ION CHANNELS PARALLY MEDICINE IN CARDIOMYOSITES (HEART CONDUCTIVE SYSTEM AND MYOCARDAL CONSTRUCTION)

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Annotation: *The article provides detailed information on the means of paralysis of ion channels in cardiomyocytes.*

Keywords: *cardiomyocytes, diastolic depolarization, arrhythmia*

The pharmacotherapeutic effect of such antiarrhythmic drugs is based on the control of their automatism, the effect on conduction and the prolongation of the effective refractory period. Changes in such parameters are associated with the effect of reparations on ion channels, in addition, they can be compensated by the effect on adrenergic and cholinergic receptors in the heart.

This is mainly due to the expansion of diastolic depolarization and increased excitability. All this prevents the activation of the natural rhythm of the heart and prevents the emergence of ectopic foci of excitation.

Many antiarrhythmic drugs, such as quinidine, novocaineamide, reduce permeability. This leads to a decrease in the rate of rapid depolarization (phase 0) and a slowing of the depolarization-repolarization process in general. The ECG shows a decrease in permeability with P-Q interval elongation and dilation of the ventricular complex. The positive effect of conduction compression is manifested in cardiac tachyarrhythmias.

The ability to lose conduction has a positive effect on the developing arrhythmia by the mechanism of recurrence. In this case, the one-way block changes to two-way.

Also an important parameter is the effective refractory period. Under the influence of many antiarrhythmic drugs (quinidine, quinidine-like drugs) it increases.

This effect is usually associated with an increase in the duration of the action potential. It can be seen that the duration of the effective refractory period limits the frequency of the propagating stimulus. Many drugs reduce the excitability. When using antiarrhythmic drugs, many of them reduce myocardial contractility. Decreased myocardial contractility has a negative effect on heart function, especially in heart failure.

When evaluating the carditropic effects of antiarrhythmic drugs, it is not possible to take into account the changes in the extracardiac effect through the sciatic nerve and sympathetic nerves. It is known that a decrease in the transmission of impulses from stray nerves to the heart (due to the effect of m-cholinoblockers) increases sinus rhythm, improves conduction in the cardiac and ventricular nodes and shortens its refractory period. Especially when it comes to quinidine and a number of quinidine-like substances, their vagolytic effect weakens the cardiac effect of these substances, which provides antiarrhythmic effect. The effect of antiarrhythmic agents from the groups that block the ion channels from adrenergic innervation is less pronounced, and in general the antiarrhythmic effect is negligible or minimal.

Thus, the effect of antiarrhythmic drugs is associated not only with the direct effect of cardiomyocytes on ion channels, but also with changes in their efferent innervation.

The most typical representative of the antiarrhythmic drugs in subgroup A is quinidine. Quinidine sulfate is used in medical practice. By blocking sodium channels, quinidine reduces sodium entry, blocking the distribution of action potentials.

Quinidine affects all parts of the heart. Loss of automatism, increased repolarization duration and, accordingly, increased effective refractory period and decreased permeability have the anti-arrhythmic effect of quinine, which is used in arrhythmias associated with automatism and conduction disturbances.

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Sodium channel blockers (membrane enhancers; group I). The most typical representative of antiarrhythmic drugs in subgroup I A is quinidine. Quinidine sulfate is used in medical practice. By blocking sodium channels, quinidine reduces sodium entry, blocking the distribution of action potentials. Quinidine affects all parts of the heart. Loss of automatism, increased repolarization duration and, accordingly, increased effective refractory period and decreased permeability have the anti-arrhythmic effect of quinine, which is used in arrhythmias associated with automatism and conduction disturbances. ECG and P-R, QRS, Q-T. a small expansion of Quinidine reduces the transmission of excitability from the sciatic nerve to the heart (due to the m-cholinolytic effect), and slightly reduces the cardiotropic and sympathetic (adrenergic) effects. in peripheral blood vessels due to the

paralyzing effect of α-adrenoceptors (slightly reduces the overall peripheral resistance). Quinidine significantly reduces myocardial contractility.

Quinidine is usually recommended for oral administration. It is completely absorbed from the gastrointestinal tract. The drug is broken down in the liver, the duration of action depends largely on liver function (usually 6-8 s). Unchanged quinidine (~ 20%) and its modified products are mainly excreted by the kidneys. There are quinidine drugs that have a prolonged effect. The use of quinidine can cause various side effects and toxicity: tinnitus, headache, visual disturbances. Occasionally there is diarrhea, nausea, and vomiting. Idiosyncrasy is noted. Severe complications include thrombostitopenic purpura. There may be ventricular and interventricular block, as well as toxic tachyarrhythmias.

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