

# Pathological Changes in Cardiospasm

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Anatomicopathological changes of the esophagus and stomach at different stages of cardiospasm are shown. As the disease develops, macroscopic changes of the esophagus, manifesting themselves in its enlargement, and microscopic changes - perivascular lymphocyte and plasmacyte infiltration and muscle fiber hypertrophy and edema - are noted. During the development of cardiospasm inflammatory-degenerative changes progress in all esophageal layers.

**Key Words:** *esophagus; cardiospasm; extirpation*

Cardiospasm (CS), characterized by a triad of major symptoms (dysphagia, substernal pain, and regurgitation), is a severe form of benign injury of the esophagus (EP). According to clinical data from 1980 to 1993, CS ranks second (26.1%) among different stricture injuries of the EP (46.6%). The incidence of esophageal tumors of different localization constituted 11.2% during this period. Few studies of CS or achalasia have been published in Russia and the West. Two major types of pathological changes have been revealed in achalasia of the cardia [2,4].

The first type is characterized by a narrowing in the distal portion of the EP, sometimes along a considerable length, and a spindle-shaped or cylindrical dilation of its ascending portions. The EP is not elongated or crooked. A macroscopic study of the anatomical parameters of the cardia does not show any marked changes. The supracardial segment of the EP is narrowed. Its muscle lining is compact and, frequently, thickened.

The second type of changes of the EP is characterized by an abrupt narrowing in its distal portion and a marked widening in its ascending portions. Such a phenomenon is termed megaesophagus, or idiopathic esophageal dilation. The EP takes the

form of a bag, and sometimes, due to its elongation, becomes S-shaped. The volume of the EP markedly increases, and it can hold more than 2 liters of liquid, instead of the normal 50-150 ml. As a rule, when the patient is on an empty stomach, the EP contains liquid and food residues. Macroscopic examination shows a markedly narrowed, as if atrophied, supracardial segment of the EP and an enormous ascending portion emerging from the mediastinum. The muscle lining in the narrowed portion of the EP is thin and does not exhibit a clearly pronounced muscular structure; on the other hand, in some cases it is thickened.

In types 1 and 2 achalasia manifestations of chronic inflammation of varying severity, sometimes, including sclerosis of the submucous layer, muscle dystrophy, and, frequently, muscle atrophy and scarring, are observed. Muscle hypertrophy is noted in places.

In types 1 and 2 achalasia esophagitis develops as a result of food congestion and decay [2]. In some cases erosions and foci of leukoplakia are observed in the mucous membrane. Cells of the mediastinum may be implicated in the inflammatory process; periesophagitis develops.

## MATERIALS AND METHODS

Material obtained at the Research Center of Surgery of the Russian Academy of Medical Sciences

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during transthoracic and transabdominal surgery in different diseases of the organs of the thoracic cavity, mediastinum, and diaphragm enabled us to compare the anatomical parameters of the EP, cardia, and surrounding structures in CS and other diseases. CS was studied for several years, and macro- and microscopic investigations of the EP, cardia, fundal portion of the stomach, diaphragm, and mediastinal pleura were performed in clinics using material obtained during surgery and endoscopic biopsy.

The data presented here were obtained by analyzing the results of investigations of the EP and stomach of 18 patients undergoing extirpation of the EP and one-stage gastropasty. The operations were scheduled, only one of them being an emergency (when the EP was ruptured during cardi-odilation).

It should be mentioned that scarring of the mediastinum, which to some extent is always observed, markedly complicates isolation of the EP. In some cases, due to previously developed mediastinitis, the scarred EP and the surrounding organs (trachea, bronchi, pleura, and pericardium) formed one conglomerated mass, from which the EP had to be dissected with scissors [3].

The material for histopathological investigation was obtained from patients with stage IV CS [1] who had undergone surgery on the terminal portion of the EP and cardia, and a female patient was diagnosed with esophageal rupture stage II-III CS.

## RESULTS

Anatomicopathological changes in CS were polymorphous and, according to our data, depended on the stage of the disease. It was possible to identify these stages from the clinical-instrumental standpoint; pathomorphologically, there were transition stages. In stage I of the disease there were no macroscopic changes, and microscopic changes were borderline. As the disease developed, microscopic changes appeared, while abnormal macroscopic changes were not detectable.

In the initial period of stage II, macroscopic narrowings of the cardia and a widening of the EP up to 3-4 cm were noted. Microscopic investigations demonstrated a slight dilation of the vessels, perivascular lymphocyte and plasmacyte infiltration, and muscle fiber hypertrophy and edema. The submucous and muscular layers were 2-3-fold thickened due to hypertrophy and lymphoid infiltration of the circular layer. Cell dystrophy, fiber degeneration and fraying, and destruction of the ganglia and of some nerve trunks were observed in

the intramural nerve plexuses. In stage III of the disease the cardia was markedly narrowed, and the lumen of the EP was dilated by more than 5 cm. Different shapes and distortions of the EP promoted mechanical occlusion of the vessels, which in turn aggravated sclerotic changes in tissues. The mucosa was thickened, edematous, and hyperemic. The submucous and circular muscular layers of the EP were sclerotized and thinned, their lability being impaired. Stricture of the nervous system was also altered. Perineural sclerosis, fibrosis, reduction and disappearance of cells in the ganglia, and a decline in the number of nerve cells in the ganglia (degeneration also resulting from myogenic dilation) were revealed. Cell dystrophy was noted in all anatomical systems. In stage III all soft tissues surrounding the EP (the mediastinal pleura, the mediastinal tissue, and the diaphragm) were changed. Surgery revealed thickening of the mediastinal pleura and its adnation with the walls of the EP, periesophageal sclerosis, mediastinitis, and sclerosis around the esophageal port of the diaphragm.

In stage IV the supracardial portion was greatly narrowed, while the EP was dilated. However, the narrowed portion was usually shorter than during stage III. Its capacity was as large as 3 liters. The EP was elongated and S-shaped. In all cases manifest esophagitis was observed, and various erosions, ulcers, and esophageal strictures were macroscopically evident. Histological investigation of the ulcers demonstrated granulation tissue and a necrotized layer at their bottom. Microscopic study showed atrophy and dystrophy of the inferior esophageal sphincter, sclerosis of the intradiaphragmal and abdominal segments of the EP, and disturbed trophics and innervation, sometimes to the point where the ganglia were absent altogether.

The layers of the wall of the EP at the incision did not exhibit a clear-cut anatomical differentiation, and pronounced sclerosis of the vascular walls was observed.

It may be assumed that the severity of morphological changes of the EP depends on the stage of the disease. It was noted that morphological changes in CS had no marker and were characteristic of nonspecific inflammation.

Pathological changes of the cardia and of the lesser curvature of the stomach were less pronounced than those of the EP. Histopathological investigation demonstrated morphological changes typical of associated diseases (more often of chronic gastritis).

It should be mentioned that in stage III and IV CS there was lymphocyte infiltration of the

mucous and submucous layers with sclerotic manifestations. We also noted that the lymph nodes became enlarged to 1.5 cm, exhibiting a picture of reactive lymphadenitis. Stromal edema and plethora of the muscular and outer layers of the stomach were also observed.

Anatomicopathological investigation of the cardia and of the lesser curvature of the stomach demonstrated a nonspecific inflammatory reaction in the form of reactive lymphadenitis.

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## METHODS

# The Multimioestim-04, a Portable Compact Device for Electroneurostimulation and Electropuncture

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The Multimioestim-04, a multichannel electrostimulator, has been developed. Its advantage lies in its powerful effect on the neuromuscular structures and neurons around the focus of injury, due to an enhancement of the impulse flow from several muscle groups of the injured extremities. The afferent effect on neurons in the zone where the synaptic contacts have been lost is also boosted by electrostimulation of the symmetrical muscles of the healthy extremities.

**Key Words:** *electroneurostimulation; electropuncture; neuromuscular system; treatment of locomotor apparatus*

The Multimioestim-04 device, designed to improve the functional state of the organism and to treat diseases of the locomotor apparatus, is intended to be used for electrostimulation (ES) of the human neuromuscular system and for electrical treatment via acupuncture points. The device was developed on the basis of earlier prototypes [1,2].

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A block-diagram of the device is shown in Fig. 1. The electronic assembly consists of two plates connected by permanent connections to the control unit and power unit. The numbering of the amplitude regulators corresponds to the numbers of the regulated channels. The signal attenuator, which reduces the signal to the level of the ES signal for electropuncture (EP), is installed in the power-unit assembly.

The main units of the device are a master oscillator, a unit producing the stimulating pulses,