



☒ Sheet

☐ Slides

Number

9

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### Quick recap of some important notes:

- Chloride channels prevent reaching threshold so they inhibit depolarization, but if we have reached it and action potential started they can't inhibit it, however, potassium channels can have an inhibitory role during action potential.
- The more negative a resting membrane potential is the more concentration of  $\text{Na}^+$  channels which are closed and capable of opening (the number of these channels in an area unit) are present. So, the membrane becomes more sensitive to any stimulus; thus we reach the Threshold faster. (more excitable)

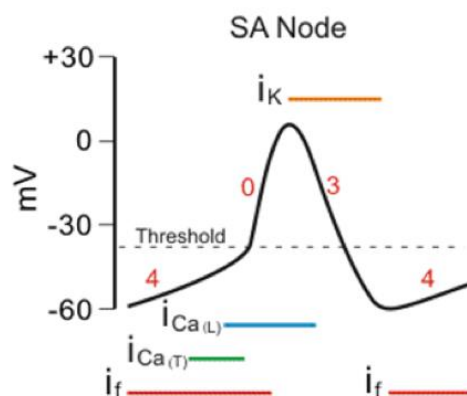
Up until now, we have been studying the action potential affected by  $\text{K}^+$  and  $\text{Na}^+$  ions. Now, we will talk about the involvement of other ions in action potential.

### Conductive tissue:

In this tissue, we can generate other configurations of action potentials than the previously-mentioned action potential. This tissue is present in the heart.

-Now, Cardiac conductive cells have a property of " $\text{Na}^+$  leakage" and this property causes automatic (spontaneous) **Depolarization**.

\*Types of  $\text{Ca}^{++}$  channels: 1- T-type (Transient).      2-L-type (Long-lasting).



$I(f)$ : Funny current ( $\text{Na}^+$  current)

### Cardiac conductive cells action potential (as in Pacemakers):

For example, we have the resting potential at -60 mV (which is not enough negative voltage to keep the sodium channels totally closed) and that causes the slow depolarization ( $\text{Na}^+$  leakage). By that we reach -50 mV, at this potential we activate the 1<sup>st</sup> type of  $\text{Ca}^{++}$  channels which is the T-type. By activating it  $\text{Ca}^{++}$  is moving from

**outside to inside** which means more **Depolarization**. Then we reach another point where we activate the L-type channels, the activation of the 2<sup>nd</sup> type helps reaching the Threshold which results in having the phase 0. At the tip, L-Ca<sup>++</sup> channels get inactivated and the conductance of K<sup>+</sup> increases which causes K<sup>+</sup> current **Repolarization**(Phase 3) returning to the resting membrane. (Phase 4)

**Note that:** This action potential is produced without a stimulus; it is produced by the automatic leakage of Na<sup>+</sup>.

**-Note:** If you increase Na<sup>+</sup> leakage you get faster depolarization → Higher rate of action potential.

If you decrease Na<sup>+</sup> leakage you get slower depolarization → Slower rate of action potential.

## Cardiac muscle action potential:

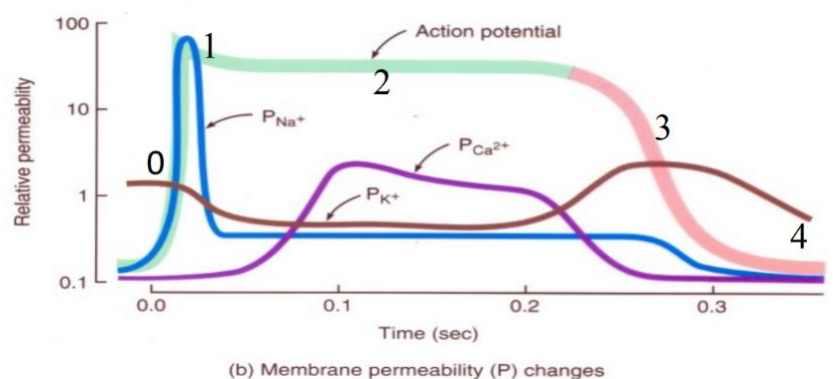
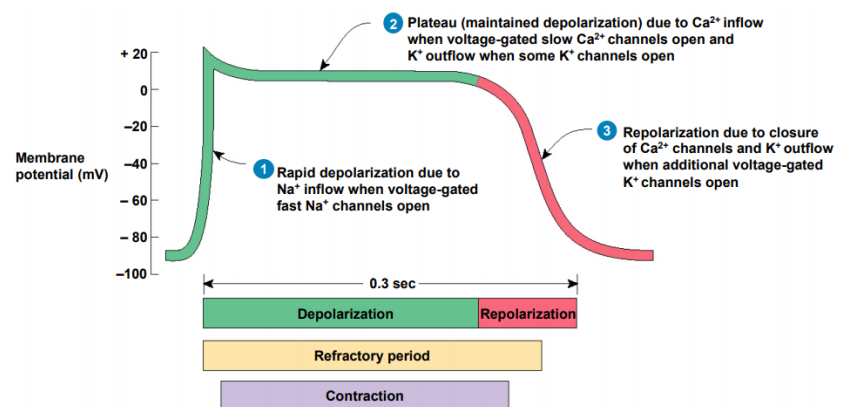
There are 5 phases:

### Phase 0 (depolarization):

When a cardiac cell is stimulated, the membrane potential becomes more positive. Voltage-gated Na<sup>+</sup> channels (**fast** Na<sup>+</sup> channels) open and allow Na<sup>+</sup> to flow inside the cell and depolarize it.

**Phase 1 (initial repolarization):** Na<sup>+</sup> channels getting inactive, the cell begins to repolarize and K<sup>+</sup> ions leave the cell through the opened K<sup>+</sup> channels.

**Phase 2 (Plateau):** The voltage-gated Ca<sup>++</sup> channels open **slowly** during phases 0 and 1 so Ca<sup>++</sup> enters the cell. Meanwhile, this opening of L-type Ca<sup>++</sup> channels make the opening of K<sup>+</sup> channels slower than usual. The combination of decreased K<sup>+</sup> conductance and increased Ca<sup>++</sup> conductance causes the action potential to plateau.



**Phase 3 (rapid repolarization):**  $\text{Ca}^{++}$  channels close and  $\text{K}^{+}$  conductance increases permitting  $\text{K}^{+}$  ions to exit the cell returning the cell membrane potential to its resting level.

#### **Phase 4 (Resting membrane potential)**

→ Muscle activity is divided into: Contraction and Relaxation. When there is a high concentration of  $\text{Ca}^{++}$  the muscle contracts.

**-The importance of the Plateau:** The Plateau makes the refractory period of the cardiac muscle much longer in order to prevent receiving another stimulus during contraction which is called **Tetanisation**, if this happens then the muscle will remain in contraction without relaxation.

**Note:** Calcium's function is not only generating the plateau action potential but it also plays a role in contraction.

The action potential in the cardiac muscle is different from the conductive tissue according to the function of each.

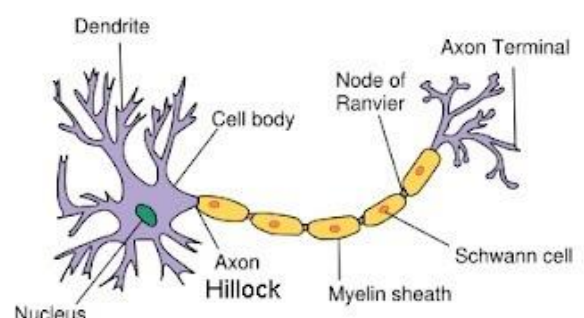
### **Generation of action potential in neural cells:**

As we learnt in the previous lectures, in our neural cells, as well as skeletal muscle cells, only Sodium channels and Potassium channels are involved in action potential.

The function of our nervous system (in general): is to generate action potential and synthesizing neurotransmitters sending them towards the terminals of neural cells at which they are released.

In motor neural cells the impulses (action potential) is transmitted from the CNS to the effector.

But in sensory neural cells (the cell bodies are in the central nervous system, at the periphery we have terminals) the action potential is generated at the periphery and conducted

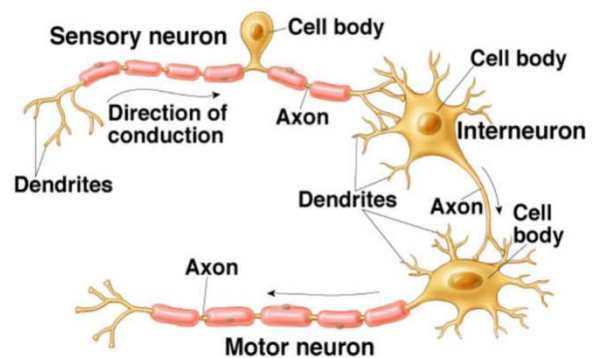


towards the central nervous system (not toward the neural cell body).

- The neural cell consists of: 1) Cell body 2) Dendrites 3) Axon which ends as axon terminals.

## The conduction of impulse through the neural cell:

The action potential is generated at the axon hillock (the junction between the axon and the cell body) and then conducted to the axon terminals then towards the central nervous system, the first sensory neuron is called T-neuron (there are two axons in different sides as T shape).



The cell body of a neuron has no role during the action potential, since an action potential is transmitted from one terminal to a second terminal.

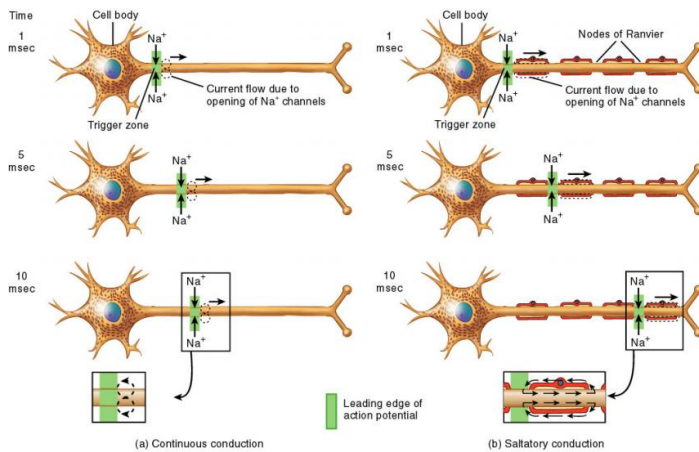
### How an action potential is transmitted towards the terminal?

There are two types of axons **myelinated axons** and **unmyelinated axons**.

1) *Myelinated axons*: they have myelin sheath wrapped around the axon.

\*myelin sheath is a cell forming a sheath around the axon and covering many parts of the axon, but it leaves small parts uncovered (between the myelin sheaths) these small parts are called nodes of Ranvier we will discuss their importance in the conduction of action potential.

2) *unmyelinated axons*: they don't have myelin sheath wrapped around the axon.



\*There are many types of **supportive cells** around the neurons, they perform different functions such as:

A. cleaning the medium around the neuron: since neural cells are functioning all the time, potassium ions are released continuously, the neural cells return back some of these ions and these supportive cells help them, and also they can uptake the excess of K<sup>+</sup> and neurotransmitters from the interstitial fluid around the neurons.

→ This is important for having a healthy medium and so a healthy function of the neurons (maximal function).

**Note:** as we have taken, sodium-potassium pumps have a similar function, but a neural cell can't handle this situation (action potential and returning the ions back) for a short term that's why supportive cells are needed.

B. phagocytic activity: when a pathogen is trying to beat, a barrier called blood brain barrier prevents passage of any pathogen from the blood towards the central nervous system, but at any time we have some passage of these pathogens these supportive cells defeat them.

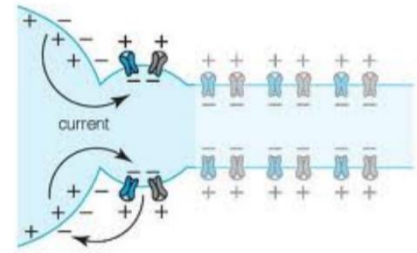
C. Releasing neurotrophic factors: since neural cells can't be replaced if they are destroyed, neurotrophic factors are released to maintain the survival of these cells as long as possible.

- The myelinated and unmyelinated fibers don't have the same conductance for an impulse.

(nerve axon is also called nerve fiber, an impulse refers to action potential)

## How is an impulse travelling along the nerve fiber?

As an action potential is generated in one area, it becomes positive inside and negative outside while the nearer area is still polarized (negative inside and positive outside)



→ This creates local ionic current as in the picture:

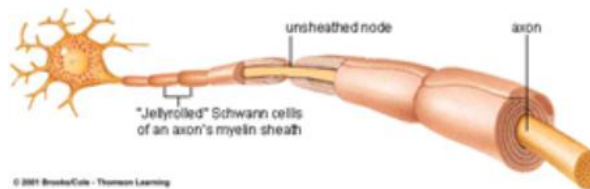
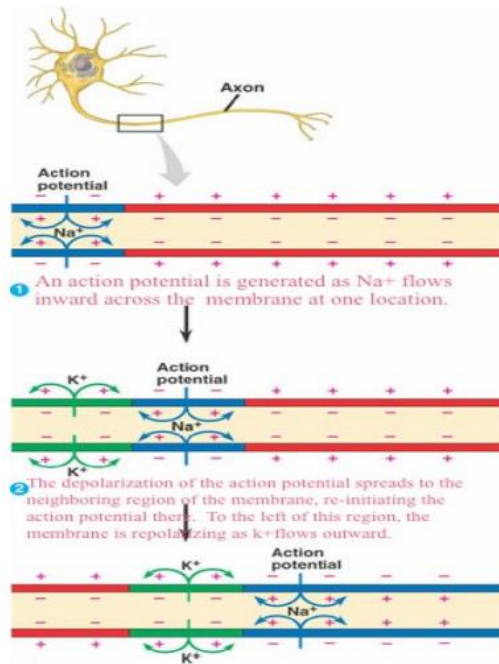
current flow from the positively charged to the negatively charged (resting) regions at both sides of the membrane (but the internal current is more important) and this changes the membrane potential (becoming less negative inside and less positive outside in the neighboring resting regions) → so the membrane is getting depolarized, reaching the threshold for action potential at that regions.

Notes: the membrane has high resistance to the passage of current flow across it (no current flow can pass through the membrane).

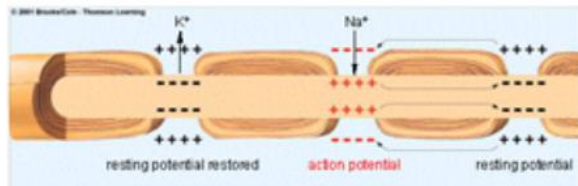
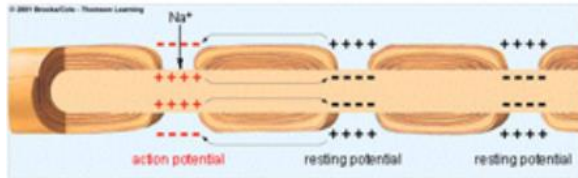
Methods of action potential propagation:

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 inactive area, which is still in resting potential. This flow will cause activation of  $\text{Na}^+$  channels in 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  $\text{Na}^+$  channels in the adjacent node, which is still in resting potential. The process is repeated until the impulse activates the axon terminals.





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

\*\*Refractory period ensures the one way (unidirectional) propagation of action potential.