Schatzki's ring and peptic stricture in the lower esophagus.

Pathophysiology and differential diagnosis.

I. **Introduction.** Schatzki was not first who reported a ring-like structure in patients with dysphagia. Однако с 1953 по 1963 гг он с соавторами изучал эту проблему. They showed that dysphagia occurs with a narrowing diameter of 13 mm. Schatzki ring (SR) is diagnosed from 0.2% to 18% of all patients having routine upper gastrointestinal examinations. There is still no uniform agreement as to its exact location, etiology, or clinical importance [1]. Some authors believe that the SR ring is a submucosal, fibrotic thickening located at the gastroesophageal junction [2]. To understand the origin of SR, researchers determine what problems this pathology combines with. Towbin et al found SC in 25 children (0.2% of all patients). In 24 patients (96%) had hiatal hernia (HH), 10/25 (40%) had eosinophilic esophagitis (EE), and 10/25 (40%) had GERD [3]. In adults, one hundred and sixty-two (97%) patients showed a sliding hiatal hernia. Erosive reflux esophagitis was found in 47 (28.1%) patients. Twenty-six (15.6%) of 167 patients showed single or multiple esophageal webs; five (3.0%) patients exhibited EE; and four (2.4%) had esophageal diverticula. Four (7%) of 57 patients undergoing esophageal manometry had non-specific esophageal motility disorders [4]. Other studies emphasize the combination of SR with EE. Müller et al diagnosis of EE was made in 11 (9%) patients with ≥ 20 eosinophils / hpf. Patients with EE were more likely to have a history of allergies (73% vs 29%, P = 0.007). The criteria for active EE were defined as: (1) eosinophilic tissue infiltration ≥ 20 eosinophils/hpf; (2) symptoms of esophageal dysfunction; and (3) exclusion of other causes of esophageal eosinophilia. Gastroesophageal reflux disease was excluded by proton pump inhibitor treatment prior to endoscopy [5]. In another study, by histologic criteria, two groups of children were defined. Eight had clinical and histologic criteria of EE and 10 of peptic esophagitis. There were no

differences in the symptoms or radiographic findings in the two groups. Those with peptic esophagitis had a significantly higher acid exposure than did those with EE (12.6 ± 2.9 v 2.0 ± 1.1%; $P < 0.01$) by esophageal pH probe. Patients with peptic esophagitis responded to proton pump inhibitors and/or dilatation, whereas those with EE did not have a good response and required specific therapy for EE [6]. Thus, the etiology, and pathogenesis of the SR have remained obscure, and little is known about their association with other structural and functional abnormalities of the esophagus. Theories about their origin include congenital, anatomical, and inflammatory factors as the most likely events that lead to a circular constriction of the esophagogastric junction [4]. In a previous study, we concluded that SR occurs in patients with GERD at the site of the functional sphincter (proximal sphincter), where its contraction closes the cranial part of the phrenic ampulla during ampullary contraction and evacuation of the bolus into the stomach [7]. Our hypothesis contradicts generally accepted ideas about the pathogenesis of SR. This leads to the fact that our articles on this subject are rejected by the editors of journals without any justification. The purpose of this work-study the pathological physiology and pathogenesis of SR by analyzing articles published in PubMed.

II. Material and method. We selected 38 articles on the examination of patients with SR, which were published in PubMed. Among them were 21 articles that were published in the open access with radiographs. Radiographs were analyzed by comparing them with radiographs of normal individuals as well as patients with GERD.

III. 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 positions. 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, because 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 LES does not differentiate. Provocative tests, including compression of the abdomen or deep breath, in healthy did not affect the speed of cleansing the esophagus. 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 lead to a reflex sphincter contraction (**Figure 1**).

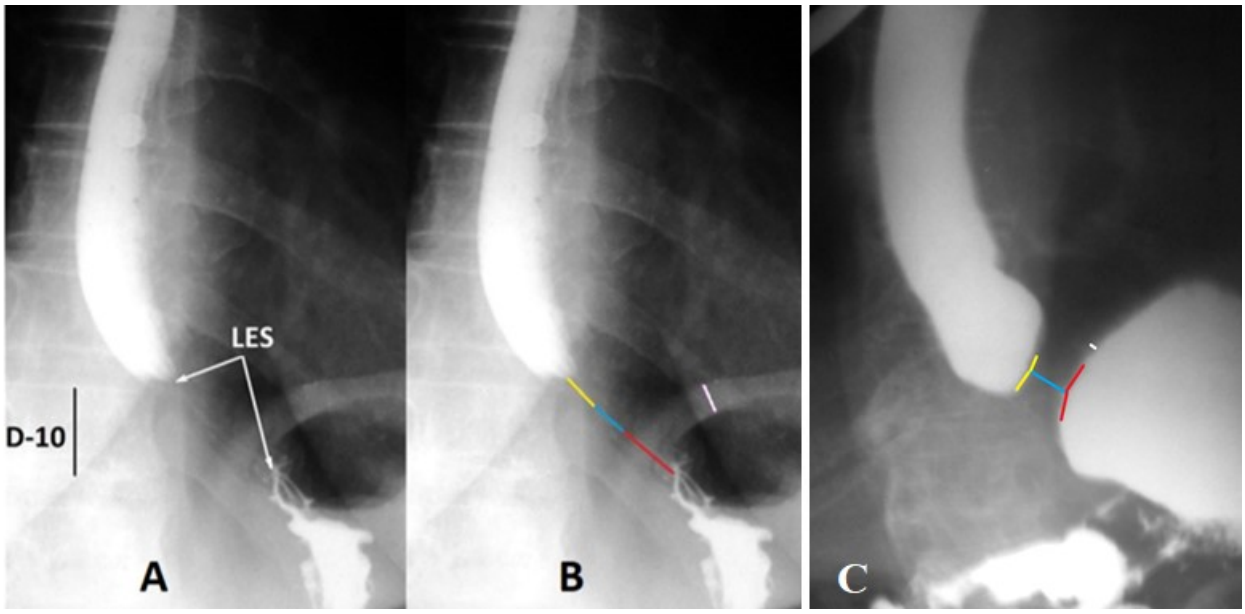

Figure 1. (**A)** Radiograph of a GEJ in the vertical position of an adult with GERD. The abdomen compression during barium swallowing caused a contraction of the LES. (**B)** The same radiograph for analysis. Since it is known that the height of the 10th thoracic vertebra (D-10) is ≈2 cm, we calculated 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 abdominal portion of the LES (1.3 cm versus 2.1 cm). The image shows 2 folds of the open portion of the abdominal portion of the LES caudally to redline. (C) In a horizontal position, after taking a large amount of barium during abdominal compression, the pressure in the stomach increased (stomach wall thickness decreased), which led to an additional opening of the LES. The length of the gap

between the barium in the esophagus and in the stomach at the level of the diaphragm is 1 cm.

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**). Thus, in a healthy person, it is impossible to cause a contraction of LES and visualize it in a vertical position.

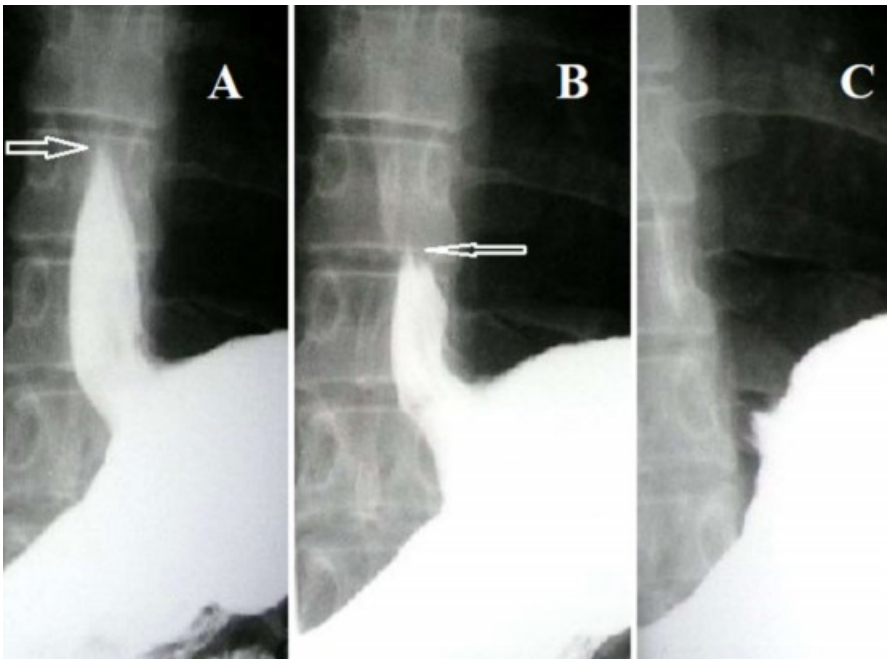

Figure 2. Elderly patient with GERD. A series of pictures taken during the reception of barium without provocative tests. It is known that at rest the pressure in the lower esophagus is lower than in the stomach. For the bolus to penetrate the stomach during the opening of the LES, the last peristaltic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

In the horizontal position 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 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 [8]. 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).**

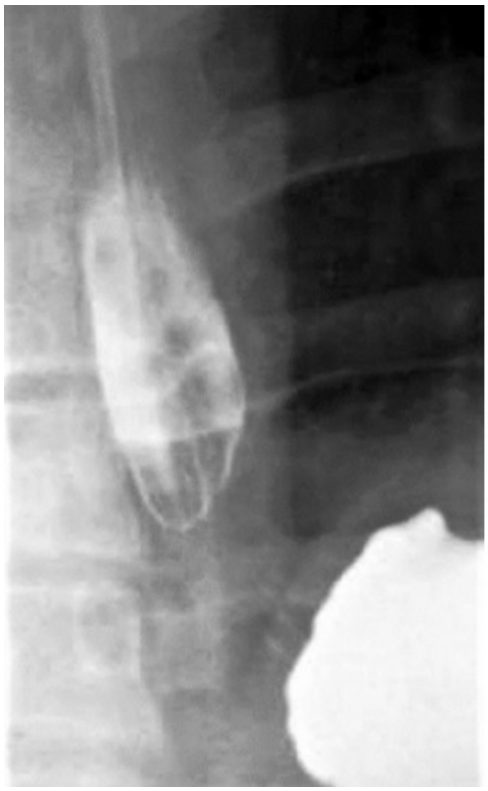

Figure 3. During the abdominal compression, a gap without contrast agent was formed between barium in the esophagus and in the stomach. It is the contracted LES.

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 the LES length in healthy adults measured by the manometric method (3.82±0.953 cm), (3.5±0.4 cm), (3.4±0.9 cm), 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 [8].

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

In healthy 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.

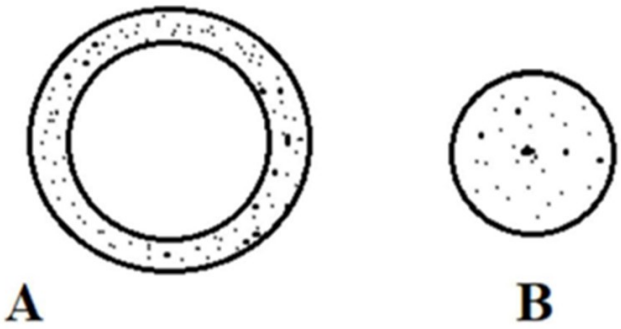

Figure 4. A diagram of the LES cross section during the bolus passage (A) and after its contraction (B).

VI. Pathological physiology of the GEJ in GERD.

It is known that the inflammatory process in GERD leads to the expansion of the esophageal lumen. This also applies to the LES. The wider the lumen of the esophagus, the weaker the peristaltic wave. This symptom can be identified in the earlier stages of the pathological process if to use abdominal compression. An

increase in the gastric pressure causes a reflex 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. A weak peristaltic wave is not able to overcome the tone of the contacted LES, which allows us to fix this moment and measure the length of the LES. Since the pressure in the stomach is higher than the resting pressure in the esophagus, to evacuate the bolus into the stomach, it is necessary to create a high pressure so that it corresponds to the threshold pressure for the opening of the LES and which should be higher than the pressure in the stomach. Normally, the last peristaltic wave creates this pressure. In patients with GERD, in whom the strength of the peristaltic wave is weakened, this pressure is created by the phrenic ampulla. It closes with a proximal sphincter(PS), as a result of which, at some point, high pressure arises between the LES and PS, which leads to the opening of the LES and a contracting ampulla injects a bolus into the stomach (**Figure 5).**

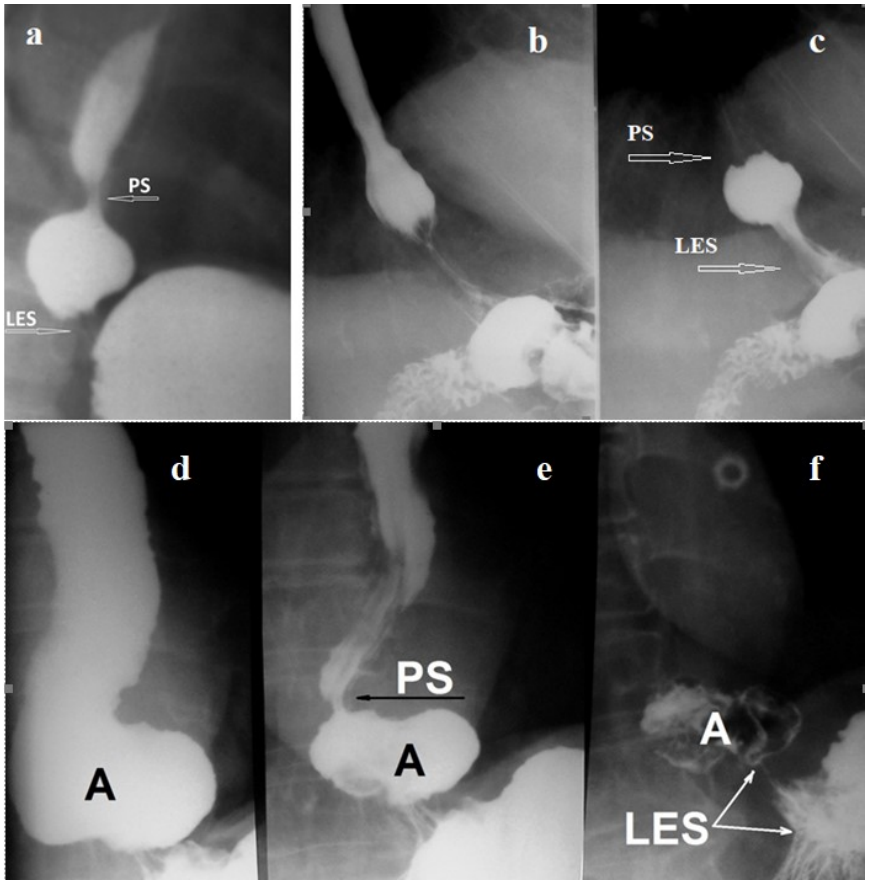

Figure 5. Radiographs of GEJ during compression of the abdomen. **(a)** A 5-year old child with symptoms of GERD. The compression of the abdomen during the swallowing of barium caused a contraction of the LES. The last peristaltic wave formed a closed chamber (phrenic ampulla), which was closed proximally by a proximal sphincter (PS). (**b-c)** The phrenic ampulla during contraction creates high pressure to open the LES. (**d-f)** A series of radiographs of the elderly patient with GERD (**d**) During abdominal compression, LES contracted and barium filled the wide ampulla and esophagus. (e) After the end of the abdominal compression, ampulla (A) injects a barium into the stomach. This has been made possible thanks to a contraction of PS. (**f**) The shortening of LES is due to the incompetence of the abdominal part of LES that appears as angular deformity (opening) of the cardia.

The literature is dominated by an unreasonable opinion that the expansion of the intestine over the diaphragm to a width of 2 cm is a phrenic ampulla, and more than 2 cm is hiatal hernia (HH). The fact that HH is found only in a horizontal position is explained by the fact that only in a horizontal position the stomach glides into the chest. Therefore, HH has a different name - sliding hernia. I propose to dwell briefly on all evidence of the absence of HH. You can find more detailed links to sources here [8].

1. The presence of rugal folds above the diaphragm is considered folds of the stomach. Meanwhile, the inflammatory process in the esophagus and the LES leads to the expansion and rigidity of their walls, because of which rough folds form during the contraction of the LES (**Figure 6**).

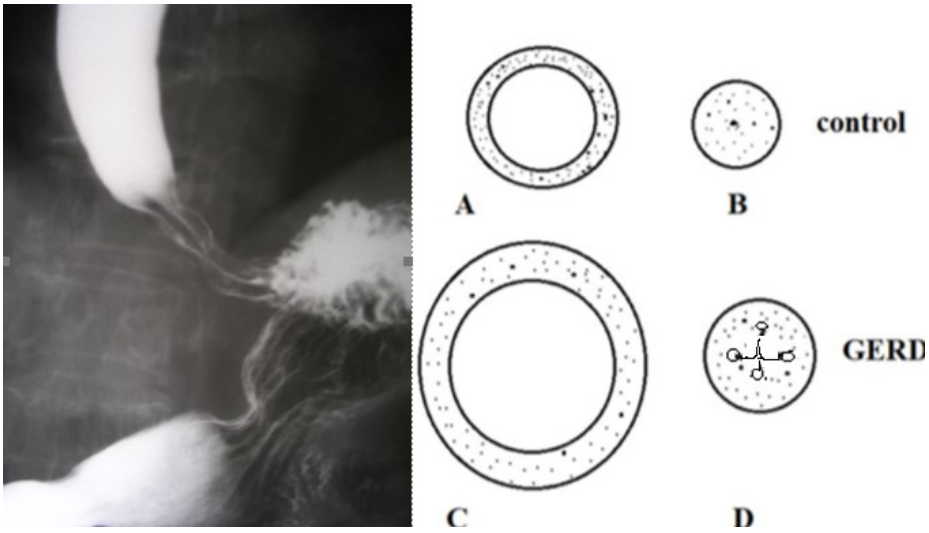

Figure 6. A radiography patient with GERD. Parallel folds above the stomach about 3 cm long were formed because of the contraction of the stretched LES. The diagram next to the radiograph shows cross-sections at the LES level in normal and GERD. Expanded and inflamed walls during a contraction of the LES form folds in which barium is retained.

2. "There is the false dogma that the cardiac epithelium normally lines the proximal stomach. Cardiac metaplasia of the squamous epithelium due to exposure to gastric juice results in the cephalad movement of the squamocolumnar junction (and not the stomach). This creates the squamo-oxyntic gap and the dilated distal esophagus, which is distal to the endoscopic GEJ" [9].

3. The double peak of pressure is due to the contraction of the PS and LES, not LES and diaphragmatic crura.

4. 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.

5. 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 in GERD this whole complex is shortened due to the shortening of the LES. Its abdominal segment opens, and the LES and whole complex becomes 2.2 cm shorter. No movement of the stomach, LES, and esophagus occurs.

Thus, the expansion of the intestine over the diaphragm, which is commonly believed to be HH, is a phrenic ampulla, regardless of size, and it is evidence of GERD.

V. Results of analysis of radiographs with lower esophageal ring.

Results We analyze 21 radiographs from the articles published in open access in PubMed, where it was possible to measure the distance from the PS to the LES, as well as the diameter and length of the PS. We proceeded from the classic definition of SR: a Schatzki ring is a submucosal, fibrotic thickening located at the gastroesophageal junction [2]. The narrowing is caused by submucosal fibrosis. The overlying mucosa is intact, and the underlying muscle is normal. The narrowing may slowly progress. Hiatal hernia is usually present [10]. The diagnosis of SR is based on the presence of a fixed, symmetric, thin (< 4 mm) structure, which intersects the esophagus perpendicular to its long axis, at the squamocolumnar junction [5]. Only X-ray data of 5 (24%) radiographs corresponded to this definition of SR (**Figure 7**).

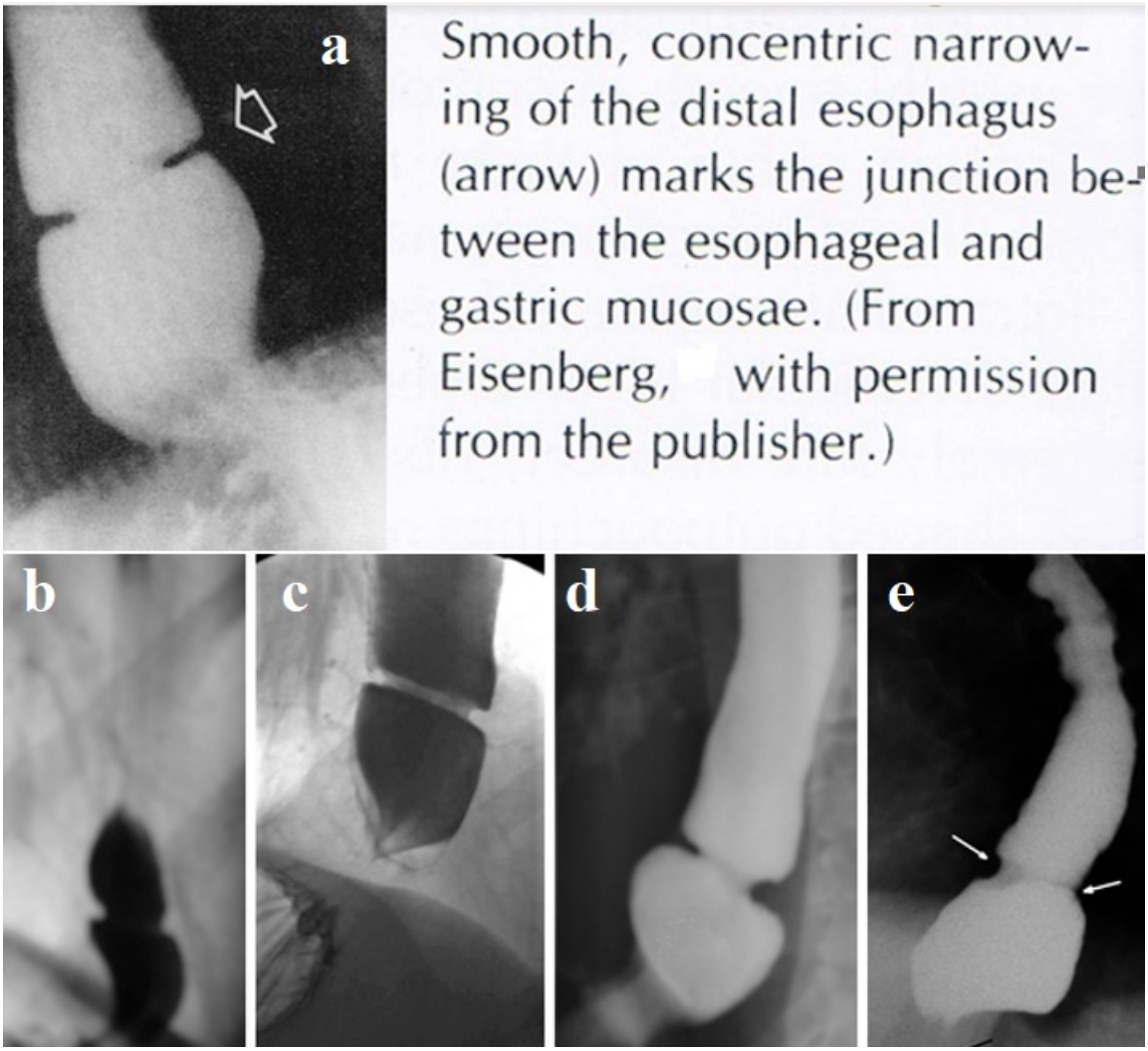

Figure 7. Five radiographs with lower esophageal ring. **(A-C)** correspond to the description of SR. (D-E) These were 2 (9%) cases recognized as doubtful due to asymmetry in the thickness of the ring and phrenic ampulla.

In all patients with SR, the size of the phrenic ampulla seems to be the same. Its length from PS to the LEC is greater than the width. However, it is impossible to judge the exact size due to the absence of markers with known lengths on radiographs According to rough estimates, on all radiographs the ampulla is about 3 cm long. On all radiographs, the LES was almost 2 times shorter than normal.

In 14 (67%) cases the esophageal stenosis was in the lower esophagus, but the radiological symptoms did not correspond to the classical description of SR. The length of the expanded segment of the esophagus between the NPS and the narrowing ranged from 4.5 cm to 1.5 cm. In cases with a short segment, its width was greater than the length. The narrowing of the esophagus was asymmetric and often with a thickness more 4 mm. As a rule, these were elderly patients with an average age of 60 years with radiological, clinical, and endoscopic symptoms of GERD. The following is an analysis of such cases.

Case 1. Figure 8 shows a study of remarkable quality from an article by Novak et al, where it is known that the width of the "SR" (**Figure 8 C**) is 10 мм, and after treatment, it increased to 15 mm (**Figure 8 D**) [11].

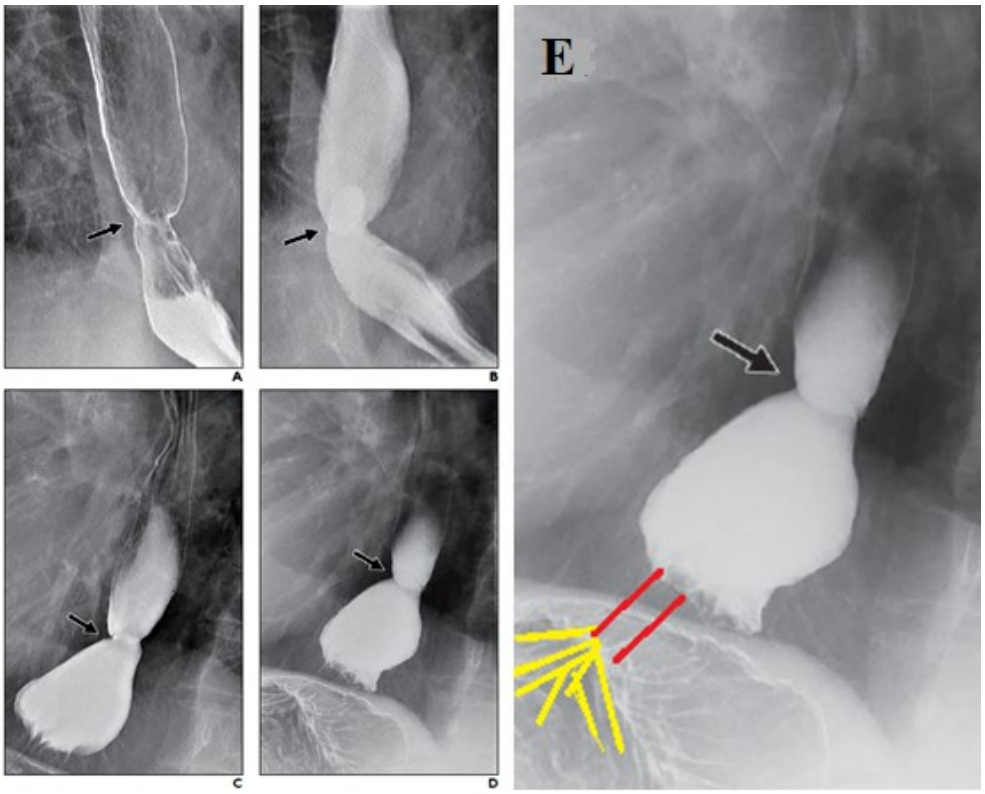

Figure 8. (A-D) A 77-year-old man with dysphagia treated with omeprazole for 24 months. (E) Scheme to figure 8.D.

On radiographs A-B performed in an upright position, barium freely penetrates the stomach through the open LES. In Figure A, barium continues to exit the esophagus, despite low hydrostatic pressure. This is evidenced by the liquid level near the LES. The esophagus over the narrowing (black arrow) is significantly expanded (2.7 cm). The folds are determined at the level of the LES. In a horizontal position (C-D), the expansion of the phrenic ampulla is visible. The rigid ring with a diameter of 10 mm before treatment (C) and 15 mm after treatment (D) is 4.5 cm from the upper edge of the LES. The thickness of the narrowing is difficult to determine, but it does not exceed 2 mm. The PS does not function and does not overlap the proximal lumen of the ampoule. Therefore, the ampoule cannot create a threshold pressure for the disclosure of the LES. The length of the contracted LES is 2 cm (red lines). Inside the stomach, a fan-shaped divergence of the folds of the open abdominal part of the LES (yellow lines) is visible, the length of which is usually 2.1-2.3 cm. Conclusion.

Patient with GERD has a sharp shortening of the LES and obvious symptoms of reflux esophagitis. The narrowing at the lower esophagus is asymmetric. The asymmetry of the narrowing and the phrenic ampulla, as well as the effect of PPI treatment, indicate stenosis due to peptic ulcer of the esophagus. The fibrous ring of SR cannot change in diameter under the drug treatment.

Important observation: In a vertical position, the LES opens under the hydrostatic pressure of the column with the level of liquid (mass) in the upper part of the esophagus. As picture 8A shows, the LES remains open until the entire bolus penetrates the stomach. Two conclusions follow from this: (1) the opening of the LES is a reflex act; (2) the opening of the LES is not only relaxation of some muscle fibers, but also an active contraction of others.

Case 2. A 69-year-old man with a history of dysphagia and esophageal food impaction who was treated with omeprazole for 2 years (**Figure 9**) [11].

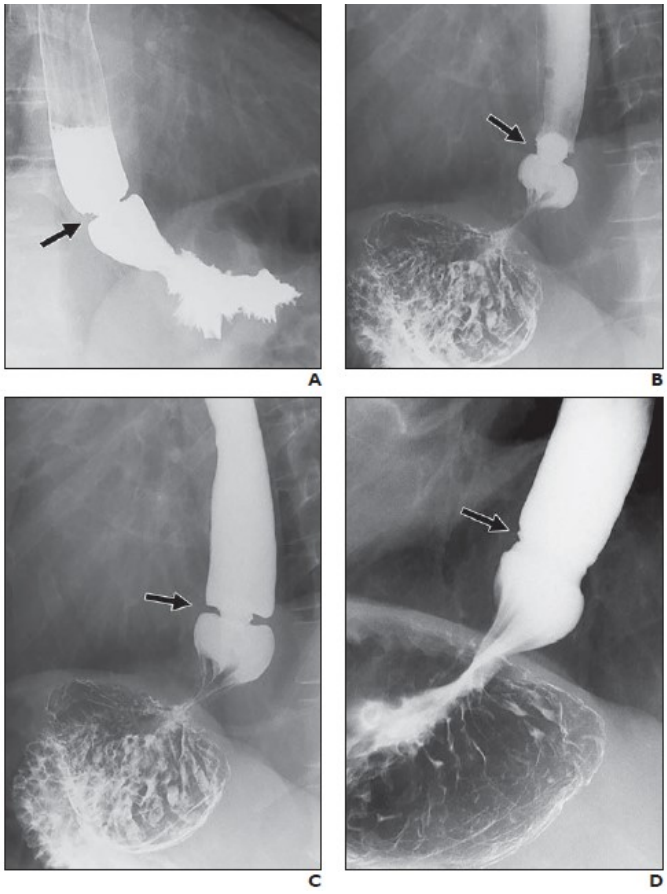

Figure 9. (**B-C**) Pretreatment esophagram reveals a 13- mm barium tablet (arrow) lodged at the level of the ring. The length of the deformed ampoule (1.9 cm) is less than its width. The length of the LES is 2 cm. (D) Posttreatment esophagram reveals significantly improved ring (arrow) lumen diameter at 25 mm.

Conclusion: In a patient with GERD, a concentric narrowing of the esophagus occurred in the phrenic ampulla distal to the proximal sphincter. A significant expansion of the narrow zone after PPI treatment indicates the peptic nature of stenosis.

Case 3 from article Novak et al (**Figure 10**) [11].

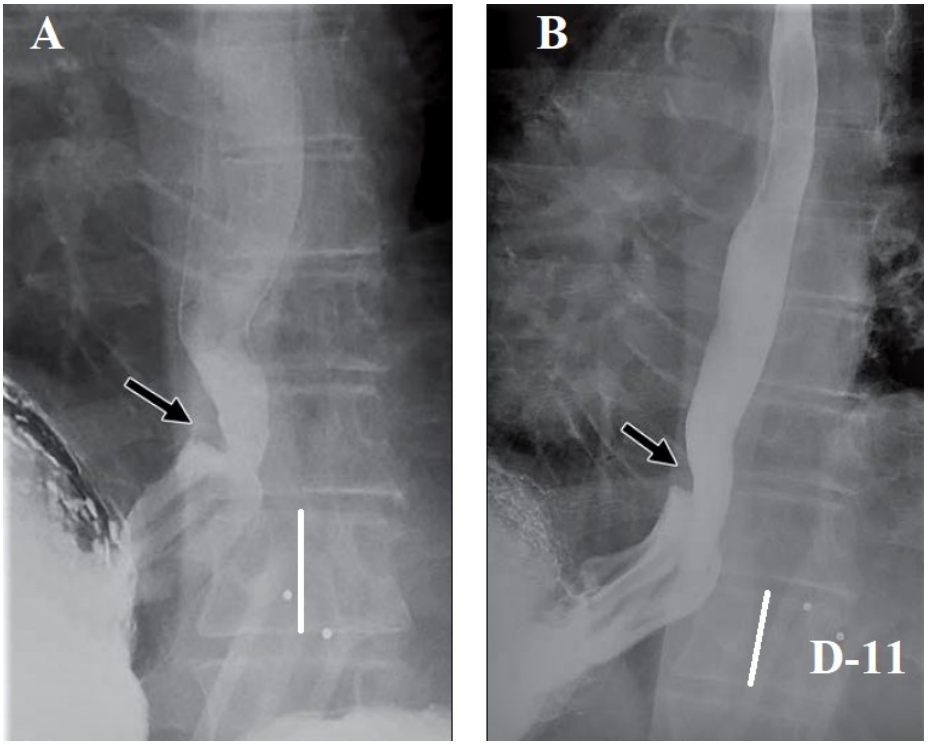

Figure 10. A 76-year-old man with dysphagia treated with omeprazole for 4 months. (A) Pretreatment esophagram. Tablet paused briefly above the ring. (B) Posttreatment esophagram reveals significantly improved ring (arrow) lumen to diameter at 13 mm. Tablet passed easily into the stomach.

As can be seen from the height of D-11, radiographs were taken with different projection magnifications. The ratio B / A = 0.8. Since it is known that the height of L-1 in adults is 2.2 cm [12], the height of D-11 is approximately 2 cm. Simple calculations show that the true width of the "narrowing" of the esophagus indicated by the arrow before treatment (A) is 6 mm, and after treatment (B) - 8 mm. This supposedly "narrowing" of the esophagus is located 2 cm from the stomach, that is,

above the LES, which is 2 times shorter than normal LES. Very thick folds indicate an inflammatory process. After treatment (B), the folds of the mucosa become much thinner. Passing through this “narrowing” of a tablet 13 mm in diameter indicates the elasticity of the esophageal wall and proves that there is no concentric narrowing. Conclusion: In a patient with GERD, dysphagia was caused by an exacerbation of the inflammatory process mainly at the level of LES. There are no signs of a concentric narrowing, both before and after treatment.

Case 4 from article Novak et al **(Figure 11)** [11].

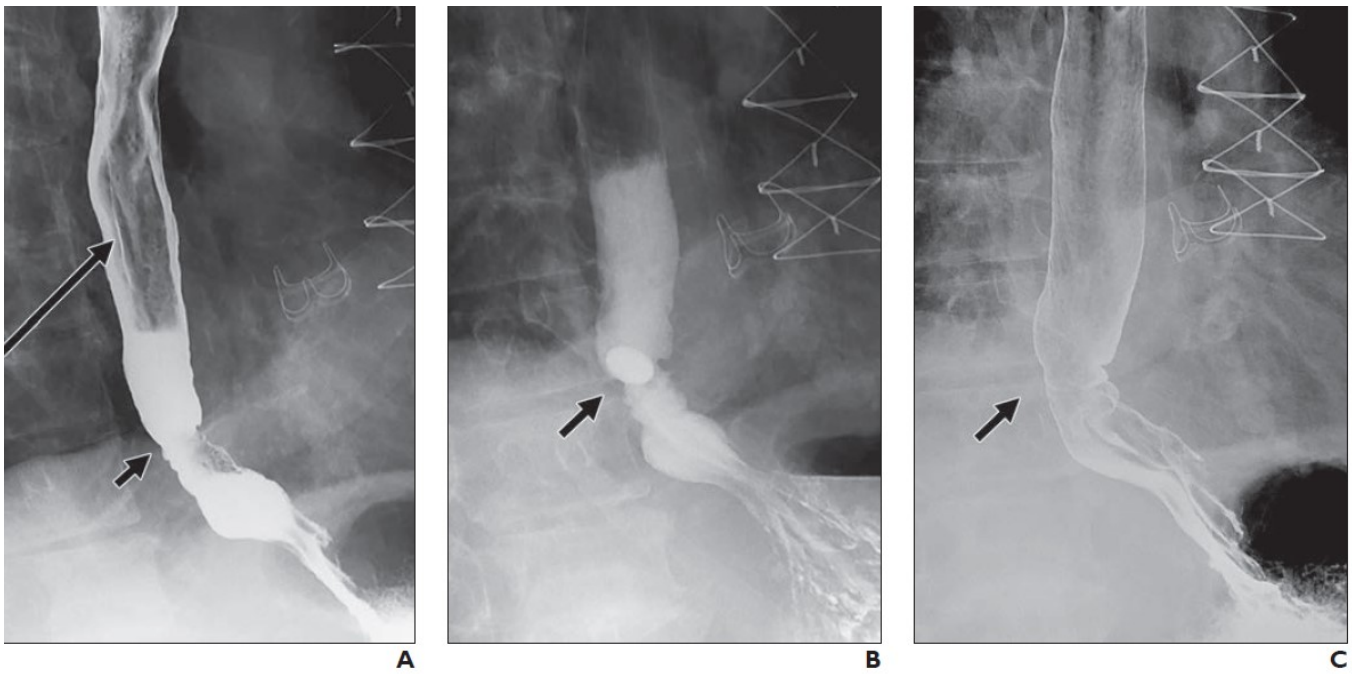

Figure 11. An 87-year-old man with endoscopically confirmed Barrett esophagus treated with omeprazole for 2 months. (**A-B**) Pretreatment barium esophagram shows prolonged stasis of a 13-mm barium tablet (arrow). (**C**) Posttreatment esophagram reveals improved ring (arrow) lumen diameter at 13 mm. Tablet passed easily into the stomach.

On radiographs (A-B), a long (6 mm) concentric asymmetric narrowing in the lower esophagus and a sharp shortening (8 mm) of the LES with folds in its lumen are determined. After PPI treatment, the lumen of the esophagus widened, but the cicatricial deformity of the left wall remained. Conclusion: In an elderly person with severe reflux esophagitis in the stage of intestinal metaplasia, there was a narrowing

in the lower part of the esophagus distal to PS. It resolved because of conservative treatment.

In the article by Novak et al, all 4 patients with dysphagia, presented as cases with SR, had elderly people with GERD, 3 of which had reflux esophagitis complicated by peptic stricture. Remarkably executed X-ray diffraction patterns in none of the parameters corresponded to the classical SR description. The same can be said about the cases described in other articles (**Figure 12**).

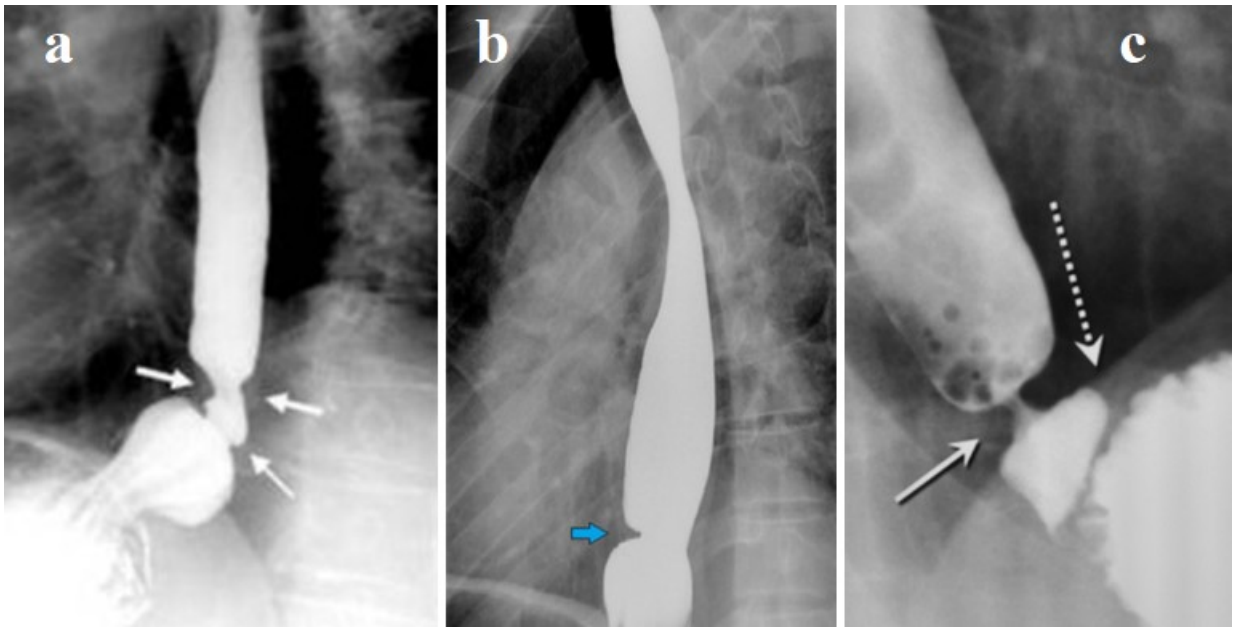

Figure 12. Radiographs from various articles described as examples of SR. **(a)** During the gag reflex, an asymmetric and long stricture above the short LES is visible **(b)** A 37-year-old woman with a history of GERD for more than 5 years with good symptomatic PPI control developed dysphagia. On the radiograph and on the endoscopy image, pronounced asymmetry of the ring is visible [12]. **(c)** Asymmetric stricture is near the short LES. Erosions are visible in the extended esophagus above the narrowing.

In these cases, in addition to the fact that the esophageal stricture was asymmetric, it was located significantly distal to the proposed site of the proximal sphincter. Radiological, clinical symptoms and endoscopic signs testified to GERD with reflux esophagitis.

VI. Discussion

We analyzed articles with about 1000 patients with low esophageal constriction, who combined with the symptoms of dysphagia, and the presence of the so-called HH. Although they were all named as the Schatzki Ring, in fact, they were two different diseases. From 1953 to 1963, Schatzki and Gary published several articles in which they concluded that the ring was fixed in the location in the gastroesophageal junction (1). It was distensible but had a fixed maximum diameter (2). They showed that all rings less than 13 mm in diameter are symptomatic, none greater than 25 mm are symptomatic, and symptoms for those in between are dependent on eating habits (3) [13,14].

Histopathology of SR is defined as a diaphragm like a mucosal shelf, triangle-shaped in cross-section of muscularis mucosa and smooth muscle fibers forming an annular bend. Above the ring is squamous epithelium and below is the columnar epithelium. The authors believed this to be the mucosal gastroesophageal junction and its location proximal to the diaphragm to be a sign of HH [15]. The narrowing is caused by submucosal fibrosis. The overlying mucosa is intact, and the underlying muscle is normal. The narrowing may slowly progress. Hiatal hernia is usually present [10]. These findings and conclusions subsequently shared by many others [2,5,10]. Schatzki et al believed the ring is congenital, not caused by inflammation [13], and that narrowing over time is uncommon and minor [16].

The X-ray diagnosis of SR is based on the presence of a fixed, symmetric, thin (< 4 mm) structure, which intersects the esophagus perpendicular to its long axis, at the squamocolumnar junction [5]. Schatzki et al recommended an upright study to expand the esophagus under high hydrostatic pressure of barium columns [14,16]. To distend the lower esophagus maximally, Müller et al asked the patient to take a deep breath and to perform a Valsalva maneuver during swallowing. However, in 14 (70%) of the 20 patients who underwent radiological examinations, the diagnosis could not be determined [5].

Endoscopically, patients present with a mucosal ring-like structure in the lower esophageal area above the squamocolumnar junction. The Bolster technique facilitates the identification of a hidden ring within the hernia sac that may otherwise not be visualized without the application of internal abdominal pressure. The Bolster technique is a procedure in which an endoscopist applies deep and steady abdominal pressure in the epigastric region of the patient's abdomen, immediately below the ribs, with his or her fist. During this time, the endoscope should be localized in the hiatal hernia sac. After several seconds of applied pressure, the Schatzki ring within the hiatal hernia sac is exposed and identified (**Figure 13**) [17,18].

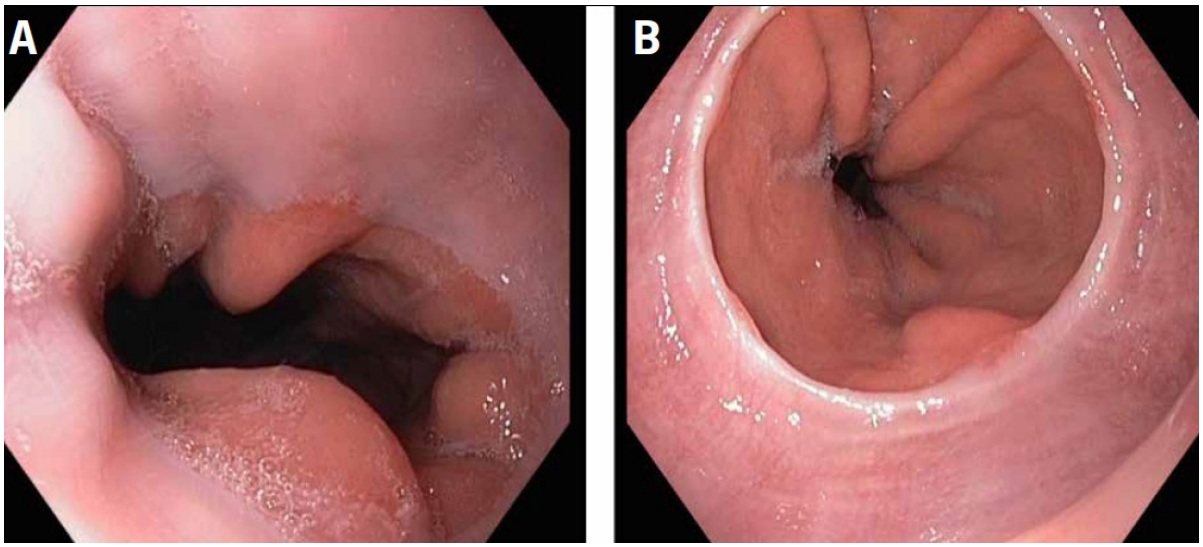

Figure 13. Endoscopic views of the gastroesophageal junction prior to compression **(A)** and after abdominal compression, revealing a Schatzki ring (**B**). (From article of Jouhourian [17].

It is known that an increase in pressure in the stomach causes a reflex increase in the tone of the LES. We performed abdominal compression during the X-ray examination of the GEJ. In patients with GERD, abdominal compression causes a contraction of the LES, which allows us to visualize and measure its length [8,19,20]. Figure 13 shows that abdominal compression caused a contraction of the LES with folds convergence. The LES is not proximal to the "hernia" but distal to it. Thus, no

shift of the LES occurs, and therefore, the extended part of the intestine distal to the SR is not a HH, but a phrenic ampulla. Secondly, the folds in the ampoule are evidence of a chronic inflammatory process that has led to the expansion and rigidity of the wall in the lower part of the esophagus. And, as Chandrasoma and DeMeester showed, cardiac metaplasia of the squamous epithelium due to exposure to gastric juice results in cephalad movement of the squamo-columnar junction [9].

VII. Pathophysiology and pathogenesis of the SR.

Contemporaries called a certain type of ring narrowing in the lower esophagus Schatzki's ring not because he described this ring first (he was not the first), but because he and his colleagues devoted much research to this pathology and achieved significant success. Unfortunately, most of our contemporaries use only the name, ignoring significant scientific evidence, and all narrowing's in the lower esophagus are called the Schatzki ring.

Since what is commonly called HH is actually a phrenic ampulla, which in turn is evidence of GERD, all patients with the SR was GERD. This is also evidenced by our measurements of LES. In all 5 patients with SR, the LES was almost 2 times shorter than normal. In all patients, the SR was at the same distance from the proximal end of the LES – about 3 cm. This place corresponds to the location of the proximal sphincter. The length of PS in children is 5 mm and in adults 7 mm [20].

An analysis of the facts described above gives reason to argue that:

1) SR is a fibrotic modified PS.
2) GERD is the cause of these changes.
3) It occurs against the background of chronic changes in the esophagus.
4) Degenerative changes in PS against the background of GERD occur in all patients, but most often the ring does not completely lose its elastic properties, and the maximum diameter during stretching of the esophagus can be of different sizes **(Figure 14)**.

We hypothesize that PS is a genetically determined anatomical formation, the role of which is:

1) to close the phrenic ampulla to create a threshold pressure in it to open the LES.

2) its contraction prevents the penetration of an acid bolus above the phrenic ampulla (**Figure 15**). Thus, our assumption coincides with the opinion of Schatzki and co-authors about the congenital nature of SR.

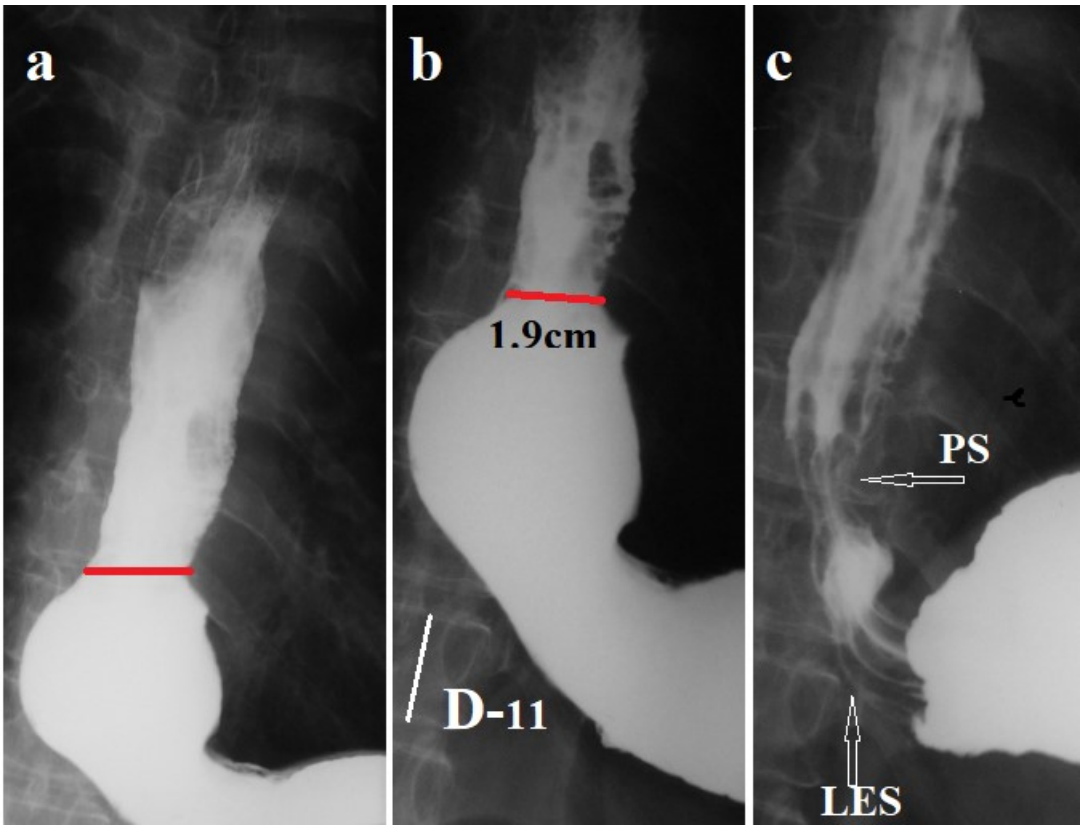

Figure 14. During upper gastrointestinal examination vomiting occurred. (**a-b**) A contracted stomach under high pressure displaces barium into the esophagus through a wide-open LES. While the phrenic ampulla continues to expand, the width of the maximum disclosure of the SR (red line) remains unchanged. The true height of D-11 is approximately 2 cm. (**c**) After stopping vomiting, rough folds are visible throughout the esophagus and in the LES.

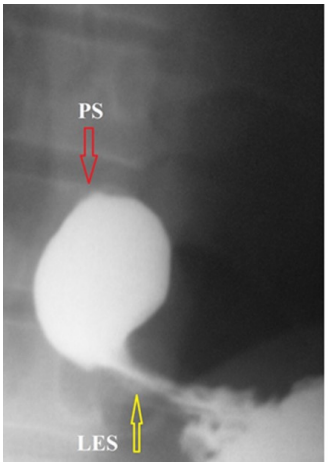

Figure 15. Upper gastrointestinal examination. During abdominal compression, the reflux of barium from the stomach to the esophagus was recorded. As a result of the PS contraction barium did not penetrate from the expanded ampulla into the proximal part of the esophagus. A contracting phrenic ampulla squeezes barium into the stomach through the LES.

VIII. Peptic stricture at the lower esophagus.

The second and more numerous group of patients in whom dysphagia occurred as a result of stricture in the lower esophagus is characterized by an acute inflammatory process as a result of esophagitis reflux. In contrast to SR, which locates at the same distance from the proximal point of the LES (≈ 3 cm), which corresponds to the location of the PS, in patients with peptic stricture, the distance from the narrowing to the proximal point of the LES ranges from 1.5 to 4.5 cm. Only in one case, it was cranial than SR. In other cases, it was in different places in the esophagus ampoule.

The concentration of peptic strictures in the esophagus ampoule is explained by the fact that a wide ampule with weak walls is not able to squeeze the whole bolus into the stomach. Part of the food remains in the ampoule after eating. The rotting this remainder throughout the day is responsible for the putrid breath. Reflux of gastric contents leads to acid retention in this pocket, which causes ulcerative damage to the wall and can lead to stenosis of the esophagus.

Peptic strictures, unlike SR, is asymmetric, and not ring-shaped. They are located at different distances from the LES (1.5-4.5 cm) and often the width of the ampoule exceeds its length. There is obvious evidence of a severe GERD: a short LES with folds, a wide and asymmetric phrenic ampulla. The diagnosis of GERD is confirmed by clinical and histological studies and the apparent effect of PPI treatment. Unlike SR, which is an exceedingly rare pathology, peptic stenosis in the lower esophagus is a frequent disease and increases from 0.2% of all studies of the esophagus in childhood [3] to 13.3% in adults [21].

Reflux esophagitis in patients with allergies is characterized by an increase in the number of eosinophils in the mucosa of the esophagus. In these cases, there are features in the treatment of the esophageal stricture, but from the point of view of pathological physiology, the diagnosis of GERD with reflux esophagitis is the main one. If peptic stricture occurs at the level of LES, then why is it called achalasia? (**Figure 16**) [22].

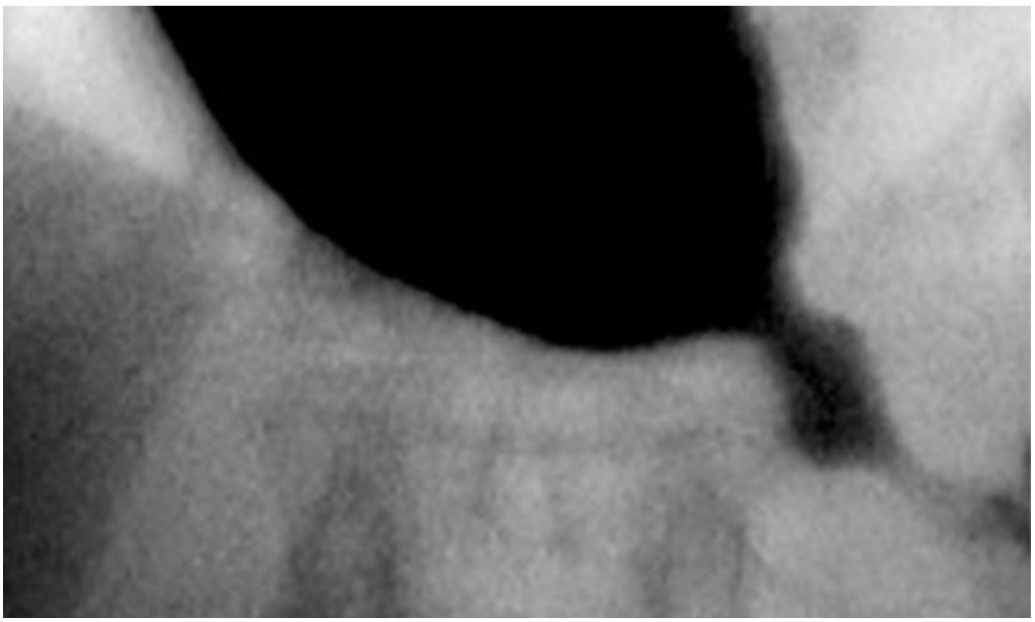

Figure 16. On the radiograph of the GEJ expanded esophagus above a contracted and deformed LES is determined (From an article by Vereczkei et al). The case described as achalasia following reflux disease [22]. This is GERD with peptic stricture at the level of LES and treatment should be consistent with the diagnosis.

IX. Practical guidelines based on the pathophysiology of SR and peptic stricture

X-ray diagnosis. The double-contrast study significantly increased the accuracy of diagnostic tumors and inflammatory changes in the upper digestive tract in the second half of the 20th century. However, with the introduction of widespread use of esophago-gastro-duodenoscopy, this method has lost its role. A study by Müller et al with Valsalva's maneuver during swallowing could not be determined diagnosis SR in 14 (70%) of the 20 patients who underwent radiological examinations [5].

Pathophysiological analysis. To see the narrowing of the esophagus, it is necessary to expand the phrenic ampulla as much as possible. Firstly, since the esophagus ampoule functions in a horizontal position, the study must be performed in the position of the patient lying on his back. Secondly, you need to fill the esophagus with a sufficient volume of barium. For this, the patient must continuously drink

barium through a straw from a can located near his head. Thirdly, it is necessary to close the LES so that barium tightly fulfills the ampoule. Valsalva's maneuver increases intra-abdominal pressure and pressure in the stomach, which leads to a reflex contraction of LES. However, it is not possible to simultaneously drink barium and perform Valsalva's maneuver. If you perform Valsalva's maneuver after stopping swallowing, the chances of contraction LES are reduced. To increase the likelihood of the LES contraction, it is necessary to fill the stomach with barium, i.e., to conduct a study of the esophagus after examination of the stomach and duodenum. In young children we produce the abdominal compression while swallowing barium. In older children and adults, we ask to raise straighten legs while swallowing barium.

SR treatment. Since SR is a fibrous ring that without an acute inflammatory process, conservative treatment aimed at suppressing the release of hydrochloric acid makes no sense. The long-term results indicate that recurrent dysphagia is common among patients with Schatzki's ring after a successful dilation. During follow-up, 35 patients (63%) developed recurrent dysphagia and required repeated dilations: 19 patients (34%) had one to two dilations, 9 patients (16%) had three to seven dilations, 6 patients (11%) had more than seven dilations; 1 patient underwent surgery for resection of the Schatzki's ring (2%) [23]. Unfortunately, in all articles under the diagnosis of SR, the authors describe lower esophageal stricture, so there is no way to unambiguously evaluate balloon dilatation for SR. It can be assumed that, regardless of the nature of the narrowing, balloon distension causes ruptures in the narrowed section of the esophagus, which causes the formation of scar tissue and leads to repeated narrowing.

Peptic stricture treatment. The literature provides compelling evidence that PPI treatment causes an increase in stenosis width and eliminates dysphagia. The use of Budesonide, regardless of the number of eosinophils in the biopsy, as well as

antihistamines, can speed up the healing process and will allow abandon other methods of treatment.

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