

6. M. Drake, *Gastro-Oesophageal Cytology*, Vol. 11, No. 40, Basel (1985), pp. 93-112.
7. F. Keibell and F. Mall, *Manual of Human Embryology*, Philadelphia (1912), pp. 331-403; 570-709
8. B. M. Patten, *Human Embryology*, New York (1959).
9. D. Rudnic, *Ann. New York Acad. Sci.*, **55**, No. 6, 109 (1952).
10. C. Zaino and H. G. Jacobson, *Radiology*, **89**, No. 4, 639 (1967).

EFFECT OF BURNS ON ULTRASTRUCTURE OF THE GANGLIA NODOSA

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Burn trauma leads to severe structural and functional disturbances of all organs and systems of the human body [6, 11, 12]. According to data in the literature, in burns all parts of the central and peripheral nervous system are involved in the pathological process [2, 7-9]. Light-optical studies of autonomic ganglia in various diseases, including burns, have shown combined lesions including, on the one hand, atrophic and degenerative changes, and on the other hand, compensatory and adaptive changes [1, 3-5, 10]. The paucity of information on ultrastructural changes in autonomic ganglia in burns is highly characteristic of this condition, more especially because the use of classical neurohistological methods of investigation has shown that the nervous system plays an important role in the pathogenesis of the burn syndrome [2, 4, 5, 7]. The aim of this investigation was to study ultrastructural changes in the ganglia nodosa of the vagus nerves at different stages of burn trauma.

EXPERIMENTAL METHOD

The investigation was conducted on autopsy material from 30 persons dying at different periods of burn trauma (shock, toxemia, septicotoxemia, burn cachexia), and 10 hitherto clinically healthy persons dying accidentally. The victims were from 14 to 75 years of age. Autopsy was carried out soon (1-3 h) after death. The test objects were the ganglia nodosa of the vagus nerves. Material was fixed in 1% glutaraldehyde solution and postfixed in buffered 1% osmium tetroxide solution, dehydrated with alcohols, and embedded in a mixture of Epon and Araldite. Sections cut on an LKB-4801A Ultramicrotome were stained in uranyl acetate and counterstained with a solution of lead monoxide and studied in the JEM-100B electron microscope. Material for histological study was fixed in 10-12% neutral formalin and embedded in paraffin wax. Sections were stained with hematoxylin and eosin, with picrofuchsin by Van Gieson's method, and also by Nissl's method.

EXPERIMENTAL RESULTS

Histological investigation revealed marked congestion of capillaries and ectasia of the lymphatics. In sections stained by Nissl's method degenerative changes were observed in the ganglion cells in the form of swelling of neurons and their central and total chromatolysis. In most neurons the chromatophilic substance was in a state of solution or was diffusely scattered in the cytoplasm in the form of tiny granules. In some neurons the chromatophilic substance was located at the periphery of the cells in the form of single small clumps, but sometimes it was concentrated around the nucleus. Neurons with total chromatolysis also were found. Degeneratively changed nerve cells, in a state of necrobiosis, often were undergoing neuronophagy by satellite cells and connective-tissue cells, especially in the later stages of burn trauma.

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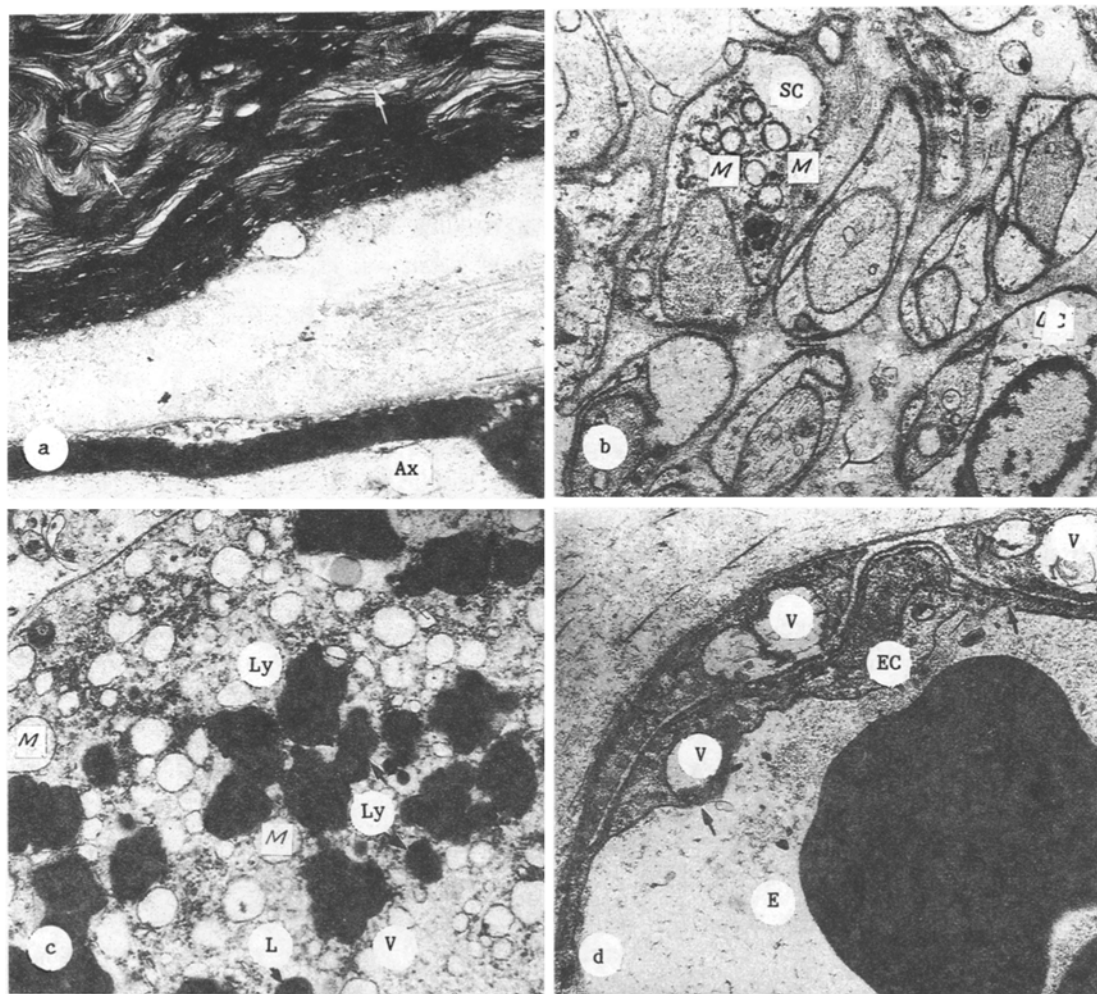


Fig. 1. Ultrastructure of ganglia nodosa at different periods of burn trauma: a) swelling and splitting (arrow) of myelin sheath of medullated axon (Ax) (burn shock); b) translucency of cytoplasm, swelling of mitochondria (M) with destruction of outer membranes of Schwann cells (SC) and decrease in number of microtubules of nonmedullated processes (burn toxemia); c) increase in number of lysosomes (Ly), lipid inclusions (L), and swelling and transparency of matrix of mitochondria (M) and vacuolation (V) in cytoplasm of neurons (burn septicotoxemia); d) formation of large vacuoles (V) with myelin inclusions in cytoplasm of endothelial cells (EC) of blood capillaries and in pericytes (arrow), lysis of plasma membranes, erythrocyte (E) visible in lumen of vessel (burn septicotoxemia). Magnification: a-d) 12,000 \times .

Analysis of the ultrastructure of neurons of the ganglia nodosa in patients dying in the early period after burn trauma (a period of burn shock and toxemia) revealed considerable changes in the mitochondria, endoplasmic reticulum, and nuclei. Many nerve cells had an electron-dense cytoplasmic matrix, against the dark background of which the swollen mitochondria and dilated tubules of the endoplasmic reticulum of which the swollen mitochondria and dilated tubules of the endoplasmic reticulum stood out in sharp contrast. The nuclei of these hyperchromic neurons were distinguished by tortuous outlines and an osmiophilic nucleoplasm, in which aggregation of chromatin and its concentration on the inner aspect of the nuclear membrane were observed. The outer layer of the neuron membrane was partly destroyed. Changes in nervous processes corresponded largely to the structural changes in the perikarya. The nerve fibers contained lipofuscin granules, the cytoplasmic matrix of some of which had increased electron density. The compact arrangement of many of the parallel microtubules in the nervous processes in some places created the impression of marked osmiophilia of the matrix of the nerve fiber, with signs of swelling and splitting of the myelin sheath of the medullated axon (Fig. 1a). Nerve fibers containing myelin-like bodies, numerous pale and osmiophilic polymorphic vesicles, and mitochondria also were found in the ganglia. The number of microtubules and neurofilaments was reduced (Fig. 1b).

In the study of the ultrastructure of the ganglia nodosa in patients dying in the late stages after burn trauma (the period of burn septicotoxemia), more profound changes were observed in the neurons and their processes. In the cytoplasm of the neurons there was a marked increase in the number and size of the lysosomes, which occupied a large part of the area of the cytoplasm. The appearance of many lysosomes in the cytoplasm of the nerve cells of the ganglia nodosa of patients dying in the late stages after burn trauma must be regarded as the initial stages of destruction of the cytoplasm of the neurons. They were mainly located in the perinuclear zone. Translucency of the nucleoplasm and accumulation of chromatin granules in the form of a thin uneven band along the inner aspect of the nuclear membrane, was observed in the nuclei. The outer layer of the nuclear membrane was destroyed in some places. The mitochondria were swollen, with a translucent matrix, and their inner membranes were almost invisible. The number of ribosomes and glycogen granules was sharply reduced. Tubules of the endoplasmic reticulum were fragmented and distributed among lysosomes in the form of separate vesicles. Areas of disintegrated membranes and of small osmiophilic granules, signifying the initial stages of intracellular necrosis, were present in the cytoplasm. In these areas the organelles were disconnected, the cytoplasm was translucent and contained a mass of small vacuoles, and scattered fragments of membranes formed by aggregation of osmiophilic granules. The appearance of numerous lipid inclusions and lysosomes in the cytoplasm of the neurons was noted (Fig. 1c). Processes of the glial cells were mainly disconnected and swollen, with lysis of the neurofilaments in them. Because of the apposition of the processes to the body of the neurons, free spaces were formed, with eventual disturbance of their contacts with the neuron body.

In the later stages of burn trauma, ending in death, marked changes in the blood capillaries were observed in the ganglia nodosa of the vagus nerves. The first feature to be noted was disturbance of the integrity of the cytoplasmic membrane of the endothelial cells. This was seen most clearly in the intima of the blood vessels. Accumulation of erythrocytes and platelets was observed in the lumen of the blood capillaries. Processes of the endothelial cells were somewhat swollen. The mitochondria were swollen, with a translucent matrix, and were located in the perinuclear space, where they came to resemble vacuoles. Translucency of the nucleoplasm and partial destruction of the outer layer of the nuclear membrane were observed in the nuclei. The basement membrane could be traced in the endothelial cells but it was destroyed in places. Destructive changes also were observed in the pericytes (Fig. 1d).

Thus on the basis of analysis of the ultrastructure of neurons and endotheliocytes of the ganglia nodosa of the vagus nerves in patients dying at different periods of burn trauma, changes were found in the structure of the mitochondria and endoplasmic reticulum, and in the nuclei and cell membranes. As a result of disturbance of energy metabolism in the cells, as shown by disappearance of glycogen granules and vacuolation of the mitochondria, gradual destruction of the intracellular organelles and accumulation of breakdown products took place, leading to an increase in the number and size of lysosomes involved in the utilization of these products. In extensive zones of damage to cell membranes and of insufficiency of lysosomal function, and also of destruction of the lysosomes themselves, intracellular proteolytic enzymes were activated and areas of necrosis appeared in the neurons. The most marked destructive changes in the neurons and endotheliocytes of the blood capillaries of the ganglia nodosa were observed in patients dying in the later periods of burn trauma.

LITERATURE CITED

1. S. S. Vail', *Arkh. Patol.*, No. 1, 43 (1937).
2. P. V. Voloshin, *Pathology of the Nervous System in Burn Trauma* [in Russian], Kiev (1982).
3. D. Yu. Guseinov, *Structural Changes in Receptors and Synapses in Pathology* [in Russian], Baku (1965).
4. I. M. Isaev, *Byull. Éksp. Biol. Med.*, No. 12, 754 (1988).
5. I. M. Isaev, L. I. Muzykant, V. P. Tumanov, et al., *Arkh. Patol.*, No. 2, 47 (1989).
6. L. M. Klyachkin and V. M. Pinchuk, *Burns* [in Russian], Leningrad (1969).
7. R. A. Perevereva and M. I. Dolgina, *Arkh. Patol.*, No. 9, 47 (1970).
8. D. S. Sarkisov, B. V. Vtyurin, and V. P. Tumanov, *Abstracts of Proceedings of the First All-Union Conference on Thermal Burns* [in Russian], Moscow (1972), pp. 64-66.
9. V. P. Tumanov and M. D. Malamud, *Changes in the Central Nervous System in Thermal, Radiation, and Combined Trauma* [in Russian], Kishinev (1977).
10. N. E. Yarygin and V. N. Yarygin, *Pathological and Adaptive Changes in Neurons* [in Russian], Moscow (1973).
11. Chen Yi-Cheng, L. Ngao Shi Jing Gutan, Li Yuan-Ping, et al., *Burns*, 11, No. 6, 408 (1985).
12. R. E. Salisbury and G. P. Dingeldein, *Clin. Orthop.*, 163, 92 (1982).