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ELECTRON-AUTORADIOGRAPHIC STUDY OF VIABILITY OF HUMAN HEART CELLS AFTER DEATH

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KEY WORDS: heart; autoradiography; RNA metabolism

The study of functional and morphological changes in the heart cells after death gives a deeper insight into their metabolism and function during life, and also is of direct practical significance for the development of resuscitation and transplant surgery. Determination of the duration of viability of the heart cells after cardiac arrest gives different values depending on the criterion used. A few hours after death the heart beat can be restored [2]. Morphological changes, in the form of severe swelling of the mitochondria, the formation of homogeneous electron-dense residues in their matrix, and fragmentation and vesiculation of elements of the sarcoplasmic reticulum have been observed in rat cardiomyocytes 15-60 min after death [5]. A normal catecholamine content has been found in the hearts of the same animals in the course of 8 h after death [1]. The criteria mentioned above and some others used for the same purpose do not enable the ability of the heart, after arrest, to maintain the normal life of the individual to be reliably confirmed or refuted.

To influence processes taking place in the heart after arrest the first essential is to have information about the basic changes in metabolism in the different cells of the organ. In this paper we give data on a fundamental biological process, namely RNA synthesis, in heart cells at various times after cardiac arrest.

EXPERIMENTAL METHOD

Pieces of atria removed during operations for congenital heart defects in two patients aged 7 and 29 years, and also at autopsy on cadavers of four patients aged 6, 7, 37, and 50 years, the immediate cause of death of whom was combined cardiac and respiratory failure, served as the test material. The time from death to removal of the fragments was 3, 4, 8, and 13 h. During this time the cadavers were kept at room temperature. The excised atrial fragments were cut into pieces measuring 1 mm^3 and were incubated at 37°C for 80 min in medium 199 containing ^{3}H-uridine 100 μCi/ml, with specific activity of 26 Ci/mmole. After incubation the pieces were washed to remove unincorporated ^{3}H-uridine with cold medium 199, fixed in a 2.5% solution of glutaraldehyde and 1% solution of osmium tetroxide, dehydrated, and embedded in a mixture of Epon and Araldite. From each block autoradiographs of semithin sections were prepared ("M" emulsion, exposure 3 days, developer D-19). On the basis of inspection of the autoradiographs of the semithin sections, pyramids

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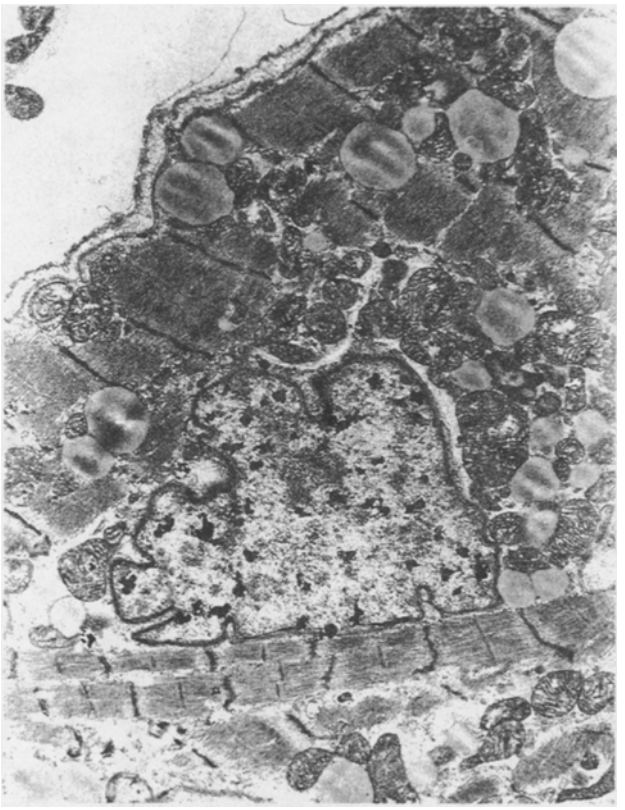

Fig. 1. RNA synthesis (multiple grains of silver above the nucleus) in a cardiomyocyte incubated with ^{3}H-uridine, 4 h after death. Accumulation of heterochromatin throughout territory of nucleus. 12,000×.

were cut out for ultramicrotomy. Electron-microscopic autoradiographs were prepared by the method described by Sarkisov and co-workers [3, 4], using "M" emulsion, an exposure of 1 month, and D-19 developer.

EXPERIMENTAL RESULTS

Autoradiographs of the myocardium removed during operations served as the control in this investigation, and demonstrated with some degree of approximation the intravital state of the heart. All cells of blood vessels in these preparations, irrespective of their position in the medium, were intensively labeled. Labeling of the myocytes differed, depending mainly on their position in the fragment. Cells located near the surface were densely labeled. Intensively labeled, weakly labeled, and unlabeled cells were found in the center of the fragments. A blood vessel could often be seen next to a labeled myocyte.

In our view this distribution of the label can be explained on the grounds that uridine penetrates slowly along the intercellular spaces, and cells of blood vessels, which can incorporate uridine from the nutrient medium penetrating into their lumen, are therefore labeled comparatively uniformly. Irregularity of labeling of the myocytes can most probably be explained, not by the fact that not all these cells can synthesize RNA, but that uridine penetrates only into those cells that are close to the nutrient medium bathing the fragment or to the lumen of the vessels filled with medium.

In autoradiographs of the myocardium obtained 3 and 4 h after death, the distribution of the labeled cells was about the same as in the control. However, it could be observed that unlabeled myocytes differed from labeled in a change in the electron density of the chromatin and its uneven distribution throughout the nucleus (Figs. 1 and 2). Consequently, absence of the label could be due not only to slow diffusion of uridine, but also to destruction of some myocytes.

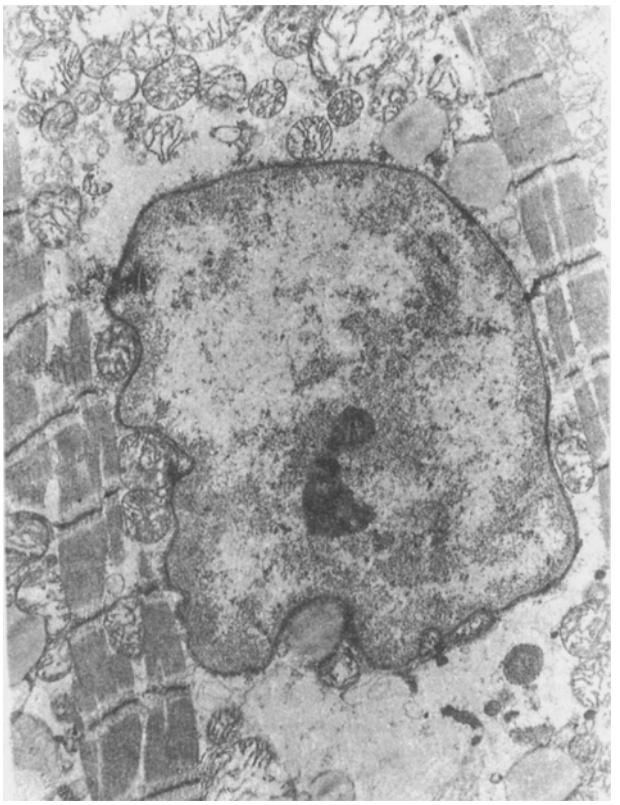

Fig. 2. Atrial myocyte incubated with ^{3}H-uridine, 4 h after death. Labeling absent. Heterochromatin converted into homogeneous floccular masses, occupying only part of territory of nucleus. 12,000×.

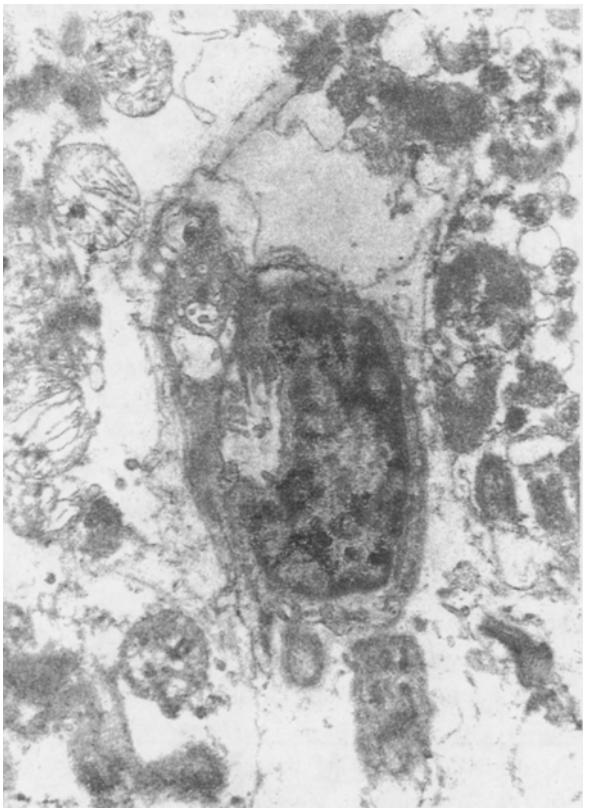

Fig. 3. RNA synthesis in endothelial cell of atrium 13 h after death. Destruction of muscle cells. 13,000×.

In the later stages after death (8 and 13 h) significant morphological changes were observed in the myocytes, indicating autolysis: destruction of cristae of the mitochondria, conversion of the myofibrils into a structureless mass, and reduction of the electron density of the chromatin. Cells of small blood vessels remained capable of synthesizing RNA: they were labeled irrespective of their position in the incubated fragment, and the labeling density was not significantly lower than in the control (Fig. 3). Myocytes 8 and 13 h after death as a rule did not preserve their ability to synthesize RNA. Most myocytes located in the center and at the periphery of the fragment did not contain the label. However, single muscle cells located near the vessels had the label, and its density did not differ from that in the control myocytes. Morphologically these cells were changed much less by comparison with the control than the main mass of myocytes in these preparations. Thus during the first 4 h after death, in all or at least in most cells of the atrial myocardium, when the appropriate conditions are created, the function of the genome (RNA synthesis) is renewed. It can be tentatively suggested that all other functions derived from this basic function can also be restored. The cells forming the wall of small blood vessels are distinguished by their great resistance to postmortem decomposition and remain viable for more than 13 h after death.

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EFFECT OF DESYMPATHIZATION ON DEVELOPMENT OF ADAPTIVE AND COMPENSATORY REACTIONS OF THE THYROID C-CELL APPARATUS AND ADRENAL CHROMAFFIN CELLS IN YOUNG RATS

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KEY WORDS: desympathization; thyroid gland; adrenals; compensation

An important place in the combination of complex morphological and physiological reactions of the thyroid (TG) and adrenal glands (AG) is played by the functional state of the adrenergic nervous system. Sympathetic influences on peripheral glands can be effected both indirectly through the pituitary gland and directly on an innervated organ [1, 2, 7]. In this connection a combined study of the C-cells of TG and the chromaffin cells of AG during inhibition of sympathetic influences is interesting. We know that the above-mentioned cell populations are formed from neuroendocrine-programed cells and are able to synthesize neuroamines as well as producing oligopeptide hormones [2, 8, 10, 11, 15]. Their secretory activity is not significantly influenced by the adenohypophysis, but is regulated entirely by nervous impulses and depends on

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