

KEY WORDS: burns, autonomic ganglia; adrenergic and cholinergic nerve structures; lungs

The study of the structure of the sympathetic ganglia in burns by classical neurohistological methods has demonstrated a combination of destructive lesions with compensatory and adaptive reactions in the ganglia. There is little information in the literature on changes in the autonomic ganglia in burns. Nevertheless, it has been shown that extensive burns give rise to pathological changes in internal organs and systems [2-6]. In view of the current urgency of the problem of the after-effects of burns, it is interesting to study the reactions of different portions of the autonomic nervous system by modern neurohistochemical methods, capable of detecting cholinergic and adrenergic nerve structures.

The absence of neurohistochemical data on changes in the peripheral nervous system during burns, by contrast with morphological changes observed in the CNS and internal organs in thermal burns is very characteristic. There have been few electron-microscopic studies of changes in the peripheral nervous system in burns. Yet research of this kind is interesting because of the important role played by the peripheral nervous system in the pathogenesis of burns, as one of the most important channels for the transmission of pathological impulses to the internal organs. A study of the structural-chemical organization of cholinergic and adrenergic structures of the human autonomic ganglia under normal and pathological conditions, such as burns, is not only of considerable theoretical interest, but also of definite practical importance. Similar investigations have been described in the literature in diseases of the cardiovascular system, essential hypertension, and so on [1, 7, 8]. However, this problem has not been adequately investigated and such information as is available on the possibility of studying the adrenergic and cholinergic innervation on cadaveric material is contradictory [10, 11].

Thus modern views on the response of the autonomic ganglia to thermal burns are based on data obtained mainly by classical neurohistological methods.

Meanwhile, no attempt has been made to analyze changes in adrenergic and cholinergic nerve structures of the autonomic ganglia in burns by modern neurohistochemical methods.

#### EXPERIMENTAL METHOD

The experimental material consisted of autonomic ganglia (superior cervical sympathetic and stellate ganglia, ganglia nodosa, ganglia of the solar plexus) and lung tissues obtained from 20 cadavers of men and women aged from 19 to 85 years, dying from various complications of burns. The ganglia taken at various times (up to 10 h) after death were frozen with dry ice. Frozen sections through the ganglia and lungs were treated with 2% glyoxylic acid solution by the method of Shvalev et al. [9], and examined in the luminescence microscope. Cholinergic nerve structures were revealed by the method of Karnovsky and Roots. Ganglia from clinically normal individuals, dying from accidental trauma, and with no other significant pathology, were used as the control.

#### EXPERIMENTAL RESULTS

Neurohistochemical investigation revealed cholinergic nerve structures in the sympathetic ganglia and ganglia nodosa.

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Department of Pathological Anatomy, N. Narimanov Azerbaidzhan Medical Institute, Baku. (Presented by Academician of the Academy of Medical Sciences of the USSR, D. S. Sarkisov.) Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 108, No. 7, pp. 107-109, July, 1989. Original article submitted November 4, 1988.

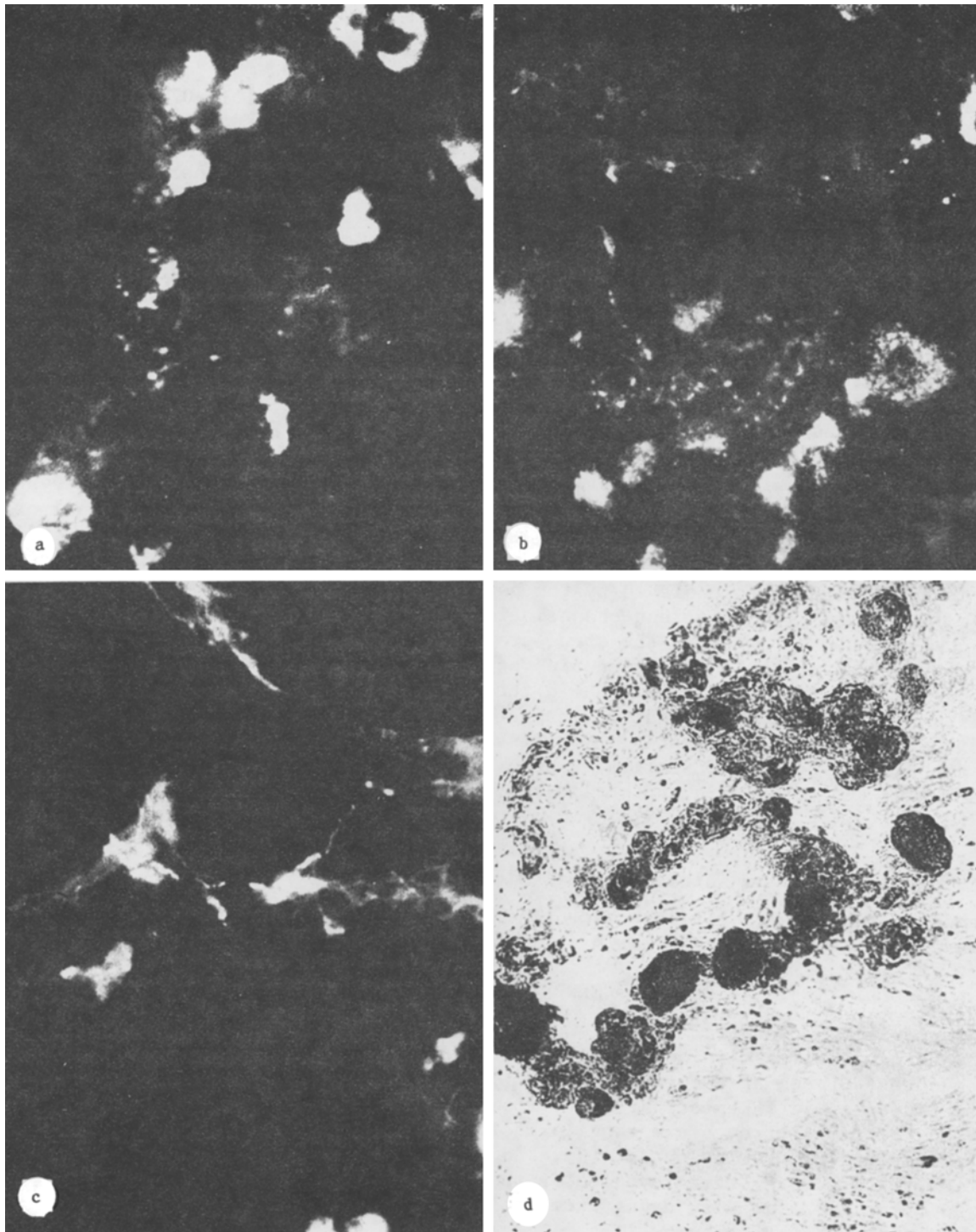


Fig. 1. Changes in adrenergic and cholinergic nerve structures of autonomic ganglia and lungs in burns. Magnification 120; objective 40, ocular, Homa 13. a) Bright fluorescence of catecholamines in cytoplasm of neurons of superior cervical sympathetic ganglia. Man aged 19 years. Incubation of sections in 2% solution of glyoxylic acid; b) uneven distribution of catecholamines in cytoplasm of nerve cells of stellate ganglia. The same case; c) adrenergic nerve plexus around artery in parenchyma of lungs. The same case; d) high concentration of AChE and lipofuscin granules in neurons of ganglia nodosa and destruction of nerve fibers. Woman aged 75 years. Method of Karnovsky and Roots.

In the superior cervical sympathetic ganglion bundles of nerve fibers were distinguished by their comparatively high acetylcholinesterase (AChE) activity. In some preparations areas of destruction of nerve cells with deposition of pigmented masses in many of them were observed. In another preparation, destruction of most sympathetic nerve cells also was found. These features indicate a severe disturbance of adaptive and trophic processes of the nervous system. The study of adrenergic nerve structures revealed bright fluorescence of catecholamines in the cytoplasm of neurons of the superior cervical sympathetic ganglia, whereas terminals of bundles of nerve fibers in the ganglia and tissues of the lungs were distinguished by a sharp decrease in their level of luminescence (Fig. 1a, c).

When the stellate ganglion was studied, the lower activity of AChE in the nerve cell bodies compared with the adjacent bundles of nerve fibers was noted. Meanwhile high AChE activity was found in walls of blood vessels surrounding the ganglia. Among neurons of the stellate ganglia, especially at the periphery of the ganglion, nerve cells with high AChE activity were found.

In these cases catecholamine luminescence in nerve structures of the stellate ganglia was observed quite clearly, although in many areas of the ganglion, foci with a sharply reduced luminescence or its total absence could be seen against this background (Fig. 1b). Incidentally, fluorescent adrenergic structures were found more often in the peripheral zones of the stellate ganglia, in agreement with data in the literature [1].

Analysis of the adrenergic and cholinergic nerve structures of the solar plexus reveal a picture which was largely reminiscent of the structure of the stellate and superior cervical sympathetic ganglia. The low AChE activity in the nerve cells was noted. In many sections through the ganglia of the solar plexus, many dying nerve cells were found.

In the ganglion nodosum, unlike the sympathetic ganglia, a high AChE concentration was found in the cytoplasm of the neurons, with accumulation of lipofuscin granules yet, at the same time, with considerable destruction of nerve fibers (Fig. 1d). Evidently in burns the afferent stage is more severely affected than the efferent [6].

Besides changes in the parenchyma of the lungs, damage also was found to the receptor apparatus, with marked destruction of sensory nerve endings. Unchanged thin myelinated nerve fibers also were found along the course of the bronchi. It can be concluded from the changes in the lungs that in burns there is visceral damage to adrenergic and cholinergic nerve structures.

Analysis of the results indicate that the autonomic ganglia are complex centers for nervous regulation of various systems of the body. Different kinds of adrenergic and cholinergic structures, which undergo marked changes in pathology, are represented in them.

We found two types of changes of nerve cells in the autonomic ganglia. Changes of one type had the appearance of homogeneously stained cells, reacting intensively for the presence of mediators, whereas in others numerous small vacuoles were found. Where adrenergic and cholinergic nerve structures were discovered, changes also were found in the staining properties of the nerve fibers and pooling of the neuropil.

In the elderly and old subjects adrenergic fluorescence was much weaker than in young subjects. Weakening of luminescence was more marked in the cytoplasm of the neurons than in plexuses of adrenergic fibers. Toward the onset of old age, the catecholamine concentration falls sharply in many adrenergic structures of the ganglia. This is in agreement with data of other workers [1, 12, 13]. Similar changes also were observed, depending on the cause of death, at different stages of burn trauma.

In elderly subjects activity of adrenergic and cholinergic nerve structures was usually reduced both in the ganglia and in the lung tissues. In the case of adrenergic nerve structures, fluorescence of many sympathetic nerve endings in the composition of nerve plexuses around the vessels of the lungs was no longer found. Individual adrenergic nerve fibers were discovered in the ganglia, but there were more sympathetic and parasympathetic neurons containing numerous lipofuscin granules than in young subjects dying from burns.

Thus in young subjects cholinergic and adrenergic nerve structures are distinguished by their high reactivity and their more marked compensatory powers, even in severe burns, and this must be taken into account when the strategy of treatment is chosen.

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# STRUCTURAL CHANGES IN THE LIVER IN EXPERIMENTAL CHRONIC HEPATITIS AND ITS CORRECTION BY BENZONAL

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UDC 616.36-002.2-092.9-085.213-036.8-076

KEY WORDS: structure of the liver; chronic hepatitis; correction

The essence of the problem of reversibility of pathological changes in the liver is stimulation of regeneration, and its solution depends on the level of cellular and intracellular repair processes [3]. The search is currently in progress for new and effective preparations which will stimulate regeneration of the liver when affected by chronic disease. The anticonvulsant drug benzonol, a barbituric acid derivative with a stimulating effect on activity of the microsomal enzymes of the liver, is particularly valuable in this respect and has been accepted by the pharmacologic committee of the USSR for wide use in the treatment of liver diseases.

## EXPERIMENTAL METHOD

Experiments were carried out on 30 male albino rats divided into three groups. Animals of group 1 were intact, in animals of group 2 experimental chronic hepatitis was induced by injection of CCl<sub>4</sub> twice a week for 60 days in a dose of 2 mg/kg body weight; in the animals of group 3 experimental chronic hepatitis also was induced but they were treated with benzonol in a dose of 50 mg/kg for 10 days. Liver sections were stained with hematoxylin and eosin and by histochemical reactions (PAS, for RNA, by Brachet's method). The material was studied in the electron microscope by a morphometric method. The number of binuclear cells and mitoses to every 10,000 hepatocytes was calculated, and areas of necrosis were determined planimetrically.

## EXPERIMENTAL RESULTS

Marked structural changes were discovered in the liver of the animals of group 2 in the form of swelling of the trabecular structure, static congestion of the vessels, and degenerative and necrotic changes. Destruction of the limiting membrane by lymphohistiocytic infiltration led to the development of foci of periportal necrosis, with the appearance of pale patches, occupying a considerable area, as a result of reduction of the RNA and glycogen content (Table 1). Similar changes constitute the picture of active chronic hepatitis of virus etiology [2, 6, 7]. Electron-microscopic examination revealed disturbances of the fine structure of the hepatocytes. In most of them the number of tubules of the smooth (SER) and rough

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Department of Morphology, Central Research Institute, Tashkent Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR, A. P. Avtsyn.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 108, No. 7, pp. 109-112, July, 1989. Original article submitted May 3, 1988.