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ANALYSIS OF ESOPHAGEAL STITCHES EFFICIENCY WITH BOERHAAVE SYNDROME (EXPERIMENTAL STUDY)

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Abstract:

The experimental study was conducted in two stages: at the first stage modeling of Boerhaave syndrome was performed, at the second stage – examination of strength of the stitches applied to the esophagus and histologic examination of the esophageal wall at various times after its rupture. The experiment was carried out using three-month-old male rats with the weight of 265-285 grams as the study material.

Original devices were developed for modeling of Boerhaave syndrome and esophagus stitch strength determination. The data obtained showed that after the esophagus wall rupture in the esophagus wall and surrounding tissues and chest organs, the inflammatory phenomena and bacterial load increase synchronously and progressively with the increase of the time interval that has passed since the moment of rupture. Which explains esophagus, lungs, and trachea inflammatory response and destruction. This proves experimentally that the main reason for the frequent failure of stitches among patients with the Boerhaave syndrome is the increase in infiltration and destructive changes in the esophageal wall, pleura and adjacent organs.

Keywords: *Boerhaave syndrome, experiment, modeling, surgery.*

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INTRODUCTION:

The first description of the condition at which a spontaneous rupture of all layers of the esophagus wall takes place was given by a Dutch physician Hermann Boerhaave in 1724 [1,2,3]. Spontaneous esophageal ruptures (SER) account for 2-3% of all cases of esophageal injury and it is more likely to occur among men over 50. Boerhaave syndrome occurs at intense vomiting after a heavy meal, water and alcohol intake, the vomiting of the central genesis (due to the vomiting center disfunction), increased intragastric and intraesophageal pressure due to weight lifting. Those result in a sudden increase of pressure inside the esophagus with a closed pharyngeal esophageal sphincter in combination with negative intrathoracic pressure, which is the direct cause of esophagus rupture. In 90% of cases the esophagus ruptures are localized in the lower third of the esophagus. Despite the advances in oesophageal surgery, insufficiency of stitches applied during the spontaneous rupture of the esophagus occurs in 30% to 90% of patients [4,5,6,7,8]. The high risk of esophagus stitches failures in case of Boerhaave syndrome has become the basis for further study of this topic and the development of experimental methods for studying the causes of the esophagus stitches failure.

Purpose: Development in the experiment of new methods of surgical treatment of patients with Boerhaave syndrome.

MATERIAL AND METHODS:

Three-month-old male rats weighting 265-285 grams were used as the study material. All experiments were carried out in accordance with the European Convention on the protection of vertebrate animals rights used for experimental or other scientific purposes (ETS No. 123 issued on 18.03.1986).

The experimental study was conducted in two stages: at the first stage modeling of Boerhaave syndrome was performed, at the second stage – examination of strength of the stitches applied to the esophagus and histologic examination of the esophageal wall at various times after its rupture.

An original device for Boerhaave syndrome modeling was developed to perform the experiment (Fig. 1) [2,9,10].

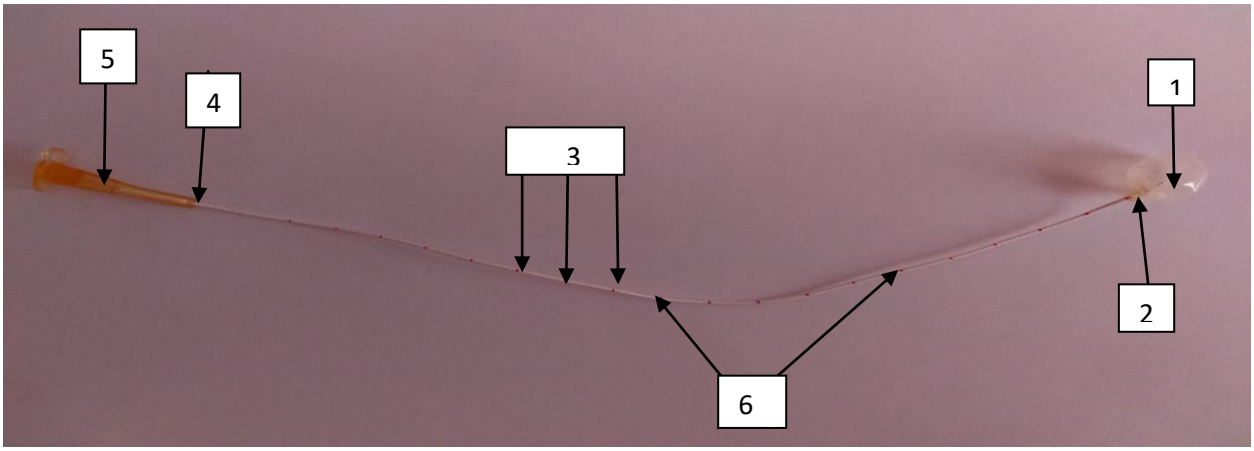

Fig. 1: Boerhaave syndrome modeling device (1. vessel, 2. connection point of the tube with the vessel, 3. marking, 4. connection point of the tube to the connector, 5 connector, 6. tube).

The device consists of the catheter of original design (Fig. 1), represented by a tube with the total length of 200 mm and diameter of 1 mm with marking (10 mm interval). At the distal end of the catheter there is a vessel of cylindrical shape, with the volume of 4.2 ml/cm3, wall thickness 0.07 mm, length and the width of 20 mm, made of natural high-quality latex, which connects with the tube lumen. The catheter tube is connected to the syringe by the connector.

The original device for Boerhaave syndrome modeling works as follows: the catheter is guided into an esophagus so that a vessel is located in the lower third of the esophagus, after which air is injected into the catheter by the syringe with a volume of about 4-5 ml. When the air is pressurized up to the specified volume, the esophagus ruptures, accompanied by a specific sound resembling a balloon explosion.

The first stage of the study. The experiment was performed under inhalation anesthesia (Isoflurane). After the animal was anesthetized, the depth of

introduction of the special catheter of original design into the esophagus for the esophagus rupture was calculated by measuring the distance from the larynx to the xiphoid process using a ruler. The catheter with the vessel was introduced in the esophagus through the animal's mouth so, that the vessel to be located in the lower third of the esophagus (according to the catheter markers, 5 - 6 cm approximately). Then the catheter was fixed by the clamp so that it would not migrate during a vessel inflation. Then a medical syringe (10 ml) was connected to the catheter connector and air was injected into it. The volume of the collected air made 4.2 ml on the average. Thus, a faint sound was heard resembling the sound of balloon explosion. After that, the collected air was removed by the syringe.

The animal's chest opening was performed to ascertain the rupture of the esophagus (Fig. 2).

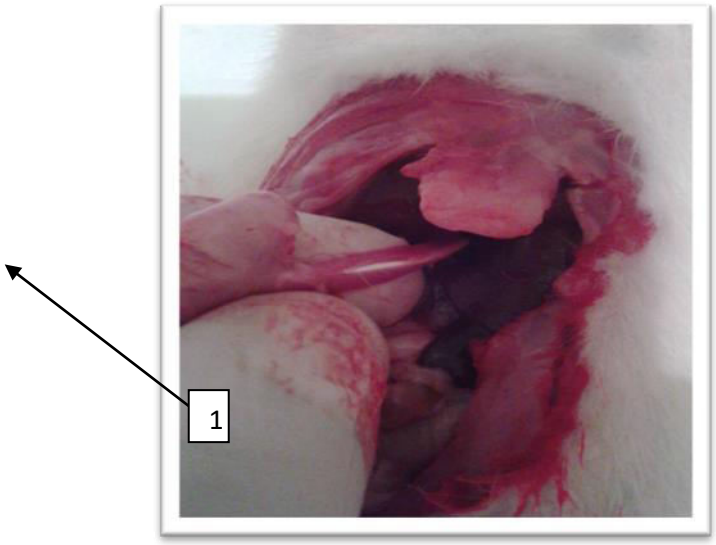

Fig. 2: Boerhaave syndrome modeling among rats (1. Esophageal rupture).

The second stage of the study was performed as follows. After modeling of the Boerhaave syndrome, the ruptured esophagus was sutured with nodular sutures with a gap of 2 mm and 1 mm indentation from the rupture edge with atraumatic sutures (Fig. 3).

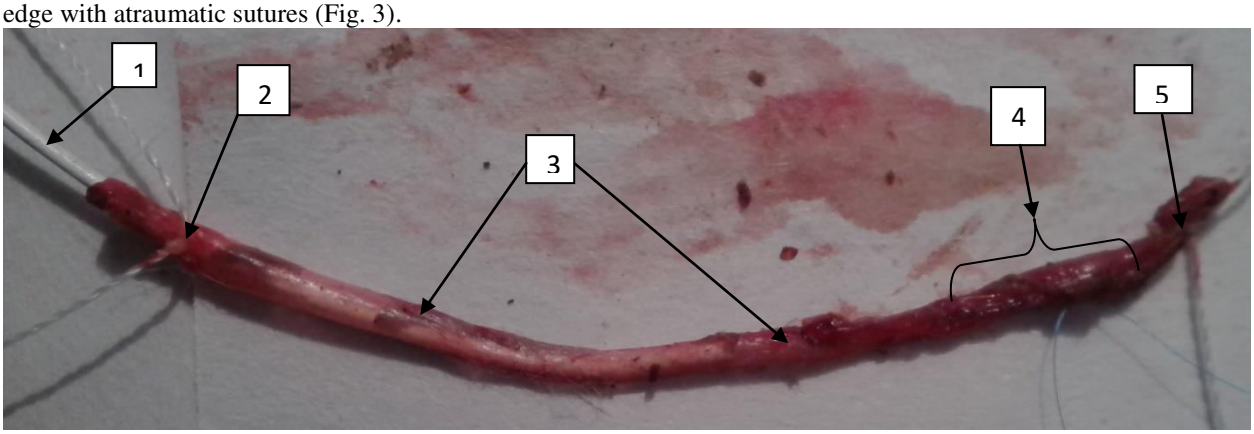

Fig. 3: Suturing of the esophagus (1. catheter, 2. ligated proximal esophagus department, 3. esophagus, 4. sutured part of esophagus, 5. ligature-stranded distal esophagus department).

For determination of the stitches strength we developed an original device as well (Fig. 4) [2,9,10].

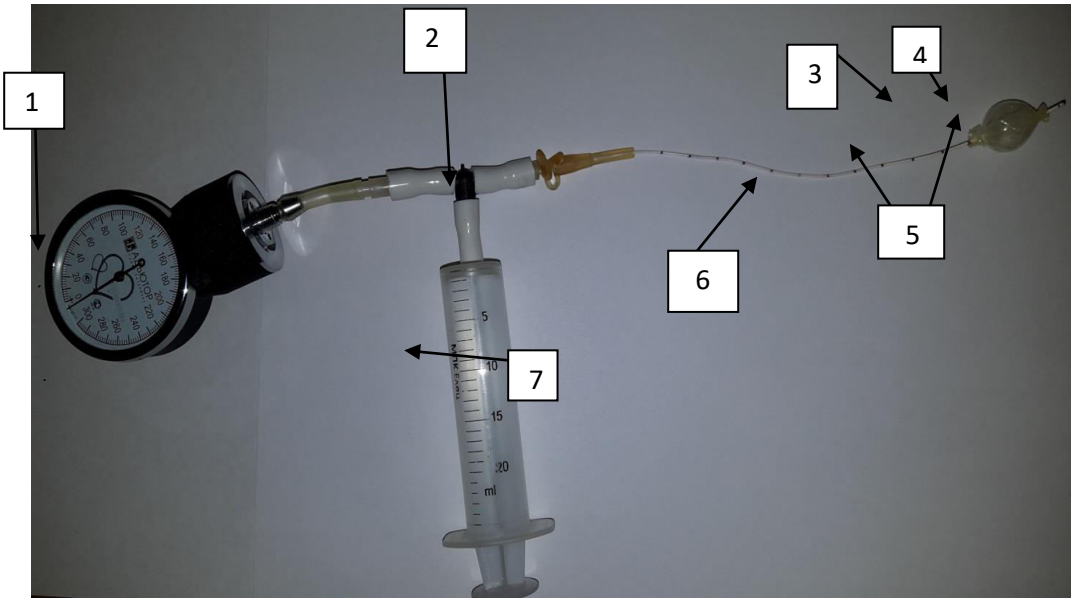

Fig. 4. The device which determines the strength of the esophagus stitches (1. manometer, 2. tee, 3. vessel, 4. distal catheter section, 5. tube joint with the vessel, 6. catheter, 7. syringe).

The measurement of the stiches strength was carried out on days 1,2,3 as follows: the esophagus was removed from the deceased animal, the catheter of the original device was inserted into it to determine the strength of the stitches (Fig. 4) so that the vessel was on the esophagus at the level of the applied stitches. The distal and the proximal parts of the esophagus were fixed to the catheter by means of ligatures. Air was injected in the catheter using the syringe, thereby inflating the vessel. During the rupture of the esophagus stitches, the pressure was fixed on the manometer. After the rupture of the esophagus stitches, the air was evacuated from the vessel with a syringe, and the device was removed from the esophagus, the samples were taken for histological and bacteriological examination. The sowing from the pleural cavity was performed for bacterial study.

OBTAINED RESULTS:

Studying of the lethality of experimental animals after modeling the Boerhaave syndrome showed that the majority of animals (54%) died on the first day after the esophagus rupture (Table 1).

Table 1: Lethality of experimental animals (rats) after Boerhaave syndrome modeling

Term	Abs. (%) during the first day	Abs. (%) during the second day	Abs. (%) during the third day
Absolute amount (%)	54 rats (54%)	37 rats (37%)	9 rats (9%)

The measurement of pressure by the manometer during the rupture of esophagus stitches showed the decrease of the esophagus stitch strength on the third day after the modeling Boerhaave syndrome (Table 2).

Table 2:Measurement of pressure by manometer (mm Hg)

Pressure (mm Hg)	First day	Second day	Third day
	180 – 160 mm Hg.	159 – 140 mm Hg	139 – 120 mm Hg
Average pressure	170 mm Hg	149.5 mm Hg	129.5 mm Hg

The bacteriological study data of the pleural cavity of experimental animals showed bacterial contamination increase of the mediastinum and pleural cavities with an increase in the time interval that has passed from the moment of the esophagus rupture (Table 3).

Table 3:Bacteriogram from the pleural cavity of experimental animals, which died at different periods of the experiment

Time	Number of animals	Pathogenes
On the first day	21	Proteus vulgaris 10*3
	33	Proteus vulgaris 10*3 Enterococcus spp 10*4.
On the second day	10	Proteus vulgaris 10*3 Staphylococcus epidermidis 10*4.
	12	Proteus vulgaris10*3 Enterococcus spp10*3, Staphylococcus epidermidis10*5.
	15	Proteus vulgaris 10*4 Enterococcus spp 10*5, Staphylococcus epidermidis 10*5, Escherichia coli 10*4.
On the third day	3	Proteus vulgaris 10*4 Staphylococcus epidermidis 10*6, Enterococcus spp 10*6
	6	Proteus vulgaris 10*5 Staphylococcus epidermidis 10*5, Enterococcus spp 10*8, Escherichia coli 10*8.

Results of histological examination

1. Histological examination of animals that died during the first day represented on Figures 5,6,7,8.

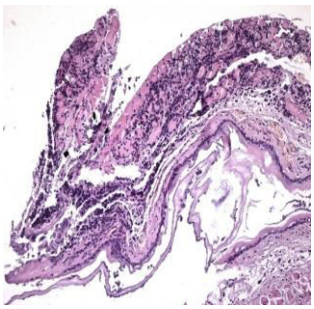

Fig.5: The esophagus (increase x10, Hematoxylin and eosin stain)

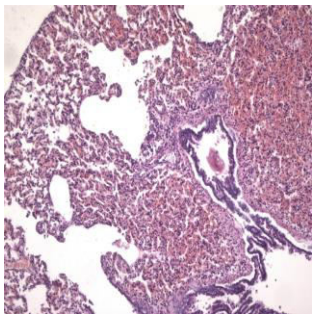

Fig.6: The lungs (increase x10, Hematoxylin and eosin stain).

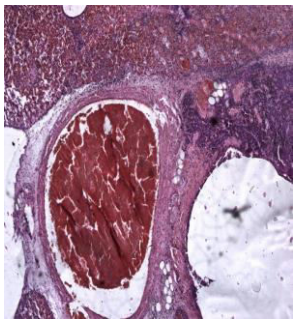

Fig.7: The trachea (increase x5, Hematoxylin and eosin stain)

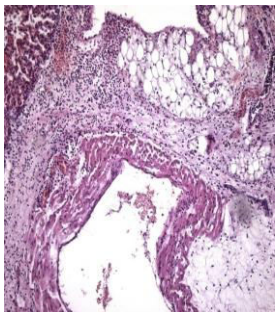

Fig.8: The mediastinum (increase x10, Hematoxylin and eosin stain)

Fig. 5 Preparation: ruptured esophagus. Conclusion: The oedematic wall, pronounced plethora, diffuse inflammatory infiltration of the esophagus muscles, infiltration of the esophagus outer wall, penetrates into the cellulose, some vessels are injured, focal hemorrhages in a rupture zone. The development of abscess in the wall.

Fig. 6. Preparation: lungs. Conclusion: Extensive hemorrhages, the areas of distelectases and emphysema with the rupture of interalveolar septa and the development of foci, bronchospasm, blood overflow in

microcirculatory bed with dilelectases and atelectasis, shock lungs.

Fig. 7. Preparation: trachea. Conclusion: Inflammatory infiltrate penetrates into the trachea wall, breaks through the lumen of the trachea along the intercartilaginous spaces.

Fig. 8. Preparation: mediastinum. Conclusion: diffuse inflammatory infiltration, decay of leukocytes.

2. Histological examinations of the animals that died during the second day are performed on Figures 9,10,11,12.

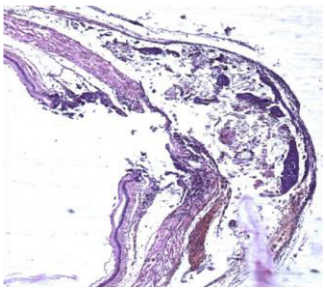

Fig. 9: The esophagus (increase x5, Hematoxylin and eosin stain)

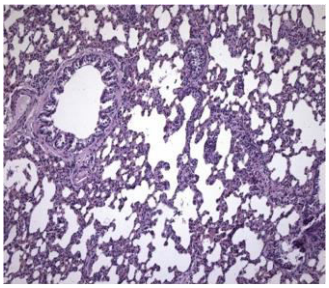

Fig. 10: The lungs (increase x10, Hematoxylin and eosin stain)

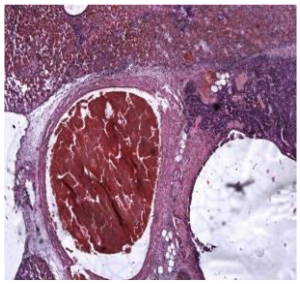

Fig.11: The trachea (increase x5, Hematoxylin and

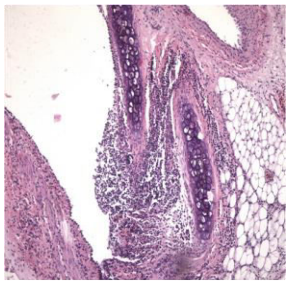

Fig.12: The mediastinum (increase x10, Hematoxylin and eosin stain)

Fig. 9. Preparation: ruptured esophagus. Conclusion: Extensive hemorrhages, venous thrombosis, non-focal spot diffuse infiltration, edematous wall, pronounced plethora, diffuse inflammatory infiltration of the esophagus muscles, infiltration of the esophagus outer wall, penetrating into the cellulose. Some vessels are injured. Focal hemorrhages in the rupture zone. The development of abscess in the wall.

Fig. 10. Preparation: lung. Conclusion: Extensive hemorrhages persist, the areas of distelectases and emphysema with the rupture of interalveolar septa and the development of foci, bronchospasm, microcirculatory bed blood overflow with dilelectases and atelectasis, shock lungs.

Fig. 11. Preparation: trachea. Conclusion: The inflammatory infiltrate persists, penetrates into the trachea wall, breaks through the lumen of the trachea along intercartilaginous spaces.

Fig. 12. Preparation: mediastinum. Conclusion: Diffuse inflammatory infiltration persists, decay of leukocytes.

3. Histological examinations of animals that died during the third day are shown on Figures 13, 14, 15, 16.

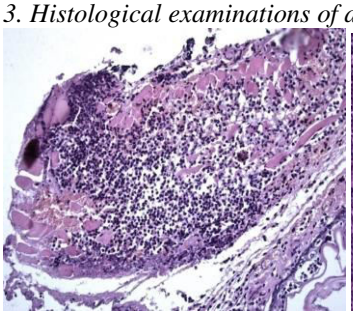

Fig. 13: The esophagus (increase x20, Hematoxylin and eosin stain)

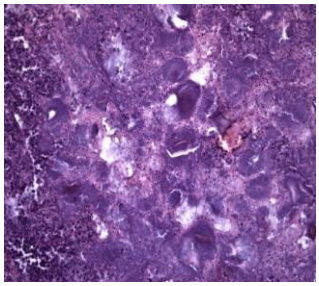

Fig. 14: The lungs (increase x20, Hematoxylin and eosin stain)

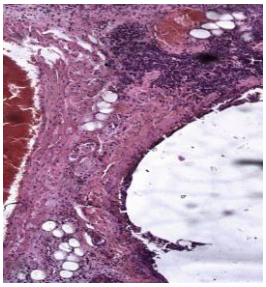

Fig.15: The trachea (increase x5, Hematoxylin and eosin stain)

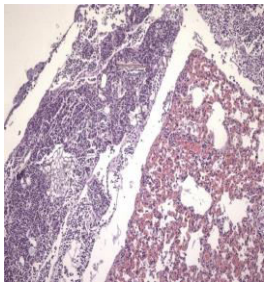

Fig. 16: The mediastinum (increase x10, Hematoxylin and eosin stain)

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Fig. 14. Preparation: lung. Conclusion: Bronchospas... persists, there are some branches of the pulmonary vein, a pronounced emphysema with intermalveolar septal ruptures, interstitial

pneumonia.

Fig. 15. Preparation: trachea. Conclusion: the inflammatory infiltrate penetrating into a trachea wall, breaking out in the lumen of a trachea along intercartilaginous spaces persists.

Fig. 16. Preparation: mediastinum. Conclusion: Diffuse inflammatory infiltration persists, leukocyte decay.

SUMMARY:

After the rupture of the esophagus the inflammatory phenomena in esophagus wall, destructive processes in organs surrounding the esophagus increase progressively and synchronously with the increase in the time interval that has passed since the moment of rupture. The increase of bacterial contamination of the mediastinum and pleural cavities explains the increase of the inflammatory response and the destruction of the esophagus, lungs, and trachea, as through the esophagus rupture edible masses enter the mediastinal fiber. Which in its turn explains the titer increase of the microflora that causes the inflammatory response of the mediastinum and adjacent organs.

CONCLUSIONS:

1. The increase in the time interval that has passed since the moment of esophagus rupture is associated with inflammatory phenomena and destructive mediastinum and adjacent organs, which cause death of the animals. The bacterial colonization of the pleural cavity at rupture of the esophagus occurs from mono culture during the first day, in the next 24 hours titer of microorganism increases and polyflora appears.

2. The strength of the stitches applied on the esophagus rupture line decreases inversely with the time elapsed since the rupture.

3. The increase of infiltration and destructive changes in the esophagus wall, pleura and adjacent organs is the main reason for the frequent failure of stitches.

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