

Action potential (physiology)

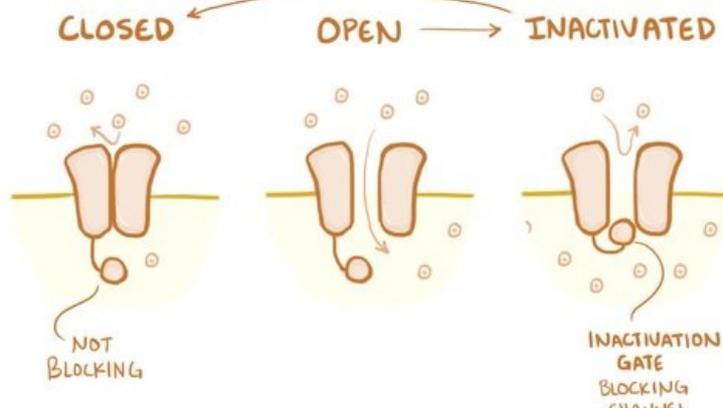
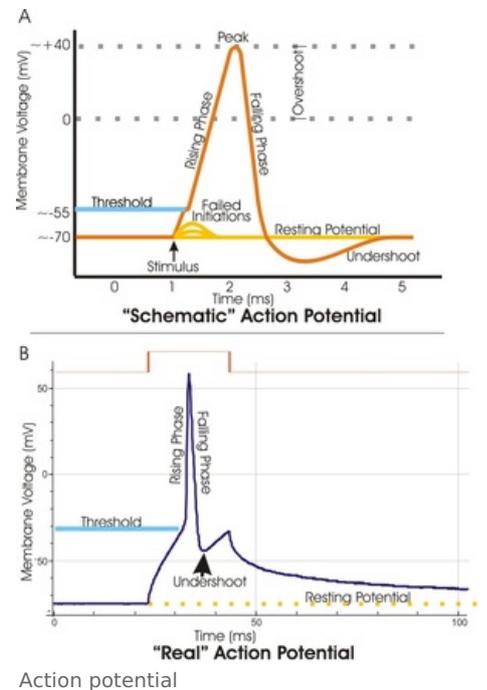
The action potential (excitation) arises from a **change resting equilibrium** (change in polarization) on the membrane. This change is caused by a spreading voltage that alters the activity of **voltage-gated** ion channels.

Principle of action potential formation

Excitation occurs at the point where voltage-gated ion channels predominate. They follow the law of **all or nothing**. This means that the excitation will only be produced by a sufficiently intense stimulus, which we call a threshold stimulus (usually 5 to 15 mV higher than the KMP value)^[1]. At this value, **opening of Na⁺** voltage-gated ion channels occurs. Sodium cations then penetrate into the intracellular space (according to the concentration and electrical gradient). The inner side of the membrane becomes more **positive** compared to the outer side. We call this phenomenon "depolarization". In the case when the outer side of the membrane is more electronegative than the inner side, we speak of transpolarization.

Along with Na⁺ channels, **delayed K⁺ channels** are also slowly opening. The opening of these channels reaches its maximum when the Na⁺ channels close. Due to the flow of potassium ions in the direction of its concentration gradient, the polarity begins to **return to the original values** and we speak of **repolarization**. If there is a **deepening** of the original membrane potential, we speak of **hyperpolarization**. Achieving the original values of the resting membrane potential is conditioned by the activity of the Na⁺/K⁺ATPase, which pumps ions against their concentration gradient.

 For more information see *Action Potential (Biophysics)*.

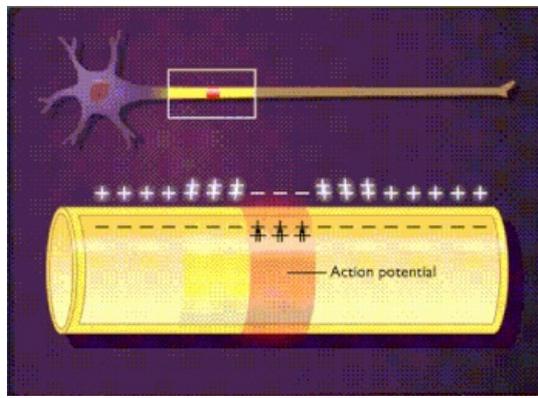


Refractory period

A condition where it is not possible to excite a nerve cell so that an action potential occurs. Refractory period can be **relative**, which is a state where we are able to create an action potential with a **suprathreshold stimulus**. Furthermore, we can talk about the **absolute refractory period**, during which **no stimulus** can cause excitement.

Excitement conduction

The action potential spreads **without decrement**, in other words, it does not lose its intensity. This is due to the creation of **local currents**. Local currents arise above the place where the ion distribution has changed. The consequence of the formation of these currents is the depolarization of the membrane of the **adjacent section** of the fiber. Local currents **accelerate conduction of excitation**. If the sections are extended (e.g. by insulation with myelin sheath), local currents only occur in the next non-insulated section and this enables a much faster transmission of excitation than in unmyelinated fibers. This is called **saltatory conduction**.



Action potential of various tissues

The action potential described here applies generally to all cells capable of generating an excitation. This general model is described on the nerve cell. For different cells, the action potential differs in e.g. the value of the threshold stimulus, the duration, the ion channels that maintain it, the course of the AP curve, etc.

 For more information see *Action potential in the heart*.

 For more information see *Pacemaker potential*.

Kalemia

Potassium = higher ECT potassium values slightly depolarize the membrane, as the concentration gradient for K^+ is limited, so complete repolarization after depolarization is not possible. The cell is not excitable. This is the principle of spreading potassium depression. [2]

In this hyperkalemia, first the excitability increases, then there is a block of voltage-gated channels and the excitability decreases (>5.3 mmol/l) - AP shortening. The voltage reaches the threshold more easily. = depolarization.

On the contrary, with hypokalemia (<3.7 mmol/l), hyperpolarization, prolongation of AP occurs, it is not easy for the neuron to reach the voltage threshold.

Links

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- Membrane potentials
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- Cell membrane
- Ion channel
- Ion pumps
- Sodium-potassium pump
- Action and Summation potential
- Action potential in a heart
- Pacemaker potential
- Action potential (biophysics)

Used literature

- KITTNAR, Otomar. *Lékařská fyziologie*. 1st edition. Praha : Grada, 2011. 790 pp. ISBN 978-80-247-3068-4.
- MYSLIVEČEK, Jaromír. *Základy neurovědy*. 2nd edition. Praha : TRITON, 2009. 390 pp. ISBN 978-80-7387-088-1.
- TROJAN, Stanislav. *Lékařská fyziologie*. 4th edition. Praha : Grada, 2003. 772 pp. ISBN 80-247-0512-5.

References

1. KYMPLOVÁ, Jaroslava. *Katalog metod v biofyzice* [online]. [cit. 2012-09-20]. <<https://portal.lf1.cuni.cz/clanek-793-katalog-metod-v-biofyzice>>.
2. MYSLIVEČEK, Jaromír a Vladimír RILJAK. *Fyziologie: repetitorium*. Praha: Stanislav Juhaňák - Triton, 2020. ISBN 978-80-7553-818-5.

