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ULTRASTRUCTURE OF THE SINUS AND ATRIOVENTRICULAR NODES IN EXPERIMENTAL MYOCARDIAL INFARCTION

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KEY WORDS: myocardial infarction; conducting system; ultrastructure

It has now been established that death from myocardial infarction is often based on disturbances of the cardiac rhythm. Lesions of the conducting system of the heart are a significant factor. However, recognition of this fact alone does not explain the morphological changes taking place in the nodes of the conducting system in myocardial infarction.

The aim of this investigation was an electron-microscopic study of the sinus (SA) and atrioventricular (AV) nodes of the rat heart in experimental myocardial infarction, using colloidal lanthanum as electron-microscopic tracer, and using morphometric methods to evaluate the ultrastructural changes.

EXPERIMENTAL METHOD

Myocardial infarction was induced in 20 noninbred albino rats weighing 180-200 g by ligation of the left coronary artery. The operation was performed under ether anesthesia. Ten rats served as the control. The experimental rats were killed under ether anesthesia 1 day after the beginning of the experiment. The heart was stopped by exposure to cold. The SA node material was taken with the lower part of the superior vena cava and adjacent right atrial myocardium, whereas material of the AV node was taken with the upper part of the ventricular and lower part of the atrial septum [4]. Pieces of tissue were fixed for 2 h in 2.5% glutaraldehyde at 4°C and washed with phosphate buffer (pH 7.4). The material was postfixed in 1% OsO_4 for 2 h. Oriented embedding [8] was used when the fragments were embedded in Araldite.

Ultrathin sections were cut on the Ultrotome-5 (LKB, Sweden), stained on the Ultro Stainer (LKB, Sweden), and examined under the UÉMV-100K electron microscope. Permeability of the sarcolemma was estimated with the aid of colloidal lanthanum, prepared from lanthanum nitrate (from Serva, West Germany) by the method in [9] in the modification [6]. The electron micrographs were analyzed quantitatively by the method in [2] under a magnification of 10,000. The mean number of mitochondria per electron micrograph, the mean number of cristae per mitochondrion, the mean area of 1 mitochrondrion, the mean total area of the mitochondria per electron micrograph, and the mean total number of cristae per electron micrograph were determined. The energy efficiency of the mitochondria (EEM) was calculated

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TABLE 1. Quantitative Analysis of Electron Micrographs (EM) of SA and AV Nodes of Rat Heart 1 Day after Ligation of Left Coronary Artery (M ± m)

Parameter	SA node			AV node		
	control	experiment	P	control	experiment	P
Number of MCh per EM	9,5±1,6	9,83±1,4	>0,05	10,62±1,6	8±1,32	>0,05
Number of cristae per MCh	7±0,55	4,25±0,27	<0,001	5,91±0,26	3,62±0,23	<0,001
Number of cristae per EM	66,83±6,5	41,77±7,4	<0,01	62,87±5,5	29±6,73	<0,01
Mean area of 1 MCh, μ^2	0,42±0,01	0,36±0,06	>0,05	0,27±0,03	0,51±0,04	<0,01
Mean area of MCh per EM, μ^2	4,01±0,21	3,52±0,1	>0,05	2,91±0,3	4,1±0,16	>0,05
EEM, %	100	54,9		100	64,9	

Legend. MCh) mitochondrion (mitochondria).

in percent as the ratio of the product of the mean total number of cristae and the total area of the mitochrondria per electron micrograph in the presence of pathology to the same parameters for the normal heart. All numerical data were subjected to statistical analysis on the Iskra 001-45 computer and the significance of differences was estimated by Student's test.

EXPERIMENTAL RESULTS

The investigation showed that 1 day after ligation of the left coronary artery, the infarct, located in the anterior wall of the left ventricle, and partly involving the ventricular septum, became transmural. In no case did the infarct spread to the region of the AV node. However, ultrastructural changes to different degrees were found in the conducting cells of the AV node. In some cells these changes were characterized by swelling of the mitochondria with destruction of their cristae, and also by the appearance of electron-dense residues in the matrix of the mitochondria. The mean area of 1 mitochondrion was increased to 0.51 ± 0.04 μ^2 (0.27 ± 0.03 in the control, $P < 0.01$, Table 1). The number of cristae per mitochondrion decreased to 3.62 ± 0.23 (5.91 ± 0.26 in the control; $P < 0.001$). The significant decrease in the number of cristae in the mitochrondria led to a decrease in EEM, which fell to 64.9% after 1 day. A varied degree of aggregation of nuclear chromatin was found in the nucleus, with dilatation of the tubules of the sarcoplasmic reticulum. Colloidal lanthanum was present in the intercellular space. Colloidal lanthanum was found in irreversibly altered cells of the AV node in the form of electron-dense granules on the outer membranes of the mitochrondria in the sarcoplasm.

A varied degree of ultrastructural changes was found in the SA cells in the present experiments. The number of cristae per mitochondrion decreased to 4.25 ± 0.27 (7.0 ± 0.55 in the control; $P < 0.001$). Swelling of the mitochondria was less marked than in the AV node. The mean area of one mitochondrion did not differ statistically significantly from that in the control. However, because of the decrease in the number of cristae, EEM fell to 54.9%. In cells located at the periphery of the SA node colloidal lanthanum was often found on the membranes of the mitochondria and in their matrix, in the form of electron-dense intracristal inclusions. Colloidal lanthanum in other cells did not pass through the sarcolemma, but the number of glycogen granules in these cells was reduced.

The appearance of colloidal lanthanum particles in the lumen of the intracellular organelles is a morphological sign of calcium overloading of the cells [5]. The reasons why different cells undergo different changes when exposed to caclium overloading are not yet known [7].

The study of the conducting cells of the SA and AV nodes, which are composed of cells of different types [3], revealed heterogeneity of their changes in experimental myocardial infarction; this may be due, on the one hand, to population differences and, on the other hand, to differences in the mechanism of their injury.

In accordance with Sharov's classification ultrastructural changes in cells of the AV node after ligation of the coronary artery must be classed as lesions of primary ischemic type. Meanwhile ultrastructural changes in cells of the SA node, which have a better blood supply, could be due to exposure to an excess of catecholamines, which arises in acute myocardial infarction [1], and they must be regarded as belonging to the primary calcium type of cell degeneration.

Changes in ultrastructure of cells of the SA and AV nodes as well as differences in the degree of disturbance of permeability of the sarcolemma and intracellular membranes for colloidal lanthanum enable the point of application of the ischemic factor to be identified in these formations. Heterogeneity of the ultrastructural changes, the reduction in the number of cristae, and the decrease in energy efficiency of the mitochondria in nodes of the conducting system of the heart lead to a disturbance of their function, on the basis of which rhythm disturbances may arise under the conditions of acute myocardial infarction.

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BRONCHOALVEOLAR CELL COUNT DURING COMPENSATORY HYPERTROPHY OF THE LUNG

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KEY WORDS: bronchoalveolar lavage; bronchoalveolar cell count; compensatory hypertrophy of the lungs

Identification of the structural basis for disturbed functions of the damaged lung has not yet been finally achieved. We know that after resection of various kinds the residual lung volume increases rapidly [5, 6, 14]. After leftsided pneumonectomy in rats the relative volume of the right lung only 5 days after the operation amounts to more than 80% of the combined volume of both lungs of control animals [6]. The question arises whether there is an increase in the number of cells settling in the respiratory part of the lung takes place parallel with compensatory growth of the lung. The morphological and functional features of cell populations existing inside the lung are nowadays studied by cytological analysis of material obtained by bronchoalveolar lavage (BAL) [2, 4].

The aim of this investigation was to discover the characteristic features of the bronchoalveolar cell count (BACC) in the normal and postoperative lung and to determine the degree of increase in the number of cells colonizing the internal medium of the lung, in the course of its compensatory hypertrophy.

By bronchopulmonary cell count is meant the ratio between the number of cells obtained by BAL (cells of the monocytic-macrophagal series, lymphocytes, neutrophils, eosinophils, and basophils). Cells of the bronchial epithelium and erythrocytes are not included. The alternative term "endopulmonary cytogram" was first introduced by Avtsyn et al. in 1982 [2].

EXPERIMENTAL METHOD

Experiments were carried out on noninbred male albino rats weighing 215-345 g, kept under animal house conditions. Only animals with no outward signs of disease of any kind were used in the experiments. Under deep pentobarbital anesthesia the left lung (about 37%

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