PATHOMORPHOLOGY OF GANGLION NODOSUM OF THE VAGUS NERVE IN BURNS

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The study of the pathogenesis of burns is one of the most important problems in medical biology. Thermal burns belong to the category of extremely severe injuries, frequently terminating in death or in prolonged and, sometimes, permanent unfitness for work. An important role in the complex pathogenesis of burns is played by the nervous system [12], but no systematic investigations of the time course of the morphological changes in peripheral nerve ganglia at successive stages of burn trauma have yet been undertaken. The state of the peripheral nerve ganglia has been sufficiently well studied under normal conditions and also in various diseases [1, 3-5, 7, 8, 10, 13-15]. The scarcity of information about changes in peripheral autonomic nerve ganglia in burns is very characteristic in this connection, although it has been proved that an extensive deep burn of the body skin can give rise to various functional and morphological disturbances of the internal organs and systems [2, 6, 9]. It was accordingly decided to study the pathomorphology of the ganglian nodosum of the vagus nerve at different stages of burn trauma.

EXPERIMENTAL METHOD

The investigation was conducted on autopsy material from 65 persons dying at different periods of burn trauma (shock, toxemia, septicotoxemia, burn emaciation) and of 10 clinically healthy persons dying from accidental trauma. The victims were aged from 19 to 85 years. Autopsy was carried out soon (within 3-6 h) after death. The test objects were the ganglia nodosa of the vagus nerves. After fixation in 12-20% neutral formalin solutions the material was embedded in paraffin wax. Sections were stained with hematoxylin and eosin and with picrofuchsine by Van Gieson's method and were impregnated with silver nitrate by the methods of Bielschowsky—Gros and Campos. Nissel's method and neurohistochemical methods of staining adrenergic structures by incubation of sections in 2% glyoxylic acid solutions also were used.

EXPERIMENTAL RESULTS

The pattern of the structural changes in the ganglia nodosa of the vagus nerves at different stages of burn trauma is one of extreme diversity of dystrophic and regenerative processes. These changes as a rule are twofold in character: dystrophic or atrophic changes in the bodies and processes of sensory neurons, on the one hand, and their hypertrophy, on the other hand. The earliest type of change is swelling of the ganglionic nerve cells. In this phase the Nissl's corpuscles break up into smaller clumps and granules, and the ground substance of the nerve cell stains pale blue or pale violet. Changes in the chromatophilic substances are always found in swollen ganglionic nerve cells.

In patients dying in the period of burn shock marked congestion of the capillaries and lymphangiectasia were often observed in the parenchyma of the ganglion nodosum. In the impregnated preparations the swollen nerve cells took up silver salts less intensively and different stages of dystrophy and destruction of sensory neurons could be clearly seen in them (Fig. 1a). A no less frequent form of change in the ganglionic nerve cells was atrophy and shrinking of the neurons. The nerve cells were reduced in volume and became 2-3 times smaller than unchanged neurons of average size, and often they became elongated or irregular in shape. In some nerve cells the intercellular net consisted of thickened and argyrophilic neurofibrils, whereas in others the neurofibrillar apparatus was completely invisible. The perikaryon of the nerve cells in these cases became homogeneous in appearance and stained diffusely black.

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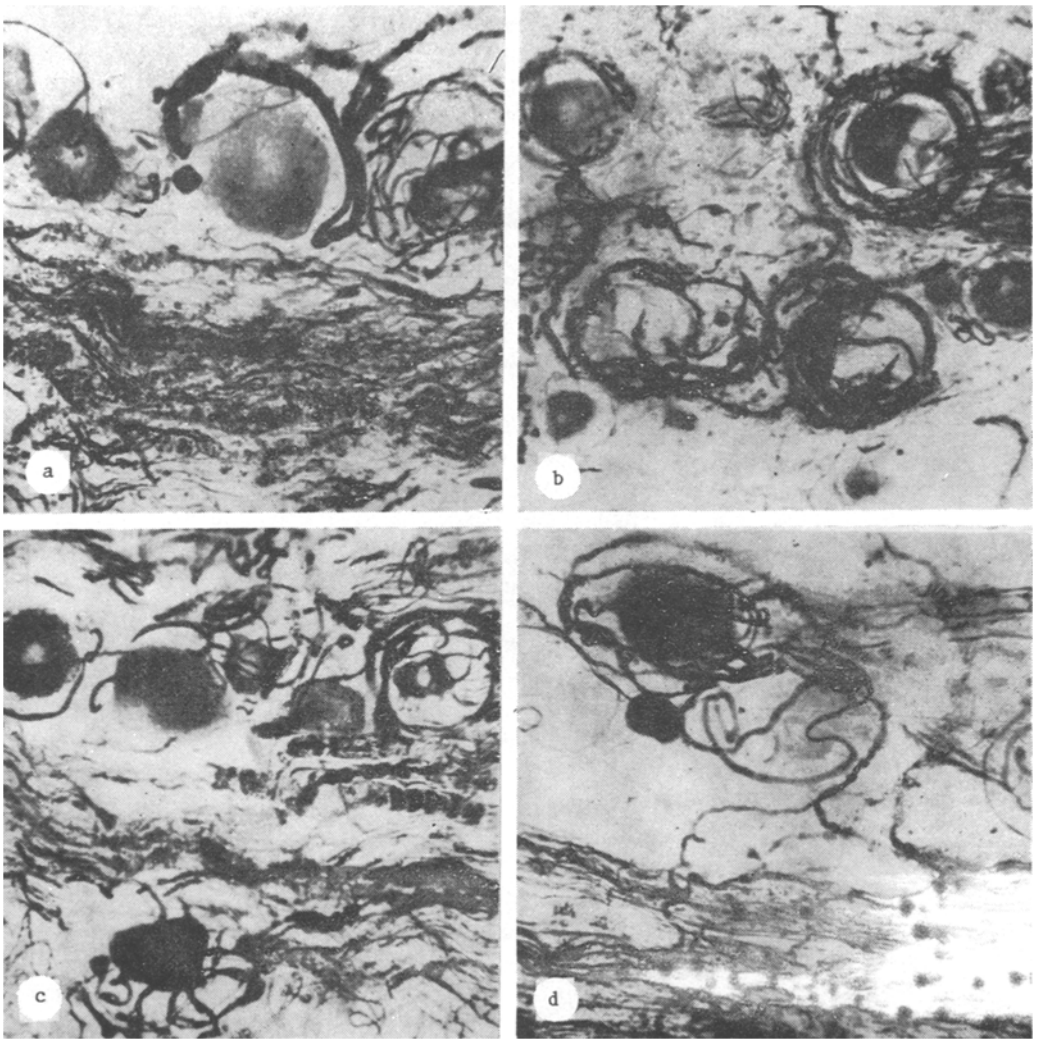

Fig. 1. Changes in ganglia nodosa at different stages of burn trauma. a) Swollen neuron weakly impregnated with silver (burn toxemia); b) atrophy of neuron bodies and "basket" spread of processes (burn septicotoxemia and emaciation); c) different stages of atrophy and dystrophy of neurons and cloudy-swelling degeneration of thick myelinated fibers inside the ganglion (burn septicotoxemia); d) "irritation spheres" near neurons (burn toxemia). Impregnation by Bielschowsky–Gros method. Magnification: a) 400 ×, b, c, d) 280 ×.

In some preparations accumulation of a yellowish brown pigment (lipofuscin) in the nerve cells was observed. As a rule this took place in neurons in a state of atrophy and shrinking, especially in patients who died in the stage of burn emaciation. Processes of the dying body of the nerve cells were in a state of compensatory hypertrophy, in the form of a widely looped coil, containing a large quantity of cytoplasmic substance. Atrophy of the bodies of sensory neurons was characteristic of the stages of burn septicotoxemia and emaciation.

During atrophy of the nerve cell bodies, the spreading processes of other neurons swelled and assumed irregular outlines (Fig. 1b).

In the stage of septicotoxemia atrophy of the nerve cell bodies developed with accumulation of edema fluid beneath the capsule; the processes of these cells underwent fragmentation and destruction. In some preparations different stages of necrobiosis of the bodies and processes of sensory neurons could be seen. In advanced cases of dystrophy the neurons died. In such cases fragmentation of myelinated fibers joining nerve bundles could be traced (Fig. 1c). Meanwhile varicose thickenings of preserved processes were noted, with evidence of compensatory hypertrophy in the form of "growth bulbs."

Not only nerve cells of the ganglia nodosa, but also their processes giving rise to thick afferent myelinated fibers were involved in the pathological process; both the myelin sheath and the axis cylinders in these fibers showed changes. In some places marked destruction of the spreading processes of the sensory neuron was visible, and was evidently associated with severe changes in adjacent regions of the ganglion. Sometimes a sensory glomerulus with coarse threads pressing on the capsule of the neurons could be clearly seen.

Dystrophic and necrobiotic changes in ganglionic nerve cells described above caused death of most of them. This state of affairs was reflected in the functional state of both nerve cells which were unchanged or only slightly changed. As a result of this, besides dystrophic and necrobiotic changes in the nerve cells in some cases structural changes were found in them, reflecting the adaptive response of the nervous system in pathological states. We know that the compensatory and adaptive changes in nerve cells include hypertrophy of their bodies, spreading out of their processes, and an increase in the number of nuclei [3, 4, 11]. Often many spherical homogeneous formations stained intense black with silver are found near nerve cells. Some of these spheres, located on myelinated nerve fibers, resemble "growth bulbs." The number and size of the "irritation spheres" described above are extremely variable. Near some nerve cells solitary "spheres" may be found, whereas near others, they may number as many as 4-6, and are mainly located within the capsules of the nerve cells (Fig. 1d). In some preparations spherical swelling of a process of a hypertrophied nerve cell compressed the capsule. Sometimes these hypertrophied knob-like nervous formations resembled synapses. The most frequent reactive phenomenon observed in nerve cells was excessive growth of their processes. In burn toxemia many neurons entangled in processes spreading out from them were seen. These changes gave the nerve cell the appearance of a "basket", as a result of which they have been called "basket cells" (Fig. 1b). In some preparations receptor apparatuses located between the nerve cells and in the pericapsular region could be observed in the stroma of the ganglia nodosa. Sometimes a spreading process formed multiple interweaving spirals near a nerve cell, lying in different planes, as a result of which each such process appeared as a curiously shaped coil, resembling a neuroma.

At different stages of burn trauma, besides changes in the bodies and processes of the neuron, nerve fibers traversing the ganglion nodosum and running in the trunk of the vagus nerve also were involved in the pathological process. Swelling, varicose thickenings, vacuolation, and cloudy swelling degeneration mainly of the thick and medium-sized myelinated nerve fibers, running at different distances from the nerve cell body, also could be observed (Fig. 1a, c). In burn trauma the thin unmyelinated nerve fibers as a rule suffer less than thick and medium-sized myelinated nerve fibers. In the later stages of burn trauma the receptor formation of the ganglia nodosa were also involved to a certain extent in the pathological process. Dystrophic changes were frequently observed both in incoming nerve fibers and in their terminal ramifications.

The changes in the structural elements of the ganglia nodosa described above at different stages of burn trauma are thus characterized by the diversity of their neuromorphological pictures. This is evidence that stimulation of sensory nerve endings in the organs during the development of the clinical course of burns is constantly changing. Optimal changes in the ganglia nodosa are probably of nervous reflex nature and are the result of long-term stimulation of the sensory nerve endings of the vagus nerves in the affected organs and tissue. The above statement is confirmed by certain morphological features of the structural changes in the sensory neurons of the ganglia nodosa at different stages of burn trauma. The first phenomenon to attract attention is that of restructuring of the bodies and processes of the neurons; the time course of these structural changes can be ascertained. Changes in the sensory neuron in the early stages of burn trauma commence mainly with its enhanced reactivity, as a result of which the body of the nerve cell undergoes hypertrophy. In burn septicotoxemia and emaciation, the bodies of the sensory neurons are particularly severely affected, they undergo distrophy and atrophy, and if advanced dystrophic changes are present the neurons die. This rule is not always seen to be obeyed, for in some cases the processes quickly die and swelling of the nerve cell body takes place, with subsequent conversions.

It can be postulated on the basis of this investigation that the ganglia nodosa of the vagus nerves play a very important role in the mechanism of the compensatory and adaptive function of the affected organs and tissues in burn trauma, on account of morphological restructuring of the residual neurons.

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