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MECHANISM AND PATHOLOGY OF HEART MUSCLE CONTRACTION

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ABSTRACT

In the article the cellular structure of your heart muscle , contraction mechanism , its working principle and reflections are made on the negative consequences that occur when the cardiac mechanism of the muscles is disturbed.

Keywords: muscle, sarcomere, actin, myosin, excitability, myocardium, troponin, tropomyosin, myocardial infarction, thrombus, plaque.

INTRODUCTION

Muscles, in turn, have skeletal, smooth and cardiac muscles, and we will now divide them according to the structure of the heart muscle and its mechanism of action. Cardiac muscle is composed of cylindrical cells parallel to each other to rise in the transverse direction. The heart of the muscle is found. A distinctive feature is that in the area of the discs with the heart muscle, the interconnected cells of the heart muscle contract rhythmically, and the person does not obey according to his will.

Cardiac muscle has other muscle properties such as excitability, conduction and contractility. It also has a heart automation feature. Excitability - occurs as a result of an impulse to tissues, then biochemical and biophysical changes occur (arrival of the action potential in the body). Contraction of the heart accumulates in the muscles and the response to the action potential is manifested. The layer of the heart muscle is called the myocardium, the contraction of the heart muscle is carried out by the sarcomeres located in its cells. If actin and myosin proteins are present in sarcomeres, their sliding occurs, resulting in contraction of the heart muscle, if actin tropomyosin and troponin proteins do not contract, your heart is at rest. When it comes to the binding of calcium ion to troponin and the function of tropomyosin, the potential for action on the body changes. And the myosin head combines with actin and it is pulled forward and moves. From this comes myosin bonds for ATF, and the energy consumption is that your heart is in constant motion the amount of energy spent by the heart muscle is more in the mitochondria cells.

Heart activity from violation come coming out diseases about

As a result of violation of the mechanism of contraction of the heart muscles, many serious diseases appear. The most common disease behind them is myocardial infarction.

arteriosclerosis begins with the formation of plaques. When a person is engaged in heavy physical work or when he is under strong excitement and stress, the standing endothelial cell is eaten. From this place, a thrombus is formed and the blood clot begins to block the passage of blood. co-located and that's it. brings it to sink in the area of compression. A sufficient amount of oxygen reaches the cardiomyocytes, it does not go to the central nervous system, the impulse affects and pain is created, only the heart pain is not in the field, but in the left shoulder, arm, neck area, etc. In some cases, the right hand and the body. will come . The lack of oxygen gradually decreases, until the cardiomyocytes are reduced and stop. Clots in a blood vein restrict the movement of completely closed blood _ Your heart activates other areas, and more starts in a short time _ If the necessary measures are not taken in time, it can occur (within 20 minutes) serious complications may occur.

Myocardial oxygen requirements. Normal myocardium consumes about 8-10 ml of oxygen per minute for every 100 g of mass. Of this amount, about 2 ml of O2 per minute is spent exclusively on maintaining the viability of myocardial cells, the rest of the oxygen is consumed in connection with muscle contraction. Oxygen consumption by the myocardium increases with increasing external work of the heart. However, the increase in O2 consumption occurs with different intensity, depending on whether the increase in work is due to an increase in the volume of pumped blood or an increase in the pressure developed by the heart. Direct experiments have shown, for example, that if the external work of the heart doubles as a result of a doubling of pressure, myocardial oxygen consumption more than doubles (increases by 108%). If doubling external work is due to doubling the volume of ejected blood, oxygen consumption increases by only 8%. Thus, the first most important determinant of oxygen consumption by the heart is pressure in the ventricular cavity. The second important determinant is heart rate. The same external work (moving the same volume at the same pressure) per minute can be performed by the heart contracting at different frequencies. However, if this work is performed at a lower frequency of strokes, oxygen consumption will be lower. The third determinant is the value of the end-diastolic volume. If the ventricular cavity is dilated, the end-diastolic volume is increased compared to normal, the release of the previous volume of blood against the previous pressure will require more oxygen consumption. The reason for this is that, in accordance with Laplace's law, an enlarged heart must develop greater contractile tension than normal in order to achieve equal pressure in the ventricular cavity. Laplace's law establishes the relationship between the stress (T) inside the wall of the sphere and the pressure in the cavity of the sphere (P). This dependence is determined by the radius of the sphere (r) and the thickness of the sphere wall It is clear that when the ventricular cavity is expanded (the radius is increased), the previous value of pressure in the ventricle, allowing systolic expulsion of blood, will be achieved only due to the development of greater wall tension.. Due to the primary role of tension in myocardial oxygen consumption, the tension-time index (TTI) is used clinically to assess cardiac oxygen consumption. In this case, the measure of tension is most often the average pressure at the aortic mouth during the blood expulsion phase, which is multiplied by the time that determines the duration of blood expulsion (the time the aortic valves remain open). Finally, another important determinant of oxygen consumption by the heart is the rate of muscle shortening. It strongly depends on the tone of the sympathetic nerves of the heart and on the content of catecholamines in the blood.

Thus, the amount of oxygen consumption by the heart is determined by three main factors: The amount of tension developed by the heart during the isovolumic phase and during the ejection phase of the blood. It depends on the pressure in the aorta and the radius of the cavity (curvature) of the ventricles. 18 The speed of contraction , which depends on the tone of the sympathetic nerves and the content of catecholamines circulating in the blood. Heart rate. There are many reasons to believe that a chronic increase in oxygen consumption by the myocardium is the most important stimulus for its hypertrophy. This is why hypertrophy is detected when blood pressure in the aorta increases (chronic hypertension, aortic stenosis). On the other hand, an increase in stroke volume causes only a slight increase in the stress-time ratio and does not lead to significant hypertrophy, especially when the increase in stroke volume is not associated with an increase in pressure. With fully developed hypertrophy, the energy expenditure of the myocardium per unit mass decreases to normal values and some equilibrium is established (stable stage of hypertrophy). The total coronary blood flow per unit mass of hypertrophied myocardium at rest is close to normal. The contractility of the hypertrophied myocardium is reduced. Let us now consider, using the above concepts, the pathophysiological mechanisms of some specific diseases.

SUMMARY

This article is based on the information provided by the mechanism of reduction of actin and myosin in the muscles of cardiac sarcomeres and the negative consequences that arise from the disruption of this reduction mechanism, and from this information there is information about the understanding of the work of the heart and the mechanism of cardiac contraction.

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