is the signal that passes along a neuron. **Sensory neurons** conduct information from the tissues and organs of the body to the brain and spinal cord and **motor neurons** relay messages from the CNS to the response organs. Some specialised neurons pass information from one neuron to another and as a result are called **interneurons** or **association neurons**.
The nervous system

Nerve cells, such as those responsible for bringing about muscular contraction of the lower leg and feet, are over one metre in length. Like other cells within the body, the cell body (soma or perikaryon) comprises of a cell membrane, a nucleus and cytoplasm. The plasma membrane of the cell body and dendrites is called the neurilemma. However, the cell membrane of the axon is usually referred to as the axolemma because the function of axons necessitates differences in structure when compared with the rest of the cell. In a similar way the cytoplasm of the cell body is known as neuroplasm whereas that in the axon is called axoplasm. The cytoplasm of neurons contains many organelles common to other cells, such as lysosomes, ribosomes, mitochondria and a Golgi apparatus. In addition to generalised organelles, they contain some neuron-specific components, such as neurofibrils, neuron-specific microtubules and Nissl bodies. Neurofibrils are comprised of protein filaments and are designed to maintain the structure of the cell body. The neuron-specific microtubules provide a transport structure through which substances pass between the cell body and the axon. Nissl bodies are small rough endoplasmic reticulum structures that are responsible for synthesising proteins to be used for growth and repair.

Axons propagate action potentials to the receiving neuron or organ. Due to their often great length and lack of Nissl bodies, axons have two specialist transport systems (slow and fast axonal transport) that enable essential substances to be moved between the axon and the cell body. Slow axonal transport (transports at a speed of about 0.1 mm per hour) is responsible for the movement of axoplasm and fast axonal transport (around 12 mm per hour – ten times quicker than its slow counterpart) is responsible for moving

Neuron structure

Neurons are normally made up of three main components, dendrites, a cell body and a single axon. Dendrites receive information (sensory or neurotransmitter stimuli) which is conveyed toward the cell body for processing. The axon then generates nerve impulses and conducts them away from the cell body, along the axolemma (axon plasma membrane), to the secretory axon terminals. The axon is a specialised cellular filament that arises from the cell body at a site known as the axon hillock. There are three main structural classifications of neurons related to the number of processes extending from the cell body. These are shown in Figure 4.2 with the direction of impulse travel indicated by an arrow. Although all neurons have only one axon leaving the cell body, multipolar neurons have many dendrites connecting to the cell body, bipolar neurons have a single dendrite coming to the cell body, and unipolar neurons have a joint dendrite axon process connecting with the cell body. Generally, motor neurons are multipolar in structure and sensory neurons are mainly unipolar although some specialist neurons, such as those of the rods and cones of the eye, are bipolar. Figure 4.3 shows a multipolar neuron in more detail.

Neurons vary greatly in both length and diameter depending on their function within the body. Some

Key point

There are two types of nervous system cells:
- **Neurons** carry action potentials and form nerves
- **Neuroglia** provide structural and metabolic support
Schwann cells form a sheath around peripheral axons. Wherever a Schwann cell covers an axon, the outer surface of the Schwann cell is called the neurilemma (noor-i-LEM-uh). Most axons in the PNS, whether myelinated or unmyelinated, are shielded from contact with interstitial fluids by Schwann cells.

Figure 4.3 The structure of a multipolar neuron.

organelles (such as mitochondria) and proteins between the cell body and axon terminals. After information from the dendrites is received and processed by the nucleus the nerve impulse is propagated from a trigger zone which is situated just below the axon hillock. Although each neuron has only one axon leaving the cell body, axons occasionally have branches from them called axon collaterals which lead to another set of axon terminals. Whether an axon is undivided or has collaterals, it usually has profuse branching at the end, enabling communication with many target cells (see Figure 4.2). The axon terminals form the link with other tissues or neurons. When the axon terminals of
a motor neuron meet a muscle fibre the junction is called a neuromuscular junction, whereas the junction between two neurons is called a synapse.

The axons of many neurons are myelinated, meaning they are covered by a myelin sheath. This is a protective layer of segmented, electrically insulating, fatty material and is found particularly on neurons that are long or large in diameter. The principle function of the myelin sheath is to greatly increase the speed of transmission of nerve impulses. Type A axons, which comprise all efferent neurons and many afferent neurons (e.g. proprioceptors) within the peripheral nervous system, are the largest diameter fibres (between 4–20 μm) and are myelinated. The larger surface area and myelinated nature of the axons means that nerve impulses travel at the highest speeds in these fibres which can be as fast as 140 m•sec⁻¹ or over 300 mph. Type B axons are smaller (between 2–4 μm), but still myelinated and as a consequence impulses still travel at relatively fast speeds, up to 18 m•sec⁻¹ or around 36 mph. Type B axons form the majority of pre-ganglionic autonomic nervous system neurons (described in the section on the PNS). Type C, the smallest axons, are up to 2 μm in diameter and are unmyelinated, consequently, nerve impulses travel at slower speeds of 1–2 m•sec⁻¹ or up to 4 mph. Post-ganglionic neurons within the autonomic nervous system predominantly comprise Type C axons.

Knowledge integration question

Explain the differences between neurons and neuroglia.

Neuroglia

Neuroglia are much smaller cells than neurons and are the most common type of nerve tissue cell found in the body, comprising over half of the nervous system. They provide structural and metabolic support for the nervous system and are found in both the central nervous system and the peripheral nervous system. There are four types of neuroglia found in the CNS; astrocytes, ependymal cells, microglia and oligodendrocytes, which can be seen in Figure 4.4, and two types found in the PNS, Schwann cells and satellite cells (see Figure 4.3).

Astrocytes are the most abundant CNS neuroglia and have several functions which include providing support for neurons, maintaining chemical balance and regulating nervous tissue growth. Ependymal cells line the central cavities of the brain and spinal cord and have hair-like structures protruding from them called microvilli and cilia which help with the flow of cerebrospinal fluid (CSF). Microglia are involved in repair of neurological tissue and help by removing debris from the interstitial environment. Oligodendrocytes are responsible for creating a myelin sheath around neurons of the CNS and to create a structural framework for the CNS. Schwann cells are the equivalent of oligodendrocytes in the PNS and, as can be seen in Figure 4.3, each Schwann cell is responsible for myelination of one section of a neuronal axon. Schwann cells also have a role in providing a coordinated structure and protection for unmyelinated neurons and can enclose up to 10 or 20 of these neurons. Satellite cells (Figure 4.3), the second form of neuroglia found in the PNS, help to supply nutrients to the surrounding neurons.

Nerves

Neurons are found collected in groups or bundles, with unmyelinated neurons forming the grey matter of nervous tissue and myelinated neurons forming the white matter (myelin is whitish in colour and formed from lipid and protein). The structure of a nerve, which can be seen in Figure 4.5, has some similarities with muscle in that individual fibres or neurons are grouped together into fascicles and are surrounded by three protective sheaths. The protective sheaths for nerve fibres are the epineurium, the perineurium and the endoneurium.

The structure of an unmyelinated nerve also includes Schwann cells that wrap around the fibres, but unlike myelinated nerves, many fibres are encapsulated by one Schwann cell, but are not individually myelinated (see Figure 4.3). The majority of nerves, however, are myelinated which increases the speed of nervous transmission. Within the PNS, the presence of Schwann cells, and the neurilemma surrounding each axon, provide a protective tube in the event of injury (providing the nerve fibres remain aligned). In this situation, the protective tube created by the Schwann...
Many oligodendrocytes cooperate in the formation of a myelin sheath along the length of an axon. Such an axon is said to be myelinated. Each oligodendrocyte myelinates segments of several axons. The relatively large areas of the axon that are thus wrapped in myelin are called internodes. The small gaps of a few micrometers that separate adjacent internodes are called nodes. In dissection, myelinated axons appear glossy white, primarily because of the lipids within the myelin. As a result, regions dominated by myelinated axons constitute the white matter of the CNS. Not all axons in the CNS are myelinated. Unmyelinated axons may not be completely covered by the processes of oligodendrocytes. Such axons are common where relatively short axons and collaterals form synapses with densely packed neuron cell bodies. Areas containing neuron cell bodies, dendrites, and unmyelinated axons have a dusky gray color, and they constitute the gray matter of the CNS.

Figure 4.4 The structure of neuroglia.
Every segment of the spinal cord is connected to a pair of spinal nerves. Surrounding each spinal nerve is a series of connective tissue layers continuous with those of their associated peripheral nerves. These layers, best seen in sectional view, are comparable to those associated with skeletal muscles.

1. Connective Tissue Layers of a Spinal Nerve
   - **Epineurium**: The outermost covering of the nerve, consisting of a dense network of collagen fibers.
   - **Perineurium**: The middle layer, extending inward from the epineurium. These connective tissue partitions divide the nerve into a series of compartments that contain bundles of axons or fascicles.
   - **Endoneurium**: The innermost layer, consisting of delicate connective tissues that extend from the perineurium and surround individual axons.

2. Blood vessels penetrate the epineurium and branch within the perineurium. Capillaries leaving the perineurium branch in the endoneurium and supply the axons and Schwann cells of the nerve and the fibroblasts of the connective tissues.

3. Arteries and veins penetrate the epineurium and branch within the perineurium. Capillaries leaving the perineurium branch in the endoneurium and supply the axons and Schwann cells of the nerve and the fibroblasts of the connective tissues.

**Figure 4.5** The structure of a nerve.
The brain has 12 pairs of cranial nerves that emerge from and return to the CNS and serve sensory and motor roles in and around the head for both the somatic (muscular system) and autonomic nervous systems. The spinal cord has 31 pairs of nerves that serve somatic and autonomic function roles for the rest of the body. The cell bodies of the neurons that form these 43 pairs of nerves are normally located within ganglia, small masses of nerve tissue that exist on the outside of the CNS. As a consequence, it is the axons of each neuron that form the nerves that extend from the CNS.

Neuron junctions

A synapse is formed at the junction between two neurons or a neuron and its effector cell, as shown in Figure 4.6. Synapses enable an action potential to pass from one neuron to another. The neuron carrying the action potential (already stimulated and conducting a nerve impulse) is called the pre-synaptic neuron, and the one to which it will pass is the post-synaptic neuron. The sequence of events for the transfer of the action potential from one neuron to the next is very similar to that at a neuromuscular junction which will be described in the next chapter. As can be seen from Figure 4.6, the arrival of the nerve impulse at the axon terminal of the pre-synaptic neuron causes voltage-gated transmembrane protein channels to open. These channels allow Ca^{2+} to enter the axon terminal. The presence of calcium within the pre-synaptic neuron causes vesicles containing a neurotransmitter (chemical message) to fuse.
with the plasma membrane and open the vesicle to the synaptic cleft.

The neurotransmitter is able to diffuse across the extracellular fluid within the synaptic cleft and bind with ligand-gated channels to enable sodium to pass into the post-synaptic neuron and initiate a nerve impulse. Ligand-gated channels are trans-membrane proteins which have special receptor cells on their outside (open to the synaptic cleft) that will only enable a particular neurotransmitter to bind with them and open the channel. The neurotransmitter for many neurons in the PNS and neuromuscular junctions is acetylcholine. Within the CNS, along with acetylcholine, there are several proteins that are involved in synaptic neurotransmission. Aspartic acid, glutamic acid, and glycine work directly as neurotransmitters, and the catecholamines adrenalin (also known as epinephrine), noradrenalin (also known as norepinephrine) and dopamine are neurotransmitters within the brain.

**Knowledge integration question**

Describe how an action potential crosses from one neuron to another.

**Key point**

Neuron cell bodies are located in ganglia (knots of nervous tissue located outside the brain and spinal cord). Axons form the structures for nerves.

Neurons are generally:

- sensory (afferent) – relaying information to the CNS, or
- motor (efferent) – transmitting responses from the brain.

The 43 pairs of nerves are composed of sensory and motor axons.

**Nerve impulse conduction**

Stimulation of a neuron results in the generation of an electrical current, known as an action potential or nerve impulse, which is propagated along the length of the axon. The ability of a neuron to generate an action potential is dependent on the movement of electrolyte ions, primarily sodium and potassium, across the axolemma via the sodium-potassium pump. To fully appreciate how an action potential is generated, we must first understand the basic principles of electricity.

**Principles of electricity**

The same number of positive and negative charges exist within the body. A simple rule of electricity is that ‘like charges repel’, whereas ‘opposite charges attract’ each other. Due to this attraction, the separation of oppositely charged molecules, for example by a plasma membrane, requires work and so creates potential energy. This energy is termed the potential difference, also called voltage, and is measured in volts, a measure of the energy of electricity between two points, e.g. intracellular compared to extracellular. When the potential difference results in the movement of charged particles (ions within the body) this is called a current which is responsible for the propagation of an action potential.

**Establishment of resting membrane potential**

The electrical potential across the axolemma is created by the sodium-potassium pump (Na⁺-K⁺ pump) which was described earlier in Chapter 2 and illustrated in Figure 3.6. It is well worth reading this section again before reading about nerve impulse conduction. The Na⁺-K⁺ pump creates a differential in the concentration of Na⁺ and K⁺ on either side of the plasma membrane, by expelling three Na⁺ from, and importing two K⁺ into, the cytoplasm. It is this (resting) membrane potential or potential difference that enables nerve impulse conduction. The selectively permeable nature of the cell’s plasma membrane, along with the actions of the sodium-potassium pump, enable the resting membrane potential to be established (Figure 4.7).
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The Na⁺-K⁺ pump expels three Na⁺ for every two K⁺ brought into the cell, but in addition:

1. The plasma membrane is more ‘leaky’ to potassium ions than to sodium ions. Both ions will leak from high concentration to low concentration; however, more potassium ions will leak from the cell thereby increasing the potential difference (voltage) between the inside and outside of the cell.

2. There are many large negatively charged protein molecules and phosphate ions within the cell, which again increases the potential difference across the plasma membrane.

Figure 4.7 Resting membrane potential. (Source: Martini, F. H., Ober, J. and Nath, J. L. (2011) Visual Anatomy and Physiology, New York: Benjamin Cummings)
The negative ions immediately inside the plasma membrane are attracted to the positive ions on the outside of the cell and this creates a narrow band of ions either side of the membrane that have a relatively large difference in charge. This difference in charge between the inside and outside of the cell, the resting membrane potential, can be measured by a voltmeter and in most cells is around −70 mV. The negative sign indicates that the inside of the cell is negatively charged relative to the outside. Cells that maintain a resting membrane potential are said to be polarised (there is a difference in the electrical charge inside and outside the cell). Nervous and muscle tissue are able to use this potential difference to propagate an electrical current.
Knowledge integration question

Explain how a resting membrane potential is established.

Action potential generation and propagation

Although all cells have a resting membrane potential, it is only those with an excitable plasma membrane (muscle fibres and neurons) that are able to generate and propagate action potentials. When a stimulus reaches a neuron it changes the permeability of the plasma membrane and triggers the opening of special voltage-gated transmembrane protein channels. The Na\(^+\) voltage-gated channels open and enable Na\(^+\) to flood into the cell before they close again. The influx of Na\(^+\) changes the electrical charge from \(-70\) mV to around \(+35\) mV and as a consequence the cell is said to be depolarised (Figure 4.8). This depolarisation is the actual action potential or nerve impulse. Once an action potential has been initiated, it is then self-propagating along the length of the axon. The action potential does not flow across the plasma membrane in one moment, especially as axons and muscle fibres can be quite long. Instead, the action potential moves down the membrane as a wave of depolarisation. As the action potential travels along an axon or muscle fibre it depolarises the next segment in the plasma membrane. As soon as the membrane has depolarised, the sodium voltage-gates are closed and potassium channels open and enable potassium ions to leave the cell. This action, along with the function of the Na\(^+\)-K\(^+\) pump, returns the neuron or muscle fibre to its resting membrane potential and thus it has been repolarised (Figure 4.8). A repolarised cell is then ready to conduct a new action potential. The whole process of depolarisation and repolarisation, that is, the duration of an action potential, is very quick taking a thousandth of a second (1 msec or 0.001 sec).

Key point

Nerve impulses can travel to the brain (sensory neurons) or from the brain (motor neurons). An action potential or nerve impulse is an electrical current propagated along the axon of neurons. The initial change in electrical charge initiating an action potential is called depolarisation. An action potential travels as a wave along a neuron axon or muscle fibre triggering the depolarisation of the next segment of the plasma membrane.

Generation of Action Potentials

Step 1: Depolarisation to threshold

• A graded depolarisation brings an area of excitable membrane to threshold (\(-60\) mV).

Step 2: Activation of sodium channels and rapid depolarisation

• The voltage-gated sodium channels open (sodium channel activation).
• Sodium ions, driven by electrical attraction and the chemical gradient, flood into the cell.
• The transmembrane potential goes from \(-60\) mV (the threshold level), toward \(+30\) mV (the sodium equilibrium potential).

Step 3: Inactivation of sodium channels and activation of potassium channels

• The voltage-gated sodium channels close (sodium channel inactivation occurs) at \(+30\) mV.
• The voltage-gated potassium channels are now open, and potassium ions diffuse out of the cell.
• Repolarization begins.

Step 4: Return to normal permeability

• The voltage-gated sodium channels regain their normal properties in 0.4-1.0 msec. The membrane is now capable of generating another action potential if a larger-than-normal stimulus is provided.
• The voltage-gated potassium channels begin closing at \(-70\) mV. Because they do not all close at the same time, potassium loss continues and a temporary hyperpolarisation to approximately \(-90\) mV (the potassium equilibrium potential) occurs.
• At the end of the relative refractory period, all voltage-gated channels have closed and the plasma membrane is back to its resting state.

Nerve impulse conduction for unmyelinated neurons is conducted in this way, as a wave of depolarisation along the axon, and is called **continuous conduction**. The myelination of a neuron's axon, however, enables the action potential to 'jump' along the axon. The process of axon myelination is performed by Schwann cells, each of which is about 1 mm in length. The gaps in myelination between the Schwann cells are called nodes of Ranvier (see pp. 86 and 96). The insulating property of myelin means ions, and hence electrical currents, are only able to pass through the membrane at these nodes, thus an action potential in a myelinated neuron travels from one node of Ranvier to the next. The jumping of an action potential from node to node, called **saltatory conduction** from the Latin ‘saltare’ meaning to leap, greatly enhances the speed of nerve impulse conduction. This, along with axonal diameter differences, is the reason why nerve impulse conduction in Type A and B axons is faster than for Type C axons (refer to previous section on neuron structure for Types of axon). Figure 4.9 provides an illustration of how the conduction of action potentials takes place for myelinated and unmyelinated neurons.

**Key point**

Unmyelinated neurons carry action potentials by continuous conduction.

Myelinated neurons carry action potentials by saltatory conduction.

**Knowledge integration question**

Explain why saltatory conduction is more rapid than continuous conduction.

**Central nervous system**

The central nervous system is composed of the brain and the spinal cord which are enclosed by bony structures, the skull and the vertebral column. In addition to bones, the delicate tissues of the brain and spinal cord are protected by connective tissue membranes, adipose tissue and fluid-filled cavities. Three protective, connective tissue membranes surround the brain and spinal cord; the **dura mater**, **arachnoid mater** and **pia mater** which are known collectively as the **meninges** (see Figure 4.11). The outer layer of the meninges, the dura mater, is a dense connective tissue found
immediately beneath the skull which covers the whole brain and, in a tube-like extension from the brain, the spinal cord. One difference in the meninges between the brain and the spinal cord is that the brain has two dura maters compared with only one protecting the spinal cord. Between the spinal dura mater and the vertebrae, within the epidural space, are connective tissue and fat as a second level of protection. A further difference between the brain and spinal cord is that no epidural space is found in the brain as the dura mater is fused to the periostium of the cranial bones.
The middle meningeal layer, the arachnoid mater, is attached to the inner surface of the dura mater. The subarachnoid space (between arachnoid and pia maters) is filled with cerebrospinal fluid (CSF) which is a clear liquid produced by the walls of the ventricles within the brain. In addition to acting as a shock absorber, the CSF plays an important role in regulating the extracellular environment of nerve cells, and provides the brain and spinal cord with nutrients and electrolytes. The pia mater, the innermost meningeal layer, sticks to the surface of the brain, following every contour. Blood vessels run along the surface of the pia mater, supplying the brain and spinal cord with nutrients and oxygen.

Unique to the brain is the blood-brain barrier, a vital protective mechanism to ensure a stable internal environment is maintained. The majority of capillaries in the brain are surrounded by the blood-brain barrier which functions to separate the general circulation from the neural tissue of the brain. This prevents the free flow of substances into the brain and is effective in protecting the brain from chemicals that might disrupt neural function, or from blood-borne bacterial infections.

A basic pattern of the distribution of matter in the CNS exists with slight variation depending on the region of the brain or spinal cord. In general, the central cavity is surrounded by grey matter with white matter lying externally. A slight variation on this is found in the cerebellum where an outer layer of gray matter exists which is known as the cortex. The grey matter contains neuronal cell bodies and their dendrites, whereas the white matter consists of myelinated neurons.

**The brain**

The human brain contains almost 97% of the body’s neural tissue and typically weighs 1.5 kg (3 lb). There is a wide variability in brain size between individuals with no difference between genders when corrected for body mass. Although making up less than 3% of total body mass, the brain is comprised of around 100 billion neurons, and at rest uses up to 20% of oxygen and glucose consumed. It has a relatively high demand for oxygen because its neurons and neuroglia almost exclusively synthesise ATP through aerobic means, with glucose being the only nutrient it utilises as an energy source.

The brain is commonly divided into four regions, the brain stem (comprising the medulla oblongata, pons and midbrain) the cerebellum, the diencephalon (incorporates the thalamus, hypothalamus and the epithalamus) and the cerebrum. Figure 4.10 provides an illustration of the brain.

**Key point**

The brain is usually divided into the:

- Cerebrum
- Diencephalon (thalamus, hypothalamus and epithalamus)
- Cerebellum
- Brain-stem (medulla oblongata, pons and midbrain)

The **brain-stem** is a small, yet extremely important, part of the brain as it is responsible for providing a link between the spinal cord and the brain and it allows passage of all the sensory and motor nerve connections from the main part of the brain to the rest of the body. It controls autonomic processes that are independent of our consciousness, such as breathing, heart rate, blood vessel diameter and a variety of reflexes such as coughing, vomiting and sneezing. The brain stem is also vital for the regulation of the sleep cycle. The **cerebellum**, is very important to sports performers as it represents the brain’s feedback centre. Its most well-known role is in controlling motor performance as it contributes to posture, balance, coordination, accurate timing and precision, and fine-tuning motor activity. The cerebellum judges movements made against planned movements and then makes adjustments as necessary. This is very important in the development of fine motor movements. The **diencephalon**, through the structures that in contains, plays numerous roles in the human body – it regulates the activity of the autonomic nervous system (hypothalamus), controls our circadian rhythms (body clock) (hypothalamus and epithalamus), regulates behaviour and emotion (hypothalamus), provides a relay of information to the cerebrum (thalamus), regulates body temperature (hypothalamus) and is involved in endocrine function (hypothalamus and pituitary gland – part of the epithalamus). The **cerebrum** is the largest, most well developed, region of the brain, accounting for 83% of total brain mass. It represents our conscious mind, directing volitional motor functions, processing sensory information and controlling higher cognitive functions such as language and communication, learning, memory, intelligence and personality.

**Knowledge integration question**

Why is the cerebellum important to athletes?
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... sacral (S₁ – S₅), and coccygeal (C₁ – C₄). In maturity, the vertebrae of the sacral and coccygeal regions are fused together, with no intervertebral discs.

The spinal cord

The spinal cord, illustrated in Figure 4.11, is on average 45 cm long and 1 cm wide. It is comprised of around 100 million neurons and is protected by a number of structures including the three protective meningeal sheaths, 33 vertebrae that form the vertebral column, surrounding fluids, connective tissue and fat (discussed at the start of this section). The vertebral column is divided into five regions, with a letter indicative of the region. Within each region, the vertebrae are numbered beginning with the uppermost; cervical (C₁ – C₇), thoracic (T₁ – T₁₂), lumbar (L₁ – L₅),...
The nervous system

CNS, form the ventral roots with the cell bodies contained within the grey matter of the spinal cord. Distal to the dorsal root ganglia, the dorsal (sensory) and ventral (motor) roots are bound together into a single spinal nerve. The structure of the spinal cord, its protective tissues and spinal nerves are illustrated in Figure 4.11.

In the upper region of the vertebral column, spinal nerves leave the spinal cord directly, whereas, in the lower region, spinal nerves travel further down the vertebral column before exiting. This is because, at around the age of 4 years, the longitudinal growth of the spinal cord ceases. However, as we continue to grow in height, the vertebral column continues to elongate. This results in the adult spinal cord extending only to the first or second lumbar vertebra, hence the origins of the sacral spinal nerves are actually in the upper lumbar region.

Peripheral nervous system

The PNS, which structurally includes the ganglia, sensory receptors and nerves, can be further divided into the somatic nervous and autonomic nervous systems. The
differences between the autonomic and somatic nervous systems can be identified.

The somatic nervous system motor neurons comprise Type A axons, large diameter and myelinated, for fast innervation of the relevant skeletal muscle. The cell bodies of these neurons are located within the CNS and their axons form part of one of the 43 paired nerves that exit the CNS. Within the somatic nervous system a single motor neuron carries the nerve impulse from the CNS to the skeletal muscle fibres. Acetylcholine is the neurotransmitter released by the motor neuron and received by post-synaptic muscle receptors, thereby stimulating the muscle fibre to contract. In the somatic nervous system, excitation of a neuron will only lead to muscle contraction, that is, there are no inhibitory neurons.

**Autonomic nervous system**

In contrast to the somatic nervous system, two synapsed (linked by a synapse) neurons form the efferent pathway of the autonomic nervous system. In addition, there are two branches of efferent autonomic neuron, those that have a sympathetic effect (preparing the body for activity, e.g. increasing heart rate during exercise) and those that carry a parasympathetic response (restorative functions, e.g. decreasing heart rate). The first autonomic or visceral (pertaining to the internal organs) neuron has its cell body within the brain or spinal cord and conducts a nerve impulse to small clusters of neuron cell bodies called an autonomic ganglion (similar to ganglia described above). This type of visceral neuron is called a pre-ganglionic neuron because it is the neuron that conducts the autonomic response from the CNS to the autonomic ganglion. As was mentioned in the section on neurons (4.1.2 Nervous tissue) pre-ganglionic neurons normally have Type B axons and as a consequence nerve impulse conduction is slower than in somatic (motor) neurons. The second autonomic neuron, the post-ganglionic neuron, is usually a Type C fibre (unmyelinated) which carries impulses more slowly from the ganglion to the tissue where they have their effect.

Figure 4.12 provides a diagrammatic representation of differences and similarities between sympathetic visceral, parasympathetic visceral and motor neurons.

**Somatic nervous system**

The cranial and spinal nerves provide the afferent and efferent neuron pathways for the somatic and autonomic divisions. Sensory neurons for the somatic and autonomic systems have the same structure, and function in the same fashion, receiving information from receptors throughout the body and passing it to the CNS. At the CNS the sensory information is processed and a response generated. The efferent pathways are where structural and functional

**Autonomic nervous system**

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**Sympathetic division**

The location of the autonomic ganglia, and therefore the lengths of the pre- and post-ganglionic fibres, differs between the sympathetic and parasympathetic divisions.
The nervous system is acetylcholine; however, norepinephrine is released into the nerve-organ junction by post-ganglionic fibres.

Sympathetic pre-ganglionic neurons (myelinated Type B axons) have their cell bodies located only in the thoracic or lumbar regions of the spine and their axons originate from the spinal cord. There are three sympathetic autonomic ganglia which are located very close to the vertebral column, hence the pre-ganglionic neurons are short in length. In contrast, post-ganglionic neurons are unmyelinated (Type C axons) and much longer, emerging from the autonomic ganglia to complete the nerve impulse journey (see Figure 4.12). Similar to the somatic nervous system, the neurotransmitter of sympathetic pre-ganglionic fibres is acetylcholine; however, norepinephrine is released into the nerve-organ junction by post-ganglionic fibres.

**Knowledge integration question**

Explain the differences between the SNS and the ANS.

**Parasympathetic division**

*Parasympathetic pre-ganglionic neurons* originate only from cranial nerves or the sacral region of the spinal cord.
and connect with the parasympathetic autonomic ganglia. The parasympathetic autonomic ganglia are located close to or within the walls of the organ they innervate, so by the time the nerve impulse reaches the parasympathetic ganglion it has almost completed its journey. In contrast to the sympathetic system, therefore, the parasympathetic division has long pre-ganglionic neurons and short post-ganglionic neurons (see Figure 4.12). The pre- and post-ganglionic neurons of the parasympathetic system release acetylcholine from the axon terminals as the neurotransmitter.

Key point
The nervous system can be divided into the CNS (the brain and spinal cord) and the PNS (the nerves and ganglia outside the CNS).

The PNS is comprised of the:

- **somatic nervous system** (for control of skeletal muscles) over which we have control (the voluntary system).
- **autonomic nervous system** (for control of smooth muscle, cardiac muscle, glands and other tissues and organs) over which we do not consciously have control (involuntary system).

4.2 The endocrine system

The endocrine system is the body’s second control system. Working alongside the nervous system, it is responsible for the regulation of the other systems within the body. Many life processes, such as growth, development and reproduction, are of a long-term nature, and it is these processes that are regulated by the endocrine system. Endocrine comes from the Greek, *endo* meaning inside and *crine* to secrete and the name underlies the system’s function. Three elements are required for endocrine messaging, a **production organ** that secretes a **hormone** into the extracellular fluid and specific **target cells**, reached via the blood, upon which the hormone would take effect. A **hormone**, from the Greek ‘*horma*’ meaning to set in motion, is a chemical message that initiates a change in the function of the target cells. Each hormone is highly specific and impacts upon only the target cells for which it is intended. All cells are exposed to hormones but it is only those with the specific receptors for a particular hormone that respond to the changing levels of that hormone in the bloodstream. As hormones are transported in the bloodstream, target cells can be located anywhere in the body, hence, the metabolic functioning of multiple tissues and organs can be simultaneously altered by a single hormone. An example of this is human growth hormone that alters the functioning of a wide number of cells within the body. Although the effects of hormones may be slow to initiate, they are prolonged in their response, generally continuing for a few days. It is thanks to the combination of hormones and neurons that the body is able to, in most circumstances, maintain or regain a state of homeostasis.

Key point
The endocrine system is the body’s second control and regulation system.

Endocrine messaging includes:

- a production organ
- a hormone
- a target organ

Commonly mentioned hormones include adrenalin, insulin and testosterone.

There are two associated types of hormone producing cells that are sometimes included within the endocrine system, **paracrines** and **autocrines**. The inclusion of these chemical messengers is not strictly correct, however, for their hormones take effect close to (paracrines), or at (autocrines), the site of production, rather than travelling some distance within the body to take effect, as is the case with endocrines. Paracrines are local messengers and secrete hormones into the extracellular fluid around their cell with target cells very close and so consequently their hormones do not need to enter the bloodstream. Autocrines secrete hormones that have an effect on the same cell from which they are produced. For example the **eicosanoids** (fatty acid-based hormones), which exert control over many bodily functions, primarily immune function and inflammation, are released by nearly all cells and typically take their effect locally and therefore act as autocrines or paracrines. Normally, however, hormones are carried in the blood to the target cells, for example, erythropoietin (the hormone that stimulates erythrocyte production) is secreted from the kidneys and travels via the blood to the target cells in the bone marrow.

Endocrine organs

The organs of the endocrine system can be seen in Figure 4.13. The primary function of those within the
The endocrine system and homeostatic balance. The hypothalamus is the interaction point between the endocrine and nervous system, for not only is it the driver of endocrine function, but it also controls autonomic nervous system functions. In its role within the nervous system it is responsible for controlling homeostatic processes such as body temperature, thirst and hunger. The hypothalamus integrates sensory information received from receptors throughout the body, and determines an endocrine and/or autonomic response as required. As a result of its dual nervous and endocrine system functions, the hypothalamus is referred to as a neuroendocrine organ.

It has recently been shown that both adipose tissue (reviewed in Singla et al. 2010) and skeletal muscle (reviewed in Pedersen & Febbraio, 2008) have endocrine purple-headed boxes, such as the thyroid gland, is endocrine and so they are classified within the endocrine system. Other organs, however, such as the heart and kidneys, have a separate primary role in addition to their endocrine function. The total weight of the endocrine organs is relatively small, around 0.5 kg in an adult; however, their importance to the body as a control system is considerable. Organs with an endocrine function, examples of hormones they produce and target cells for each specific hormone are shown in Table 4.1. The hypothalamus represents the controlling mechanism for endocrine function, and along with the pituitary gland is responsible for the production of 16 hormones (nine from the hypothalamus and seven from the pituitary gland) which regulate growth, maturation, metabolism and homeostatic balance. The hypothalamus is the interaction point between the endocrine and nervous system, for not only is it the driver of endocrine function, but it also controls autonomic nervous system functions. In its role within the nervous system it is responsible for controlling homeostatic processes such as body temperature, thirst and hunger. The hypothalamus integrates sensory information received from receptors throughout the body, and determines an endocrine and/or autonomic response as required. As a result of its dual nervous and endocrine system functions, the hypothalamus is referred to as a neuroendocrine organ.

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functions. In addition to its primary role as an energy store, adipose tissue has been found to be an important source of various hormones. Several of these hormones play important roles in weight control, such as leptin, resistin, visfatin and adiponectin. In 2005, Pedersen & Febbraio first suggested that skeletal muscle be termed an endocrine organ, due to the discovery that contracting skeletal muscle is a major source of the circulatory cytokine interleukin-6 (IL-6), an intercellular signalling molecule with a role in inflammation. The term ‘myokines’ is now commonly used to refer to substances (cytokines or other peptides) released by skeletal muscle during physical activity. It is thought that some myokines work in a hormone-like fashion, exerting effects in other tissues such as adipose tissue and the liver, whereas others will work in a paracrine or autocrine manner. The discovery of myokine release during exercise
contributes to our understanding of why regular physical activity is important for the protection against a range of chronic diseases.

Function of hormones

Hormones regulate the functioning of the body by impacting upon processes such as growth, metabolism, reproduction, and circadian rhythms. There are two main groups of hormones based on chemical structure, those that are lipid (steroid) based and those that are protein-based (further classified as amino acid derivatives, peptide, polypeptide or protein hormones depending on their molecular size). Table 4.2 provides details of some of the most common hormones, their classification and the organ that produces them. The majority of hormones are produced by a host cell and secreted into the extracellular fluid and from there diffuse into the blood where they are carried to their target cells.

Lipid-based and protein-based hormones are both carried in the blood to their target cells. However, they differ as to the method by which they are transported and how they are received by their target cells. An illustration of lipid- and protein-derived hormone transport and message entry to a target cell is provided in Figure 4.14. As a result of their insolubility in water, lipid-based hormones attach to carrier proteins for transport within the blood. As they reach the target cell they disconnect from the carrier protein, which is left in the bloodstream to bind with other lipid-based hormones, and diffuse into the extracellular fluid surrounding the target cell. Upon reaching the plasma membrane of the target cell, lipid-based hormones diffuse into the cell and attach to an intracellular receptor on the nuclear membrane (Figure 4.14). In contrast, protein-based hormones (e.g. human growth hormone) travel unbound in the blood and, upon reaching the target cell, diffuse into the extracellular fluid and attach to extracellular receptors (a trans-membrane protein) on the surface of the target cell plasma membrane. The binding of a protein-based hormone to the receptor causes the release of a secondary messenger within the cell that stimulates the desired response (Figure 4.14). Cells typically have between 2,000–10,000 receptors for each hormone it requires as part of its functioning and regulation.

An example of hormone functioning can be found in the actions of glucagon and insulin, both of which are produced by the pancreas. The level of glucose within the bloodstream is regulated by the secretion of glucagon and insulin. The control process is carried out by way of negative feedback. Following eating and the subsequent digestion of carbohydrates, blood glucose levels rise above normal creating a hyperglycaemic state which stimulates the β (beta) cells of the pancreas to produce insulin.

<table>
<thead>
<tr>
<th>Protein-based hormones</th>
<th>Produced by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growth hormone</td>
<td>Pituitary gland</td>
</tr>
<tr>
<td>Insulin</td>
<td>Pancreas</td>
</tr>
<tr>
<td>Prolactin</td>
<td>Pituitary gland</td>
</tr>
<tr>
<td>Antidiuretic hormone</td>
<td>Pituitary gland</td>
</tr>
<tr>
<td>Glucagon</td>
<td>Pancreas</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Pancreas</td>
</tr>
<tr>
<td>Endorphins</td>
<td>Pituitary gland</td>
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<tr>
<td>Oxytocin</td>
<td>Pituitary gland</td>
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<tr>
<td>Adrenalin</td>
<td>Adrenal medulla</td>
</tr>
<tr>
<td>Noradrenalin</td>
<td>Adrenal medulla</td>
</tr>
<tr>
<td>Serotonin</td>
<td>Platelets</td>
</tr>
<tr>
<td>Melatonin</td>
<td>Pineal gland</td>
</tr>
<tr>
<td>Thyroxine</td>
<td>Thyroid</td>
</tr>
<tr>
<td>Erythropoietin</td>
<td>Kidneys</td>
</tr>
<tr>
<td>Parathyroid hormone</td>
<td>Parathyroid glands</td>
</tr>
<tr>
<td>Thyroid stimulating hormone</td>
<td>Pituitary gland</td>
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<table>
<thead>
<tr>
<th>Lipid-based hormones</th>
<th>Produced by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testosterone</td>
<td>Testes</td>
</tr>
<tr>
<td>Oestrogen</td>
<td>Ovaries</td>
</tr>
<tr>
<td>Progesterone</td>
<td>Ovaries</td>
</tr>
<tr>
<td>Aldosterone</td>
<td>Adrenal cortex</td>
</tr>
<tr>
<td>Cortisol</td>
<td>Adrenal cortex</td>
</tr>
<tr>
<td>Prostaglandins</td>
<td>All cells but RBC</td>
</tr>
<tr>
<td>Leukotrienes</td>
<td>All cells but RBC</td>
</tr>
</tbody>
</table>

Table 4.2 Classification of some well-known hormones.
Insulin diffuses into the blood and takes its effect on the many cells throughout the body, especially those of the liver and skeletal muscles, stimulating them to take up glucose and form glycogen (glycogenesis). As a result, the level of glucose in the blood decreases. This change is detected by receptors in the blood which relay information to the brain. When glucose levels return to normal there is no longer a need for insulin production and so the brain signals to the pancreas to discontinue insulin secretion (negative feedback). Glucagon, produced by pancreatic \(\alpha\) (alpha) cells, is secreted at times of low blood glucose (hypoglycaemia). As blood glucose is used as an immediate energy source, levels will fall during a long training session. Detection of this change by the brain results in the stimulation of the pancreas to secrete glucagon which, following diffusion into the blood, takes effect on the hepatocytes of the liver. The liver, along with the muscles, is a major store of glycogen. When the muscles are exercising glucagon stimulates the liver to covert its glycogen back to glucose (glycogenolysis) and release it into the blood. Glucagon has an additional effect in the blood, for not only does it stimulate glycogenolysis, it also stimulates the conversion of lactic acid (produced during exercise) and amino acids to glucose through the process of gluconeogenesis in the liver. When blood glucose levels reach normal the secretion of glucagon is inhibited (negative feedback).

**Key point**

Hormones target specific cells and change the functioning of those cells. Hormones impact upon metabolism, growth, immune function, the body’s internal environment and many other aspects of human functioning.

**4.3 Acute responses to exercise**

The metabolic requirements of the body at rest are low. These requirements, however, are greatly enhanced during exercise with the oxygen consumption of skeletal muscle increasing by up to 40-fold during intense exercise. In addition to the increased demand for oxygen, the active skeletal muscles must oxidise a greater amount of fuel for the generation of ATP required for muscle contraction, hence glucose and fats are mobilised from the body’s stores. Many physiological responses within the body are altered during exercise to meet the increased metabolic demands, particularly of skeletal muscle. As we have discussed in this chapter, alterations to the body systems are under the control of both the nervous system and the endocrine system.
At the start of exercise, the sympathetic nervous system is the key driver of the physiological changes within the body, having immediate effects on the cardiovascular and respiratory systems, the details of which will be discussed in Chapter 6. The sympathetic nervous system also stimulates endocrine function resulting in the release of exercise-related hormones. These hormones provide a less immediate, but longer-enduring, reinforcement of stimulation initiated by the sympathetic nervous system. The hormonal response to exercise is mainly associated with increases in the cardiovascular response, metabolism and substrate availability through the increased release and action of the catecholamines (adrenaline and noradrenaline), thyroxine, cortisol, glucagon and human growth hormone. The roles of these hormones in the control of metabolism and substrate availability, particularly relating to skeletal muscle tissue, are discussed in Chapter 5 (The movement systems) while their role in the functioning of the cardiovascular system are discussed in Chapter 6 (The transport and exchange systems).

**Key point**

The alterations to body system function with exercise are under the control of the nervous system and the endocrine system, which work together to increase the transport and exchange of metabolites, and to increase fuel substrate availability.

**Knowledge integration question**

Describe what happens to your body at the start of exercise.

**Check your recall**

Fill in the missing words.

> The human body has two control and regulatory systems: the nervous system (immediate response) and the ____________ ____________ (slow, prolonged response).

> Under control of these systems, the body is able to maintain ____________, a stable internal environment, required for cell survival.

> The ____________ system functions in the control of metabolic activity, and initiates the breakdown of glycogen to glucose when blood glucose levels fall during prolonged exercise.

> The nervous system comprises of the brain, ____________, ganglia, nerves and sensory receptors.

> These components are often divided into the ____________ nervous system (the brain and spinal cord) and the peripheral nervous system.

> The peripheral nervous system (PNS) can also be further divided into the ____________ division, which carries information to the central nervous system (CNS), and the ____________ division, which carries signals away from the CNS.

> Another useful distinction is to divide the nervous system into the ____________ nervous system, over which we have conscious control, and the autonomic nervous system, which functions without our conscious control.

> The autonomic nervous system has ____________ and ____________ divisions, which speed up (______ ) and slow down (______ ) the body’s autonomic responses.

> There are two types of cell that make up the nervous system: neurons and ____________. The junction between two neurons is referred to as a ________.

> The electrical impulse in the pre-synaptic neuron triggers the release of a chemical messenger known as a ________, which is capable of crossing the synaptic cleft and in turn triggers the electrical impulse in the post-synaptic neuron.

> The endocrine system is the second _________ and regulation system for the human body.

> It involves the transmission of chemical messengers, called ________, through the blood.

> These can be either protein-based or ________-based messengers.

> The ________ provides a junction between the two communication systems as the controlling mechanism for the autonomic nervous system and endocrine functioning. This junction ensures that the two systems respond in harmony.

> The initiation of exercise induces a rapid increase in the ________ nervous activity. This is by the release of exercise-related ________ (adrenaline, noradrenaline, cortisol, human growth hormone, thyroxine, glucagon).

> Together, the ________ and ________ systems function to increase the transport and exchange of metabolites and to enhance the availability of fuel substrates.
Chapter 4 The control systems: nervous and endocrine

Review questions

1. How are messages transmitted by the nervous system? How do electrical and chemical processes contribute to communication?

2. How do myelinated and unmyelinated neurons differ? What advantages do myelinated neurons have?

3. What is the difference in the function of neurons and neuroglia?

4. How is the resting membrane potential established?

5. Describe the propagation of an action potential along both a myelinated and unmyelinated axon.

6. What are the components of endocrine messaging?

7. What is the difference between endocrine, paracrine and autocrine signalling?

8. Name five hormones, their site of production and their target cells.

9. How is a lipid-based hormone transported from the organ of production to the target cells?

10. Describe the differences between the sympathetic and parasympathetic divisions.

Teach it!

In groups of three, choose one topic and teach it to the rest of the study group.

1. Explain, using a diagram, the divisions within the nervous system and the function of each component.

2. Explain how an action potential can arise and how it travels along a neuron.

3. Choosing one example, explain how a hormone can take its effect on cells within the body. Describe the components of endocrine messaging (for that hormone) and how it is regulated.
High-intensity aerobic endurance sports

As a result of the diversity of aerobic sports, both in terms of the intensity and duration, two chapters have been devoted to the aerobic endurance sports. The current chapter focuses on high-intensity aerobic endurance sports, while Chapter 12 examines the physiology of lower-intensity aerobic endurance sports. The relative intensity of aerobic endurance sports is closely related to the duration of the event. By way of example, Figure 11.1 provides an illustration of a velocity–distance curve for the world records for a number of athletics events. As can be seen from this figure, the highest velocities occur for the events in the anaerobic zone.

Within the anaerobic zone, as a result of the maximal intensity involved and the increasing duration of the effort, the velocity curve falls sharply through this zone. We then enter the aerobic high-intensity zone and we see the shape of the curve begin to plateau. The lesser velocity decline in this zone reflects the ability of the aerobic energy system, as the main fuel source, to sustain high-intensity exercise across a wide duration of activity from 1,500 m (~3.30 min) to a full marathon (~2.00–2.15 h). As we enter the lower-intensity aerobic zone we see a further drop in intensity for marathon performance at the end of an ironman triathlon. Given that the duration of a full ironman triathlon is in excess of 8 hours for elite athletes, their times for the marathon (between 2 h 30 min and 2 h 48 min) are a further...
Chapter 11 High-intensity aerobic endurance sports

High-intensity aerobic endurance sports require a high intensity of performance. Clearly, it would not be possible for an athlete to maintain the physiological intensity they achieve in a 1,500 m race over the distance required for a marathon. When planning training for an aerobic endurance sport the duration of the event should be central to decisions made about the preparation programme. A high degree of physiological specificity is required for training to improve performance in high-intensity aerobic endurance sports, as for all the sports covered in Part II of this book.

The pace judgement involved in aerobic endurance sports means that athletes will operate in a zone close to the transition between aerobic and anaerobic exercise. This transition point is often referred to as the anaerobic threshold. The anaerobic threshold and the related lactate threshold, have been shown to have significant correlation with race performance. It is likely that a delayed reflection of the sustained performance ability of the aerobic system. In events of a duration longer than an elite full marathon (just over two hours), we begin to see a further decline in intensity and velocity, and these will form the focus of Chapter 12. The current chapter considers a range of sports that fit within the high-intensity aerobic endurance category, of which some can be seen in Figure 11.2.

As a consequence of their higher relative intensity, the duration of the activities in Chapter 9 (2–10 s) tended to be shorter than those covered in Chapter 10 (10–90 s). Despite this, the high-intensity endurance activities covered in this chapter still have a relatively wide range in duration, from events like the 1,000 m sprint kayak or canoe (3–4 min duration) to the marathon run (2–3 h duration). As can be seen from Figure 11.1, the duration of an individual aerobic endurance event has a direct impact upon the relative intensity of performance. Clearly, it would not be possible for an athlete to maintain the physiological intensity they achieve in a 1,500 m race over the distance required for a marathon. When planning training for an aerobic endurance sport the duration of the event should be central to decisions made about the preparation programme. A high degree of physiological specificity is required for training to improve performance in high-intensity aerobic endurance sports, as for all the sports covered in Part II of this book.

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Figure 11.1 The velocity duration curve for athletics world records. ½ M = half marathon, M = marathon, IMM = Ironman marathon time.

Figure 11.2 The duration continuum for high-intensity aerobic sports.
Aerobic endurance refers to the ability of the body to produce energy for exercise involving the whole body during sustained exercise. There are a number of different terms, often used interchangeably, that have been used to refer to this aspect of fitness: these include aerobic capacity, aerobic power, cardiovascular fitness and cardiorespiratory fitness. The metabolic pathways within the aerobic system become the predominant energy source for sports or events that last for longer than 60–90 s. The aerobic system has specific pathways through which carbohydrates, lipids and proteins can be catabolised in the presence of oxygen to produce energy for exercise.

The functional capacity of the aerobic system has traditionally been assessed by conducting a test for maximal oxygen uptake (\( \dot{V}O_{2\text{max}} \)). An individual’s \( \dot{V}O_{2\text{max}} \) represents the maximum volume (V) of oxygen (O\(_2\)) he or she can consume per minute (dot above the V) to provide energy via aerobic metabolism during an exercise test of increasing intensity. There is a variety of ways this can be measured or estimated, such as through the multi-stage fitness test (Bleep or Beep test) or in a laboratory on a cycle ergometer, a treadmill or a rowing ergometer. In a physiology laboratory, gas analysis equipment can be used to measure the exact volumes of oxygen consumed and carbon dioxide produced during a maximal test. If such a precise measurement of aerobic endurance is not required, or for safety reasons is not advisable, there is a variety of sub-maximal methods that can provide an estimate of an individual’s aerobic power. A number of these methods are described in Chapter 12.

More recently, the transition between aerobic and anaerobic systems has been measured as a way to assess the functional capacity of the aerobic system. This change has occurred because stronger correlations have been observed between the aerobic-anaerobic transition and race performance than between \( \dot{V}O_{2\text{max}} \) and race performance. In other words, the point at which an athlete makes the transition to an increasingly anaerobic performance is a better predictor of race pace than his or her \( \dot{V}O_{2\text{max}} \). Consequently, because the focus is on high-intensity aerobic endurance sports, we will examine the aerobic-anaerobic transition in this chapter and then in Chapter 12 move on to examine the concept of \( \dot{V}O_{2\text{max}} \).
acid concentration increased during anoxic (low or no oxygen) conditions. Later, Hill and Lupton (1923) suggested the increase in lactic acid during exercise was the consequence of insufficient oxygen supply.

The terms aerobic and anaerobic glycolysis arose from the conclusions drawn by Lupton and Hill regarding the formation of lactic acid during exercise. ‘Aerobic glycolysis’ was used to describe slower-rate glycolysis where the presence of oxygen is sufficient to enable pyruvate to be completely oxidised. More correctly, it refers to a pattern of glycolysis where lactate removal is equal to lactate synthesis (resulting in no or only a small net gain in lactate during exercise). ‘Anaerobic glycolysis’ described the fate of pyruvate during fast-rate glycolysis where lactate synthesis exceeds lactate removal resulting in an accumulation of lactate within muscle fibres and the blood.

As described in Chapter 10, the use of these terms has led to some confusion in physiological pedagogy, due to the fact that glycolysis is an anaerobic (not requiring oxygen) metabolic process. It is the fate of pyruvate, the end-product of glycolysis, which is determined by the intensity of exercise and the supply of oxygen. This is why the terms slower-rate and fast-rate glycolysis have been used throughout this textbook.

Traditionally, the metabolic processes are described by starting with short-term high-intensity exercise and then moving on to consider endurance activities. In this way textbooks move from anaerobic to aerobic metabolism. For some activities this matches the transition in energy production from the onset of exercise. However, for the high-intensity aerobic endurance activities that are the focus of this chapter there are times when the reverse pattern of energy system contribution occurs. Sports such as 1,500 m running, 400 m swimming and rowing are predominantly aerobic sports but draw upon anaerobic metabolism for short in-race bursts or for the finishing ‘kick’. An understanding of this conceptual difference is important when considering the transition from aerobic to anaerobic metabolism.

During an aerobic endurance race athletes attempt to maintain an exercise level that is as high as possible, thereby maximising aerobic metabolism, while avoiding substantial reliance on anaerobic (fast-rate glycolysis or phosphagen system) metabolic processes that would lead to early fatigue. This point of exercise intensity, maximising aerobic metabolism while remaining just below substantial reliance on anaerobic metabolism, represents the AAT.

The term AAT has been adopted for this textbook as an umbrella term encompassing the wide number of terms that have been used previously to describe this transition. For want of a better term, the anaerobic threshold has most commonly been used to describe the transition from aerobic to increasingly anaerobic exercise. The term anaerobic threshold, however, has a specific meaning which, as was clearly argued by Brooks and Davis in the 1980s, precludes its use as an umbrella term. The AAT is an important physiological transition point for high-intensity aerobic endurance athletes. As a consequence, the various terms and concepts related to the AAT will be discussed in this section prior to describing a number of methods that can be used to identify this transition point.

**Key point**

The aerobic-anaerobic transition (AAT) represents the point during exercise at which maximal aerobic metabolism switches to rely mainly on anaerobic metabolism resulting in the subsequent accumulation of lactate.

**Anaerobic threshold**

Interest in the identification of an individual’s AAT arose from a clinical rather than a sport-related concern for cardiac and cardiovascularly-compromised patients. It was thought that the AAT provided a sub-maximal method for the assessment of exercise capacity for cardiac patients. Since then, interest in the AAT has spread to a sports performance context. The AAT is traditionally identified using invasive blood lactate analysis techniques or through the use of non-invasive gas analysis. The gas analysis methods were originally developed in the 1960s for a clinical setting where non-invasive techniques for AAT might be preferred. The use of gas analysis to identify the AAT was originally termed the *anaerobic threshold* (AnT) by Wasserman and McIlroy (1964) but is sometimes referred to as the *ventilatory threshold*. Wasserman and McIlroy suggested that a breakaway in pulmonary ventilation equated with the rise in lactate accumulation associated with increasing anaerobic exercise. During exercise $V_E$ initially rises linearly with increasing intensity; however, at a specific point for each individual a breakaway in ventilation occurs. This point was identified by Wasserman and McIlroy as the AnT and was said to coincide with the rise in CO$_2$ produced during blood lactate and H$^+$ buffering (introduced in Chapter 10). Thus, the AnT indicates non-invasively the point at which the body begins to accumulate lactate, i.e. the lactate threshold. Later research highlighted difficulties in the identification of the ventilatory breakpoint and consequently other aspects of gas kinetics were examined to assess their possibility as a more clearly defined marker of AnT. Currently, the most widely used non-invasive indicator of AnT is the combined...
The $\dot{V}O_2\max$ test, which will be described in Chapter 12, has traditionally been the gold standard measure of aerobic endurance. It is now more common, however, for exercise physiologists to conduct an LT test and, more recently, critical power (CP) tests to assess aerobic endurance as it relates more closely to race performance. The point at which an athlete's LT or CP occurs is a more accurate predictor of race success than his or her $\dot{V}O_2\max$ in aerobic endurance events such as 10 km running. In the next section we will move on to describe the LT, prior to introducing the concept of CP.

**Lactate threshold**

The assessment of LT is based on research that has shown that as an athlete increases their effort towards maximum, they make a transition towards increasingly anaerobic exercise, and lactate begins to accumulate in muscle fibres and blood. Lactate is produced continuously and removed during metabolism. At rest, and during lower-intensity exercise, lactate removal matches its synthesis and so blood lactate concentration remains relatively constant. As exercise intensity increases, such as during a $\dot{V}O_2\max$ test or for the finish of a race, lactate is produced at a faster rate than it can be removed. As a consequence, lactate and $H^+$ begin to accumulate in the muscles and blood.

**Key points**

In aerobic endurance sports athletes attempt to race at an intensity level close to the point at which exercise shifts from being predominantly fueled by the aerobic system to being increasingly fueled by the anaerobic systems. This point is known as the aerobic-anaerobic transition (AAT).
Chapter 11 High-intensity aerobic endurance sports

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for assessment of AAT was completed by Krista Svendahl and Brian MacIntosh in 2003 and provides a useful additional resource.

Onset of blood lactate accumulation

The OBLA test represents an incremental test designed to identify the exercise intensity at which blood lactate accumulation reaches 4 mM. This test was developed in the 1970s by Mader et al. because, at a blood lactate concentration of 4 mM, there appears to be a relationship between the lactate concentrations in the muscle and blood, which is not found as clearly above or below this concentration level. This test has been used quite widely; however, the assumption that a blood lactate concentration of 4 mM is synonymous with AAT ignores the wide individual variation in AAT. As a consequence, the use of LT or MLSS tests for AAT identification, and the monitoring of training induced changes, would be more beneficial for athlete populations.

Lactate threshold test protocol

The LT represents the point after which the body’s lactate removal mechanisms cannot keep pace with the rate of lactate synthesis. When exercising at an intensity above the LT, lactate and H⁺ accumulate in the blood and are, in part, thought to lead to fatigue. The higher the percentage of an athlete’s VO₂max at which the lactate threshold occurs, the better for race performance. In other words, two 10 km runners with the same VO₂max but with differing LT (occurring at 80% and 70% of VO₂max) would differ in race performance. The race winner would be the athlete who could run at the higher percentage of VO₂max before an increasingly heavy reliance on anaerobic metabolism. An illustration of this can be seen in Figure 11.4. Research indicates that an athlete’s lactate threshold is a better predictor of his or her performance than VO₂max alone. In addition to exercise intensity, heart rate (HR) at the point at which LT occurs can be used as a basis for training programme development. The practical use of LT testing for the development of training programmes is discussed in a following section ‘Practical use of AAT determination’.

Lactate-concentration-related measures of AAT

There is a wide variety of lactate-based measurements for the AAT including mathematical transformations of the data to identify the transition point more clearly. The assessments of AAT based on lactate analysis includes the onset of blood lactate accumulation (OBLA), lactate minimum speed, LT (as introduced in the previous section) and the maximal lactate steady state (MLSS). As the most common methods for lactate analysis of AAT are LT and MLSS, they are the focus of this section. However, a brief mention of OBLA is included as it is often linked with LT assessment. A more complete review of the variety of methods

Figure 11.4 Lactate threshold occurring at different points for two athletes with the same VO₂max.

The LT represents a level of work after which the body’s lactate removal mechanisms cannot keep pace with the rate of lactate synthesis. When exercising at an intensity above the LT, lactate and H⁺ accumulate in the blood and are, in part, thought to lead to fatigue. The higher the percentage of an athlete’s VO₂max at which the lactate threshold occurs, the better for race performance. In other words, two 10 km runners with the same VO₂max but with differing LT (occurring at 80% and 70% of VO₂max) would differ in race performance. The race winner would be the athlete who could run at the higher percentage of VO₂max before an increasingly heavy reliance on anaerobic metabolism. An illustration of this can be seen in Figure 11.4. Research indicates that an athlete’s lactate threshold is a better predictor of his or her performance than VO₂max alone. In addition to exercise intensity, heart rate (HR) at the point at which LT occurs can be used as a basis for training programme development. The practical use of LT testing for the development of training programmes is discussed in a following section ‘Practical use of AAT determination’.

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Lactate threshold test protocol

The LT represents the point after which lactate begins to accumulate in the blood. The LT test, in common with OBLA and AnT, is conducted as an incremental test (most commonly 4-min stages). It can be conducted in the laboratory on a treadmill, cycle ergometer or rowing ergometer. In this situation, the starting intensity is dependent upon the athlete’s current level of competition and training programme, that is, it is based on an estimation of the athlete’s current fitness level. The increase in intensity during the test is easily achieved on the equipment in the laboratory. Blood lactate concentration, HR, and sometimes the rating of perceived exertion (RPE) (a psychological response to exercise), are measured at the end of each stage. The test is ended when a significant rise (>4 mM) in lactate is observed.

The LT test can also be administered in the field. A set distance (rather than time), dependent upon the sport, is completed during each stage of the test. Suggested repetitions for the three major endurance sports are: swimming, 300 or 400 m; cycling, 3 or 4 km; running, 1 or 1.6 km (Hellemans, 2000). As with the starting intensity selected in the laboratory, the distance selected in the field is dependent on the athlete’s fitness level. The more difficult aspect of lactate threshold testing in the field, compared with the laboratory, is the increase in intensity between test stages. A five-point subjective scale of exercise intensity is used by coaches of national athletes 1–easy, 2–steady, 3–moderately
practically possible, with the duration for each step being around four minutes to enable blood lactate concentration to reflect the increment for that step. Sometimes it is beneficial during an initial athlete assessment to conduct the LT test twice, with smaller steps being made during the second test to identify more closely the exercise intensity at which LT occurs for that individual. An LT test should be conducted with specificity in mind, for example, on a treadmill for a runner.

The simplest way to identify the LT is by visual inspection of the lactate concentration data. As with the detection of AnT (ventilatory breakpoint) however, the LT can sometimes be hard to pinpoint by visual inspection. Because of this, a number of test protocols and mathematical approaches to LT identification have been developed to identify the LT more clearly. Three of these approaches are illustrated in Figure 11.6. Beaver and colleagues (1985) recommended the use of a logarithmic transformation of oxygen consumption and corresponding lactate data (log-log transformation). The Dmax method, developed by Cheng and co-workers (1992), involves the creation of a plot of oxygen consumption and lactate concentrations during an incremental test. As is illustrated in Figure 11.6, a general direction (GD) line is drawn between the first and last points on the plot. The LT represents the point at which the greatest distance (distance maximum or Dmax) occurs between the lactate-VO₂ curve and the GD line. The individual anaerobic threshold (IAT) method involves a data collection period beyond the termination of exercise. The IAT test, proposed by Stegman and colleagues (1981), requires lactate samples to be collected post-exercise until, following the initial rise, blood lactate concentration returns to the level at maximal exercise (as can be seen in Figure 11.6). Having plotted the blood lactate data, the IAT is identified by drawing a tangent line from (a), the point at which lactate returned to the maximal exercise concentration to (b), the blood lactate curve. The tangent line is drawn as a straight line that just touches, but does not intersect, the lactate concentration data. The best LT method for the prediction of performance may depend on the sport and the duration of event.

The identification of lactate threshold

A number of different protocols have been used to identify LT. These have included continuous and discontinuous test protocols with stages of varying duration and sampling periods. The duration of each increment, along with the increasing intensity of exercise, affects the identification of LT. Consequently, increments should be as small as

<table>
<thead>
<tr>
<th>Key points</th>
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</thead>
<tbody>
<tr>
<td>Lactate threshold test:</td>
</tr>
<tr>
<td>➤ An incremental workload to identify AAT</td>
</tr>
<tr>
<td>➤ Can be performed in the laboratory or in the field</td>
</tr>
</tbody>
</table>

Figure 11.5 provides an illustration of a model lactate curve, along with representation of the effects of training on the lactate curve. Threshold training results in a shift of the lactate curve to the right, indicating that an athlete can, for example, run, cycle or row at a higher intensity before crossing the LT. This, in turn, leads to an improved performance in high-intensity endurance sports. Lactate samples are relatively easy to obtain, do not present great practical difficulties and represent an efficient method for AAT identification. The lactate threshold point appears to provide a valid and reliable marker of the AAT; however, this will be influenced by the quality of the protocol employed.

<table>
<thead>
<tr>
<th>Key points</th>
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<tbody>
<tr>
<td>Methods of lactate threshold identification:</td>
</tr>
<tr>
<td>➤ It can be identified visually or with mathematical transformation</td>
</tr>
<tr>
<td>➤ Mathematical approaches include: log-log transformation, the D-max method or the individual anaerobic threshold</td>
</tr>
</tbody>
</table>
Chapter 11 High-intensity aerobic endurance sports

Maximal lactate steady state test (MLSS test)

An alternative approach to obtaining the AAT point through the use of lactate sampling is provided by the use of an MLSS test. The tests already described for ATT identification, AnT, OBLA and the various forms of LT, are based around the execution of an incremental test. The aerobic-anaerobic transition is identified through MLSS by use of a series of 30-minute constant-intensity tests. The MLSS exercise intensity is the maximum that can be maintained for a 30-minute period with a less than 1 mM change in lactate concentration during the last 20 minutes of the test. Although first developed in the 1980s, one of the most useful methods for the identification of MLSS was that proposed by Dekerle and colleagues (2003). The first stage in MLSS testing is for an athlete to complete a \( V_\text{O}_2\text{max} \) test. The \( V_\text{O}_2\text{max} \) test is required to calculate the work intensity for the MLSS which is completed on a different day. In the first MLSS assessment the athlete completes a \( T_{30} \) (MLSS 30 min) at an exercise intensity equivalent to 75% of \( V_\text{O}_2\text{max} \). Blood lactate samples are collected at the start of testing and every five minutes until the last collection at the end of the 30th minute. On a separate day the athlete completes a second \( T_{30} \), the intensity of which is determined by the result in the first test. If an MLSS was reached or lactate concentration fell during the final 20 minutes of that test, the second test is completed at an exercise intensity 5% higher than in the first test. If the lactate concentration rose by more than 1 mM during the first test the exercise intensity is decreased by 5%. The \( T_{30} \) tests would need to be repeated, increasing or decreasing the exercise intensity until an MLSS was identified. The obvious disadvantage with this method relates to the time taken for testing, although this would probably decrease in future tests. Laplaud and colleagues (2006) developed a single incremental test to estimate MLSS. However, the mean 10 beats \( \cdot \text{min}^{-1} \) HR differences between the incremental test and the constant-intensity test for MLSS determination may preclude its use with athletes. A 10 beats \( \cdot \text{min}^{-1} \) difference in HR can represent a large difference when establishing training zones for athletes.

Practical use of AAT determination

As a high-intensity aerobic endurance athlete, whether a runner, cyclist, rower or cross-country skier, you aim to push yourself as hard as possible without straying too far into your anaerobic reserve. Knowledge of your AAT enables you to train at the most effective intensity to optimise training success and subsequent sporting performance.
Critical power

The concept of critical power (CP) is not a new one, and dates back to the mid-1960s. At that time, the concept was suggested as a theoretical maximal exercise intensity that could be sustained for a period of time without fatigue. In the 1980s this concept was developed into a test that could be used in a sporting context. Conceptually, CP refers to an exercise intensity that is between the LT and \( VO_2\text{max} \), and approximately equivalent to MLSS. Exercise at an intensity that is at, or close to, an athlete’s LT can be maintained for relatively long periods of time, e.g., for the completion of a marathon run. The maximal intensity required at the end of a \( VO_2\text{max} \) test, however, can be sustained for only a limited period of time. The exercise intensity for an MLSS tests can be sustained for 30 min and is consequently closely related to CP. Critical power was suggested as an exercise intensity above LT, but sustainable for a duration typically between 20–30 min. Figure 11.7 provides a representation of exercise intensity zones as they relate to the concepts of LT, MLSS and CP, such that LT and MLSS/CP represent the upper and lower limits of the heavy intensity exercise zone. The point at which MLSS and CP are achieved appears to represent a threshold above which exercise intensity becomes very heavy. It is important to note that while CP and MLSS have been shown here as occurring at the same time, research indicates that they are only approximately equal: in reality CP often occurs at an exercise intensity that is slightly above MLSS (approximately 5%).

Research has shown that CP is highly individual and generally occurs between 70–90% of \( VO_2\text{max} \). As a consequence, the power output or HR at CP for one person might represent the LT for another. Over time the concept of critical power has evolved, with the introduction of the concept of critical speed, which is particularly relevant to cycling. In this context, critical power is defined as the speed at which an athlete can maintain a constant speed on a bicycle, with no external forces, for a sustained period of time.
of CP, and tests of CP, have been extended and applied to a wider number of sports including running, rowing and swimming. In running, CP has been found to have a correlation of $r = -0.85$ with 10 k running time and a $r = -0.79$ correlation with half marathon time. For cycling $r = -0.71$ and $r = -0.91$ correlations have been found between CP and 17 km and 40 km time trial performance. In swimming, where the concept has been converted to a critical velocity (CV), a correlation of $r = 0.86$ was found between CV and 400 m freestyle swim mean velocity. These findings show that, similar to LT and MLSS, CP is highly related to sports performance.

Figure 11.8 provides a theoretical example of results from a CP test displayed in three related panels which illustrate the relationship between power output, time to exhaustion (TTE) and work completed. When originally developed by Scherrer and Monod (1965), the authors suggested that two key parameters could be calculated through critical power testing, both of which are illustrated in Figure 11.8. The first, CP, is the main focus of the test, while the second parameter is the anaerobic work capacity (AWC or $W'$). The anaerobic work capacity is illustrated in Figure 11.8a by the shaded area $W'$. While CP (also shown in Figure 11.8a) represents the maximal power attainable for a sustained period of time, the AWC represents a finite store of energy available at the start of exercise from anaerobic sources.

Critical power can be determined by anywhere between 2 and 10 exhaustive exercise trials depending on the protocol being used. Typically, the exercise intensity (power) for each trial is set such that exhaustion is reached between 1 and 30 min. In this example the exercise intensity was set at 450, 300, 250 and 225 W. The time to exhaustion for these examples was 90, 180, 300 and 420 s respectively which

Figure 11.8 Figures depicting the nature of critical power in relation to power, work and time to exhaustion.
11.1 Aerobic endurance sports and the importance of the aerobic-anaerobic transition (AAT)

gave rise to the plot in Figure 11.8a. The CP for the athlete in this model was calculated at 162 W (details of how to calculate CP are provided below). As can be seen from Figure 11.8a, when the power output is plotted against the time to exhaustion we see the characteristic hyperbolic curve that is typical of the decline in power with increasing duration of exercise (refer to Figure 8.1 in Chapter 8). The higher the power output required the shorter the time period for which the output can be maintained. As shown in Figure 11.8a the line representing CP acts as an asymptote to the power curve, such that the distance between the power curve and CP approaches zero as the lines trend towards infinity. In this way, in line with the classical model developed by Scherrer and Monod (1965), CP represents an exercise intensity that can be sustained for a relatively long theoretically infinite period of time.

In the example (Figure 11.8), four exercise trials have been illustrated and when work completed (W’) and power are plotted against time to exhaustion and the inverse of time to exhaustion, the linear relationship between these parameters is revealed. The longer the time to fatigue the greater work is completed (panel b) and the greater the power output required, the shorter the duration of time to exhaustion (remember that for mathematical reasons time is inverted in panel c). The work limit (Wlim) for a test is calculated by multiplying the power output (P) by the time limit (Tlim) – the time for which work can be sustained before exhaustion in the following equation:

\[ W_{\text{lim}} = P \times T_{\text{lim}} \]

The simplicity of CP testing (very little equipment is required and the test does not require invasive techniques such as lactate sampling) and its relationship with MLSS and performance make this test a useful one for athletes. There are a number of sport-specific protocols that can be used to establish an athlete’s CP. Such protocols vary but can be complex and time-consuming depending on the accuracy required; however, a straightforward laboratory method for calculating CP can be completed using a cycle ergometer equipped with power measurement and a stopwatch. Results are then generated with the use of statistical software.

A typical protocol involves three tests to exhaustion, ideally on separate days; however, these can be completed on the same day with suitable rest between each effort. After a 5-min warm-up the athlete should complete a constant cadence and intensity ride to exhaustion. Exhaustion would be defined as when the cadence cannot be maintained within 5 rev·min⁻¹ for a period of over 5 s. The intensity for each of the tests should be set such that ideally, exhaustion is reached within 1, 6 and 10 min respectively for each test. Table 11.1 suggests power outputs to reach exhaustion within these timelines for individuals of differing fitness levels.

<table>
<thead>
<tr>
<th>Fitness estimation</th>
<th>~10 min duration</th>
<th>~6 min</th>
<th>~1 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average level</td>
<td>170</td>
<td>195</td>
<td>220</td>
</tr>
<tr>
<td>Competitive sport player</td>
<td>230</td>
<td>265</td>
<td>300</td>
</tr>
<tr>
<td>Endurance athlete</td>
<td>300</td>
<td>345</td>
<td>390</td>
</tr>
<tr>
<td>Higher level endurance athlete</td>
<td>370</td>
<td>425</td>
<td>450</td>
</tr>
</tbody>
</table>

(Source: Adapted from School of Sport and Health Sciences, University of Exeter)

Table 11.1 Suggested power outputs (W) for critical power testing using a cycle ergometer.

Table 11.2 Example data for a CP test of a 13-year-old male participant.

<table>
<thead>
<tr>
<th>P 1 (W)</th>
<th>P 2 (W)</th>
<th>P 3 (W)</th>
<th>TTE 1 (s)</th>
<th>TTE 2 (s)</th>
<th>TTE 3 (s)</th>
<th>1/t 1 (s)</th>
<th>1/t 2 (s)</th>
<th>1/t 3 (s)</th>
<th>CP (W)</th>
<th>AWC (J)</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>140</td>
<td>147</td>
<td>170</td>
<td>429</td>
<td>332</td>
<td>159</td>
<td>0.002331</td>
<td>0.003012</td>
<td>0.006289</td>
<td>123.635</td>
<td>7401.392</td>
<td>0.996</td>
</tr>
</tbody>
</table>

Note: When using Statistical Package for the Social Sciences (SPSS), after running the regression model, CP is found from the constant value which and AWC from the predictor constant. \( R² \) is provided here so that when using this data set as a test model the model can be checked for accuracy. \( R² \) relates to the meaningfulness of the relationship between the variables. An \( R² = 0.996 \) indicates that was a meaningful relationship between the variables TTE and Power - which is shown very clearly in the linear relationship revealed in Figure 11.9.
The physiology of high-intensity aerobic endurance sports is closely related to the transition between aerobic and anaerobic metabolism. As such, an understanding of the concepts of LT, AnT and CP is important when considering them. The primary macronutrient for energy supply in these sports is carbohydrate in the form of glucose or glycogen. This has implications for glycogen storage, the enhancement of which is discussed in the next section: ergogenic aids for high-intensity aerobic endurance sports.

Firstly, this section discusses steady-state exercise, cardiovascular drift, exercise economy and oxygen debt as they all are important physiological concepts that relate, in particular, to aerobic endurance exercise. Following this we move on to discuss conditioning training, probable causes of fatigue and adaptations to training.

Steady-state exercise

In Chapters 9 and 10 the focus was upon anaerobic sports of short duration and high intensity during which a steady-state exercise level would not be achieved. Many of the sports that are the focus of this chapter and Chapter 12 enable a steady state to be achieved due to their sub-maximal exercise levels and longer duration. A steady-state exercise level is illustrated in Figure 11.10, and occurs when the energy supply for exercise meets the energy demands. At the start of exercise, respiration, heart rate and oxygen consumption rise to meet the body’s energy requirements. If an athlete exercises against a constant and sub-maximal workload, the heart rate and oxygen consumption will begin to plateau and after 3–4 minutes, a steady state will be reached (the nature of the steady-state plateau is an essential part of the Åstrand-Åstrand aerobic capacity test which is described in Chapter 12).

Figure 11.10 shows the plateau associated with steady state for three exercise intensities or workloads. As illustrated, the time to reach steady state increases as workload is increased and takes around 4 minutes in response to an initial workload. If the workload is subsequently increased the athlete will require a shorter time, of around 1–2 minutes to reach steady state for the second workload.

When exercise intensity or workload is increased above a critical level for any athlete he or she will not be able to attain a steady state for heart rate, oxygen consumption or respiration. This critical level is closely related to the concepts of MLSS and CP which were

![Figure 11.9 Example figure of CP using the data from Table 11.2.](image-url)
The physiology of high-intensity aerobic endurance sports

Figure 11.10 Steady-state exercise heart rate, respiration rate and oxygen consumption plateau.

Figure 11.11 Illustration of cardiovascular drift.

described in the previous section. In effect, these concepts relate to a steady-state max ($SS_{\text{max}}$), that is, a maximal intensity of exercise that can be sustained for a period of time. Although MLSS and CP were conceptualised as unique physiological measurements, research suggests the exercise intensities at which MLSS and CP are reached are similar. While during a CP test, given the exercise to exhaustion nature of the test, it is not appropriate to measure lactate concentration, the exercise intensity (power output) an athlete could maintain during an MLSS test would be closely related to his or her calculated CP or $SS_{\text{max}}$. Exercise intensity (demand) is related to physiological response (HR, lactate accumulation, etc.) and consequently, both CP and MLSS relate to a notion of a $SS_{\text{max}}$.

The research interest in the AAT and $SS_{\text{max}}$ should not be surprising, given that both constructs are important functional aspects of high-intensity aerobic endurance performance. Race performance is certainly correlated with MLSS, CP, the AAT and $SS_{\text{max}}$. Research also indicates that individuals who train for a specific aerobic endurance sport reach steady state for any given exercise intensity more quickly than sedentary individuals, and can exercise against heavier workloads before crossing the threshold above which steady state cannot be achieved. Improved aerobic capacity, demonstrated through increases in the number of mitochondria, development of capillaries and enzyme functioning, are responsible for the improved steady-state capacity.

Cardiovascular drift

An interesting response to prolonged, steady-state exercise is that of cardiovascular drift. This is associated with long-duration exercise and/or exercise in a hot environment. Figure 11.11 provides an illustration of cardiovascular drift.

Recalling from Chapter 6, cardiac output ($Q$) is the resultant of stroke volume (the blood volume ejected from the heart in one beat) and heart rate (the number of times per minute the heart beats). The rise in heart rate associated with cardiovascular drift is thought to be caused by a drop in stroke volume which is associated with prolonged exercise or exercise in the heat. To maintain $Q$ for a particular steady-state exercise intensity, heart rate must rise if the stroke volume drops. The drop in stroke volume is thought to occur for a number of reasons, all or some of which could occur during exercise. The upright body position during most aerobic endurance sports, for example running, kayaking and cycling, means that venous return must compete against gravity. It is thought that during exercise at a constant workload there is a progressive decrease in venous return which in turn leads to a decrease in stroke volume and necessitates a rise in heart rate. The
rise in body temperature and sweating associated with exercise are also thought to contribute to a decrease in stroke volume during prolonged exercise. As body temperature rises during prolonged exercise or exercise in the heat blood is diverted to the skin for cooling, which results in a decreased venous return and, consequently, stroke volume. This situation can be further exacerbated by the incidence of water loss through sweating which can lead to a decrease in blood volume and hence venous return through plasma removal to help maintain tissue hydration levels and body core temperature during exercise.

**Key points**

Cardiovascular drift:
- rise in exercise HR that is not attributed to increased exercise intensity
- associated with long-duration exercise or exercise in a hot environment
- occurs when blood flow is diverted to the skin to aid heat loss resulting in decreased SV and increased HR to maintain $Q\dot{}$

**Exercise economy**

Exercise economy relates to the energy demands of a given workload for any individual athlete. The relative efficiency of an athlete will have a significant impact on his or her response to a given workload. Exercise economy provides the interaction point between physiology and the skilful aspects of performance in sport. An example of efficiency can be found in rock climbing. When comparing climbers of different abilities the more skilful climber tends to be more efficient and smoother in their movements. Each movement and grasp of a new hold requires less effort and fewer muscle fibres to grip the hold. In a similar way, when learning a new route a climber grips tighter to each hold and requires more effort to make each movement. When the moves are learned the movement becomes smoother, more efficient, the effort required to grip each hold decreases and the overall energy demands of the climb are reduced. The more efficient and smoother a climber becomes in their movement, the greater exercise economy they possess.

A further example can be found in rowing. The ability of the same individual to move through the water at a set speed is influenced not only by their physiological ability, but many other factors such as the angle of the blade entry and exit, the duration of the stroke, and body movement with each stroke. With an optimised technique, exercise economy is improved, with the movement of the boat through the water becoming smoother and more efficient.

Exercise economy is typically measured by the assessment of oxygen consumption across a range of workloads or exercise intensities. As with other aspects of physiology, exercise economy has been studied most with regard to running and indicates that race performance is improved when the relative oxygen consumption for a given workload is lower than that for other athletes. In sports like swimming, which has also been the focus of exercise economy research, the findings have been similar. Not surprisingly, higher level swimmers were more efficient than less skilful swimmers who spent more time and energy maintaining a horizontal body position in the water. The results from swimming have application to other more technical aerobic endurance sports such as rowing, kayaking and cross-country skiing for they stress the importance of the skilful aspects of the sport. In sprint kayaking for example the interaction between the paddler’s physiology, their boat awareness, feel for the water and use of the blade are critical for performance. The development of technical aspects alongside improved physiological performance are essential for exercise economy. Research indicates that if you improve exercise economy you delay fatigue and improve performance.

**Oxygen deficit and Excess Post-exercise Oxygen Consumption (EPOC)**

When we start to exercise there is an immediate rise in the body’s demand for oxygen, which results in an increase in heart rate and breathing frequency to match oxygen delivery to the exercise requirements. As has been described, it takes time for the supply of oxygen to the active muscles and aerobic metabolism to meet the demands of exercise and therefore to reach a steady state during sub-maximal exercise. This delay in the rise of aerobic metabolism and oxygen consumption at the start of exercise, first identified by Archibald Hill and Hartley Lupton in 1922, creates what they termed an oxygen deficit. The maximal accumulated oxygen deficit (MAOD) test described in Chapter 10 represents a measure of oxygen deficit. The higher the exercise intensity the longer it takes for the body to reach steady state and the larger the oxygen deficit incurred.
As the temperature drops over time, post-exercise oxygen consumption also drops. It is believed that a further part of EPOC is associated with the removal of CO₂ and the conversion of lactate to glycogen. As was described in Chapter 8, the onset of exercise causes the release of adrenaline and noradrenaline which serve to stimulate an increase in metabolism. These sympathetic nervous system hormones are not immediately removed from the blood and continue to stimulate metabolism, and consequently oxygen consumption, prior to their removal. It is likely that the EPOC is also related to tissue repair and the re-sequestering of Ca²⁺, K⁺ and Na⁺ ions to their non-exercising compartments within muscle fibres. Further research is ongoing in this interesting area of physiology; however, it appears there are a number of mechanisms behind the continued elevation of oxygen consumption post-exercise.

Knowledge integration question

Upon cessation of exercise we can feel that HR and ventilation remain elevated. What are the likely reasons for this and how do exercise intensity and duration affect the rate of recovery?

Key points

The commencement of exercise results in:

- an immediate rise in muscle demand for oxygen = increased HR and \( V_e \)
- a time delay to match oxygen supply to demand (i.e. to reach steady-state exercise) is the oxygen deficit

Oxygen deficit is an important factor in the increased \( O_2 \) consumption after exercise – known as the excess post-exercise oxygen consumption (EPOC) or oxygen debt.
Conditioning for aerobic endurance sports

Due to the wide variation in intensity and duration that can be found in aerobic sports, the physiology of these sports is covered in two chapters (this chapter and Chapter 12). There would consequently be overlaps in the training methods covered if conditioning was considered in both chapters. To avoid such overlap, aspects of conditioning training for competitive aerobic sports are covered in this chapter, while in Chapter 14 we will consider exercise prescription for health.

In Chapter 8 a brief outline of general aspects for conditioning training was provided. Table 8.5 provides an overview of a number of different forms of training that could be considered for any athlete. An aerobic athlete might use all of these forms of training depending on the duration and intensity of the sport in which he or she takes part. There is, however, a much greater range of training methods that could be employed by a coach or an athlete which would be beneficial to aerobic endurance performance. Some of these are illustrated for marathon kayaking in Anna Hemmings’ Sport in Action piece (p. 348). In this section we will consider a number of types of conditioning training that could form the basis for an aerobic endurance athlete’s programme.

In making decisions about the types of training to include it is very important that attention is paid to the principle of sport specificity. Clearly, following this principle of training (which was outlined in Chapter 8), a conditioning programme for a runner should be based around running whereas that for a triathlete would need to include all three disciplines; swimming, cycling and running. Additionally, however, choices for a training programme should be specific to the intensity and duration of the sport.

Training intensity

Regular physiological testing can help with the design of a sport specific training programme. As was outlined in Chapter 8, HR monitors can be used to set and check the intensity of training for any particular session. Such intensities could be set from two possible starting points and careful consideration should be paid to which is more appropriate for use with a particular athlete. A test, such as a VO$_{2\text{max}}$ (which will be described in more detail in Chapter 12), could be used to identify an athlete’s HR$_{\text{max}}$. Once this is determined it is possible to specify the intensity of training session relative to his or her HR$_{\text{max}}$. This method was used to identify the intensity of training for types of conditioning training shown in Table 8.5.

Increasingly, however, the results of LT, MLSS and CP tests have been used to prescribe the intensity of training for aerobic endurance athletes. An athlete’s HR response for one of these given measures can be used to prescribe the intensity of sessions, with sessions differentially designed to be below, at or above the HR at LT, MLSS or CP. The choice of which of these measures would be most beneficial for an athlete would be based upon the duration of the sport and the type of sport itself. Training based around the HR at LT has most commonly been used in running, whereas CP has been used as the basis for exercise prescription in cycling and swimming (where the parameter critical velocity is used). An illustration of training based around LT, using the terminology developed by Bompa, can be seen from Figure 11.13.

Anaerobic threshold/pace-tempo training

As can be seen from Figure 11.13, anaerobic threshold training (AnTT) refers to training that is just above LT. Very similar in concept to (race) pace-tempo training, the idea with such training is to increase the speed at which LT occurs and to improve running economy. Depending on the duration of the event or sport an athlete is training for, LT will be close to race pace for an athlete. Pace-tempo training, as the name suggests, is designed to improve race pace by working at a tempo just above normal race pace. Bompa suggested that repetitions for AnTT might vary between 90s –1 hr depending on the duration of the competition event, and the tempo is relative to race pace for the repetition or session. Research suggests that AnTT or pace-tempo training will enhance both aerobic and anaerobic metabolism, making this form of training particularly useful for high-intensity aerobic endurance sports.
The physiology of high-intensity aerobic endurance sports

Fartlek training

As for anaerobic endurance athletes, interval sessions provide an incredibly versatile form of training for aerobic endurance athletes. As is shown in Table 8.5, the intensity and duration of the work phases, along with the number of repetitions, sets and length of recoveries can be manipulated to meet specific training goals. As such, interval sessions should provide an integral part of any aerobic athlete’s training programme. Additionally, fartlek and long slow distance (LSD) sessions, also illustrated in Table 8.5, are beneficial forms of training for aerobic endurance athletes.

The word fartlek is a Swedish word meaning speedplay and relates to a form of continuous training in which the intensity of exercise is varied throughout the effort. Fartlek training has mainly been associated with running; however, it can have application to other sports. In middle distance running, Kenyan athletes have become famous for changing the pace of a race throughout its duration such as applying a surge in race pace followed by a drop in pace. This race strategy has enhanced fatigue for athletes not used to this type of pace change; however, fartlek training can help to alleviate the fatiguing effects of race pace changes. An example of a 35-min fartlek session might include 3 exercise intensities (individually devised, but based around high-, moderate- and low-intensity efforts) with efforts at each intensity lasting for varying duration (1–5 min).

Long slow distance training

Long, slow, distance training, which should be carried out at an exercise intensity between 50–70% of HRmax, has a number of potential benefits for athletes. This form of training can lead to improved oxidative capacity and thermoregulation as well as facilitating a sparing of glycogen. It is worth noting that, because this form of training is by definition below race pace, there should not be an over-reliance on LSD as this might negatively affect exercise economy.

Strength training

An overview of strength training can be found in Chapter 9 and a well balanced training programme for an aerobic endurance athlete should include both strength and conditioning training. Although not always considered for aerobic endurance athletes, strength training has been shown to provide a number of benefits to performance. Strength training is thought to benefit aerobic endurance athletes by reducing muscle imbalances and assisting with recovery from injury. In addition, strength training can serve to improve hill climbing and the ‘kick’ during a race.

Key points

Conditioning for aerobic endurance events should include:

➤ anaerobic threshold training – to increase speed at which LT occurs and to improve exercise economy
➤ fartlek training – to increase ability to cope with changes in race pace
➤ long slow distance training – to improve oxidative capacity and thermoregulation
➤ strength training – to reduce muscle imbalances and aid recovery from injury

Fatigue in high-intensity aerobic endurance sports

Short-duration events

The mechanisms for fatigue in high-intensity aerobic endurance sports relate to the duration of the event. For endurance sports, those on the left of the duration continuum (Figure 11.2), a number of possible contributors to fatigue have been postulated. Fatigue in sports, such as rowing and cycling (3,000 and 4,000 m pursuit), is closely linked with anaerobic endurance fatigue. The most commonly reported mechanism for fatigue is the increasing acidosis, associated with increasing H+ as a consequence of an extended reliance on fast-rate glycolysis due to the high intensity of effort. More recently, however, Pi and K+ have been implicated as possible alternatives or contributary explanations of fatigue. The possible mechanisms, along with current thinking about the role of decreasing pH in fatigue, were described in Chapter 10, but have clear implications for performance in shorter-duration aerobic sports that are the focus of this chapter.

Long-duration events

As the duration of aerobic endurance sports increases the relative intensity falls (see Figure 11.1), with glycogen depletion increasingly becoming the major mechanism behind fatigue. Glycogen stores in the muscles and liver are the major substrate for ATP regeneration, particularly during high-intensity aerobic endurance sports. It is likely that the intensity and duration of these sports is such that,
Physiological adaptations to aerobic endurance training

The completion of an aerobic training programme, based on the conditioning training methods described above, will lead to a variety of physiological adaptations in response to the training stress. This section describes the key adaptations that occur in response to aerobic training and so provide the basis for improvement in performance for any of the sports in this chapter or the lower-intensity sports covered in Chapter 12. The adaptations to aerobic training include alterations in cardiac, respiratory, muscular and metabolic function.

Aerobic power adaptation

The most obvious response to aerobic training is an improvement in aerobic power ($\dot{V}O_{2\text{max}}$) which results in an increased ability to maintain a higher intensity and duration of exercise. The intensity, duration, type and specificity of training will all influence the extent of any physiological adaptation. The extent to which adaptation occurs will also be dependent upon the individual’s previous training and their individual response to the training stimulus. For instance, improvements in $\dot{V}O_{2\text{max}}$ have been shown to occur primarily within the first 6–12 months of training. After this time, improvements in performance tend to be related to improvements in their LT and exercise economy. Consequently, if an individual is new to aerobic training their improvement in $\dot{V}O_{2\text{max}}$ will be significantly greater than that for an experienced athlete. Nevertheless, through training, experienced athletes can still continue to improve their performance. Individuals respond differently to any training stimulus and therefore coaches, or the athletes themselves, need to monitor the response to training and alter the programme if the desired results are not being realised.

Knowledge integration question

How does the duration of an aerobic endurance event relate to the mechanism of fatigue? How can fatigue be delayed in such sports?
Adaptations of skeletal muscle fibres

Muscle fibre size and type

In Chapter 6 the properties of different muscle fibre types were described in detail. During aerobic activities type I fibres are primarily recruited for exercise. In response to aerobic training type I fibres tend to decrease slightly in size. At first this might seem contrary to logic; however, during aerobic training type I fibres are not required to produce maximal forces, but to complete repeated lower-intensity contractions. As was described in Chapter 9, strength training is associated with hypertrophy (increase in muscle fibre size). Consequently, aerobic endurance athletes should undertake a strength training programme, to maintain muscle strength and size, in conjunction with aerobic training. By combining strength and aerobic training an athlete can maintain muscle size and strength, along with making aerobic gains.

The protein degradation within muscle fibres in response to aerobic training is thought to arise from changes in the production of the hormones cortisol and testosterone. In response to aerobic training, levels of cortisol tend to increase. Cortisol has been shown to be associated with protein degradation in type I muscle fibres. In contrast to this, plasma levels of testosterone, an anabolic hormone (stimulates protein synthesis) tend to fall in response to aerobic training. Together, these hormonal changes combine to create a predominantly catabolic environment within type I muscle fibres.

In addition to the reduced size of type I muscle fibres, research findings indicate there are also changes in the properties of type II fibres. In response to aerobic training, type IIx fibres alter their structure and functioning to that of type IIa fibres and, similarly, type IIa shift towards the properties of type I fibres. These adaptations of type II fibres enhance their oxidative capacity, particularly in response to high-intensity aerobic endurance exercise. Training-induced changes in the metabolism of type I muscle fibres are discussed below.

Muscle fibre structure and metabolism

Although aerobic training results in a decrease in the size of type I muscle fibres there are a number of structural changes within these fibres that promote increases in aerobic endurance performance. In response to aerobic training there is an increase in the oxidative capabilities of type I muscle fibres. Endurance training brings about increases in the capillary density, myoglobin concentration, mitochondrial size, number and function, which together serve to improve the aerobic capability of the trained fibres. The number of capillaries surrounding each muscle fibre can increase by 10–15% through training, thereby enhancing the amount of oxygen available for active muscle fibres.

In addition, more oxygen can be stored due to an increase in myoglobin concentration within type I muscle fibres. Myoglobin concentration has been shown to increase by as much as 75–80% above pre-training levels.

Further to these improvements in oxygen delivery and storage, aerobic metabolism is also improved through changes in mitochondria. These are the site of oxidative phosphorylation within muscle fibres and have been shown to increase by up to 35% in size and 15% in number in response to aerobic training. Further to this, their increased efficiency has been demonstrated to be due to greater enzyme activity. As an example, the Krebs cycle enzymes citrate synthase and succinate dehydrogenase (SDH) increase their activity in response to endurance training (the Krebs cycle is described in detail in Chapter 12). Interestingly, the intensity of training appears to dictate the degree to which SDH activity is increased; higher-intensity training promotes a greater increase in SDH activity compared to lower-intensity aerobic training.

In addition to increases in enzyme activity within the Krebs cycle, the enzymes involved in β-oxidation (the start of lipid catabolism: refer to Chapter 12) respond to aerobic training. The resultant increases in enzyme activity for fatty acid metabolism can be as much as 25% (or even higher) and have a glycogen-sparing effect. In addition, the muscles can store greater amounts of triglyceride which creates an increased lipid pool immediately available for β oxidation and fatty acid oxidation. Training has been shown to more than double the resting muscle triglyceride stores. These changes result in a decrease in RER values for a given work intensity. A decrease in RER during aerobic endurance exercise provides evidence of the shift towards fat oxidation as a consequence of aerobic training. It is thought that the shift to fat oxidation is accompanied by a decreased response of the sympathetic nervous system (SNS) to exercise. At the start of exercise the SNS, through

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**Key points**

Muscle fibre type adaptations to aerobic endurance training:

- diameter of type I muscle fibres falls – increased cortisol and decreased testosterone promote a catabolic environment
- strength training should be included in programmes to maintain muscle strength and size
the release of the hormones adrenaline and noradrenaline, increases metabolism and the body’s reliance on glycogen as a fuel source. As a result of training the SNS response to exercise is blunted, decreasing the emphasis on carbohydrate as the main fuel source.

As well as improvements in fatty acid storage and oxidation, aerobic training appears to result in increases in glycogen synthesis and storage. Aerobic training appears to trigger an improved insulin response, leading to an increased uptake of glucose by muscles. The enhancement in glycogen storage brought about through this mechanism has been demonstrated to lead to improved aerobic performance and to result in a delayed onset of fatigue. Glycogen sparing also appears to be enhanced through a training-induced improvement in mitochondrial functioning which serves to slow glycogen oxidation and decrease lactate production.

**Knowledge integration question**

What training-induced adaptations contribute toward the increased reliance on fat oxidation by the muscles?

**Key points**

Type I muscle fibres show:

- increased capillary network – enhanced oxygen availability
- mitochondrial adaptations: increased, number, size, and function (increased aerobic enzyme activity) = improved oxygen storage and aerobic metabolism
- increased triglyceride storage
- increased metabolism of fat has a glycogen-sparing effect
- improved insulin response increases muscular glucose uptake and glycogen storage

Combined, these adaptations result in an improved aerobic endurance performance.

**Cardiovascular changes with training**

Aerobic training results in a lower resting heart rate and a decreased heart rate response to exercise (maximal HR is normally little altered in response to training). The mean resting heart rate for a sedentary individual is about 72 bts · min⁻¹. As a result of aerobic training the resting heart rate can fall below 50 bts · min⁻¹. Despite a lower resting and sub-maximal heart rate response, an athlete’s Q (the resultant of heart rate and stroke volume) has been shown to increase in response to endurance training. The increase in Q is brought about through improvements in stroke volume. Aerobic training results in the heart increasing in size, causing an increase in the volume of blood ejected from the left ventricle with each contraction.

**Training-induced LT adaptations**

In sustained endurance training one of the key adaptations is an improvement in LT. In an AAT test, improvements in LT can be found by a shift to the right of the lactate curve as is illustrated in Figure 11.5. Aerobic training results in improvements in LT, conversely, research indicates that the most effective way to improve oxidative functioning is to train at or just above the exercise intensity at which LT or AAT occurs. Improvements in LT have been found to be specific to the exercise mode. Running training results in a greater shift in the point at which LT occurs in a treadmill test compared to a cycling test. Consequently, LT test apparatus should as far as possible match the demands of the sport.

**11.3 Ergogenic aids for high-intensity aerobic endurance sports**

Beyond a healthy balanced diet, the key legal manipulation that could be beneficial to high-intensity aerobic endurance performance is carbohydrate loading. Certainly for longer-duration sports such as marathon running, where glycogen depletion represents a significant possible cause of fatigue, the employment of a glycogen loading strategy and the use of in-race carbohydrate feeding can help to maintain performance. As a consequence, this section begins with a review of carbohydrate loading. Although aerobic metabolism is the predominant source of ATP, a significant energy contribution from fast-rate glycolysis continues in short-term aerobic endurance events. The use of bicarbonate and β-alanine supplements (as discussed in Chapter 10) may therefore also be beneficial as pH buffering agents in events
such as a 1,500 m run or a 400 m swim. Following discussion of carbohydrate loading, we will consider the case of caffeine, as a widely available, legal supplement which has a wealth of research describing its possible ergogenic effects.

**Carbohydrate loading**

Carbohydrate loading (also referred to as carbo-loading, glycogen loading or glycogen super-compensation) involves the manipulation of diet to increase the muscle glycogen stores prior to an endurance event. As was discussed in the section on fatigue, glycogen depletion leads to feelings of ‘hitting the wall’ and has been shown to be detrimental to performance. Research indicates that for prolonged, high-intensity sports (over 60 min), detrimental to performance. Research indicates that for prolonged, high-intensity sports (over 60 min, \( \geq 75\% \text{ of } V_{O_{2max}} \)), carbohydrate loading can improve performance by increasing the time to exhaustion, or, in other words, delaying fatigue. The classic studies into glycogen depletion and carbohydrate loading took place in the 1960s and form the basis from which modern forms of carbohydrate manipulation were developed. Research by Bergström and colleagues, published in 1966 and 1967, provided a major breakthrough in the understanding of the effects of glycogen loading on performance. Bergström and Hultman (1966) carried out a one-legged cycle test to deplete muscle glycogen stores in the exercising leg. The participants exercised for several hours after which muscle biopsies were taken to record the levels of glycogen stored within the leg muscle. After a 3-day period, during which a high-carbohydrate diet was followed, the glycogen levels of the exercised muscle were nearly double those in the non-exercising leg. This study indicates that a bout of glycogen-depleting exercise prior to adopting a carbohydrate rich diet results in a higher storage of glycogen in muscle. In 1967, Bergström and colleagues published findings regarding the effect of diet on performance. In this study, nine participants followed a normal diet, then three days of a low-carbohydrate diet and finally three days of a high-carbohydrate diet. After each diet they completed a cycle ergometer ride to exhaustion (at 75% \( V_{O_{2max}} \)). The mean exercise time following a normal diet was 115 minutes, which decreased to 60 minutes after the low-carbohydrate diet. However, after three days of a high-carbohydrate diet the time to exhaustion increased to a mean of 170 minutes. These and subsequent studies have indicated that carbohydrate loading can increase muscle glycogen stores by 40–100%, depending upon the regimen followed.

A classic model for carbohydrate loading was developed from the results of these findings. This model involves an athlete altering their diet and exercise pattern in the final week before a competition. After three days following a low-carbohydrate diet (i.e. a fat and protein based diet) an athlete performs an exhaustive exercise bout to deplete glycogen stores. In the remaining days before the event the athlete adopts a high-carbohydrate diet. Following this model glycogen stores are severely depleted before bouncing back above normal resting levels, hence the term glycogen super-compensation.

The use of this dietary manipulation undoubtedly led athletes to increase their pre-race glycogen stores, but a number of drawbacks meant this was not reflected in optimal performance, hence alternative strategies were developed. The three days following a low-carbohydrate diet and exhaustive exercise caused feelings of fatigue, dizziness and led to mood change, including decreased self-confidence and self-belief prior to competition. These changes appeared to have a detrimental effect on subsequent performance. Further to this, following the classic model also interfered with an athlete’s final taper to the event. Identification of these drawbacks led to the development of a modified carbohydrate-loading regimen by Sherman and colleagues (1981, 1982). The Sherman model involved a decrease in exercise in the week prior to competition and an increase in carbohydrate consumption to represent 70% of the total calorific intake. Following the Sherman model, athletes were found to have very similar pre-race gains in muscle glycogen storage to the classic model, but without the side effects. This model not only provided a more straightforward method for increasing muscle glycogen storage, but also presented a regimen that matches the alterations in training associated with the taper for competition.

Research indicates that a typical glycogen loading diet should contain around 500–600 g of carbohydrate per day. Table 11.3 provides an example of a one-day menu where the carbohydrate contribution would be within this range. As indicated in the table, increases in total carbohydrate ingestion should come from complex carbohydrates as found in foods such as wholewheat pasta, bread and cereals, not through the consumption of simple sugars. Consuming a high-carbohydrate diet prior to competition can improve performance and delay fatigue. Athletes should consider one further aspect before the adoption of a pre-race carbohydrate loading regimen. The improvements in performance for cycling have been better than those for running and the difference may well relate to the weight-bearing nature of running. Each gram of glycogen stored results in the storage of 2.7 grams of water. This additional stored weight (2.7 g H₂O + 1 g glycogen) creates a 0.5 kg to 2.0 kg increase in total body weight that must be carried at the start of a race, having a larger...
A further aspect to consider for high-intensity aerobic endurance sports is that of in-race carbohydrate feeding. In events lasting more than one hour the consumption of a carbohydrate sports drink or gel has been shown to result in improvements in performance and delaying of fatigue. The use of a carbohydrate-electrolyte sports drink, the most effective for delaying fatigue (Maughan et al. 1989), has not only a glycogen-sparing effect, but also helps to maintain hydration, electrolyte balance and to make fluid intake more palatable. The effects of dehydration can have a major impact on performance in endurance events with a 2% drop in bodyweight (through water loss during exercise) associated with a resultant drop in speed. Consumption of a 6–8% carbohydrate drink during exercise is recommended for improved performance. A higher concentration can affect gastric emptying and lead to discomfort so ideally should be avoided. If employing a carbohydrate-drinking schedule during a race, ingestion should begin within the first 30 minutes of exercise and continue at a rate of 500mL Hr⁻¹ for the duration of the race. A 6–8% carbohydrate solution would represent an intake of about 30–40 g of carbohydrate per hour (about 6–8 grams per 100 ml).

In addition to carbohydrate, electrolytes are commonly included in drinks consumed during endurance events. As mentioned previously, the primary mechanism of heat loss during exercise is the evaporation of sweat from the surface of the skin. Sweat contains a variety of electrolytes, especially sodium and chloride. The loss of sodium through sweating is of greatest concern to the athlete due to its potential link with muscle cramping. The composition of sweat varies greatly between individuals, hence an athlete must not only work out their sweat rate in different conditions, but also analyse their sweat sodium concentration. This will aid in determining the ideal composition of drink for them.

An additional advantage obtained through the use of a carbohydrate-electrolyte drink relates to the rates of water and sodium absorption which are enhanced when combined with carbohydrate. As with carbohydrate loading, it is important to practise a drink strategy before a major event. An athlete’s rate of fluid loss (pre-weight minus post-weight, compensating for fluid intake and urine loss) should be determined. The volume of fluid consumed during prolonged exercise must be sufficient to prevent a loss of body mass of ≥2%. The timing and volume of fluid intake should be individualised to determine what can be tolerated without gastric discomfort.
Ergogenic aids for high-intensity aerobic endurance sports

An exercise intensity of 80% of \( V\dot{O}_{2\text{max}} \) Time to exhaustion was nearly 20% higher during the caffeine trial and the cyclists exhibited a lower respiratory exchange ratio (RER) value indicating a shift towards fat metabolism, thereby sparing glycogen stores. In addition, the athletes reported the caffeine trial as being easier than the placebo trial. Subsequent studies have produced similar findings with caffeine resulting in a 10–20% improvement in time to exhaustion.

At rest, caffeine is a diuretic and this presents a potential problem regarding its use as an ergogenic aid. The diuretic effect, however, is negated during exercise due to catecholamine release at the start of exercise that stimulates the release of anti-diuretic hormone; this increases water re-absorption and counteracts the diuretic effects of caffeine. The findings of a variety of studies suggest that an ingestion of 3–5 mg of caffeine per kg bodyweight is sufficient to create an ergogenic effect. For those who consume caffeine regularly, however, any ergogenic effect would not be realised unless the athlete completed a wash-out phase (did not consume caffeine) prior to the event. Research suggests that omission of caffeine from the diet for 5–6 days should be sufficient to establish an ergogenic effect for regular caffeine consumers. For those that do not normally consume caffeine, or when a dose is higher than normally encountered, there are potential side effects which include restlessness, elevated heart rate, insomnia and headaches. Unless consumed in excess doses caffeine ingestion does not generally present a health risk and as such represents an ergogenic aid that can positively affect performance in a wide range of sports.

Caffeine was a restricted substance until 2004 when WADA removed it from the banned list. Although a monitored substance, athletes are now able to use caffeine as a performance supplement. Again, any individual using caffeine as an ergogenic aid has to make a moral decision about its ingestion despite the recent changes in the regulations regarding caffeine and sports performance.

**Key points**

Ergogenic aids for high-intensity aerobic endurance sports are:

- short duration – β-alanine and bicarbonate
- longer duration – glycogen loading and in-race feeding
- caffeine – can improve time to exhaustion by 10–20%

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Caffeine as an ergogenic aid

Caffeine, one of the most widely-consumed drugs, is found naturally in tea, coffee and chocolate. A tea bag contains around 65–100 mg, ground coffee 125 mg per cup and milk chocolate in the region of 50 mg per small bar. Caffeine is a purine, similar in structure to adenine which forms the basis for ATP, and is a central nervous system stimulant. The effects of caffeine are widespread and include increasing mental alertness, concentration, mood state, fatty acid mobilisation, catecholamine release (adrenaline and noradrenaline) and muscle fibre recruitment. Caffeine consumption also results in a decrease in perception of effort, prolongs the time to fatigue and reduces reaction time. As a consequence, caffeine represents the most wide-ranging of the ergogenic aids across the eight chapters in Part II of this textbook. Increases in mental functioning can impact on all sports. Decreases in reaction time, and increases in muscle fibre recruitment and catecholamine release, can result in performance improvements for power and power endurance activities (Chapter 9). The increased catecholamine release and reduced perception of effort represent potential improvements in performance for anaerobic endurance events (Chapter 10). The lowered perception of effort, the glycogen-sparing effect of increased fatty acid mobilisation and usage, along with the delaying of fatigue, can significantly improve performance in intermittent and aerobic activities (Chapters 11, 12 and 13).

The benefits of caffeine ingestion before exercise have been most widely researched and reported for endurance sports. A classic study by Costill and colleagues published in 1978 provides an excellent example of the effects of caffeine on endurance performance. In this study nine cyclists consumed caffeine in one trial, and a placebo in the other trial, during which they cycled to exhaustion at

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**Key points**

A carbohydrate-electrolyte drink consumed during exercise can:

- reduce rate of dehydration
- have a glycogen sparing effect
- maintain electrolyte balance
- increase fluid palatability
- delay fatigue and improve performance
Chapter 11 High-intensity aerobic endurance sports

Check your recall
Fill in the missing words.

➢ Aerobic endurance sports primarily utilise the ___________ energy system; however, a switch to ___________ energy pathways occurs during a finishing sprint.

➢ The transition from aerobic to anaerobic metabolism is known as the ___________ ___________ threshold.

➢ The anaerobic threshold is also referred to as the ___________ threshold.

➢ The point at which ___________ begins to accumulate is commonly used to identify an athlete’s AAT.

➢ As a non-invasive measure, ___________ corresponding to lactate threshold or maximal lactate steady state can be used to set training zones.

➢ When performing sub-maximal exercise, steady state is achieved when energy demand is met by energy ___________.

➢ Beyond a critical level, the ___________ threshold, it is no longer possible to achieve steady state.

➢ The delay in the rise of oxygen consumption at the start of exercise is termed an oxygen ___________.

➢ The elevated oxygen consumption and ventilation upon completion of exercise is known as the excess post-exercise oxygen consumption (EPOC), also referred to as the oxygen ___________.

➢ In long-duration endurance sports, fatigue results from ___________ depletion.

➢ A gradual increase in HR during prolonged steady-state exercise is known as ___________ ___________.

➢ In short-duration aerobic activities, fatigue results from the accumulation of ___________.

➢ As the exercise intensity falls and duration increases, ___________ depletion becomes the predominant mechanism behind fatigue.

➢ Aerobic power improvement with training occurs mainly in the first ___________ to ___________ months of training.

➢ Following training, there is a metabolic shift toward ___________ oxidation by muscle fibres.

➢ A regimen commonly used by long-duration aerobic athletes to enhance performance is that of ___________ loading.

Review questions

1. Define the Aerobic-Anaerobic Transition. Why is it important to know the exercise intensity at which this occurs when training a high-intensity endurance athlete?

2. Why do the increases in exercise intensity need to be relatively small when conducting an incremental lactate threshold test? Why will some physiologists have a performer complete a second LT test with smaller intervals?

3. With the use of graphs, discuss the different methods used to identify the lactate threshold from a lactate curve.

4. How does testing for critical power differ from that carried out for lactate threshold or maximal lactate steady state?

5. Why are lactate threshold, maximal lactate steady state and critical power tests more useful to coaches and athletes than VO2max tests?

6. In practice, how are training zones identified during the development of a training programme for an athlete?

7. What is meant by cardiovascular drift? Explain why it may occur during high-intensity aerobic endurance activity?

8. Why is it important to consider the sport of an athlete before conducting aerobic power testing? Why is it difficult to compare meaningfully the VO2max scores of sea kayakers and fell runners?

9. What is an oxygen deficit? Why might the continued elevation of oxygen consumption continue after the oxygen deficit appears to be repaid?

10. What happens to levels of cortisol and testosterone as a result of aerobic training? How does this impact on type I muscle fibres?

Teach it!

In groups of three choose one topic and teach it to the rest of the study group.

1. Explain the importance of the aerobic-anaerobic transition to the endurance athlete. What tests can be used to assess aerobic endurance? In practice, which tests are most commonly adopted with elite athletes?

2. Discuss the structural and functional changes in muscle tissue with aerobic training, linking the adaptations to improvements in performance.

3. Taking into account the range of endurance event durations, what are the primary mechanisms of fatigue? Discuss ways in which the development of fatigue can be delayed and performance enhanced.
Biography: Tim Brabants

Adventuresport: Kayak sprint racing

Tim, a 29-year-old doctor based in Nottingham, is a K1 kayak sprint racing specialist. He started canoeing at the age of 10 when taken by his mother to a come-and-try-it session at Elmbridge Canoe Club. The club has a strong tradition in sprint and marathon racing and it wasn’t long before Tim was taking part in domestic competitions. His discipline is K1 1,000 m and he has won titles and medals at national and international levels. In 2008 he won a K1 gold medal in the K1 1,000 m and a bronze in 500 m at the Beijing Olympics to add to his World K1 1,000 gold medal in 2007 and Olympic bronze in 2000. He is the current world record holder for the 1,000 m sprint, a time he set in the heats at the Athens 2004 Olympic Games. He was junior World Champion in 1995, senior European Champion in 2002 and 2006. Away from the 1,000 m distance he joined with Conor Holmes in 1998 to take a K2 silver medal at the Marathon World Championships.

Sydney 2000 Olympic Games

Sprint kayak racing is held on a 2,000 m regatta lake, racing over a distance of 1,000 m or 500 m in single, double or four-man kayaks. Each race has nine lanes with stationary starts and events are organised into heats, semis and finals.

My first shot at qualifying for an Olympic Games was whilst still a junior in 1995, narrowly missing out on competing in the Atlanta Olympics 1996. Three years later I was fitter, stronger and faster and knew that to qualify for Sydney I would need to come in the top eight at the World Championships in 1999. I performed well, finishing sixth. From that moment all my focus was on training and preparing for the Olympic Games in Sydney 2000. I took time out of my medical degree to make more time for training. We work on four-year cycles so this was the big year.

Training two to three times a day, six days a week is tough. The coldest months are the worst when the water freezes on your clothing, kayak and paddles, and your hands hurt for an hour after as they re-warm. Your muscles are aching every day from weight training. Training camps, World Cup and European Championship races are also that year. We race and train in a multitude of climates and altitudes so one has to develop effective ways of acclimatising to each venue quickly and successfully. The physical demands of my sport require a high aerobic capacity combined with good strength endurance as well as an explosive element. Training has to be carefully planned and my coach Eric Farrell was excellent at this. Every day follows the same routine but every day you’re closer to your dream of competing in the Olympic Games.

As a nation we had never medalled in sprint kayak racing at the Olympics. The build-up was different from anything I had experienced. All of a sudden there was a large increase in media attention with radio, television and newspaper interviews. Also there was lots of information about the Olympics, travel and boat transport arrangements, measuring for kit etc. It would be easy to become caught up in all of this and get distracted. I knew I wanted to go to Australia and race the best race of my life so far; it wasn’t just about competing at the Olympics. To do that would require me to stay focused, train hard and manage my time better with the increased demands. Good quality recovery time enables good quality training.

I’d raced at major events before but I knew this would be different mainly from a psychological...
viewpoint. The other main challenges would be the time difference, climate and length of travel. These we tackled by travelling four weeks before competition to fully acclimatise. This allowed a good block of quality training to sharpen up for the start of racing. We stayed away from Sydney for the first three weeks to avoid too many distractions. The Olympic village is an amazing place. The best athletes in the world all staying in the same place, effectively everyone having their World Championships at the same time. I found it a very positive and motivating experience. Our competition venue was a 2,000 m lake just like everywhere else I race, apart from the 30,000 spectators, high security, TV cameras and media attention.

Come race day I was ready. I was acclimatised and used to the different atmosphere at the Olympics. I’d watched other people standing on the Olympic podium and that’s where I wanted to be too. The elation and look on their faces was very motivating. When I was in my boat on the water, that was my familiar territory. I was there to do what I’d done many times before and had spent many years training for. The first race went well and then I was drawn in probably the hardest of the three semi-finals, up against the European champion and current Olympic and World Champion. Then it was the final, this was it, my first Olympic final. An Olympic rowing friend, Tom Kay, had said to me that if I didn’t feel I was going to die when I crossed the finish line then I hadn’t done enough. The important thing was to prepare and warm up like I do for any other race. On the start line when the gun went off my gate didn’t drop. For a second I thought that was it, race over. On looking across I was aware that no one else’s had gone down either, a system fault. Time to re-focus, paddle round and get back on the start line. I stuck to my race plan not worrying about where anyone else was. With 50 m to go I was in fifth place, really digging deep now, nothing to lose. I gave it everything and in that last 50 m I passed two people to finish third, Britain’s first ever Olympic medal in sprint kayak racing. I’d raced the best race of my life and came home with a medal.

Eight years later at the Beijing Olympic Games Tim went on to better this result with a Gold medal in K1 1,000 m and a Bronze in the K1 500 m. He was the first British kayaker in slalom or sprint to win an Olympic gold medal.

Biography: Anna Hemmings

Adventuresport: K1 kayak marathon

Anna Hemmings, for many years Britain’s leading female marathon canoeist, six times World and three times European Champion, was told in 2003 by medical experts she might never race again. In 2005, however, she signalled a miraculous return to fitness by regaining her status as the world’s leading marathon canoeist at the Marathon Racing World Championships in Perth, Australia. It is her fourth gold medal in the 13-year history of the event, and her ninth World and European Championship medal, confirming her status as Britain’s most successful ever female canoeist. Anna’s achievements were recognised at the 2005 Sunday Times Sports Woman of the Year Awards, where she won the Champions Award and in 2010 when she was made a MBE. Anna, 35 years old, from Surrey, was diagnosed with chronic fatigue syndrome which had threatened to end her career. However, reverse therapy enabled her to overcome the illness and return to the highest level of marathon performance. After her success at the 2005 World Marathon Championships Anna she went on to take two further World Marathon gold medals (2006 and 2007) and compete at the Beijing 2008 Olympic Games prior to retiring in 2009.

Marathon Racing World Championships 2005

The sweetest victory

We were about 150 m away from the final portage when I took the lead; I arrived at the portage (a portage is where you have to get out of your kayak and run with it for 200 m before re-entering the water for the next lap), I leapt out of my boat, and, totally focused, I ran, ready to embrace the hardest part of the race. Imagine this, you’ve already been racing for an hour-and-three-quarters, you get out of the boat and...
run with it as rapidly as you can for 200 m, the first 170 m being on grass around a bend and then the last 30 m on sand. Just as your legs are getting tired it gets even tougher - the sand completely zaps the energy from your legs! You get back in, your legs have seized up, your arm is tired from carrying the boat, your lungs are bursting and then you have to pull away and paddle as fast as you can. I managed to establish a 100 m lead but I knew that if I relaxed they would catch me. I paddled hard but this was the bit that hurt - the pain was kicking in big time, then I realised that I'd been there before, in a number of races and in a thousand training sessions. I remembered that this is what I trained for – being able to push through the pain barrier in the last 6 km of the World Championships . . .

How do you prepare yourself for that? When you think about marathon canoeing, most people compare it to running a marathon; however it is more akin to cycling. In the same way that cyclists sit on the slipstream and ride along in the peloton; canoeists 'sit on the wash' (or ride the wave that comes off the side of the boat) and race in a pack. The pack leader changes every so often, thus causing the paddlers to jostle for the best positions in the group. By sitting on the wash you can conserve about 30% more energy than the person leading the pack. So my plan is usually to sit on the wash as much possible and conserve as much energy as possible!

The idea of riding waves and racing in groups means that the pace fluctuates; there are numerous sprints, particularly as a group approaches a turn or portage or when the pack leader changes. The start of a marathon race is pretty rapid too. Despite the fact that the race is 18 miles (28.8 km) long, if you want to win, it is essential that you make the front group; otherwise you are playing catch up for the rest of the race. With between 20-30 canoeists on the start line the first 1000 m are fairly frantic as everyone sprints to make the front group and fights to gain the best position. Equally a sprint finish is not uncommon. A successful marathon canoeist, therefore, doesn't only require a high level of endurance; a powerful sprint is also of paramount importance.

In order to prepare my body for this, in a typical training week (during the summer) I would clock up around 50–60 km on the water. This would consist of 10-11 individual training sessions; 8–9 of those would be on the water and 1-2 would be running sessions. I would normally do weight training twice a week. However, in the summer of 2005 I was challenged with a wrist injury which forced me to omit weight training altogether. Although weight training is important for building power and strength, with a number of years of weight training behind me it wasn’t going to be detrimental.

I run a couple of times a week for two reasons. The first being that I believe running is a great way to build general fitness and stamina (crucial for marathon racing). The second reason is because we have to do portages which are usually around 200 m long. Although you don’t have to be an elite runner, the more comfortable you are running through the portage, the less it will take out of you and affect you when you get back on the water.

If during one week I did eight sessions on the water, they would be broken down in the following way:

- 5 × endurance sessions, including:
  - 1 × 10 km time trial or 21 km race
  - 1 × long intervals at core aerobic pace (CAP)
  - 2 × intervals at threshold eg
    - 4 × 4 mins on 1 min rest
    - 3 × 5 mins on 1 min rest
    - 2 × 6 mins on 1 min rest, with 3–4 mins rest between the sets
  - 1 × 4 km time trial followed by long intervals at threshold
  - 1 × speed endurance
  - 2 × speed or speed-strength

A speed-strength session is resistance training; in a similar way that a sprinter on the track might run dragging a tyre or some other form of resistance, a canoeist will tie a bungee around the kayak and place 1-3 tennis balls underneath it to create extra drag and resistance.

In my preparation for the world championships, the only time that I paddled the full race distance was at the European Championships. With

(Source: Mark Lloyd)
so many years of endurance training behind me and the solid base that I built at the beginning of my career, it is not necessary for me to race regularly or train over the race distance. With all this training in place I was finally ready to head to Australia. I flew out to Perth 14 days prior to the race, this gave me sufficient time to overcome the jet-lag, acclimatise and complete the finishing touches of the training programme. The race was on a Saturday and I completed my final training session on Tuesday evening, this left me with three full days to rest and build up my glycogen stores. I consumed a high-carbohydrate diet and plenty of fluids. Race day arrived; I was rested and ready to endure the toughest part of the race...

I raced through the pain barrier and finally I entered the home straight, there was no chance of anyone catching me now, the challenge was maintaining my focus, because all of a sudden I was distracted by the thought of another world title and that made me emotional! This was not the time to be getting emotional, I hadn’t won yet! I needed to focus!

I did and finally after 2 hours 16 minutes of racing I crossed the line in first place – world title number four! I threw my arms in the air and gave a yelp of joy and relief. It was the sweetest victory yet. (If you would like to see video footage of me in action please go to my website: www.annahemmings.com.)

The use of the aerobic-anaerobic transition in determining endurance training intensity by Kindermann et al. (1979)

In the 1970s, many researchers held differing views on the best criteria for the selection of the most appropriate training intensity for endurance athletes. The anaerobic threshold was suggested, in 1978, to be a suitable guideline for the determination of exercise training intensity. Kindermann and colleagues aimed to build on this suggestion by establishing an exercise intensity that could be maintained for prolonged periods of time, yet adequate for endurance training.

Kindermann and colleagues tested seven national-level cross-country skiers. Initially they completed an incremental treadmill test to volitional exhaustion. The treadmill incline was maintained at 5% throughout the test whereas velocity was increased from a starting level of 8 km·h⁻¹ by 2 km·h⁻¹ every three minutes. Exercise was stopped for 20 s after each three minute period to allow the testers to obtain a blood sample from the ear lobe for the analysis of arterialised blood lactate concentration. In addition, a final blood sample was taken three minutes following volitional exhaustion for the determination of maximal post-exercise lactate concentration. With the use of an Oxycon gas analysis system, V̇O₂ and V̇E were recorded and heart rate was monitored with electrocardiograms. The aerobic-anaerobic threshold was defined in this paper as a blood lactate concentration of 4 mM. Two additional treadmill tests were then completed: (1) 30 min running at the aerobic-anaerobic threshold HR (treadmill speed was continually reduced to maintain HR at this level), and (2) 30 min running at the aerobic-anaerobic threshold velocity. Heart rate was recorded every minute while V̇O₂ was continuously monitored, and blood samples were taken from the ear lobe at rest and every 5 min during the 30-min run.

The primary finding of this paper was that running at an intensity sufficient to elevate blood lactate concentration to around 4 mM (as determined with an incremental treadmill test) can be performed by the majority for 45–60 min, with a few able to continue for even longer. This exercise elevated HR to an average of 170 beats·min⁻¹, although some individuals experienced HRs in excess of 180 beats·min⁻¹. Kindermann and colleagues suggested that training at an intensity around the ‘aerobic-anaerobic’ threshold (~4 mM blood lactate) would lead to adaptations in both the cardiovascular system and the muscle cells. It has since been determined that blood lactate measures of the aerobic-anaerobic transition are strong predictors of endurance performance in 30–60-min races, hence its improvement with endurance training is critical for optimal performance.