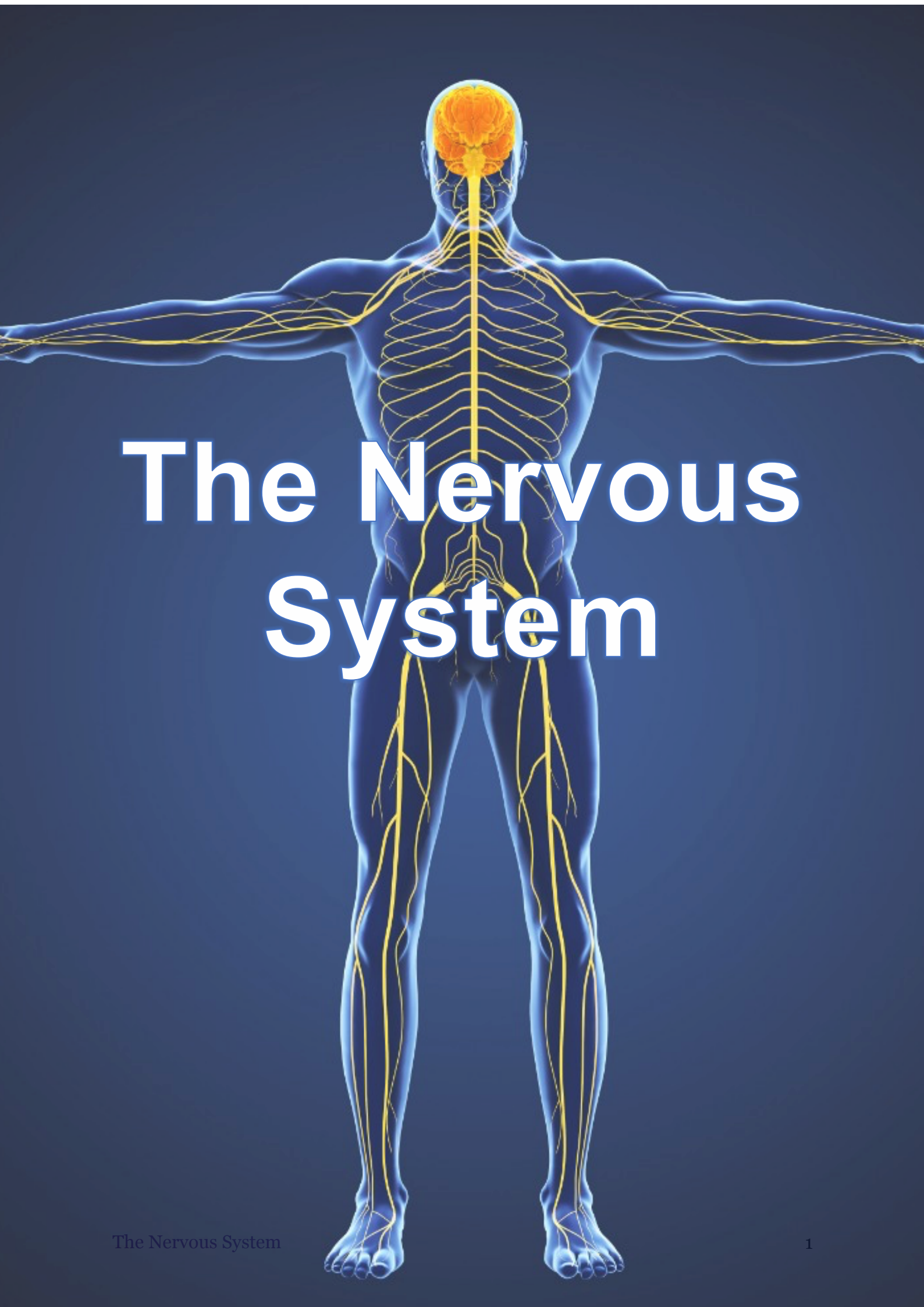

The Nervous System

Table of Contents

The human brain

Our brain contains billions of neurons that communicate with nearly 10,000 others within the body. The brain and subsequently the neurons control all aspects of the human body (motor performance to psychological factors). It is beyond the scope of this e-book to completely discuss the brain; some essential areas, however, need to be discussed to help develop an understanding of adaptations to training.

Essentially, the brain stem (consisting of the medulla oblongata, midbrain, and pons) contains the neural centre. For example, the brain stem regulates heart rate, the force of contractions, blood pressure, breathing, vision and consciousness. The thalamus, hypothalamus, and pineal body or epithalamus (collectively termed diencephalon) is the control centre for sleep and relay mechanism of the brain (pineal body and thalamus respectively). The hypothalamus is the main link between the nervous and endocrine systems because it is an endocrine gland under neural control.

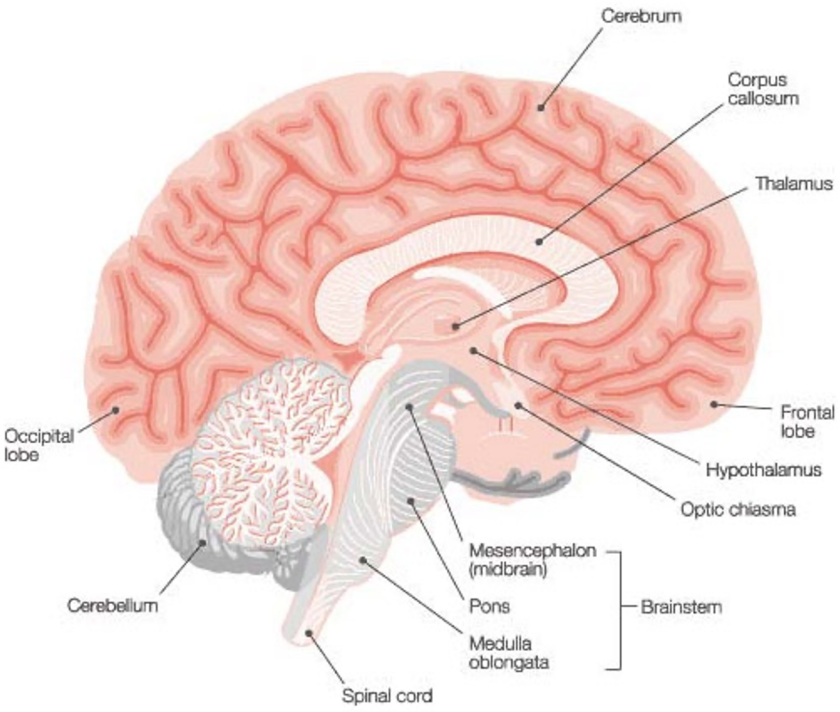

Figure 1. Various areas of the human brain.

The hypothalamus releases numerous hormones that inhibit or release hormones from the anterior pituitary glands controlling most essential functions of the body (e.g., homeostasis, autonomic control, body temperature, emotions). The largest part of the brain called the cerebrum contains 75% of the neurons within the nervous system and is in the cerebral cortex. Critical areas include the premotor cortex (where a voluntary muscle contraction begins), the primary motor cortex (where voluntary muscle contraction is controlled) and the primary sensory area (where sensory information is integrated). The basal ganglia are involved with control and planning of muscular function, posture, and regulating undesirable movements. Finally, the cerebellum integrates sensory information and coordinates muscle activity.

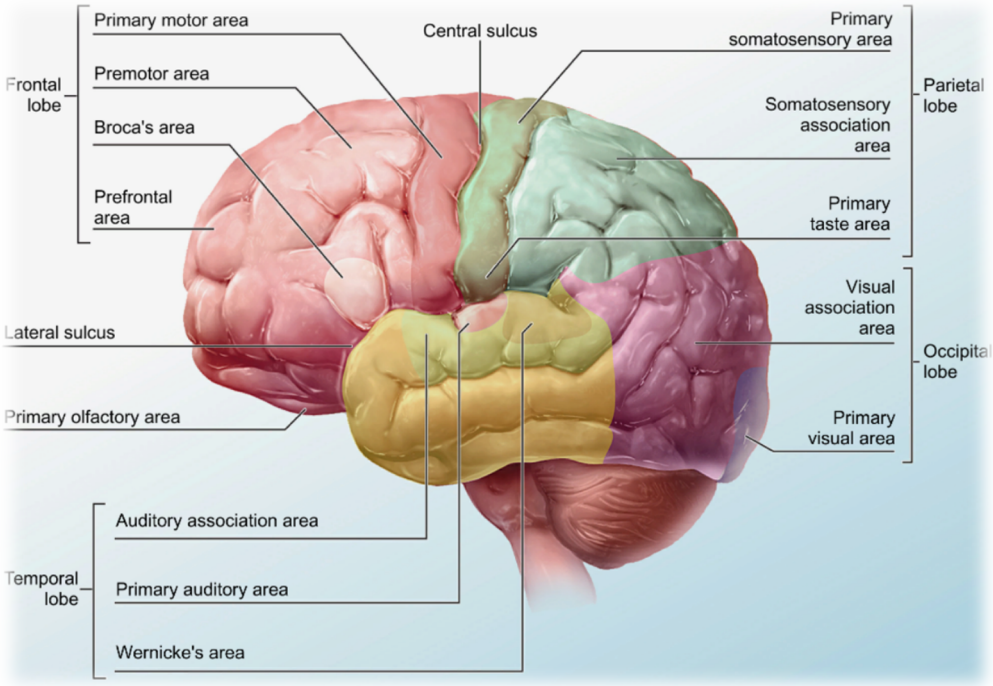

Figure 2. Higher brain centres including the motor cortex.

It is important to understand that the ability to increase neural output is initiated in the higher brain centres such as the motor cortex (Figure 2). This is especially true when individuals attempt to produce high levels of force and power. Evidence has suggested that neural activity increases in the primary motor cortex as force increases (Dettmers, et al., 1996). Motor learning results in the functional organisation of the cerebral cortex. This has been demonstrated through visualisation training with untrained subjects demonstrating significant strength increases

(visualising lifting weights without lifting them). Therefore, cerebral adaptations are essential for developing coordination, motor learning, skill acquisition, strength, power, and speed.

Descending corticospinal tracts

The descending corticospinal tracts are a collection of axons linking the cerebral cortex to the spinal cord. The motor pathway is characterised by neurons in the brain (primarily in the motor areas) forming synapses with other nerves that ultimately proceed down the spinal cord to the anterior root of exit for innervation of skeletal muscle (**Figure 3**).

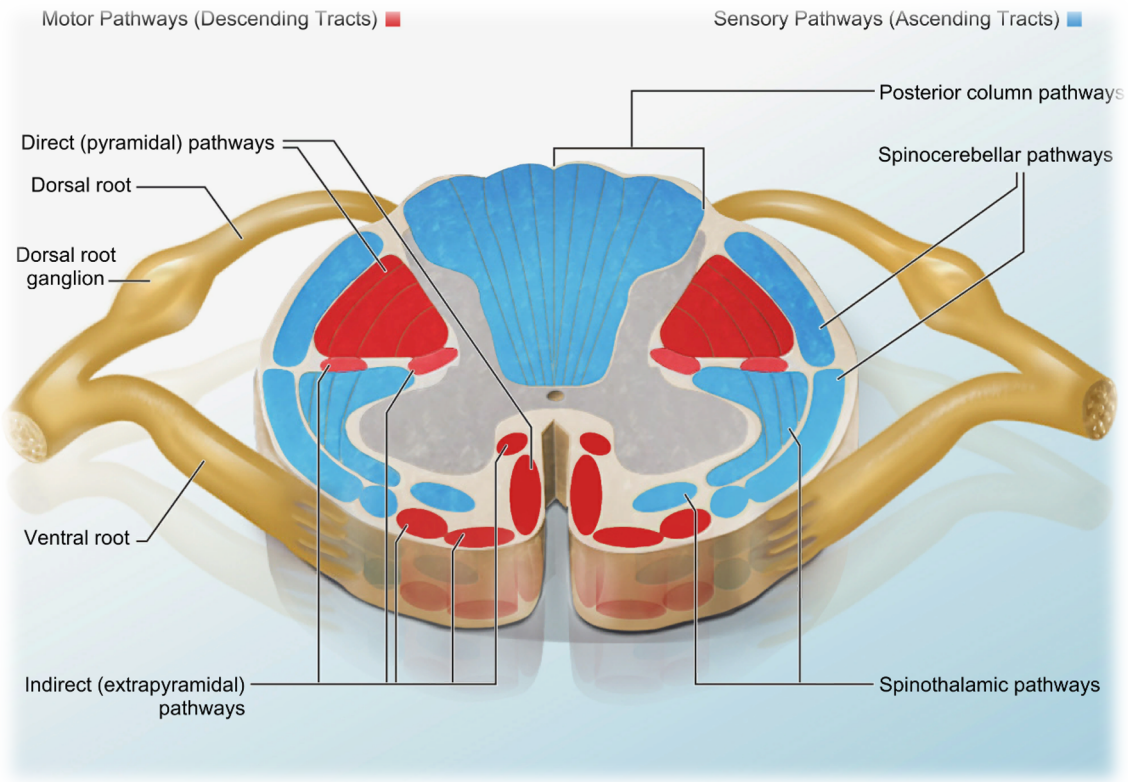

Figure 3. Corticospinal tracts.

A substantial proportion of potential neural changes take place in the spinal cord along the descending corticospinal tracts. Untrained individuals typically display restricted ability to maximally recruit all their muscle fibres. A study by Adams et al., (1993) demonstrated that only 71% of muscle mass was activated during maximal effort in untrained individuals. Carrol and colleagues (2002) reported that a limitation

in subjects central drive reduces strength and power with much of the inhibition originating from the descending corticospinal tracts. However, Pucci et al., (2006) reported that training can greatly reduce this deficit, thereby demonstrating a greater potential to recruit a larger per cent of muscle mass with training.

The nervous system

The nervous system is critical for regulating acute exercise performance and ensuing training adaptations. The main lines of communication between the brain and the muscular system are via these nerve networks, ensuring that signals travel to the appropriate locations within the body (**Figure 4**). From an exercise training perspective, the magnitude of these signals is essential in regulating the final strength or power output. This neural drive is critical to the individual motivated to maximise athletic performance. The neural drive is believed to occur in agonist muscle recruitment, firing frequency, and the pattern of discharge during high-intensity muscular contractions. It is thought that a reduction in the inhibitory mechanisms also occurs, although limited understanding is available on how these mechanisms co-exist. However, it is apparent that the neural adaptation is multifaceted and may precede changes in the muscle.

Functional organisation of the nervous system

The nervous system serves predominately as the foremost control mechanism of the body (excluding the endocrine system). This system receives various sensory information in the form of pressure, temperature, joint position, muscle length and pain. It can process, integrate, and respond to every tissue, gland, and organ controlling all outputs or responses. Additionally, the nervous system also controls our emotions, personality, and other cerebral (brain) functions. The nervous is comprised of two major branches the central and peripheral nervous system (Figure 4 and 5). The central nervous system (CNS) consists of the brain and the spinal cord. The peripheral nervous system is separated with two segments namely the motor and sensory divisions. There are 31 pairs of spinal nerves that exist and exit on the sensory (posterior) and motor (anterior) roots of the spinal cord.

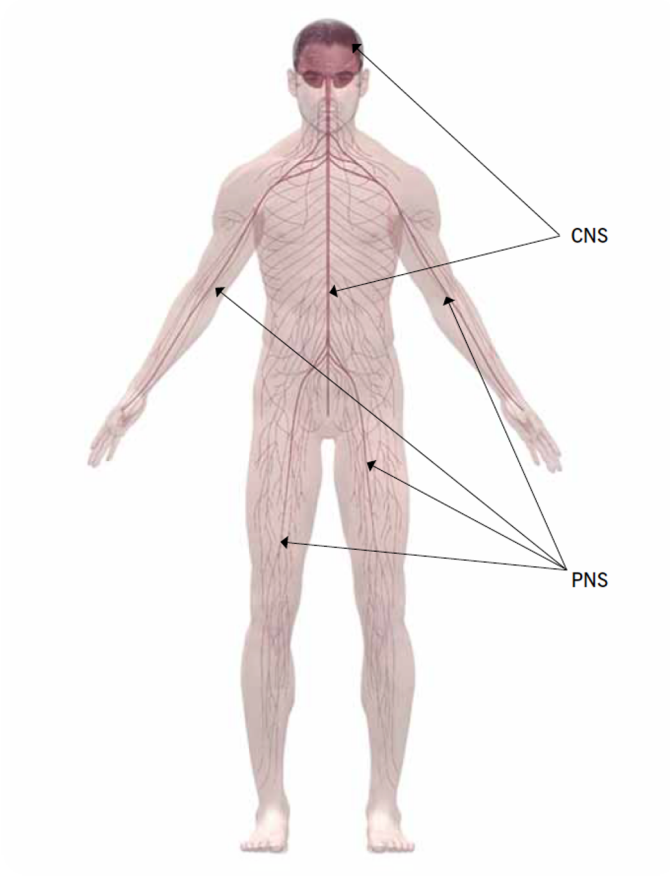

Figure 4. Central nervous system (CNS) and peripheral nervous system (PNS).

The sensory nervous system detects varies stimuli and relays this information afferently to the CNS. The motor nervous system consists of two divisions (i) the somatic and the autonomic nervous system (ANS) (**Figure 5**). The somatic system conveys information from the CNS efferently (away from the CNS) to the muscles leading to muscle contractions. The ANS consists of nerves relaying efferent information to cardiac, tissues, glands, and muscles. The ANS comprises of the sympathetic and parasympathetic nervous system, both of which are necessary for preparing the body for the physical stress of exercise and returning the body to pre-exercise state. It has been suggested that physical training and exercise may produce adaptations along the neuromuscular chain, instigating in the higher brain centres and cascading down to individual muscle fibres. Aerobic training imposes specific neural demands although the rate coding or pattern of neural activation appears less complex than high-intensity anaerobic training stimuli, where high levels of strength and power are required.

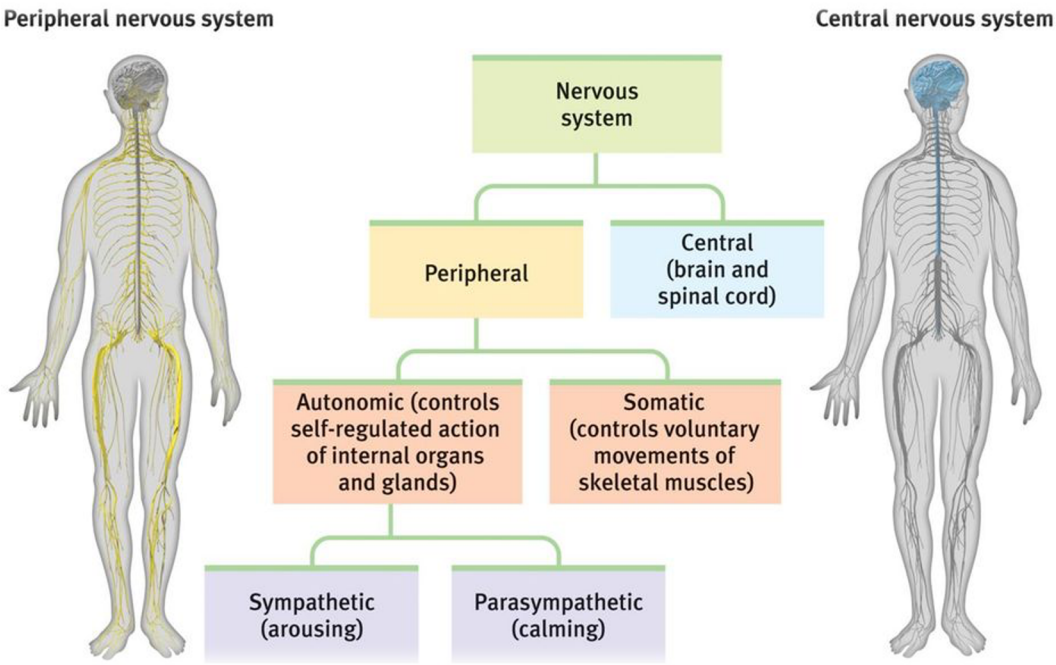

Figure 5. Divisions of the nervous system.

Nerve cells

The nervous tissues comprise of supporting cells and neurons (**Figure 6**). The supporting cells are essential, and they ensure constancy throughout the CNS, with neurons (nerve cells) communicate with other nerves and tissues. Motor neurons tend to be multipolar whereas sensory neurons tend to be unipolar. The key structures of the motor neuron include dendrites which receive input from other nerve cells. The cell body contains the organelles which are responsible for energy metabolism, protein synthesis, and transport. These cell bodies play a key role in integrating the stimuli from other neurons within the CNS and determine how much stimuli will transfer to the muscles. Other components of the neuron comprise of the axons which are long processes that are responsible for communicating with target tissues. The axon hillock is the area where the action potential is initiated once the critical threshold is reached. There is fatty tissue which is wrapped around the axon called the myelin sheath, which significantly increases the speed of signal transmissions.

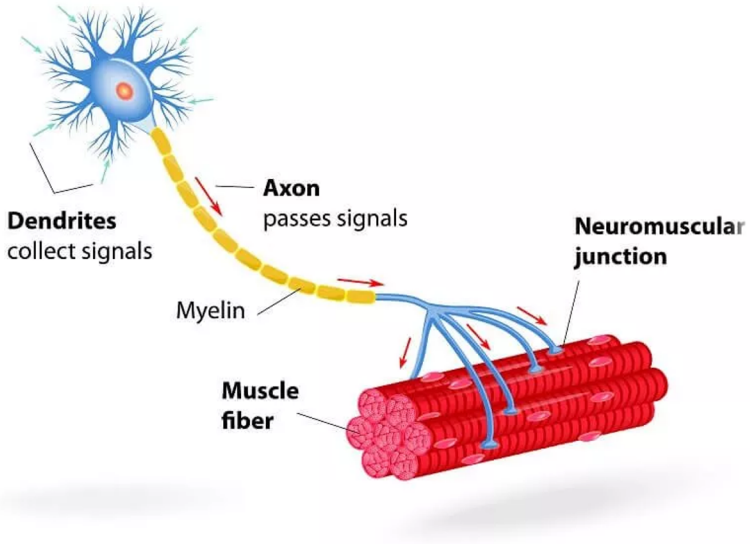

Figure 6. Key structures of the motor-neuron such as the dendrites, cell body, axon.

Neural communication

Communication occurs between nerves and tissues through the generation of an electrical signal (current) called the action potential (AP). The AP consists of three key actions: (i) integration, (ii) propagation, and (iii) neurotransmitter release. Integration ensues within the cell body and determines whether the Ap will be transmitted to the target tissue (**Figure 7**). The cell body integrates the charges from other neurons and if the threshold voltage is reached then the AP will travel in an all-or-none mode to the end of the nerve terminal. Propagation is then brought about by ion movement (sodium and potassium) down the axon at the nodes of Ranvier through a process called saltatory conduction.

The electrical current is quickly driven down the axon to the terminal with the myelin sheath further accelerating this process. At the nerve terminal, neurotransmitters are released allowing communication to occur with the target tissue. This entire process occurs rapidly and enables several APs to be conducted in under a second. A further detailed breakdown of the processes is on the next few pages.

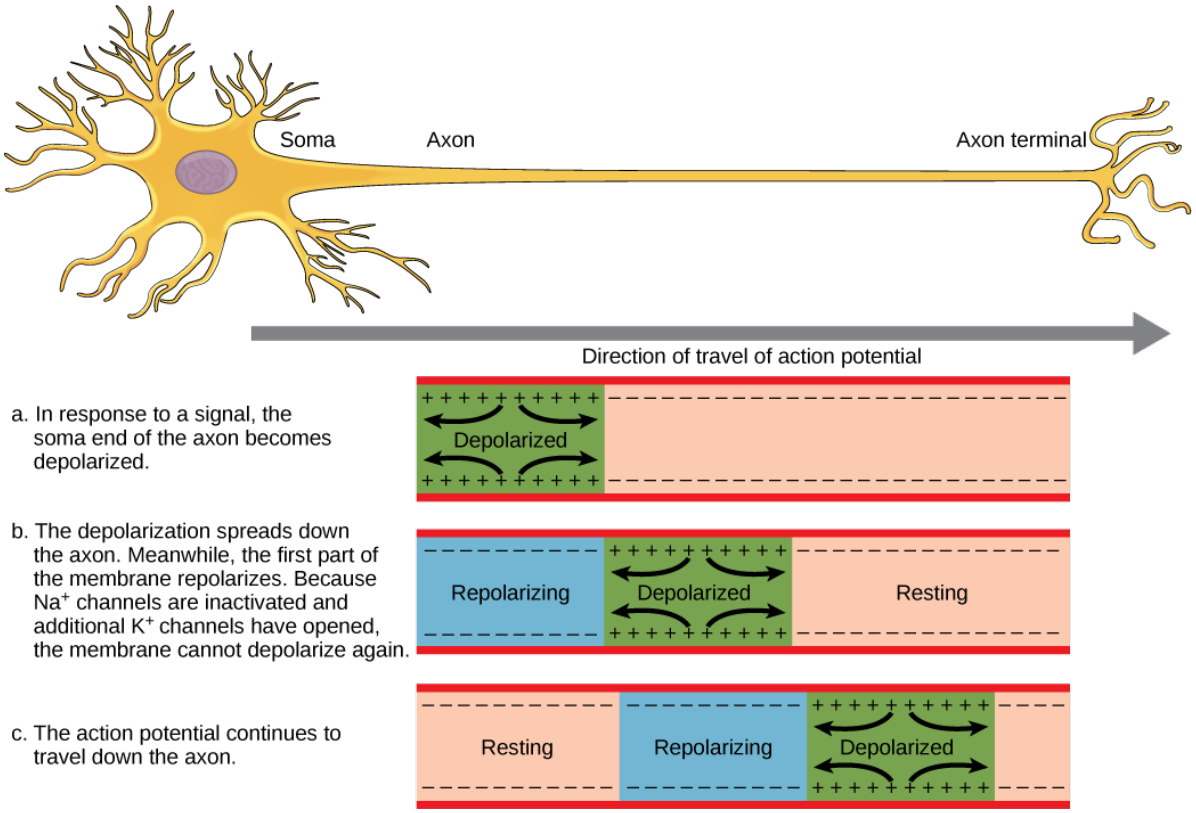

Figure 7. Communication occurring between nerves and tissues through the generation of an action potential.

Cell membrane activity

The main roles of the nervous system are sensation, integration, and response, depending on the functions of the underlying neurons underlying these pathways. It is important for sports coaches and trainers to understand how neurons communicate and how action potential occurs if specific training exercises or practices are developed.

The cells of the human body use charged ion particles to generate a charge across the cell membrane. For muscles to contract, excitation-coupling requires input from a neuron. The cell membrane, therefore, is responsible for the regulation and movement between ions from the extracellular fluid and the cytosol. The cell membrane, therefore, is responsible for the regulation of what crosses the membrane via a phospholipid bilayer. This aids in the diffusion (passage) of only substances that pass through the hydrophobic core, with charged particles unable to pass through without support (**Figure 8**). Channel proteins (transmembrane) allow this to ensue,

with numerous passive channels and active transport pumps generating a transmembrane potential and an action potential (AP). Carrier proteins (sodium/potassium pumps) help to regulate the ion concentration on both sides by moving sodium ions (Na+) out of the cell and potassium ions (K+) into the cell.

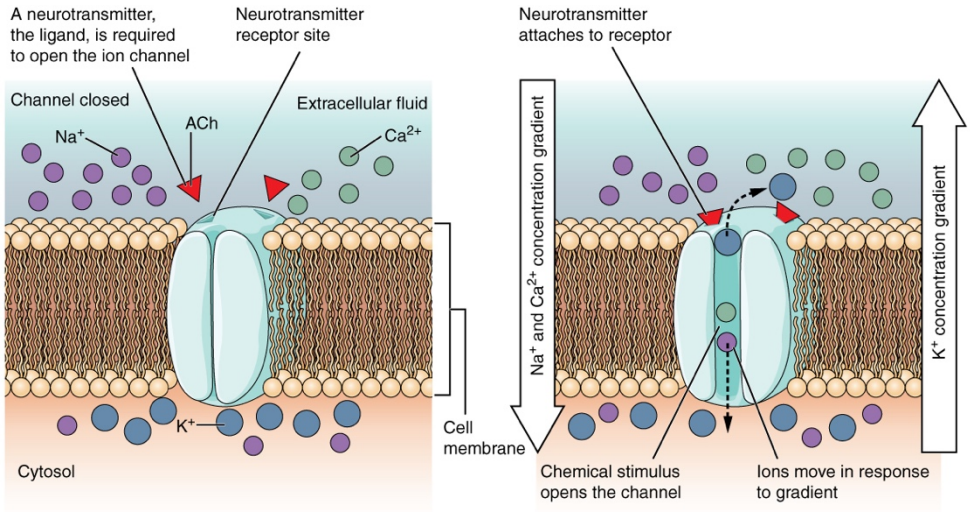

Figure 8. The Cell membrane.

For the sodium/potassium pumps to function, they require energy in the form of adenosine triphosphate (ATP) (also referred to as an ATPase). The Na+ concentration is higher outside than inside of the cell and the concentration of K+ is higher inside than outside. As a result, this pump is regulating ions against a concentration gradient for sodium and potassium which is why ATP energy is required. Ion channels are openings that allow charged particles to cross the membrane in response to a current concentration gradient. Proteins can span the cell membrane (including the hydrophobic core) and can interact with ions charge because of the various properties of the amino acids located within the regions of the protein channels. For example, hydrophobic amino acids are in the areas that are interconnected to the hydrocarbon tails of the phospholipids.

Hydrophilic amino acids are exposed to the fluid environments of the extracellular fluid and cytosol. Hydrophilic amino acids are exposed to extracellular fluid and cytosol. Furthermore, the ions will interact with hydrophilic amino acids, which will be selective for the charge of the ion. Channels for positive ions (cations) will have negatively charged side chains in the opening (pore). Whereas negative ions

(anions) will have positively side chains in the pore. This is called electrochemical exclusion, meaning that the channel opening is charge specific.

The diameter of the pore has a specific impact on the ion channels. The is because the distance between the amino acids is specific for the pore diametre when it detaches from the water module surrounding it. Due to the size of the surrounding water molecules, the larger pores are not suitable for smaller ions because the water molecules will interact (due to hydrogen bonds) more willingly than with the amino acid chains (termed size exclusion).

Ion channels do not always permit diffusion of ions across the membrane. Some channels are gated (ligand-gated) and are primarily found in the cells of the muscle tissue, epithelial, and connective tissues. A ligand-gated channel opens because a signalling molecule binds to the extracellular region of the channel. This type of channel is sometimes known as an ionotropic receptor because when the ligand (sometimes termed a neurotransmitter) in the nervous system, binds to the protein, ions cross the membrane changing its charge.

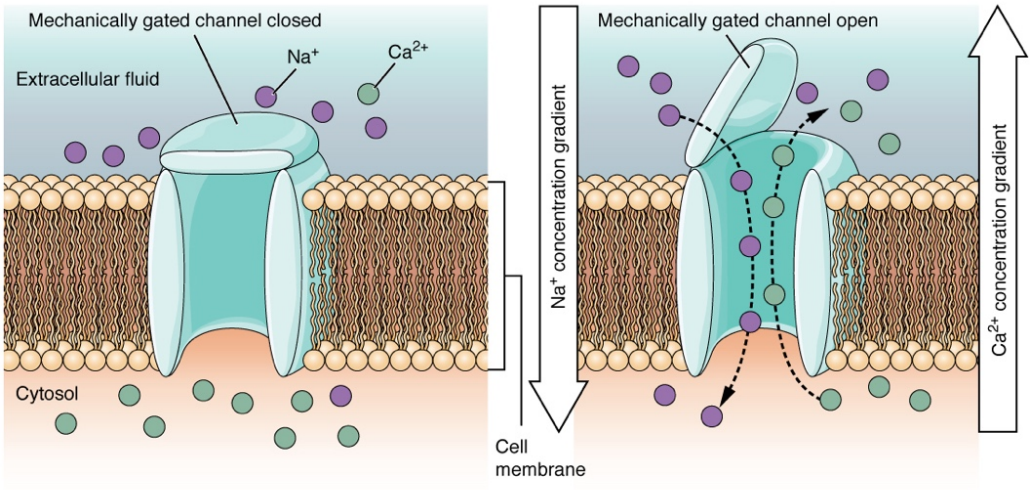

Figure 9. Ligand-Gated Channels.

Types of gated channels

A mechanically gated channel (**Figure 10**) opens because of a physical alteration of the cell membrane. Channels coupled with the sense of touch (somatosensorial) are

mechanically gated. For example, as pressure is applied to the skin, these channels open and permit ions to enter the cell. Similar to this type of channel would be the channel that opens due to temperature changes, as in testing the water in the shower.

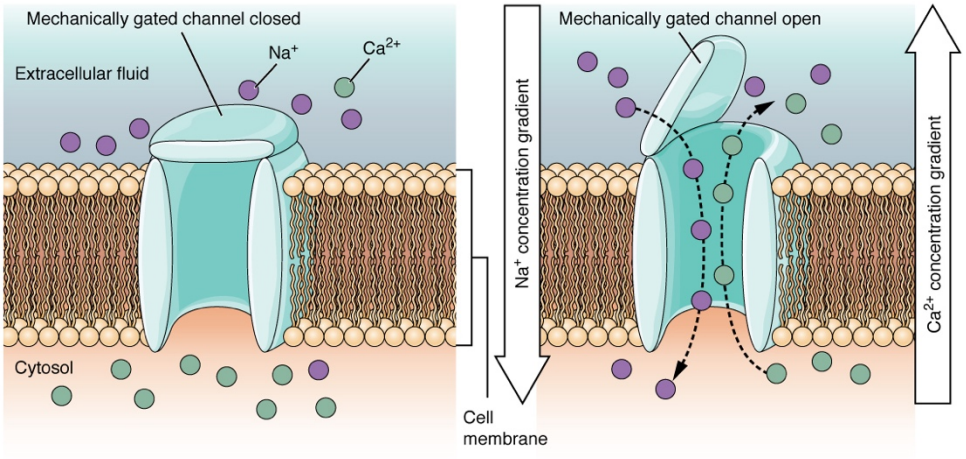

Figure 10. Mechanically gated channels.

A voltage-gated channel (Figure 11) is a channel that responds to changes in the electrical properties of the membrane in which it is embedded. Typically, the inner portion of the membrane is at a negative voltage. When that voltage becomes less negative, the channel begins to allow ions to cross the membrane.

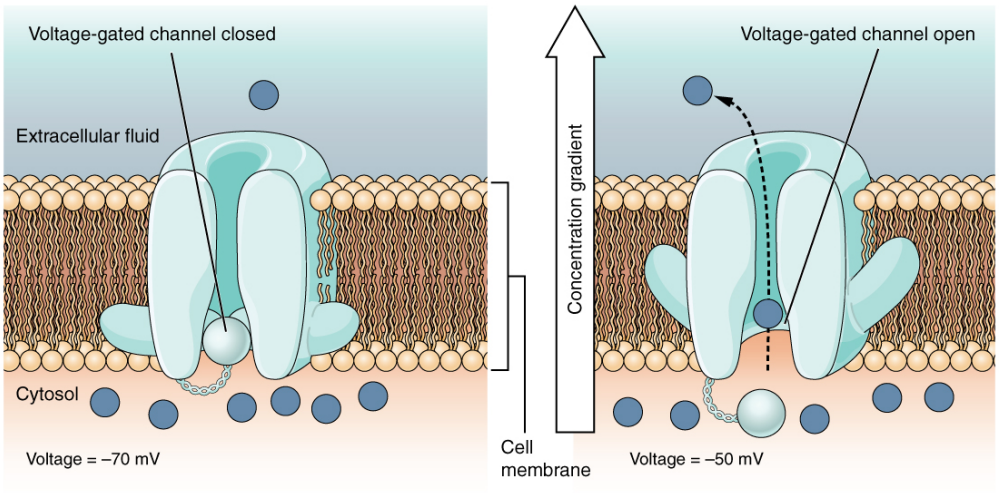

Figure 11. Voltage-gated channels.

A leakage channel (**Figure 12**) is randomly gated, meaning that it opens and closes at random, thus the reference to leaking. There is no actual event that opens the

channel; instead, it has an intrinsic rate of switching between the open and closed states. Leakage channels contribute to the resting transmembrane voltage of the excitable membrane.

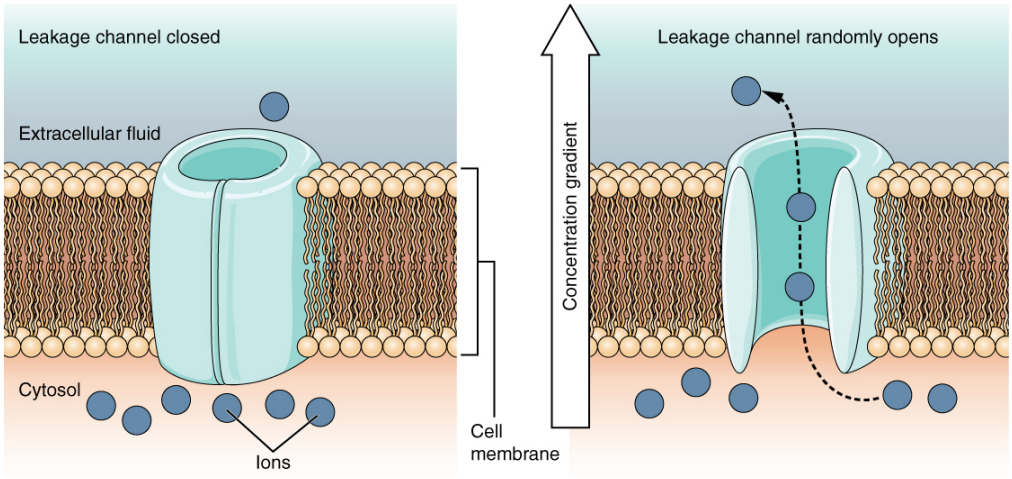

Figure 12. Leakage channels.

The membrane potential

The cell membrane electrical state can have some variants of (termed membrane potential). The membrane potential is the interference of charge across the cell membrane (measured in millivolts [mV]). Normally comparisons are made inside of the cell relative to the outside. Therefore, the membrane potential is a value compared to the charge on the intracellular side of the membrane based on the outside is zero. The concentration of ions in extracellular and intracellular fluids is mostly stable, with a net neutral charge. However, a small variation in charge occurs at the membrane surface, both internally and externally. It is this difference that has all the power in neurons to produce electrical signals, including action potentials (**Figure 13**).

However, before the electrical signals can be described the resting membrane state must be clarified. When the cell is at rest, and the ion channels are closed the ions are distributed across the membrane in a certain fashion. The concentration of Na+ outside the cell is considerably greater (10 times greater) than the concentration inside. Moreover, the concentration of K+ inside the cell is greater than outside. The cytosol contains a high concentration of anions, in the form of phosphate ions and negatively charged proteins. With the ions distributed across the membrane at these

concentrations, the variance in charge is measured at -70 mV (the value commonly defined as the resting membrane potential). This voltage would essentially be lower except for the contributions of some important proteins in the membrane. Leakage channels allow Na+ to gradually move into the cell or K+ to slowly move out, and the Na+/K+ pump restores them.

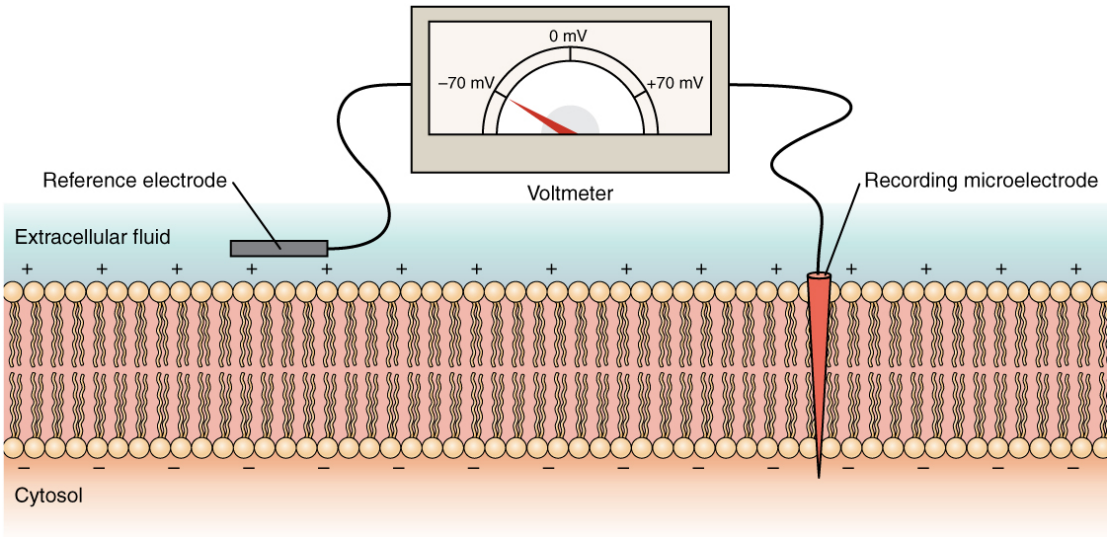

Figure 13. Measuring charge across a membrane with a voltmetre.

Action Potential

Resting membrane potential depicts the steady state of the cell, which is equalised by ion leakage and ion pumping. Importantly, without any outside stimulus, it will not change. To initiate an electrical signal, the membrane potential must change. This commences with a channel opening for Na+ in the membrane because the Na+ concentration is greater outside the cell than inside. Ion's hurry into the cell due to the concentration gradient. Due to sodium being a positively charged ion, it will immediately alter the relative voltage inside the cell comparative to directly outside. The resting potential is the state of the membrane at a voltage of -70 mV, so the sodium cation entering the cell will initiate a reduction in mV (termed as depolarisation).

Due to the strength of Na+, it will continue to enter the cell even after the membrane potential has become zero, resulting in the voltage directly around the pore becomes positive. The electrical gradient also contributes to these as negative proteins

below the membrane attract the sodium ion. As a result, the membrane potential will reach +30 mV by the time sodium has entered the cell. As the membrane potential reaches +30 mV, other voltage-gated channels are opening in the membrane. These channels are specific for the potassium ion. A concentration gradient also acts on K+. As K+ starts to exit the cell the membrane potential begins to move back toward its resting voltage (termed repolarization).

Repolarization returns the membrane potential to the -70-mV value that denotes the resting potential but overshoots the -70-mV value. Potassium ions reach equilibrium when the membrane voltage is below -70 mV, so a period of hyperpolarization occurs while the K+ channels are open. Those K+ channels are slightly delayed in closing, accounting for this short overshoot. The change in the membrane voltage from -70 mV at rest to +30 mV at the end of depolarization is a 100-mV change. (Also written as 0.1-V change).

The membrane potential will stay at the resting voltage until something changes. The description above just says that a Na+ channel opens. However, there are several different types of channels that allow Na+ to cross the membrane. A ligand-gated Na+ channel will open when a neurotransmitter binds to it and a mechanically gated Na+ channel will open when a physical stimulus affects a sensory receptor. Whether it is a neurotransmitter binding to its receptor protein or a sensory stimulus activating a sensory receptor cell, some stimulus initiates the process. Sodium starts to enter the cell and the membrane becomes less negative.

Another channel that is an essential part of depolarisation in the AP is the voltage-gated Na+ channel. The channels that start depolarizing the membrane because of a stimulus help the cell to depolarise from -70 mV to -55 mV. Once the membrane reaches that voltage, the voltage-gated Na+ channels open. This is what is known as the threshold. Any depolarization that does not change the membrane potential to -55 mV or higher will not reach the threshold and thus will not result in an AP.

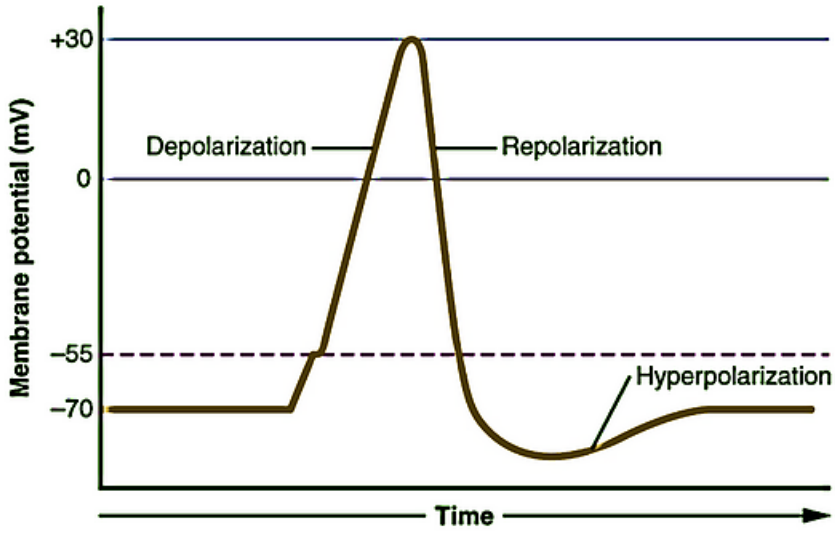

Figure 14. Graph of Action Potential.

A stronger stimulus, which might depolarise the membrane past the threshold, will not create a greater AP. Action potentials are “all or none”, either the membrane reaches the threshold, and everything occurs, or the membrane does not reach the threshold and nothing else happens. All APs peak at the same voltage (+30 mV), so one action potential is not bigger than another. Stronger stimuli will initiate multiple AP more rapidly, but the individual signals are not bigger.

As previously described, the depolarisation and repolarisation of an AP are dependent on the voltage-gated Na+ channel and the voltage-gated K+ channel. The voltage-gated Na+ channel has two gates. One is the activation gate, which opens when the membrane potential crosses -55 mV. The other gate is the inactivation gate, which closes after a specific period. When a cell is at rest, the activation gate is closed, and the inactivation gate is open. However, when the threshold is reached, the activation gate opens, allowing Na+ to rapidly enter the cell. Timed with the peak of depolarization, the inactivation gate closes. During repolarization, no more sodium can enter the cell. When the membrane potential passes -55 mV, the activation gate shuts. After that, the inactivation gate re-opens, making the channel ready to start the entire process again.

The voltage-gated K+ channel has one gate, which is sensitive to a membrane voltage of -50 mV. However, it does not open as rapidly as the voltage-gated Na+

channel. It takes only just under a millisecond for the channel to open once that voltage has been reached. This timing coincides precisely when the Na+ flow peaks, so voltage-gated K+ channels open just as the voltage-gated Na+ channels are being inactivated. As the membrane potential repolarises and the voltage passes -50 mV, the channel closes. Potassium continues to exit the cell and the membrane potential becomes more negative, resulting in the hyperpolarising overshoot. Then the channel closes again, and the membrane can return to the resting potential because of the ongoing activity of the non-gated channels and the Na+/K+ pump. All these processes take place within 2 milliseconds (**Figure 15**).

While an AP is in progress, another one cannot be initiated, this is denoted as the refractory period. There are two phases of the refractory period: the absolute refractory period and the relative refractory period. During the absolute phase, another AP will not start, this is because of the inactivation gate of the voltage-gated Na+ channel. Once that channel is at its resting state (less than -55 mV), a new action potential may be started only if a stronger stimulus other than the one that initiated the current AP. This is due to the flow of K+ out of the cell. Because that ion is rapidly exiting, any Na+ that attempt to enter will not depolarise the cell but will only keep the cell from hyperpolarizing.

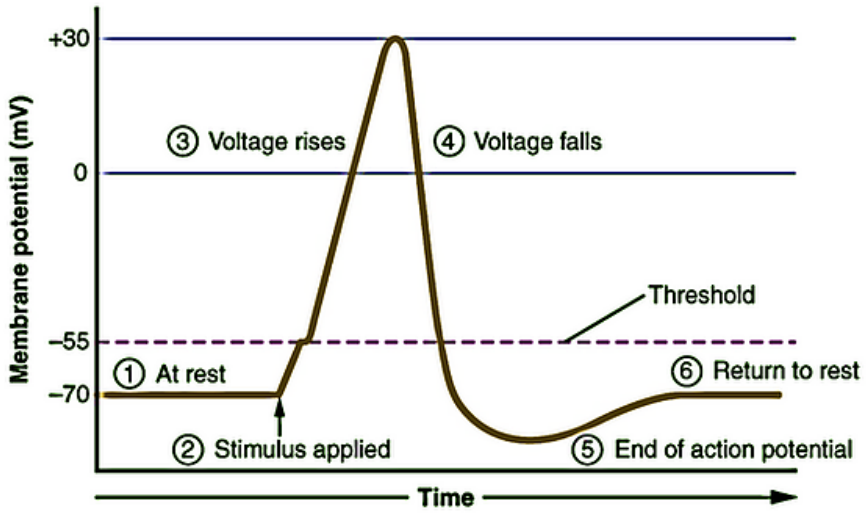

Figure 15. Stages of an Action Potential.

Propagation of the action potential

The AP is initiated at the beginning of the axon (termed the initial segment). There is a high density of voltage-gated Na+ channels so that depolarization can occur here. Progressing down the length of the axon, the AP is propagated because more voltage-gated Na+ channels are opened as the depolarization extends. This dispersal occurs because Na+ enters through the channel and travels along the inside of the cell membrane. As the Na+ flows its positive charge depolarizes more of the cell membrane. As the depolarization spreads, new voltage-gated Na+ channels open and more ions enter the cell, spreading the depolarization farther.

Since the voltage-gated Na+ channels are inactivated at the peak of the depolarization, they cannot be opened again for a brief time (the absolute refractory period). The AP must propagate toward the axon terminals; as a result, the polarity of the neuron is maintained. Propagation applies to unmyelinated axons. When myelination is present, the AP propagates differently. Sodium ions that enter the cell at the initial segment start to extend along the length of the axon segment, but there is no voltage-gated Na+ channels until the first node of Ranvier. The distance between nodes is the optimum distance to keep the membrane still depolarized above the threshold at the next node. As Na+ spreads along the inside of the membrane of the axon segment, the charge starts to dissipate. If the node were any further down the axon, the depolarization would have dropped off too much for the voltage-gated Na+ channels to be activated at the next node of Ranvier. If the nodes were any closer together, the speed of propagation would be longer.

Propagation along an unmyelinated axon (termed as continuous conduction) travels along the length of a myelinated axon (termed as saltatory conduction). Continuous conduction is slow because there is always voltage-gated Na+ channels opening, and more Na+ entering the cell. Saltatory conduction is faster because the AP 'jumps' from one node to the next (saltare = "to leap"), and the new influx of Na+ renews the depolarized membrane. Along with the myelination of the axon, the diameter of the axon can influence the speed of conduction.

Potassium concentration

Glial cells, especially astrocytes, are accountable for maintaining the chemical environment of the CNS tissue. The concentrations of ions in the extracellular fluid are the basis for how the membrane potential is established and changes in electrochemical signalling. If the balance of ions is upset, severe outcomes are possible. Normally the concentration of K+ is higher inside the neuron than outside. After the repolarizing phase of the action potential, K+ leakage channels and the Na+/K+ pump ensure that the ions return to their original locations. Following a stroke or other ischemic event, extracellular K+ levels are elevated. The astrocytes in the area are equipped to clear excess K+ to aid the pump. But when the level is profoundly out of balance, the effects can be permanent. Astrocytes can become reactive in cases such as these, which impairs their ability to maintain the local chemical environment. The glial cells enlarge and their processes swell. They lose their K+ buffering ability and the function of the pump is affected or even reversed.

Synaptic Transmission

Synapses link nerve cells to other nerve cells and sensory and effector cells). Electrical synapses are direct, ion-conducting cell-to-cell junctions through channels in the region of gap junctions. They are accountable for the conduction of impulses between neighbouring smooth or cardiac muscle fibres and ensure communication between neighbouring epithelial or glial cells.

Chemical synapses use (neuro) transmitters for the conduction of information and provide not only 1-to-1 networks but also assist as 'switching elements' for the nervous system. They can enable or inhibit the neuronal transmission of information or process them with other neuronal input. At the chemical synapse, the onset of an AP in the axon initiates the release of the transmitter from the presynaptic axon terminals. The transmitter then diffuses across the narrow synaptic cleft to bind post-synaptically to receptors in the subsynaptic membrane of a neuron or a glandular or muscle cell. Depending on the type of transmitter and receptor involved, the effect on the postsynaptic membrane may either be excitatory or inhibitory.

Transmitters are released by controlled exocytosis of synaptic vesicles. Each vesicle contains a certain number of neurotransmitters. In the motor endplate, approximately 7000 molecules of acetylcholine (ACh) are released. Some of the vesicles are already anchored and ready to exocytose their contents. An inbound AP functions as the signal for transmitter release. The higher the AP frequency in the axon the more vesicles releases their contents. An AP increases the open probability of voltage gated Ca2+ channels in the presynaptic membrane, thereby leading to an increase in the cytosolic Ca2+ concentration. Extracellular Mg2+ inhibits this process. Ca2+ binds to synaptotagmin, which initiates the interaction of syntaxin and SNAP-25 on the presynaptic membrane with synaptobrevin on the vesicle membrane, thereby triggering exocytosis of already anchored vesicles. Equally, Ca2+ activates calcium- calmodulin-dependent protein kinase-II (CaM-kinase-II), which activates the enzyme synapsin at the presynaptic terminal. This results in vesicles dock again on the active zone.

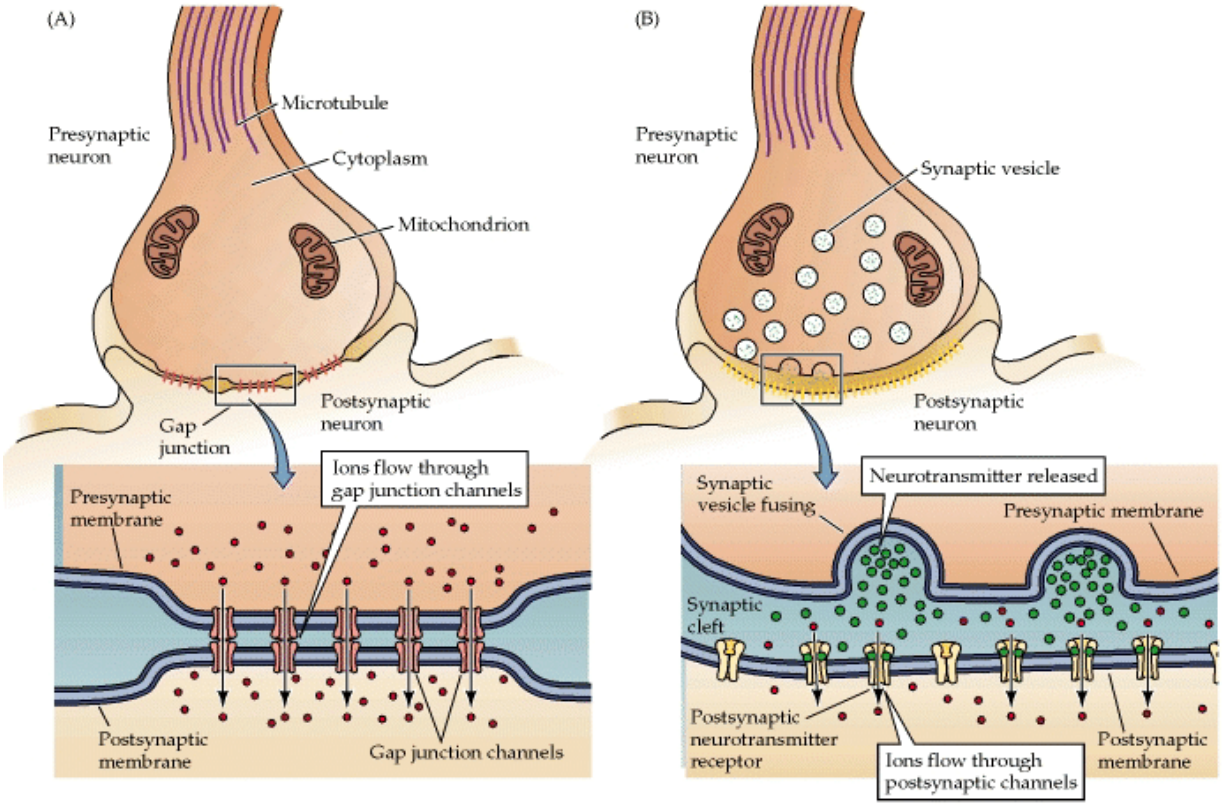

Figure 16. Chemical synapses and electrical gap junctions. In the electrical gap junctions (left), voltage is transferred via touching membranes and signals may pass in both directions. In chemical synapses (right), signals are transferred through ion channels from the pre-to post-synaptic neuron.

Synaptic Potentiation

If an AP reaches the presynaptic terminal immediately after another AP, the cytosolic Ca2+ concentration will not decrease to the resting value, and residual Ca2+ will accrue. As a result, the new rise in [Ca2+] i forms on the former one. [Ca2+] i increases to a higher level after the second stimulus than after the first and releases more transmitters. Consequently, the first stimulus assists in the response to the second stimulus. Muscle strength increases at high stimulus frequencies for similar reasons. Among the many substances that act as excitatory transmitters are acetylcholine (ACh) and glutamate (Glu). They are frequently released together with co-transmitters which modulate the transmission of a stimulus. If the transmitter's receptor is an ion channel itself the channels open more often and allow a larger number of cations to enter and leave the cell. Other, metabotropic receptors affect the channel via G proteins that control channels themselves. Because of the high electrochemical Na+ gradient, the number of incoming Na+ ions are much larger than the number of exiting K+ ions. Ca2+ can also enter the cell. The net influx of cations leads to depolarization: excitatory postsynaptic potential (EPSP). The EPSP begins at approx. 0.5ms after the arrival of an AP at the presynaptic terminal. This synaptic delay is caused by the moderately slow release and diffusion of the transmitter.

A single EPSP generally is not able to generate a postsynaptic (axonal) action potential (APA) but requires the activation of many local depolarizations in the dendrites. Their depolarizations are transmitted electrotonically across the soma and summed on the axon hillock. Should the individual stimuli arrive at different times, the prior depolarization will not have dissipated before the next one arrives, and summation will make it easier to reach the threshold. This type of temporal summation, therefore, increases the excitability of the postsynaptic neuron.

Inhibitory transmitters include substances as glycine, GABA (Gamma-aminobutyric acid), and acetylcholine. They increase the conductance, g, of the subsynaptic membrane only to K+. The membrane usually becomes hyperpolarized in the process. Increases in gK occur when Em approaches EK. However, the main effect of this inhibitory postsynaptic potential IPSP is not hyperpolarization–which works to counter EPSP-related depolarization. Instead, the IPSP related increase in membrane

conductance short circuits the electrotonic currents of the EPSP. Since both EK and ECl are close to the resting potential, stabilization occurs, that is, the EPSP is cancelled out by the high K+ and Cl– short circuit currents. As a result, EPSP-related depolarization is reduced, and the stimulation of postsynaptic neurons is inhibited.

Termination of synaptic transmission can occur due to inactivation of the cation channels due to a conformational change in the channel like the one that occurs during an action potential. This very rapid process called desensitization also functions in the presence of a transmitter. Other terminating pathways include the rapid enzymatic decay of the transmitter (e.g., acetylcholine) while still in the synaptic cleft, the re-uptake of the transmitter (e.g., noradrenaline) into the presynaptic terminal or uptake into extra neuronal cells (e.g., in glial cells of the CNS), endocytotic internalization of the receptor, and binding of the transmitter to a receptor on the presynaptic membrane. In the latter case, a rise in gK and a drop in gCa can occur, thus inhibiting transmitter release.

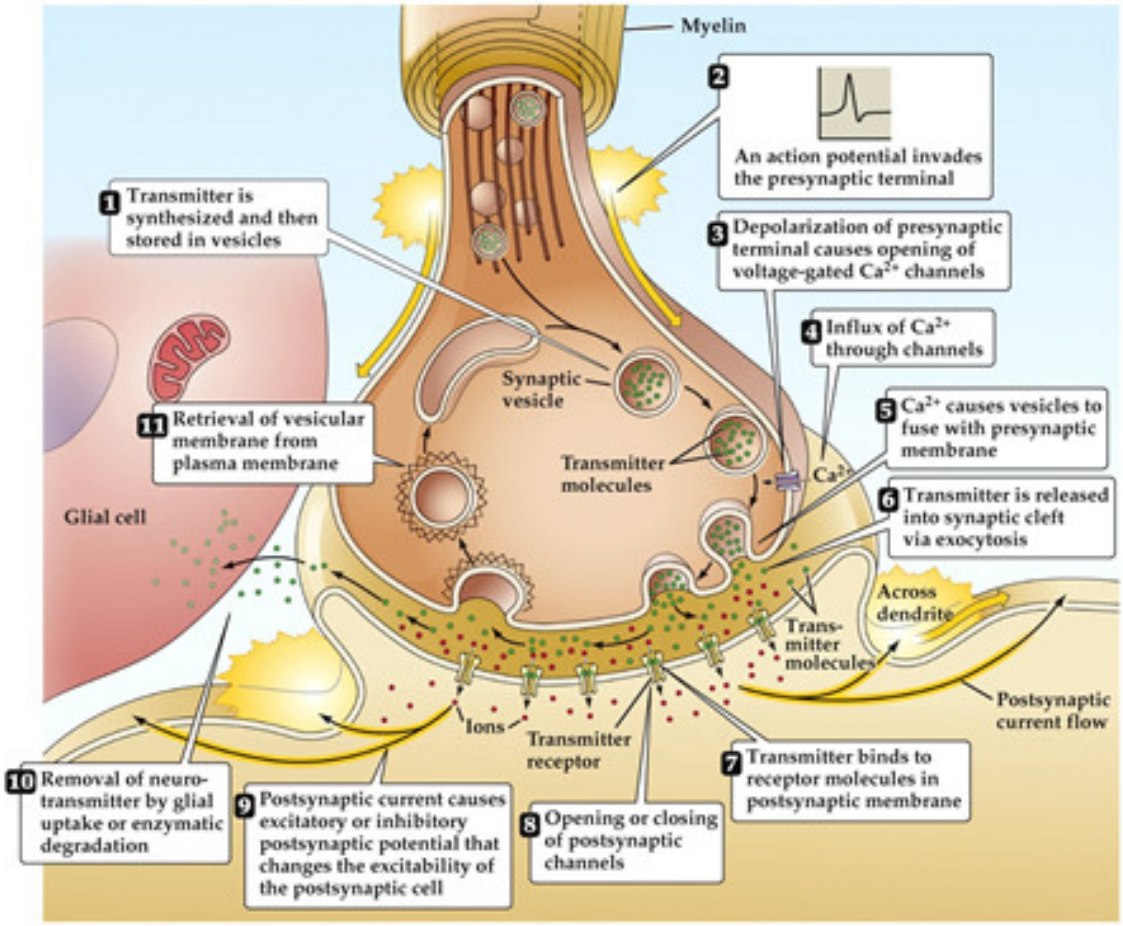

Figure 17. Summary of the synaptic transmission.

Neurological adaptations to resistance training

Training for increased muscular strength not only affects muscle tissue but also results in changes in the central nervous system. Unfortunately, early evidence on neurological adaptations has been generated [in part] from indirect observations and inferences drawn from techniques that were applied to animal studies. However, evidence over the last 20-years suggests that neural adaptations result from alterations in co-ordination and learning that aid in improved recruitment and activation of muscles during resistance training (RT). This evidence has transpired due to non-invasive methods that capture electrical and twitch responses of muscle.

Motor unit recruitment, rate coding, and synchronisation

Early observations on muscular strength reported that when performing a RT program, the muscle responds to the demands of increasing amounts of resistance lifted (Hellebrandt and Houtz, 1956). This increase in muscle strength results from the ability of the nervous system to recruit motor units (MU) involved in the activation of the muscle and the adaptations at the muscle fibre (Sale, 1988; Tesch, 1988). These alterations in the nervous system following RT have been referred to in scientific literature as neural adaptations (Sale, 1988). Studies have reported increases in strength with no or minimal changes in the cross-sectional area of the muscle (Moritani and deVries, 1979; Tesch, 1988).

Motor unit recruitment

Henneman and Olson (1965) developed the selective recruitment theory, which states that MUs are composed of the same fibre types and are innervated by motor neurons with different thresholds for each fibre type. The MU is considered the functional unit of the muscle and consists of a motor cell, the axon and terminal branches and all the individual muscle fibres supplied by the axon (Freund, 1983). A skeletal muscle, together with the motor neurons controlling it, comprises several hundred MU of different sizes (**Figure 18**). A single motor unit can innervate a few to several hundred muscle fibres with each specific muscle containing ten to several hundred MU. For example, muscles involved in eye movement have an innervation ratio of 1:4 (the number of motor axons divided by the total number of muscle fibres). Large muscles

that do not require the same degree of motor control have an innervation ratio as large as 1:300 (i.e., gastrocnemius muscle) (Garnett et al., 1979). The force output of a muscle is determined by the sum of the force outputs of the active MU (Senn et al., 1997). An increase in force production is, therefore, a result of the systematic activation of a greater number of MU being recruited and an increased frequency of activation (rate coding) (**Figure 19**).

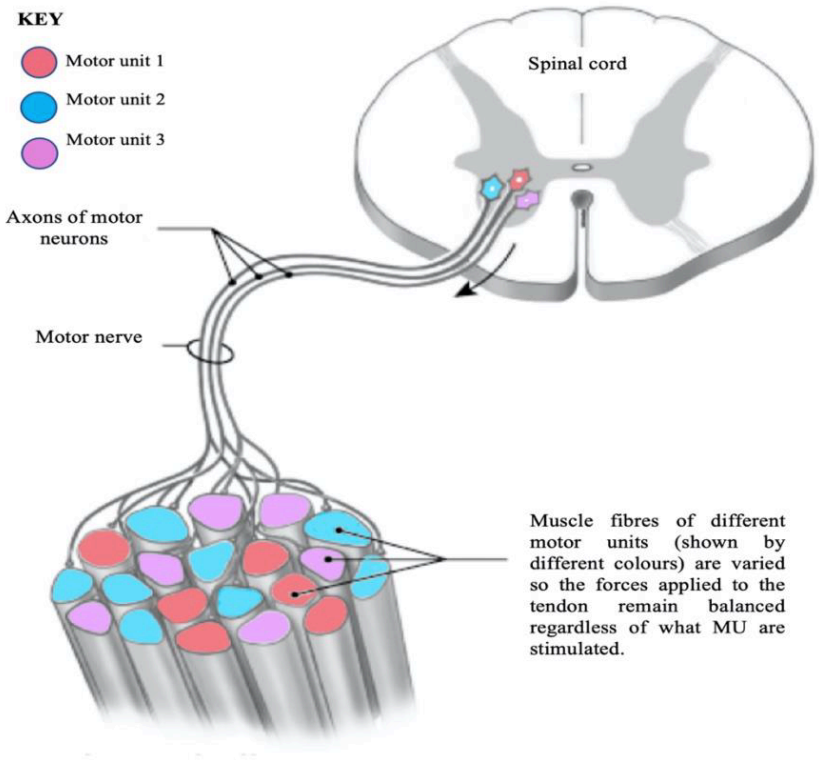

Figure 18. Muscle innervation by the motor neurons. The ventral horn of the spinal cord contains motor neurons that innervate skeletal muscle fibres.

The size principle and the application to training

The Henneman size principle states that when the central nervous system recruits MU for a specific activity, it initiates smaller, easily excited MU's and proceeds to the larger more difficult to excite MU's (Henneman et al., 1974). This orderly recruitment of MU provides a smooth graduation of force (Guyton, 1991). If the input to a pool of MU's exceeds the excitation threshold of the motor neuron, the neuron will generate nerve impulses (action potential) to activate the innervated fibres (Guyton, 1991; Senn et al., 1997). Cope and Pinter (1995) specified that the motor neuron recruitment threshold

is determined by the interaction between the strength and organisation of the synaptic inputs and motor neuron responses. Depending on the muscle, studies have suggested that maximal recruitment occurs between 30-to-90% of maximal voluntary contraction (Enoka and Fuglevand, 2001; DeLuca, Foley and Erim, 1996). Linnamo et al., (2003) reported that the threshold for maximal recruitment was higher in static compared to dynamic muscle actions. Most peer-reviewed evidence suggests that there are no functionally significant violations of the size principle (Cope and Pinter, 1995).

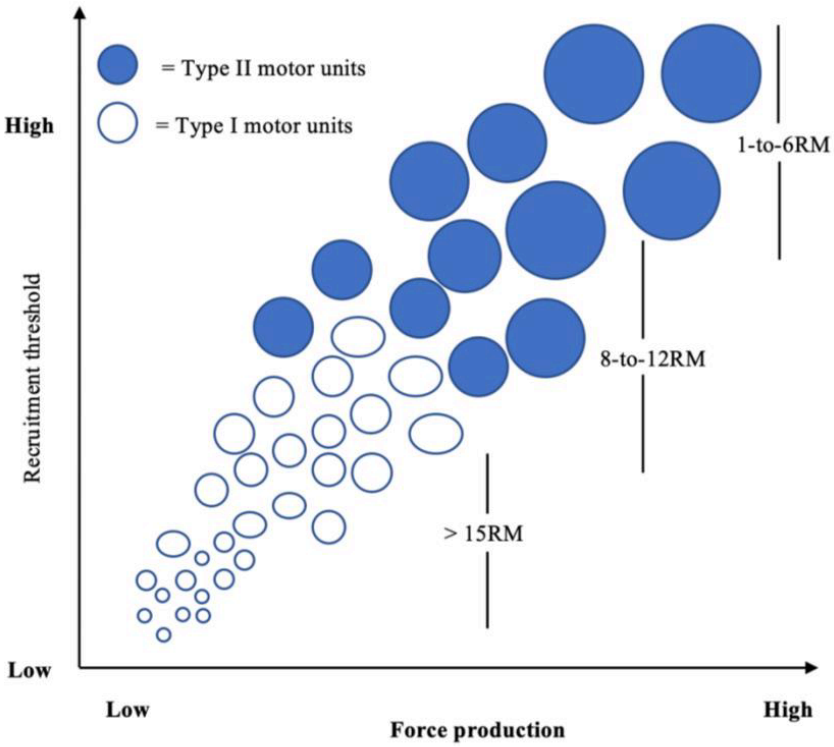

Figure 19. The size principle. Each circle denotes a motor unit made up of different types and numbers of fibres. The blue outlined circles are Type I motor units, and the blue circles are Type II motor units, with larger circles depicting larger motor units containing more fibres. As one goes up the line of orderly recruitment, heavier and heavier resistances recruit more motor units and their associated muscle fibres.

Until recently, the body of scientific evidence on neurological adaptations was derived indirectly with varied techniques used to evaluate neural adaptations. However, recent technological advancements have more accurately defined the specific neural mechanisms contributing to RT induced increases in maximal strength. Folland and Williams (2007) stated that neural adaptations are positive alterations in

co-ordination and learning that enable better MU recruitment and activation of the muscles involved during a specific strength task. This is apparent, particularly in the early stages of when untrained individuals perform strength training, as there are disproportionately larger increases in muscle strength than muscle size (**Figure 10**).

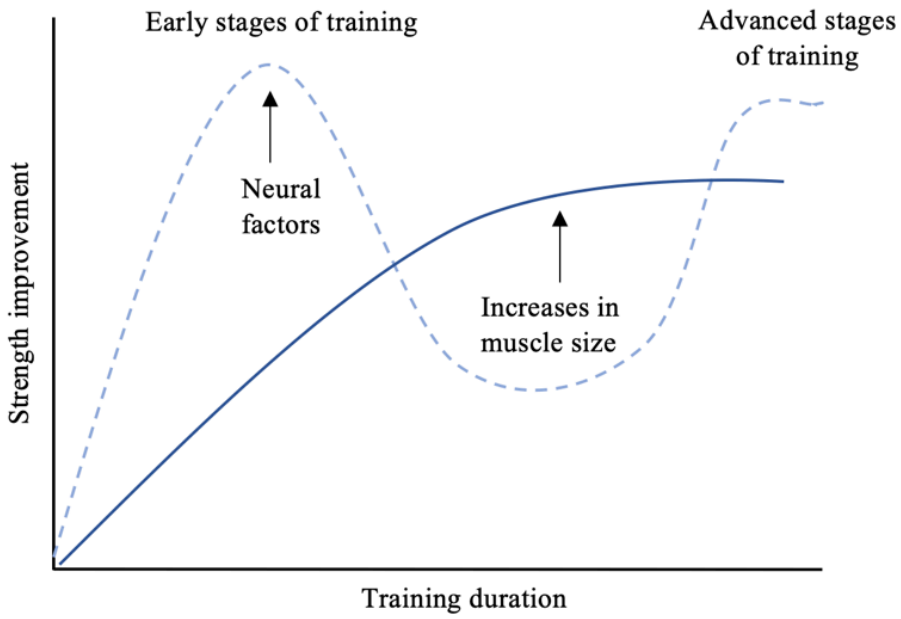

Figure 20. Neuromuscular interplay to resistance training stimulus over time.

However, there are several morphological and neurological factors that contribute to increased strength following RT. Typically, increased size of an exercised muscle is regarded as the long-term adaptation, although this varies to the exposure to the training stimulus. The body of indirect evidence suggests substantial neurological adaptations may be due to learning and changes in the intermuscular co-ordination of agonist and antagonist interactions. During the early stages of RT, strength is associated with greater neural adaptations (within several weeks). This is in contrast with trained and elite individuals as the extent of the adaptations are within the muscles, promoting an increase in muscle mass (hypertrophy) (**Figure 21**).

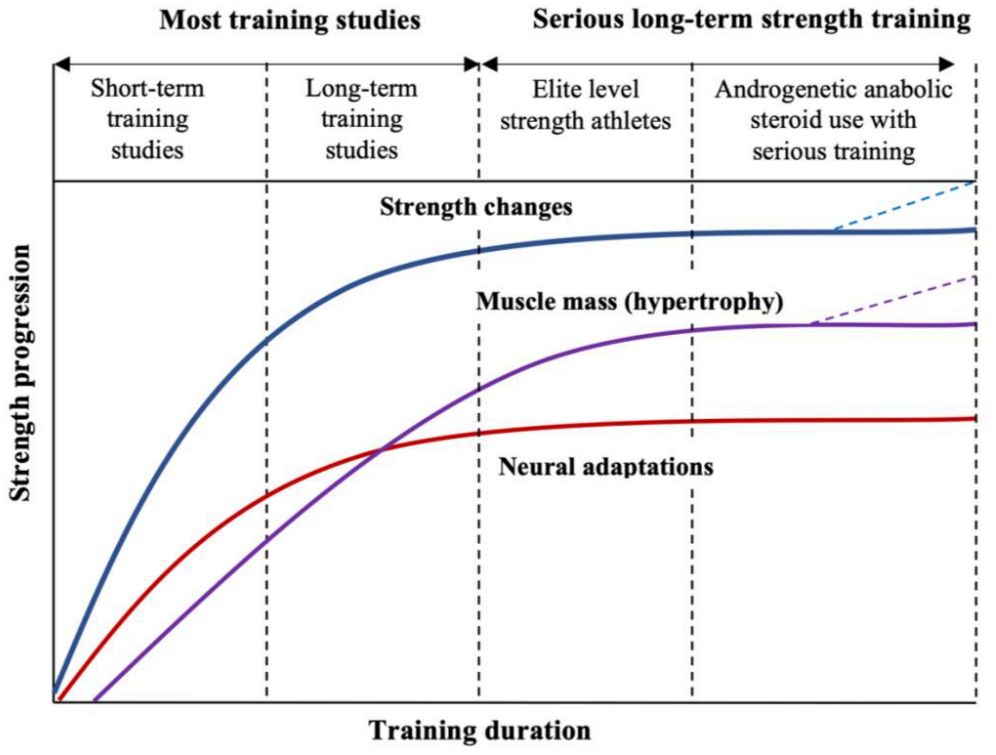

Figure 21. Short-term to long-term neural and muscle adaptations to strength training.

Rate coding and synchronisation

As discussed previously, to activate and deactivate MU requires structured sequencing based on the size principle (Henneman et al., 1974). This sequence depends on the size of the MU with the smallest motor neurons recruited first and deactivated last, and MU with the largest motor neurons recruited last and deactivated first. According to Henneman and colleagues (1974), the order of MU recruitment is encoded by size and is not directly organised by the brain. The production of muscle force depends on the number of active MU and the rate at which the MU discharge action potentials. There are two kinds of rate coding with evidence suggesting that muscle force exerted during a voluntary contraction is dependent on the number of MU and the rates and frequency in which these MU discharge action potentials (Milner-Brown et al., 1973). These properties are known as recruitment and rate coding with early studies suggesting that recruitment is a more significant factor for low forces, whereas rate coding is more responsible for changes in muscle force at intermediate and high forces. Milner-Brown et al., (1973) introduced electromyography measurements that quantified MU synchronisation by measuring MU activity. This six- week resistance study indicated increased MU synchronisation in the first dorsal interosseous muscle

of the hand. Milner-Brown, Stein and Lee (1975) performed a further cross-sectional study comparing weightlifters to a control group. Results from this study observed that all seven weightlifters showed a significant degree of synchronisation. Semmler and Nordstrom (1998) indicated that athletes possess greater MU synchronisation than untrained individuals with heavy RT, increasing synchronisation.

Muscle adaptations to resistance training

RT promotes structural and functional changes in the body as indirect observations made on the muscular size and strength of highly trained strength athletes. The magnitude of these muscular adaptations is directly proportional to the physical stress placed on the body (training volume, loading and frequency) and the recovery period necessary to produce favourable changes.

Muscle fibre types

Humans comprise of varying percentages of muscle fibres, with fibre composition varying between muscles and individuals (Gollnick and Matoba, 1984). Several classifications have been used to distinguish different fibre types established from biochemical, histochemical, and physiological evidence (Morris, 1969; Morris, 1970; Peter et al., 1972; Sale et al., 1983). Early researchers developed physiological techniques that assessed the contractile properties of muscle and the speed at which fibres can generate peak tension (**Table 1**). Two fibre types (slow-twitch [ST] and fast-twitch [FT]) were identified (Morris, 1969). These fibres developed different tension characteristics with ST developing tension more slowly than FT fibres but were resistant to fatigue. Morris (1969) reported that ST fibres are recruited for long term, low-intensity work and FT for short duration, high-intensity work (i.e. resistance training).

Early studies on RT programs demonstrated that FT and ST fibre ratio increased and selective hypertrophy of individual fibres following heavy progressive loading and explosive jump training (Thorstensson et al., 1976; Häkkinen, Alen and Komi, 1985). Thorstensson et al., (1976) examined the effects of progressive strength training on eight healthy male subjects (22-to-31 years) leg extensor muscles over

eight-weeks. Thorstensson and colleagues reported that no or minor alterations were observed in anthropometrics, muscle enzyme, and fibre composition. However, the muscle fibre ratio suggested a specific effect of ST on fast-twitch muscle fibres. Häkkinen and colleagues (1985) examined 11 male subjects performing 24-weeks of high-load ST. It was reported that subjects increased maximal isometric strength by 26.8%. The increases in strength correlated with significant increases in neural activation of the leg extensor muscles during an intensive period of training. Häkkinen et al., (1985) suggested that selective RT induced hypertrophy contributed to strength gain. Other researchers have also reported that heavy RT can influence hypertrophy on FT fibres, with Prince, Hikiia and Hagerman (1976) and Tesch (1988) suggesting that weightlifting increases FT fibre area more than ST. Research suggests that resistance-trained athletes possess hypertrophied FT fibres, while endurance athletes have more hypertrophied ST fibres.

Table 1. Overview of the evolution of identifying muscle fibre types,

Date	Fibre type conceptualisation	Author (year)	Pioneering studies and milestones
1960-to-1967	Two muscle fibre types: Slow twitch (red muscle fibres) Fast twitch (white muscle fibres)	Dubowitz and Pearse (1960) Buller et al., (1960) Bárány (1967)	Enzyme histochemistry exposes a relationship between glycolytic and oxidative enzymes in muscle fibres. Cross-innervation of cat muscles (fast and slow) leads to a partial conversation of their contractile properties. Myosin ATPase activity is higher in fast than slow muscles and correlates with speed of muscle shortening.
1967-to-1975	Three muscle fibre types: Slow type 1 Fast type 2A Fast type 2B	Guth and Samaha (1969) and Brooke and Kaiser (1970) Burke et al., (1971) Peters et al., (1972)	Identification of type 1, 2A and 2B fibres by myosin ATPase histochemical staining. Identified in cat muscles motor units composed of type 1, 2A or 2B. Slow oxidative, fast oxidative glycolytic and fast glycolytic fibres identified by enzyme biochemistry.
1986-to-1991	Slow type 1 Fast type 2A Fast type 2X Fast type 2B	Larsson et al., (1991) Bottinelli et al., (1991;1994) Smerdu et al., (1994) Ennion et al., (1995)	Identification of type 2X motor units. Single fibre analyse reveals different speeds of shortening of the four fibre types. Human muscle fibres classified by ATPase histochemistry contain 2X myosin heavy chain.

However, the development of staining techniques and electron microscopy measurements led to additional fibre classifications (Brooke and Kaiser, 1970; Burke et al., 1971). Researchers identified a third fibre type in addition to ST and FT fibres (Peter et al., 1972; Brooke and Kaiser, 1970; Burke et al., 1971). Rose and Rothstein (1982) combined the different classification schemes and described muscle fibres based on biochemical, histochemical, and physiological. The three muscle fibre types were reclassified: Type I (slow oxidative, ST) fibres Type IIA (fast oxidative glycolytic, FT-fatigue resistant) fibres Type llB (fast glycolytic, FT-fast fatigable) fibres Staron et al., (1984) applied this fibre type classification and performed a cross-sectional investigation on weightlifters, distance runners and sedentary individual's fibre type ratios. The authors observed that weightlifters had a significantly larger type IIA area than the other two groups (distance runners [aerobically trained] and untrained [control] subjects).