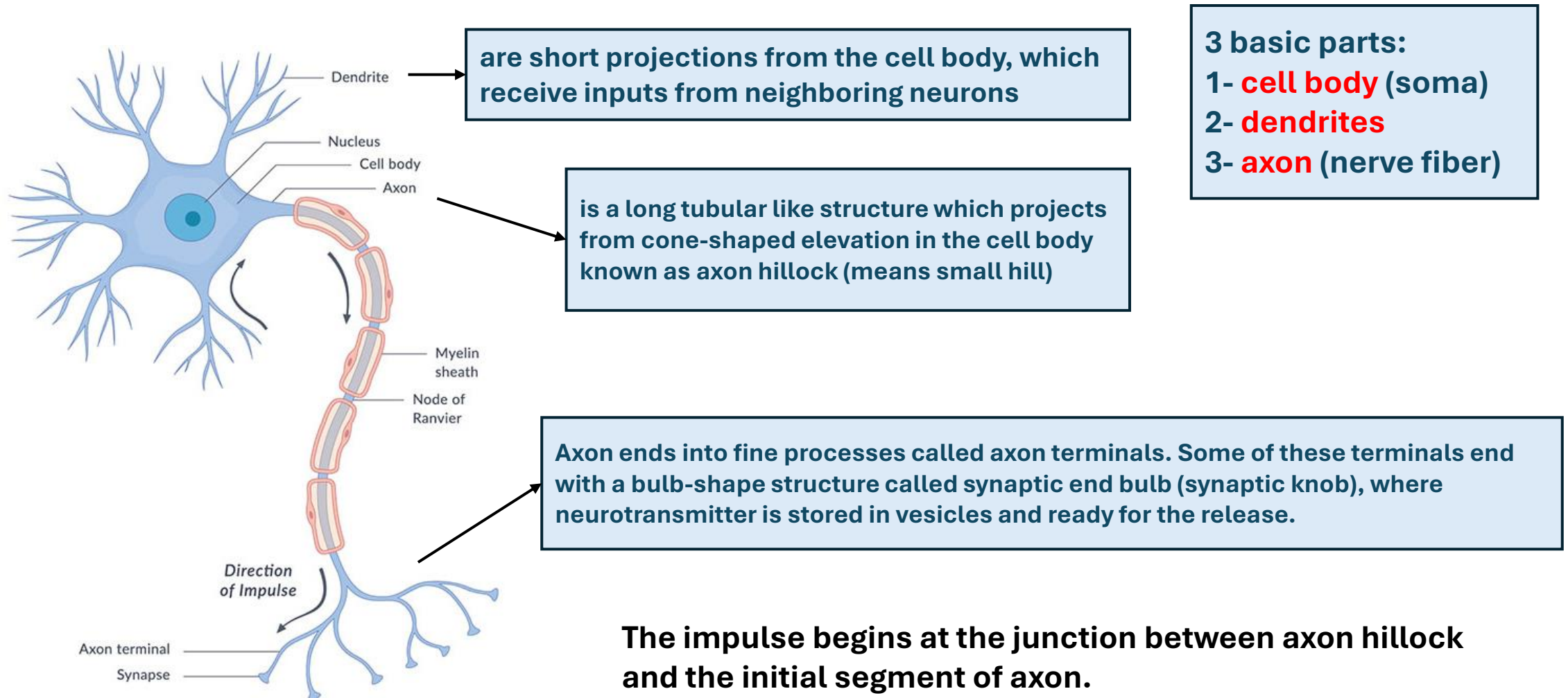


Membrane potential

Physiology

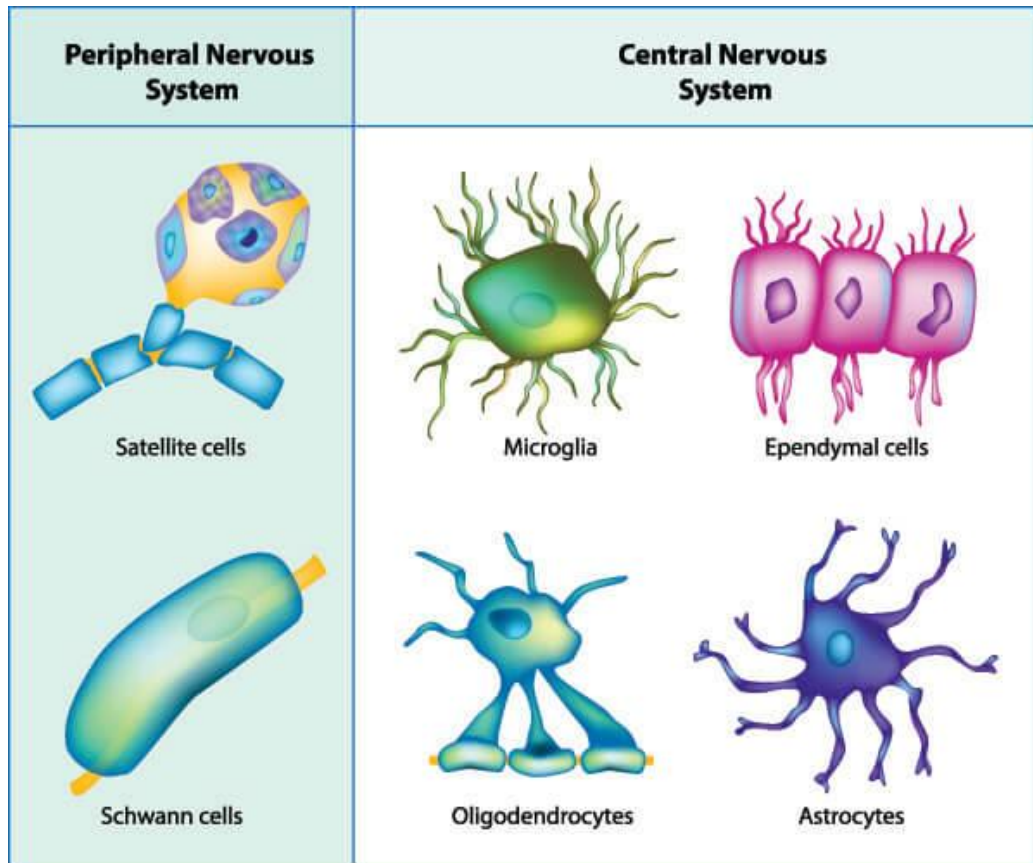
By Abdallah Al-Saraireh

The functional and structural unit of the nervous system is called (Neuron OR nerve cell)



Supportive cells (NEUROGLIA)

Many types of supportive cells around neurons have been described (at least 6)



- **Maintenance of the neural environment:** They take up excess K^+ and neurotransmitters from the interstitial fluid around the neurons.

- **Protection and survival:** They synthesize and release neurotrophic factors that maintain the survival of neurons.

In the Gastrointestinal (GI) Tract: Specific glial cells that are like the **astrocytes** found in the CNS

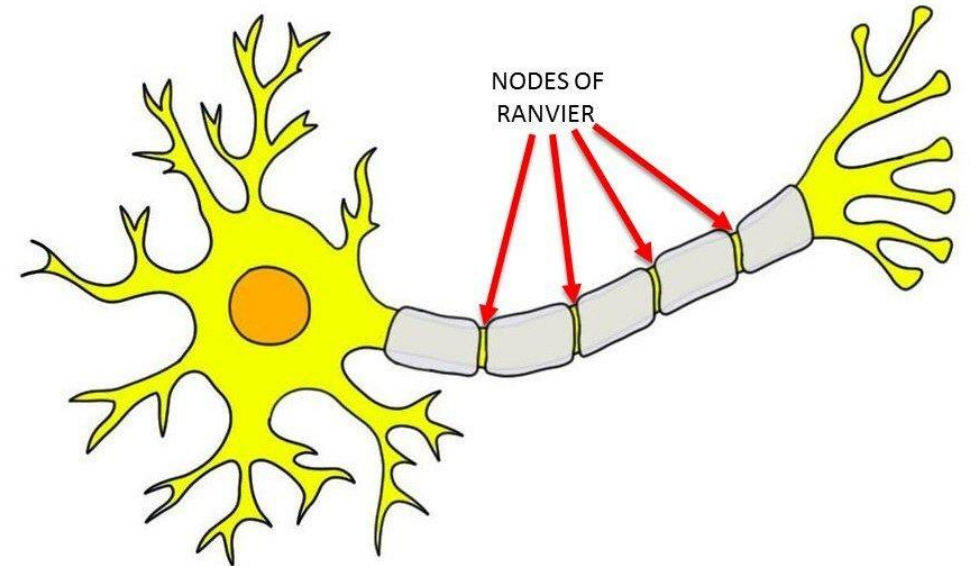
Specialized supportive cells

are responsible for *myelination*

In the **CNS** (central nervous system) are **Oligodendrocytes**

In the **PNS** (peripheral nervous system) are **Schwan cells**

- These cells wrap around axon segments and secrete myelin sheath (a protein lipid complex that insulates nerve fiber). There are **gaps** in myelin sheaths known as **nodes of Ranvier**, which appear at **intervals** along axon. These gaps are used for **transmission** of impulse along myelinated nerve fiber.



What is the primary function of the Nernst equation in membrane physiology?

The Nernst equation is used to calculate the **equilibrium potential** for any **univalent ion** at normal temperature. It determines the point at which the electrical potential difference across a membrane completely opposes the net diffusion of that specific ion despite a remaining concentration gradient.

Simply put, it is the exact electrical charge needed inside the cell to **stop** a specific ion from moving across the membrane

At this specific voltage, the electrical force perfectly balances the concentration gradient, so the net movement of that ion becomes **zero**

$$E \text{ (mV)} = - 61.\log (C_i/C_o)$$

E = equilibrium potential for a univalent ion

C_i = concentration inside the cell.

C_o = concentration outside the cell.

Goldman-Hodgkin-Katz equation

When more ions are involved in creating the potential, we can calculate the potential according to Goldman-Hodgkin-Katz equation

$$E_m = \frac{RT}{F} \ln \left(\frac{P_{Na^+} [Na^+]_o + P_{K^+} [K^+]_o + P_{Cl^-} [Cl^-]_i}{P_{Na^+} [Na^+]_i + P_{K^+} [K^+]_i + P_{Cl^-} [Cl^-]_o} \right)$$

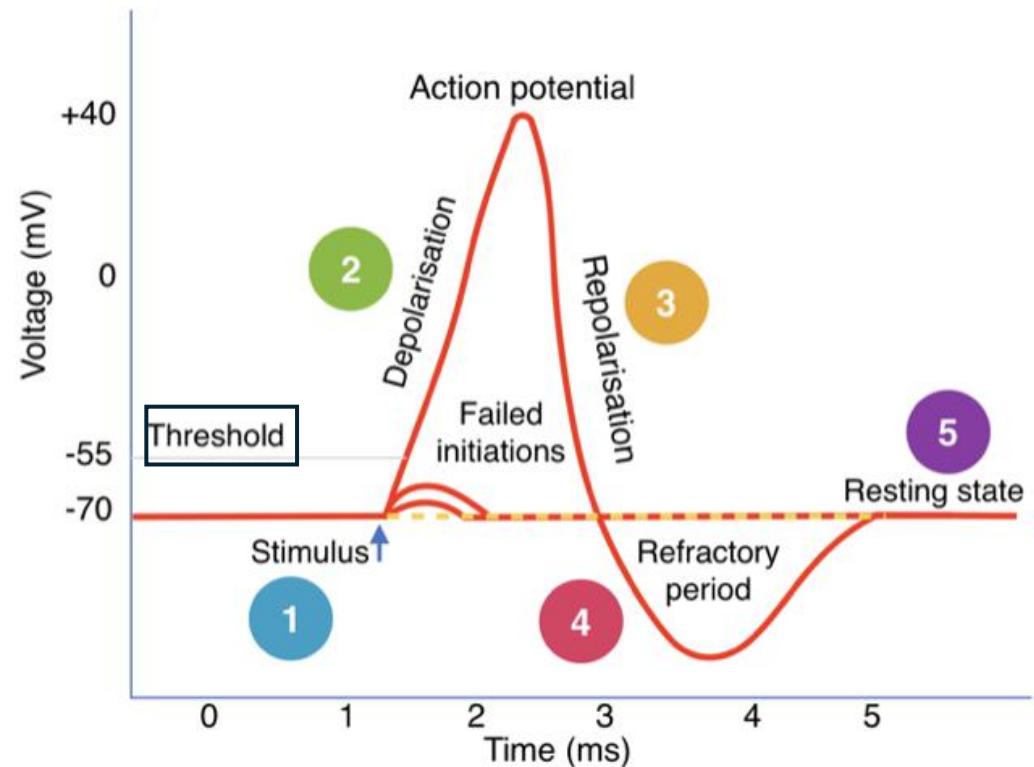
P = permeability of the membrane to that ion

In this equation, Goldman and his colleagues considered that these ions are mostly involved in the development of membrane potential.

According to this equation, the permeability of the membrane to an ion is very important in determining the membrane potential.

Explain the "None or All Principle" as it relates to excitable cells.

If a stimulus is strong enough to depolarize the membrane to its **threshold**, it triggers a full firing stage (action potential). If the threshold is **not achieved**, the membrane **returns** to its resting level **without** an action potential, meaning the response is either full or non-existent.



None or All Principle

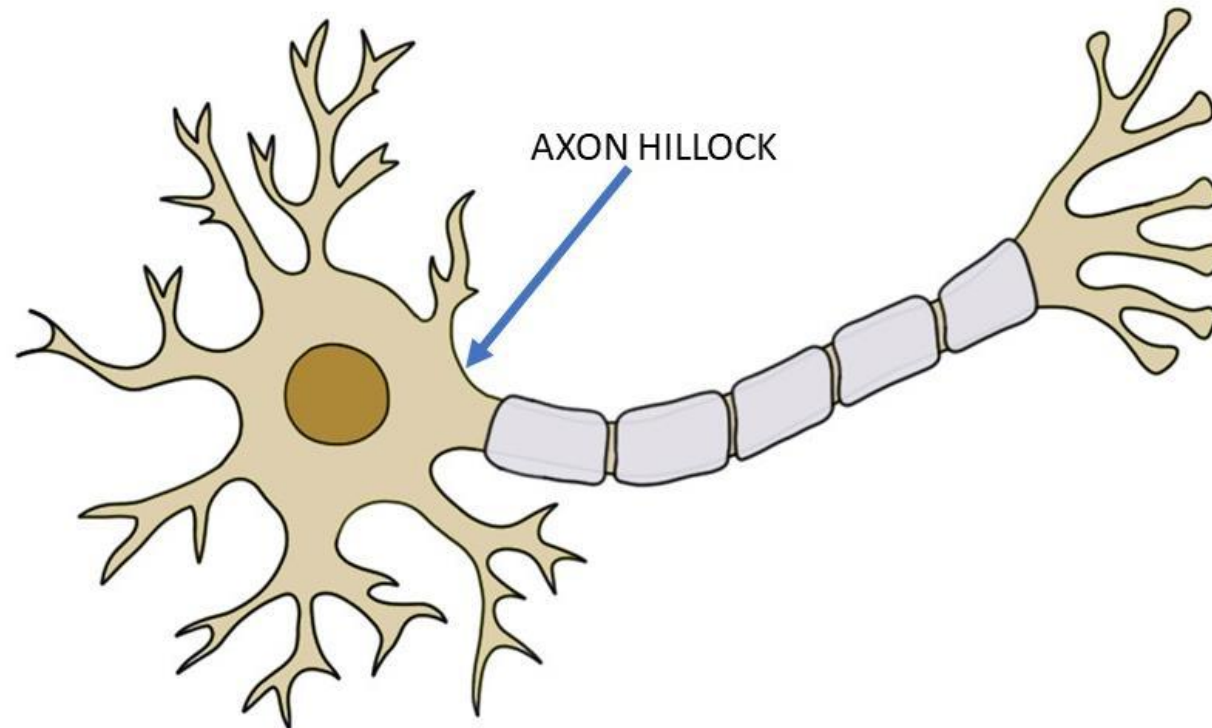
If depolarization in the membrane **has not reached threshold**, the membrane will not enter firing stage, and instead, the potential returns to its resting level. Therefore, the response in the membrane will be either by an action potential when threshold is achieved or no appearance of an action potential when the membrane potential has not reached threshold. For that reason, induction of an action potential in excitable cells follows the **NONE OR ALL PRINCIPLE**.

Action Potential

A rapid change in membrane potential involving depolarization and repolarization that propagates along the membrane of an excitable cell.

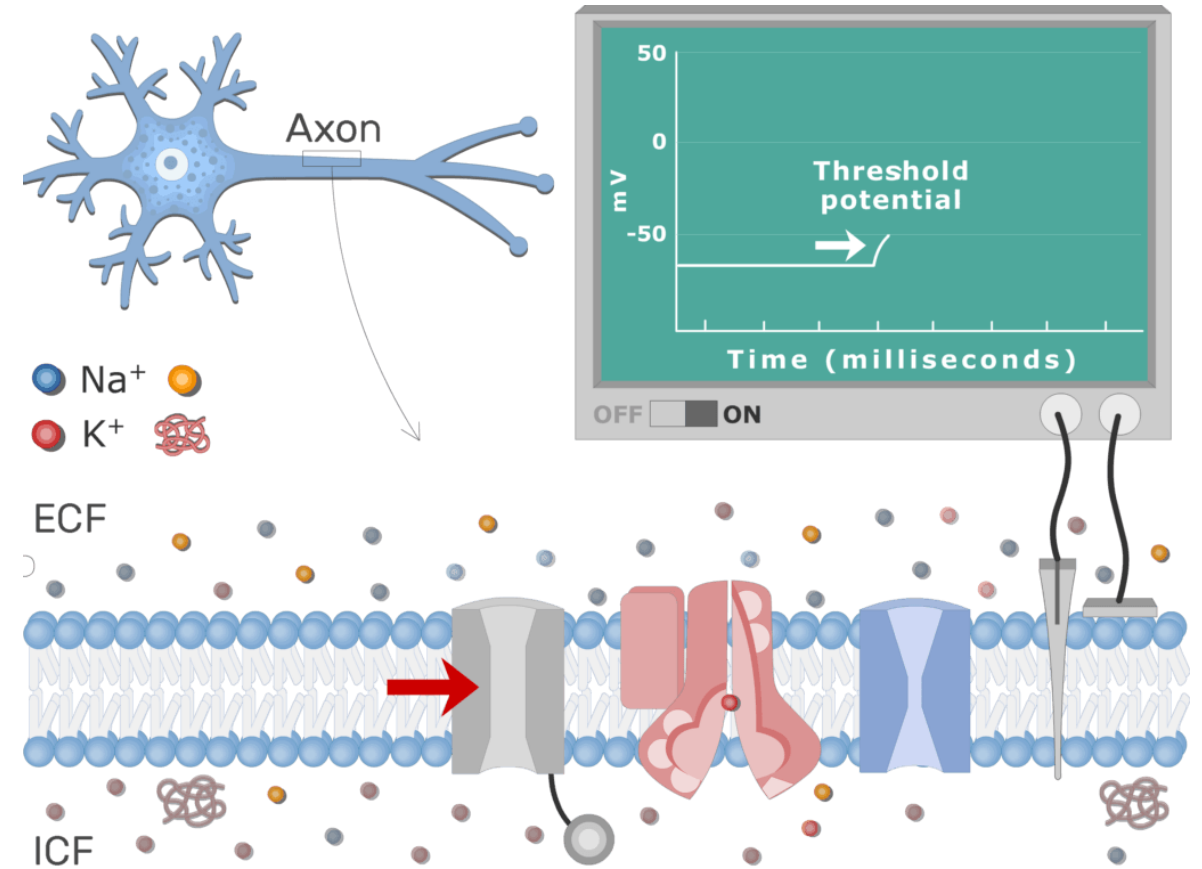
Axon Hillock

The cone-shaped elevation of the cell body from which the axon projects and where the neural impulse typically begins.



Threshold

The critical level of depolarization required to trigger the firing stage of an action potential.

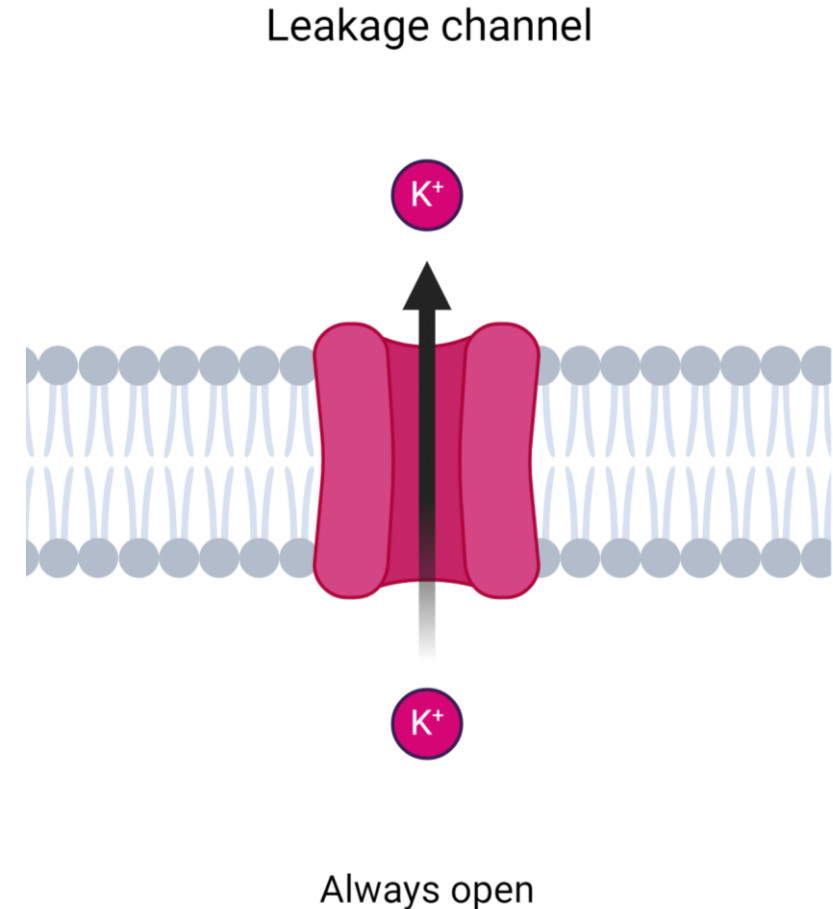


Influx: The movement of ions or molecules **into** a cell.

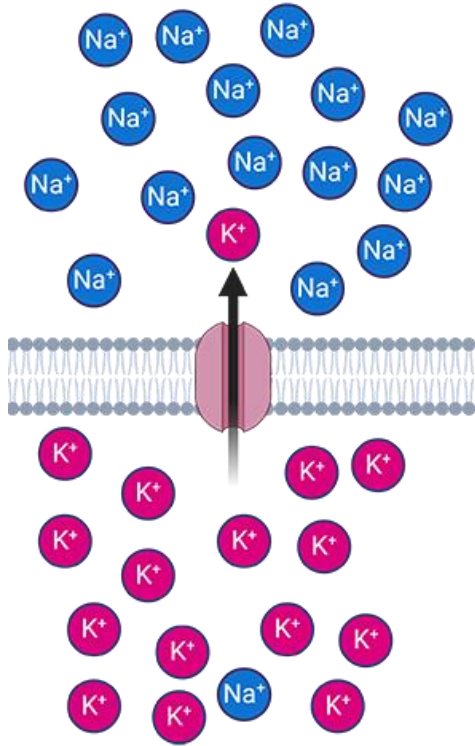
Efflux: The movement of substances **out** of a cell.

leak channels

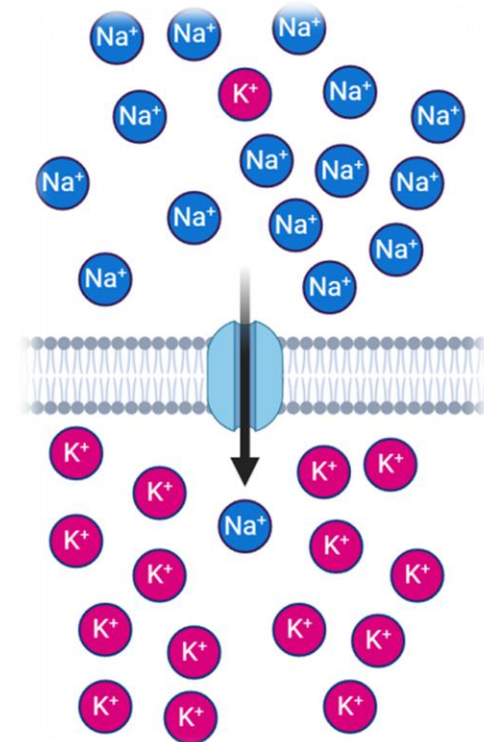
are **passive** ion channels that remain "**always open**" allowing specific ions to move across the cell membrane following their **electrochemical gradients**.



leak channels



The concentration of potassium (K+) is almost always higher inside the neuron than outside. If we open a potassium channel, potassium goes from where it's at higher concentration to where it's at lower concentration: it goes from inside to outside the neuron.

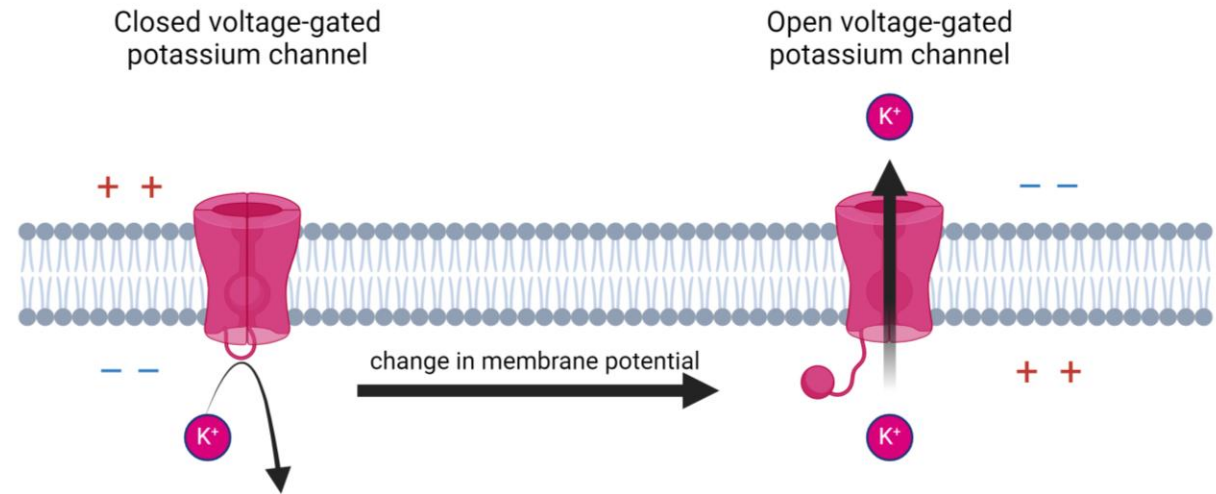


The concentration of sodium (Na+) is almost always higher outside the neuron than inside. If we open a sodium channel, sodium goes from where it's at higher concentration to where it's at lower concentration: it goes from outside to inside the neuron.

At rest, a cell membrane is significantly **more permeable** to potassium (K^+) than to sodium (Na^+) because it **contains many more leak channels.**

Voltage-gated K⁺ channels

A voltage-gated potassium channel is a specialized **transmembrane protein** that **opens** or **closes** in response to changes in the cell's electrical membrane potential, specifically allowing the **efflux** of potassium ions.

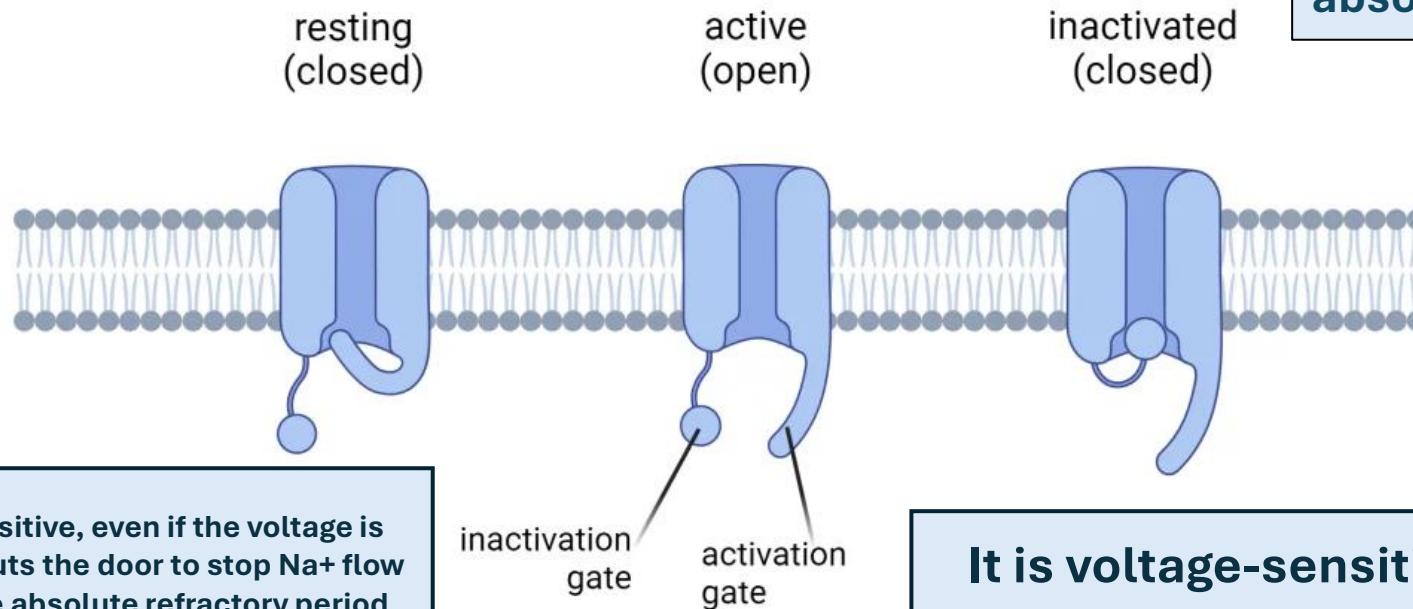


Voltage-gated Na⁺ channels

This is the **default state** at the **resting** membrane potential (typically). The channel is "primed" and ready to open if a threshold stimulus is reached.

This state is triggered by membrane depolarization to a threshold level (around -55 mV). It lasts for less than 1 millisecond.

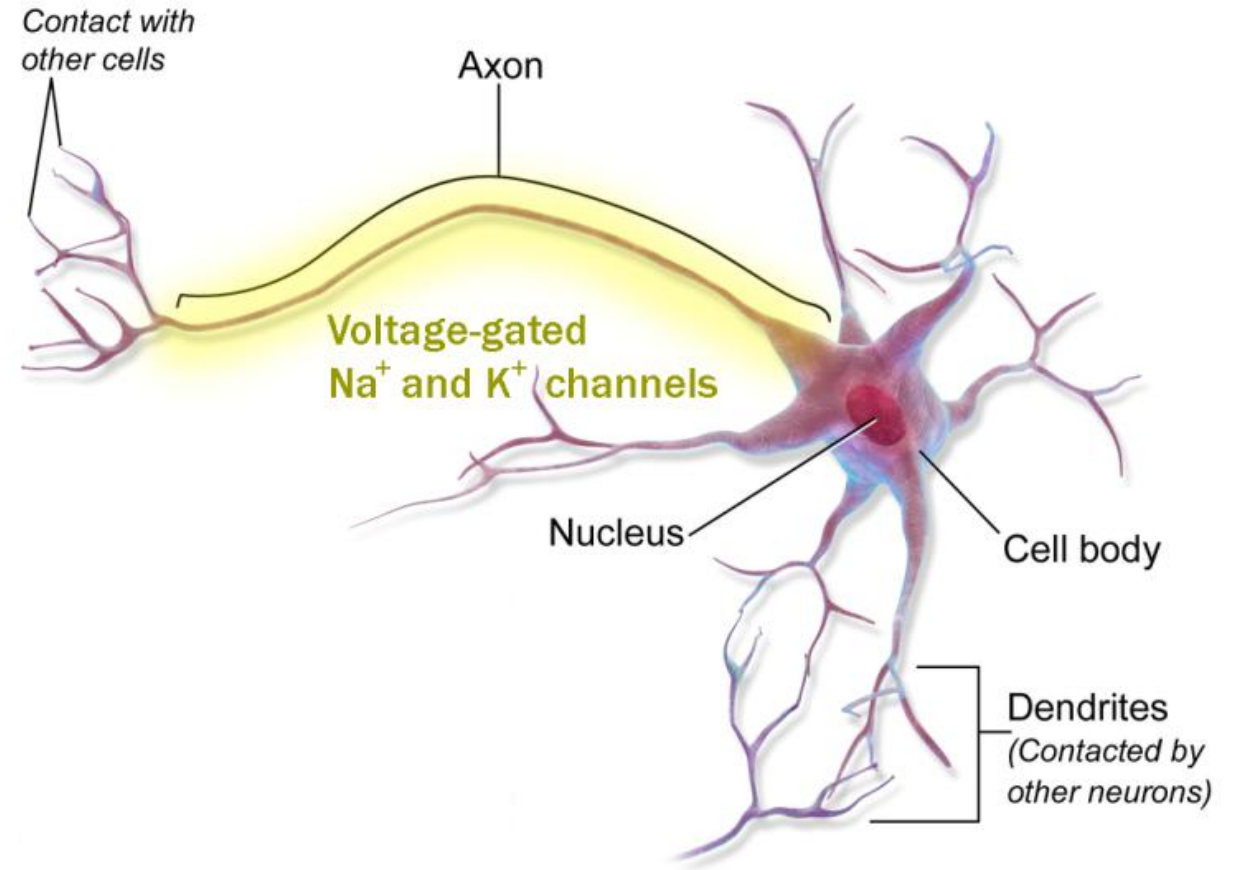
This occurs at the peak of the action potential (around +30 mV). The channel cannot be reopened by further depolarization until it "re-primed" back to the resting state. This state is responsible for the absolute refractory period.



It is time-sensitive, even if the voltage is still high, it shuts the door to stop Na⁺ flow and starts the absolute refractory period

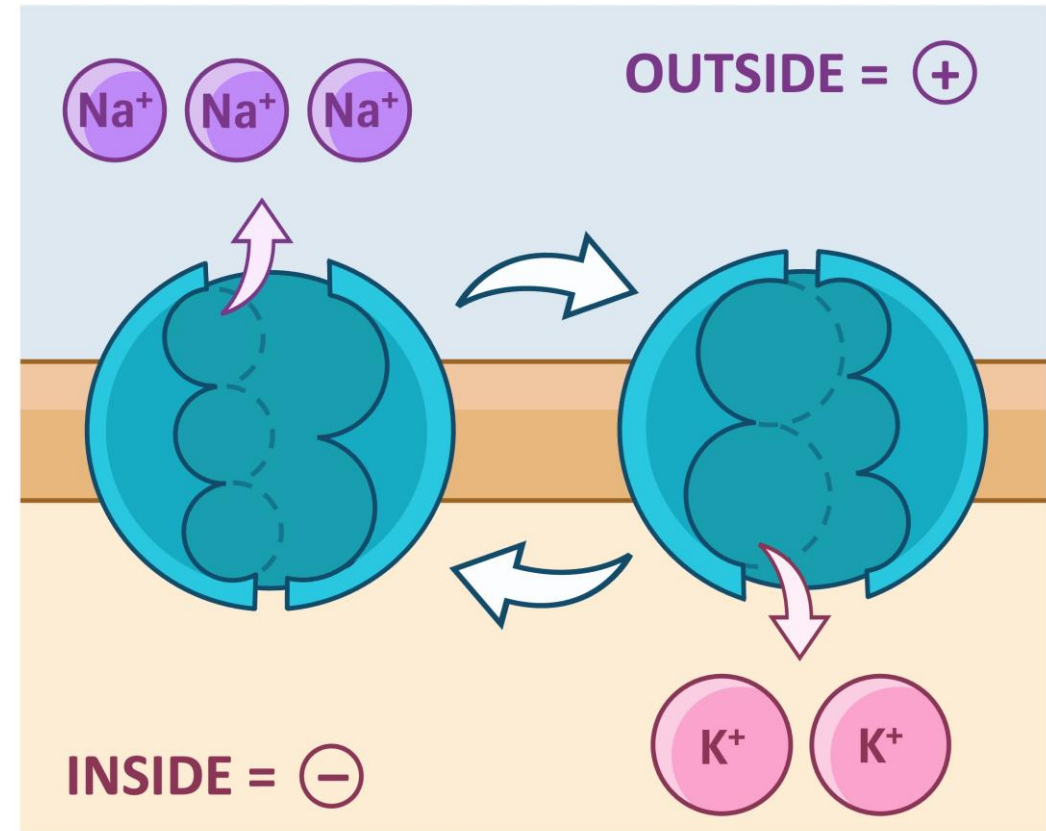
It is voltage-sensitive

Voltage-gated channels are only found in a **limited region** of the neuron, typically on the **surface of the axon**, as shown by the yellow shading here.



The sodium-potassium pump (Na⁺/K⁺- ATPase)

It acts as a primary **active** transporter, meaning it uses energy from **ATP** to move ions **against** their concentration gradients.



Resting membrane potential

In **excitable cells** the membrane potential is **not constant**. When the cell is **stimulated**; the membrane potential is **changing**. These changes in membrane potential are **due to changes in permeability of plasma membrane to different ions**. For example, when a neuron is stimulated, this will result in **increased permeability to Na⁺**. This will bring the membrane potential closely to (E_{Na}). The recorded membrane potential for a cell under resting conditions when no stimulus is involved is known as **resting membrane potential**. For neurons, the recorded resting membrane potential is about **(-90 mV)**. This represents a potential difference between the inside to the outside when the neuron is **not active**.

Origin of resting membrane potential

Contribution of K⁺ diffusion:

As mentioned earlier, if the membrane is permeable only for K⁺ the calculated E_{K⁺} is about (-94mV).

$$C_{oK^+} = 4\text{meq/l} , C_{iK^+} = 140\text{meq/l}$$

$$E_{K^+} = -61 \cdot \log 140/4 = -94\text{mV}$$

Which is not far from the recorded membrane potential but not exactly.

Origin of resting membrane potential

The contribution of Na⁺ diffusion:

Membrane is also permeable to Na⁺. The permeability of the plasma membrane for Na⁺ is much less than that of K⁺. If the membrane is permeable only to Na⁺, the calculated $E_{Na^+} = +61\text{mV}$.

..... ($C_{oNa^+} = 142\text{meq/l}$, $C_{iNa^+} = 14\text{meq/l}$).

Because of the permeability of the membrane for the two ions, the E would be between (-94mV and +61mV). The calculated E for the two ions is -86mV, which is not far from the E_{K^+} because of the higher permeability of membrane for K⁺ than for Na⁺ (100 times more).

So the Na⁺ contribution in resting potential is by bringing the membrane potential to a lower value than the calculated E_{K^+} .

Origin of resting membrane potential

Contribution of Na⁺ - K⁺ pump:

As mentioned earlier, this pump is electrogenic. It moves more positive charges outside the cell (3 for 2). This will induce loss of positive charges from the cell and bring the membrane potential to a higher negativity (about -4mV additional negativity).

Therefore all these factors, during **rest**, will give a net membrane potential of -90mV (called **Resting Membrane Potential**).

Action potential

- As we have seen, the plasma membrane is **polarized** (has ability to separate opposite charges) during resting state.
- When the membrane potential decreases (becomes less negative), the membrane is in **depolarization stage**.
- While the change in membrane potential in opposite direction (becomes more negative than resting potential) is known as **hyperpolarization**.

Action potential

- When a cell is depolarizing, it reaches a **maximum** according to stimulus, then the membrane potential returns to its resting state. The phase of returning from depolarized state to resting state is known as **repolarization**.

Recordings of action potential

Recording of **monophasic action potential** is by placing one electrode **inside** the cell and the other electrode **outside** the cell.

While a different configuration of an action potential can be obtained by placing **the two electrodes outside** the cell membrane. The later recording is known as **biphasic action potential**. Two waves are obtained in the recording of biphasic action potential, the first represents depolarization, and the second is in the reverse direction of the first and represents repolarization.

Action potential

- **To induce a change, a stimulus must be applied to change activity of channels at the membrane.**
- **Any increase in permeability of membrane to Na^+ will result in diffusion of (+) charges inward. This event will decrease the membrane potential (becomes less negative). And conversely any increase in K^+ diffusion (movement outward) will result in an increase in membrane potential (becomes more negative).**
- **Activation of Na^+ channels will induce depolarization, while activation of K^+ channels will increase the potential difference across membrane.**

Action potential and the role of Na⁺ channels

On the membrane, most Na⁺ channels during resting state are **inactive** (closed). According to channel type, these channels can be activated by a **chemical** stimulus (in case of chemical gated channels), **electrical** stimulus (in case of voltage gated channels), or **mechanical** stimulus.

In the case of **chemical gated channels**, binding of ligand to its receptor will induce activation of chemical gated Na⁺ channels. Once activated, the membrane potential will decrease (becomes less negative). Which means that the membrane depolarizes

The voltage changes in the membrane will cause the other type of channels (Na⁺ voltage gated channels) to be activated.

Activation of these channels will cause more changes in membrane potential (more depolarization). More and more depolarization will occur in the membrane by a **positive feed back mechanism**.

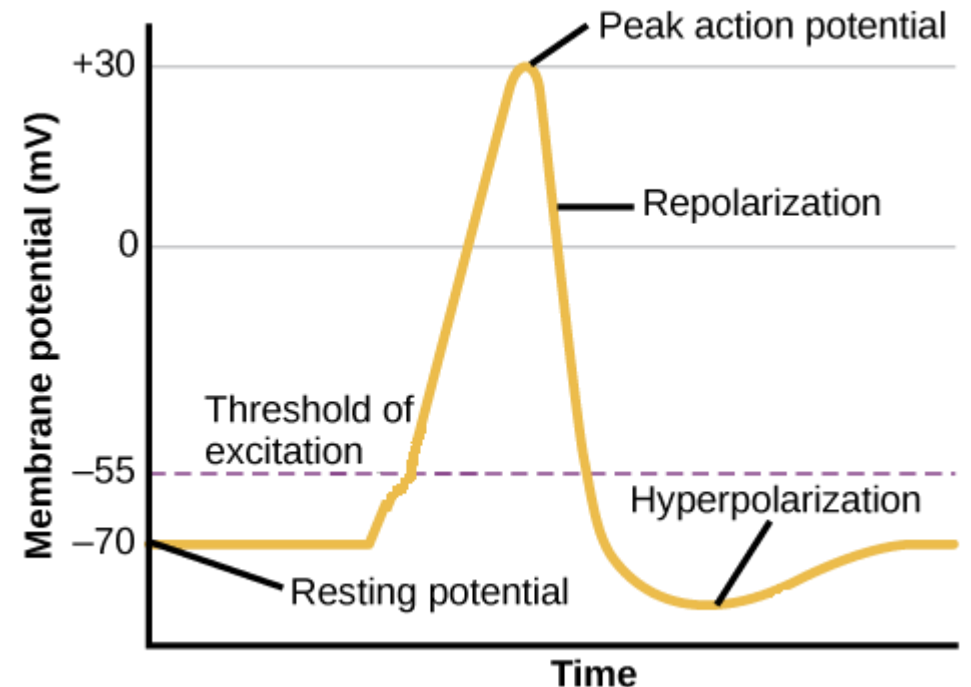
Action potential and the role of Na⁺ channels

If we reach a point at which **most** voltage gated Na⁺ channels are activated, this will cause a **sudden increase** in Na⁺ permeability.

This increase in Na⁺ permeability will even **reverse** the membrane potential (becomes positive inside and negative outside) (this is known as the **overshoot** in the action potential), because Na⁺ is trying to approach its equilibrium potential (E_{Na})

At this point, the membrane has reached maximal changes in membrane potential (**a peak of an action potential**).

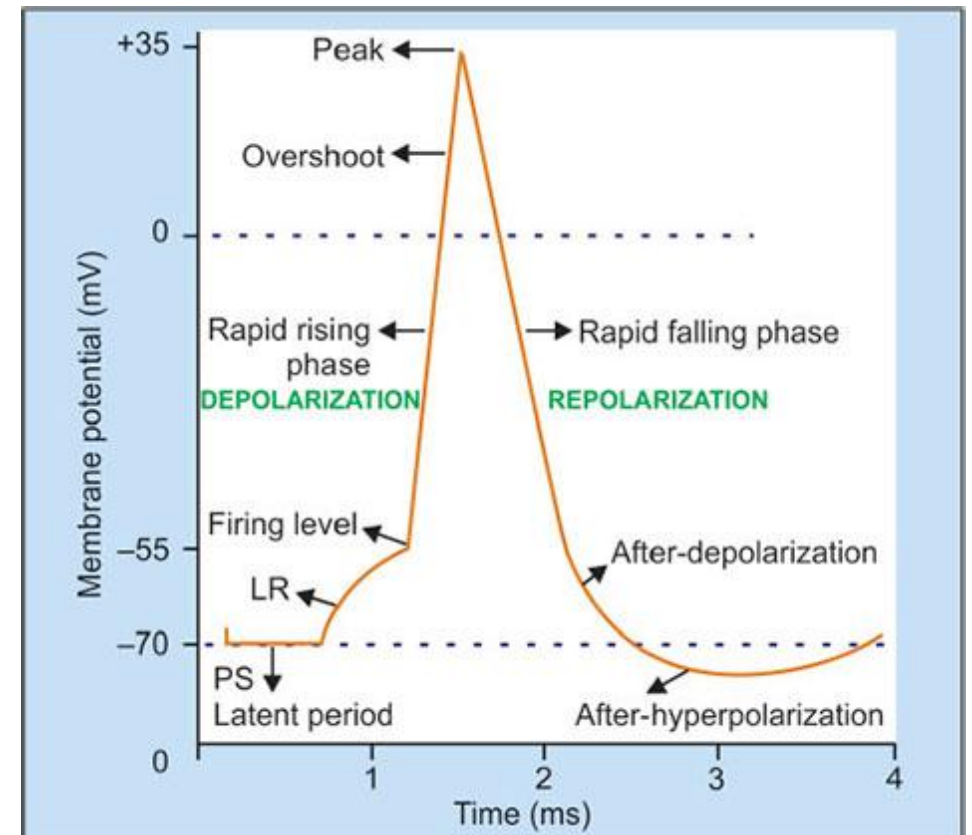
As we have seen, during depolarization there is a point at which a sudden increase in Na⁺ influx which induces rapid and maximal change in membrane potential. This point is known as the **threshold** of an action potential.



Action potential and the role of Na⁺ channels

The rapid change in membrane potential during the raising phase of an action potential is known as **firing stage**.

When a stimulus causes a depolarization that brings the membrane potential to the threshold, the membrane will respond by the **firing stage of an action potential**.



Action potential and the role of Na⁺ channels

The voltage changes in membrane potential **not only activate voltage dependent Na⁺ channels**, but also inactivate these channels at **certain potential difference**. This inactivation appears because channels have changed their state from opened channels to closed channels due to voltage changes. The closing event of Na⁺ channels does not make these channels as the only responsible for bringing membrane potential to its resting level. But also, activation of voltage dependent K⁺ channels is the main player in returning the membrane potential to its resting level.

Action potential and K⁺ channels

Although there is some leakage of K⁺ during resting state, which maintains the resting membrane potential close to (E_{K^+}), depolarization causes activation of voltage gated K⁺ channels. The activation of these channels is **much slower** than activation of Na⁺ channels. This results in a delay in the maximal activation of K⁺ channels.

The delayed activation of K⁺ channels combined with inactivation of Na⁺ channels will result in a rapid returning of the membrane potential to its resting level, causing the **falling phase** in the action potential.

The membrane potential may go **for a while** to more negative potential than during resting potential, which is known as positive afterpotential (**after hyperpolarization**).

Followed by full recovery in the membrane potential (returns completely to its **resting level**). The positive after potential is probably due to an **excess in K⁺ efflux**, which causes more deficit of positive ions inside the cell.

Na⁺ -K⁺ pump and action potential

This pump has **no role** in the electrical activity that are taking place **during action potential**. But it plays an important role in **restoring** ionic composition that has been altered during action potential. This role is important in maintaining the ionic composition of the intra- and the extracellular fluids.

Action potential and Ca^{++}

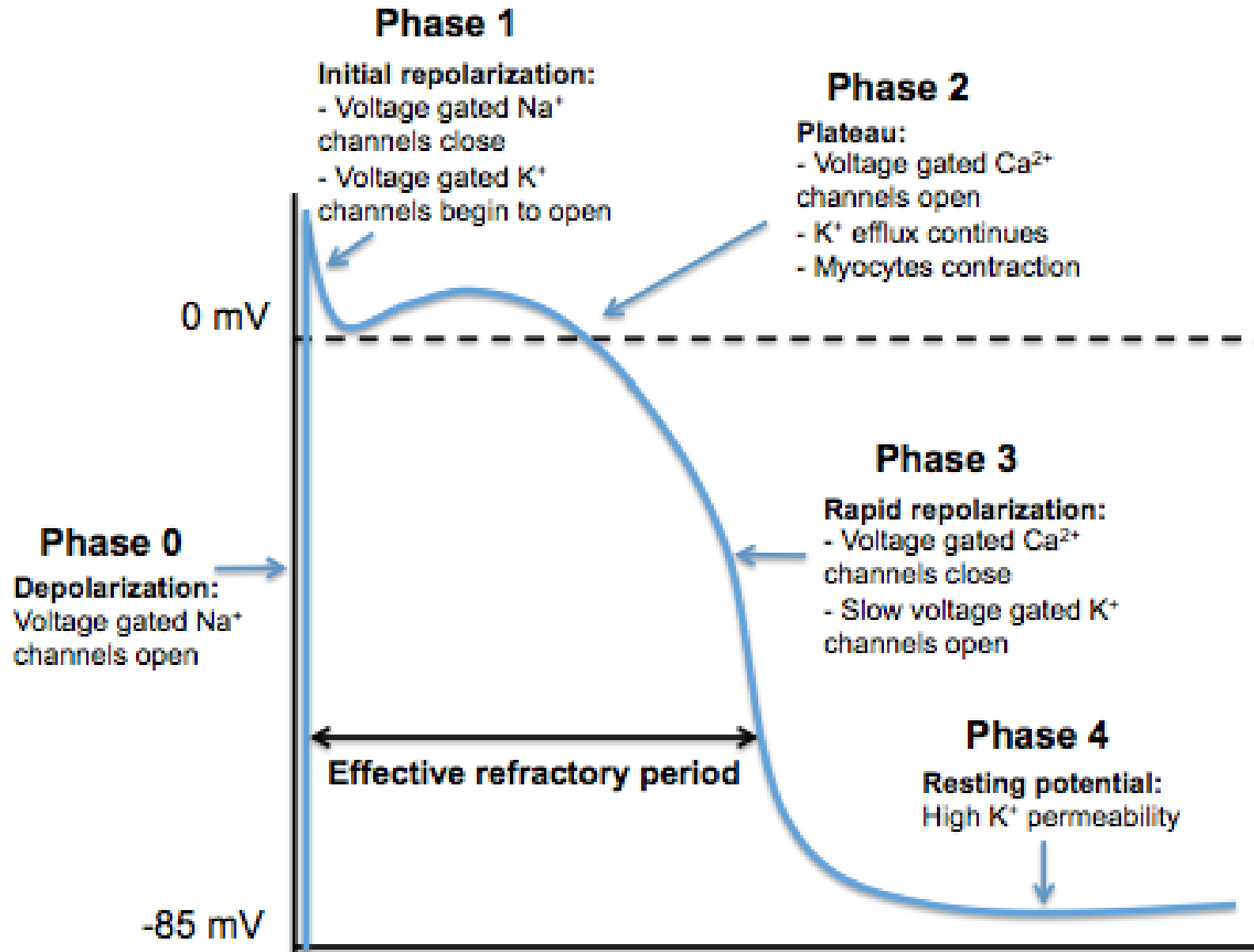
As discussed before, the raising phase of an action potential results by fast activation of Na^+ channels. These are called **fast channels**.

In some excitable cells, like **cardiac muscle** and **uterine muscle**, cells are equipped with another type of channels known as **slow $\text{Na}^+ - \text{Ca}^{++}$ channels**.

The slow and prolonged opening of slow channels will cause mainly Ca^{++} to **enter** the cell and prevents the rapid fall induced by activation of K^+ channels, and the membrane potential is **maintained** for a while then the potential falls to its resting level.

These channels are activated at slower rate than Na^+ channels.

This is known as a **plateau** in action potential. The presence of plateau in this type of cell is important in **prolonging the time of an action potential**, giving more time for the cell to be able to respond to another stimulus, because the cell remains longer time in refractory period.



During an action potential, the cell cannot respond to another stimulus. **From the firing stage to the end of the first third of falling phase** the cell will not respond at all even by a stronger stimulus.

In this stage the cell is said to be in **absolute refractory period**.

From the beginning of the second phase until the resting membrane potential is achieved, the cell cannot respond to the usual stimulus, but a **stronger stimulus** can change the membrane potential. In this period, the cell is in **relative refractory period**.

Refractory periods of an action potential

Refractory periods of an action potential

The periods depend **on the activity of Na⁺ channels**. These channels pass **three** states during action potential.

- 1- During resting potential, Na⁺ channels **are closed** but capable for opening when stimulated.
- 2- During the raising phase (firing), **almost all Na⁺ channels are opened**. And any other stimulus (even stronger one) will not cause activation of more Na⁺ channels. During this period, the membrane is in **absolute refractory period**.
- 3- In the third state, **when voltage dependent Na⁺ channels become closed** after the membrane potential has reached positive values. At this state, Na⁺ channels are not capable for opening. During all the falling phase of an action potential, these channels remain closed and not capable for opening.

Refractory periods of an action potential

They can pass to the first state (closed and capable for opening) when the membrane potential returns to its normal level or to a more negative potential than resting potential.

During this period, the membrane is in **relative refractory period**. This means that a stronger (**suprathreshold**) stimulus may activate the closed channels that are not capable for opening by normal stimulation. In addition to the role of voltage gated Na⁺ channels in establishing the relative refractory period, the presence of widely opened K⁺ channels during falling phase, which cause excess flow of positive charges to the outside, may also play a role by opposing stimulating signals.

TRANSMISSION OF ACTION POTENTIAL ALONG NERVE FIBERS

Once an action potential is generated at the axon hillock, no more triggering events are needed to activate the whole nerve fiber (axon). The generated impulse is conducted along the nerve fiber by one of the followings 2 methods of propagation

1. Continuous conduction (conduction by local current flow):
occurs in **unmyelinated** fibers. Local currents flow between the active area, which is at the peak of action potential and the inactive area, which is still in resting potential. This flow will cause activation of Na⁺ channels in the inactive area and reduce the membrane potential to the threshold, which triggers an action potential in this area (that was previously inactive). This process is repeated all along the nerve fiber until the impulse has reached nerve terminals.

2. Saltatory conduction:
In **myelinated** fibers, the impulse **skips** the myelinated regions in the axon and jumps from one node of Ranvier to the adjacent node. This process ensures **faster propagation** of an action potential along the myelinated axons (**50** times faster than in unmyelinated fibers of the same size). The conduction also involves current flow between two adjacent nodes of Ranvier, which results in activation of Na⁺ channels in the adjacent node, which is still in resting potential. The process is repeated until the impulse activates the axon terminals.

TRANSMISSION OF ACTION POTENTIAL ALONG NERVE FIBERS

Note: current flow in both types of conduction is from the **positively charged to the negatively charged regions at both sides of the membrane**, and the membrane has high resistance to the passage of current flow across it (**no current flow can pass through the membrane**).

Not only myelination can influence the velocity of conduction, but also the diameter of nerve fibers. Larger fibers conduct impulse with higher velocity.

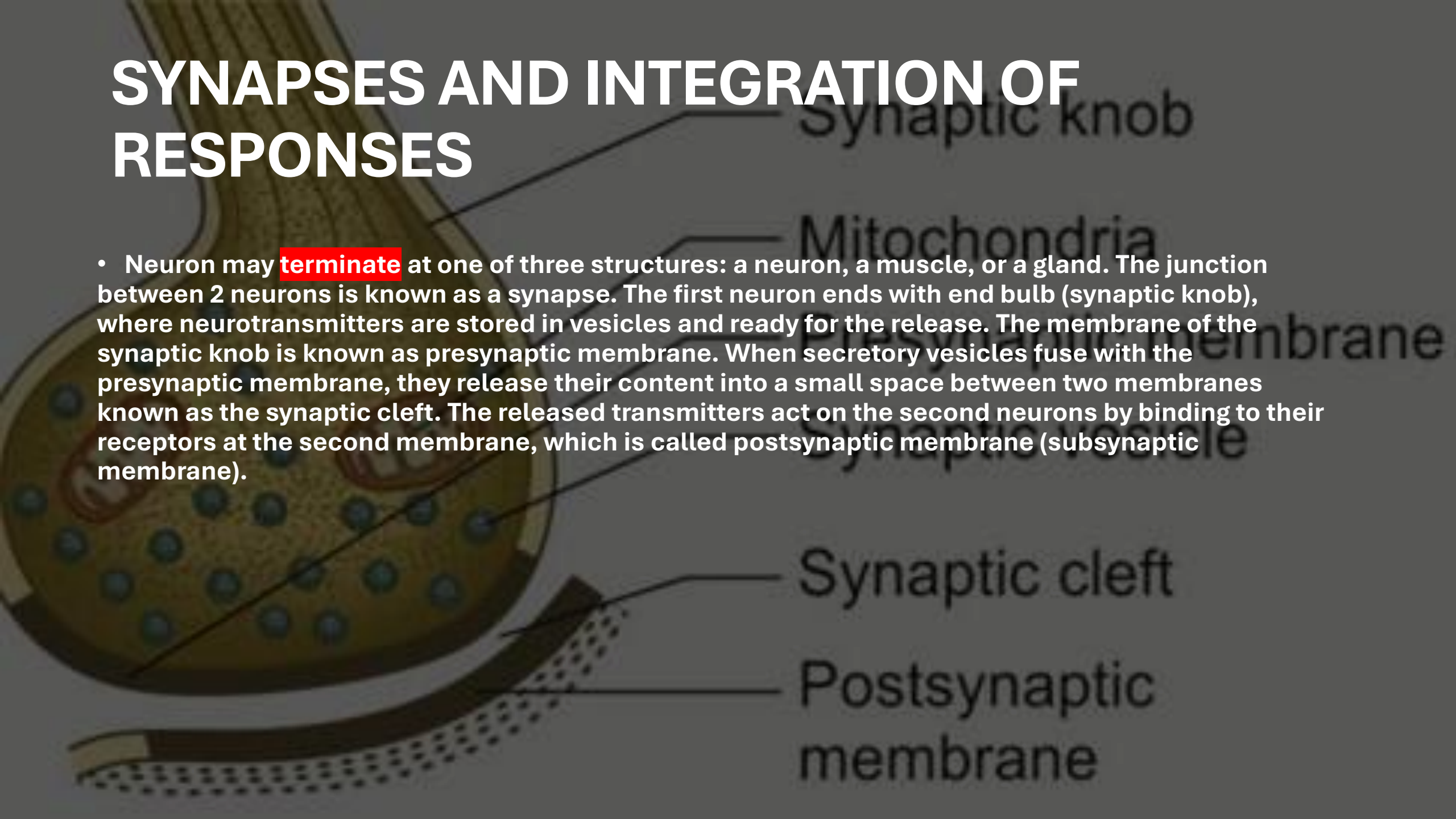
Nerve fibers have been classified in (A, which includes as subtypes (α , β , γ , δ) fibers, B, C). The diameter and the velocity of conduction is the highest in $A\alpha$, and is the lowest in C fibers.

The importance of refractory periods in conduction

The presence of refractory periods during action potential is very important in the conduction of impulse. The refractory periods **ensure the one-way (unidirectional) propagation of action potential**. Once an area has developed an action potential, the previous region is still under refractory period (unresponsive area). This area will not develop another action potential. But the following area that is at resting potential is capable to initiate an action potential.

SYNAPSES AND INTEGRATION OF RESPONSES

- Neuron may **terminate** at one of three structures: a neuron, a muscle, or a gland. The junction between 2 neurons is known as a synapse. The first neuron ends with end bulb (synaptic knob), where neurotransmitters are stored in vesicles and ready for the release. The membrane of the synaptic knob is known as presynaptic membrane. When secretory vesicles fuse with the presynaptic membrane, they release their content into a small space between two membranes known as the synaptic cleft. The released transmitters act on the second neurons by binding to their receptors at the second membrane, which is called postsynaptic membrane (subs synaptic membrane).

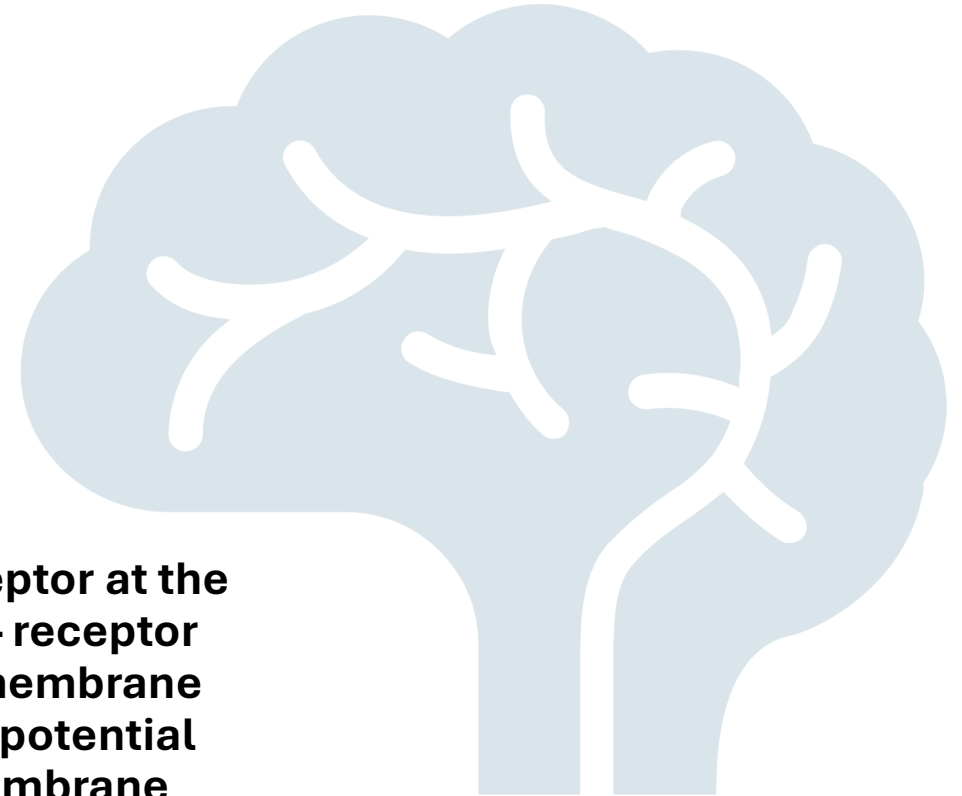


SYNAPSES AND INTEGRATION OF RESPONSES

Synapses operate in one direction. Transmit signals from one neuron to an adjacent neuron. When the impulse from the presynaptic neuron reaches the synaptic knob, this will cause **activation of voltage dependent Ca⁺⁺ channels**. This will result in Ca⁺⁺ diffusion into the synaptic knob. The increase in Ca⁺⁺ concentration inside the axon terminal will **trigger the release of neurotransmitter** from vesicles into synaptic cleft by a process of **exocytosis**. **Inactivation of synaptic knob** by **inhibitory** inputs that may synapse with the membrane at the nerve terminal may **induce inhibition of the release of transmitter**. This inhibition that appears at this site reduces the effectiveness of transmission in the synapse. This type of inhibition is known as **presynaptic inhibition**.



SYNAPSES AND INTEGRATION OF RESPONSES



- Once released, neurotransmitter binds to its receptor at the **postsynaptic membrane**. According to transmitter – receptor combination, this will induce either a decrease in membrane potential (depolarization) or increase in membrane potential (hyperpolarization). When there is a decrease in membrane potential, the developed postsynaptic potential is called EPSPs (**Excitatory Post Synaptic Potentials**), while the increase in membrane potential is called IPSPs (**Inhibitory Post Synaptic Potentials**).

SYNAPSES AND INTEGRATION OF RESPONSES

After inducing the appropriate response at the postsynaptic membrane, the transmitter is **inactivated** or **removed**, leaving the postsynaptic membrane ready to receive additional messages from the same presynaptic membrane.

The inactivation of transmitter takes place by postsynaptic membrane bound **enzymes**. An example of these enzymes is **acetylcholine esterase**, which destroys **acetylcholine** (Ach) into **acetyl** and **choline** molecules, which then transported back to the synaptic knob, where they combine again to form new **Ach** molecules. Some types of transmitters are transported back, without inactivation, into synaptic knob. Conditions that alter the activity of destroying enzyme, uptake of transmitter by nerve terminal, or induce release of high concentration of transmitter (*presynaptic facilitation*) alter the activity of synapse by prolonging the activation of receptors at the postsynaptic (subs synaptic) membrane. In addition to that, some drugs may combine with receptor and prevents binding of transmitter to its receptor. These drugs are known as **blockers**. An example of these is **hexamethonium**, which can bind to acetylcholine (Ach) receptor at postsynaptic membrane and prevents Ach from binding. This will inhibit transmission induced by Ach neurons.

SYNAPSES AND INTEGRATION OF RESPONSES

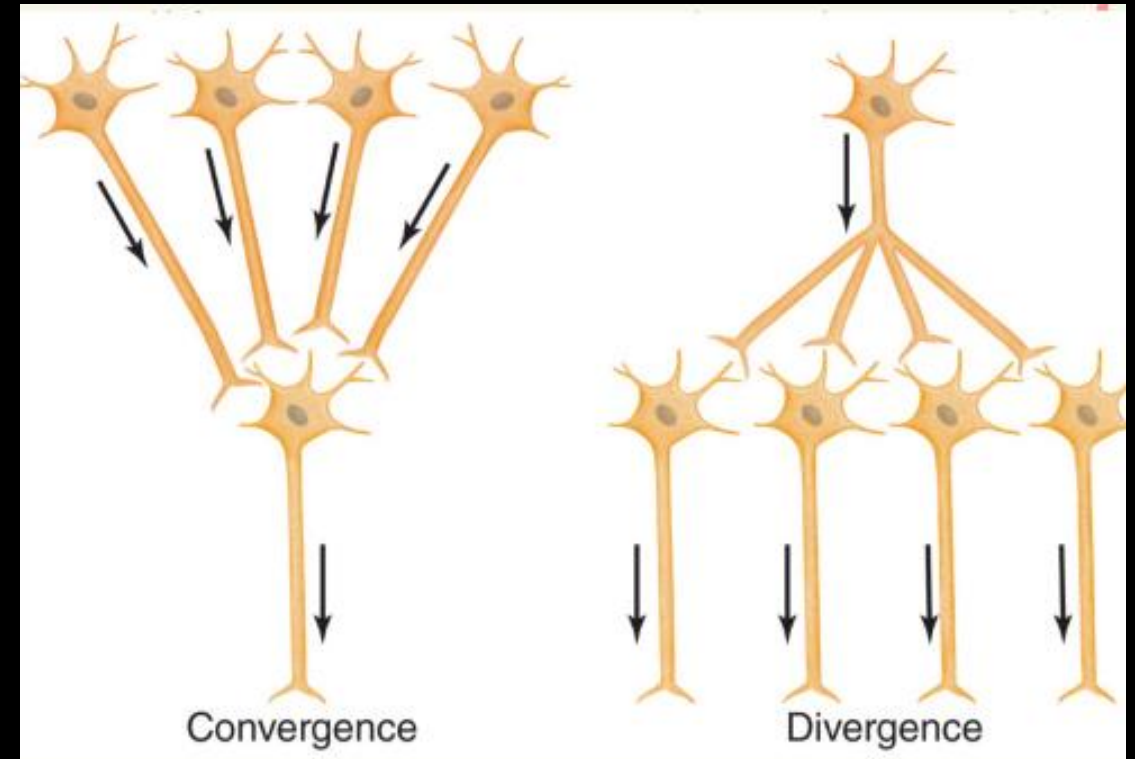
The EPSPs are not action potentials. They are small depolarization (*subthreshold potential*) that can be induced by activation of few Na⁺ channels.

The IPSPs are usually induced by **activation of K⁺ channels**. Which result in efflux of K⁺ and change in the membrane potential to more negative potential. Some transmitters activate Cl⁻ channels, the activation of these channels will not induce hyperpolarization (during rest, neural cell is near the (E_{cl}), and the opening of Cl⁻ channels will not induce inward diffusion of Cl⁻). But this activation is inhibitory on neural activity. This inhibition is achieved by holding the membrane at its resting potential and preventing depolarization.

The time it takes for a signal from the first neuron to induce changes in membrane potential in the second neuron is known as *synaptic delay*.

Integration of responses at postsynaptic membrane

- Usually, the complexity of neural network connections permit synapsing of many axonal terminals from different neurons to one neural cell body (called **convergence**), and branching of one nerve fiber to many terminals that synapse to different neurons (**divergence**). This complexity results in converting the signal from one neuron to many postsynaptic neurons in the case of divergence, and many inputs from presynaptic neurons can be received by a single postsynaptic neuron in the case of convergence.



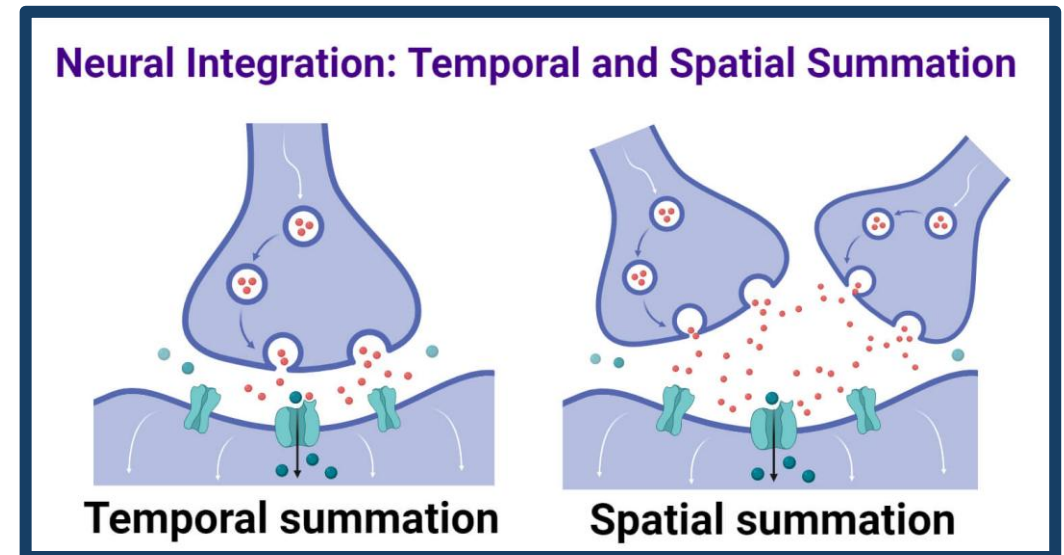
Integration of responses at postsynaptic membrane

As mentioned before, one stimulus may induce depolarization or hyperpolarization at the postsynaptic membrane. The induced depolarization is not an action potential, but it is a **subthreshold potential**. The action potential will develop only when the threshold is achieved. In a neural network, to have subthreshold potentials eliciting an action potential, **summation** (two depolarizations can sum to elicit a higher depolarization) must take place between responses at the postsynaptic membrane.

Integration of responses at postsynaptic membrane

Two types of summation are known at the postsynaptic membrane. **Spatial summation** appears when 2 or more responses from 2 or more different neurons have appeared simultaneously (at the same time) at the same site of postsynaptic membrane, which result in summing of these responses into a final response. This summation can take place between 2 or more **IPSPs** to elicit more hyperpolarization, two or more **EPSPs** to elicit more depolarization in the membrane, or between excitatory and inhibitory potentials which results in **cancellation** of potentials and induce postsynaptic inhibition.

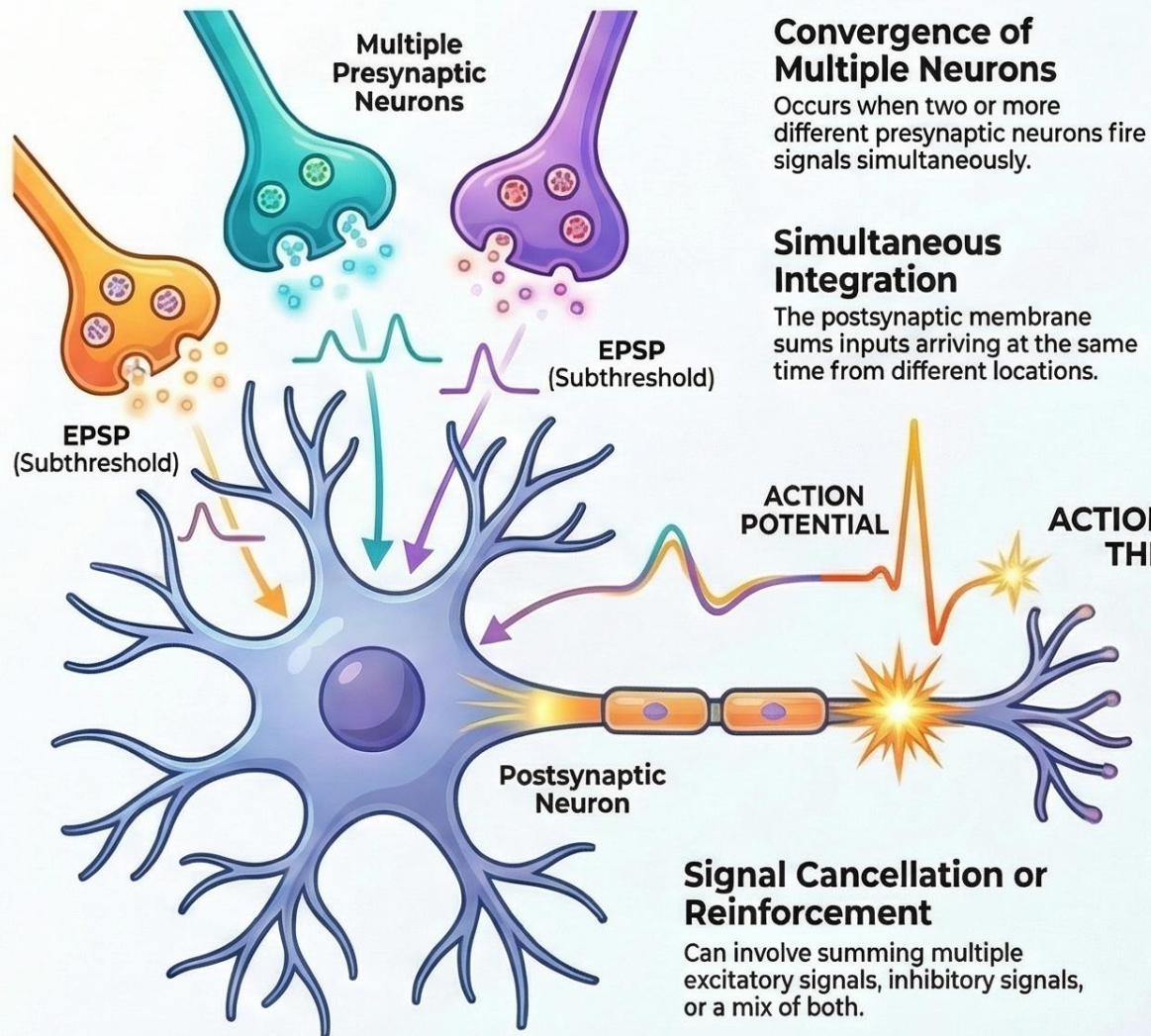
The second type of summation is called **temporal summation**. Appears when 2 or more postsynaptic potentials, which were elicited by one presynaptic neuron at different times, sum to induce more depolarization in the membrane potential. In this case, the repetitive excitation of postsynaptic membrane from a single input induces a higher depolarization that may elicit an action potential at the postsynaptic membrane.



Neural Integration: Spatial vs. Temporal Summation

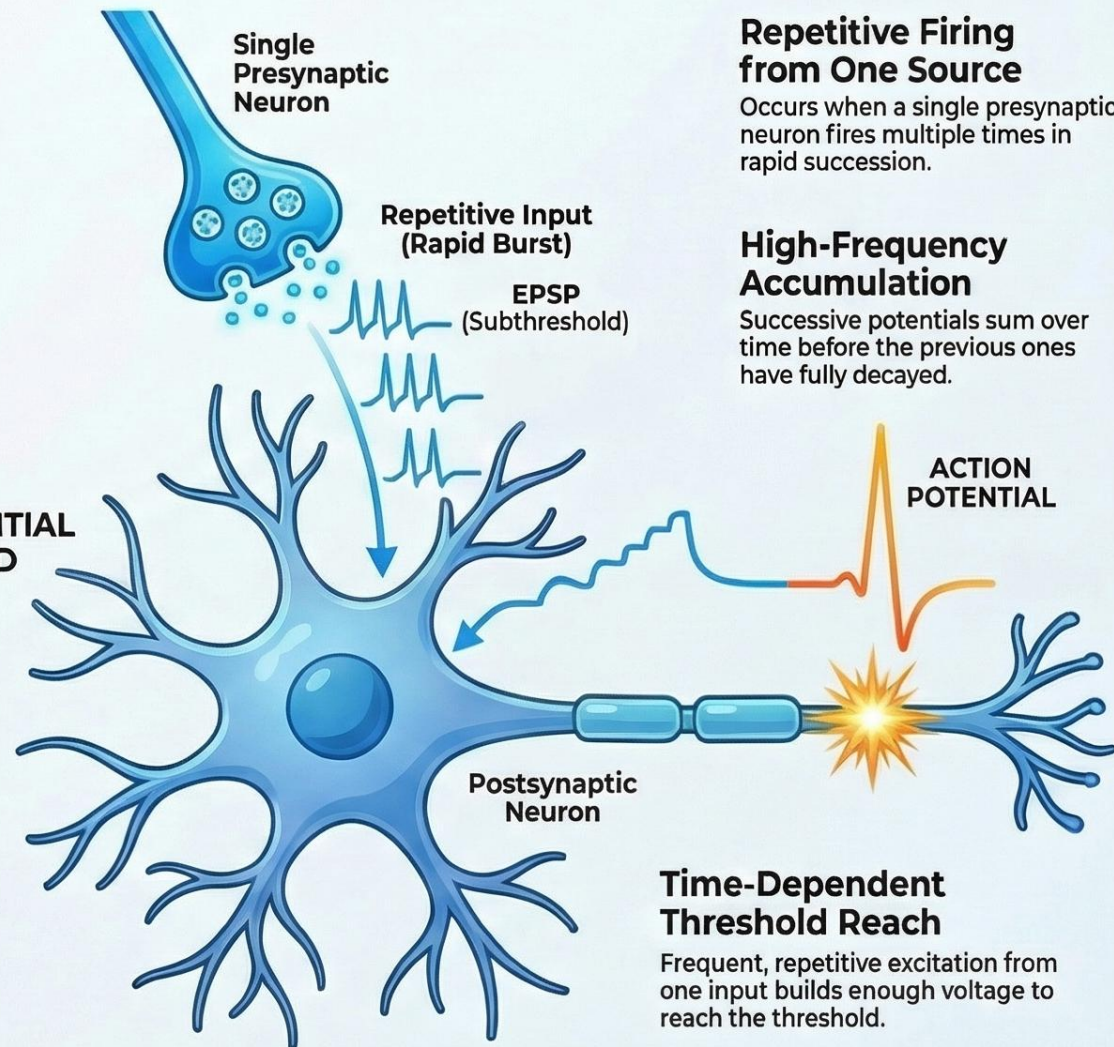
SPATIAL SUMMATION (Multiple Sources)

(Multiple Sources)



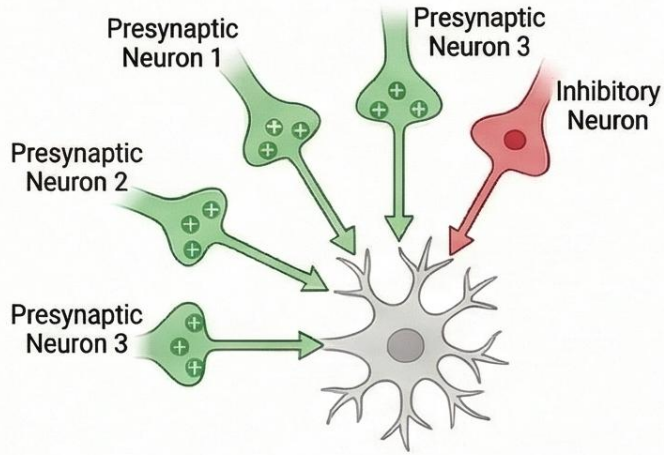
TEMPORAL SUMMATION (Single Source)

(Single Source)



How Neurons "Add Up" Signals: Spatial vs. Temporal Summation

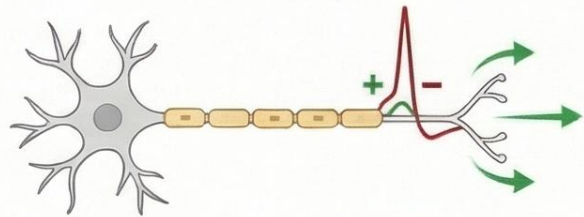
Spatial Summation (Multiple Sources)



The Potentials Involved

EPSPs (Excitatory Postsynaptic Potentials): Small depolarizations (subthreshold) caused by Na⁺ channel activation, moving towards threshold (**Green +**).

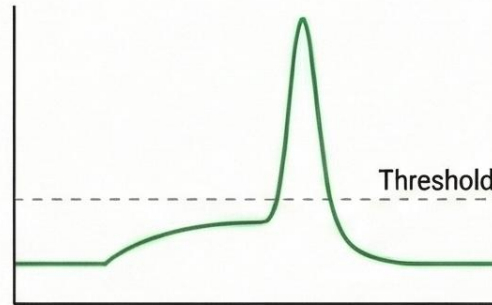
IPSPs (Inhibitory Postsynaptic Potentials): Hyperpolarizations via K⁺ or Cl⁻ channels, moving away from threshold (**Red -**).



Summation Goal: Reach threshold

Definition: Occurs when two or more responses from different presynaptic neurons appear simultaneously at the same site on the postsynaptic membrane.

Input Source: Many vs. One: Relies on "convergence," where many axonal terminals synapse onto one cell body.



Graph: Simultaneous inputs cause a large, sudden depolarization, reaching threshold.

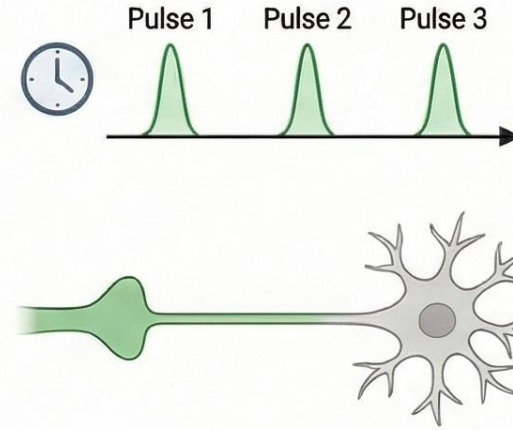
Final Outcomes

Reaching the Threshold: If the sum reaches threshold, Na⁺ permeability triggers firing.

Cancellation Effect: An EPSP and IPSP occurring simultaneously can cancel, resulting in postsynaptic inhibition.

"None or All" Principle: If subthreshold, membrane returns to resting level (-90mV).

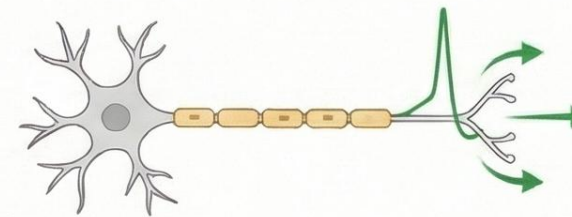
Temporal Summation (Single Source)



The Potentials Involved

EPSPs (Excitatory Postsynaptic Potentials): Small depolarizations (subthreshold) caused by Na⁺ channel activation, moving towards threshold (**Green +**).

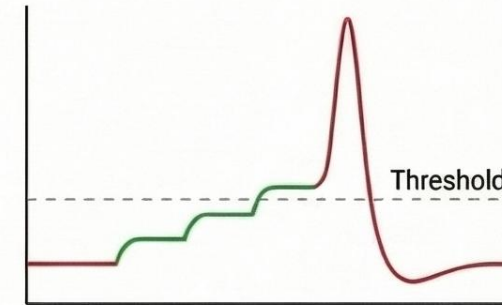
IPSPs (Inhibitory Postsynaptic Potentials): Hyperpolarizations via K⁺ or Cl⁻ channels, moving away from threshold (**Red -**).



Summation Goal: Reach threshold

Definition: Occurs when a single presynaptic neuron fires multiple times in rapid succession, allowing potentials to sum over time.

Timing: Simultaneous vs. Successive: Requires signals to arrive at different times but close enough together to overlap.



Final Outcomes

Reaching the Threshold: If the sum reaches threshold, Na⁺ permeability triggers firing.

Inhibition: Summation of multiple IPSPs results in greater hyperpolarization, making firing harder.

"None or All" Principle: If subthreshold, membrane returns to resting level (-90mV).

Potential Types: EPSPs & IPSPs (Simultaneous)

Final Result: Action Potential or Cancellation

Potential Types: EPSPs or IPSPs (Repetitive)

Final Result: Action Potential or Inhibition

Question 1

How does the $Na^+ - K^+$ pump contribute to the resting membrane potential value?

- A. It adds approximately -4 mV of additional negativity by being electrogenic.
- B. It triggers the firing stage by rapidly increasing Na^+ permeability.
- C. It facilitates the plateau phase by allowing Ca^{2+} to enter the cell.
- D. It brings the membrane potential from -94 mV to -86 mV .

(A)

Question 2

Which phase of the action potential is characterised by a 'positive feedback mechanism' involving voltage-gated channels?

A. The Plateau phase

B. Repolarisation

C. Depolarisation (Firing stage)

D. Hyperpolarisation

Question 3

In excitable cells like cardiac muscle, what is the physiological significance of the 'plateau' in the action potential?

- A. It accelerates the repolarisation phase to allow for faster heart rates.
- B. It allows for saltatory conduction to occur between the nodes of Ranvier.
- C. It prolongs the refractory period, allowing the cell more time before it can respond to another stimulus.
- D. It ensures that the action potential follows the 'None or All' principle.

Question 4

During which period is a neuron completely unresponsive to even an exceptionally strong stimulus?

- A. Absolute refractory period
- B. Relative refractory period
- C. Resting membrane potential
- D. Positive afterpotential stage

(A)

Question 5

Which type of neuroglia is specifically responsible for the myelination of axons within the Central Nervous System (CNS)?

A. Microglia

B. Schwann cells

C. Oligodendrocytes

D. Astrocytes

Question 6

In the context of synaptic transmission, what is the role of Ca^{2+} ions entering the synaptic knob?

- A. It provides the positive charge required for the saltatory conduction of the impulse.
- B. It binds to postsynaptic receptors to induce EPSPs.
- C. It inactivates enzymes like acetylcholine esterase in the synaptic cleft.
- D. It triggers the release of neurotransmitters from vesicles via exocytosis.

(D)

Question 7

Which phenomenon occurs when a single presynaptic neuron fires rapidly, causing multiple subthreshold potentials to combine at the postsynaptic membrane?

A. Spatial summation

B. Temporal summation

C. Saltatory propagation

D. Presynaptic inhibition

(B)

Question 8

When recording a biphasic action potential with two electrodes placed outside the cell membrane, what do the two obtained waves represent?

- A. The first represents Na^+ influx and the second represents Ca^{2+} influx.
- B. The first represents depolarisation and the second represents repolarisation.
- C. The first represents the absolute refractory period and the second represents the relative refractory period.
- D. The first represents an EPSP and the second represents an IPSP.

(B)