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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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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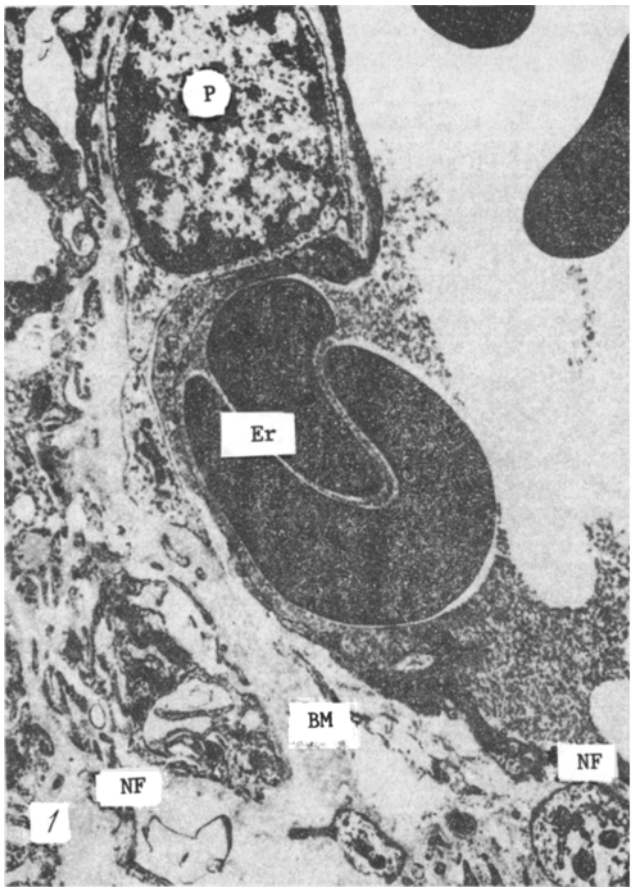

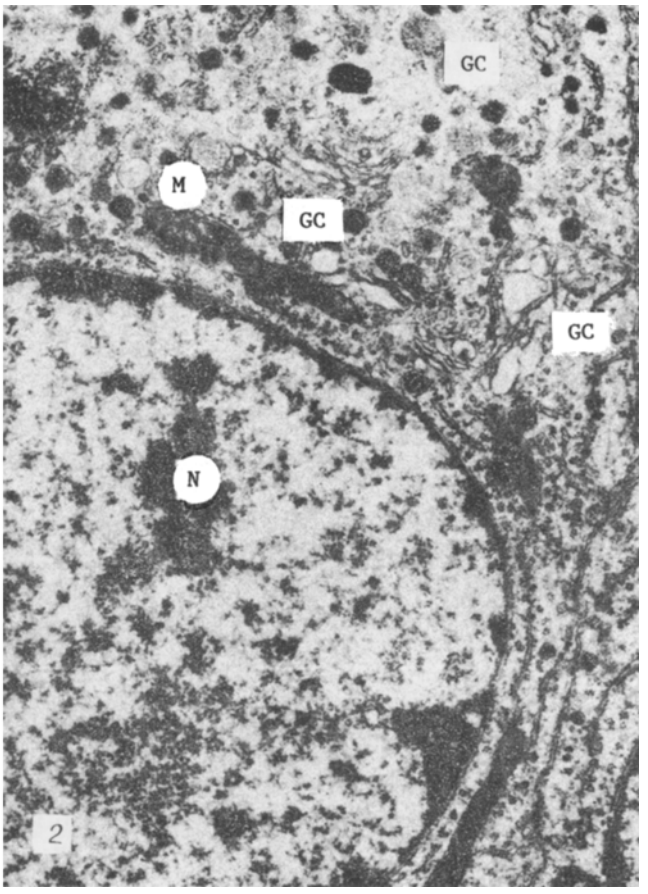

Fig. 1 Fig. 2

Fig. 1. Fragment of venule in TG of experimental rats. Edema of basement membrane (BM), nerve fibers (NF) in state of destruction, sludging of erythrocytes (Er). P) Pericyte. 7700×.

Fig. 2. C-cell in TG of experimental rat. Hyperplasia of lamellae of Golgi complex (GC). N) Nucleus, M) mitochondria.

the blood levels of Ca^{2+} and inorganic phosphorus [1, 2, 14]. Mechanisms of adaptive and compensatory reactions of C and chromaffin cells during chemical desympathization have virtually not been studied.

The aim of this investigation was to study structural and functional changes in the C cells of TG and chromaffin cells of AG in young rats during partial desympathization by guanethidine.

EXPERIMENTAL METHOD

Experiments were carried out on 24 normally developing and desympathized male albino rats of infantile age (1 month). Desympathization was carried out by the method described by the present writers previously, leading to death of more than 80% of sympathetic cells in the rats' nervous system [5], but leaving cholinergic fibers intact [9]. On reaching the age of 1 month the animals were decapitated under ether anesthesia. The serum calcitonin (CT) level was determined by radioimmunoassay, using the RIA-Mat Calcitonin II commercial test kit (Mallinckrodt Diagnostica, West Germany). The calcium concentration was estimated by biotest kits from "Chemapol" (Czechoslovakia). For histological investigation TG and AG were fixed in Bouin's fluid and embedded in paraffin wax. Serial sections were impregnated with silver nitrate [13] and stained with hematoxylin and eosin. The number of C-cells per field of vision of the microscope (objective 40×, ocular 15×) in TG was counted and the approximate volumes of their nuclei and cytoplasm were calculated by the formula for an ellipsoid. In sections of AG the diameter of the medulla and the area of the chromaffin cells were determined. Material for electron microscopy was fixed in glutaraldehyde, postfixed in OsO_4, and embedded in a mixture of Epon and Araldite M

TABLE 1. Morphological and Functional Parameters of TG and AG after Partial Desympathization by Guanethidine (M ± m)

Parameter	Control	Experiment
Serum CT, pM	35,9±5,3 (n=7)	18,5±3,5* (n=7)
Number of C-cells per field of vision of TG, conventional	6,96±0,3 (n=7)	10,3±0,9* (n=7)
Volume of nuclei of C-cells, μ^3	45,8±2,1 (n=7)	22,0±2,2* (n=7)
Volume of cytoplasm of C-cells, μ^3	134,2±6,35 (n=7)	214,4±10,1* (n=7)
Density of adrenergic parafollicular fibers of TG, conventional units	15,8±0,9 (n=5)	4,0±0,6* (n=5)
Density of adrenergic fibers of perivascular plexuses of TG, conventional units	2,3±0,2 (n=5)	1,4±0,1* (n=5)
Diameter of adrenal cortex, μ	602±9 (n=7)	635±13 (n=7)
Diameter of adrenal medulla, μ	370±9 (n=7)	575±11* (n=7)
Area of nuclei of chromaffin cells, μ^2	19,2±0,6 (n=7)	23,5±0,7* (n=7)

Legend. n) Number of animals; *$p < 0.05$ compared with control.

resins. Ultrathin sections were studied in the JEM-100C electron microscope. Frozen sections for fluorescence-histochemical analysis were treated with glyoxalic acid [6]. The density of adrenergic fibers in the interfollicular nervous network and perivascular plexuses was determined with the aid of a planimetric grid. For cytospectrofluorometric estimation of catecholamines, the LYUMAM-2 microscope with FMÉL-1 attachment were used. The results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

A state of hypofunction of the C-cell apparatus was found in the thyroid parenchyma of the experimental rats, and was confirmed by the results of radioimmunoassay of CT in the blood serum. The volume of the nuclei of the C-cells, the most informative criterion of their functional activity, was significantly reduced. The dimensions of the cytoplasm of the C-cells showed a distinct increase (Table 1) and solitary forms with reduced granulation or completely degranulated forms were seen among them. These cells could be divided into two types. The type 1 cells were characterized by destructive changes of the Golgi complex and mitochondria, with the formation of myelinlike structures. These processes are known to be most marked when the trophic function of the vessels is disturbed. Changes in the microcirculatory bed following sympathetic denervation could be clearly traced in virtually all the vessels of TG: the endotheliocytes were swollen or flattened, the basement membrane edematous, and stasis and sludging of the erythrocytes were observed, with the formation of a niche in the walls of the venules and blood capillaries (Fig. 1). In the type 2 C cells hyperplastic changes were observed in the lamellae and vesicles of the Golgi complex (Fig. 2).

The greater part of the C-cell population consisted of cells with a well marked argyrophilic reaction and no signs of degranulation, possibly because of slowing of the rate of evacuation of hormonal products into the blood stream. Weakening of the internal secretory activity of the C-cells of the desympathized rats was evidently due to a decrease in the number of incoming sympathetic impulses. Microspectral analysis showed that the catecholamine concentration in adrenergic nerve fibers was reduced in sections through TG. Luminescence microscopy revealed a decrease in density of the fluorescent fibers of the parafollicular network and perivascular plexuses. Since the number of C-cells per field of vision of

the microscope (Table 1) and the mass of TG of the experimental animals (11.4 ± 0.5 mg) were higher than the corresponding parameters in rats of the control group (8.6 ± 0.7 mg, $r < 0.01$), it can be concluded that after desympathization, hyperplasia of the C-cell population was observed. Incidentally, an increase in proliferative activity against the background of desympathization also has been observed in the epithelium of the follicles [4]. This was due to a decrease in the catecholamine concentration in the thyroid parenchyma and also, evidently, to reduced formation of adrenalin-chalone complexes, which have an antimitotic action on tissue cells [4], as a result of disturbance of sympathetic mediation. Under these circumstances hyperplastic changes in C-cells after desympathization can also be regarded as the result of adaptation, aimed at restoring the hormonal balance in the blood stream, with the aim of depositing circulating mineral substances in the bone tissue, including calcium (the blood serum calcium concentration of the experimental rats was 2.9 ± 0.2 compared with 2.1 ± 0.3 mM in the control, $P < 0.05$). These results are in agreement with those obtained by other workers [12] who observed slowing of deposition of bone substance on the surface of the dental alveolae of rats after removal of the superior cervical sympathetic ganglia.

The amplitude and vector of compensatory and adaptive reactions of the chromaffin cells of AG of the experimental rats differed significantly from those in the C-cells of TG. The volume of AG increased mainly on account of an increase in size of the medullary layer. Changes in the medulla were manifested as a vascular reaction: dilatation of the sinusoids and disturbance of the structure of the endotheliocyte nuclei. The area of the nuclei of the chromaffin cells (Table 1) and the number of polymorphic mitochondria in the cytoplasm were increased, while some secretory granules were completely without electron-dense contents. The catecholamine concentration in sections through AG was a little higher (1.07 ± 0.08 conventional unit) in the experimental series compared with the control (0.85 ± 0.06 conventional unit). Thus the chromaffin cells of AG have greater structural and functional lability than the C-cells of TG in response to inhibition of the sympathetic innervation of the body.

It is interesting to compare these results with those obtained by other workers [3] who observed an increase in the catecholamine concentration in the blood stream of desympathized animals. Consequently, the structural and functional changes which we found in AG point to active involvement of the chromaffin cells of the medulla in adaptive and compensatory reactions in desympathized rats aimed at restoring the neurotransmitter concentrations in the tissues in order to correct the deficit of sympathetic mediation.

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