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ROLE OF THE VAGUS INJURY IN THE DEVELOPMENT OF THE CARDIAC ESOPHAGUS DYSFUNCTION

Abstract. *During the experiment with a dissected cervical part of the right vagosympathetic trunk on 23 adult cats, we actually simulated partial cardiospasm, a trigger of which, in our opinion, is the loss of preganglionic chain of parasympathetic reflex arch and an expressed autonomy of innervation as well as independence on the central nervous effects of the cardiac sphincter of the esophagus. The results must be considered when performing a surgery on the organs of the thoracic cavity with the aim of preventing disorders of motor activity of the esophagus (especially its abdominal region).*
Key words: *esophagus, neuron, myenteric plexus, ganglia.*

Introduction. The esophagus is known to belong to the organs that are under a significant influence of parasympathetic innervation - the vagus nerve [1, 2, 3].

Mechanical compression of the nerve, a trauma or its involvement in the inflammatory process is observed in many diseases of the chest cavity and mediastinum and cause significant changes in the esophagus [4], which are often accompanied by the cardiac sphincter spasm [3, 5, 6].

Based on the above, there is no doubt of the relevance and validity of studying the processes that occur in intramural esophageal interlacement in case of its innervation dissorders.

Objective: to study the changes in the structural elements of the esophageal myenteric plexus (EPMP) and their effects on the cardiac sphincter function after a high dissection of the vagosympathetic trunk.

Materials and methods. The experiment was performed on 23 adult cats of about the same age and weight, which underwent a partial denervation of the esophagus under an ether anesthesia in sterile conditions by dissecting the right vagosympathetic trunk in the cranial department. Time of the experiment was 1, 3, 7, 15, 30, 45, 60 and 90 days.

To study the nervous system and the esophageal bloodstream we used the following methods: 1) Bilshovsky- Gross impregnation of neural elements with silver nitrate; 2) injection with finely ground paints (Paris blue and black ink) into the esophageal bloodstream; 3) a combined detection of blood vessels and nerve elements of the esophagus with previous injection of blood vessels; 4) morphometric method and correlation analysis.

Results and discussions. On the 1^{st}-3^{rd} day after the vagus dissection the myenteric plexus neurocytes exhibit different tinctorial properties. Some of them become argentophil ones, others – argentophobic ones. It is difficult to identify nerofibrils in their cytoplasm, their nuclei become smaller or bigger, often being located eccentrically, on the move, or some varicose thickenings occur at the ends of the nerve cell processes.

Along with the above-described phenomena in some neurons of the cranial and middle esophagus there is often an acute swelling, and total chromatolysis and karyolysis, hydropic degeneration and vacuolization of cytoplasm, whose nucleus is difficult to distinguish . These changes lead to the death of a large number of neurons. For instance, in the cranial department of EPMP their number decreases by 1.99, in the middle part by 2.45 and in the cardiac department by 1,97 times. The nerve fibers take irregular contours, sometimes they become considerably thinner or thicker. Sometimes we observe a myelin fragmentation and a disintegration of axial cylinders.

The number of glial cells around each of the neurocites remains virtually unchanged at this term of the experiment, although the surface of

the neuron area, which accounts for one gliocyte, is slightly reduced and is in the cranial esophagus 181,5-243,9 microns², in the middle part - 169,4-240 7 microns² and in the cardiac one- 192,7-247,2 microns².

In the bloodstream of EPMP ganglia the changes only start after 3 days and result in uneven filling of blood vessels with injection masses, watering the bloodstream, increasing the size of capillary loops, a decrease in cross-sectional area of blood vessels, which accounts for a conventional unit of the area of EPMP ganglia. It should be noted that these changes in EPMP angioarchitectonics are mainly found in the cranial and middle esophagus, while its bloodstream cardiac segment remains more or less intact.

7-15 days after the operation in all parts of the EPMP there is a significant number of neurons, changes in which indicate the beginning of a degenerative process. Some neurocites impregnate intensively, their outlines are not clear or deformed, the nuclei are not seen, the processes only get coloured at the beginning of their branching or do not contrast at all. On the other hand some nerve cells are poorly impregnated, their nucleus shrinks, deforms, becomes hypo and hyperchromic, it is often shifted to the periphery of the cell. There are "shadow cells" when instead of a nerve cell we can see its residues with blurred contours formed by the products of granular lumpy disintegration of neurofibrils and lysis of nuclei.

The specific gravity of small neurons decreases in the cranial department by 1.33, in the middle one by 1.12 and in in the cardiac one by 1.07 times. The specific gravity of medium neurocytes increases, and large neurons in the cranial department disappear completely.

At this time of the experiment there is a significant proliferative response of glia. The number of glial cells in an environment of neurons reliably increased in all parts of the esophagus just around neurocytes with a transverse diameter of 16-20 microns and 21-25 microns. The surface area of neurons to one gliocyte decreases in all departments of EPMP more than in previous periods. Changes in the microvasculature of the nerve ganglia of EPMP of the cranial and middle esophagus amplified. They are complemented by plethora of the venous link of MCS. There are some disorders in the cardia: the bloodstream is poorly filled with injection masses, we observe the arteriolar tortuosity and gaps in capillary loops links, the venous part of the blood flow ways is slightly dilated. The percentage of small vessels with a diameter of 10 microns increases due to a decrease of vessels with larger caliber, but these changes in the cardia are minor. Due to this the cross-sectional area of blood vessels, which accounts for a conventional unit of ganglion area decreases by 2,00-2,15 times (in the cardia - only by1.18 times). The bloodstream capacity, which accounts for one neurocyte, decreases in the cranial and middle parts by1.78 and 1.72 times, respectively, but increases in the cardia by 1.59 times. The area of capillary-neuro-cellular contacts reduces.

This period of the experiment is characterized by a conical transition of the extended esophagus in the cranial and middle parts to the constricted one in the cardiac department, accompanied by dysfunction of the cardiac sphincter by cardiospasm pattern.

The process of degeneration of nerve cells and nerve fibers ends by 30 days. There are much fewer degenerative modified structures in ganglia and their decay products and glia proliferative processes continue. Adverse changes in the bloodstream of EPMP continue to grow, as evidenced by the sharp drop in the area of the capillary-neurocellular contacts in the cranial department by 1,37-1,84, in the middle part by1,43-2,08 and in the cardia by 1, 65-1,79 times. On the 45th day of the experiment degenerative changes in ganglia of EPMP stop, regenerative hypertrophy begins instead, continuing to 90 days. It is accompanied by the appearence of hypertrophied, uniformly impregnated neurons with distinct contours, a distinct nucleus and neurofibrils in the structure of ganglia. Processes of such neurons have a uniform thickness and form numerous collaterals.

At this time of the experiment the proliferative response of glial cells subsides and the number of glial cells around each of the neurocytes does not virtually differ from the norm. The index of the neuron area, which accounts for one gliocyte is not significantly different from the norm either.

On the 60-90th day of the experiment the microcirculatory bed of intramural nerve ganglia of the esophagus become normal too. Arterioles and venules that are around them, take their usual appearence. Their contours turn into smooth lines, there are not any deformations in

the vessel walls. The caliber of component parts of the venous link of MCS approaches to normal parameters. Morphometric data in this period also indicate a normalization of the blood supply of the components of EPMP and restoration of the capillary-neurocellular relationships.

Functional status of the cardiac sphincter comes to normal starting with the 45th day of the experiment and its complete recovery occurs on the 60-90th day.

Conclusions: 1. We believe that two points are essential in the pathogenesis of cardiac dysfunction of the esophagus: a) loss of preganglionic link of the parasympathetic reflex arc as a result of a damage to the right vagus nerve; b) expressed innervation autonomy and independence on the central nervous influences of the cardiac esophagus, which is confirmed by morphometry in our studies.

2. The distinctive conical transition from the dilated part of the esophagus to the narrowed one is due to the gradual transition from the area with transneurona ganglia denervation of EPMP (departments dependent on the central nervous influences) to the areas where the Dogel type I neurocytes remain due to the autonomous innervation of the department.

Prospects for further research. The etiology of primary disorders of the esophageal motility and in particular, which is the primary factor in the vicious circle of gastroesophageal reflux remains unclear. According to Edwards they are due to the damage of postganglionic neurocytes [8]. We think that lesions in EPMP preganglionic nerve fibers cause them. The question remains arguable and requires more detailed research.

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