Function of biomembrane and it's excitability with reference to membrane potential

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Introduction

- Nerve cell are specialized for the collection, conduction and transmission of information, which is coded in the form of fast moving impulse.
- The cell body bears the nucleus and organelles.
- The Denderites receive the signals.
- The axons conduct signals.
- The mylin sheath surroundes the axon in a discontinuous manner and

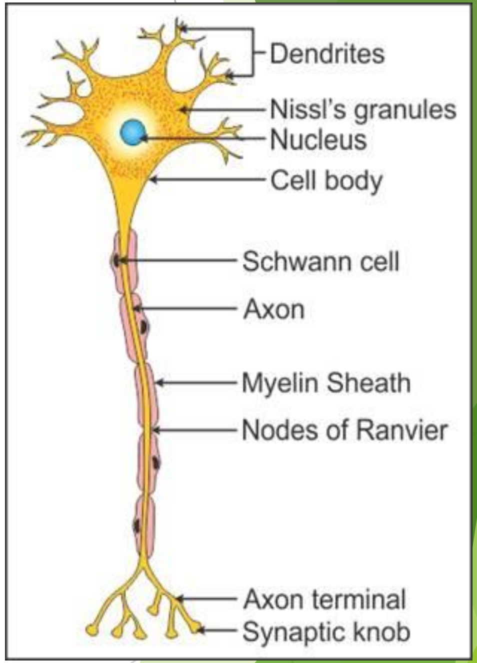
  Form the nodes of Ranvier.

Nerve cells can be long (eg – a motor neuron 's cell body in the spinal cord

And The axon ends in your toes).

An axon ends with synaptic knobs that transmit the signal through a

Specialized junction – the synapse.



Membrane potential and action potential

- Every cell in the body is electrically active : to a greater or lesser degree, they all pump ions across the cell membranes to maintain an electrical potential difference across the membrane.
- The difference in electrical charge between the inside and outside of the membrane is the basis for many types of physiological processes. Including transport of particles across the membrane and signalling among cells.
- In some cells up to 40% of energy is used to power active transport, a process that maintains or restores Membrane potential

Membrane potential

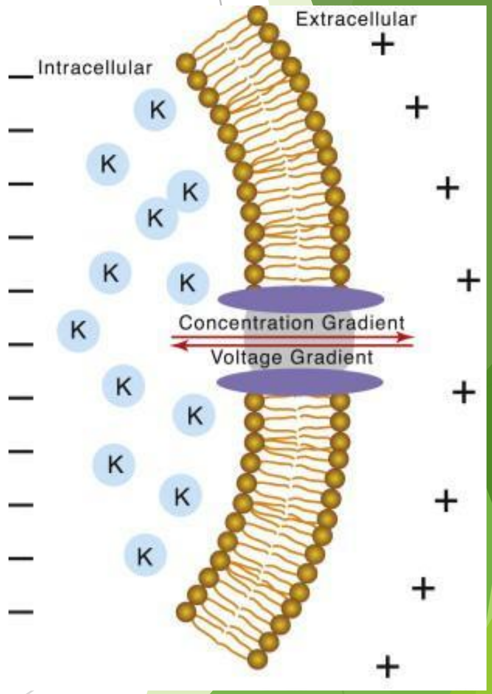
- Membrane potential is a property of all cells and reflects a difference in charge on either side of the cell membrane. Normally, cells are net negative inside the cells which results in the resting membrane potential or Vm (a negative resting membrane potential).
- The cytosol has a much higher concentration of potassium and

  mpermeable anion relative to the intercellular fluid.

  As potassium ion diffuse out of the cell the impermeable anion are left

  Behind, creating a membrane potential
- The magnitude of the membrane potential increases until and an

  Equillibrium is reached .



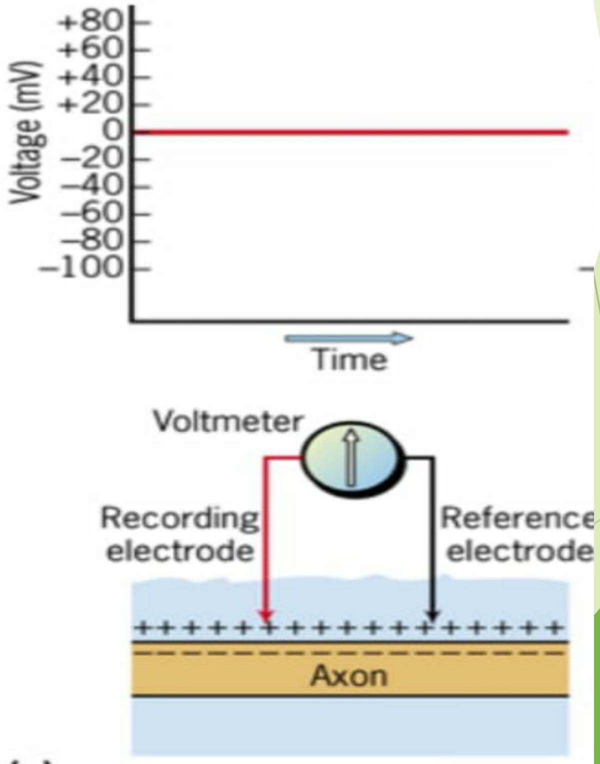
Resting membrane potential

- When nerve or muscle cell is in unexcited state, the membrane potential is reffered to as the resting potential.
- The resting membrane potential depends on differing concentration of ions inside (cytoplasm) and outside the membrane (extracellular fluid).
- Large negatively charged molecules (proteins and RNA) do not pass through the membrane to set up the negative resting membrane potential.
- If the cell membranes were simply permeable to these ions, they would approach an equilibrium with equal concentration on each side of the membrane and no voltage difference , but there is a voltage difference, so the processes which produce the membrane potential are not simply diffusion and osmosis.

- Electrical excitibility depends upon “ion channel “ Acting like gates for the movement of ions through the membrane to produce an action potential
- Passive channels, ion may freely move diffusively through the channel. Leakage channels is the simplest type, since their permeability is more or less constant.
- Chemically gated channels pump Na+ out of the cell ,while pumping in K+in the ratio of 2k+ for every 3 Na+ pumped out.
- The flow of oppositely charged ions toward each other is the potential or voltage when the ions move,this is current.
- Eventually electrochemical equilibrium (chemical vs electrical) is established and the equilibrium membrane potential is reached. .

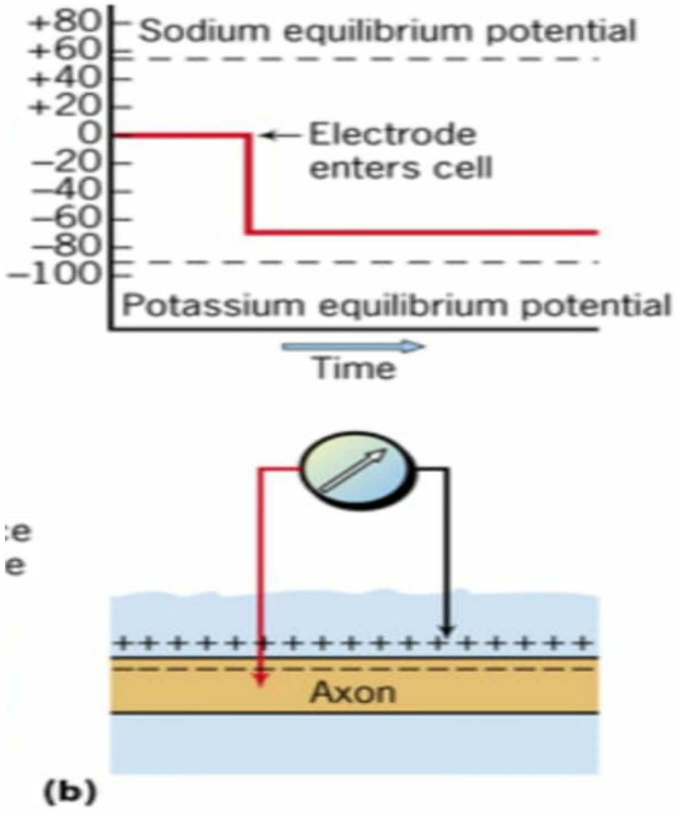
Meauring a membrane‘s resting potential

- A potential is measured when a difference in charge is detected between the reference and recording electrodes.
- In diagram, both electrodes are on the Outside of the Cell and no potential difference (voltage) is measured
- As one electrode penetrates the plasma membrane of the of the axon.



(a)

- In this diagram, the potential immediately drops to -70 mV (Inside negative) .
- Which approaches the approaches the equilibrium potential for potassium ion, that is, the potential
- That would result if the membrane were impermeable to all Ions except potassium ions.

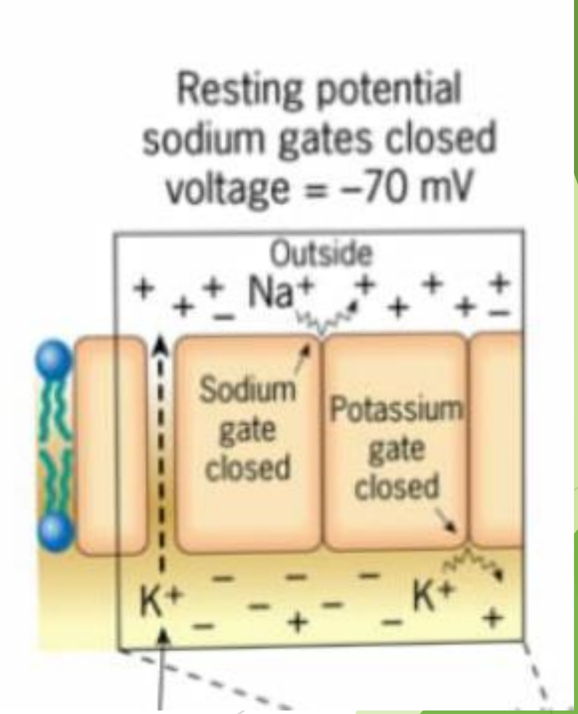


The action potential

- An action potential (also known as nerve impulse) is a pulse like waves of Voltage that passes through an axon that influences other neurons.
- The action potential has 2 main phases called depolarisation and repolarisation.
- Formation of an action potential
- During resting potential- the membrane in this region of

  Nerve cell exhibit the resting potential .
- In which only the k+ ions leak channels are open and the

  Membrane voltage is approximately -70 mV.



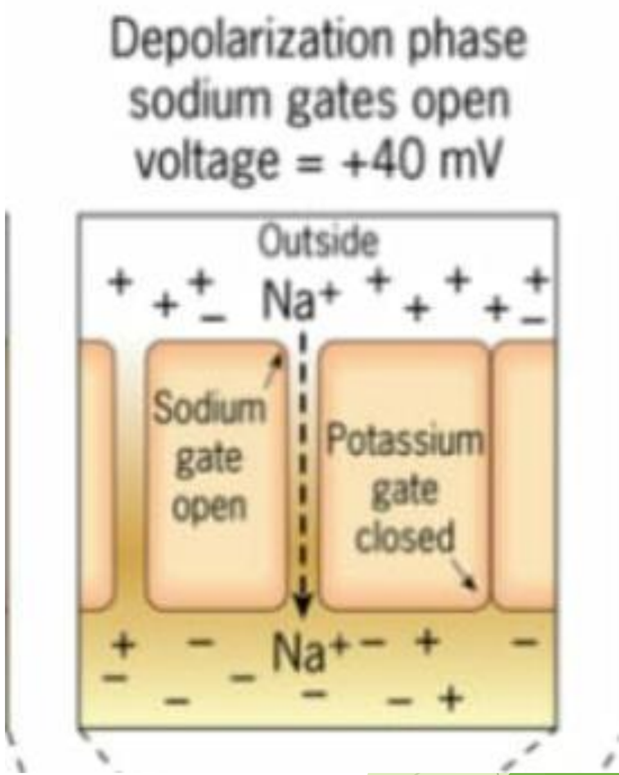
- During depolarisation phase –
- The membrane has depolarized beyond the threshold

  Value , opening the voltage regulated sodium gates, leading

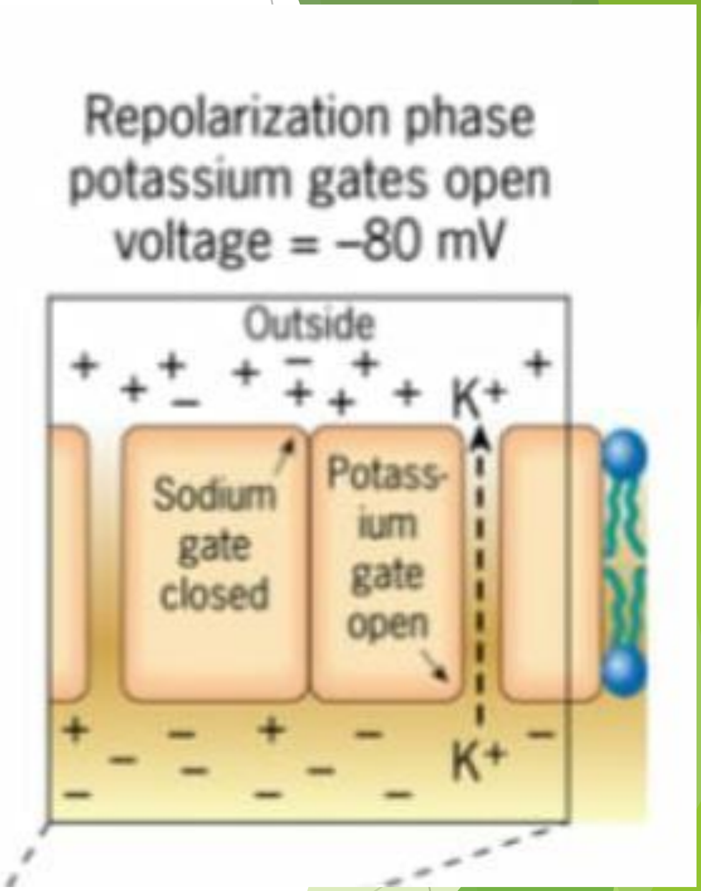
  To an to an influx of an Na+ ions.
- The increased Na+ permeability causes the membrane voltage

  To temporarily reverse itself, reaching a value approximately

  +40 mV in the giant squid axon.

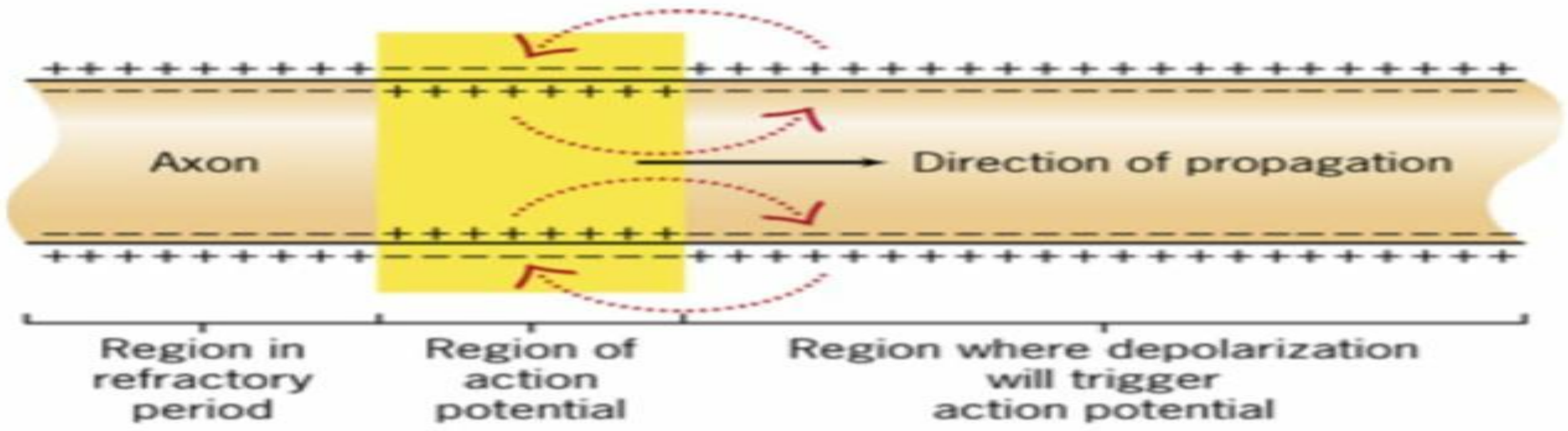


- During Repolarization phase-
- Within a tiny fraction of second, the sodium gates are Inactivated and the potassium gates open, allowing potassium Ions to diffuse across the membrane and establish an even more Negative potential at at the location (-80mV) than that of the Resting potential.
- Almost as soon as they open, the potassium gates close, leaving The potassium leak channels as the primary path of ion Movement across the membrane and re-establishing the resting Potential.



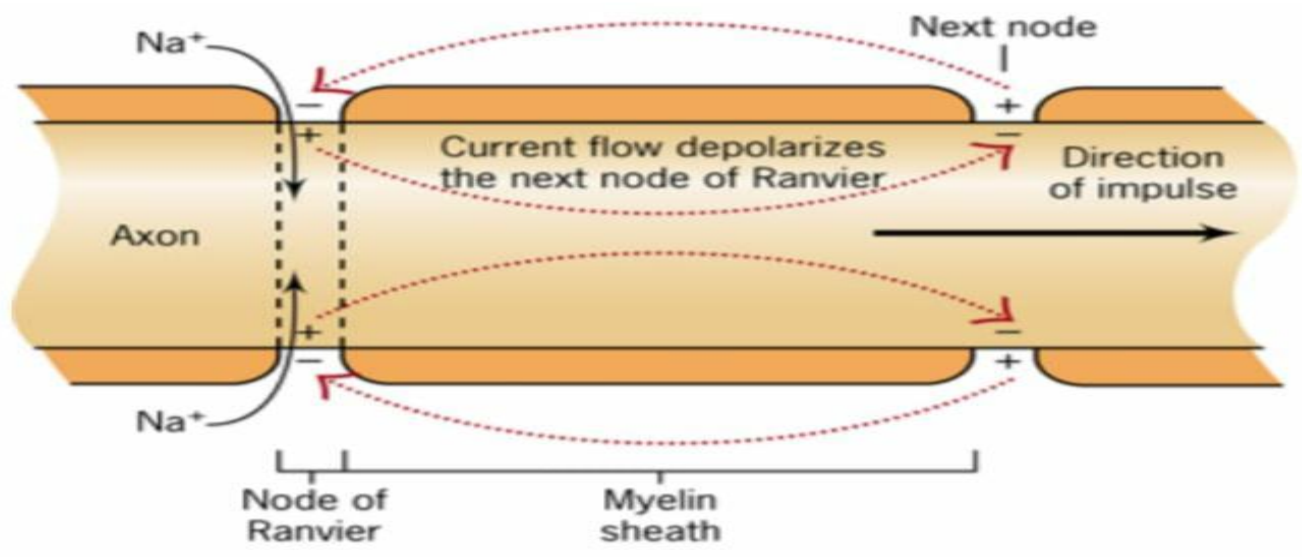
Propagation of action potential as an impulse.

- Propagation of an impulse results from the local flow of ions.
- An action potential at one site on the membrane depolarizes an adjacent region of the membrane, triggering an action potential at the second site.
- The action potential can only flow in the forward direction because the portion of the membrane that has just experienced an action potential remains in a refectory period.



Saltatory conduction

- During saltatory conduction, only the membrane in the nodal region of the axon becomes depolarized and capable of forming an action potential.
- This is accomplished as current flow directly from an activated node to the next resting node along the axon.

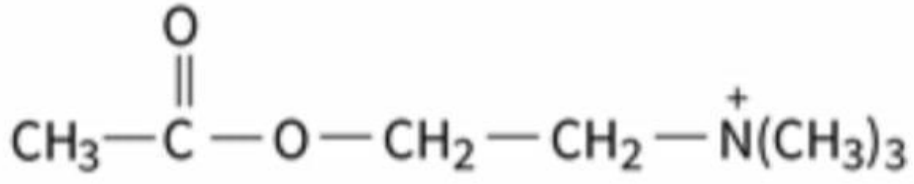


Neurotransmission : jumping the synaptic cleft

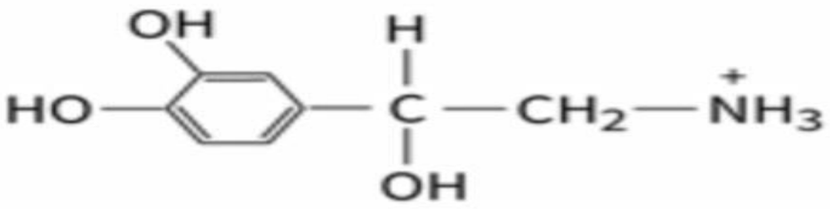
- Neurons are linked with their target cells at sowciallized juction called synapses.
- Two neuron do not make direct contact but are seprated from each other by a narrow gap of 20-50 nm, this gap is called synaptic cleft.
- Presynaptic cell conducts impulse toward a synapses.
- Postsynaptic cell receives them on the other side of the synapse.
- A number of synapses between the terminal branches of an axon and a skeletal muscle cell, synapses of this type are called neuromuscular junction.

Neurotransmitter

- A Neurotransmitter is a small molecule that, through the interaction with a specific receptor, relays a signal across nerve synapses.
- Neurotransmitter molecules that are kept in the terminal bulb or synaptic knob are secreted into the synaptic cleft and then bind to the receptors in the post-synaptic neuron.
- This generates an electrical signal to stimulate or inhibit a new action potential .



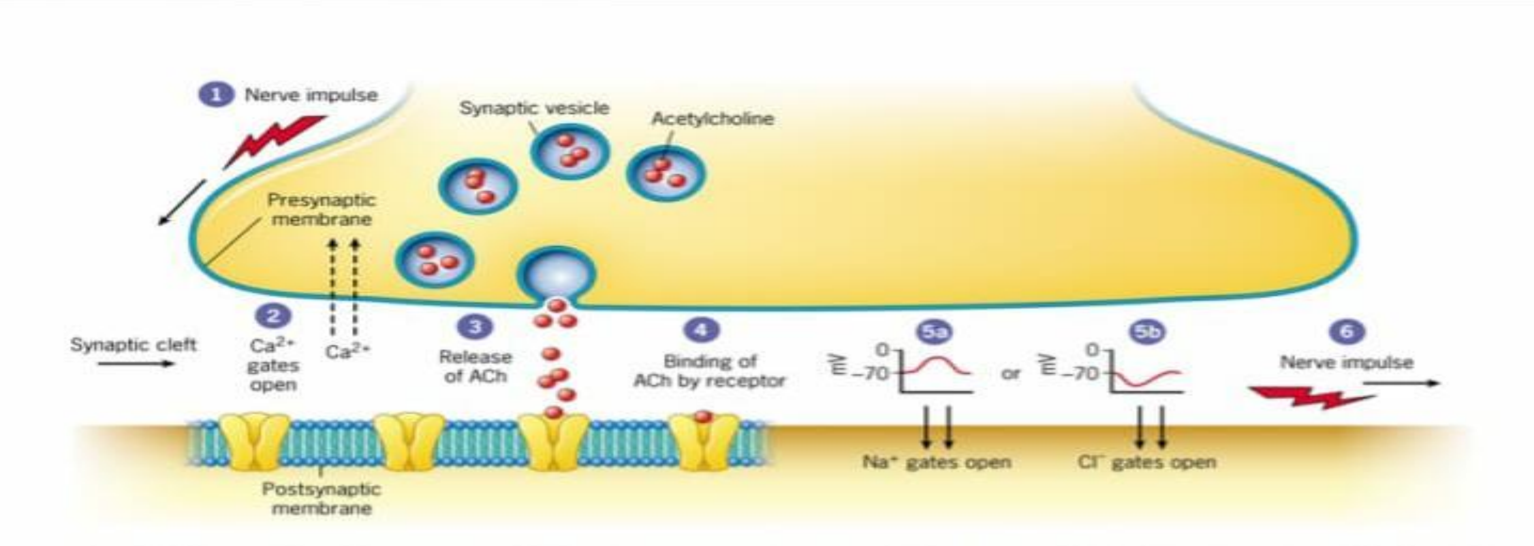
Acetylcholine (ACh)



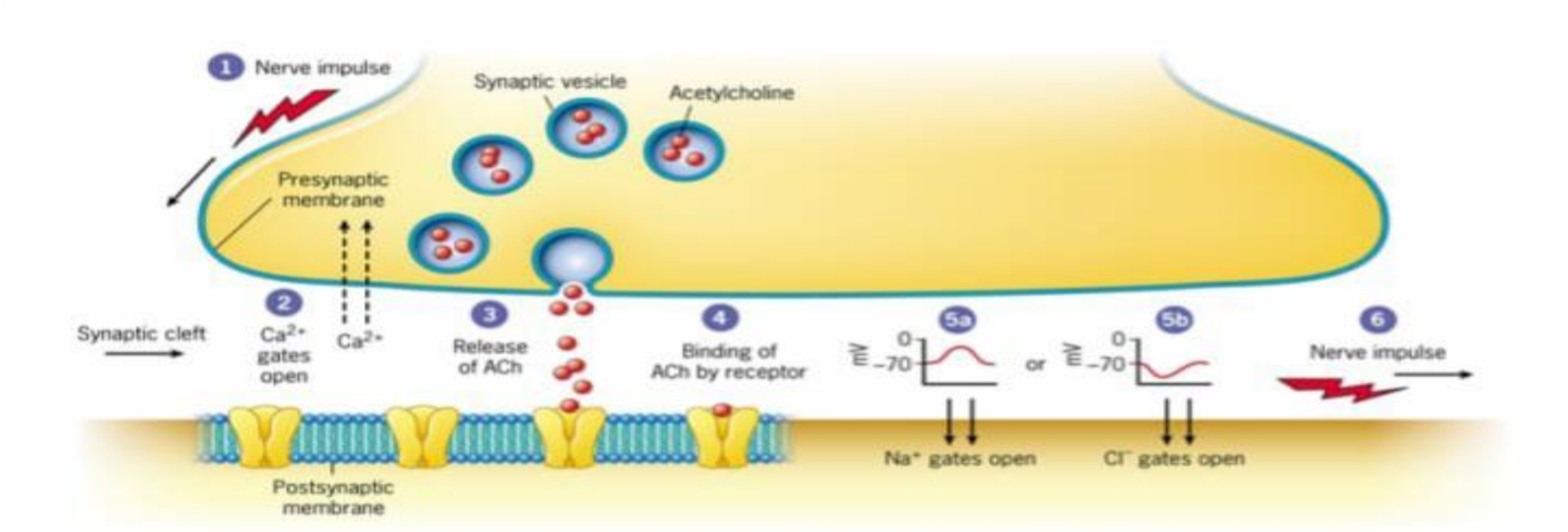
Norepinephrine

Synaptic transmission

- The sequence of events during synaptic transmission with acetylcholine as the neurotransmitter.
- A Nerve impulse reaches the terminal knob of the axon, calcium gates open leading to an influx of Ca2+ And acetylcholine is released from the synaptic vesicles and binds to receptors on the post-synaptic membrane.



- If the binding of the neurotransmitter molecules causes a depolarisation of the post – synaptic .membrane, a nerve impulse may be generated there.
- If the binding of neurotransmitter causes a hyperpolarization of the post-synaptic membrane., the target cell is inhibited, making it more difficult for an impulse to be generated in the target cell by other Excitatory stimulation.



Action of drug on synapses

- Some drugs mimic neurotransmitters.
- Heroin and opioid, for example chemically resemble the brains natural opioids (endorphir and enkephalin) sufficiently to engage and stimulate their specialized receptors.
- Since heroin stimulates many more receptors strongly than the natural opioids, the massive amplification of opioid receptor activity.
- Marijuana mimics cannabinoid neurotransmitter, the most important of which is anandamide.
- Nicotine attaches to receptors for acetylcholine, the neurotransmitter for the cholinergic system.

- Other drug alter neurotransmission by interacting with molecular components of the sending and receiving process other than receptors.
- Cocaine for example attaches to the dopamine transporter, the molecular conduits that draws free floating dopamine out of the synapses and back into the sending neurons.
- As long as the cocaine occupies the transporter, dopamine cannot renter the neuron.
- It builds up in the synapses, stimulating receiving neuron receptors more copiously and producing much greater dopamine impact on the receiving neurons than occurs naturally.

Synaptic plasticity

- Synapses act like gates stationed along the various pathway, allowing some piece of information to pass from one neuron to another, while holding back other pieces or rerouting them in another direction.
- While synapses are often perceived as fixed, unchanging structure, they can remarkable dynamic quality known as synaptic Plasticity.
- The term synaptic plasticity refers to the variability of the strength of signal transmitted through a synapse.
- Synaptic plasticity is most readily observed in studies on neurons from the hippocampus.
- Synaptic plasticity is vitally important in learning and short term memory.

- The synapses that connect these neurons are repeatedly stimulated over a short period of time, the synapses that connect these neurons to their adjacent one become “strengthened by a process known as long term potentiation (LTP).
- Which may lasts of days, weeks and even longer.
- There are numerous other reasons the study of synapses is so important.
- For example – number of diseases of nervous system including myasthenia gravis, Parkinson’s disease, schizophrenia and even depression are thought to have their roots in synaptic dysfunction.

References

Jwasa J., Marshall W., (2020), Karp‘s cell and molecular Biology, Ninth edition,

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