

# 1.3

## Action potentials

Converting energy into nerve impulses, resting potentials and action potentials

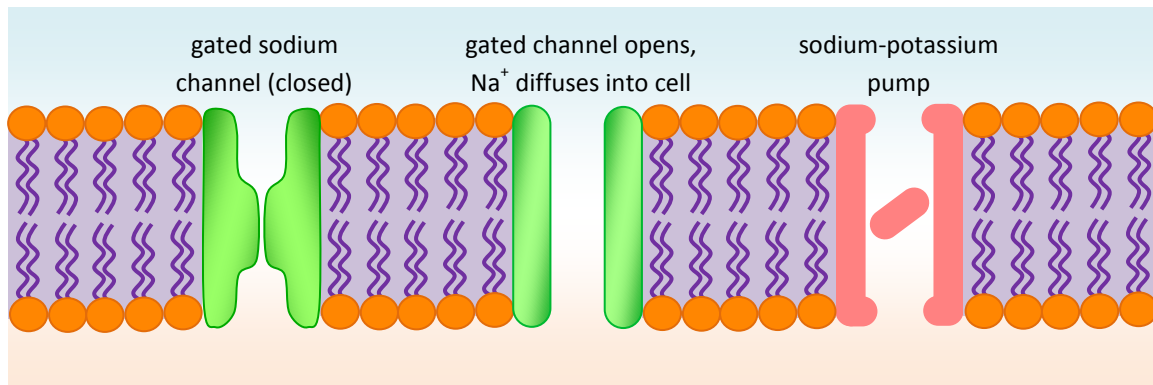
### Sensory receptors

A **receptor** converts an external or internal stimulus into an electrical signal. Sensory receptors detect changes in their surroundings. The table below outlines the main stimuli and the receptors which detect them:

Stimulus	Receptor	Location
sound	auditory receptors (the cilia in the cochlea)	inner ear
temperature	thermoreceptors	skin and hypothalamus
light	rods and cones	retina
taste	chemoreceptors	taste buds
smell	chemoreceptors (olfactory cells)	nose
pressure	Pacinian corpuscles	skin
position/stretch	propioreceptors (stretch receptors)	muscles

A *thermoreceptor* detects changes in temperature. These are found all over the skin. Similarly, *chemoreceptors* in the nose and mouth detect taste and smell (in the nose, these are in olfactory cells lining the inner surface in the nasal cavity). All muscles have *propioreceptors* which are also known as 'stretch receptors' – these detect the change in muscle fibre length. This is how we know the position or stretch of our body (for example, knowing when our arm is stretched out without seeing it). Also in the skin are *Pacinian corpuscles* which are your pressure receptors detecting pressure on the skin.

Neurons have very specialised cell surface membranes filled with many channel proteins. These are specialised to sodium and potassium channel proteins. One type of channel protein, the **sodium-potassium pump** actively transports sodium ions ( $\text{Na}^+$ ) out of the cell, and potassium ions ( $\text{K}^+$ ) into the cell. There are also **voltage-gated channels** which are channel proteins which have a gate which when open allows a certain ion through. Gated channels are specific to ions, so one gated channel will only allow through sodium or potassium ions.



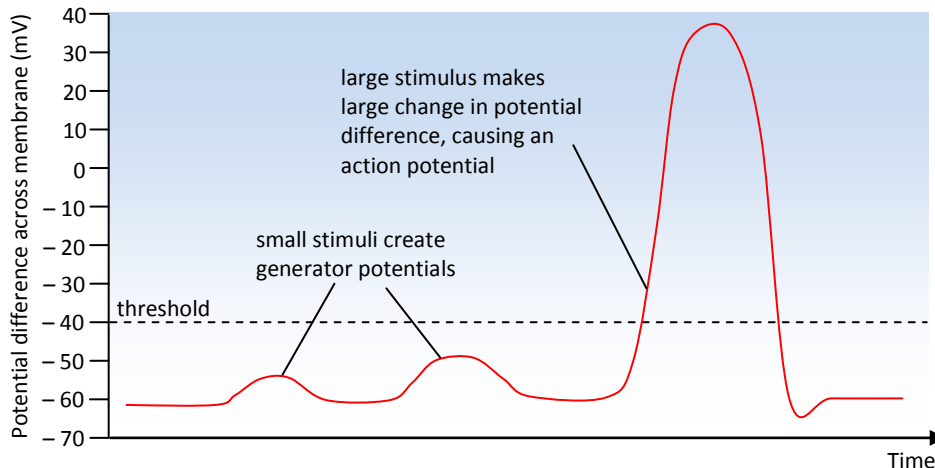
The inside of the membrane is more negatively-charged than the outside, although they both have a negative charge. The membrane is said to be polarised, meaning it has a voltage (potential difference) across it. There is a high concentration of *sodium ions* outside of the cell, and a high concentration of *potassium ions* inside the cell.

Sensory receptors are known as **energy transducers**. This means that they convert energy from one form to another. They detect **stimuli** (energy changes) – whether it be a thermoreceptor detecting a change in thermal energy, or a Pacinian corpuscle detecting a pressure change – and convert this energy into a form of electrical energy, called a **nerve impulse**. It is this stimulus, or energy, which causes the sodium voltage-gated channels to open along the membrane.

When the sodium gated channels open, this causes an influx of sodium ions, as the permeability of the membrane to sodium ions increases. This movement of sodium ions across the membrane (down the concentration gradient, so into the cell) causes a change in charge, so the inside of the cell becomes *less* negative. This is called **depolarisation**. The bigger the energy of the stimulus, the more sodium gated channels open, and so the more permeable the membrane to sodium ions and therefore the more sodium ions enter the cell – resulting in a larger depolarisation.

## Triggering an action potential

Because the number of sodium ions which can enter the cell during the process varies so greatly, sometimes there is only a small depolarisation, where the result is only a slight increase in voltage across the membrane. This small depolarisation is known as a **generator potential**. However, if enough channels open and enough ions enter the cell, and the voltage reaches the threshold value (known as the **threshold potential**) of  $-40\text{mV}$  (millivolts), a full impulse – or **action potential** – is generated. The graph outlines how different amounts of depolarisation can cause either a generator potential or an action potential.



This concept of a stimulus needing to reach the threshold value (of approximately  $-40\text{mV}$ ) is called the **all or nothing response**. This term describes the idea of anything below that voltage not generating an action potential at all, whereas if it does reach that value – a full action potential is generated, which will travel along a nerve cell as a nerve impulse.

## The resting neurone

Of course, a neurone is not constantly transmitting an action potential. Whilst during times of not generating nerve impulses the neurone is said to be 'at rest', it is in fact doing a lot of work to maintain a stable membrane. This state is called a **resting potential**.

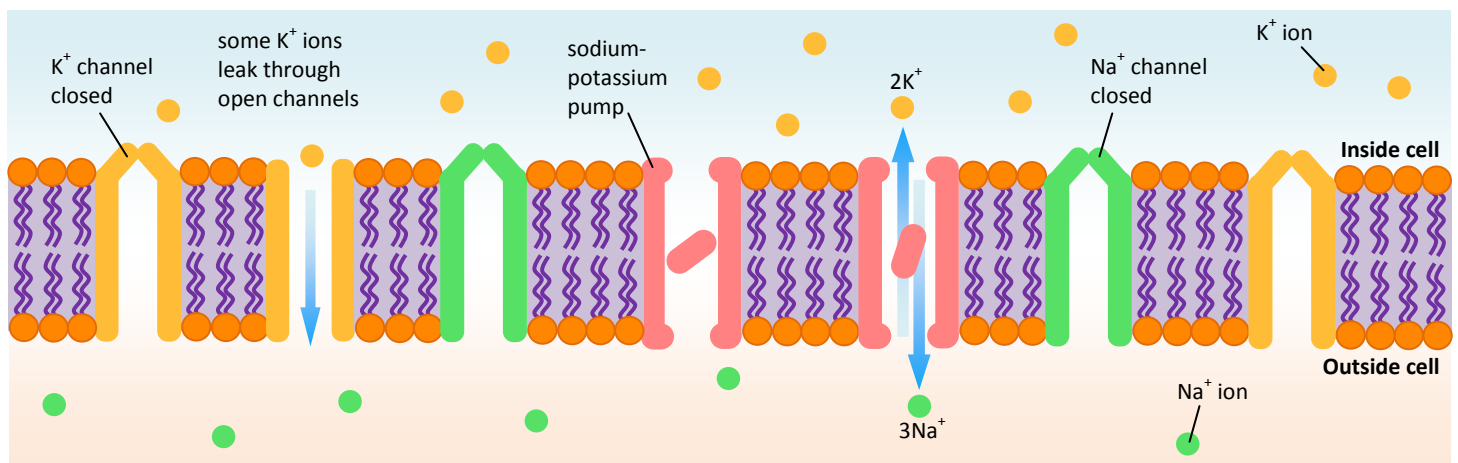
In this state, there are more potassium ions ( $\text{K}^+$ ) inside the cell and more sodium ions ( $\text{Na}^+$ ) outside the cell. Using ATP (reducing it to ADP in the process), the sodium-potassium pump can actively transport both ion types across the membrane. It pumps three sodium ions out of the cell for every two potassium ions it pumps into the cell. This is because the membrane is more permeable to potassium ions and many diffuse back out of the cell. The neural membrane is often described as a "leaky" membrane as it has this characteristic. The inside of the cell has a negative overall charge compared to the exterior, as it contains many other *anions* (negative ions) in the cytoplasm. The resting potential maintains a voltage of around  $-60\text{mV}$ .

The **threshold potential** is the potential difference across a membrane of around  $-40\text{mV}$  which if met or exceeded will trigger an action potential

A **generator potential** is caused by slight depolarisations in the membrane from small stimuli

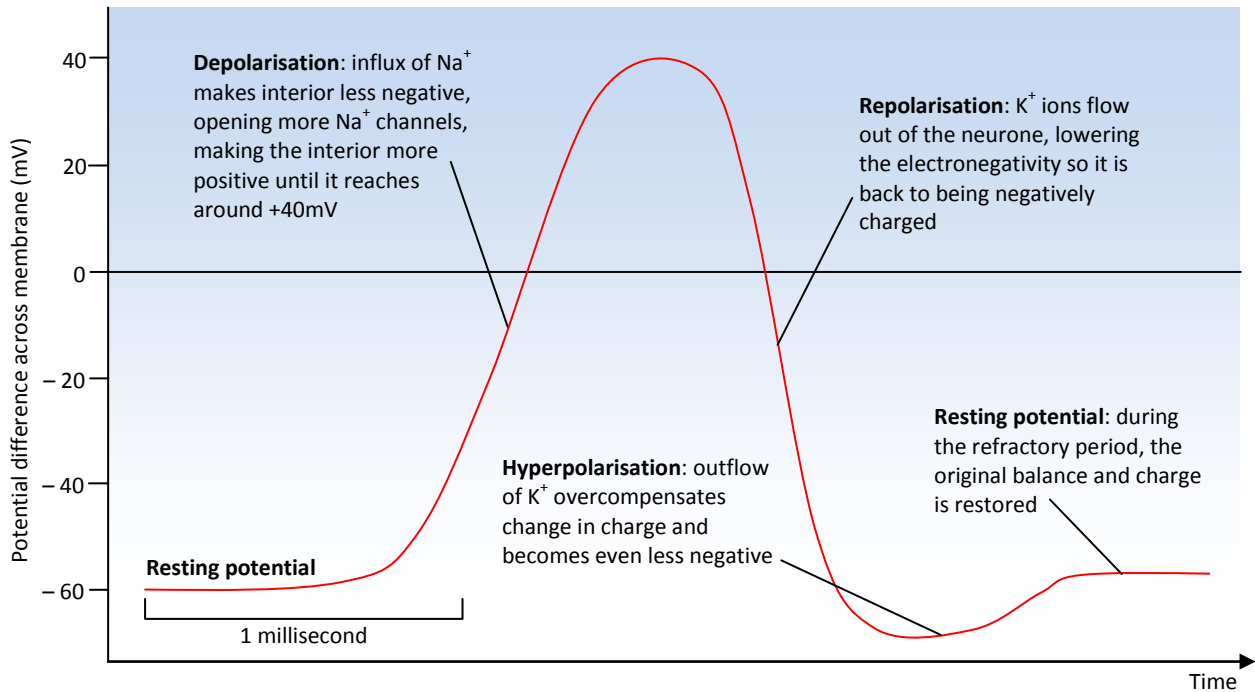
An **action potential** is a depolarisation of the surface membrane where the inside is more positive than the outside, transmitted along a neurone, reaching a high point of  $+40\text{mV}$

The **resting potential** is the state a neurone is in when it is not transmitting an action potential, but is still actively transporting sodium and potassium ions to maintain the potential difference of around  $-60\text{mV}$



## Events of an action potential

Now that you understand the behaviour of a neural membrane at its resting state and you know what *causes* an action potential to be generated, you should be able to understand the events of a single action potential. Action potentials are triggered by ionic movements over the membrane which bring about changes in electronegativity, and all of the events which make up one action potential are also due to ionic movements. The sodium-potassium pump actively transports sodium ions out of the cell and potassium ions into the cell (against their respective concentration gradients), and when the voltage-gated sodium and potassium channels are open, those ions can flow through those channels also.



- 1 The membrane begins in its resting state – a *resting potential* – at around -60mV
- 2 As some sodium ion channels open, the permeability of the membrane to  $\text{Na}^+$  is increased, so these ions move into the cell – which increases the electronegative charge (making it less negative)
- 3 As the charge increases, the voltage-gated  $\text{Na}^+$  channels open, which further increases sodium permeability, so more sodium ions diffuse into the cell – further increasing the charge – this process is called **depolarisation**, and the overall charge of the cell should increase so approximately +40mV (only if an action potential is triggered by reaching the initial threshold potential of the neurone)
- 4 At the peak of the action potential, around +40mV, the sodium channels close and the potassium channels open, which causes  $\text{K}^+$  ions to flow out of the cell – bringing the charge inside the cell back down – this is **repolarisation**
- 5 This outflow of potassium ions overshoots the potential difference slightly to just below -70mV, which overcompensates the change in electronegativity – this is called **hyperpolarisation** – and then  $\text{K}^+$  channels close
- 6 During what is known as the *refractory period*, the cell restores its original balance of ions and continues to be in a resting potential state until stimulated by the next action potential

## The refractory period

After *hyperpolarisation*, the ionic movements during the action potential have left the sodium ions inside the cell and the potassium ions out of the cell – which is not where they need to be for a resting potential. So during the **refractory period** the cell is given a chance to recover from the action potential and restore the correct balance of ions on either side of the membrane. It is the role of the sodium-potassium pump to actively transport said ions across the membrane.

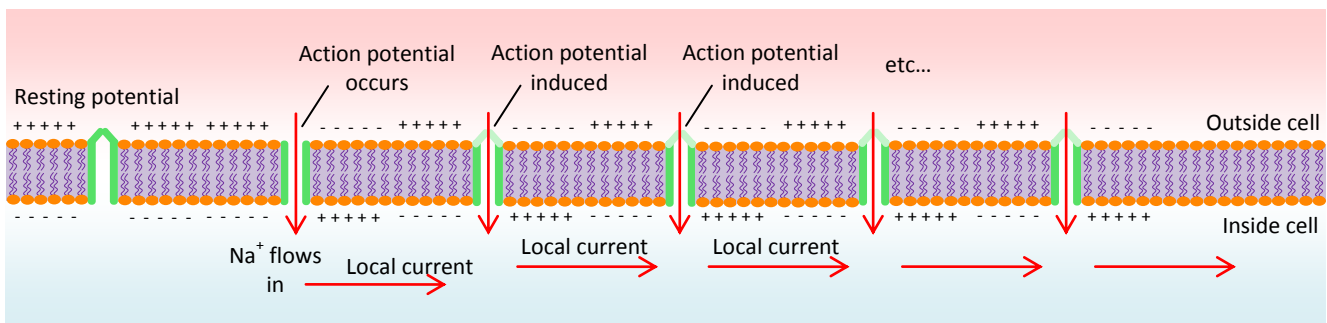
The refractory period is beneficial because it means that (given the ionic concentrations both inside and outside the cell) another action potential cannot be stimulated in the cell membrane so soon after the first – it needs to have this recovery time. A second role of this period is that it ensures that action potentials are being transmitted in one single direction.

Of course, an action potential is no use in one place. It needs to be transmitted along a neurone and between different neurones in order to transmit a signal effectively – and this next section will explain how this process takes place.

## Transmission of an action potential

The resting neurone has a stable balance of sodium and potassium ions, which is created by the sodium-potassium pump. The ions are sorted so that there are mainly sodium ions outside the cell and mainly potassium ions inside the cell, also so that the inside of the cell is more negatively charged than the outside (the cell interior usually has a stable charge of  $-60\text{mV}$ ). When an action potential arrives, or is induced, sodium channels open, which causes an influx of sodium ions into the cell, depolarising that section of the membrane. This actually disrupts the careful balance of ions in surrounding areas so that **local currents** are created in the cytoplasm, causing sodium channels further along the membrane to open.

A *local current* is a movement of charged particles within a cell along a concentration gradient. In this case, when sodium ions move into the cell as an action potential occurs, at the location in the membrane where the sodium voltage-gated channels are, the concentration of  $\text{Na}^+$  increases. This creates a **concentration gradient** with adjacent sections of the membrane, and the local current occurs where sodium ions then diffuse sideways along the membrane, down to areas of a lower concentration of these ions.



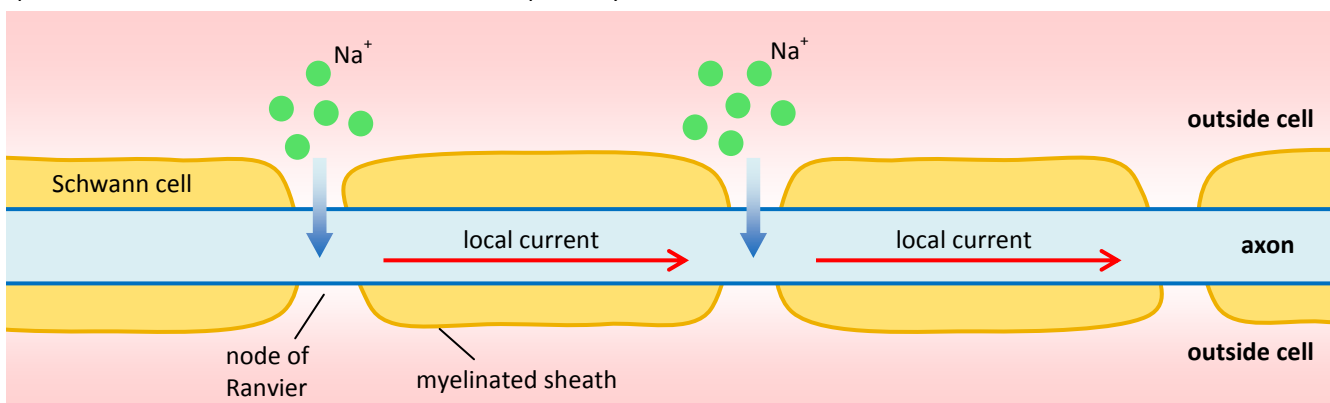
The diagram shows how this process can transmit a nerve impulse along the entire membrane of a neurone:

- an action potential is generated at one point in the membrane as sodium voltage-gated channels open and  $\text{Na}^+$  ions flow into the cell, depolarising the membrane at that point
- this causes the cell exterior at that area to become more negative due to the loss of positively-charged  $\text{Na}^+$
- as the ions enter the cell, the concentration at that point increases, creating a concentration gradient to the adjacent areas which have a low concentration of sodium ions, causing the ions to diffuse along to those areas
- this in turn depolarises slightly that part of the membrane, causing the voltage-gated sodium channels to open in that area, so even more sodium ions diffuse into the cell, depolarising the membrane further – inducing an action potential
- this leads to another concentration gradient of sodium ions to the next area along the membrane and the process repeats – transmitting the impulse all along the membrane

Although there are quite a few steps involved in the process, this all happens in mere fractions of a second. It is also important to note that this is an example of **positive feedback**: the arrival of sodium ions depolarises the membrane, causing channels to open so more sodium ions diffuse into the cell further depolarising the membrane, causing more ions to move down the membrane, causing more channels to open, etc.

## The myelinated sheath

Neurones may have areas of their axons coated in an insulated layer of fatty material. This is called the **myelin sheath**. Specialised cells known as Schwann cells make up the myelin sheath.



The myelinated sheath won't allow sodium or potassium ions to diffuse through it, and so the transmission of an action potential along the axon happens in a 'jumping action'. The areas between the myelination, called **nodes of Ranvier**, will receive the influx of sodium ions as per usual, but the *local current* is elongated so that the ions diffuse from one node of Ranvier along to the next, inducing another action potential at the following node. This gives the impression of an action potential 'jumping' from one node of Ranvier to the next. Neurones which have a myelinated axon transmit the nerve impulse in this way, which is called **saltatory conduction** (meaning to jump).

Neurones with these myelinated sheaths have an obvious benefit in that because an action potential can only be induced at the gaps between the myelinated areas, the transmission of the action potential will be sped up, as it goes further in less time. Myelinated neurones conduct action potentials much more quickly than non-myelinated neurones.

### Sensory, relay and motor neurones

Once an action potential is induced – it needs to be transmitted, not only along the neurone, but around the body. There are a variety of forms of neurone which have different roles, including **sensory neurones** (which take the action potential from the sensory *receptor* to the central nervous system), **relay neurones** (which carry the action potential from a sensory neurone to a motor neurone) and **motor neurones** (which receive a signal from relay neurones and carry it to an *effector* such as a muscle).