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The Physiological Role Of Mediators In The Central Nervous System.

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ABSTRACT

In the central nervous system, the mediator function is performed by a large group of dissimilar chemicals. According to the latest data, there are about 30. According to the principle of Dale, each neuron in all its synaptic endings allocates one mediator. Proceeding from this principle, it is customary to designate neurons as a mediator, which distinguish their endings. In response to a nerve impulse from the presynaptic nerve endings through a small time interval, called a synaptic delay, there is an ejection from several units to several hundred quanta of the neurotransmitter, which, interacting with the receptors on the membrane and the effector cell, generate postsynaptic responses. The regulation of interactions in the central nervous system through the mediators produced by cells is an important component of intercellular communication. At present, there are many methods of active release of humoral factors from cells, which exert paracrine and autocrine influence in multicellular ensembles (for example, exocytosis, secretion in membrane microparticles, release through connexin and pannexin channels). Therefore, further study of the mechanisms of intracellular interaction in the central nervous system with the help of biologically active molecules is of great importance for fundamental science and medical practice.

Keywords: brain, mediators, central nervous system, synapses, regulation.

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INTRODUCTION

The main structural unit providing the process of intercellular communication of information in the nervous system is a synapse of chemical type. The main principles of the operation of such synapses are similar both in the central nervous system and in the peripheral nervous system [1].

In response to a nerve impulse from the presynaptic nerve endings through a small time interval called synaptic delay, a release from a few to several hundred quanta of neurotransmitter that interacts with receptors on the membrane and effector cells, generate postsynaptic responses [2].

According to modern ideas, there are two forms of stimulation-induced release of quanta of the neurotransmitter: phase synchronous and delayed asynchronous, which differ in intensity, degree of synchronism of release of quanta, mechanisms involved in their implementation, and contribution to the process of information transfer in the synapse [3].

The time parameters (kinetics) of the evoked release of the mediator, along with the number of released quanta, are characteristic of the neurosecretory process [4].

In the central nervous system, the mediator function is performed by a large group of dissimilar chemicals. The list of newly discovered chemical mediators is steadily increasing. According to the latest data, there are about 30. According to the principle of Dale, each neuron in all its synaptic endings singles out the same mediator. Proceeding from this principle, it is customary to designate neurons as a mediator, which distinguish their endings [5].

Thus, for example, neurons releasing acetylcholine are called cholinergic, serotonin-serotonergic. This principle is used to denote various chemical synapses.

Purpose: to trace the basis of presynaptic mechanisms of regulation of the nervous system in humans.

Quantum bases of secretion of mediators

By analogy with other fields of knowledge in neurobiology, a quantum of a neurotransmitter is understood as a relatively stable portion of signal molecules capable of simultaneously being released into the extracellular space spontaneously or by depolarization of the nerve ending by the action potential [6].

The overwhelming number of experimental data obtained using a wide range of methodological approaches, starting with Katz's works up to the present day, fit within the framework of the so-called "quantum-vesicular hypothesis" of mediator isolation, which is based on the postulate: one quantum is the contents of one synaptic vesicle. The release of the quantum is carried out by means of calcium-dependent exocytosis, during which the membrane of the synaptic vesicle merges with the membrane of the nerve end and the subsequent release of the contents of the vesicle into the synaptic cleft. The mediator released into the extracellular space diffusion rapidly reaches the postsynaptic membrane, where it interacts with the target cell receptors. In the event that these receptors are chemically activated ion channels, when interacting with them the mediator molecules stored in one synaptic vesicle through the postsynaptic membrane, an electric current flows, leading to a small change in the potential in the synaptic zone, denoted as a miniature postsynaptic signal. With regard to the neuromuscular synapse, these signals range from a few tenths to millivolts, called miniature potentials (or currents) of the end plate. In response to the action potential of the axon, enough synchronously is allocated from units to several hundred quanta of the mediator. The number of quanta of the mediator that led to the appearance of a specific induced postsynaptic response began to be denoted by the term "quantum composition" of the postsynaptic signal [7].

Thus, the characteristics of the intensity of spontaneous quantum secretion is the average frequency of miniature postsynaptic responses, and the induced frequency is the average quantum composition of the induced signals.

Biological action of acetylcholine

Acetylcholine is a neurotransmitter mediator. It is synthesized in the body from the amino alcohol of choline and acetic acid. Biologically, it is a very active substance.

Acetylcholine has a multifaceted effect on the body. The main function is the mediation of nerve impulses. Nerve fibers and the corresponding neurons that carry out nerve impulses through acetylcholine are called cholinergic. These include motor neurons, innervating skeletal muscles; preganglionic neurons of parasympathetic and sympathetic nerves; postganglionic neurons of all parasympathetic and some sympathetic nerves (uterus, sweat glands) and some neurons of the central nervous system. All cholinergic fibers contain choline acetyltransferase, a specific enzyme that synthesizes acetylcholine. Acetylcholine is in the nerve endings in the vesicles, from which it pours out into the synaptic cleft at the moment of the arrival of the nerve impulse. The release of acetylcholine by nerve endings is of a quantum nature. Apparently, the contents of the bubble constitute the smallest portion of acetylcholine (quantum) that can be isolated. Under normal conditions, each nerve impulse causes the release of several hundred quanta of acetylcholine. Interacting with a specific macromolecule on the postsynaptic membrane - cholinergic receptor, acetylcholine increases the permeability of the membrane for ions: a postsynaptic potential arises that changes the excitability of the effector cell, and in the case of the neuromuscular synapse is the direct cause of the generation of the action potential. The effect of acetylcholine stops under the influence of the enzyme acetylcholinesterase, which hydrolyzes acetylcholine to low-activity choline and acetic acid, and also due to the simple diffusion of acetylcholine from the synaptic cleft. In the molecule of acetylcholine there are two active groups providing interaction with the cholinergic receptor: a charged trimethylammonium group (cationic "head") that reacts with the anionic group in the cholinergic receptor and a highly polarized ester group reacting with the so-called esterophilic region of the cholinergic receptor.

There are two types of action of acetylcholine: muscarinic and nicotine-like. Muscarinic-like action is manifested by effects analogous to those that occur when the parasympathetic nerves of smooth muscles, heart, glands are stimulated, and are removed by atropine; nicotinic is expressed by the excitation of vegetative ganglia and adrenal medulla, as well as skeletal muscles and is removed with large doses of nicotine, hexonium, tubocurarine. Accordingly, cholinergic systems of different organs are designated as m-cholinergic (muscarinic-sensitive) and n-cholinergic (nicotine-sensitive) [8].

Under normal conditions, the muscarinic-like action of acetylcholine predominates. When instillation of acetylcholine in the eye there is a narrowing of the pupil and a spasm of accommodation, the intraocular pressure decreases. When entering the bloodstream, there is a decrease in blood pressure caused by vasodilatation (coronary blood vessels of the person acetylcholine narrows) and to a lesser extent slowing down the cardiac activity, increasing the motor activity of the gastrointestinal tract, contracting the muscles of the bronchi, bile and bladder, uterus, with cholinergic innervation, especially salivary and sweat [9].

The nicotine-like effect of acetylcholine on the autonomic ganglia and adrenal gland is manifested after atropinization and when higher doses are used. It is expressed in the pressor effect. Acetylcholine also stimulates nicotine-sensitive systems of carotid glomeruli and reflexively stimulates respiration.

In experiments on the isolated right atrium of the rat, it was shown that the slowing down of the sinus rhythm and the decrease in the duration of the cardiomyocyte action potential is achieved not only by the addition of exogenous acetylcholine, but also by the inactivation of acetylcholinesterase.

Recently, results have been obtained showing that neither hexamethonium (blocker of neuronal acetylcholine receptors) nor tetrodotoxin (sodium channel blocker) nor botulinum toxin (exocytosis blocker) influence the development of the effects of anticholinesterase agents [10].

Thus, acetylcholine is secreted, despite the "shutdown" of possible intracardiac reflex arcs, the lack of action potential and the actual process of exocytosis of the vesicles. At the same time, the inactivation of the high-affinity choline capture system by hemicholinium-3, as well as in the neuromuscular synapse, led to the elimination of the effects of anticholinesterase agents, which gives serious grounds for talking about the mechanism of non-quantitative isolation of acetylcholine from the terminals of parasympathetic neurons [11].

Effects of serotonin in the human body

Serotonin helps to transfer information from one area of the brain to another. In addition, it affects a variety of psychological and other processes in the body. Of the 80-90 billion brain cells, serotonin has a direct or indirect effect on most of them. It affects the work of cells that are responsible for mood, sexual desire and function, appetite, sleep, memory and learning ability, temperature and certain aspects of social behavior [12]. It has been proved that when the serotonin decreases, the sensitivity of the body's pain system increases, that is, even the weakest irritation responds with severe pain [13].

Serotonin can also affect the functioning of the cardiovascular, endocrine systems and the work of muscles.

Studies have shown that serotonin can play a role in the formation of breast milk, and its deficiency can become the root cause of sudden death of an infant during sleep [14].

- Serotonin normalizes blood clotting; in patients with a tendency to bleeding, the amount of serotonin is reduced; The introduction of serotonin helps reduce bleeding
- Stimulates the smooth muscles of blood vessels, respiratory tract, intestines; at the same time it intensifies the intestinal peristalsis, reduces the daily amount of urine, narrows the bronchioles (branching of the bronchi). Lack of serotonin can cause intestinal obstruction [15,16].
- Excess of the serotonin hormone in the regulatory structures of the brain acts depressingly on the functions of the reproductive system [17].
- Serotonin is involved in the pathogenesis of diseases of the gastrointestinal tract, in particular, carcinoid syndrome and irritable bowel syndrome [18]. Determination of serotonin concentration in the blood in clinical practice is used primarily in the diagnosis of carcinoid tumors of the abdominal cavity (the test is positive in 45% of cases of rectal carcinoma). The study of blood serotonin is expedient to use in combination with the determination of excretion of the metabolite of serotonin with urine [19].

Thus, serotonin, found in the sympathetic and parasympathetic nuclei of the lumbosacral spinal cord and the delimited region of the lateral-ventral horn, attests to the participation of bioamine in the reflex, cardiovascular, endocrine system and muscle activity.

CONCLUSION

The regulation of interactions in the central nervous system through the mediators produced by cells is an important component of intercellular communication. At present, there are many methods of active release of humoral factors from cells, which exert paracrine and autocrine influence in multicellular ensembles (for example, exocytosis, secretion in membrane microparticles, release through connexin and pannexin channels). Therefore, further study of the mechanisms of intracellular interaction in the central nervous system with the help of biologically active molecules is of great importance for fundamental science and medical practice.

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