protein "for export," including secreting cells, among them, fibroblasts. On the basis of the morphological differences between the cells as regards the type of protein synthesis, described above, two hypotheses can be put forward: either two types of cells with myogenic and fibrogenic directions of differentiation, i.e., cells differing in their nature, are present beneath the sarcolemma of the muscle fiber in an undifferentiated state, or the satellite cell is pluripotent and is a cambial cell which can develop in at least two directions. It is impossible to settle this problem finally at the present stage because of the absence of reliable cell markers [5].

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RAT DIAPHRAGM NEUROMUSCULAR JUNCTION IN EXPERIMENTAL HYPOPARATHYROIDISM

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Calcium ions (Ca^{++}) play an essential role in the regulation of mediator secretion [5]. However, the bulk of the experimental data obtained by the study of the effect of Ca^{++} on synaptic transmission has been obtained in vitro [2], whereas several factors exist that require the introduction of certain corrections into results obtained on isolated nerve-muscle preparations in vitro [4]. The aim of the present investigation was accordingly to study the ultrastructure of the neuromuscular junction in experimental endogenous hypocalcemia induced by partial destruction of the parathyroid glands.

EXPERIMENTAL METHODS

Experiments were carried out on noninbred albino rats weighing 180 g. Under ether anesthesia the parathyroid glands were partially destroyed by means of a thermocautery. The effectiveness of the operation was assessed by determining the plasma calcium level before and after the operation. The diaphragm was fixed in vivo by injecting a cold solution of formol-sucrose into the peritoneal and pleural cavities of the anesthetized animal. After removal of the synaptic zone from the muscle, the tissue was dehydrated in ethanol and acetone and embedded in Araldite.

Ultrathin sections were cut on the LKB III Ultratome, stained by Reynolds' method, and examined in the JEM-7a electron microscope. The electron micrographs were subjected to morphometric analysis with the aid of the Leitz ASM semiautomatic image analyzer. The number of synaptic vesicles was counted and their perimeter measured. The perimeter of the vesicles was calculated relative to the equivalent volume of a sphere.

EXPERIMENTAL RESULTS

The most marked ultrastructural changes in the neuromuscular junction were observed on the 7th day after the operation. The cellular structure of the synapse was unchanged this

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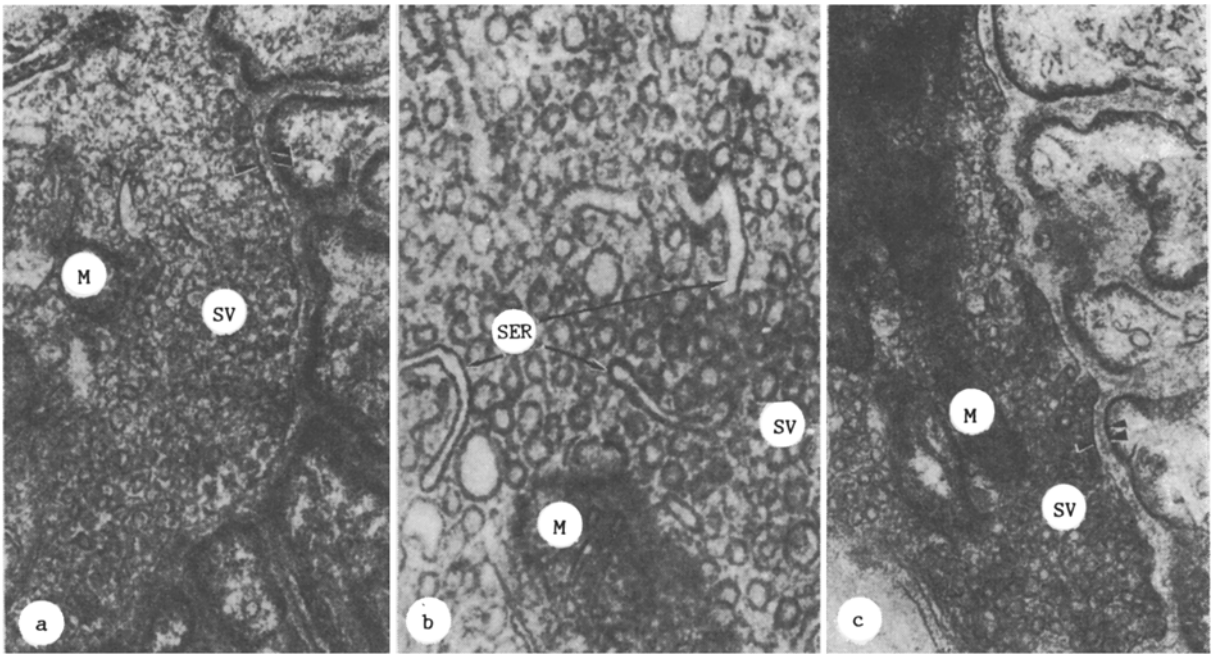

Fig. 1. Ultrastructure of neuromuscular junction in rat diaphragm after partial destruction of parathyroid glands. a) Synapse on 7th day after destruction of parathyroid glands (80,000×); b) elements of smooth endoplasmic reticulum (SER) in axon terminal, 7th day after destruction of parathyroid glands (112,000×); c) synapse on 12th day after destruction of parathyroid glands (80,000×). One arrow) presynaptic membrane, two arrows) postsynaptic membrane. SV) Synaptic vesicles, M) mitochondria.

time, but considerable changes were observed in subcellular structures: the electron density of the axoplasm of the mitochondrial matrix, and of the contents of the synaptic vesicles in the axon terminals was reduced (intact rats 290 ± 20, on the 7th day after the operation 190 ± 30). The distribution of vesicles by size differed significantly from the control, intact rats. Mitochrondria were displaced toward the presynaptic membrane. Elements of the endoplasmic reticulum were dilated (Fig. 1b). The electron density of the contents of the synaptic cleft was increased. The cholinoceptive zone on the postsynaptic membrane was appreciably widened. On the 12th day the structural changes were less marked (Fig. 1c), although the average number of synaptic vesicles was reduced even more (160 ± 40). These changes were less marked on the 20th day than on the 7th day, the number of vesicles was increased (210 ± 30), and their distribution by volume differed as before from the control.

Electrophysiological investigations revealed maximal changes on the 5th-8th days after the operation. They consisted of an increase in the frequency and amplitude of miniature end-plate potentials (MEPP) and the appearance of giant MEPP, sometimes changing into full end-plate potentials (EPP) [1].

It will thus be clear that disturbances of calcium metabolism in experimental hypoparathyroidism leads to changes in the structure of the synapse and a significant change in the character of mediator secretion. This confirms the important role of Ca^{++} in the regulation of intracellular processes especially synaptic.

The probable cause of the changes described above is the accumulation of Ca^{++} within the axon terminals. Although no direct proof of this was obtained, some indirect evidence can be cited: 1) the external appearance of the axon terminals resembles the picture produced when a nerve-muscle preparation is placed in medium with a high calcium concentration; 2) the increase in frequency of MEPP and the decrease in the number of synaptic vesicles in nerve endings reflect in an increase in the probability of secretion of a "quantum" or transmitter; 3) characteristic changes took place in the organelles, associated with Ca^{++} accumulation (endoplasmic reticulum, mitochondria).

Structural-functional parallels: a combination of increased release of transmitter with a reduction in the number of synaptic vesicles in the terminals, and also changes in the volume of the vesicles and in the size of the "quantum" of transmitter demonstrate a connection

between the "quantum" of transmitter and the synaptic vesicles. The state of affairs must be noted because recently doubts have been expressed on this problem [3].

Attention may also be directed toward the high reserves of reliability of the neuromuscular junction, for despite changes in a factor as important for the regulation of synaptic processes as the calcium ion concentration, synaptic function remains adequate for performance of the vital functions, in this case respiration.

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MORPHOLOGICAL ANALYSIS OF THE EFFECT OF DESYMPATHIZATION ON INTESTINAL IMMUNE MECHANISMS

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Investigators in recent years have shown increased interest in the immunologic defense of the intestine, in preventing adsorption of foreign, including pathogenic, agents and their passage through the intestinal barrier. The main components of the effector stage of this system are the intraepithelial lymphocytes (IEL), responsible for the cell-mediated response [14], and the plasma cells, producing 7S IgA components and the j-chain of secretory immunoglobulins [13]. Synthesis of the secretory component and assembly of 7S IgA take place in enterocytes [12]. Whereas the basic principles governing the function of this system and their structural basis have been investigated in sufficient detail [4, 5, 9], the importance of the neural factor in the maintenance of its structural and functional integrity has received little study. Only lymphoid infiltration of the stroma and an increase in the number of IEL in the villi of the jejunum have been demonstrated in rats after vagotomy [3]. The role of the sympathetic division of the autonomic nervous system in the regulation of the structure and function of the immune mechanisms of the intestine, however, has not been investigated. Yet the elucidation of this problem is of definite theoretical and practical interest, first, for a deeper understanding of the pathogenesis of neurodystrophic processes and, second, in connection with the use of operations involving disturbance of the sympathetic innervation of the intestine in clinical practice [7].

On the basis of the facts described above, it was decided to study the state of the effector components of the immunologic defense system of the small intestine and its epithelium after surgical desympathization.

EXPERIMENTAL METHODS

Experiments were carried out on 72 noninbred male albino rats weighing 200-220 g. Periarterial sympathectomy was performed on the cranial mesenteric artery of 36 of them and the remaining animals served as the control. The effectiveness of intestinal desympathization

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