

## 6.5 Neurones & Synapses

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## 6.5.1 Neurones: Function & Structure

### **Function & Structure of Neurones**

#### The nervous system

- The human nervous system consists of:
  - Central nervous system (CNS) the brain and spinal cord
  - Peripheral nervous system (PNS) all of the nerves in the body
- It allows us to make sense of our surroundings and respond to them, and to coordinate and regulate body functions
- Information is sent through the nervous system in the form of electrical impulses these are electrical signals that pass along nerve cells known as neurones
  - A bundle of neurones is known as a nerve
- The nerves spread out from the central nervous system to all other regions of the body and importantly, to all of the sense organs
  - The **CNS** acts as a **central coordinating centre** for the impulses that come in from, and are sent out to, any part of the body



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• The following features are found in neurones:

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- Neurones have a main, long, fibre known as an axon
- They have a **cell body** that contains the **nucleus** and other cellular structures
- Their cell bodies and axon terminals contain many extensions called dendrites
- These **dendrites** allow them to **connect** to **many other neurones** and receive **impulses** from them, forming a **network** for easy **communication**



Neurones have a characteristically elongated structure which allows them to transfer information between the central nervous system and the rest of the body

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### Research

NOS: Cooperation and collaboration between groups of scientists; biologists are contributing to research into memory and learning

- Some of the so-called 'higher' functions of the brain e.g., memory and learning, are still not fully understood and are the focus of much current research
- Biologists are becoming increasingly involved in this research, which uses techniques from the fields of neurobiology, molecular biology, and biochemistry to understand the mechanisms behind these brain functions
- The Centre for Neural Circuits and Behaviour (CNCB) at the University of Oxford is a good example of an
  institution in which scientists with different areas of expertise collaborate, or work together, with a
  common research goal
  - The research team at the CNCB contains experts in various fields of biological science, including medicine, physiology, genetics, molecular biology, neurobiology, and neurogenetics
- Research into functions of the brain such as memory and learning not only involves collaboration between scientists from different specialities, but also from different countries



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## **Myelination**

- Neurones have a main, long, fibre known as an axon
- The axons of neurones are surrounded by specialised cells called Schwann cells
- Schwann cells wrap themselves around the axon, forming a structure known as a myelin sheath
  - Myelin contains the phospholipids of the Schwann cell membranes; it is built up in layers as the Schwann cells grow around the axon
  - The lipid content of the myelin sheath gives it a high electrical resistance
- The myelin sheath acts as an **electrical insulator**; impulses cannot pass through the myelin sheath
- The myelin sheath has **small, uninsulated sections** in the gaps between the individual Schwann cells
  - These gaps are called nodes of Ranvier
- Electrical impulses effectively **jump** from one node of Ranvier to the next
  - This process is known as **saltatory conduction**
  - It greatly speeds up the rate of transmission of impulses along myelinated neurones
  - In non-myelinated neurones the axon is not insulated by myelin, so the impulse travels more slowly



An impulse travels down a neurone via saltatory conduction

## 6.5.2 Nerve Impulses

## **Resting Potential**

- **Neurones** transmit information in the form of **impulses**, which travel extremely quickly along the neurone from one end to the other
  - Note that an impulse is **not** an electrical current that flows along neurones as if they were wires
  - Instead, an impulse is a momentary reversal in the electrical potential difference across the neurone cell surface membrane
    - The electrical potential difference across a membrane can also be described as the **voltage** across a membrane, the **difference in charge** across a membrane, or the **membrane potential**
- In an axon that is not transmitting an impulse the inside of the axon always has a negative electrical potential, or charge, compared to outside the axon, which has a positive electrical potential
  - This membrane potential in a resting neurone is known as resting potential
- The resting potential is usually about -70 millivolts (mV)
  - This means that the inside of the resting axon has a more negative electrical charge than the outside by about 70 mV
- Two main processes contribute to establishing and maintaining resting potential:
  - The active transport of sodium ions and potassium ions
  - A difference in rates of diffusion of sodium ions and potassium ions
- In addition to these two main processes, **negatively charged proteins** inside the axon also contribute to the negative resting potential

#### The active transport of sodium ions and potassium ions

- Carrier proteins called sodium-potassium pumps are present in the cell surface membranes of neurones
- These pumps use ATP to actively transport sodium ions (Na\*) out of the axon and potassium ions (K\*) into the axon
- The two types of ion are pumped at an unequal rate; for every 3 sodium ions that are pumped out of the axon, only 2 potassium ions are pumped in
- This creates a concentration gradient across the membrane for both sodium ions and potassium ions

#### Difference in rates of diffusion of sodium ions and potassium ions

- Because of the concentration gradient generated by the sodium-potassium pumps, both sodium and potassium ions will diffuse back across the membrane
  - The neurone cell surface membrane has **sodium ion channels** and **potassium ion channels** that allow sodium and potassium ions to move across the membrane by **facilitated diffusion**
- The neurone membrane is much **less permeable** to sodium ions than potassium ions, so potassium ions inside the neurone can diffuse **out** at a **faster rate** than **sodium ions** can diffuse **back in**

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- This results in **far more positive ions** on the **outside** of the neurone than on the inside, generating a **negative charge inside** the neurone in relation to the outside
- The result of this is that the neurone has a resting membrane potential of around -70 millivolts (mV)



Sodium-potassium pumps in the membrane of a resting neurone generate a concentration gradient for both sodium ions and potassium ions. This process, together with the facilitated diffusion of potassium

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ions back out of the cell at a faster rate than sodium ions diffuse back into the cell, generates a negative resting potential across the membrane.



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## **Action Potential**

- Once resting potential is reached, the neurone membrane is said to be **polarised**
- To initiate a nerve impulse in a neurone, the neurone membrane needs to be **depolarised** 
  - Depolarisation is the reversal of the electrical potential difference across the membrane
- The depolarisation of the membrane occurs when an action potential is generated
  - Action potentials lead to the reversal of resting potential from around -70 mV to around +40 mV
- Action potentials involve the rapid movement of sodium ions and potassium ions across the membrane of the axon
- An action potential is the **potential electrical difference** produced across the axon membrane when a neurone is **stimulated** e.g. when an environmental stimulus is detected by a receptor cell

#### How an action potential is produced

- Some of the ion channels in the membrane of a neurone are **voltage gated**, meaning that they open and close in response to changes in the **electrical potential** across the membrane
  - Voltage gated ion channels are **closed** when the membrane is at rest, but they are involved in the generation and transmission of action potentials
  - Note that not all of the channels in a neurone membrane are voltage gated e.g. some types of
    potassium ion channel are open when a neurone is at rest to enable potassium ions to diffuse out
    of the axon and generate resting potential
- When a neurone is stimulated, the following steps occur:
  - A small number of **sodium ion channels** in the axon membrane **open**
  - Sodium ions begin to move into the axon down their concentration gradient
    - There is a greater concentration of sodium ions outside the axon than inside due to the action of sodium-potassium pumps
  - This reduces the potential difference across the axon membrane as the inside of the axon becomes less negative
  - If enough sodium ions enter the axon and the potential difference is reduced enough, voltage gated sodium ion channels open, leading to a further, large influx of sodium ions
  - Once the charge has been reversed from -70 mV to around +40 mV, an action potential is said to have been generated

#### How an action potential is propagated

- Once an action potential has been generated, it can be **propagated**, or transmitted, along the length of the axon
  - The depolarisation of the membrane at the site of the first action potential causes **sodium ions** to diffuse along the cytoplasm into the next section of the axon, **depolarising** the membrane in this new section, and causing voltage gated sodium channels to open
  - This triggers another action potential in this section of the axon membrane
  - This process then repeats along the length of the axon

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• In the body, this allows action potentials to begin at one end of an axon and then pass along the entire length of the axon membrane





Repolarisation

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- About 1 ms after an action potential is generated, all the voltage gated sodium channels in this section of membrane close
- Voltage gated potassium channels in this section of axon membrane now open, allowing the diffusion of potassium ions out of the axon, down their concentration gradient
  - Remember that the sodium-potassium pumps have not stopped working during the action potential; hence the potassium ion gradient is still present
- This movement of potassium ions causes the inside of the axon to become negatively charged again, a process known as **repolarisation** 
  - There is a short period during which the membrane potential is more negative than resting potential; this is known as **hyperpolarisation**
  - The period during which the membrane is hyperpolarised is known as the **refractory period** 
    - The membrane is unresponsive to stimulation during the refractory period, so a new action potential cannot be generated at this time
    - This makes the action potentials **discrete** events and means the impulse can **only travel in one direction**
    - This is essential for the successful and efficient transmission of nerve impulses along neurones
- The voltage gated potassium channels then close, and the sodium-potassium pumps work to restore resting potential
  - Only once resting potential is restored can the membrane be stimulated again



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The depolarisation and repolarisation of an action potential can be clearly seen in a graph of membrane potential against time



## **Threshold Potential**

- An action potential is only initiated if the threshold potential is reached
- When a neurone is stimulated, sodium ion channels in the axon membrane open and sodium ions pass into the axon down their concentration gradient
- This causes the inside of the axon to become **less negative**, but exactly how much less negative it becomes is dependent on the number of sodium ion channels that open
  - A large stimulus will cause more channels to open than a small stimulus
  - If more channels open, then more sodium ions will enter the axon, causing it to become less negative
- If the potential difference reaches around -50 mV, known as the threshold potential, voltage gated sodium ion channels open and many more sodium ions enter the axon
  - This causes the membrane potential to reach around +40 mV
- Once the charge has been reversed from -70 mV to +40 mV, an action potential is generated

#### The all-or-nothing principle

- Action potentials are either generated or not generated depending on whether the threshold potential is reached; there is **no such thing as a small or large action potential** 
  - If a stimulus is **weak**, only a few sodium ion channels will open and the membrane won't be sufficiently depolarised to reach the **threshold potential**; an action potential will not be generated
  - If a stimulus is **strong enough** to raise the membrane potential above the **threshold potential** then an action potential will be generated
- This is the **all-or-nothing principle** 
  - An impulse is **only transmitted** if the **initial stimulus is sufficient** to increase the membrane potential above a **threshold potential**
- Stimulus size can be detected by the brain because as the intensity of a stimulus increases, the frequency of action potentials transmitted along the neurone increases
  - This means that a small stimulus may only lead to one action potential, while a large stimulus may lead to several action potentials in a row





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## Local Currents

- The propagation of nerve impulses along axons occurs due to local currents that cause each successive section of the axon to reach the threshold potential
- Inside the depolarised section of the axon
  - There is a **high concentration** of sodium ions due to their recent **influx**
  - This creates a **concentration gradient** between the section of the axon that has depolarised and the section next to it
  - Sodium ions diffuse along **inside** the axon to the neighbouring section of axon that has not yet become depolarised
  - This reduces the negative membrane potential in the new section of axon and, if a threshold is reached, begins the initiation of an action potential
    - This enables the original action potential to be propagated
- On the **outside** of the axon
  - There is a higher concentration of sodium ions outside the section of axon that has **not yet become depolarised** due to the diffusion of sodium ions into the depolarised section
  - Sodium ions diffuse from here along the outside of the axon to the section of axon that has just become depolarised
- These movements of sodium ions are known as **local currents**
- These local currents cause a **wave of depolarisation** and **repolarisation** to travel along the axon, resulting in the **propagation of a nerve impulse**



## The propagation of nerve impulses along axons occurs due to local currents created by the diffusion of sodium ions

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## 6.5.3 Synapses

### Synapses

- Where two neurones meet, they do not actually come into **physical contact** with each other
- Instead, a very small gap, known as the synaptic cleft, separates them
- The ends of the two neurones, along with the synaptic cleft, form a structure known as a synapse
- Synapses act as the junctions **between any cells in the nervous system**, e.g.
  - In the sense organs, there are synapses between **sensory receptor cells** and **sensory neurones**
  - In muscles, there are synapses between **motor neurones** and **muscle fibres**



• Electrical impulses cannot 'jump' across the synaptic cleft

- When an electrical impulse arrives at the end of the axon on the presynaptic neurone, the membrane of the presynaptic neurone becomes depolarised, triggering an influx of calcium ions into the presynaptic cell via calcium ion channels in the membrane
- The calcium ions cause vesicles in the presynaptic neurone to move towards the presynaptic membrane where they fuse with it and **release chemical messengers** called **neurotransmitters** into the synaptic cleft
  - A common neurotransmitter is **acetylcholine**, or **ACh**
- The neurotransmitters diffuse across the synaptic cleft and bind with receptor molecules on the postsynaptic membrane; this causes associated sodium ion channels on the postsynaptic membrane to open, allowing sodium ions to diffuse into the postsynaptic cell
- If enough neurotransmitter molecules bind with receptors on the postsynaptic membrane then an **action potential** is generated, which then travels down the **axon** of the **postsynaptic neurone**
- The neurotransmitters are then broken down to prevent continued stimulation of the postsynaptic neurone
  - The enzyme that breaks down acetylcholine is **acetylcholinesterase**







#### Synaptic transmission using the neurotransmitter acetylcholine

#### Unidirectionality

- Synapses ensure the **one-way transmission** of impulses
- Impulses can only pass in one direction at synapses because neurotransmitter is released on one side and its receptors are on the other – chemical transmission cannot occur in the opposite direction
- This prevents impulses from travelling the wrong way

## Acetylcholine

- There are over 40 different known **neurotransmitters** 
  - Examples include dopamine and noradrenaline
- One of the key neurotransmitters used throughout the nervous system is acetylcholine (ACh)
  - ACh is produced in the **presynaptic neurone** by combing **choline** with an **acetyl group**
  - Synapses that use the neurotransmitter ACh are known as cholinergic synapses
- Acetylcholine is released into the synaptic cleft when ACh-containing vesicles fuse with the presynaptic membrane, releasing ACh molecules into the synaptic cleft
- ACh binds to specific receptors on the postsynaptic membrane, where it can generate an action potential in the postsynaptic cell by opening associated sodium ion channels
- To prevent the sodium ion channels staying permanently open and to stop permanent depolarisation of the postsynaptic membrane, the **ACh molecules are broken down** and **recycled** 
  - The enzyme acetylcholinesterase catalyses the hydrolysis of ACh molecules into acetate and choline
  - The products of hydrolysis are then absorbed back into the presynaptic neurone, and the active neurotransmitter ACh is reformed

## Inhibition of Acetylcholine Receptors

- **Neonicotinoids** are synthetic compounds similar to nicotine that are commonly found in **pesticides**
- Neonicotinoids can block synaptic transmission at cholinergic synapses in insects by binding to acetylcholine receptors
  - This binding is irreversible, as acetylcholinesterase cannot break down neonicotinoids
  - As the acetylcholine receptors are blocked, **acetylcholine is unable to bind**, which **stops impulses** from being transmitted across synapses
  - This leads to **paralysis** and **death** in insects
- Neonicotinoids are considered to be especially suitable as pesticides because they're not toxic to humans and other mammals
  - A much larger proportion of synapses in insects are cholinergic compared to mammals
  - Neonicotinoids bind much more strongly to acetylcholine receptors in insects
- There is a great deal of controversy over the use of neonicotinoid pesticides because of the impact that they are thought to have on essential pollinators such as bees



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## 6.5.4 Skill: Neurones & Synapses

### Analysis of Oscilloscope Traces

- It is possible to measure membrane potentials in neurones by placing electrodes on each side of the membrane
  - A membrane potential is the **difference in charge** between one side of a membrane and the other, sometimes described as the potential difference, or the voltage
- The membrane potential can then be visually represented and displayed using an oscilloscope
- An oscilloscope is a type of electronic test instrument that graphically displays varying signal voltages
- The display produced is **like a graph** with **time** in milliseconds on the **x-axis** and the membrane **potential** in millivolts on the **y-axis**

#### How to analyse oscilloscope traces showing resting potentials and action potentials

- If there is a resting potential, a straight, horizontal line should be shown on the display screen of the oscilloscope at a level of -70 mV
- If an action potential occurs a spike, rising up to a maximum voltage of between +30 and +40 mV, should be shown on the display
  - The rising phase of the spike shows depolarisation
  - The falling phase of the spike shows repolarisation
- Often not shown on an action potential graph is the gradual rise in membrane potential just before the membrane rapidly depolarises
  - Before threshold potential is reached, only a small number of sodium channels in the membrane are open, so the membrane depolarises slowly, but when the threshold is reached many more sodium channels open
- Instead of repolarisation causing the membrane potential to return immediately to the normal resting potential of -70 mv, the trace often shows a short period of hyperpolarisation
  - This is when the membrane potential briefly becomes more negative than resting potential



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An example of an oscilloscope trace showing resting potential and an action potential