"Tolerance and apathy,

these are the last two virtues of a dying society."

Aristotle

General patterns of the motor function of the sphincters of the digestive system. Comparison of the function of the gastroduodenal junction with the esophagogastric junction.

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Abbreviations

AS- antral sphincter; CD - crural diaphragm; GERD – gastroesophageal reflux disease; GDJ – gastroduodenal junction, EGJ - esophagogastric junction; LES – lower esophageal sphincter; PS - proximal sphincter; PyS – pyloric sphincter; HRM – high-resolution manometry; PD - pneumatic dilation, POEM – peroral endoscopic myotomy; LHM - laparoscopic heller myotomy.

The physiology of the digestive tract owes much to research in animal models of various mammals, as it is assumed that, because of artificial selection, all mammals have received the most functional digestive system like that of humans. In any case, the law of the intestine, which determines the pattern of peristalsis, is equally true for all mammals. The law of the intestine by Bayliss and Starling - "Excitation at any point of the gut excites contraction above, inhibition below". Cannon later proposed the term "myenteric reflex" for the phenomenon because it is due to the activity of the intramural nervous system [1].

A study of the basic patterns of the functioning of the digestive tract shows that natural selection is limited to the best option. This suggests that all sphincter zones of the digestive tract function similarly. Based on this, I suggested that knowing the mechanisms of functioning of any sphincter, it is possible to extrapolate these mechanisms to the function of another less studied one.

The purpose of this study is to compare the patterns of motor function of the gastroduodenal junction (GDJ) and the esophagogastric junction (EGJ).

1. Gastroduodenal junction motility

From a functional point of view, the stomach is divided into proximal and distal parts. The proximal part, including the fundus and the body of the stomach, provides reception and temporary storage of food. It regulates intragastric pressure and stimulates the tonic advancement of the chyme into the distal part. In addition, it provides space and time for pepsin and hydrochloric acid to act in the early stages of digestion. The fundus is characterized by tonic rather than peristaltic contraction. An important function of the proximal stomach is its ability to accommodate. More than a liter of food can enter the stomach without increasing intragastric pressure [2,3]. A study of pressure on volunteers showed that intragastric pressure decreases moderately after eating and returns to the initial level as soon as solid food ingredients penetrate the duodenum [4]. The motor function of the proximal stomach is regulated by reflexes: receptive relaxation and gastric accommodation. Receptive relaxation is manifested by a decrease in the tone of the proximal stomach during swallowing. For example, Shafik's study showed that «Pharyngeal distension produced a significant pressure drop of the corpus of the stomach ($p < 0.05$); the pyloric antrum shows no response. Upper, middle, or lower esophageal distension produced gastric response similar to that evoked by pharyngeal distension" [5]. Stomach accommodation is described as a relaxation reflex of the proximal stomach in response to distension. Unlike receptive relaxation, this reflex does not depend on the stimulation of the esophagus and pharynx.

In the distal part of the stomach, a negatively charged membrane potential is determined, on which rhythmic depolarization at three cycles per minute is superimposed. The frequency and direction of electrical activity are closely related to the slow peristaltic wave. There is evidence that the slow-wave is generated by the phase depolarization of Cajal interstitial cells. These cells are located on the greater curvature of the stomach. The slow-wave propagates a little faster along the greater curvature so that the myoelectric activity "running" along the greater and lesser curvatures of the stomach reaches the pylorus simultaneously. Slow waves with three cycles per minute are observed both at rest and in the phase of active gastric motility. Neurohumoral activators increase the amplitude of the slow wave, and this contributes to the crushing of food into finely dispersed.

Shafik et al showed that "gastric balloon filling with more than 20 ml of H2O showed progressively increasing LES pressure up to 110-120 ml, beyond which the pressure exhibited no further increase upon incrementally increased gastric filling volume" [6]. The distension of the proximal stomach produced no pressure

changes in the proximal stomach, pyloric antrum, or sphincter ($p>0.05$). Antral distension affected a significant rise in antral pressure, but not in the proximal stomach. A significant (pyloric) sphincter pressure decrease occurred only with antral distension volumes >50 ml [7]. Pyloric sphincter contraction and antral dilatation upon duodenal distension suggest a reflex relation [8].

The results of X-ray studies are completely consistent with the manometric data (**Figure 1**). Since antral distension causes a significant rise in antral pressure, but not in the proximal stomach, which means that two cavities with different pressures arise in the stomach. X-ray studies show that in a horizontal position every 3-5th peristaltic wave closes in the antrum and detaches a part of the contrast agent to form a closed cavity. This closed cavity is called the antral cavity, and the functional sphincter that occludes the gastric lumen above it is called the antral sphincter (AS). Continuing to contract antral systole causes a rise in pressure to a threshold level, which leads to the reflex opening of the pyloric sphincter (PyS) and injection of the bolus into the duodenum [9,10].

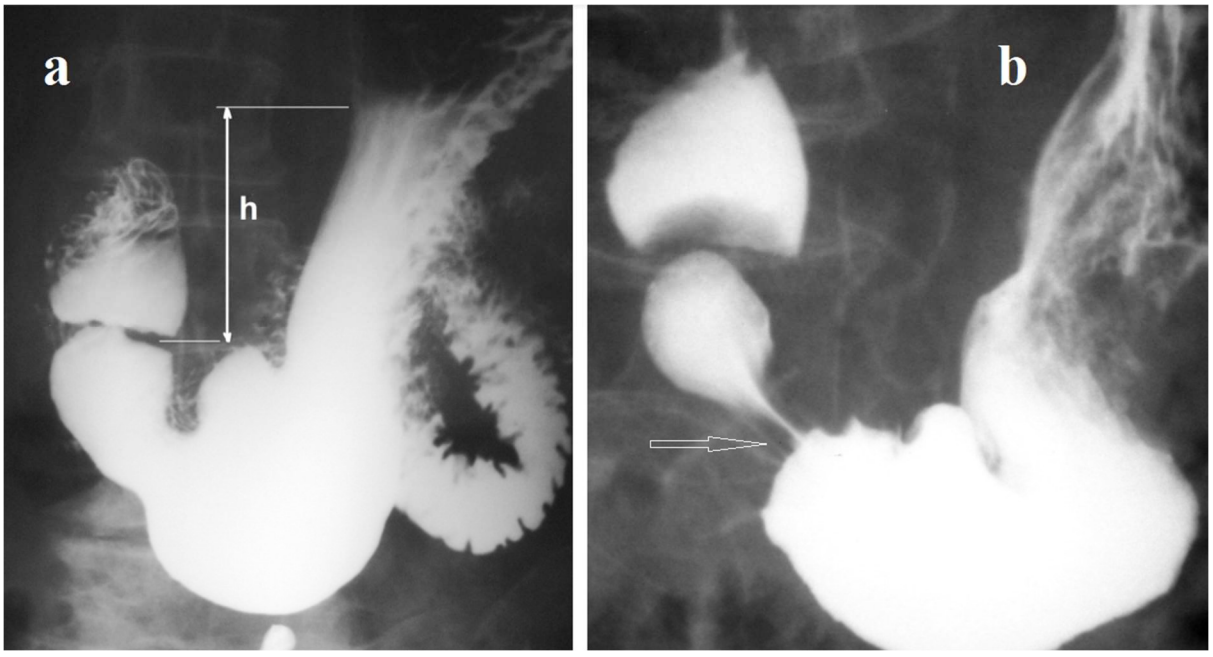

Figure 1. Two options for evacuation from the stomach. (a) In the upright position, the hydrostatic pressure of the liquid column (h) between the liquid level in the proximal part of the stomach and the pyloric sphincter creates a threshold pressure for the reflex opening of the PyS and filling of the duodenal bulb. (b) At low hydrostatic pressure in the stomach, the threshold pressure for PyS opening is created by the contraction of AS (arrow), which leads to the formation of an antral cavity and injection of a bolus into the duodenal bulb.

The reflex contraction of PyS and antral dilation upon duodenal distension is of great importance for the normal function of the stomach and duodenum. Thanks to this reflex, the bolus is evacuated in portions, the volume of which is equal to the capacity of the duodenal

bulb. The closing of the PyS occurs when the pressure in the bulbous rises to a threshold level.

This occurs because of the contraction of the post bulbar sphincter in response to its irritation with hydrochloric acid (Figure 2). Thus, as soon as the bulb is filled with food, the post bulbar sphincter contracts. The pressure in the bulbous rises, which leads to a contraction of PyS and the termination of evacuation.

Only every 3-5th peristaltic wave ends with the formation of the antral cavity and the release of a bolus into the duodenum. The rest of the waves do not close, and during the contraction of the antrum, its contents are thrown retrogradely into the body of the stomach, mixing the gastric contents. At this moment, PyS is in a closed state (see Figure 2. a).

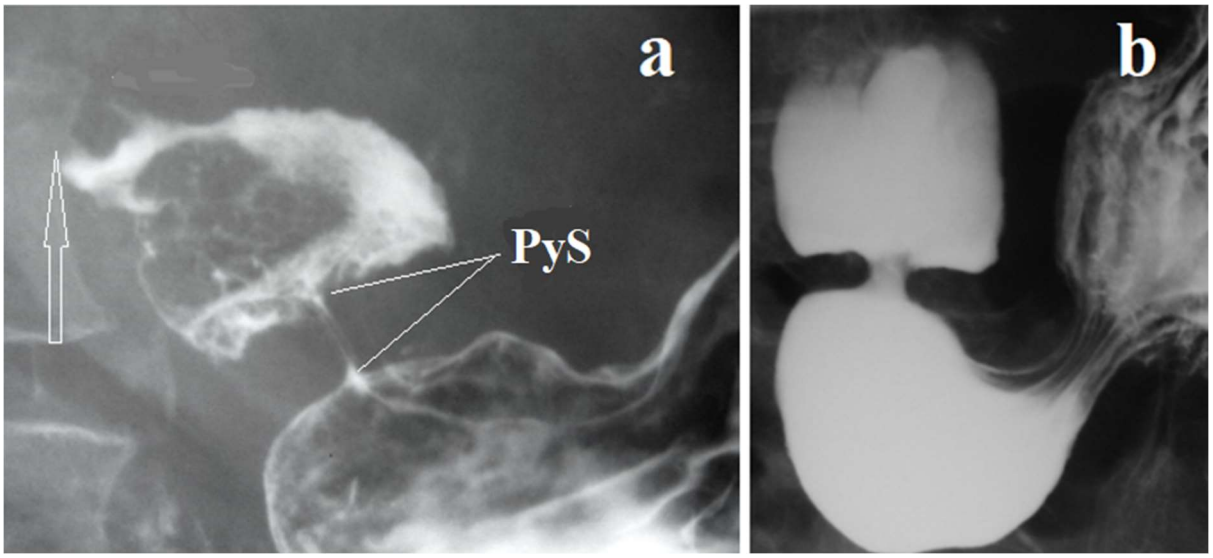

Figure 2. Radiographs of the gastroduodenal junction. (a) The PyS is in a closed state. The arrow indicates the location of the contracted post bulbar sphincter. The width of the bulbus base is almost equal to that of the opposite side of the stomach. (b) In the horizontal position, the moment of the evacuation of barium from the stomach is visible, the PyS widened, and its length decreased. The width of the base of the bulb is almost equal to the part of the stomach facing it. (The figure shows radiographs of elderly people examined for problems of the digestive tract. Therefore, the size of the pyloric canal cannot be considered normal).

The pyloric sphincter, as well as the LES and the internal anal sphincter, are not detected during anatomical examination because they do not have clear boundaries [11]. However, the contraction of muscle fibers of PyS shows clear boundaries, which proves its functional feature [7,8,12]. From a ontogenetic point of view, the duodenal bulb belongs to the stomach. It, like the stomach, originates from the foregut. Its mucous membrane is similar in structure to the mucous membrane of the antrum. The post-bulbar part of the duodenum originates from the midgut and has a structure characteristic of the small intestine [13].

The empty bulb of the duodenum (at rest) has a small diameter round shape and is no different from the small intestine. It takes on the typical triangular shape of

a bulb when filled with chyme from the stomach. At the time of filling the bulb, the width of the opened PyS ranges from 2 to 5 mm, and its length is significantly shorter than the age norm. The formation of the bulb with the simultaneous opening and shortening of PyS is explained by the structure of the muscle fibers. Superficial longitudinal muscle fibers stretch from the wall of the antrum and pass through the pylorus to the bulb. Deep longitudinal muscle fibers extend from the antral wall, penetrate through the entire thickness of the pylorus, and attach to the connective tissue of the submucosal layer of the bulb [14] (**Figure 3)**.

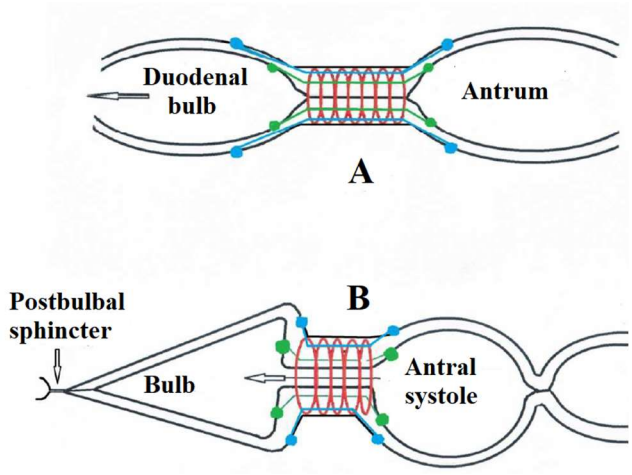

Figure 3. The scheme of the GDJ. (A) At rest, the PyS closed. (B) At the time of antral systole, concurrent with the relaxation of the circular muscle fibers of the PyS, contraction of the longitudinal muscles extending from the antrum of the stomach to the base of the bulb occurs. They stretch the base of the bulb, like parachute slings. The walls of the antrum facing the bulb are stretched to the same extent. The wider the base of the bulb, the wider the "shoulders" of the antrum. These fibers pass through the PyS and therefore when they contract, stretch its walls, actively creating a channel for the bolus to pass.

Based on the above data, the following conclusions can be drawn:

1. The formation of "shoulders" both from the side of the duodenal bulb and from the side of the stomach with simultaneous shortening and expansion of the lumen of the pyloric sphincter can only be explained by contraction of the longitudinal muscle fibers. The assumption that the contraction of the longitudinal fibers accompanied by the contraction of the circular fibers is contrary to common sense since in this case, the contraction of the circumferential fibers prevented the opening of the PyS. In accordance with this hypothesis, PyS opening occurs not only because of its relaxation of the circular muscles but also because of contraction of the longitudinal muscles, i.e. it is an active process.

2. Obviously, the advancement of the bolus from the antrum into the bulb is accompanied by contraction of the antral sphincter above and opening of the pyloric sphincter below the bolus. The advancement of the bolus from the bulb into the second part of the duodenum is accompanied by contraction of the pyloric

sphincter and opening of the post bulbar sphincter. Thus, the progression of the bolus through the GDJ follows the law of the gut. The sphincter zones differ in that the opening of the caudal sphincters occurs under a certain threshold pressure (see Figure 1).

3. The function of the antral sphincter plays an important role, firstly, to create a threshold pressure when there is no hydrostatic pressure (in the horizontal position) or if its value is below the threshold pressure (in the vertical position). Secondly, since it is involved in creating high pressure only in the anal part of the stomach, this protects the LES from high gastric pressure, which can cause GERD.

4. The peak local longitudinal shortening in the pyloric sphincter coincides in time with the maximal circular muscle contraction in the antral chamber, which is consistent with the study by Nicosia et al [15]. However, they do not coincide in localization. At the time when the longitudinal PyS fibers contract, its circular fibers relax, but at the same time, the circular fibers of the antral chamber contract. It is possible that such a shift is characteristic of the entire digestive system.

2. Esophagogastric junction motility

It is known that at rest, i.e., outside of swallowing, the pressure over the LES is lower than in the stomach [16, 17]. This means that to evacuate the bolus from the esophagus to the stomach, it is necessary to create pressure over the LES, at least more than in the stomach. In the horizontal position, esophageal distension above LES causes a significant decrease in LES pressure [18, 19]. An increase in the gastric pressure causes an increase in the tone of the LES [20, 21, 22]. I used compression of the abdomen for approximately 30 seconds during taking the barium, to increase LES tone. It has previously been found that the crural diaphragm (CD) contracts reflexively in response to an increase in gastric pressure concurrently with LES contraction. However, striated fibers of the CD quickly tire and stop contracting after an average of 18 seconds [23]. Therefore, after 30 seconds of abdominal compression, there is no longer any CD effect on EGJ.

In x-ray studies in the horizontal position, abdominal compression had no effect on the radiological examination in healthy individuals. The barium bolus passed from the esophagus into the stomach without delay because the last peristaltic wave was strong enough to overcome the increased LES tone. In patients with GERD, abdominal compression caused a contraction of the LES, which was defined as a contrast-free zone between barium in the esophagus and stomach.

The length of this interval, i.e., LES length, in patients with mild GERD, coincided with the results of a manometric study in the control group. Thus, in adults, the LES length varied from 3.2–4.2 (3.60±0.08 cm) [24]. The intraabdominal portion of the LES is near 2.0 cm [25]. In patients with GERD, the shortening of the LES was determined due to the opening of the intra-abdominal part, which was also established by manometric studies [26, 27, 28] (**Figure 4)**

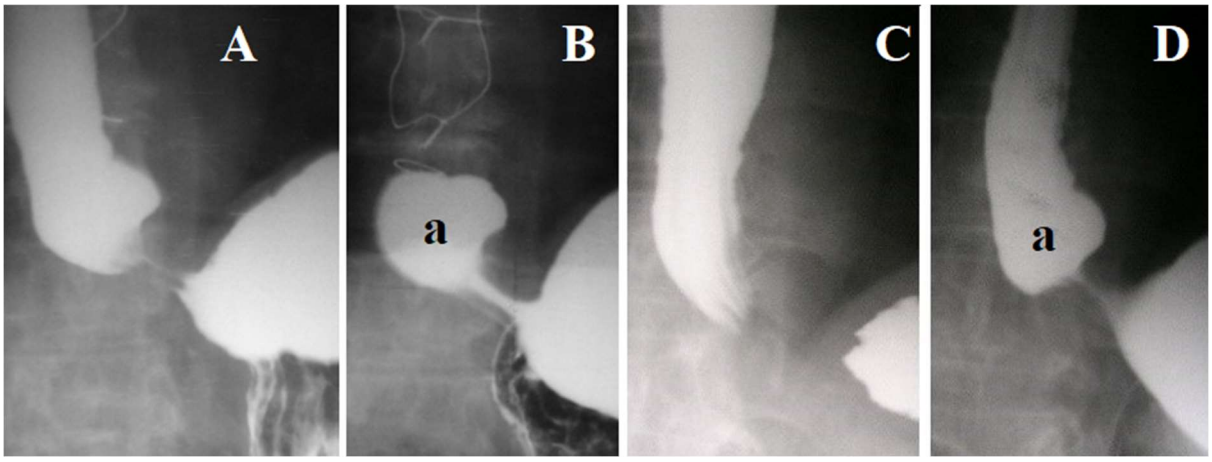

Figure 4. EGJ radiographs of patients with GERD during abdominal compression. (A-B) phrenic ampulla (a) injects a bolus into the stomach through an open LES. This means that the pressure in it is higher than in the stomach. Such a high pressure could be created due to the contraction of the functional sphincter, the contraction of which blocked the lumen of the esophagus above the ampulla. I have named this sphincter the proximal sphincter (PS). The length of the LES between the ampulla and the stomach is about 1 cm (normally about 3.6 cm), since the supraphrenic (0.5 cm) and abdominal part (2.1 cm) of it opened, unable to withstand pressure. (C-D) An example of the formation of "shoulders" in the distal esophagus and in the stomach, which face each other during the opening of the LES between them.

On x-ray studies in the upright position, the column of barium reaches the 4th thoracic vertebra and progressively descends until the barium moves into the stomach. Since at rest the pressure in the esophagus is lower than in the stomach, the hydrostatic pressure of the barium column creates a threshold pressure for the opening of the LES. Although the hydrostatic pressure decreases during the emptying of the esophagus, the LES remains open until all barium enters the stomach. This indicates that the opening of the LES is an active reflex process, and not a mechanical phenomenon. A decrease in hydrostatic pressure in the esophagus does not lead to gas reflux, since during swallowing, because of the receptive relaxation, the tone of the proximal stomach decreases.

The mechanism of this opening is explained by the location of muscle fibers in the LES. In 1979, Liebermann-Meffert et al. used “a new method of preparing dried fiber specimens. They found: "The muscle bundles of the inner muscle coat

split up 10.2 mm +/- 3.0 SD above the GER (fixed specimen) and for a length of 25 mm +/- 8 SD formed short transverse muscle clasps on the lesser curve side. Those muscle bundles on the greater curve side formed long oblique gastric fiber loops. described on the cardiac part of the stomach a short transverse muscle clasps on the lesser curve side and those muscle bundles on the greater curve side formed long oblique gastric fiber loops". They suggested that "these loops along with the clasps represent the intra-abdominal component of the LES" [29]. Obviously, when the oblique longitudinal muscle fibers contract, which are attached to the wall of the esophagus above the LES, and below the sphincter to the lesser and greater curvature of the stomach, the LES will open and become shorter **(Figure 5).**

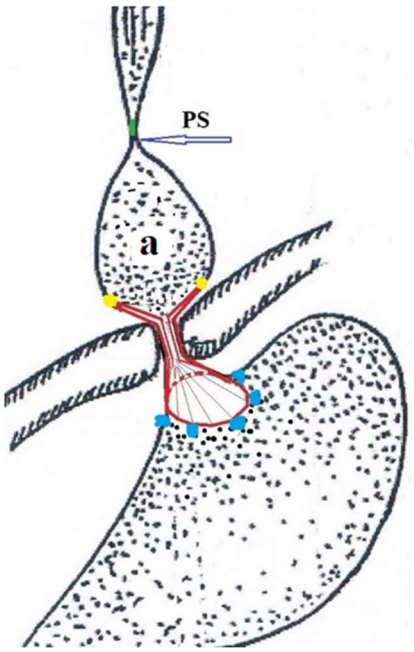

Figure 5. Diagram of bolus evacuation from the ampulla to the stomach in a horizontal position in a patient with GERD (see Figure 4.B). The shortening of the LES is due to the opening of its abdominal and supradiaphragmatic parts. The ampulla injects a bolus into the stomach due to contraction of the proximal sphincter (PC). Its length does not exceed 0.7 cm. CD is located in the hiatal canal and cannot be more than 1 cm. The yellow dots are the places of attachment of oblique fibers to the esophagus, blue dots are its attachment to the stomach.

Confirmation of this hypothesis is the formation of "shoulders" in the esophagus and stomach, facing each other, as is observed in the formation of the duodenal bulb (see Figure 4. C-D).

The anti-reflux function of the EGJ is carried out by two sphincters.

The LES is the internal sphincter. It is formed by smooth muscle fibers, which can constantly be in a contracted state because of the change in the contraction of some bundles of muscle fibers by others (like a postural reflex). The tone of the LES is proportional to the pressure in the fundal part of the stomach. Its length is about 3.6 cm. The LES consists of three parts. The distal 2.1 cm of the LES is in the abdominal cavity. One centimeter of the LES is located inside the hiatal canal, where the CD functions around it. Above the diaphragm are 0.5 cm LES. These indicators correspond to manometric, radiological, and anatomical studies **(Figure 6) [28, 30].**

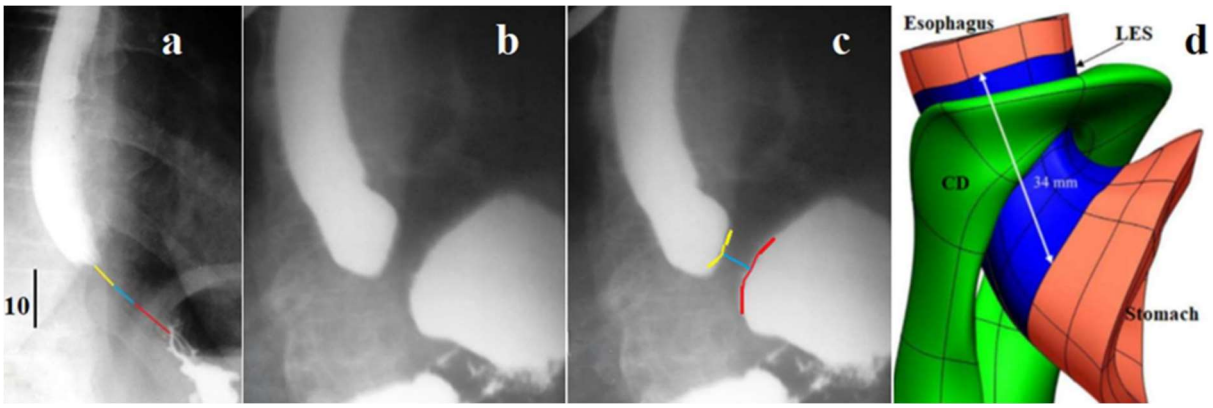

Figure 6. (a). Radiograph of a patient with GERD in an upright position taken during abdominal compression. The zone without contrast agent between the esophagus and stomach is the contracted LES. Since the actual height of D-10 is ≈2 cm, the actual length of the LES is ≈3.3 cm. Diagram of the LES parts: red - the abdominal segment, blue - inside the diaphragm, yellow - above the diaphragm. **(b, c).** The same patient during abdominal compression in a horizontal position. A sharp shortening of the LES is determined due to the disclosure of the intra-abdominal (red line) and supradiaphragmatic (yellow line) parts. Only a part of the LES 1 cm long located at the level of the diaphragm is in a contracted state. **(d)** Three-Dimensional Model of the Esophageal Gastro Junction [30]. The length of the LES is 3.4 cm (blue). Its abdominal part is ≈2 cm. About 1 cm is located at the level of the CD and 0.4 cm above the diaphragm.

CD is the external sphincter. Its length is 1 cm. It consists of striated muscles, which contracts during the rise in gastric pressure, including with deep inspiration. However, like any striated muscle, it tires quickly and relaxes after an average of 18 seconds. The contribution of each of these sphincters is evidenced by the conclusion of an experiment in dogs: «The presence of a mechanical barrier also after excision of the left half of the diaphragm, as evidenced by both pressure and reflux volume, can only be explained by an intrinsic sphincter, a lower esophageal sphincter (LES). However, the experiments also showed that the diaphragm contributed to the competence of the LES» [31].

Based on the above data, the following conclusions can be drawn:

1. The formation of "shoulders" both from the side of the esophagus and from the side of the stomach with simultaneous shortening and expansion of the lumen of the LES can only be explained by contraction of the longitudinal muscle fibers. It is likely that the opening of the LES is due to the reflex relaxation of its circular muscles and contraction of the longitudinal ones. Since we only assessed diseased individuals, we have no evidence that LES shortening occurs in healthy individuals when LES opens.

2. Obviously, the advancement of the bolus from the phrenic ampulla into the stomach accompanied by contraction of the proximal sphincter above and opening of the LES below the bolus. Thus, the progression of the bolus through the GDJ follows the law of the gut. The sphincter zones differ in that the opening of the sphincters occurs under a certain threshold pressure over them. According to the hypothesis of Sugarbaker et al [32], contraction of the circular layer of the esophagus during peristalsis, it is accompanied by a simultaneous local contraction of the longitudinal layer, which leads to local shortening of the esophagus [15]. If this phenomenon is an addition to the law of the intestine, and therefore refers to the entire digestive tract, then the contraction of the last peristaltic wave (ampulla) is normally accompanied by relaxation of the circular fibers of the LES with a simultaneous contraction of its longitudinal (oblique) fibers, i.e., relaxation and opening of the LES in normally occurs at the same time.
3. The function of the proximal sphincter, like the antral sphincter, plays an important role, for create a threshold pressure when there is no hydrostatic pressure (in the horizontal position) or if its value is below the threshold pressure (in the vertical position). The emergence of functional sphincters in the pre-sphincter zones, i.e., with a high threshold pressure or with impaired passage, is a pattern, as can be seen from the example, the appearance of several functional colonic sphincters.

Truth does not depend on the method of research. We can evaluate EGJ radiographs of elderly patients with GERD, which clearly show what is established based on manometric, histological, different radiological studies (X-ray, US, CT) and MRI. (**Figure 7)**.

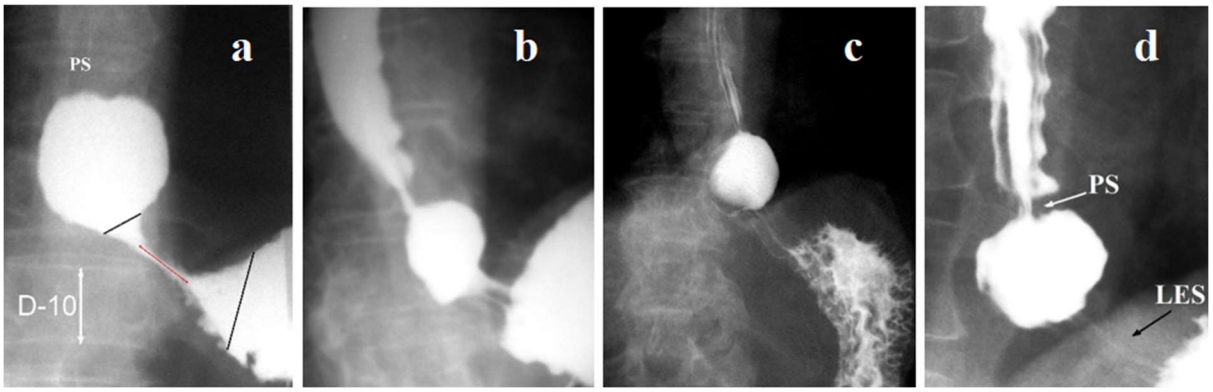

Figure 7. EGJ radiographs of patients with GERD took ≈ 30 seconds from the start of abdominal compression. In all cases, abdominal compression resulted in a reflex contraction

of LES and CD. However, by the time the radiograph was taken, the CD striated muscle had relaxed, and we only see the contracted LES.

The mathematical analysis of each radiograph is based on the knowledge that the true height of the 10th thoracic vertebra in adults is approximately 2 cm. Each reader can verify the correctness of the measurements.

(a). The width of the ampulla is 3.7 cm, the length of the contracted section of the LES is 1.7 cm (red line), and the length of the entire LES between the black lines is 4 cm. It includes an open epiphrenic part, a contracted area (red line), and an open abdominal part of the LES, which looks like an angular deformity of the stomach. A shortened section 1.7 cm long cannot be caused by CD contraction, since, firstly, by this moment the CD has relaxed, and secondly, the CD contraction cannot be more than 1 cm. Thirdly, theoretically, the esophagus shortens locally at the site of contraction of circular fibers and cannot be shortened by 4 cm. After injection of barium into the stomach, this ampulla is contracted. It gradually decreases in size and finally disappears for observation. And it is not visible in a vertical position. So, the round cavity above the diaphragm it's the phrenic ampulla, not the stomach.

(b). As a result of compression of the abdomen, only a 1-cm LES area located inside the hiatal canal was contracted. Other departments of the LES could not stand the strain. The area of contraction of the esophagus above the ampulla, 0.7 cm long, cannot be the LES, which is normally approximately 3.6 cm long.

(c). Longitudinal folds are visible throughout the esophagus. The PS contraction 0.5 cm long, seals the ampoule, which makes it possible to create a threshold pressure. The width of the ampoule is 2 cm. Wide longitudinal folds are visible at the level of the shortened LES 2 cm long. The remaining parts of the LES proved to be untenable. Longitudinal folds are the result of an inflammatory process. At the LES level, they are wider due to the higher pressure and thicker wall. These folds have nothing to do with the stomach.

(d). The longitudinal folds in the esophagus are wider than at the level of the LES, reflecting the difference in the inflammatory process. The length of the PS is 0.3 cm. The width of the ampulla is 2.4 cm, and the length of the contracted LES is 2 cm.

Each radiograph is scientific proof that:

1. Longitudinal folds over the stomach reflect inflammation in the walls of the LES.

2. LES does not move into the chest either at rest or when swallowing.

3. The stomach does not move into the chest, and the rounded cavity above the LES is the ampulla of the esophagus, regardless of its size. First, it's not a hernia. Secondly, the presence of the ampoule is evidence of GERD.

4. The proximal sphincter is a functional sphincter, like the antral sphincter and colonic sphincters, which provide threshold pressure in the pre-sphincter zones.

Conclusion.

The truth does not depend on research methods (manometric, histological, different radiological studies (X-ray, US, CT) and MRI. Each of them contributes to the understanding of the normal physiology of DGJ and EGJ, which function according to the same laws as all other sphincter zones. The results of these methods do not contradict each other.

All conclusions based on the use of HRM are false. This is the result of a violation of the methodology of science: selection of control persons based on the absence of complaints, selection of manometric limits by voting, application of manometric parameters that do not make physiological sense, the use of false hypotheses as scientific facts, the suppression of scientific discussion. The huge number of supporters of HRM in the absence of critical articles is the most eloquent evidence of the anti-science of this method.

This is dangerous because what:

1. In those cases where earlier GERD with hypertensive LES was recently diagnosed [33,34] has now been diagnosed as esophageal achalasia or other manometric abnormalities on HRM. Instead of pathogenetic treatment of a weakened and poorly functioning LES, its dissection or other methods are performed that weaken its function for the sake of temporary relief of symptoms. All articles are similar to each other, and they do not talk about the treatment of the disease, and not even about alleviating the symptoms of the disease, but only about alleviating the symptoms of achalasia, established on the basis of HRM - chest pain and dysphagia. For example, an article by Kumar et al recommends an injection of Botox into the LES, even though the symptoms of so-called achalasia after that have been replaced by heartburn, i.e., more severe pain than before [35]. This is clearly stated in Nullens et al: "POEM and LHM both have excellent 2-year **success rates for relieving achalasia symptoms**, but reflux disease and erosive esophagitis are more prevalent following POEM" [36].

The above analysis is consistent with Wilkinson et al: «Patients with esophageal dysphagia may report a sensation of food getting stuck after swallowing. This condition is most commonly caused by gastroesophageal reflux disease and

functional esophageal disorders. Esophageal motility disorders such as achalasia are relatively rare and may be overdiagnosed» [37].

2. Long-term results of achalasia surgical treatment.

First, "Although highly effective, 10 to 20% of achalasia patients treated with laparoscopic Heller myotomy (LHM) or per-oral endoscopic myotomy (POEM) remain symptomatic" [38]. However, these figures refer only to the symptoms of achalasia. Clinical success is defined as a decrease in the Eckardt score to 3 or below.

Second, "Abnormal acid reflux (total acid exposure time greater than 5%) was in 53.4% of patients [but only 24.6% reported heartburn, and esophagitis was present in only 27.4% of patients, with a minority of them reporting heartburn (9 out of 20)" [36]. These arguments mislead readers, as they are contrary to common sense. (1). Because 5% means that acid with pH < 4 acts on the wall of the esophagus for more than 1 hour per day. pH monitoring only diagnoses very severe forms of GERD. (2). It is known that GERD can occur and progress in the absence of symptoms. The above figures indicate that esophagitis can be without heartburn, and heartburn without GERD. The following reference is misleading as the authors only diagnosed GERD by a questionnaire and could not prove that: esophageal acid exposure in achalasia does not necessarily result from acid reflux, as acidification of stagnant foods can also cause a high esophageal acid exposure time" [39].

In all patients with so-called "achalasia", except for very rare cases of true achalasia, GERD is the cause of the symptoms. After surgical treatment, the antireflux function of the LES deteriorates sharply. Therefore, "of patients treated with LHM, Dor fundoplasty was the most common accompanying anti-reflux surgery, 28 (43.1%); 3 (4.6%) had a Nissen fundoplication (outside hospitals) and 22 (33.8%) had a Toupet fundoplication" [38]. This means that after dissection of about 7 cm of the muscular wall in the distal esophagus, including the LES, which is about 3.6 cm long, an additional fundoplication was performed in 82% of cases. Due to the transection of the muscular layer of the distal esophagus, some of these patients, as well as after POEM, developed a diverticulum at the site of the incision [38] (**Figure 8**).

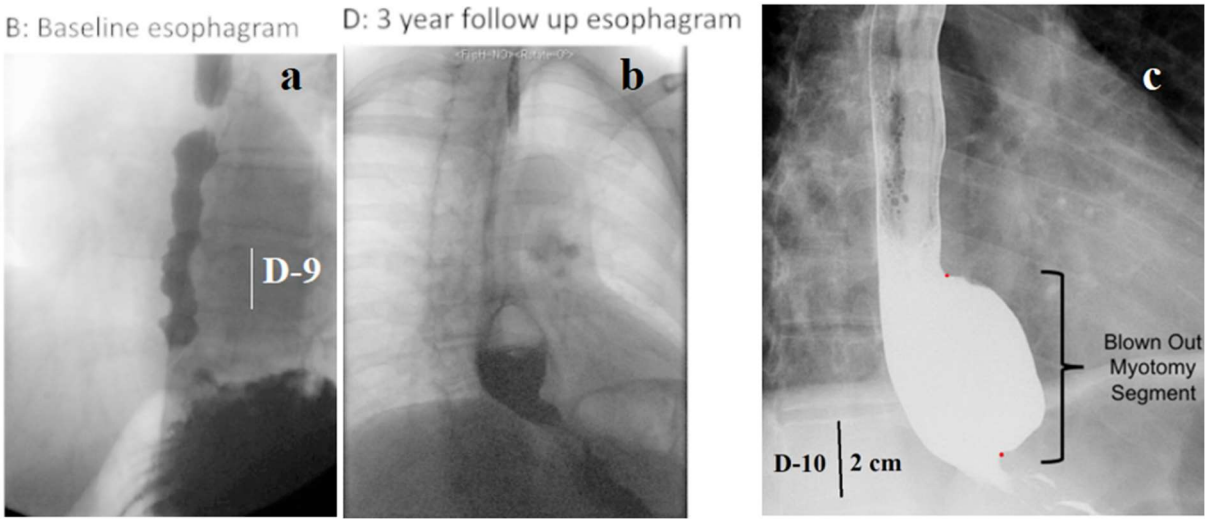

Figure 8. Radiographs from Triggs et al [38]. (a). Patient presented with dysphagia and chest pain and was referred for manometry. Manometry was consistent with Type III achalasia. The patient underwent POEM with a standard 9 cm myotomy. (b). The esophagram revealed a large pouch along the myotomy length that failed to empty consistent with a blown out myotomy. (c). Patient esophagrams were reviewed to assess for a BOM, defined as a distal wide-mouthed (>2 cm) diverticulum in the area of the prior myotomy with a greater than 50% increase in esophageal diameter.

On x-ray (a), the true height of D-9 is approximately 1.9 cm. X-ray parameters indicate GERD with severe esophagitis: The esophagus is twice as narrow as normal (0.7 vs. 1.5 cm). Its tone is high with interceptive peristalsis in the upper part. The contours are uneven and asymmetrical. The length of the LES is about 2 cm, which is almost 2 times shorter than the normal (3.6 cm). The evacuation of liquid barium into the stomach is not disturbed. There is gas in the stomach. These parameters exclude esophageal achalasia, in which the esophagus is always dilated, the LES is of normal size, evacuation to the stomach is severely impaired, and there is no gas in the stomach.

On x-ray (b), the manometric characteristic is not a diagnosis. As a result of a meaningless manometric study, in addition to crossing the muscular wall of the LES, about 5 cm of the esophageal wall was cut. First, POEM drastically weakened already weak LES function, exacerbating GERD. Secondly, a diverticulum of the esophagus has formed, which is especially dangerous because acid reflux is retained in it. In addition, such diverticula tend to increase, because the mucous membrane prolapses through the hole in the muscle layer.

On x-ray (c), the radiological picture does not correspond to achalasia. The wide mouth of the diverticulum is 5 cm. There is no justification for the operation in general, and even more so for the dissection of the wall of the esophagus.

Output

Most patients with the so-called achalasia or other forms of violation of manometric indicators is complicated forms of GERD. As a result of misdiagnosis, treatment is directed not at enhancing the anti-reflux function of the EGJ, but at reducing the function of the LES.

1. From 10 to 20% of achalasia patients treated with laparoscopic Heller myotomy (LHM) or per-oral endoscopic myotomy (POEM) remain symptomatic"

2. A significant percentage of patients develop a diverticulum of the esophagus.

3. GERD progresses in all patients.

4. “The surgery for achalasia usually improves passage symptoms, but esophageal cancer still arises in some cases and the number of tumors occurring many years later is not negligible” [40]. Obviously, the unusually high incidence of esophageal cancer after surgical treatment of achalasia is due to the lack of treatment for GERD.

Alternative (1). Combined non-operative treatment

Non-operative treatment by BT injection into the LES, and PD a month later. The results have demonstrated the long-term efficacy of combination treatment in patients with achalasia who would otherwise have to undergo a risky and costly procedure, making it a safe and effective alternative to myotomy [41].

This program is as effective as surgical methods but excludes all surgical complications. However, it does not provide for the presence, and hence the treatment of GERD, which is exacerbated after this program.

Alternative (2). Pathogenic treatment of GERD

1. Complete and prolonged exclusion of products containing lactose, which provokes hypersecretion of hydrochloric acid. See the lactose intolerance test here [42].

2. Appointment of PPI for no longer than 8 weeks with a gradual decrease in dose until complete elimination within 4 weeks.

3. Go to bed with an empty stomach. Go to bed 5 hours after a light dinner without meat and fat.

4. Reduce the volume of a single meal and the time interval between them.

5. Antimicrobial treatment if a microorganism is found on histological examination of the esophagus.

6. Anti-inflammatory treatment (antihistamines, hormones).

7. Swallowing a large tablet with a diameter of 1.8-2.3 cm to expand the lumen of the esophagus, LES, and pyloric sphincter [43].

8. In the absence of effect, fundoplication is indicated as an extreme case.

Note. This does not apply to true achalasia with a sharp expansion of the esophagus, where the LES is of normal length (≈ 4 cm) and there is no gas in the stomach. In such cases, inflation of the balloon over the LES results in contraction of the LES, rather than relaxation, as is normal.

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