GOMEL STATE MEDICAL UNIVERSITY
Normal and Pathological Physiology Department

PHYSIOLOGY OF EXCITABLE TISSUES

Physiology of nerve tissue

Lecturer:
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Lecture plan:

1. Membrane-ionic theory of the origin of the resting membrane potential.
2. Membrane action potential (AP). Changes of excitability during excitation. Laws of stimulation and assessment of excitability. Lability.
3. Physiology of nerve fiber. Laws of excitement conduction. Mechanisms of signal formation and conduction in myelinated and unmyelinated fibers.
4. Parabiosis.
5. *Physiology of synapses.* Mechanisms of signal transmission in chemical synapses. Principles and features of excitation transmission in interneuronic synapses.
6. Perception of external stimuli (reception). Transformation of stimulus energy.

1. Membrane-ionic theory of the origin of the resting membrane potential

All tissues are excitable, but conventionally they are divided into excitable and non-excitable. ***Nervous, muscular, and glandular*** tissues are excitable, as impulses which appear in them go along the membrane. These impulses have an important diagnostic value (for example, in electrocardiography, electroencephalography, electromyography, etc.).

The cell membrane is known to have an electric charge. Its **external surface** is charged **positively "+"** and the **internal one — negatively "–".**

The difference between the charges of the external and internal membrane sides is called the ***resting membrane potential***.

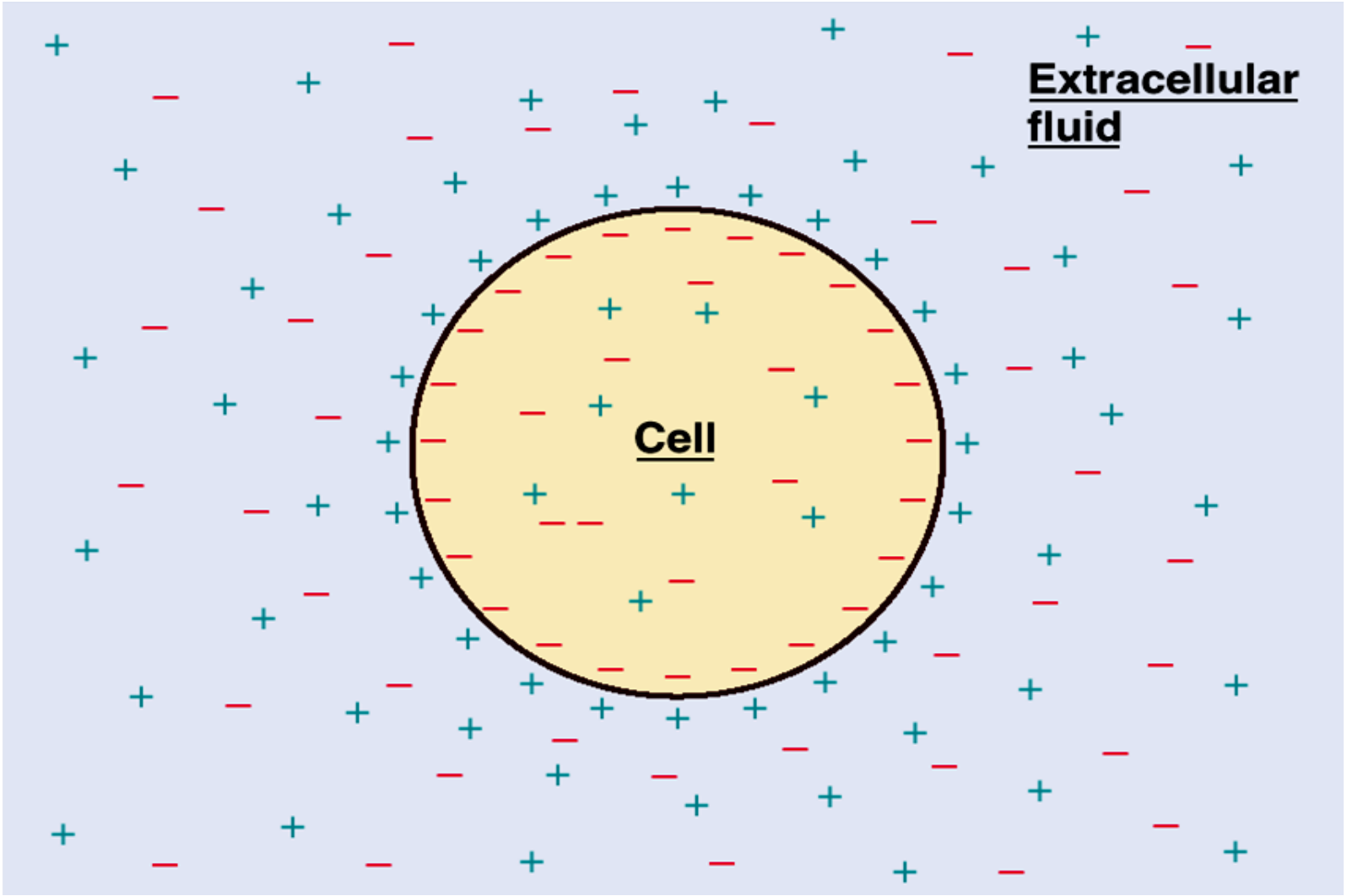


FIGURE — CHARGES DISTRIBUTION BETWEEN INSIDE AND OUTSIDE OF THE CELL IN ITS RESTING STATE

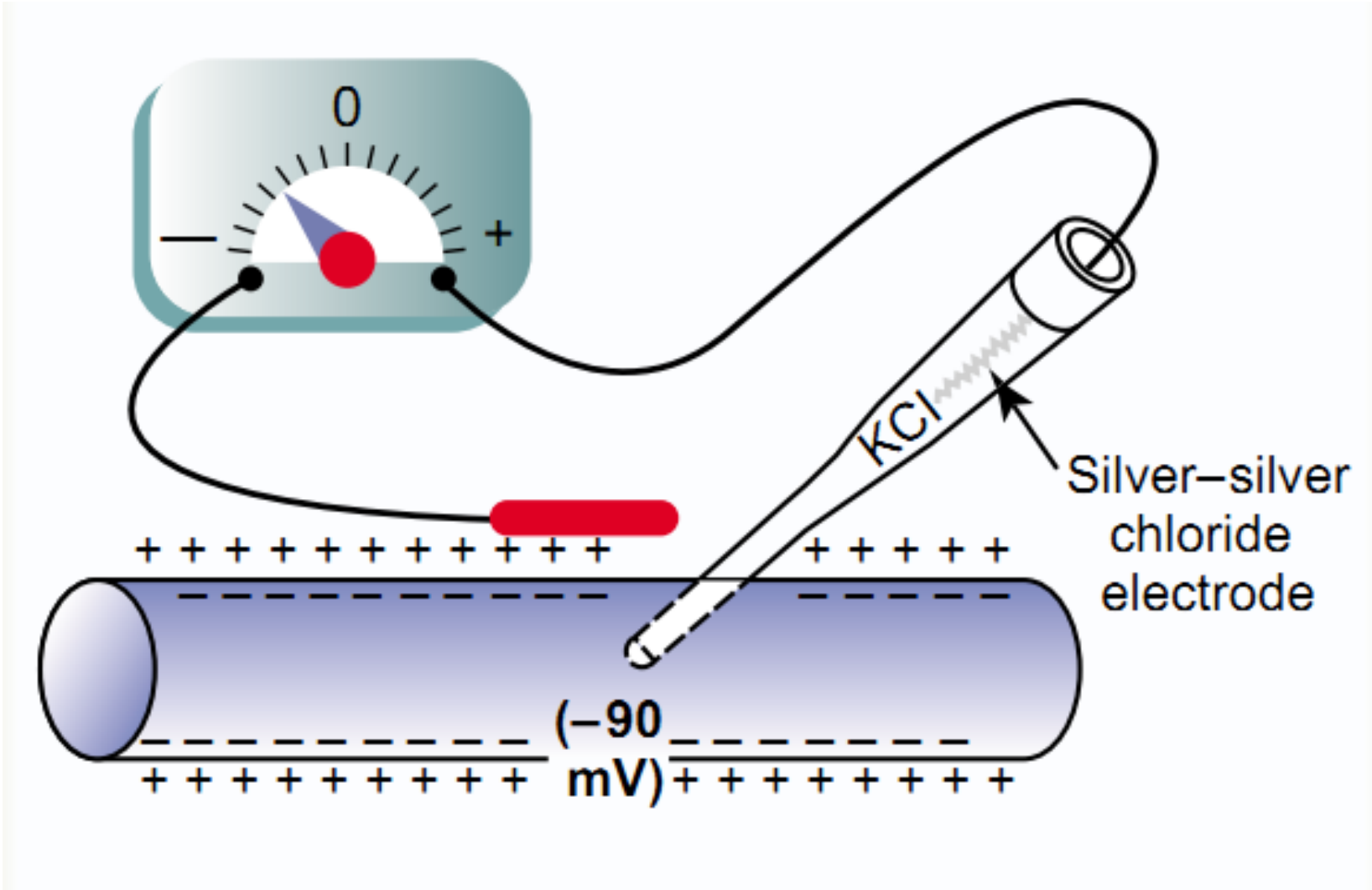


FIGURE — MEASUREMENT OF THE MEMBRANE POTENTIAL OF THE NERVE FIBER USING A MICROELECTRODE

The formation of the resting membrane potential (RMP) depends on the concentrations of K^+, Na^+, Ca^{2+}, Cl^-, as well as on the features of the cell membrane. The cell membrane has 3 layers (Figure):

- External layer — mucopolysaccharides.
- Bimolecular lipid layer.
- Internal (protein) layer.

The membrane has channels which have the properties of:

- *Selectivity* — the channels are divided into 4 groups: *sodium, potassium, calcium, chloric*. Selectivity is not obligatory yet preferable.
- *Electroexcitability.*

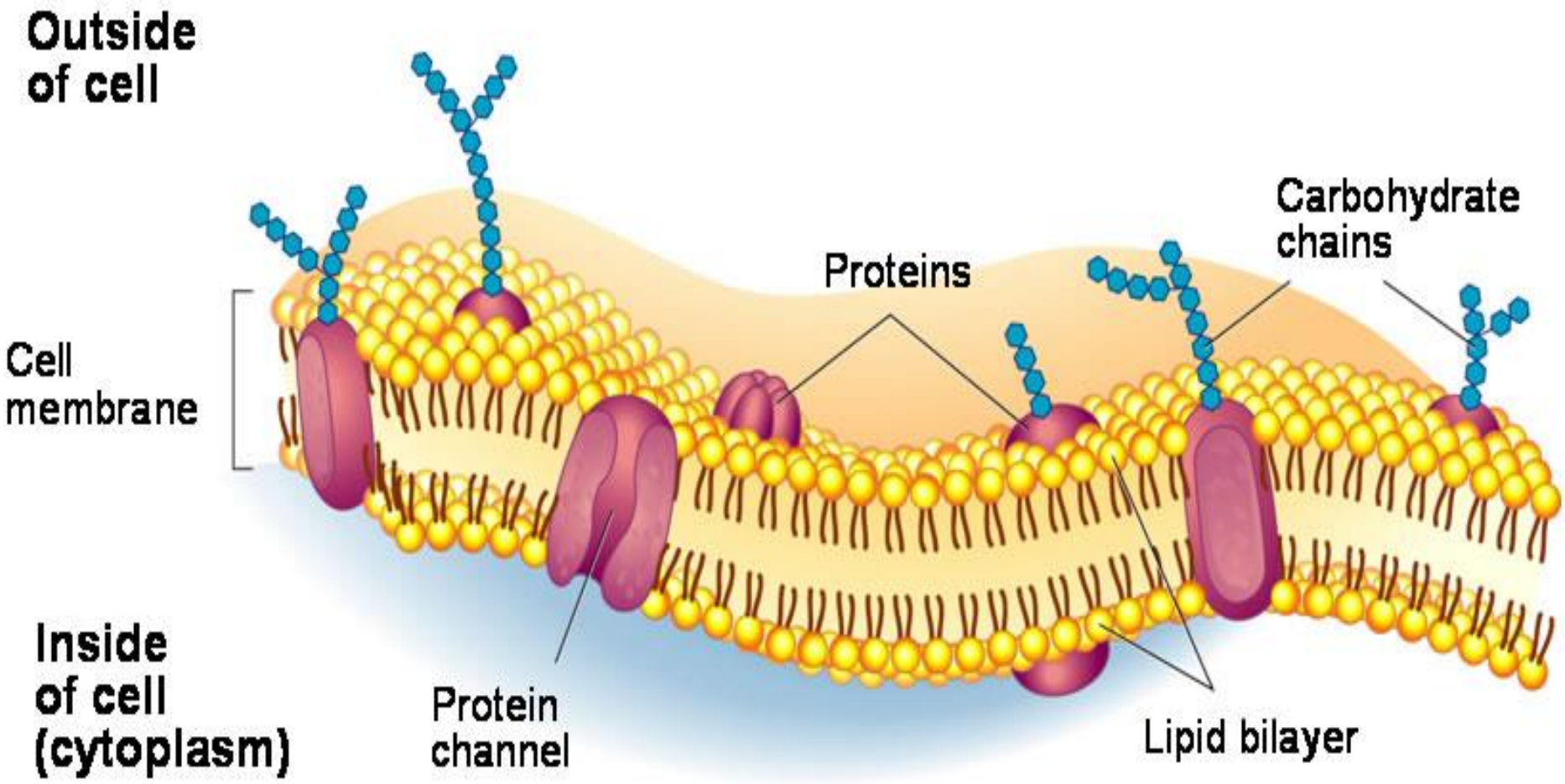


FIGURE — STRUCTURE OF THE PLASMA MEMBRANE

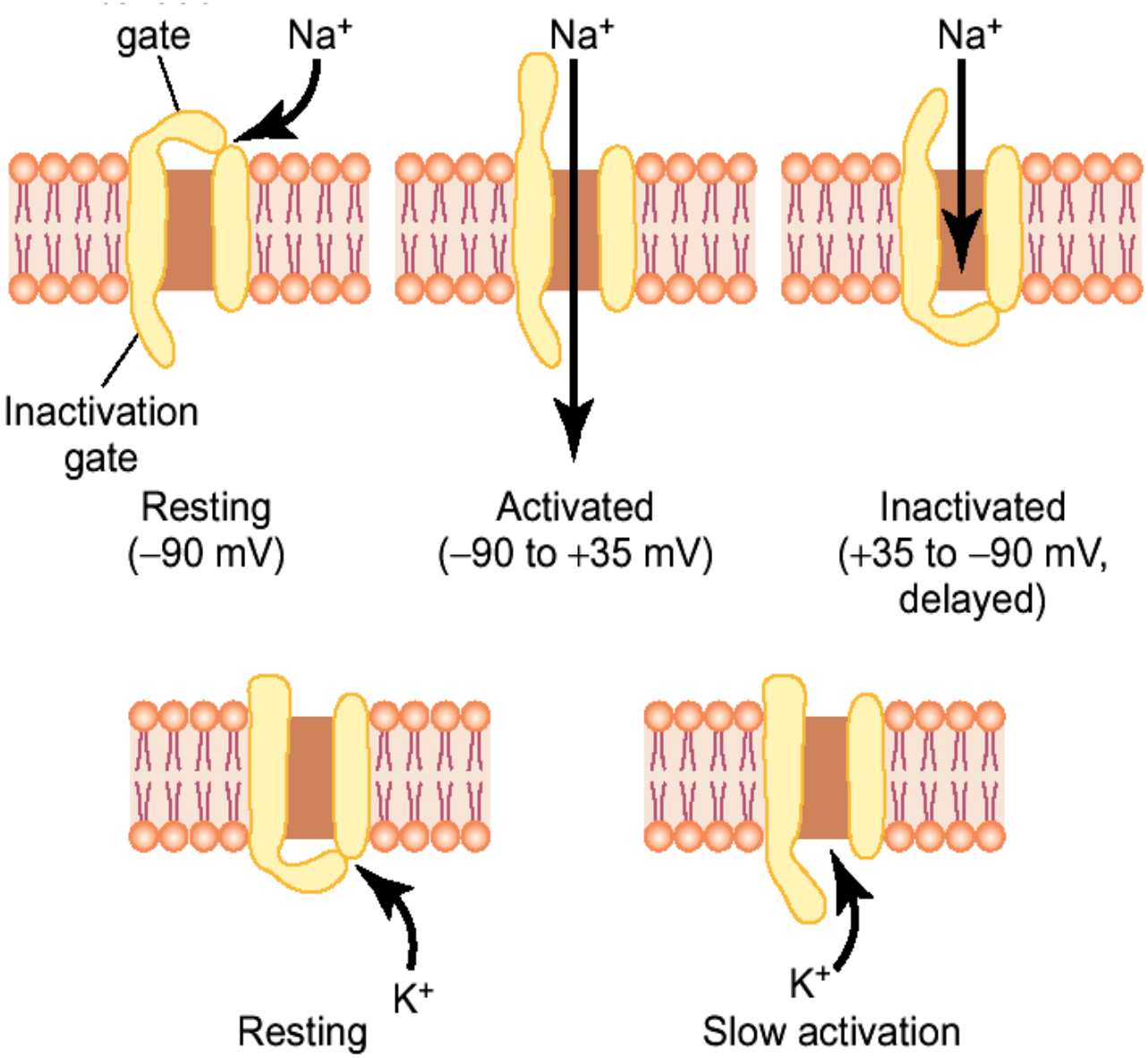


FIGURE — SODIUM AND POTASSIUM CHANNELS

Many channels can be opened or closed by gates that are regulated by electrical signals or chemicals that bind to the channels. The gating of protein channels provides a means to control their ion permeability (Figure).

Classification of ion channels:

By the amount of ions to which the channel is permeable:

— ***Selective ion channels*** (permeable to one type of ions).

— ***Non-selective ion channels*** (permeable to several types of ions).

By the type of ions the selective channels are divided into K^+, Na^+, Ca^{2+}, Cl^- channels.

By the type of regulation (gating):

— ***Voltage-gated channels****.* They react to the changes of the membrane potential. When the potential reaches a certain value, the channel becomes activated and ions pass through it down the concentration gradient.

— ***Chemically-gated channels*** *(ligand-gated channels).* In these channels the gates are opened by the binding of a chemical substance (a ligand) with receptors.

— ***Mechanically-gated channels****.* In these channels the permeability is changed if there are some mechanical actions on the membrane (these channels are present in the membrane of the mechanoreceptors of the blood vessels, skin, etc).

In cells **at rest all sodium channels are closed**. There are *leakage channels* (non-specific), which are permeable to all elements but are most permeable to potassium. They are always open, and potassium ions move through these channels down the concentration and electrochemical gradients. According to the **membrane-ionic theory,** the presence of the RMP is caused by:

- Unequal ion concentration inside and outside the cell.
- Different permeability of the channels to these ions.

There are many K^+ ions inside cells and few outside them, opposite to Na^+. There are slightly more Cl^- ions outside cells than inside them. There are *a great number of organic anions* inside cells.

The membrane of cells at rest is only permeable ***to K^+ ions***. At rest, potassium ions constantly move outside cells, where there is a high Na^+ concentration. Therefore, in cells at rest, the external surface of the membrane is ***positively*** charged. ***High-molecular organic anions (proteins)*** are concentrated on the internal surface of the membrane and determine its ***negative*** charge. Due to electrostatics they keep K^+ ions on the other side of the membrane. **The basic role in the formation of the RMP**

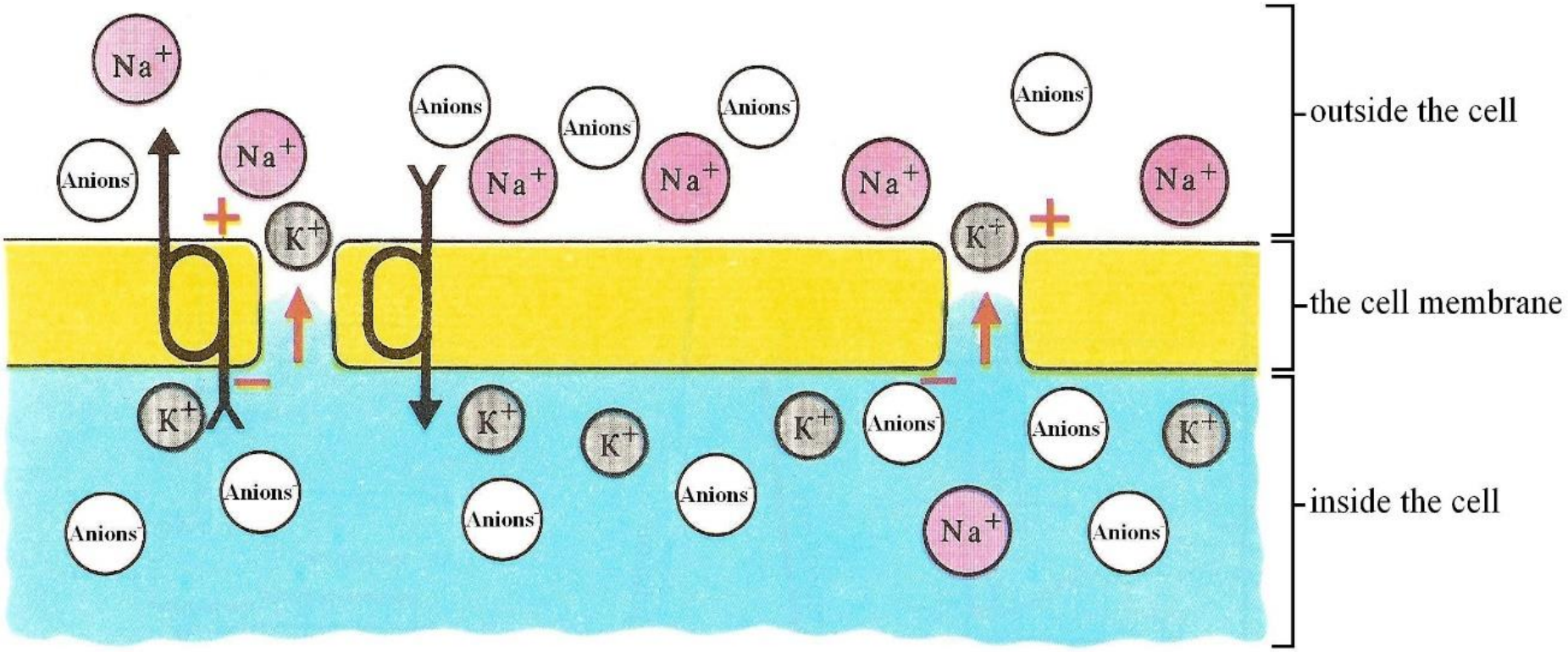


FIGURE — IONIC MECHANISM OF THE FORMATION OF THE RESTING MEMBRANE POTENTIAL

Despite the streams of ions coming through the leakage channels, the ion concentrations are not equivalent, i. e. they are always constant. This does not happen because of the existence of ***Na^+-K^+-pumps*** in the membranes (Figure).

They continuously pump Na^+ out of cells and pump K^+ against the concentration gradient into the cytoplasm. For 3 Na^+ ions removed from a cell, 2 K^+ ions are introduced into it. The transmission of ions against the concentration gradient is carried out by active transport (with energy input).

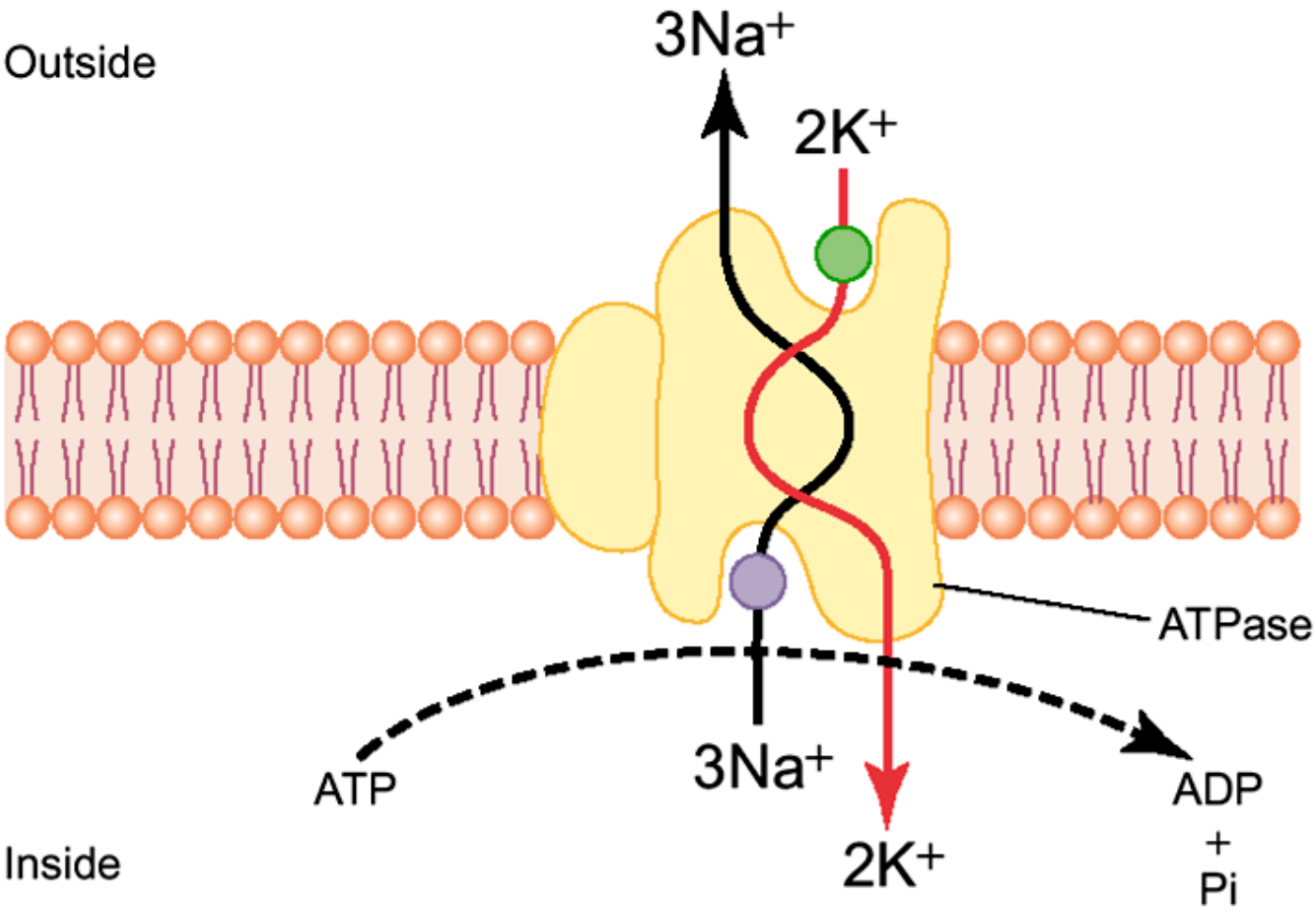


FIGURE — STRUCTURE OF THE SODIUM-POTASSIUM PUMP

Membrane potentials in different tissues are characterized by different values: the **highest one is in muscular tissue — 80–90 mV, in nervous — 70 mV, in connective — 35–40 mV, in epithelial — 20 mV**.

When the internal charge of the membrane becomes less negative, it is known as membrane ***depolarization***. If the internal charge of the membrane becomes more negative, it is called ***hyperpolarization***.

2. Membrane action potential (AP)

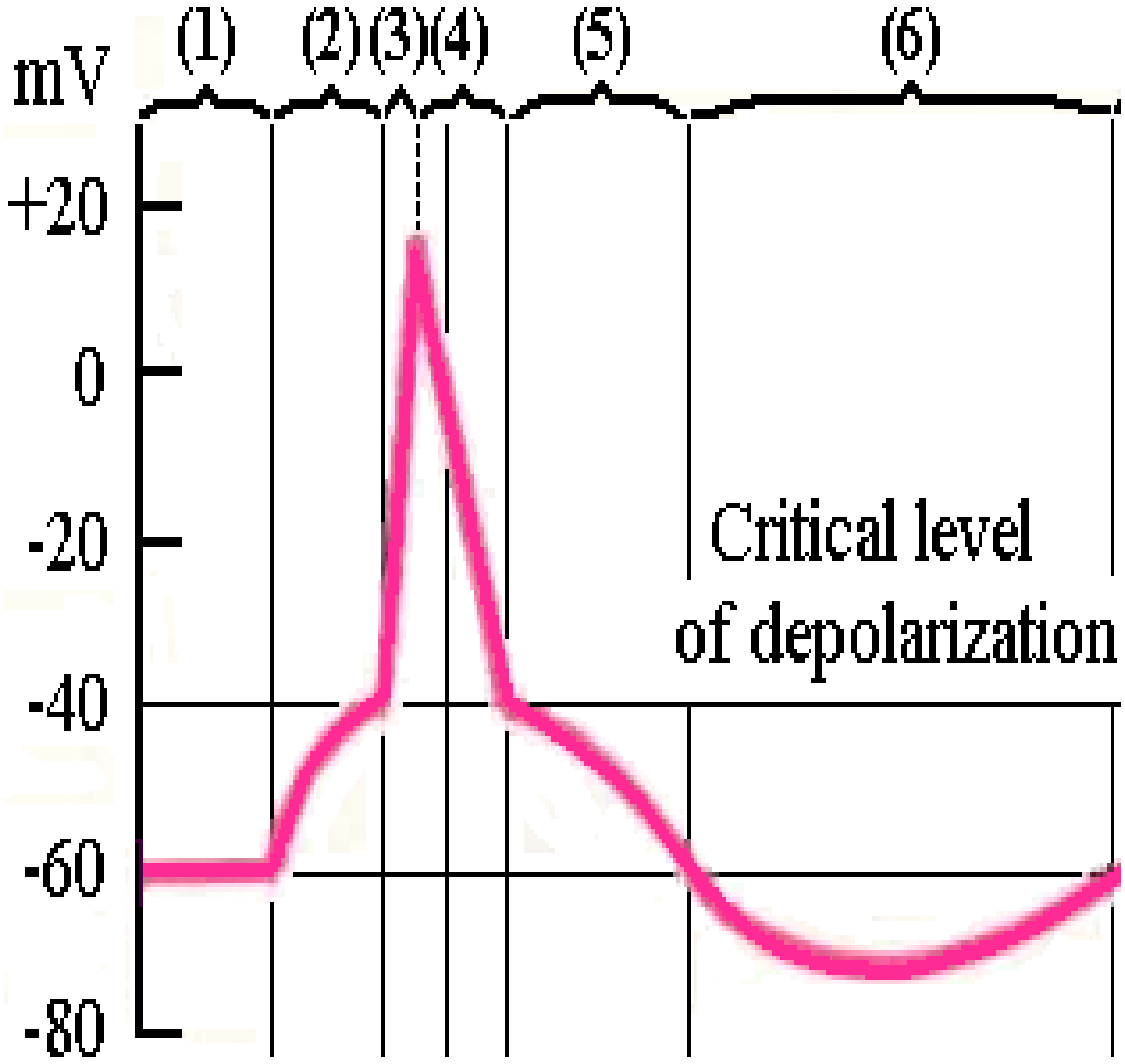
Being imposed by a threshold stimulus, the permeability of the membrane changes, and an ***action potential (AP)*** or excitation occurs (Figure). **AP is rapid fluctuations of the membrane potential during excitation.**

The threshold stimulus is the minimal strength which leads to the minimal response. To characterize the threshold stimulus, the concept of *rheobase* (in Greek, the root *rhe* translates to "current or flow", and *basi* means "bottom or foundation") is used.

Apart from the threshold stimulus, there are ***subthreshold stimuli*** which cannot generate responses but induce a shift in cell metabolism. Besides, there are ***superthreshold stimuli.***

Having arisen, AP goes along the membrane without changing its amplitude. ***It has the following phases***:

1. **Slow depolarization** (See figure «2»);
2. **Fast depolarization** (See figure «3»).
3. **Fast repolarization** (See figure «4»);
4. **Slow repolarization or negative afterpotential** (See figure «5»).
5. **Hyperpolarization or positive afterpotential** (See figure «6»).



Phases of the membrane action potential:

(2) Slow depolarization.
(3) Fast depolarization.
(4) Fast repolarization.
(5) Slow repolarization.
(6) Hyperpolarization.

FIGURE — MEMBRANE ACTION POTENTIAL

Mechanism of the AP origin (Figure).

Under the effect of the *threshold stimulus*, the cell membrane becomes permeable to ***Na^+ ions,*** which stream inside the cell at a high speed (the flow of Na^+ ions into cells is higher than the flow of K^+ ions outside cells). ***The internal side of the membrane becomes positive, and on its surface a negative charge is formed***. The changes of the charges on the internal and external surfaces of the membrane correspond to the ***depolarization*** phase (See figure «1»).

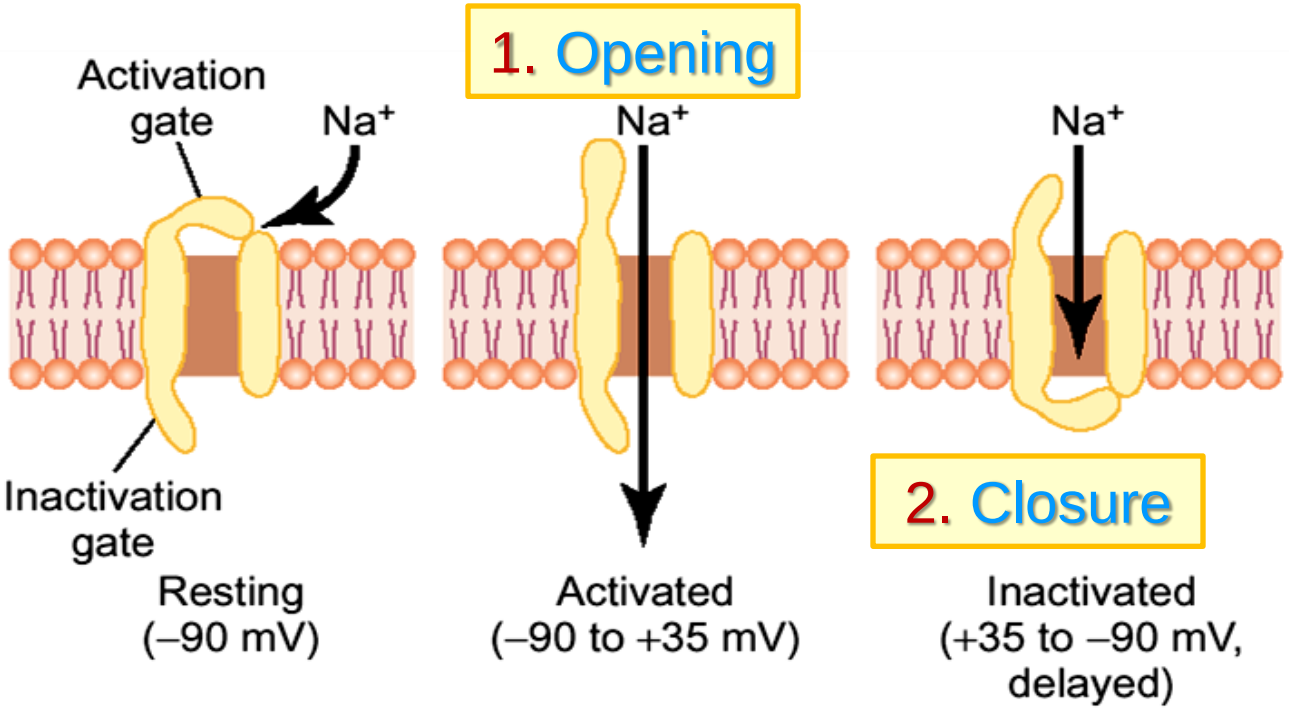
Afterwards the sodium channels close, and the potassium channels which have been partially closed open. K^+ ions go out of the cell. This AP phase is called ***repolarization*** (See figure «2»)

The action of the Na^+-K^+-pump and the RMP are restored (See figure «3»).

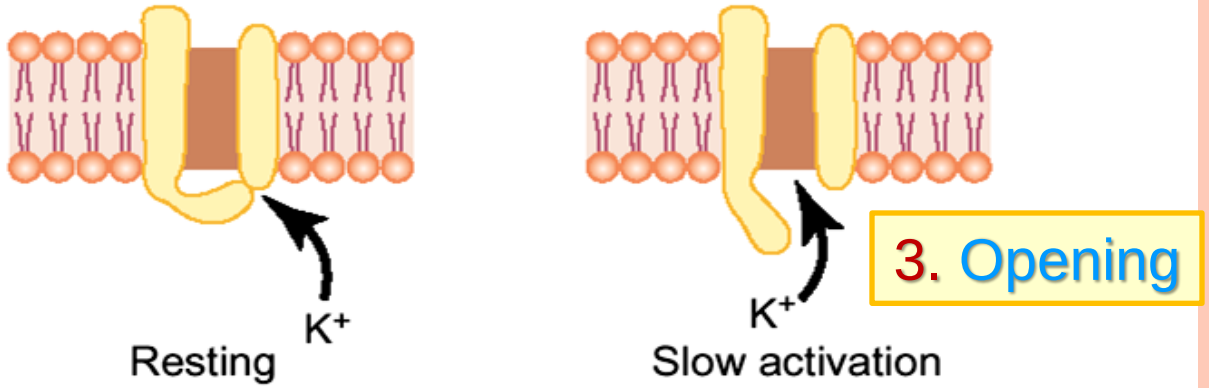
The basic role in the formation of AP belongs to Na^+ ions.

FIGURE — SUCCESSIVE OPENING AND CLOSURE OF CHANNELS

I. Sodium channel



II. Potassium channel



Changes of excitability during excitation

During the development of AP (excitation), the excitability of cells changes (Figure).

The development of the slow depolarization phase ***raises the excitability*** (hyperexcitability) creating conditions for a response. Further, when the slow depolarization phase is replaced by the fast one, the ***excitability rapidly reduces*** and, when the repolarization phase occurs, it starts ***to recover again***.

There are several periods of excitability:

1. Refractory period:

a) absolute;

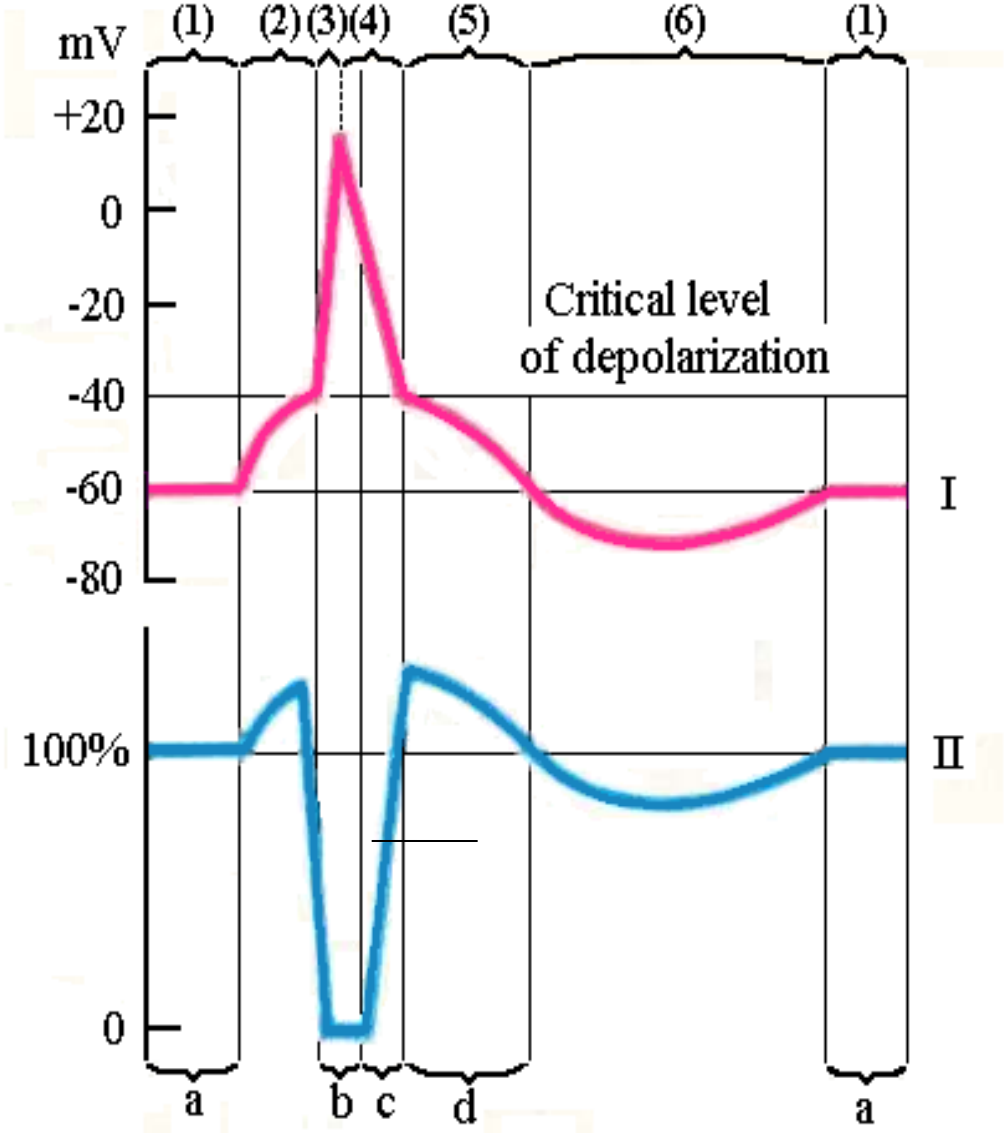
b) relative.

2. Supernormal or exaltation period.

The *refractory period* is an interval of time during which a cell cannot respond to the action of a stimulus. The sodium channels are inactivated. During the ***absolute refractory period*****,** the cell does not respond to the action of threshold or superthreshold stimuli.

The membrane repolarization leads to the reactivation of the sodium channels. This is the ***relative refractory period*****.** During this period, a response may appear under the action of the superthreshold stimulus.

During the ***supernormal period*****,** excitability exceeds the initial level. At this state the cell can respond to a stimulus the strength of which is a bit lower than the threshold one. The threshold of excitation is decreased because the values of the membrane potential are close to the critical level.



I — Changes of the membrane potential:	II — Changes of excitability:
(1) Membrane resting potential	(a) Normal excitability
Phases of membrane action potential:	
(2) Slow depolarization;	(b) Absolute refractory period
(3) Fast depolarization.	(c) Relative refractory period
(4) Fast repolarization;	
(5) Slow repolarization	(d) Supernormal or exaltation period
(6) Hyperpolar-tion	

FIGURE — CHANGES OF THE MEMBRANE POTENTIAL AND EXCITABILITY DURING EXCITATION

Laws of stimulation and assessment of excitability. Lability.

The excitability of tissue depends on the threshold of its irritability *(rheobase)*. ***Rheobase*** is the minimal strength of a stimulus that is able to cause excitation of tissue and induce the minimal response. The lower the strength of the threshold stimulus is, the higher the excitability of tissue is. However, the response of tissue depends on the strength of the stimulus to a certain extent.

The response of the cell also depends on the duration for which the stimulus is applied. The threshold strength of the stimulus is in the inverse relation with its duration.

The interrelations between the strength and duration of the stimulus are demonstrated by the ***strength-duration curve***. If the strength of the current is «1», to induce a response from tissue, the duration of the stimulus must be «a» (Figure).

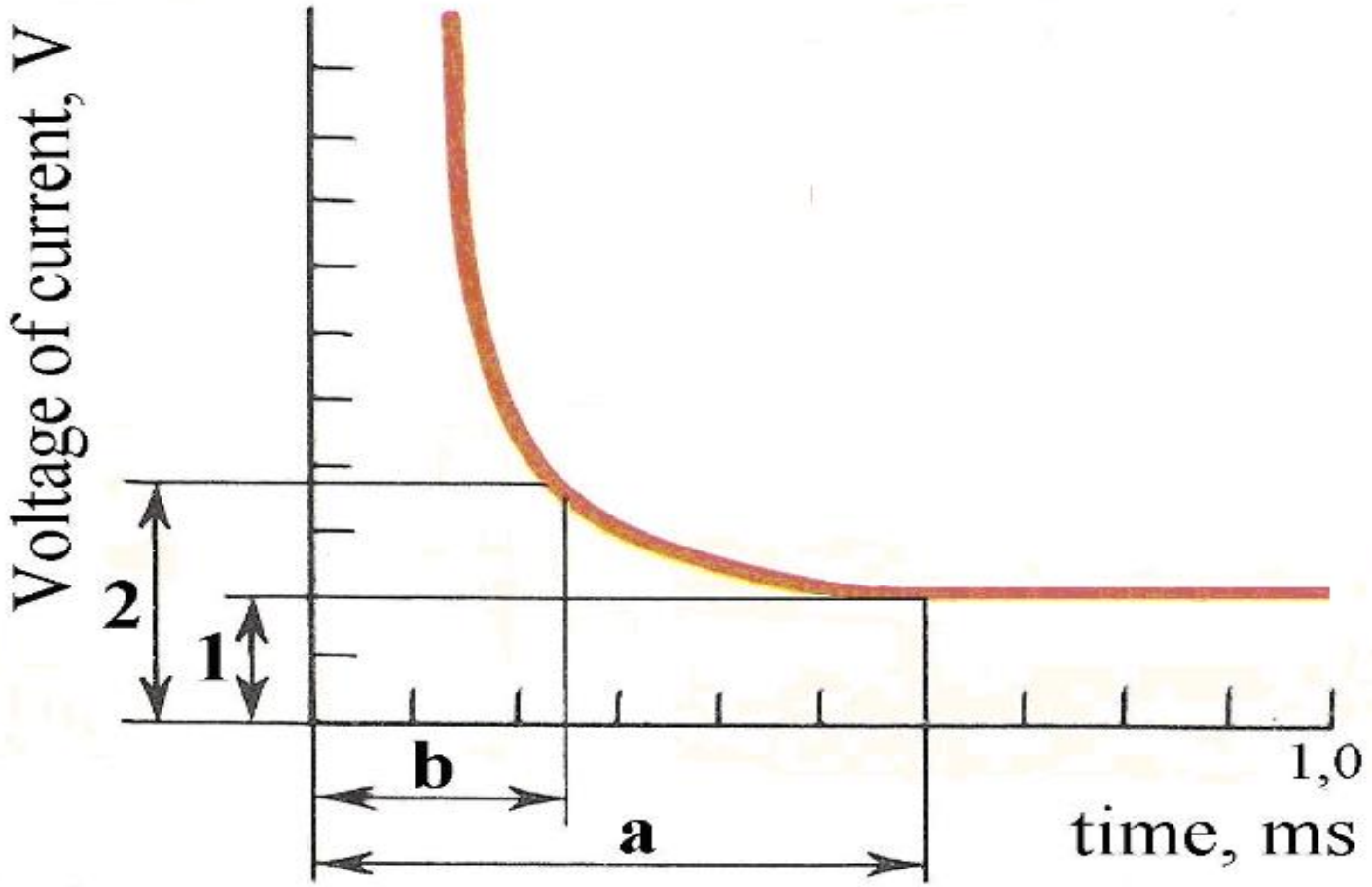


Figure — The strength-duration curve

Notes: 1 — rheobase; 2 — double rheobase; a — useful time; b — chronaxie

The shortest duration for which a stimulus equal to rheobase should react to induce a response is called ***the useful time*** (Figure). If to double the strength of the stimulus («2» — two rheobases), the duration of the stimulus necessary to induce the response decreases («b») (Figure).

The shortest duration for which a stimulus equal to double rheobase should be applied on tissue to cause a response is called ***chronaxie*****.**

If the strength of a stimulus is equal to half of rheobase (half of «1»), no response will arise regardless of the duration of the stimulus. For example, the reflex of withdrawing hands away from a cold iron will not occur.

If tissue is exposed to a stimulus whose strength is equal to triple rheobase, but whose duration is too short (half of «b»), no response will arise either. For example, if to touch a hot iron very quickly, it is impossible to feel its temperature (Figure).

Chronaxie characterizes the rate of excitation generation. In different tissues it varies, which is used for medical purposes, e.g. to determine the damage of motor nerves.

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Lability

To characterize the development of separate APs, the concept of *lability* is used. ***Lability*** **is the rate of the development of the response to a stimulus (separate APs).** The higher lability is, the more APs tissue can make per unit of time. The measure of lability is the highest number of stimuli to which tissue can respond by generating APs per unit of time. The maximal rhythm of excitation is limited by the duration of the absolute refractory period. If the refractory period lasts for 0.5 msec, the maximal rhythm is 1,000 impulses per second and more.

Nervous tissue possesses the highest lability. It can generate up to 1,000 impulses per second. Muscular tissue can conduct up to 500 impulses per second. Synapses are least labile. However, tissues cannot function at the maximum rhythm for a long time. In natural conditions tissue reacts to the excitation of a lower rhythm which can be kept for a long time. This rhythm is produced during the supernormal period and is therefore called ***optimal.*** In nerve fiber it is 500 impulses per second, in muscle fiber — 200 impulses per second.

During rhythmic excitation, lability can increase or decrease. Decreased lability leads to the development of the processes of inhibition and its increase determines the properties of tissues to adjust to a ***new higher rhythm*** of impulses. The adjustment to the higher rhythm is connected with the pumping of Na^+ ions out from the cytoplasm during excitation. Thus, muscles are capable to adjust to a more frequent rhythm of impulses coming to them from nerve fibers. For example, if after a long flight you see your parents at the airport, your tiredness disappears for a while. This is connected with the adjustment of your muscles to a higher rhythm coming from the nerve centers.

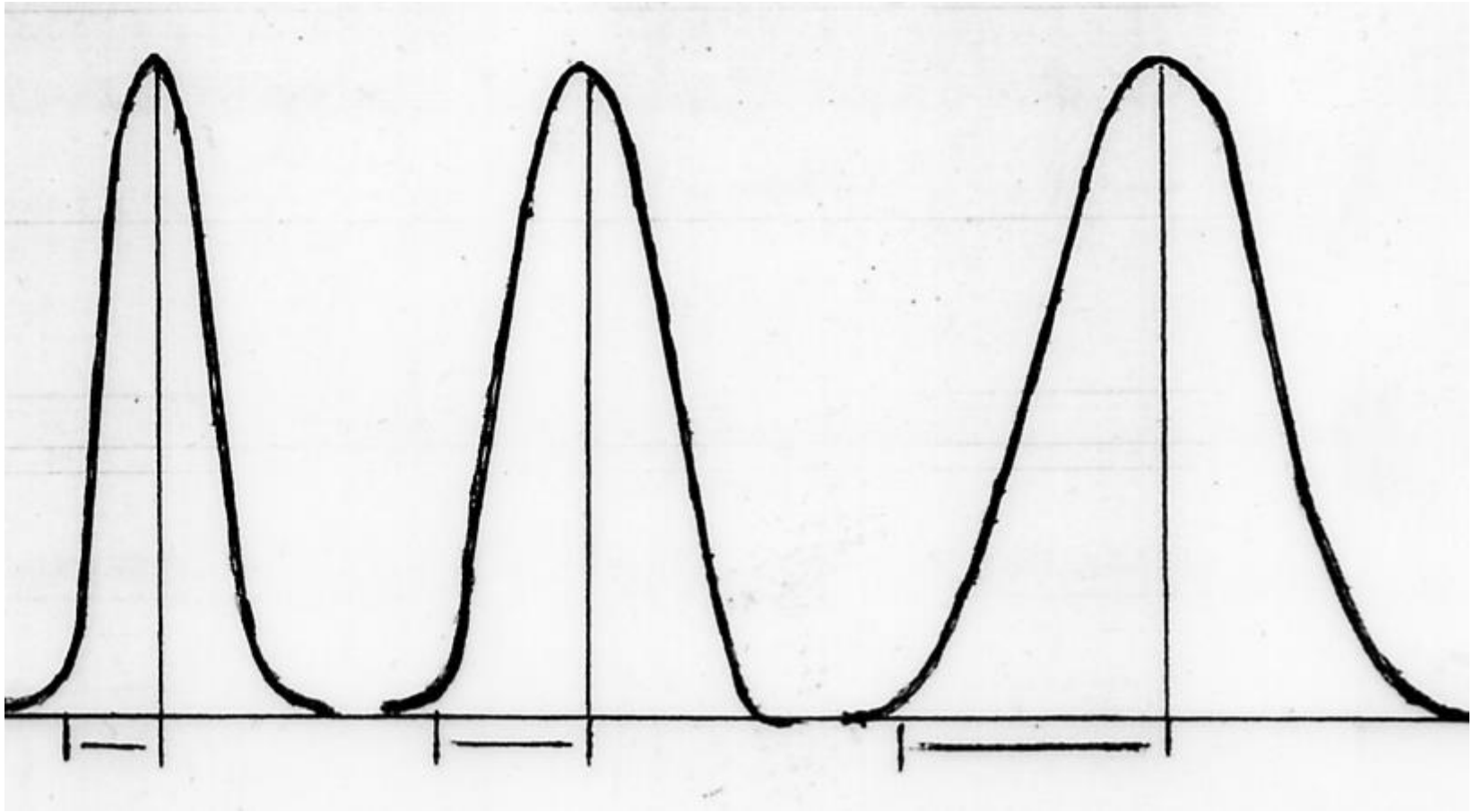


FIGURE — LABILITY OF VARIOUS TISSUES

3. Physiology of nerve fiber

Nerves specialize on the conduction of stimuli and connect the nerve centers with executing organs. Nerves consist of myelinated and unmyelinated fibers, which are coated with the connective tissue membrane (Table).

The surface of the axial cylinder of nerve fiber is coated with the plasma membrane, which performs the main role in the generation and conduction of excitement.

Table — Properties of different nerve fibers

Type of fibers	Diameter, mcm	Speed of conduction, m/sec	Functions
Aα (myelinated)	**13–22**	**70–120**	Efferent fibers conduct excitation to skeletal muscles, afferent fibers conduct excitation from muscle receptors
Aβ (myelinated)	**8–13**	**40–70**	Afferent fibers conduct excitation from touch and tendinous receptors
Aγ (myelinated)	**4–8**	**15–40**	Afferent fibers conduct excitation from touch and pressure receptors, efferent fibers conduct excitation to skeletal spindles
B (myelinated)	**1–3**	**3–14**	Preganglionic fibers of the vegetative nervous system
C (unmyelinated)	**0.5–1.0**	**0.5–1.0**	Postganglionic fibers of the vegetative nervous system, afferent fibers conduct excitation from pain, temperature, and pressure receptors

Myelinated fibers have an intercept sheath, which is formed by myelin segments 1–2 mm long (the myelin sheath). The gap between the two segments is called the ***node of Ranvier*** (Figure).

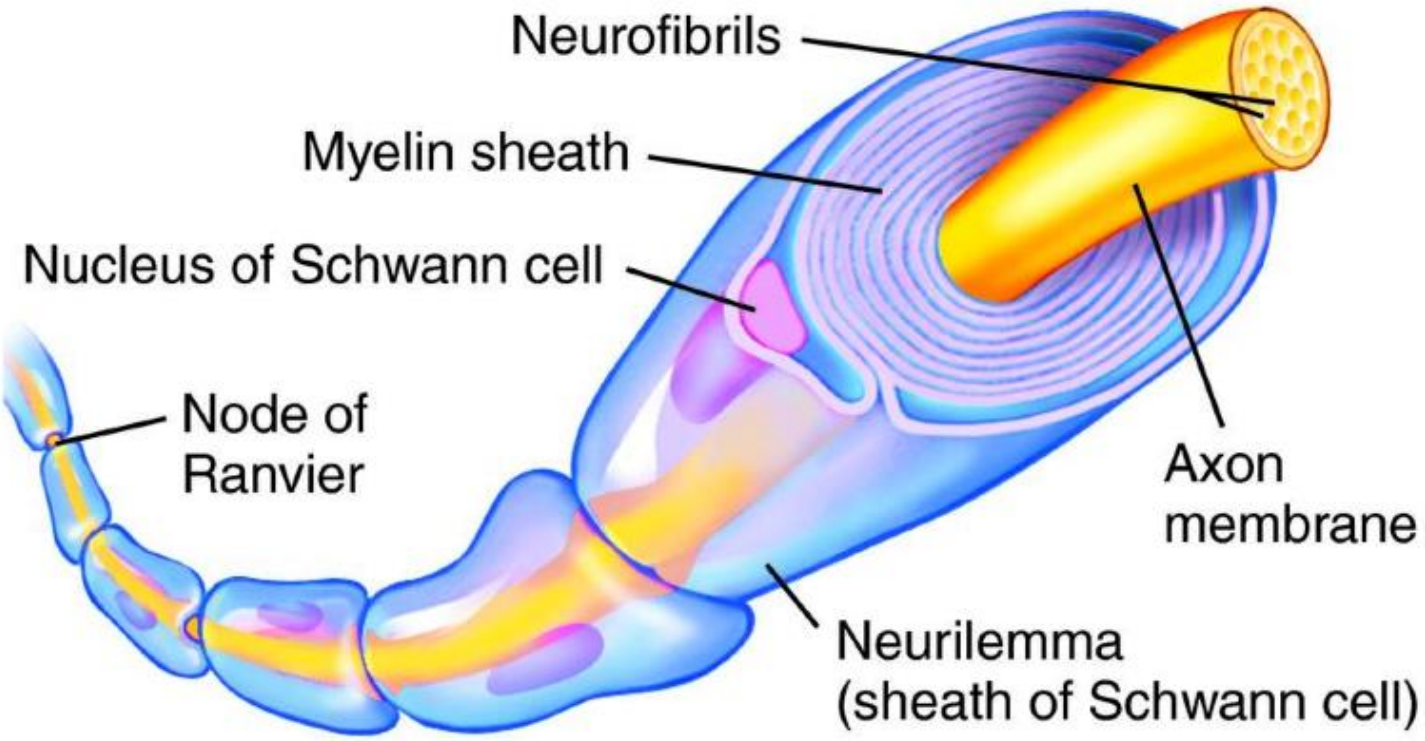


Figure — Structure of myelinated fiber

The myelin sheath is deposited around the axon by Schwann cells. The membrane of Schwann cells first envelops the axon. Then Schwann cells rotate around the axon many times, laying down the multiple layers of the Schwann cell membrane. Myelin is highly resistant and besides it performs the isolating function and takes part in the metabolism of nerve fibers. A signal along myelinated fiber goes only through the *nodes of Ranvier,* as they have many sodium channels.

Unmyelinated fibers are of the similar structure but have no myelin. Their surface is coated with Schwann cells.

If to dissect nerve fiber, its peripheral end after a while loses the ability to conduct signals and degenerates. Myelin undergoes fatty degeneration and transforms into fatty drops. The central end of nerve fiber is able to regenerate. A growth bulb is formed on it and grows towards the periphery (from 0.4 to 4.5 mm a day) and reaches the corresponding organ or tissue. Therefore, their innervations are recovered. Thus, the first signs of the regeneration of muscle innervations can appear after 5–6 weeks.

Laws of excitement conduction

- ***The anatomical and physiological integrity of fibers*** is essential. Dissection or compression affects the conductivity of nerves. If to cut a nerve and separate both the ends of the cut at a distance of 1 mm, excitation can skip from one end to the other only through myelinated fibers.
- ***Signals may propagate along nerves in both the directions***. This law is typical only for fibers isolated from the body, as inside the body signals are transmitted through synapses which conduct APs only in one direction.
- ***Isolated signal conduction*****,** i.e. a signal from one nerve fiber cannot skip to another one located in parallel.

Mechanisms of signal formation and conduction in myelinated and unmyelinated fibers

The mechanism of signal conduction in unmyelinated fibers. The action of the threshold stimulus on the unmyelinated fiber membrane changes its permeability to Na^+ ions, a great number of which flow inside the fiber. In this area the charge of the membrane changes (the internal becomes positive, the external — negative). It generates circular currents (movements of charged particles) from «+» to «–» along

Features of signal conduction along unmyelinated fibers:

- ***Signals go continuously and the whole fiber is seized with excitation.***
- ***Signals go at a low velocity.***

Along unmyelinated fibers signals go to the internal organs from the nerve centers. However, the low velocity of the signals and their fading are not always beneficiary to the human body. That is why nature made an additional mechanism: the conduction of signals along myelinated fibers.

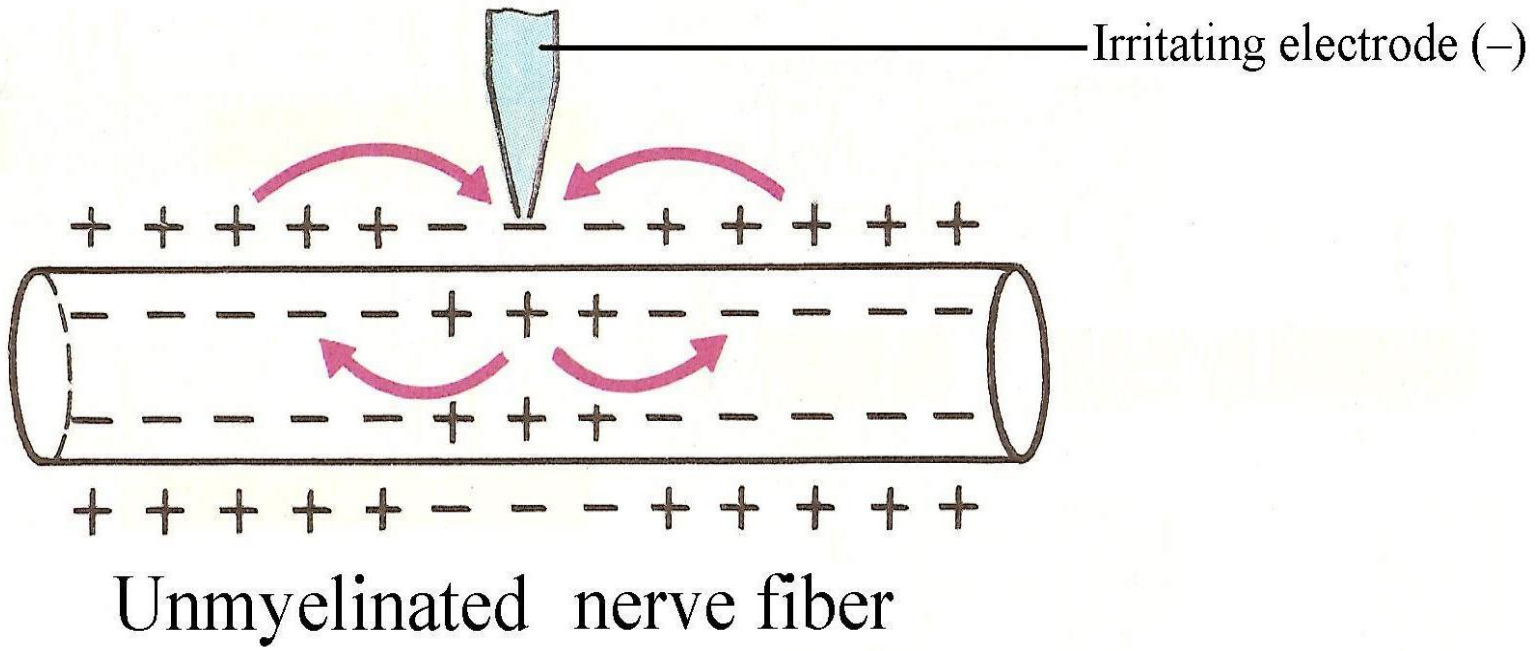


Figure — The mechanism of signal conduction in unmyelinated fibers

The mechanism of signal conduction in myelinated fibers (Figure). The action of the threshold stimulus on the membrane of myelinated fibers at the Ranvier's node changes the permeability to Na^+ ions, which go inside the fiber. In this part the charge of the membrane changes, which also generates circular currents. These currents go through the intercellular fluid to the adjacent node, where the charge changes again. Thus, the excitation transmits from one part to another. The reverse movement of the signal is impossible, as the part through which it has passed, is at the absolute refractory phase.

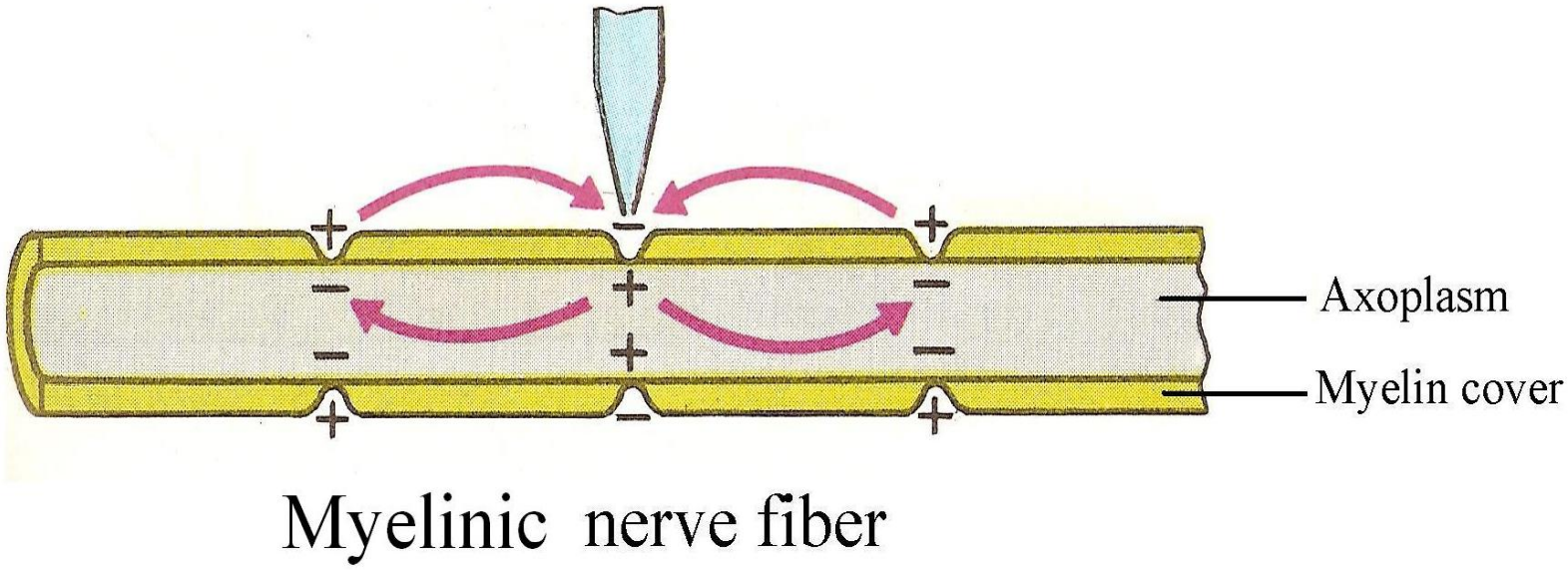


Figure — *The mechanism of signal conduction in myelinated fibers*

Thus, in myelinated fibers action potentials occur only at the nodes of Ranvier. *The action potentials are conducted from node to node,* and this is called saltatory conduction. Saltatory conduction is important for two reasons. Firstly, this mechanism increases the velocity of transmission of nerve impulses. Secondly, saltatory conduction conserves energy for the axon because only the nodes depolarize, therefore requiring less energy for re-establishing the difference between the sodium and potassium concentrations across the membrane after a series of nerve impulses.

Features of signal conduction along myelinated fibers:

- *Signals go in intermittent motion (saltatory conduction).*
- *Signals go at a high velocity.*

In myelinated fibers signals are transmitted from analyzers to the CNS, skeletal muscles i.e. where a high speed of responses is required.

4. Parabiosis

The scientist ***N.E.Vvedensky*** proved that a part of a nerve changes its lability under the effect of an alterant (irritant). This happens due to the fact that excitation lasts longer within this part and, therefore, at a certain stage of the alteration, the excitation is not transmitted through the nerve.

The condition of low lability, i.e. damage of the normal vital activity of the nerve is called *parabiosis*. Parabiosis can be observed under the action of narcotics, cold or heat, under the influence of currents and other stimuli.

The phenomenon of parabiosis was studied on the example of a nerve-muscle specimen which consisted of nervous cells, nerve fibers, and muscles, which reflected all the changes happening in nerve fibers (Figure). During the experiment, a stimulus (for example, some narcotic on cotton wool) was applied on some part of nerve fiber. Through this part the stimulus transferred and some changes could be observed.

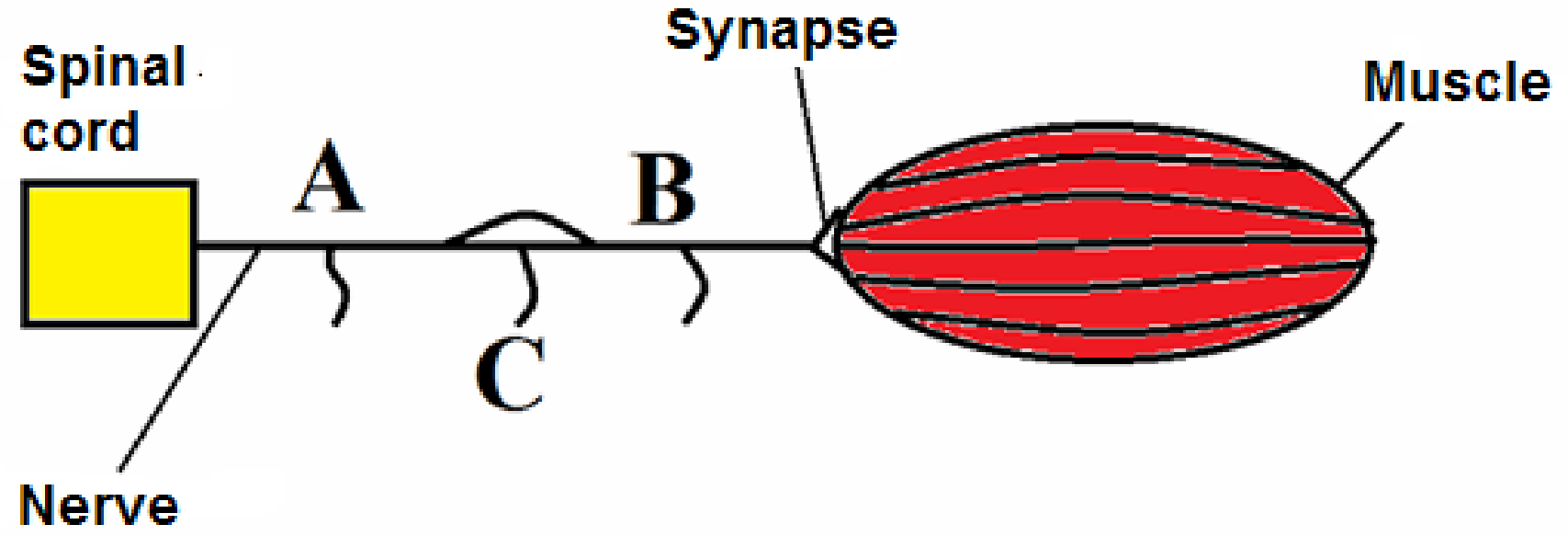


Figure — The scheme of the nerve-muscle specimen of parabiosis

A,B,C — electrodes: A — experimental, B — control,

C — in the area of the alteration influence

As a result of the experiment, **3 phases (Figure) of parabiosis** were detected:

1. Provisional or equalizing phase. If to irritate nerve fiber with stimuli of various strength (weak and strong), the response of the muscle will always be identical.

2. If the narcotic continues its action, there comes the second phase — ***paradoxical*****.** In this case strong stimuli induce weak responses, and, on the contrary, weak stimuli —strong ones.

3. If the effect of the narcotic is not stopped, neither strong nor weak stimuli can induce a response. This stage is called ***inhibitory*****.**

Then, if to terminate the effect of the narcotic and to wash the damaged part of the nerve, its properties are recovered in the inverse sequence.

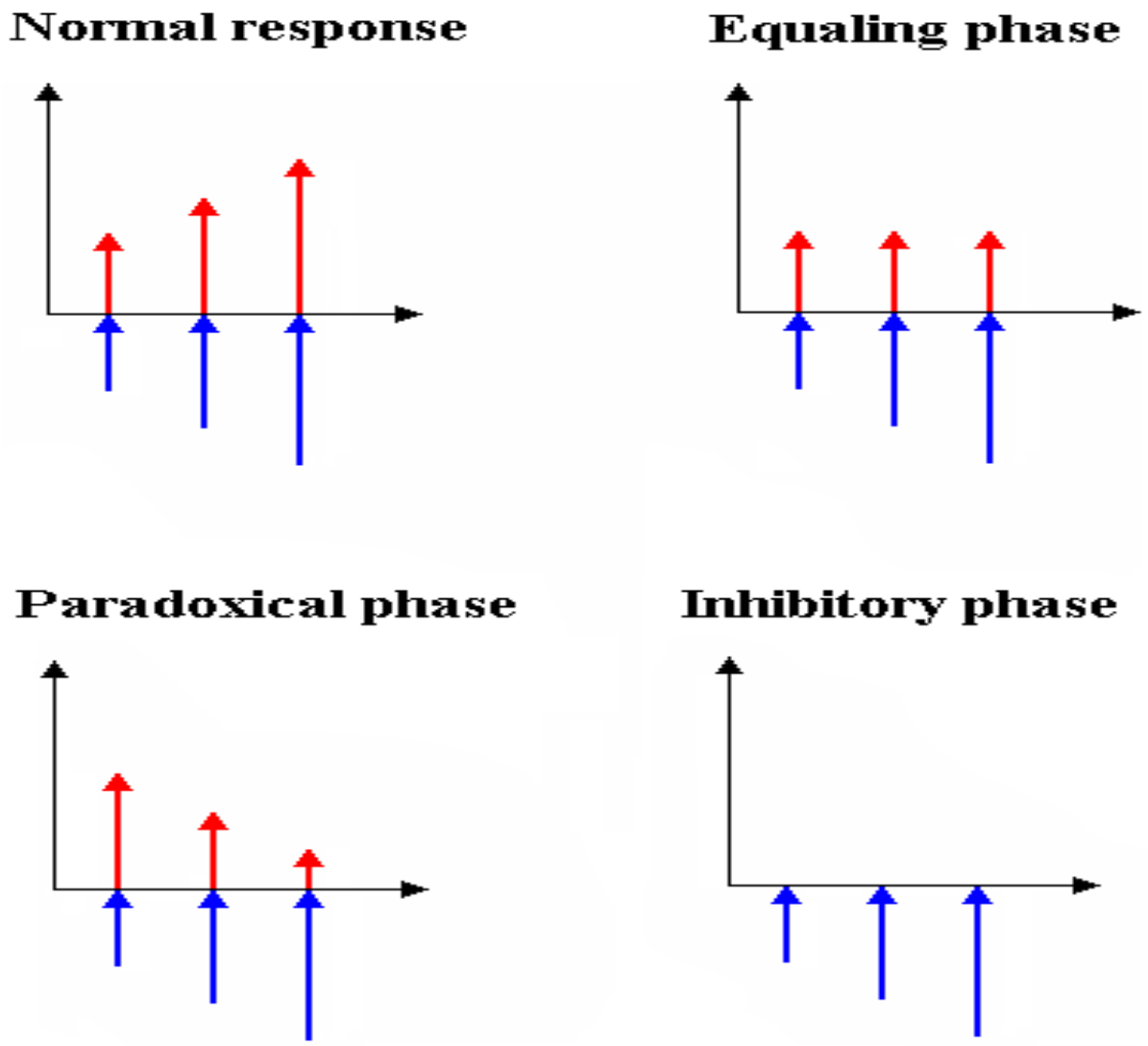


FIGURE — PHASES OF PARABIOSIS

NOTES: THE ARROWS ABOVE SHOW THE STRENGTH OF THE RESPONSE (THE FORCE OF THE MUSCLE CONTRACTION). THE ARROWS BELOW SHOW THE STRENGTH OF THE STIMULUS (CURRENT)

5. PHYSIOLOGY OF SYNAPSES

Synapses. Structure of synapses

Synapses **are specialized structures which provide the transmission of excitation from one neuron to another or to target effector cells.**

The functional role of synapses:

1. They provide functional contacts between nerves and organs.

2. They promote the regulatory activity of the CNS.

3. They have plasticity (the amount of signal which passes through a synapse can change, which is of an important functional value).

4. They participate in the formation of memory.

The structure of chemical synapses

Nerve fibers approaching a cell form a thickening which contacts with the cell. This part is the ***presynaptic membrane***. The opposite membrane is ***postsynaptic***. Between them there is a ***cleft*** filled with a plasma-like fluid. In the presynaptic terminal, there are ***neuromediators*****,** which are capable to excite or inhibit the innervated cell (Figure).

Myelinated nerve fibers approaching skeletal muscles make fanlike branchings into end fibers (terminals). The area of the synapse formation between the nerve terminations and muscles is called the ***motor end plate*****.** The postsynaptic membrane of muscle fibers is thicker and forms regular folds which increase the surface area of the postsynaptic membrane. Therefore, a big amount of the mediator may contact the postsynaptic membrane of muscle fiber.

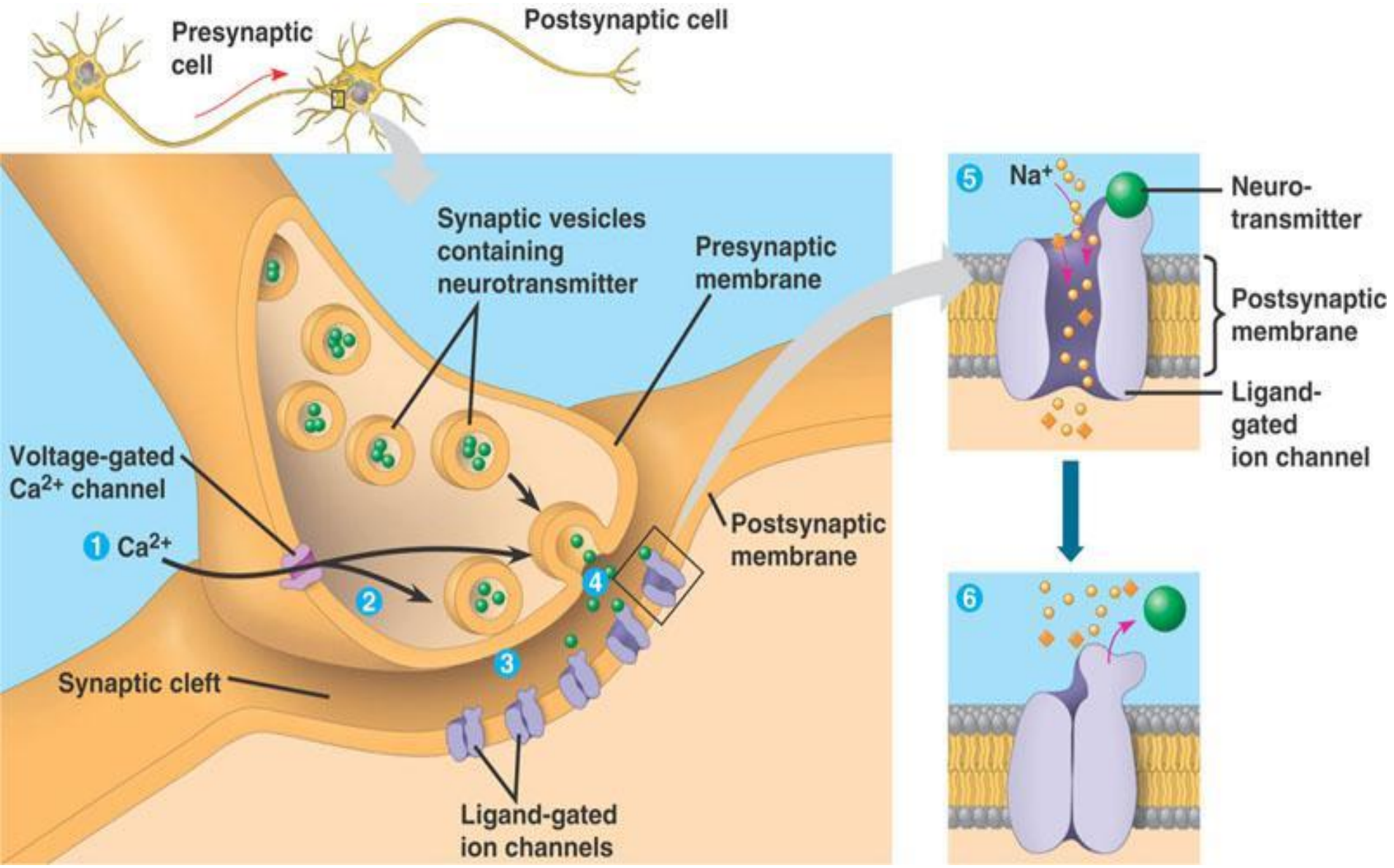
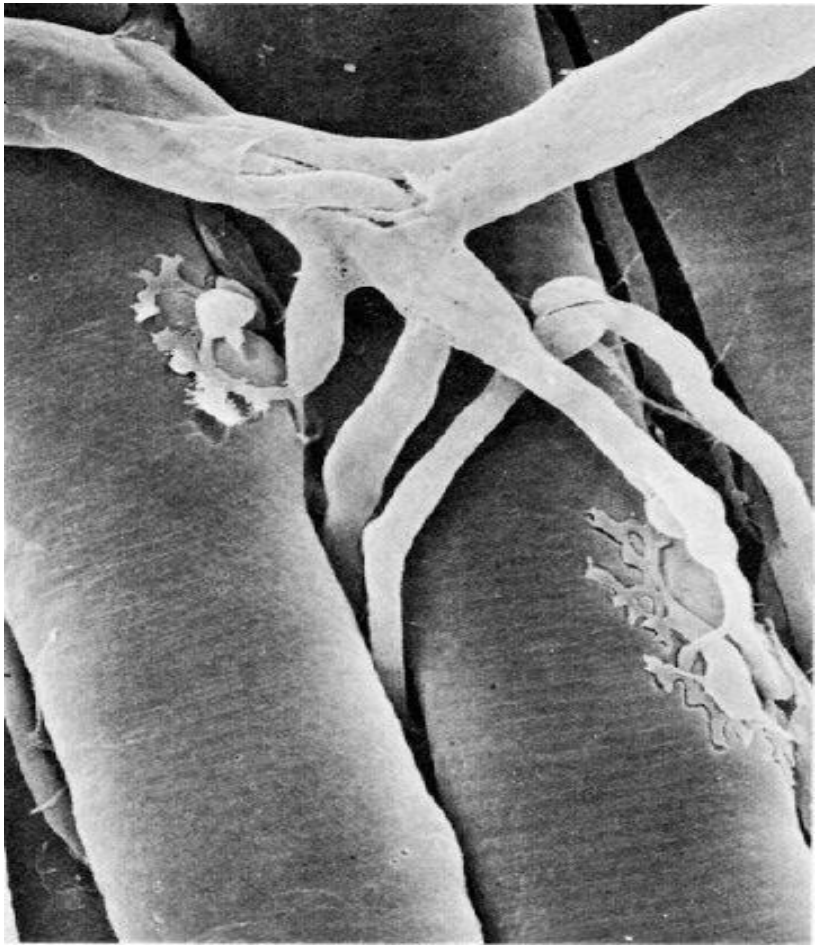


FIGURE — STRUCTURE OF THE SYNAPSE



NEUROMUSCULAR SYNAPSE IS THE SYNAPSE BETWEEN MOTOR NEURON AND SKELETAL MUSCLE CELL. NEUROTRANSMITTER – ACETYLCHOLINE

Figure — Neuromuscular synapse

Classification of synapses

1. By the location:

a) peripheral: neuro-muscular, neuro-secretory, receptor-neuronal;

b) central: axoaxonic, axosomatic, axodendritic, dendrodendritic, somatodendritic (Figure).

2. By the effect:

a) excitants;

b) inhibitors.

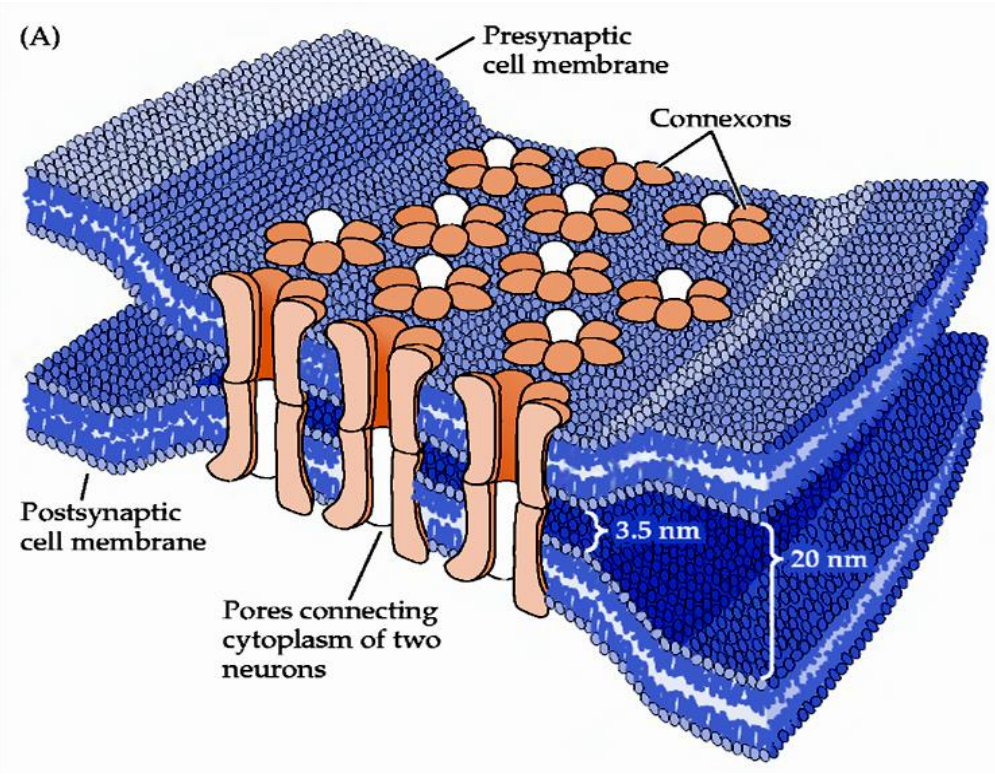
3. By the mechanisms of signal conduction:

a) chemical;

b) electrical. They conduct excitation without participation of the mediator at a high speed and have bilateral signal conduction. The structural basis of electrical synapses is the nexus. These synapses are located in the endocrine glands, epithelial tissue, CNS, and heart.

c) mixed.

In some organs excitation can be transmitted both through chemical and electrical synapses.



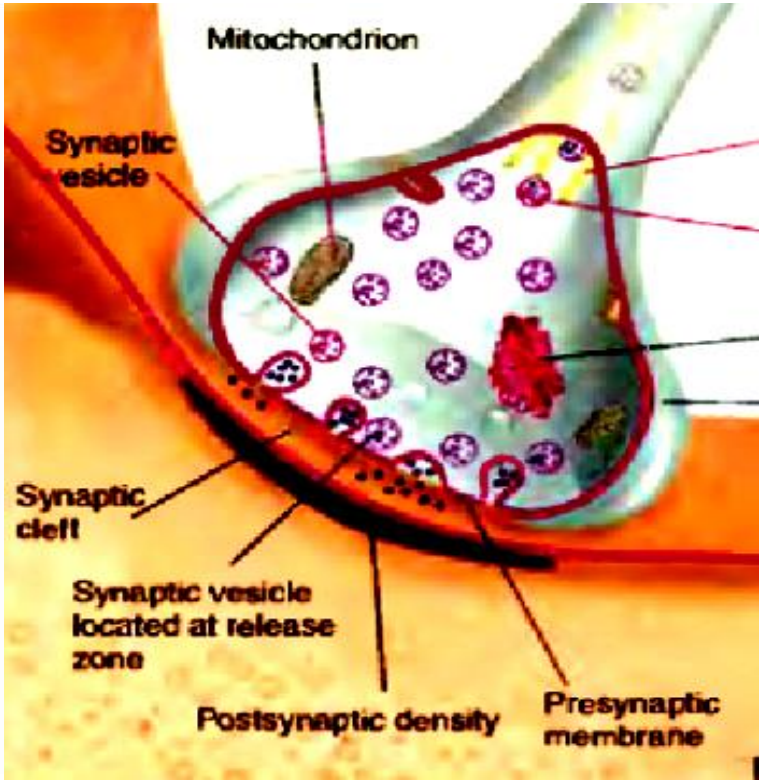


FIGURE — ELECTRICAL & CHEMICAL SYNAPSES

4. By the type of the secreted mediator, chemical synapses are classified into:

a) adrenergic (the mediator is noradrenalin);

b) cholinergic (the mediator is acetylcholine);

c) serotoninergic;

d) glycinergic and others.

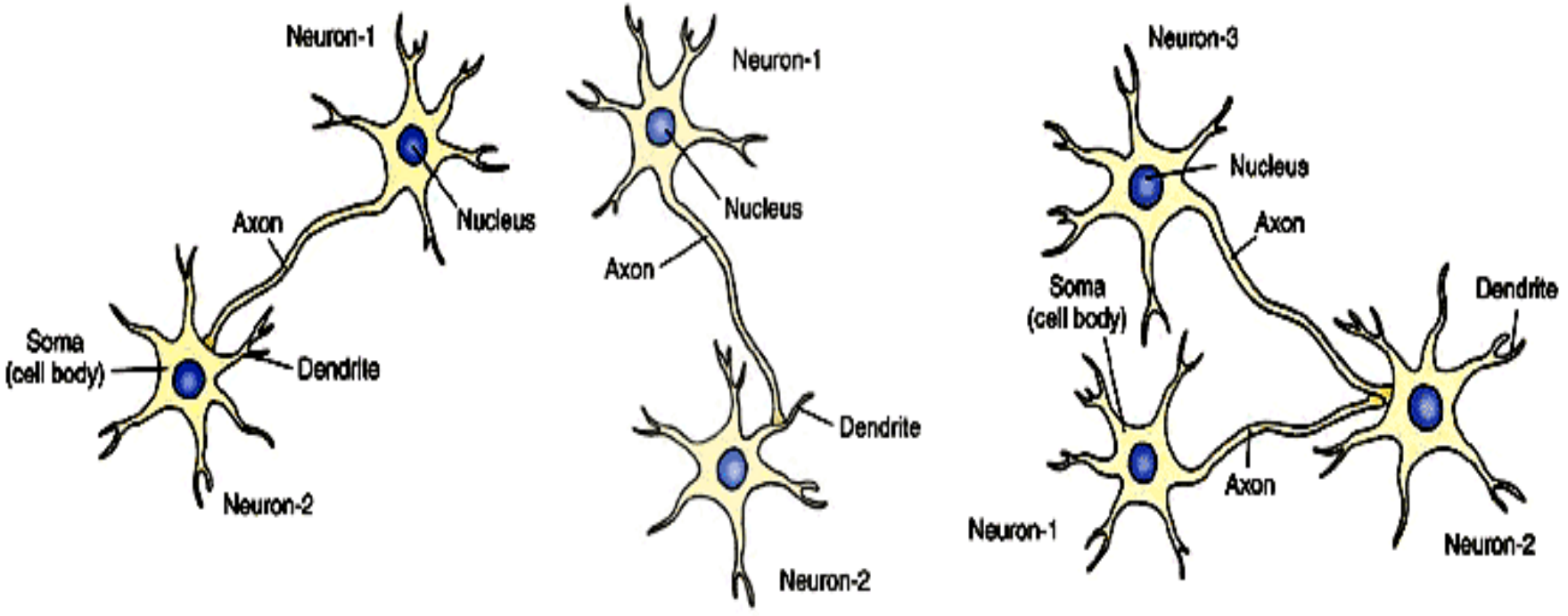


Figure — Classification of synapses

Chemical synapses have some common properties:

- Excitation in synapses is transmitted only in one direction. This is provided by the structure of synapses: the mediator is released only from the presynaptic part and it interacts with the receptors of the postsynaptic membrane.
- The transmission of excitation in synapses is slower than that in nerve fibers (synaptic delay).
- Excitation is transmitted with the help of special chemical substances – mediators (neurotransmitters).
- In synapses the transformation of the excitation rhythm occurs.
- Synapses have low lability.
- Synapses have rapid fatigability.
- Synapses have high sensitivity to chemical substances (including pharmacological drugs — blockers and others).

Mechanisms of signal transmission in chemical synapses (on the example of nerve-muscular synapses)

1. Release of the mediator into the synaptic cleft.

When APs reach the nerve termination (pre-synaptic membrane), they generate its depolarization. As a result, ***calcium ions*** go inside the terminal. The increase of the calcium concentration in the nerve termination promotes the release of ***acetylcholine*** into the synaptic cleft.

2. Diffusion of the mediator to the postsynaptic membrane and binding with receptors. The mediator reaches the postsynaptic membrane and binds with cholinoreceptors located on the postsynaptic membrane.

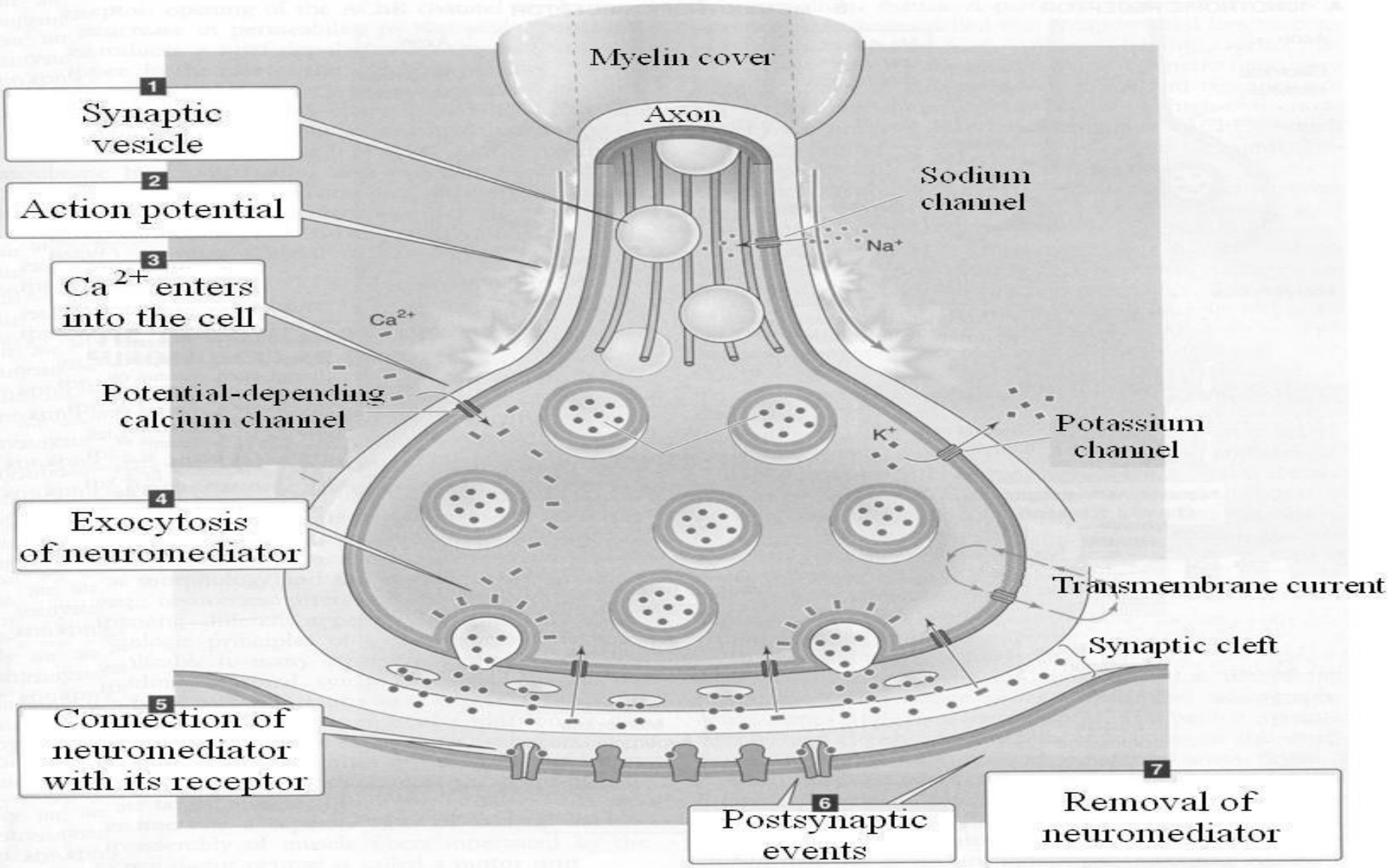
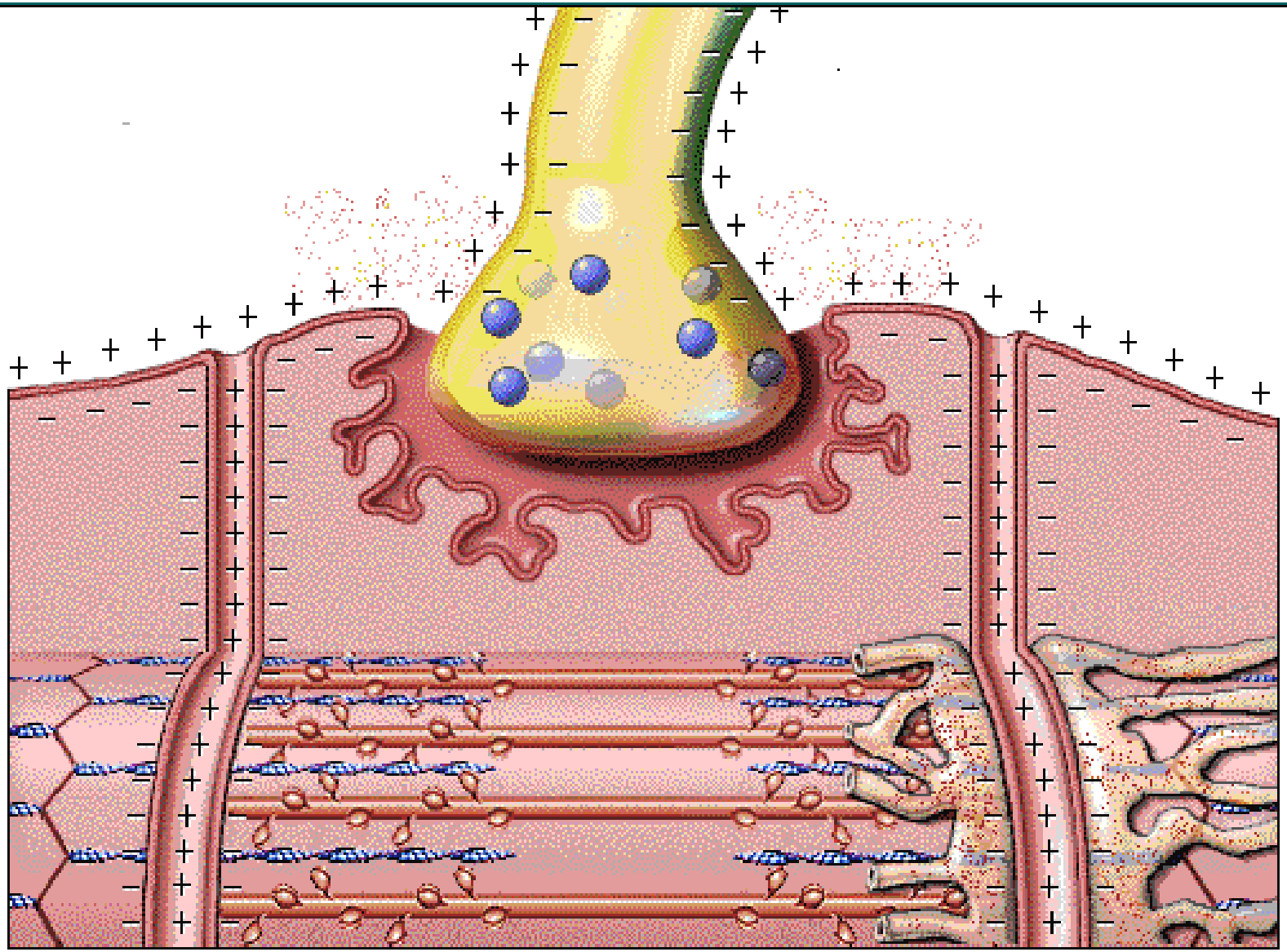


Figure — Stages of signal transmission in the synapse



3. The occurrence of excitation in muscle fiber. As a result of the interaction of acetylcholine with the receptors, sodium ions go through the postsynaptic membrane into the cell and depolarize the membrane (Figure).

If the initial level of the RMP is — 85 mV, it can decrease to 10 mV, i.e. partial depolarization occurs, the excitation does not go further, it stays in the synapse. These mechanisms cause a synaptic delay, which may last 0.2–1 ms. Partial depolarization of the postsynaptic membrane is called an ***excitatory postsynaptic potential (EPP)***.

Influenced by the ***EPP*** in the next part of the membrane of muscle fiber, there arises a ***propagating AP,*** which produces a muscle

In synapses the mediator depending on the chemical structure can cause depolarization of the postsynaptic membrane (the excitatory postsynaptic potential is formed, which provides the exciting effect) or hyperpolarization of the postsynaptic membrane (the inhibitory postsynaptic potential is formed, which provides the inhibitory effect).

4. The removal of acetylcholine from the synaptic cleft. The enzyme **acetylcholinesterase** is located on the external surface of the postsynaptic membrane. This enzyme disintegrates acetylcholine and inactivates it.

Some poisons and toxins like botulin can block the conduction of signals through synapses. For example, the poison *curare* contacts the receptors of the postsynaptic membrane and interferes their interaction with acetylcholine.

6. Perception of outside stimuli (reception)

Receptors are specific formations which transform energy of a stimulus into an electrochemical potential and then into the form of nervous excitation.

Classification of receptors.

By the character of sensations:

1) Visual.

2) Auditory.

3) Olfactory.

4) Gustatory.

5) Tactile.

By location:

1) Exteroreceptors — external (acoustic, visual).

2) Interoreceptors — internal (vestibular and proprioreceptors).

By the character of stimuli:

1) Photoreceptors — (visual).

2) Mechanoreceptors — (touches and pressure).

3) Thermoreceptors — (cold and warmth).

4) Olfactory.

5) Gustatory.

6) Painreceptor.

By the location of stimuli:

1) Distant — (auditory, visual).

2) Contact — (gustatory, temperature, receptors of pressure).

All the receptors have adaptation***.*** ***Adaptation*** is decreasing sensitivity to the long effect of a stimulus.

Transformation of stimulus energy

As a result of the interaction of a stimulus and the receptor membrane, a ***receptor potential (RP)*** appears. How does it happen?

During the contact of the stimulus with the receptor membrane there is an increase of the permeability of the membrane to ***sodium ions*** and they get into the sensory terminal, which is depolarized, and a ***RP*** is formed.

The initial conversion of the stimulus into the RP is called transformation.

The RP excites the initial segment of the sensory nerve generating a nervous impulse. The frequency of nervous impulses depends on the RP amplitude.

There are ***primary-sensitive receptors,*** which represent the endings of sensory nerves, and ***secondary-sensitive receptors*** — separate cells which receive stimulation. These cells are in contact with the endings of sensory nerves. From these cells the mediator is released and this results in the formation of a nervous impulse. A set of receptors that cause excitation of their own neurons is called ***the receptive field***; and the areas of the concentration of receptors belonging to certain sensory systems are called ***the reflexogenic zones***.