Postsynaptic potentials

Postsynaptic potentials arise at the site of the postsynaptic membrane (let's say the dendrite of a neuron) by a change in the relative permeability of the membrane to ions, which is determined by the opening of certain chemically controlled channels. There is a deviation of Resting membrane potential either to more negative values (*hyperpolarization*) or to more positive values (*depolarization*).

Hyperpolarization leads to the opening of chemically **gated** channels for:

- **K^+** : the relative permeability of the membrane for K^+ increases in this section and the KMP value shifts more towards the E_{K^+} value (−90 mV),
- **Cl^-** : similarly, there is a shift of KMP towards the EC value of Cl^- (−71 mV).

Depolarization is caused by the opening of chemically gated channels for:

- **Na^+** : an increase in membrane permeability to Na^+ causes a proportional rise in the KMP value (E_{Na^+} is 62 mV).

Conduction of postsynaptic potentials

The formation of a postsynaptic potential causes a **voltage difference** on the membrane between the place of its formation and the remaining membrane, where the original KMP value is still present. The difference in voltage causes an electric current to flow, which tries to cancel this imbalance. Since the membrane of dendrites does not contain *voltage-controlled channels* , currents spread along the membrane "like a wire" in all directions from the point of origin of the postsynaptic potential, we speak of passive conduction.

What does "like a wire" mean? To give you an idea: you apply an electrical voltage source to the middle of a conductor ("wire") for a few ms:

- this voltage pulse will of course arrive at both ends of the conductor at the same time
- its size will be smaller than the original one (current losses due to electrical resistance of the conductor)
- the speed of propagation will be close to the speed of light.

So everything has its advantages and disadvantages - the propagation almost at the speed of light is redeemed by the loss of the size of the original PSP due to the high resistance of the membrane, this voltage drop is called *decrement* , so it is a **line with decrement** .

Generation of postsynaptic potentials

Postsynaptic potentials depend on the type of mediator and synapse.

Two main types of potentials arise in the organism:

1. The **excitatory postsynaptic potential (EPSP)** is caused by excitatory mediators. In the postsynaptic membrane, Na^+ (Ca^{2+}) channels open and ions enter the cell, causing depolarization. A single EPSP represents a depolarizing change that is deeply subthreshold (2−4 mV). However, EPSPs add up (temporal and spatial summation), so a threshold level (7.5-15 mV) can be reached at which an action potential is generated in the efferent part of the synapse.
2. The **inhibitory postsynaptic potential (IPSP)** is caused by inhibitory mediators. There is an opening of K^+ and Cl^- channels and a flow of positive ions out of the cell and negative into the cell. The membrane is hyperpolarized by ion movements and the excitability of the neuron decreases. The IPSP value is in the range of 2-5 mV.

Combining EPSPs and IPSPs on the same membrane results in **signal summation**. If the inhibitory signal sufficiently reduces the excitatory one, no action potential occurs.

Slow postsynaptic potentials consist only in the regulation of K^+ channels. An IPSP occurs when channel permeability is increased, an EPSP occurs when channel permeability is decreased. These potentials have a long latency (100-500 ms) and duration (seconds-minutes).

Regulation of excitation transfer

Presynaptic inhibition can occur in two ways. The first of these is *axo-axonal inhibition* , in which the axon of an inhibitory neuron acts on a presynaptic neuron. *Autoinhibition* consists in binding of the mediator to the receptors of the own presynaptic membrane, from which it is flushed out. Further neurotransmitter release is inhibited, preventing overstimulation of the postsynaptic membrane.

Postsynaptic inhibition is also possible with the help of an inhibitory neuron, which here, however, acts on the postsynaptic neuron. Autoinhibition can occur afferent collaterally - the collateral from the presynaptic neuron activates an inhibitory interneuron that inhibits the postsynaptic neuron. Efferent collateral connections lead from

the postsynaptic neuron through the inhibitory interneuron back to the postsynaptic one.

Presynaptic facilitation and summation occurs especially when two neurons converge to one. Subthreshold stimuli can then trigger an EPSP, and the subsequent summation of these signals produces an action potential. Suprathreshold stimuli usually evoke a prolonged action potential.

Occlusion is a special type of summation in which two suprathreshold stimuli evoke a normal action potential (not, e.g., prolonged as in presynaptic facilitation and summation).

Links

Resources

TROJAN, Stanislav – ET AL.,. *Lékařská fyziologie.* 4. edition. Grada, 2003. 772 pp. pp. 65–67. ISBN 80-247-0512-5.

A KOL., Jaromír. *Základy neurověd.* 2. edition. Praha : Triton, 2009. 390 pp. ISBN 978-80-7387-088-1.

KITTNAR, Otomar, et al. *Lékařská fyziologie.* 1. edition. Praha : Grada, 2011. 790 pp. ISBN 978-80-247-3068-4.