The function of the esophagus and gastroesophageal junction is normal and in gastroesophageal reflux disease.

M.D. Levin, MD, PhD, DSc. Pediatric surgeon, radiologist,

Department of Pediatric Radiology of the 1-st State Hospital, Minsk, Belarus.

Dorot-Netanya Geriatric Medical Center, Israel.

Amnon VeTamar, 1/2, Netanya, 42202, Israel.

nivel70@hotmail.com; michael.levin@dorot.health.gov.il

https://orcid.org/0000-0001-7830-1944

https://www.anorectalmalformations.com

Scopus Author ID: 7402571390

I. Introduction. The esophagus is a part of the digestive tube that provides the passage of a food from the pharynx to the stomach and prevents a retrograde movement of a food, i.e. reflux from the stomach to the esophagus. Anatomically, this structure is divided into the upper esophageal sphincter (UES), the body of the esophagus and the lower esophagus sphincter (LES). The LES is in the distal part of the esophagus and it is a thickened circular muscular layer compared with the body of the esophagus. Its caudal part is about 2.1 cm long (in adults) is located below the diaphragm, i.e. intraabdominal (2.19±0.972 cm [1]. Since its cranial point does not have a clear localization, the length of the LES cannot be measured at the autopsy or during surgery [1]. The LES is in constant contraction, therefore the intraluminal pressure at the LES level is higher than in the esophagus and stomach. The length of the LES is measured by the manometric method as a high-pressure zone, which increases with age to 4 cm in adults (3.82±0.953 cm) [1]. The LES surrounded by the crural diaphragm at the tenth thoracic vertebra (T 10) [2].

Following the relaxation of the UES and the passage of the bolus into the esophagus, peristaltic muscular contraction propels the bolus toward the LES. Relaxation of the LES in conjunction with the peristaltic propulsion of the bolus allows the entry of the bolus into the stomach. Generally, in peristalsis, the area ahead of the bolus is relaxed, and the area behind the bolus is undergoing peristaltic contraction which allows for the bolus to be propelled forward [2]. Studies by Shafik et al. convincingly proved that an increase in pressure in the stomach and abdomen causes an increase in pressure and electrical activity not only in the LES, but also in the crural diaphragm [3-5].

The abdominal LES length in reflux group is significantly shorter than the non-reflux group [6,7]. The dilated distal esophagus is the pathologic expression of damage to the abdominal segment of the LES [8].

In recent years, the diagnosis of gastroesophageal reflux disease (GERD) is in a crisis due to there are no clear criteria of the norm. In the conclusion of the articles devoted to this problem, there are no clear recommendations. For example, the article by Masuda et al. states that "Patients with maximal tHH≥2 cm at least 1 swallow were more likely to experience pathological reflux than patients with maximal tHH < 2 cm" [9]. In the literature, at the same time a mass of contradictory and / or illogical statements circulate. For example, Chandrasoma et al. have been proving for 9 years that reflux starts only in the abdominal segment of the LES, when the chyme still does not penetrate the esophagus [8]. Despite the fact that so far no one has been able to dismiss the evidence of Dr. Chandrasoma, in the gastroenterolgy the criteria for GERD based on results of pH monitoring was defined as reflux index (RI) (% proportion of time during which esophageal pH is below 4) > 10% in infants, > 7% in older children, and > 4 in adults [10]. How can these indicators be the limit of the norm, if for a long time the esophagus is damaged by aggressive hydrochloric acid along with pepsin? These numbers indicate a poor anti-reflux function of GEJ. It is not surprising that some of the authors found the RI measured by pH-metry had a

sensitivity and specificity 50% and 82%, respectively, using history and physical examination as the gold standard method for diagnosing GERD [10]. It is surprising that the expansion of the digestive tube over the diaphragm to a diameter of 2 cm is a phrenic ampulla, and > 2 cm is a hiatal hernia. Endoscopists demarcate the gastroesophageal junction (GEJ) at the top of gastric folds, but Dr Chandrasoma argues that this is an unreliable landmark in GERD patients in whom the distal esophagus has dilated, and developed rugal-like folds easily mistaken for gastric folds.

II. X-ray anatomy and physiology of the esophagus and gastroesophageal junction (GEJ).

There are significant differences between the function of the esophagus and the GEJ in the vertical and horizontal position of the patient.

1. In the vertical position, the barium suspension forms a pillar above the lower esophageal sphincter (LES), the upper limit of which does not rise above the 4th thoracic vertebra. The pressure of this pillar causes a reflex opening of the LES, as a result of which the entire contrast material without delay falls into the stomach. The cleansing of the esophagus is quick and complete. The maximum width of the esophagus is no more than 1.2 cm. The functioning area of the LES is not determined. Provocative tests, including compression of the abdomen or deep breath, did not affect the speed of cleansing the esophagus [11]. In GERD the threshold pressure for the LES opening increases and / or the tone and contraction force of the distal esophagus decreases. In such cases, the abdominal compression which causes an increase in the tone of the LES can close it (**Figure 1**).

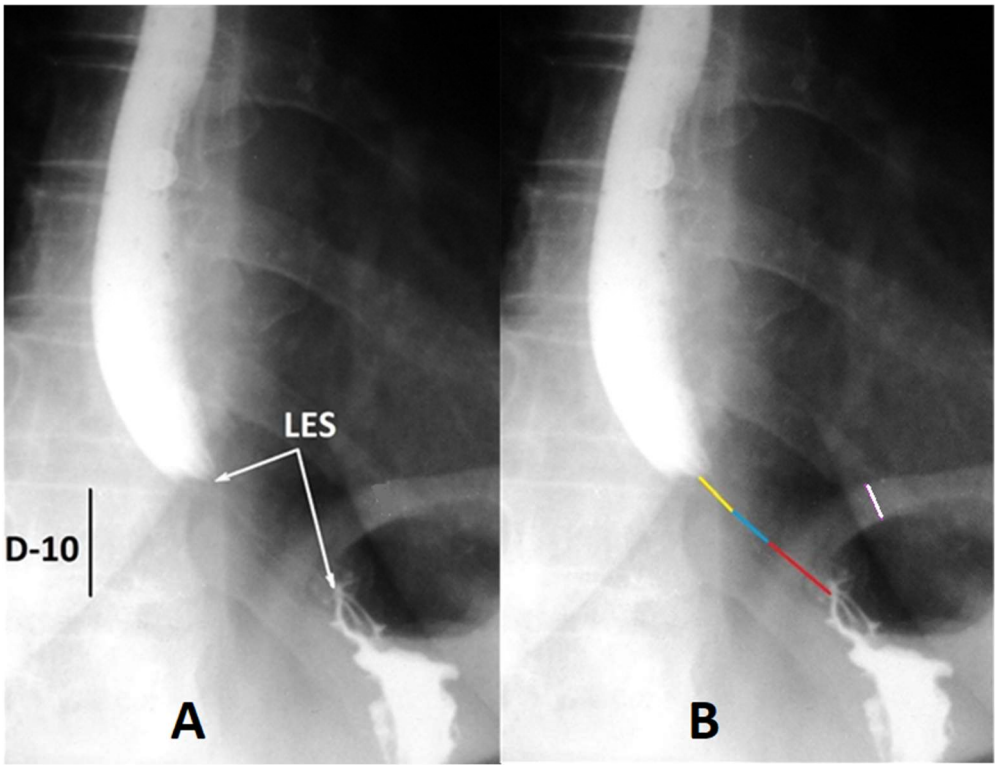


Figure 1. (A) Radiograph of a GEJ in an adult with GERD during compression of the abdomen at barium swallowing. **(B)** The same radiograph for analysis. Since it is known that the height of the 10th thoracic vertebra (D-10) in people with a height of 170-180 cm is 2 cm, we calculated the length of the LES (3.3 cm), the length of the intra-abdominal part (red line - 1.3 cm), at the level diaphragm (blue line - 1 cm), above the diaphragm (yellow line -1 cm). The thickness of the stomach wall (white line) is 0.7 cm. The shortening of the LES occurred due to the shortening of the intraabdominal portion of the LES (1.3 cm versus 2.1 cm). The image shows 2 folds of the open portion of the intraabdominal portion of the LES caudally to red line (see the study of this patient in a horizontal position in **Figure 7. AB**).

2. In a horizontal position, the bolus moves under the influence of a peristaltic wave. When the bolus approaches the distal part of the esophagus, the last peristaltic wave creates a threshold pressure above the LES, which leads to a reflex opening of the LES. Continuing to contract, the latter peristaltic wave injects a bolus into the stomach, after which the LES contracts, preventing reflux into the esophagus. When the LES is closed, the barium in the esophagus no longer exists, so it is not possible to differentiate the LES (**Figure 2**).

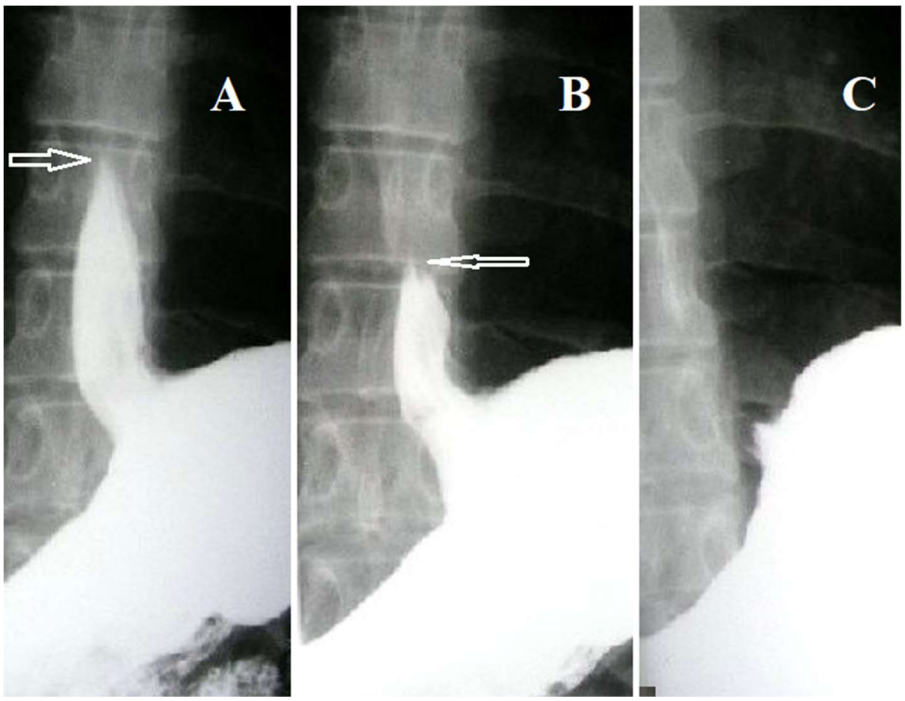


Figure 2. Elderly patients with GERD. A series of pictures taken during the reception of barium without provocative tests. It is known that at rest the pressure in the lower esophagus is lower than in the stomach [12]. For the bolus to penetrate the stomach during the opening of the LES, the last peristaltic wave must create a pressure higher than in the stomach. The arrow shows the exceptional importance of the force of contraction of the last peristaltic wave.

The maximum width of the esophagus did not exceed 1.5 cm and was the same throughout, including over the diaphragmatic zone. Normally, the use of provocative tests did not lead to barium reflux from the stomach into the esophagus. Despite the compression of the abdomen during the passage of barium through the GEJ the contrast agent passed into the stomach without delay. The cardiac part of the stomach always had a rounded configuration. The contours of the esophagus have always been smooth. There were no folds in the esophagus or in the area of the GEJ.

Thus, in patients of different ages, without any signs of the GERD, either in vertically or horizontally positions, despite the use of various provocative tests, it was impossible to see and measure of the LES.

Retrospectively, the results of the examination of 55 patients were selected who had none of the typical symptoms of GERD. In children X-ray examination

was conducted for localization of foreign bodies, the space-occupying lesions of the chest and the abdomen, as well as for determining the cause of abdominal pain. In adults X-ray examination was performed to determine the cause of anemia and / or recurrent pain in the abdomen. The results of their examination differed from patients in whom GERB was excluded by the fact that in response to the abdominal compression there was a short-term contraction of the LES, as in patients with reflux (**Figure 3**).

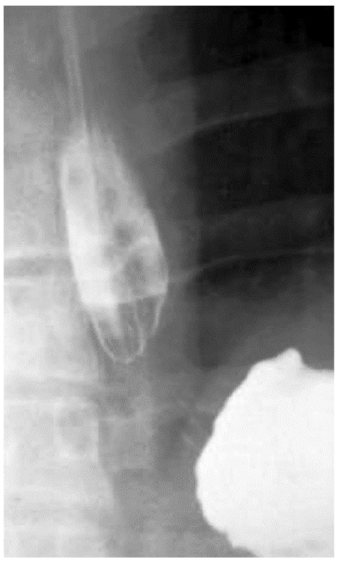


Figure 3. During the abdominal compression a gap without contrast agent was formed between barium in the esophagus and in the stomach. It is the contracted LES.

The results of the measurement of the gap between the esophagus and the stomach in 55 patients without typical symptoms of GERD are given in **Table 1.** Since they completely coincide with the results of measuring the length of a normal LES by the manometric method (3.82±0.953 cm) [1], (3.5±0.4 cm) [13], (3.4±0.9 cm) [14], we believe that this gap is a contracted LES. Probably, these patients were examined in the initial stage of GERD, when the length of the LES was not significantly changed compared to the absolute norm.

Table 1. The normal length of the LES in different age groups.

Age	Up to a year	1-3 years	4-7 years	8-10 years	11-15 years	21– 65 years
Limits	0.7 – 1.0	1.2 – 1.5	1.5– 1.8	1.9 – 2.3	2.3 – 2.9	3.2 -4.2
Average	0.86±0.03	1.40±0.02	1.72±0.07	2.10±0.05	2.45±0.11	3.60±0.08

After the passage of barium into the stomach, no trace of the contrast agent remains either in the esophagus or at the level of the LES. This indicates a surprising elasticity of tissue. A diagram of the LES cross section during the bolus passage (A) and after its contraction (B) is presented in **Figure 4.**

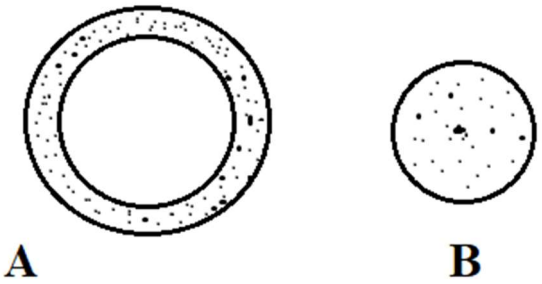


Figure 4. A diagram of the LES cross section during the bolus passage (A) and after its contraction (B).

III. Pathological physiology of the GEJ in GERD.

It is known that the inflammatory process in the esophagus in GERD leads to the expansion of its lumen [10]. This symptom can be identified in the earlier stages of the pathological process if to use abdominal compression. An increase of the gastric pressure causes a contraction of the LES. The contraction of the last peristaltic wave in front of an obstacle leads to an increase in pressure over the closed LES and expands this zone. (**Figure 5**).

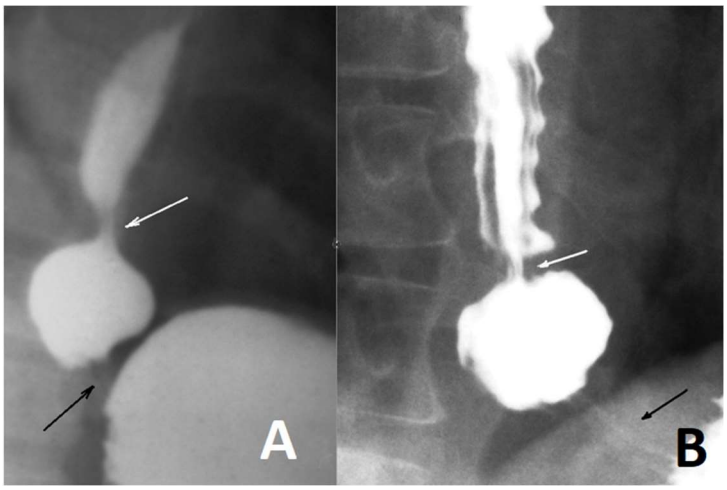
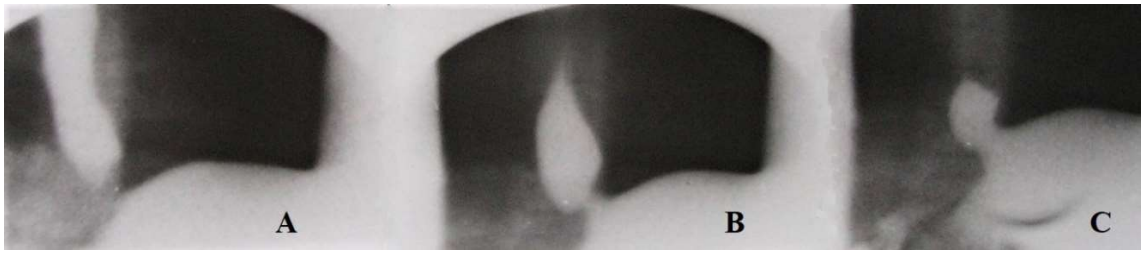


Figure 5. Radiographs of GEJ during compression of the abdomen. (A) A 5-year-old child with symptoms of GERD. Compression of the abdomen during the swallowing of barium caused a contraction of the LES (black arrow). The last peristaltic wave formed a closed circular chamber (phrenic ampulla), which was closed proximally by a functional sphincter. I call it the proximal sphincter (PS - white arrow). The phrenic ampulla during contraction creates high pressure to open the LES. Two thin folds throughout the LES indicate a decrease in the elasticity of the mucosae due to the inflammatory process. (B) Elderly patient with GERD. The LES contracted during compression of the abdomen. Its length is 2 cm. At the level of the LES, the mucous folds are thin due to the high tone. In the esophagus folds are wide, as the pressure in it is much less and the width is greater. It is not completely blocked proximally due to weak PS. I think that the PS is turning into a rigid ring - Schatzki ring is 4 mm long.

First, the ampulla of the esophagus occurs when it is filling with barium, which comes from the esophagus but not from the stomach, and then it closes proximally by the contracted PS. The point of intersection of the folds with the stomach does not change its position. The process of the ampulla formation is seen in **Figure 6.**



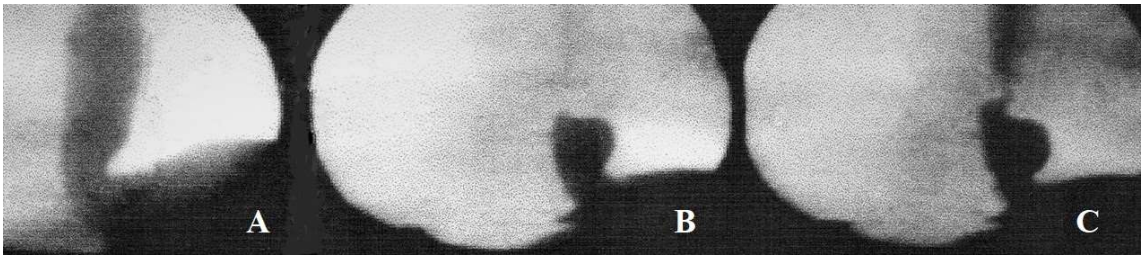


Figure 6. (Two examples). Sequential shots of filming the process of swallowing in a horizontal position of a child with severe GERD. **(A)** There is a significant expansion of the esophagus. Due to ineffective peristalsis, the threshold pressure for opening the LES is created by the contraction of the esophageal walls between the upper and lower esophageal sphincters. **(B)** In the final phase of the esophageal emptying an ampulla emerged to create a positive pressure gradient. **(C)** (down below) The PS did not withstand the pressure and the ampulla was unlocked, the pressure in it dropped and the barium began to flow into the proximal esophagus.

Secondly, the gap between the barium in the esophagus and the stomach, which is created by the constricted diaphragmatic crura, is limited by the length of the hiatus canal and cannot be 1.5 or 2 cm long. In addition, Shafic et al. proved, that "... CD (crural diaphragm) consists of striated muscle fibers, which are easily fatigable and cannot remain contracted for long period" [15]. Since this narrowing zone does not relax for 30 seconds [11], we can claim that the contraction zone, which is located caudally relative to the ampulla, is formed by the contracted LES, so the LES does not shift regardless of the width of the ampulla. The crural diaphragm act as an external sphincter of the anti-reflux barrier. Contraction of the crural diaphragm increase LES pressure during each inspiration and in situation of increased abdominal pressure [15,16]. Thus, the contraction zone in the distal part of the ampulla at the level of the diaphragm cannot only be the result of contraction of the crural diaphragm. It is formed by the joint contraction of the LES and the crural diaphragm (external lower esophageal sphincter) regardless of the width of the ampulla.

Thirdly, the functional sphincter that closes the ampulla proximally cannot be of the LES, since its length does not exceed 5 mm in children and 7 mm in adults, that is, 5-6 times shorter than the normal LES [11]. Only when a rigid, non-

closing ring (Schatzki ring) is formed in its place, its length is shortened to 3-4 cm [17]. PS is contracted only at the time of the ampulla formation and disappears (relaxed) after emptying the esophagus, while the LES, is in a state of constant contraction. Thus, the contraction zone in the proximal part of the ampulla cannot be the LES, which has moved upwards.

Fourth, it is known that the LES at GERD is shorter than normal due to shortened by its intra-abdominal segment. This is confirmed by numerous manometric studies and is manifested by a sharp decrease in pressure in the distal part of the LES [1,2,6,13,14]. Chandrasoma as a result of endoscopic and histological examinations found that "The dilated distal esophagus is the pathologic expression of damage to the abdominal segment of the LES" [8]. In X-ray examination, the shortening of the LES is manifested by a decrease in the gap between barium in the esophagus and in the stomach **(Figure 7, A and B**), or in the form of angular deformation of the stomach (**Figure 7, C and D**). The degree of damage to the LES can be judged not only by the remaining length of the LES, but also by the strength of the provocation at which these symptoms are detected [11,17].

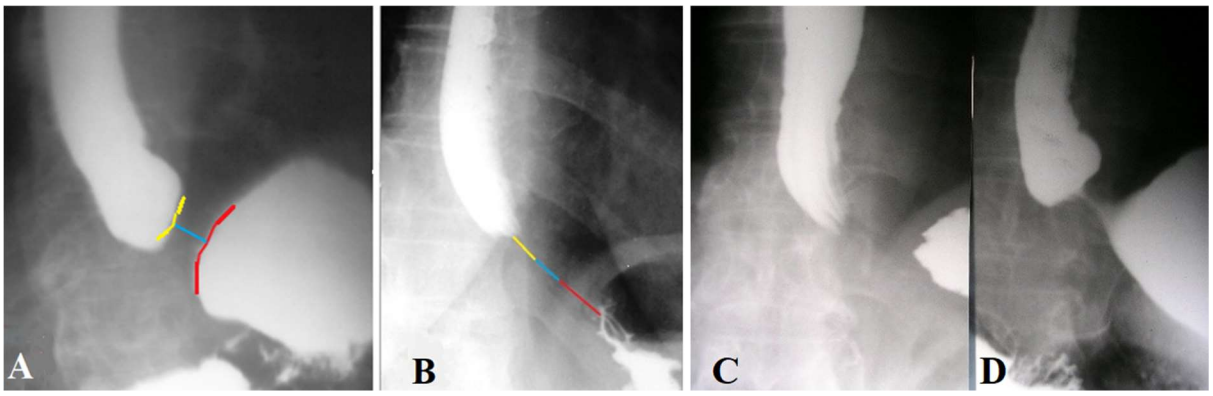


Figure 7. Radiographs of GEJ in two adult patients with GERD (**A-B** and **C-D**). (**A**) Investigation in a horizontal position during compression of the abdomen (before the formation of the phrenic ampulla). This study of the same patient, who was examined in an upright position (**B**) [see Figure 1]. The length of the gap between the barium in the esophagus and in the stomach at the level of the diaphragm is 1 cm. This happened as a result of the disclosure of the intra-

abdominal segment (red) and the segment of the LES over diaphragm (yellow) . (**C**) Short LES (about 2 cm). (**D**) During compression of the abdomen, the LES has become even shorter as a result of the full opening of its intra-abdominal segment in the form of angular deformation of the stomach.

Fifth. In a study by Rorn et al. it was found that "The average cardia perimeter was 6.3 cm in control subjects, 8.9 cm in GERD patients, and 13.8 cm in patients with Barrett's esophagus" [18]. In GERD, the thicker distal esophageal wall is observed, especially those with high-grade reflux esophagitis [19]. The authors found that diaphragmatic hiatus size significantly larger in patients high grade reflux esophagitis and the mean value more than twofold larger than that in the normal volunteers. According to their opinion, "The presence of hernia in reflux esophagitis patients seem to be fewer than examined by endoscopy and esophagogram. In this study, a diagnosis of esophageal hiatal hernia was made when a 1 cm or longer gastric wall (rugal folds?) was present above the diaphragm by CT imaging" [19].

I have not found a single study with evidence that the folds above the diaphragm are the folds of the stomach. If the wall of the LES is exposed to aggressive chyme of the stomach earlier than other parts of the esophagus; if its lumen is 2 times wider than in the control; if its wall is thicker than in the control, this indicates a strong inflammatory process. Obviously, the wall of the LES in patients with esophagitis loses its elastic properties. Therefore, folds at the LES level are the same origin (inflammation) as the folds in the inflamed esophagus (Figure 8).

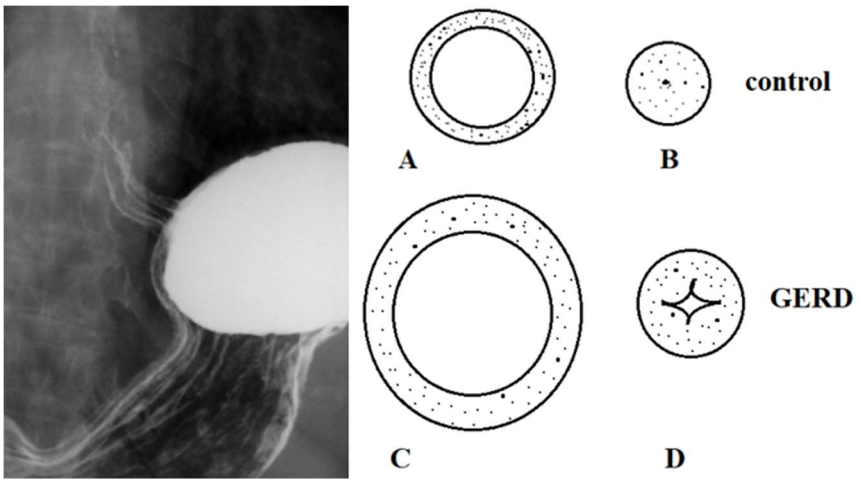


Figure 8. A gastric radiography with GEJ performed without provocation. Parallel folds above the stomach about 3 cm long were formed as a result of contraction of the sphincter. In terms of length and function (at rest) it is not a crural diaphragm. This zone cannot be a stomach, since barium has not penetrated there from the stomach. It is obvious that the folds were formed as a result of the contraction of the LES. The diagram next to the radiograph shows cross sections at the LES level in normal and GERD.

At sixth. On all radiographs, where the phrenic ampulla is fixed, including filming sections (Figures 1,2,5,8), the ampulla is formed and emptied, gradually decreasing in volume regardless of the size of the ampulla. This occurs as a result of the peristaltic wave. However, it is known that peristalsis is absent in the cardia and fundus, since there are no Cajal cells in their walls [20]. Consequently, the phrenic ampulla does not turn into a hernia if it increases in size.

Discussion. My X-ray studies and literature analysis, which are given in 6 proofs, confirm the results of endoscopic and histological studies Chandrasoma et al. Their studies reject two false dogmas that result in two widely believed fundamental errors: (1). These are the belief that cardiac epithelium normally lines the proximal stomach and (2) that the GEJ is defined by the proximal limit of rugal folds [21, 22]. From the point of view of well-established ideas, which leave many questions about the pathological physiology of the GERD

unanswered, these statements seem controversial. I am sure that this discovery opens the door to evidence-based science.

Esophageal pH-metry was initially introduced in 1969 and it was considered the gold standard for the diagnosis of GERD since the 1980s [23]. Until now is published works claiming great reliability of this method [23]. Initially, patients were selected without the typical symptoms of GERD and without signs of inflammation in the esophagus during gastroscopy to determine the normal range. As it turned out, this selection was erroneous. First, GERD for a long time can proceed without clinical manifestations. So, for example, with a screening gastroscopy examination of 6,683 healthy Koreans, 14.66% had GERD diagnosed [24]. In another study of 57 healthy subjects, 13 (23%) had an esophageal pathology in endoscopy, and 10 (17%) had an esophageal hernia [25]. Secondly, patients with GERD could be included in the group of healthy patients with endoscopy-negative reflux disease or (nonerosive reflux disease) [26,27,28]. As a proof of the erroneousness of the pH range, frequent examples of patients with GERD can be used, in which 24-hour pH-metry did not detect reflux disease, including in observations where patients needed surgical treatment [29,30]. Thirdly, as histological studies of recent years have shown, GERD begins with reflux only in the abdominal segment of the esophagus, i.e. intra-abdominal, part of the lower esophageal sphincter (LES), when the acidic gastric contents do not yet enter the esophagus, and, consequently, the disease cannot be detected using pH-metry [22].

Esophageal pH-metry was considered a standard for the diagnosis of GERD since the 1980s [23]. For more than 30 years, in all publications, without exception, pH-metry and pH-metry / impedance-24 hours were considered reliable methods for diagnosing GERD. The discussion on this topic was not allowed, about what I'm judging by the lack thereof and by my own experience. Recently, when the market for pH equipment replete and a tool for the high-resolution manometry appeared, it turned out that pH-metry is not the gold

standard for diagnosing GERD. Rosen et al. found the RI (reflux index) measured by pH-metry had a sensitivity and specificity of 50% and 82%, respectively GERD [10]. However, even these figures exaggerate the diagnostic value of pH-metry, since in a significant number of GERD patients, the clinical manifestations of the disease may be erased and not permanent.

This period left us a legacy of two serious problems: ethical and scientific.

Ethical problems:

1. In a significant number of patients, the diagnosis of GERD was not established timely, and they did not receive the necessary treatment.

2. We see that science is fading away without free discussion of scientific problems, despite the use of modern equipment and statistical processing of the data.

3. Money should not decide the direction of scientific research.

Scientific problems: Present ideas about the normal physiology of GEJ and the pathological physiology of GERD are based on the false standards defined by pH-metry.

1. The GEJ function is the prevention of gastroesophageal reflux. The reflux of aggressive gastric contents into the esophagus for 1 hour per day (4%) in adults or 2.4 hours per day (10%) in infants cannot be physiological. These data testify to the insufficiency of the function of the GEJ. This idea is also absurd, as the assumption that urine and feces incontinence several times a day is a physiological norm.

2. Transient lower esophageal sphincter relaxation indicates damage to the function of the LES.

3. The presence of phrenic ampulla is evidence of GERD, as it results from the inflammatory process that leads to the expansion of the esophagus and the weakening of the last peristaltic wave.

4. If the ampulla is wider than 2 cm, this is proof of the incompetence of the LES and esophagitis. Its is mistaken for a hernia. However, no displacement of the stomach into the chest does not occur.

(a) As it was proved above, the folds at the level of LES, due to the rigidity of the inflamed mucosa;

(b) "cardiac metaplasia of the squamous epithelium due to exposure to gastric juice results in cephalad movement of the squamo-columnar junction (SCJ)." [21];

(c) The double peak of pressure is due to the contraction of the PS and LES;

(**d)** Oral displacement of clips attached to the mucous occurs because during the formation of the ampoule area of its inner surface increases sharply. To cover this area, an additional amount of mucous is needed, which is pulled from the bottom along with the clip.

(e) The idea of shortening the esophagus during swallowing, and especially in hiatal hernia, is based on false evidence. If we consider the LES as the lower part of the esophagus, then this whole complex is shortened in GERD due to the shortening of the LES. Its abdominal segment opens, and the entire esophagus becomes 2.2 cm shorter. However, the LES is a different structure from the esophagus. It is in constant contraction around the clock, except for the moments of eating, and it does not participate in peristalsis. In this way, it resembles the internal anal sphincter, which cannot be called the rectum.

It is easy to see during fluoroscopy of the stomach that the GEJ never shifts. But if the direct observation has already become unconvincing, we can review the article by Kwiatek et al. [31].

Selection of volunteers as healthy individuals is erroneous, because the absence of symptoms does not exclude GERD. At X-ray studies, that were conducted in parallel with high resolution manometry (HRM), all radiographs presented as a study of healthy volunteers, have strong evidence of GERD: the presence of the

phrenic ampulla and the shortening of the LES due to the opening of its abdominal segment (**Figure 9)**.

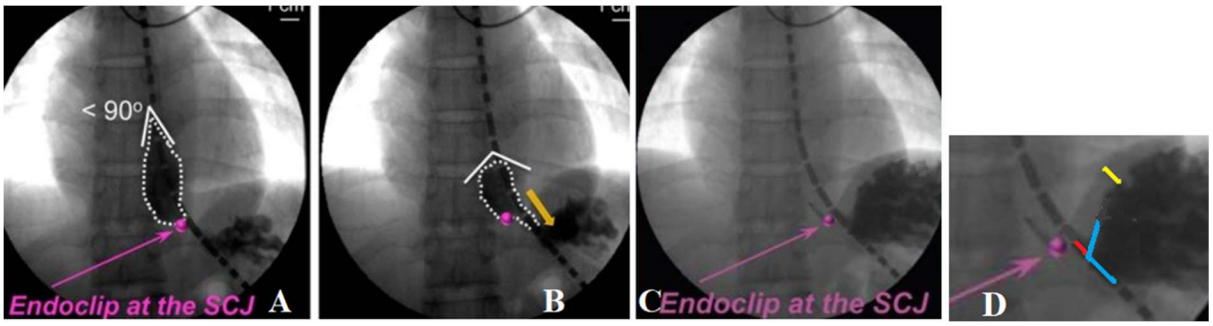


Figure 9. Radiographs from the article Kwiatek et al. [31], and scheme D to figure 1.C. In all the pictures endoclip is on the same place - to the left of the lower contour of the D-10. On Figure C, performed after numerous swallows, the pressure in the stomach increased, as evidenced by the large amount of barium in the stomach and the sharp decrease in the distance between the contour of the diaphragm and the stomach (the yellow line in the diagram). This led to the opening of the abdominal part of the LES (blue line), with the shortening of the distance between the endoclip and the stomach (the red line is the contracted part of the LES).

According to the authors, an endoclip was placed at the SCJ. If this point corresponded to the caudal border of LES displaced cranially, this would mean that the phrenic ampulla coincides with the LES. There is no doubt that the clip was attached to the proximal point of the LES. We see a sharp shortening of the LES. But there is no displacement of the GEJ and the shortening of the esophagus. The following is an analysis of a study from the article by Pandolfino et al. [32] (**Figure 10**).

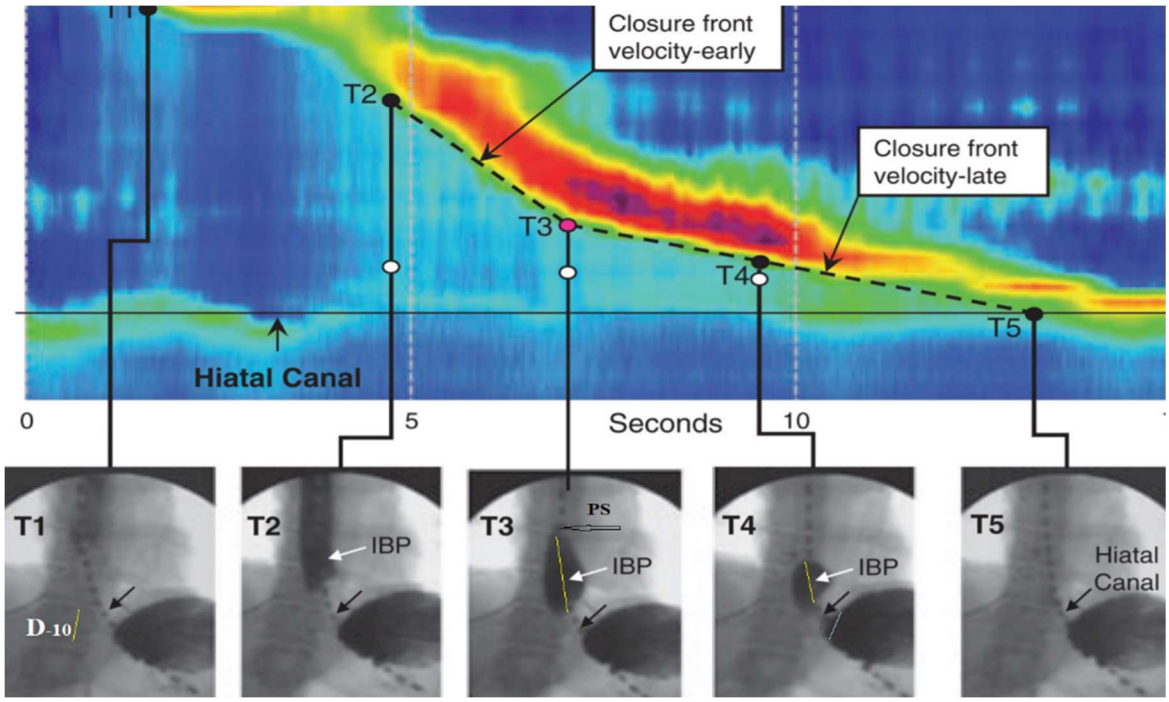


Figure 10. From the article Pandolfino et al [32]. The length of the LES (E2) is 3.2 cm. On the radiograph of T3, a phrenic ampulla appeared, as a closed cavity between the PS and the LES. The wave propagation in cranio-caudal direction causes a sharp rise in pressure (T3-red point). When the pressure reached the threshold pressure, the LES opened and the ampulla injected of barium bolus into the stomach. This is accompanied by a drop-in pressure in the ampulla. At the same time, there is a sharp shortening of the LES due to the opening of its abdominal part (the angle bounded by the blue line). Diagnosis of GERD is beyond doubt.

Figure 11 shows a diagram of the change in GEJ during the progression of GERD.

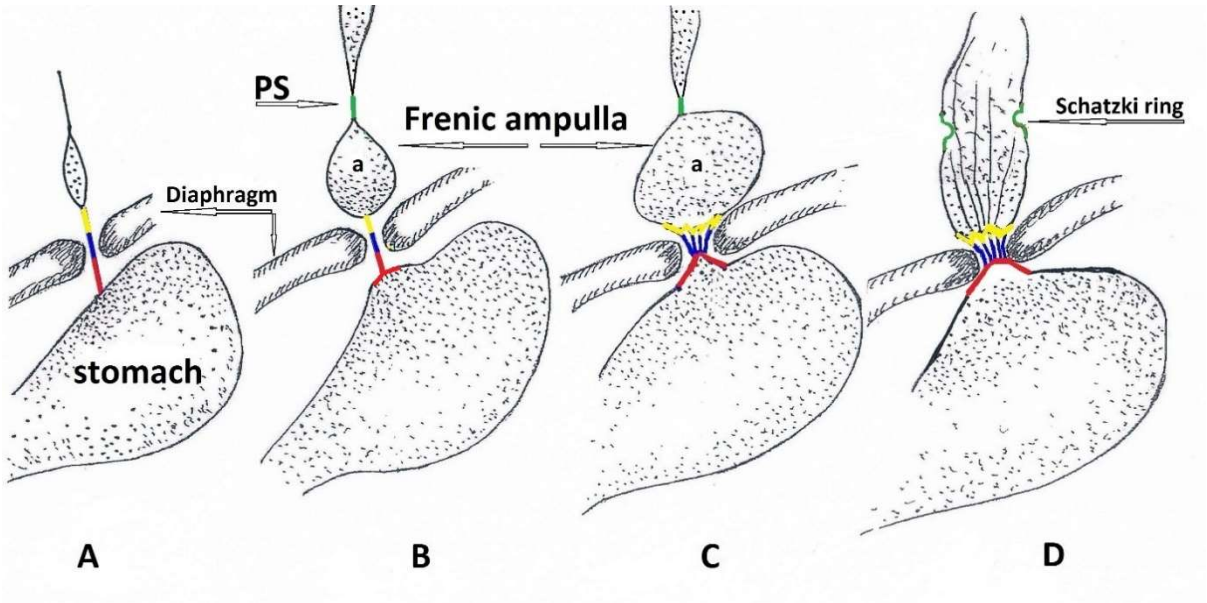


Figure 11. A diagram of the change of GEJ in the process increasing of the GERD severity. (**A**). Norm. The esophagus is not dilated, and a strong last peristaltic wave inserts the bolus into the stomach without delay, despite the compression of the abdomen. The length of the LES in the normal range. (B). The initial stage of GERD. During the compression of the abdomen there was a short-term contraction of the LES and a phrenic ampulla appeared. Perhaps a slight shortening of the LES, due to the disclosure of the distal portion of its abdominal segment (red). **(C)**. Severe GERD. Expansion of the esophagus with the formation of ampulla wider than 2-3 cm. Significant shortening of the LES during abdominal compression, widening of the hiatal canal and appearance of folds at the LES level. The proximal sphincter (PS - green) is functioning. **(D).** Short LES without the use of provocation tests. The proximal sphincter is not functioning, or in its place appears rigid fibrous ring (Schatzki ring). Symptoms of severe esophagitis.

Conclusion. An analysis of the literature suggests that GERD are diagnosed at the stage of a chronic irreversible process due to erroneous ideas about the normal and pathological anatomy and physiology of the esophagus and GEJ. Patients with the phrenic ampulla, if they do not have serious clinical symptoms, are considered healthy. What is considered to be a hiatus hernia is an ampulla of the esophagus with more severe damage of the GEJ. Therefore, the statement that hiatal hernia can be without GERD is a delusion. Due to the erroneous criteria of the norm, in a significant number of the patients with GERD this diagnosis was excluded. As a result of this, after numerous, expensive and cumbersome studies, many patients were not receiving the necessary treatment. I find it likely that the Chandrasoma research is a very important discovery. It deserves to be tested and widely discussed.

IV. Diagnosis of GERD

Recent recommendations suggest starting treatment of patients with suspected GERD based on clinical symptoms [33]. However, if the disease is not responding to treatment after eight weeks, X-rays are necessary to rule out other pathological processes mimic or predispose to GERD such as hiatal hernia,

malrotation, pyloric stenosis, duodenal web, duodenal stenosis, antral web, esophageal narrowing, Schatzki's ring, achalasia, esophageal stricture, and esophageal extrinsic compression in children [10], and gastric outlet obstruction, duodenal dyskinesia or gastroparesis in adults [33]. In the middle of the 20th century, X-ray examination was the main method of instrumental diagnosis of GERD. The spontaneous penetration of barium from the stomach into the esophagus was the proof of this pathology. This method was poorly correlated with the clinical manifestations of the disease. In a significant number of patients with a typical picture of GERD, reflux was not detected. A water-siphon test was proposed to increase the accuracy of the radiographic examination. After drinking barium, the patient, while in a horizontal position, drank water through a straw from a glass located at his head. This method proved to be highly sensitive because it detects reflux in 95% of children with positive pH-metry [34]. But it was little specific in comparison with pH monitoring [35].

I propose a new method of X-ray diagnosis, which is based on the regularities of the normal and pathological physiology of GEJ. It simultaneously uses three provocative test: water-siphon test (drinking barium in a horizontal position); provocation by gastric distension (> 200 ml of barium in adults), as well as compression of the abdomen. This method allows you to detect and measure the LES and evaluate the function of the esophagus and GEJ in comparison with the standards described above. I propose to use this method in cases where x-rays are used to rule out other diseases. Provocative tests are applied simultaneously and do not increase the exposure of ionizing radiation.

X-ray examination. Standard X-ray examination of the upper parts of the digestive tract is carried out in a horizontal position. The patient lies on his back and drinking barium through a straw from a can that was near his head. After drinking about 200 ml (out of 250), during the last sips, the abdominal wall is compressed by the radiologist hand for 30 seconds. Several radiographs are produced during the study. Babies drink a barium suspension from a bottle with

a pacifier. Abdominal compression causes an increase an intra-abdominal and intragastric pressure. The level of pressure in the stomach does not depend on the strength of the compression by the hand, but on the reactive contraction of the anterior abdominal wall.

X-ray analysis. The length of the gap without barium is a length of the contracted LES. On the radiographs, we measured the length of the LES and the width of the supra-diaphragmatic esophagus. On radiograph all objects are magnified in proportion to the distance between the object (LES) and the place of registration of the image (film). The true parameters were calculated using the formula:

d = D x k; where "d" - true size; "D" -its size on radiograph; "k" is the coefficient of magnified, which is the ratio of the true height of the first lumbar vertebra to its image on the radiograph. (The true height of L-1 in adults is 2.3 cm). The results of measuring LES in patients of different ages are shown in Table 1 (From page 6).

Table 1. Normal length of LES (см) in different age groups.

Age	Up to a year	1-3 years	4-7 years	8-10 years	11-15 years	2 – 65 years
Limits	0.7 – 1.0	1.2 – 1.5	1.5– 1.8	1.9 – 2.3	2.3 – 2.9	3.2 -4.2
Average	0.86±0.03	1.40±0.02	1.72±0.07	2.10±0.05	2.45±0.11	3.60±0.08

The normal width of the esophagus was the same throughout and did not exceed 1.2 cm in children and 1.5 cm in adults.

Patients with GERD revealed a large range of pathological symptoms. Based on radiological symptoms, it is possible to differentiate the degree of the GEJ dysfunction. In a minimal degree, the LES is contracted in response to compression of the abdomen, and its length is within the age norm. Moderate dysfunction of the GEJ is expressed in shortening of the LES relative to the norm due to the expansion of its intra-abdominal segment. In significant weakness of

GEJ the length of the LES is 2 times shorter than normal. Free reflux of barium from the stomach into the esophagus is evidence of GEJ incompetence.

A complete list of radiological symptoms of GERD can be divided into three groups (**Table 2**).

Table 2. Radiographic symptoms of GERD.

Fluoroscopic symptoms	LES changes	Esophageal changes
LES contraction during abdominal compression	Shortening of the LES with angular opening of its abdominal part (Figure 2)	Expansion of the esophagus more than 1.2 cm in children and 1.5 cm in adults
Provoked reflux during abdominal compression	Shortening of the LES almost twice with respect to the age norm (Figure 3)	Slow and / or incomplete evacuation of barium from the esophagus
Free reflux from the stomach to the esophagus	Longitudinal folds at the level of the LES (Figure 4)	Picture of the phrenic ampulla and proximal sphincter (Figure 4)

In all children who had at least one of the typical clinical symptoms of GERD, the diagnosis was confirmed by X-ray examination. The GERD was established in 91 (98%) of 93 elderly patients. The length of the LES in them was significantly less than the normal {1.96±0.19 cm vs 3.60±0.08 ($p < 0.001$)}. The length of the LES was inversely proportional to the width of the ampulla. In 64 patients with the ampulla width < 2 cm (1.56 ± 0.04 cm), the LES length was 2.21 ± 0.14 cm, and in 27 patients with the ampulla width ≥ 2 cm (2.75±0.09 cm) the LES length was 1.60 ± 0.16 cm ($p < 0.01$).

V. Treatment

From this review, the cause of GERD in children and adults is the incompetence of the GEJ function. The pathological process begins with reflux into the abdominal segment of the LES, when treatment can prevent the progression of the disease and lead to a full recovery. What used to be considered pathogenetic factors (hiatal hernia, Schatzki ring, transient lower esophageal sphincter relaxation, impaired esophageal clearance) are symptoms of the chronic and irreversible GERD. In such cases, treatment is aimed at controlling the symptoms and preventing further progression. Currently, there is no single research method

that would diagnose GERD at an early stage. Chandrasoma research is of great theoretical value. However, it is impossible to imagine that a histological examination was carried out for persons without clinical manifestations of GERD or with minimal symptoms.

1. An indication for treatment is a combination of a recurring at least one typical symptom of GERD with at least one risk factor.

In children, the **clinical manifestations** of GERD can be divided into esophageal and extra-esophageal symptoms. Esophageal symptoms include vomiting, poor weight gain, dysphagia, pain in the abdomen or chest (the fussy infant), putrid breath, wet pillow due to hypersecretion of saliva, and anemia, cough after eating, which are based on esophagitis. Extra-esophageal symptoms include cough, laryngitis, asthma and dental erosion, most of which are pharyngitis, sinusitis, idiopathic pulmonary fibrosis and recurrent otitis [36]. Adults may have the same symptoms, but heartburn, regurgitation, and pain during swallowing or in the abdomen predominate.

Among the **risk factors** associated with reflux symptoms:

1. Persons born prematurely;
2. Long vomiting in the first six months of life;
3. Cow's milk intolerance;
4. Intolerance to other products or allergy;
5.Hypersecretion of hydrochloric acid (gastritis, gastric ulcer);
6. Overeating (obesity);
7. Stress.
8. Cases of GERD in the family

This applies to both children and adults, because GERD of adults most often begins in childhood.

2. Treatment:

(a) Detection and exclusion of risk factors.

(b) Exclusion of provoking factors.

- small amounts of a single meal;
- to take a horizontal position (to sleep) with an empty stomach;
- do not squeeze the abdomen with a belt;
- do not bend over after eating;
- treatment of constipation;
- antihypertensive drugs are preferably taken in the morning.

(c) Drug treatment is well covered in the literature. I believe the statement by Kellerman and Kintanar that "GERD is a clinical diagnosis and is most effectively treated with proton-pump inhibitors (PPIs)" [37] is true. The disappearance or relief of symptoms after 1-2 weeks of GERD treatment confirms this diagnosis. Continuous treatment of 1-2 tablets per day for 3-4 weeks is enough to eliminate the inflammatory process in the esophagus. After this, can apply short courses in exacerbation or violation of the rules of conduct. "Long-term use of PPIs is associated with bone fractures, chronic renal disease, acute renal disease, community-acquired pneumonia, and Clostridium difficile intestinal infection" [37].

(e) The theoretical rationale for new surgical tactics in GERD.

LES is a functional structure that is fundamentally different from the esophagus. It, like the internal anal sphincter, is in a closed state all day and does not participate in peristalsis. Its ability to continuously contract can be explained by the fact that different groups of circular fibers contract at different time intervals. When one group of fibers relaxes to restore its contractile potential, another group that maintains the tone of the LES is contracted. LES relaxes in response to increased pressure in the esophagus above the LES to a threshold level. It also reflexively contracts in response to pressure into the stomach.

With GERD, the tone of the LES is reduced and sphincter becomes shorter as a result of the dilation of its abdominal part up to 2.1 cm long. The degree of weakness, as well as the degree dilation of the abdominal segment of the LES in

the course of disease progression, increases from the minimum to the maximum along the exponential curve. In some cases, without provocation, its length may be within normal limits, but it opens with increasing pressure in the abdomen (**see Figures. 1 and 7**). In other cases, it is only partially disclosed (**Figure 12.B**), and in severe cases - completely (**Figure 12, C**).

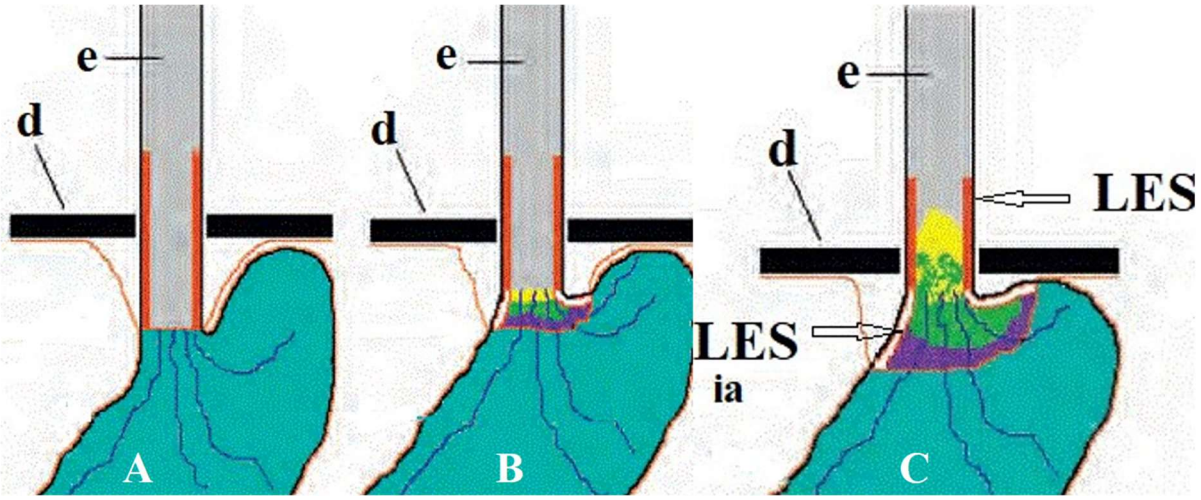


Figure 12. Scheme of progression of weakness of the LES in GERD. (A) Norm. (B) GERD - part of the abdominal segment of the LES as a result of inflammation disclosed, and its wall is a continuation of the stomach wall. (C) GERD - all abdominal segment of LES dilated and does not perform anti reflux function. d-diaphragm; e - esophagus; LES ia - intraabdominal segment of the LES (white).

Thus, with GERD, an extended and weak LES does not move into the chest. Although its abdominal cone-shaped segment is called the cardiac part of the stomach, it is a distended LES (**Figure 13**).

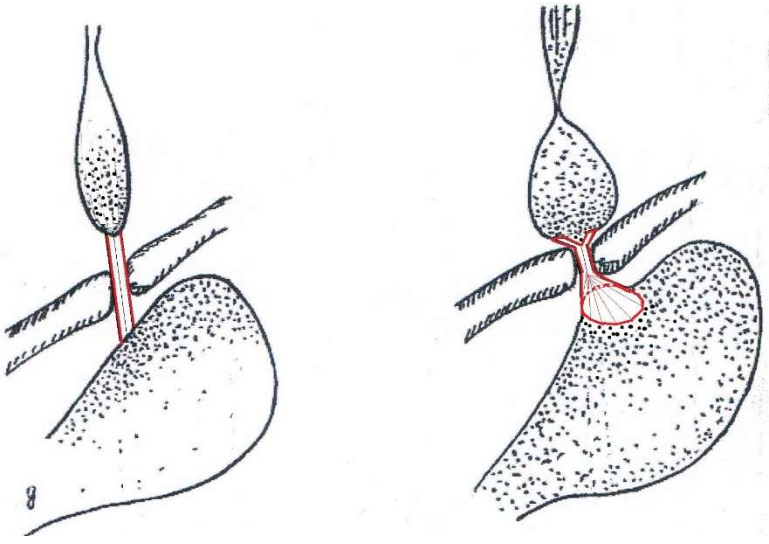


Figure 13. Diagram of the dilatation of the abdominal segment of the LES in GERD. (A) Norm. (B) Cone-like stretching of the LES in GERD.

Given that the LES remains in its place at GERD, I suggest restoring the function of the LES by plication the wall of the cardiac part of the stomach, which is the abdominal segment of the LES. This achieves two goals: a) mechanical narrowing; b) an increase in tone due to the thickening of its wall of the LES and restoration of its function. Mittal et al. showed in an experiment that surgical plication of the external anal sphincter increases the length of its sarcomere and causes an increase in pressure in the anal canal [38]. The cardioplication scheme is shown in **Figure 14**.

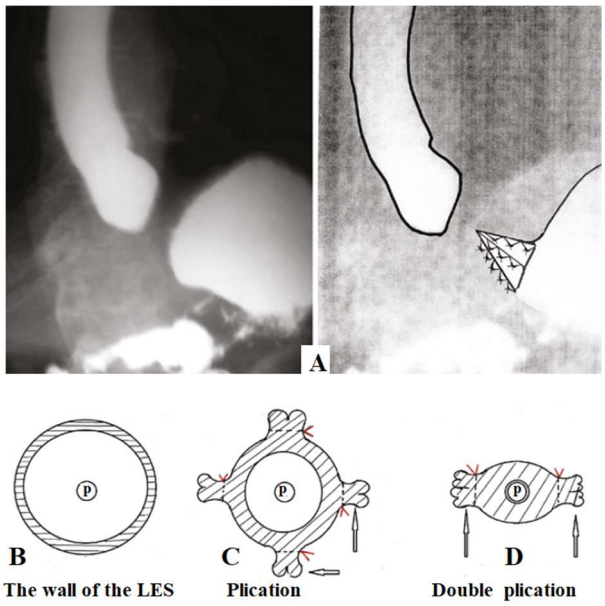


Figure 14. Scheme of the surgery with LES plication in GERD.
A) Radiograph of the GEJ, and the scheme surgery with LES plication - 3 cm below diaphragm; B - D) cross sections. B - at the level of the abdominal section of the LES before the operation; C - plication in four (three) areas, resulting narrowing of the lumen; As a possible option: D - doubling of plications, leading narrowing the lumen to the diameter of the gastric probe (p).

This method was first published in 2017 [39] but was not applied in practice.

The operation can be simplified by using a cardioplicator. Before applying the cardioplicator, it is necessary to damage the peritoneum so that the folds of the LES collected under the cardioplicator grow together. A synthetic cone, selected for each age, squeezes the abdominal segment of the LES. To develop the method, animal studies are needed (**Figure15**).

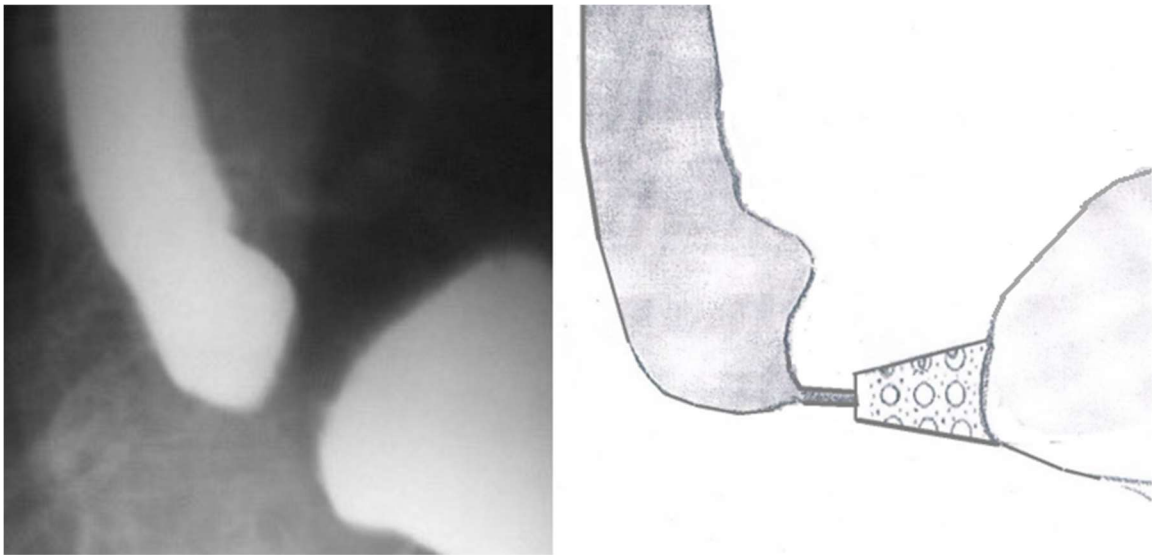


Figure 15. The method of lengthening and enhancing the function of the abdominal segment of the LES using a cardioplicator.

respectfully

Michael Levin

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