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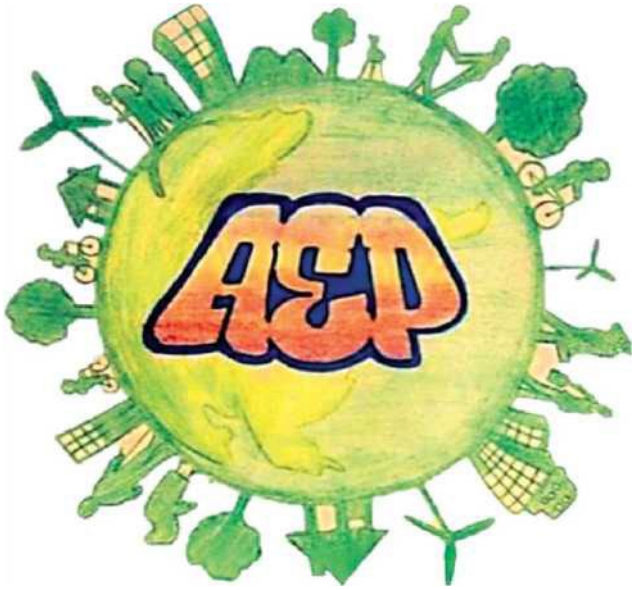

ACTUAL ENVIRONMENTAL PROBLEMS

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CHANGES IN ION CURRENTS OF NEURONS UNDER ACTION OF LOCAL ANESTHETICS

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Modern local anesthetics suppress ion currents in excitable membranes by interacting with channels from the cytoplasmic side of the cell membrane. The mechanism of the effect of local anesthetics on the permeability of the functional membrane for sodium and potassium ions has been investigated. The blocking effect of local anesthetics on the permeability of the membrane for ions is shown. A comparative analysis of the intensity and duration of the action of the anesthetic substances studied on the ion flows of Na^+ and K^+ was carried out.

Keywords: sodium channels, local anaesthetics, sodium channel blockers, pain relief.

Chronic and other types of pain are quite common phenomena today, and the transmission of nerve impulses is caused, simply, by the undulating movement of sodium ions into and out of the cell. Despite the obvious positive aspects of the sensation of pain signals, prolonged or severe painful sensations can interfere with everyday life and lead to unpleasant consequences. Therefore, there is a need to block ion channels. One of the ways to achieve this goal is the use of local anesthetics that cause reversible local loss of sensitivity, primarily pain, without loss of consciousness.

The aim of this study was to investigate changes in the ion currents of neurons under the action of local anesthetics.

Nerve fibre membranes maintain a transmembrane potential of -90 to -60mV, at which sodium channels, which include activation and inactivation gates, are closed, when a nerve impulse is transmitted, excitation occurs and the Na^+ channel opens, Na^+ ions enter the cell, depolarizing the membrane to an equilibrium potential Na^+, equal to +40mV. Inactivation occurs, followed by closure of Na^+ channels. To restore the resting potential, K^+ channels open, releasing K^+ ions outward, repolarizing the membrane to the K^+ equilibrium potential (-95mV) [1]. Restoration of the amount of Na^+ and K^+ ions inside the cell to the initial values occurs due to the ion pump, which provides transport of Na^+ ions outside and K^+ ions inside due to the energy of ATP hydrolysis.

The mechanism of action of local anesthetics is that, penetrating into the cell, they block ion channels, not allowing ions to pass through the gate, preventing the transmission of excitation. The following assumptions are taken into account for this phenomenon: there is only one binding site in the channel, there is no competition of other ions with the blocker for the binding site [2]. Hydrophobic anesthetics penetrate nerve fibres and block ion channels on the inner side of the membrane, while hydrophilic local anesthetics cause blockage by penetrating through the gate of sodium channels [3]. Other studies have shown that anesthetics inhibited not only inward Na^+ currents but also outward K^+, but to achieve these required concentrations 10-100 times greater than those required to inhibit Na^+ currents [1]. A well-known example of local anesthetics is lidocaine. By reducing the permeability of the nerve cell membrane to Na^+ ions, it reduces the rate of depolarization and increases the excitation threshold, resulting in reversible local numbness.

The structure of most anesthetics consists of three main fragments: the aromatic structure, the intermediate chain and the amino group. The middle part is usually either amide or ester, which divides local anesthetics into two types: esters and amides. Representatives of esters are cocaine, procaine, benzocaine, and amides are lidocaine, mepivacaine, and bimecaine. They differ in potency and in some pharmacodynamic parameters that affect the potency and duration of action [4]. Compound ester bonds are destroyed faster in the body due to esterases, enzymes that catalyze the hydrolysis of esters, which are common in tissues; therefore, anesthetics of this group work for a shorter period of time than the amide group [3]. Articaine, classed as an amide type, is quite unique in this respect as it also contains an ester group. Hydrolysis of this side chain renders the molecule inactive, which promotes an output identical to that of ester-type anesthetics [4].

The effect of local anesthetics is temporary due to their rather rapid destruction and connection with the blood flow. However, to increase the duration of action and enhance the anesthetic effect, they are often combined with vasoconstrictors (for example, epinephrine) [3]. This affects the possibility of side effects that may occur with good absorption of the drug. For example, epinephrine affects β_2-adrenergic receptors, increasing the risk of toxic effects of the anesthetic deposited in muscle tissues. While choosing local anesthetics and complementary drugs, doctors rely on the duration and degree of exposure to the body during the future operation in order to ensure the most effective and less traumatic effect.

Despite the positive effect of local anesthetics in terms of blocking pain, it is not recommended to abuse these drugs due to the insensitivity of ion channels to drugs at rest, as well as an increase in the threshold of excitation and slowing down the occurrence of action potentials with a prolonged increase in the concentration of local anesthetics.

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