

Nerves & Synapses

Neurone Cell Structure

Nerve cells (neurones) are adapted for transmitting nerve impulses over long distances. Each neurone has:

- a cell body which houses the nucleus and a long fibre (axon) which conducts the impulses to their destination.
- a thin sheath of pavement epithelium-like cells called **Schwann cells**. These produce a fatty layer called the **myelin sheath** which is wrapped around the axon, insulating it from adjacent axons and also speeding up the rate of impulse conduction. Some axons have very little myelin and are termed 'non-myelinated'. These occur in the autonomic nervous system and are slow conducting. In myelinated neurones, the myelin sheath is interrupted at points called the nodes of Ranvier.
- fine extension processes to the cell body called **dendrons** which branch into numerous **dendrites**. These give the cell a large surface area to link with other neurones via **synapses**.
- an axon which is also branched at its end to increase the surface area for making synapses. The end branches have synaptic knobs (boutons) in which there are cell vesicles which manufacture and release the chemicals required as synaptic transmitters, such as acetylcholine.
- numerous mitochondria throughout the neurone which provide the energy required for acetylcholine synthesis and for maintaining the sodium and potassium pumps needed for impulse transmission.
- cytoplasm (axoplasm) which streams between the cell body and axon in both directions. There are fine microtubules and microfilaments made of the protein **actin** running through the cytoplasm along the axon. These help to direct the streaming of the cytoplasm to carry out transport within the cell and also act as a cytoskeleton to maintain the cell shape.
- many organelles called Nissl granules. These consist of mitochondria, free ribosomes and rough endoplasmic reticulum. They are concerned with protein synthesis of enzymes involved in impulse transmission and with the synthesis of trophic factors which regulate the growth and differentiation of nervous tissue.

Functions of Neurones

Sensory neurones conduct impulses from receptors and sense organs to the central nervous system. They are usually myelinated and thus fast conducting. **Relay neurones** conduct impulses within the central nervous system from sensory neurones to effector (motor) neurones. In the white matter they are myelinated and fast conducting and carry impulses up and down the length of the spinal cord and brain. In the gray matter they are non-myelinated and so slow conducting. However, these only conduct impulses for short distances, for instance, from the dorsal to the ventral sides of the spinal cord. **Effector or motor neurones** conduct impulses from the central nervous system to the effector organs, muscles or glands. In the voluntary nervous system, the motor neurones are myelinated and fast conducting, supplying the skeletal muscles. In the autonomic nervous system, the motor neurones are non-myelinated and slow conducting. They supply smooth muscle in the organs, cardiac muscle in the heart, and glands. The structure of motor and sensory neuones is shown in Fig 1.

Fig 1(a). A myelinated Motor neurone



Fig 1(b). Sensory neurone



The physiology of nervous transmission

The maintenance of a resting potential

Neurones can only develop and transmit impulses if they possess a resting potential to begin with. At rest: (see Fig 2)

- 1. the neurone membrane is impermeable to the entry of sodium ions but
- 2. sodium ions are actively transported out of the neurone by a sodium pump. Thus, the resting neurone becomes deficient in positive ions with a sodium concentration of only 10mmoles dm⁻³ inside compared with 142mmoles dm-3 outside.
- 3. At the same time, the neurone membrane is freely permeable to the passage of potassium ions and these are attracted into the neurone to balance the deficit of positive ions within. This results in potassium concentrations of only 7mmoles dm-3 outside the neurone compared with 135mmoles dm⁻³ inside the neurone. However, the influx of potassium never quite catches up with the outflow of sodium which means that there is always a slight deficit of positive ions within the neurone.
- 4. Thus, inside remains negative with respect to the outside. This resting potential is usually in the range -60 to -90 millivolts.



The generation of an action potential

An action potential must be generated before an impulse can be established and transmitted. It is generated in response to a stimulus and it involves a sequence of changes in permeability of the membrane and in the distribution of ions. The stimulus must be above a certain minimum value, called the threshold stimulus and it produces a maximum all or none response in the neurone. The stimulus may come from the external environment, such as changing light or pressure, or may come from the internal environment, such as changing blood temperature or pH. The stimulus causes the neurone membrane to become permeable to the entry of sodium ions for a few ten thousandths of a second. Sodium ions thus rush into the neurone along the concentration gradient. This results in a reversal of polarity to around +35 millivolts (Fig 3). This is depolarisation. At this stage the membrane becomes even more permeable to potassium ions and these rapidly leave the neurone to balance the negative charge outside. The sluggish sodium pump is also removing positive sodium ions from the neurone but at a slow pace. As a result of these ionic movements, the membrane becomes repolarised back to near its resting potential. The neurone can then be restimulated to establish another action potential. This sequence of depolarisation and repolarisation can be repeated many times so that impulses flow in volleys. A volley of impulses is called a signal.

Fig 3. The action potential



The refractory period is the time that must elapse after a stimulus has established an action potential before a second stimulus can set up another action potential. Enough potassium outflow from the neurone must have occurred before a second action potential can arise. In the voluntary nervous system (myelinated neurones) the refractory period is about $\frac{1}{2500}$ of a second allowing a volley frequency of up to 2500 impulses per second. In the involuntary autonomic nervous system (non-myelinated neurones) the refractory period is about $1/_{50}$ of a second, allowing a volley frequency of only 50 impulses per second.

Propogation of the Impulse

Once the action potential (impulse) has been established it is propogated by local currents along the axon. In theory, these can pass either way along the axon. However, synapses can only conduct in one direction. Each local current at the edge of the area of depolarisation causes depolarisation of the next section of membrane. Thus a wave of depolarisation passes along the neurone from the point of stimulation followed by a wave of repolarisation as the sodium permeability of the membrane returns to the resting state. In myelinated neurones the electrons of the local currents jump from one node of Ranvier to the next.

This has two important consequences:

- 1. Gain or loss of ions is restricted to the nodes and so the sodium and potassium pumps have to expend less energy in recovery.
- 2. This jumping conduction (saltatory conduction) greatly increases the speed at which impulses travel. Thus, myelinated neurones in the voluntary nervouc system can conduct up to 100 m sec⁻¹ whereas the non-myelinated fibres of the autonomic nervouc system only conduct at around 5 m sec⁻¹. Saltatory conduction is illustrated in Fig 4.



Local currents leap from Node to Node

Synapses

Synapses are junctions between neurones or between neurones and muscles (neuromuscular junctions). The structure of synapses can be seen in Fig 5.

Fig 5. Synapses



Synapses transmit signals between neurones by means of chemicals. They may be **excitatory** or **inhibitory** and therefore have an important homeostatic role in switching processes on and off. Excitatory synapses transmit signals from one cell to the next by establishing impulses in the post-synaptic membrane (the membrane of the cell after the synapse). Inhibitory synapses will not transmit the signal and so stimulation of an inhibitory neurone will lead to the stopping of the activity under control.

Synaptic Transmission

The commonest transmitter substance in excitatory synapses is **acetylcholine** (ACh) and such synapses are called **cholinergic**. These include most synapses in the voluntary nervous system, the neuromuscular junction, and many synapses in the autonomic nervous system.

- 1. ACh is synthesised in synaptic vesicles from choline and acetate under the influence of the enzyme choline acetylase.
- 2. ATP from the numerous **mitochondria** present supplies the energy for this.
- 3. When an impulse reaches a synaptic knob, the depolarisation causes some of the vesicles to rupture, releasing ACh into the synaptic cleft.
- 4. The ACh attaches to specific receptors on the post-synaptic membrane and makes it permeable to sodium ions.
- 5. These flood into the neurone, causing a post synaptic potential and depolarisation.
- 6. The enzyme **acetylcholine esterase**, present on neurone membranes and on muscle sarcolemmas, then detaches ACh from the receptors and hydrolyses it to acetate and choline.
- 7. The acetylcholine is then **actively** (hence, the mitochondria) reabsorbed back into the synaptic knob for recycling.
- 8. Thus, the post-synaptic membrane is now impermeable to sodium ions again and repolarisation can occur due to potassium ion outflow.
- 9. Once the refractory period is over, the synapse can transmit another impulse.

Excitatory synapses between sympathetic motor neurones and effectors are termed **adrenergic** because they release **noradrenaline** as their transmitter substance. After depolarisation the noradrenaline is destroyed by the enzyme **monoamine oxidase**.

Various other excitatory transmitters occur in the brain, for example, serotonin, dopamine and glutamic acid.

Inhibitory synapses release transmitters which increase the resting potential thus making it more difficult to establish a reversal potential. Examples of such transmitters are glycine and gamma-amino butyric acid.

The effects of drugs on synaptic transmission

Nicotine causes facilitation by lowering the threshold for activation of neurones. It does this by mimicking the action of acetylocholine on the post-synaptic membrane. **Caffeine** can have a similar effect.

Atropine blocks the action of acetylcholine in parasympathetic synapses and so inhibits parasympathetic functions.

Curare blocks the action of acetylcholine at the neuromuscular junctions and thus causes paralysis.

Practice Questions

1. Read through the following passage, then write on the dotted lines the most appropriate word or words to complete the account.

2. The table refers to three different types of synapse. Complete the table by writing the most appropriate word or words in the empty boxes.

Synapse	Transmitter
Cholinergic synapse	
	Nor adrenaline
Neuromuscular junction (Voluntary NS)	

3. The graph below shows the changes in membrane potential in a presynaptic neurone and a postsynaptic neurone when an impulse arrives at a synapse.

membrane



- (a) Explain how depolarisation occurs in the presynaptic neurone. (4 marks)
- (b) The maximum depolarisation in the presynaptic neurone is +40mV. What is the maximum depolarisation in the postsynaptic neurone? (1 mark)
- (c) How long is the delay between the maximum depolarisation in the presynaptic and postsynaptic neurones? (1 mark)
- (d) What is the cause of this delay? (1 mark)

Answers

Marking points are shown by semicolons

 Sensory; Motor; Sodium; Pump; Negative; Sodium; Potassium; Refractory; Stimulus;

0		

Synapse	Transmitter
Cholinergic synapse	Acetylcholine
Adrenergic	Nor adrenaline
Neuromuscular junction (Voluntary NS)	Acetylcholine

 (a) Stimulus make membrane permeable to sodium; sodium ions enter; along the concentration gradient;

therefore potential reverses; = depolarisation;

(Any 4)

(b) +25 - 28mV;

- (c) 0.4 0.6 milliseconds;
- (d) Time taken for release/diffusion of acetylcholine;

Acknowledgements;

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