

MORPHOLOGICAL CHANGES IN NEUROGENIC LESIONS OF THE LEFT VENTRICULAR MYOCARDIUM IN EXPERIMENT

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Abstract. One of the solutions to the problem of detecting neurogenic lesions of the heart myocardium is modeling neurogenic heart lesions in animal experiments. In this study, neurogenic lesions in the heart were modeled in rats using chemical desympathization with guanethidine. Chemical desympathization has varying effects on cells located in the isolated layers of the left ventricular myocardium of rat hearts. The obtained data provide a new perspective on the processes of changes in organ functioning and indicate one of the possible directions for searching for patterns of mosaic functioning of the myocardium.

Keywords: experiment, rat, heart, left ventricle, myocardium, desympathization, guanethidine, histological analysis.

МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ ПРИ НЕЙРОГЕННЫХ ПОРАЖЕНИЯХ МИОКАРДА ЛЕВОГО ЖЕЛУДОЧКА В ЭКСПЕРИМЕНТЕ

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Аннотация. Одним из решений проблемы выявления нейрогенных поражений миокарда сердца является моделирование нейрогенных поражений сердца в эксперименте на животных. В настоящей работе нейрогенные поражения в сердце моделировали на крысах химической десимпатизацией гуанетидином. Химическая десимпатизация по-разному влияет на клетки, лежащие в выделенных слоях миокарда левого желудочка

сердца крыс. Полученные данные позволяют по-новому взглянуть на процессы изменения функционирования органа и указывают на одно из возможных направлений поиска закономерностей мозаичного функционирования миокарда.

Ключевые слова: эксперимент, крыса, сердце, левый желудочек, миокард, десимпатизация, гуанетидин, гистологический анализ.

ТАЖРИБАДА ЧАП ҚОРИНЧА МИОКАРДИНИНГ НЕЙРОГЕН ШИКАСТЛАНИШЛАРИДА МОРФОЛОГИК ЎЗГАРИШЛАР

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Аннотация. Юрак миокардининг нейроген шикастланишларини аниқлаш муаммосининг ечимларидан бири ҳайвонларда тажриба ўтказиб, юракнинг нейроген шикастланишларини моделлаштиришдир. Ушбу ишда юракдаги нейроген шикастланишлар каламушларда гуанетидин билан кимёвий десимпатизация орқали моделлаштирилди. Кимёвий десимпатизация каламуш юраги чап қоринчаси миокардининг ажратилган қатламларида жойлашган ҳужайраларга турлича таъсир кўрсатади. Олинган маълумотлар орган фаолиятининг ўзгариш жараёнларига янгича назар ташлаш имконини беради ҳамда миокарднинг мозайка тарзидаги фаолият қонуниятларини излашнинг мумкин бўлган йўналишларидан бирини кўрсатади.

Калит сўзлар: тажриба, каламуш, юрак, чап қоринча, миокард, десимпатизация, гуанетидин, гистологик таҳлил.

Introduction. Cardiovascular pathology is a crucial component in the structure of morbidity in most developed countries [1,7]. Diseases of the circulatory system account for approximately half of all causes of disability and mortality [2]. Activation of neurohormonal systems plays an important role in the evolution of the cardiovascular system, provoking autonomic imbalance. Currently, there are

numerous methods for assessing parameters of autonomic regulation of the circulatory system; however, only an adequate combination of registration methods and stress tests enables a comprehensive evaluation of neurogenic regulation of blood circulation [3,5]. This allows for determining the nature and possible causes of autonomic dysfunction, even in cases of combined pathology. Addressing the issues of etiology and pathogenesis of neurogenic myocardial lesions requires the development of differential morphological criteria for assessing the organ's condition using quantitative morphological methods [4,6]. One solution to the problem of detecting specific heart lesions is modeling neurogenic heart lesions in animal experiments, which allows tracing the multilevel nature of organ changes.

Study Objective. To investigate changes in neurogenic lesions of the left ventricular myocardium in an experiment.

Materials and methods. In the present study, neurogenic lesions in the heart were modeled in rats using chemical desympathization with guanethidine. Experiments were conducted on 35 white rats weighing 150–210 g. Morphological and histochemical methods were used to identify morphofunctional shifts. Accordingly, measurements were taken of the diameter of cardiomyocytes in the subendocardial, subepicardial, and central layers of the left ventricular myocardium in rats under normal conditions and after chemical desympathization. Measurements were performed on histological preparations using a drawing apparatus with a final magnification of 1800×. The obtained results were statistically processed.

Results. The data obtained show that cardiomyocytes in different layers of the left ventricular myocardium significantly differ in diameter. In control animals, the maximum diameter is observed in cardiomyocytes located in the central layers of the myocardium ($22.5 \pm 0.12 \mu\text{m}$), with slightly smaller sizes (significantly different from each other) in cells located in the subendocardial ($17.50 \pm 0.09 \mu\text{m}$) and subepicardial ($17.10 \pm 0.18 \mu\text{m}$) layers. Chemical desympathization affects cells in the isolated layers of the left ventricular myocardium of rat hearts differently. Cardiomyocytes in the central layers of the myocardium decrease in

size (diameter $17.57 \pm 0.17 \mu\text{m}$), while in the subendocardial and subepicardial layers, there is a significant increase in cardiomyocyte diameter ($23.10 \pm 0.19 \mu\text{m}$ and $21.00 \pm 0.14 \mu\text{m}$, respectively).

Conclusion. The obtained data provide a new perspective on the processes of changes in organ functioning and indicate one of the possible directions for searching for patterns of mosaic functioning of the myocardium. The literature has repeatedly noted that establishing a new level of organ functioning is determined by multidirectional processes; however, there are no indications in the available literature regarding the preferential association of these processes with specific layers of the myocardium.

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