I offer an analysis of the medical history of a 53-year-old man who came to me with the complaints listed below. Unfortunately, it was impossible to determine when he first began to feel ill. We will analyze the reason for this "forgetfulness" in the "Discussion" section.

- **Complaints:**

Depression, cough with sputum production, very frequent belching. Heartburn occurs very rarely – once a year. Symptoms occur during eating, after drinking, and during conversation. Taking PPI twice a day did not provide relief.

Gastroscopy – mild antral gastritis

- **High-resolution manometry**

1. Esophageal peristalsis is normal.

2. Normal LES length 2.7–4.8 cm

3. LES length – 2.7. Intraabdominal – 0

4. HH- 1.8 cm.

5. Distal contractile integral (DCI) – 2636,6 (Normal 500-5000)

6. Integrated Relaxation Pressure (IRP) is at the upper limit of normal.

7. Upper esophageal sphincter (pressure) – normal.

Conclusion: the function of the LES is normal. The data are close to esophagogastric junction (EGJ) outflow obstruction (EGJOO). Small hiatal hernia (1.8 cm).

- **Ambulatory reflux monitoring (pH – impedance)**

1. Acid reflux 5 cm above LES – 50.

2. Proximal reflux only upright (15 cm): Acid reflux 12, weakly acidic reflux 5, all reflux 17 – only up.

3. Mean nocturnal baseline impedance – 1.56

4. Interpretation: (a) Mild increase in acid reflux (pH-4.9%) (AET > 4 but <6) was seen during testing. This was mainly driven by upright position reflux. (b) Mild increase in overall number of reflux episodes (>40 but <80). (c) Mean NBI is low (< 2). (d) No symptoms recorded during testing.

Conclusion: Suspected reflux disease is not severe.

A course of 8 weeks of treatment with a double dose of PPI is recommended.

- **Consultation with an ENT doctor**

Diagnosis: Partial unilateral paralysis vocal cord, Cholesteatoma (LT), Eustachian tube dysfunction.

- **CT and MRI of the glottis.**

Asymmetry of the vocal cords, rotation of the arytenoid cartilage, thickening of the epiglottic tissue.

- **Radiography of the esophagus and its sphincters with high pressure in the stomach.**

This test is based on physiology. A significant increase in gastric pressure causes a reflex contraction of both the upper and lower esophageal sphincters [1]. The patient lies on the X-ray table and sips barium through a straw from a jar placed next to their head. When he feels that the barium is no longer flowing, he immediately raises his straightened legs. This signals the need to take the first radiograph **(Figure 1a).** Five minutes later, a second radiograph is taken with the patient at rest. Sometimes second radiograph gives additional information.

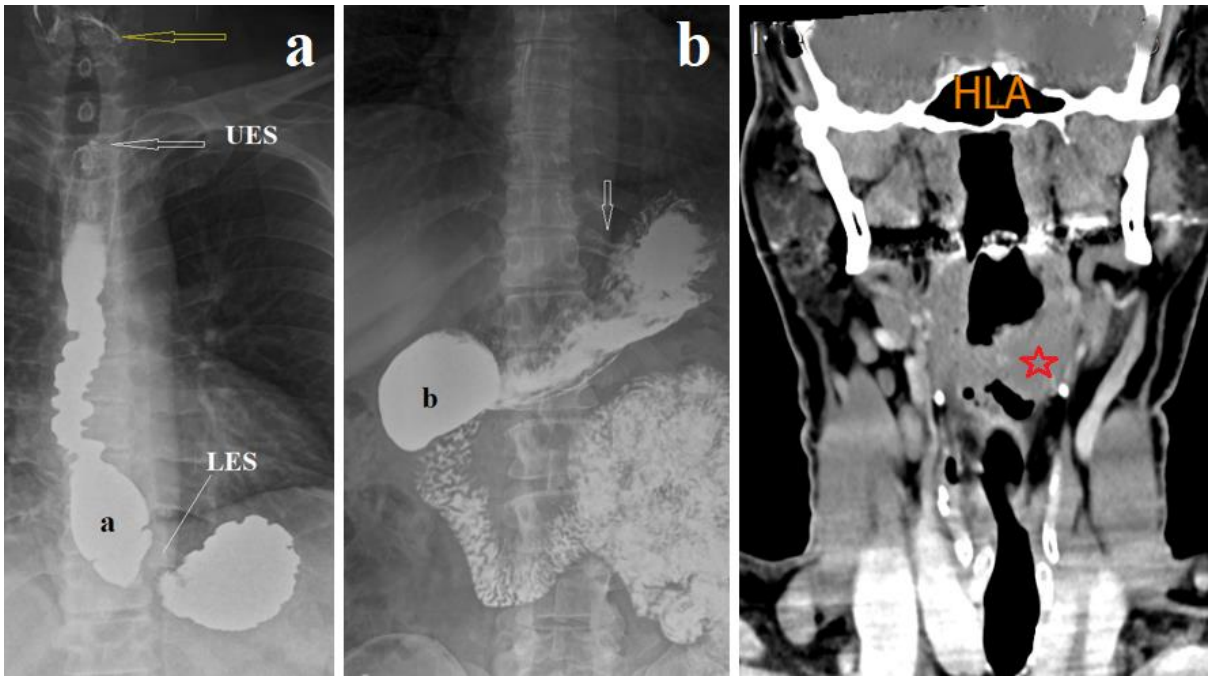

Figure 1. (a) In frontal radiography, the distance between the phrenic ampulla (a) and the stomach, which does not contain contrast, is the contracted LES. Its length is 1.6 cm, approximately 2 cm shorter than normal. The ampulla width is 3.2 cm. The contours of the esophagus are tortuous and asymmetrical, indicating dyskinesia due to esophagitis. The upper

yellow arrow indicates asymmetry with a lesion of the left hypopharynx. (b) After 5 minutes, significant evacuation of barium from the stomach is detected. A barium collection (b) resembling a diverticulum is detected in the projection of the gastroduodenal junction. The arrow shows thickened parallel folds in the LES, indicating inflammation. (c) On the section of the CT in the pharynx determine an asymmetric thickening (asterisk), suggesting an inflammatory process.

Conclusion: Extra-esophageal presentation of severe gastroesophageal reflux disease. Gastritis. Pharyngitis. Partial unilateral paralysis vocal cord, Cholesteatoma (LT), Eustachian tube dysfunction.

I gave him a tablet to swallow, 1.9 cm in diameter and 0.5 cm thick, after which in some patients the symptoms of GERD are reduced or disappear completely [2]. But in this case, it didn't help.

Discussion

Problems of methodology in historical aspect.

The criterion of truth is proven scientific facts and the physiological laws of the digestive system, including the esophagus and its sphincters. If a new assumption (hypothesis) contradicts any scientific fact, it must be rejected as incorrect. The Chicago classification version 4.0 is based on a vote of invited gastroenterologists who discuss the symptoms of the disease and the reliability of various equipment. If consensus reaches 80%, then this variant is accepted as reliable. Journal in which Chicago classification version 4.0 was published не имел права ее опубликовать из-за the presence of a conflict of interest [3]. Secondly, new knowledge in science is not created by voting.

Before the use of pH monitoring, the presence of reflux on X-ray examination was considered evidence of a disease called gastroesophageal reflux (GER). DeMeester and co-authors determined the norm for pH-monitoring examined 15 patients who claimed to have no reflux symptoms [4,5]. This idea, without scientific justification, assumed the possibility of physiological reflux. A study by Korean authors showed that 17% of individuals without complaints had signs of GERD during gastroscopy [6]. However, it is known that gastroscopy only detects complications of GER (erosions, stenosis, Barrett's esophagitis and tumors). Thus, pH monitoring and “DeMeester score” contradicted scientific data. They detected only severe forms of GERD. Some studies showing that GERD can occur and progress without symptoms

are given in my review [7]. Since pH monitoring has no scientific basis and only detects severe forms of GERD, it does not increase the accuracy of GER diagnostics compared to clinical symptoms.

The Global Consensus Group at its conference in Montreal (2006) redefined reflux disease. "GERD was defined as a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications" [8]. The new definition was necessary for clinical symptoms to match pH monitoring results, which diagnosed GERD only in advanced cases of GERD. Because of this definition, a huge percentage of patients with reflux with minor or no esophageal symptoms did not receive timely treatment. Since articles contradicting the "Global Consensus" were not accepted for publication, the impression arose of universal support for decisions made by the Delphi method. The careers of gastroenterologists would be ruined if they didn't use the recommended equipment. Many decided to follow the accepted rules. Articles that contradict the Delphi method's decisions are rejected as inconsistent with scientific requirements. As a result of the victory of Chicago classification version 4.0©, public opinion has arisen that if its decisions are accepted by all, then they are correct.

The same specialists at the Lyon Consensus 2.0 reached a consensus on more sophisticated diagnostics for GERD. First, criteria adopted in previous votes but proven poor in diagnosing treatable GERD were excluded. Second, the previous definition of GERD was replaced with recommendations that allow a diagnosis of GERD only after the use of equipment. (1) Patients without previous signs of GERD (unconfirmed GERD) are assessed using long-term wireless pH monitoring or catheter pH monitoring, or pH monitoring without antisecretory drugs. (2) Patients with confirmed signs of GERD (confirmed GERD) and persistent symptoms are assessed using pH impedance monitoring with optimized antisecretory therapy. Thirdly, using modern equipment, they stated that “Later studies have demonstrated grade A esophagitis in 5%-7.5% of healthy subjects. In healthy subjects, grades B, C, and D esophagitis are highly uncommon” [9]. A huge list of conflicts of interest confirms that the authors abandoned previous "global consensus decisions" in favor of advertising equipment whose manufacturers continually funded their financial well-being. Their decisions are not only absurd but also dangerous for patients. Obviously, patients with esophagitis cannot be considered healthy. This lack of logic has led to the emergence of several functional diseases. For example, if AET < 4% with heartburn, it may be either a hypersensitive esophagus or functional heartburn, which can only be differentiated by instrumental methods. As a result, patients with reflux are prescribed medications to alter the brain's effect on the digestive system [10].

Analysis of HRM

1. The normal limits for LES length, defined for HRM, differ by almost a factor of two. This indicates an inappropriate selection of control subjects for determining the normal range, as all anatomical parts of the body vary little even among individuals of different heights. In the article Kwiatek et al, to determine normal values for HRM, the authors selected 15 patients without gastrointestinal complaints [11]. It is not clear why did they not use pH monitoring to rule out GERD in control individuals, although they themselves promote as the ideal method for diagnosing GERD. Analysis of the radiographs shows that the case cited in the article corresponds to severe GERD. Reviewing the X-ray patterns, the authors state that "the phrenic ampulla is a transient structure comprised of the stretched, effaced, and axially displaced LES." Following ampullary emptying, the stretched LES (maximum length 4.0 cm) progressively collapsed to its baseline length of 1.9 cm ($P < 0.001$). Radiographs show that the endoclip attached to the esophago-gastro junction remains in the same position relative to the thoracic vertebra, indicating no displacement of the LES. The presence of a phrenic ampulla represents esophageal dilation and is evidence of reflux esophagitis. Shortening of the LES from 4 cm to 1.9 cm indicates that the intra-abdominal portion of the LES failed to withstand the tension and opened. Consequently, the authors selected a patient with GERD for the control group, and all the norms were compromised.
2. In the analyzed case, the function of the LES is described as normal with a length of a LES of 2.7 cm, although the absence (0) of an intra-abdominal part, that is, most of the LES did not perform the antireflux function, since it was opened. This conclusion contradicts physiology.

3. The dilation of the esophagus above the LES was called a hiatal hernia, 1.8 cm wide. Figure 1a shows that the dilation of the esophagus is located above the very short LES. On radiograph, its width has become wider (3.2 cm) because the pressure in the esophagus has increased as a large volume of barium is trapped between the contracted UES and LES. This is a phrenic ampulla, which cannot be a hernia, because it is located above the LES. The discovery of an ampulla is evidence of reflux esophagitis. The assertion that an ampulla can exist without GERD arose after the introduction of pH monitoring and is explained by the fact that pH monitoring diagnoses only severe forms of GERD.

Normal esophagus and LES physiology

On standard radiographic examination, the LES cannot be identified because the peristaltic wave rapidly propels the barium bolus into the stomach (Figure 2).

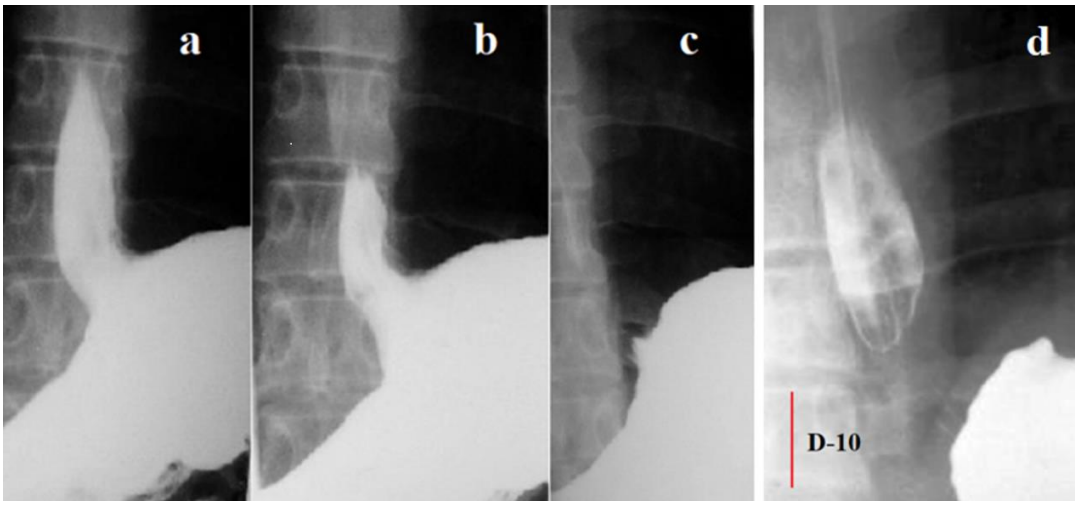

Figure 2. (a-c) In the horizontal position, a swallow of food (barium) moves rapidly by a peristaltic wave in accordance with the Baylis-Starling law of the intestine, which states: "Excitation at any point of the gut excites contraction above, inhibition below" [12]. The barium passes through the LES without stopping and therefore LES cannot be seen. However, when the pressure in the stomach increases, the LES contracts to prevent the reflux of gastric contents into the esophagus [1]. (d) Abdominal compression during barium ingestion causes an increase in gastric pressure, which leads to a contraction of the LES, the length of which can be measured as the distance without contrast between the esophagus and the stomach. Knowing the true height of the tenth thoracic vertebra, the length of the LES can be accurately determined.

The results of measuring the LES in patients of different ages are presented in Table 1 [7].

Table 1. Normal length LES in different age groups

	Length of lower esophageal sphincter (cm)					
Age	Up to 1 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
M± м	0.86±0.03	1.40±0.02	1.72±0.07	2.10±0.05	2.45±0.11	3.60±0.08

I believe that the results obtained are close to the true ones, since they coincide with the normal length of the LES measured by the manometric method in adults: 34 ± 9 mm [13], 35 ± 4 mm [14]; 36 ± 12 mm [15]; 37 ± 1 mm [16]; 4.1 cm [17]. However, it should be borne in mind that

GERD cannot be excluded based on the absence of symptoms. I examined patients who developed unpleasant symptoms only a few weeks or months before seeking medical attention, assuming that the LES length had not yet had time to significantly decrease relative to the norm. Since there were probably some patients with GERD among the control subjects, I believe that the true LES length in each age group is close to the maximum variant. In adults, the LES length is probably ≈ 4 cm.

Mapping of the esophago-gastric junction (EGJ).

On some radiographs in adult patients with GERD during the contraction of the LES, it is noticeably shorter than normal both due to the opening of its proximal and distal (abdominal) parts. In such cases contracted only the segment of the LES that is in the diaphragmic (hiatal) channel. On numerous radiographs, the minimum length of this segment was 1 cm. By comparing different radiographs and determining the true dimensions, I was able to determine the different parts of the LES and their relationship with the CD, i.e., create a map of the EGJ. A typical example is shown in Figure 3abc.

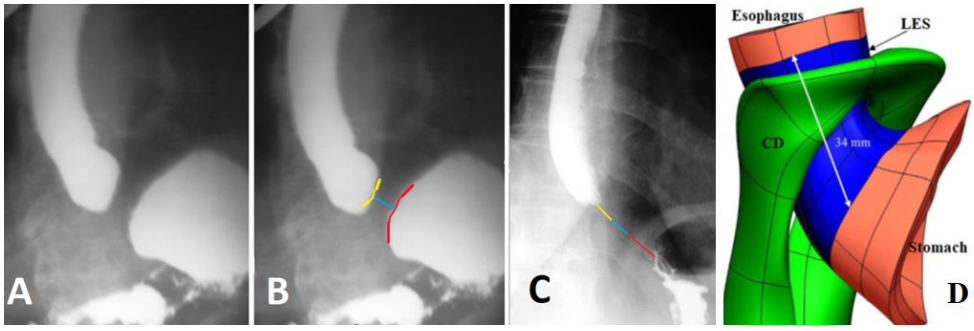

Figure 3A and scheme to it **(B)** of patient with GERD. Radiograph was done in a horizontal position with abdominal compression. The sharp shortening of the LES because of the opening of the supra-diaphragmatic part of the LES (yellow) and inside the abdominal part of the LES (red) was determined. Only the diaphragm part (blue) of the sphincter is closed. (**Figure 3C**) In an upright position during abdominal compression the LES contracted in response to the increased pressure in the stomach. It is visible as two longitudinal folds between the esophagus and stomach. Since the actual height of D-10 is ≈2 cm, the actual length of the LES is ≈3.4 cm. The LES parts: red - the abdominal segment, blue - inside the diaphragm, yellow - above the diaphragm. (**Figure 3D**) Three-dimensional model of the EGJ from the article by Yassi et al [18]. 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 crural diaphragm and 0.4 cm above the diaphragm.

Thus, in the case described above, the short LES, with a dysfunctional intra-abdominal portion, cannot withstand high gastric pressure for long periods. Large volumes of food cause it to relax, and chyme enters the esophagus. A short LES is a cause and evidence of GERD, but not one of a predisposing factors as stated in consensus decisions.

Analysis of pH impedance monitoring results

Over a 24-hour period, the patient experienced 50 episodes of acid reflux, but only in the upright position. As Figure 1b shows, his stomach empties rapidly. Since he goes to bed four hours after his last meal, his stomach is empty at that time. Therefore, he did not experience reflux in the horizontal position. However, during the day, after a large meal and an active lifestyle, intragastric pressure increases, leading to opening of the weakened lower esophageal sphincter and the reflux of chyme into the esophagus. This leads to the following conclusions: (1) To prevent reflux in the presence of a damaged LES, it is necessary to eat small meals during the day and lie down only on an empty stomach. In severe cases of GERD, sitting for at least half an hour after eating is not recommended. Without these recommendations, the pH of the chyme is of little importance, as gastric chyme, containing hydrochloric acid and pepsin, leads to the breakdown of food proteins and, when it enters the esophagus, damages its wall, which lacks the same protection as the stomach. Secondly, each episode of reflux damages the esophagus and LES. Repeated episodes lead to progressive irreversible losses, i.e., GERD is a chronic, progressive disease. (2) The idea of the possibility of physiological reflux was necessary for DeMeester to justify pH monitoring. Reflux was considered physiological if AET < 4%. His followers have brought pH monitoring to the point of absurdity. In our patient, the diagnosis of suspected mild GERD did not help him after using a double dose of PPI twice daily. He received no other recommendations (3). At the onset of the disease, reflux causes heartburn and chest or abdominal pain. However, during this period, reflux frequency is low, and damage to the LES and esophagus is minor. In such cases, pH monitoring does not diagnose reflux due to an AET < 4%. Based on this, consensus groups diagnose hypersensitive esophagus or functional heartburn. Over time, patients no longer experience esophageal pain (heartburn, pressure, etc.), and this can only be explained by the fact that gastric chyme has caused severe damage to the esophageal nerve endings responsible for sensing damage of various types of pain.

Conclusion. Modern gastroenterology emerged with the advent of pH-metry, stripping away centuries of accumulated scientific knowledge about the anatomy and physiology of the

digestive tract. Its creators came from traditional gastroenterology departments and were familiar with the advances of science. They deliberately discarded these advances to allow for seamless adjustment of instrument parameters to the patient's symptoms. For adaptation, they chose the Delphi method, which is used in economics and manufacturing to select optimal solutions. However, it has no connection to science. The same conference participants, using different types of equipment, gradually modified their recommendations, which are published in countless articles as new developments in science. This equipment promotion campaign is well-paid thanks to company profits. No gastroenterologist can make a career without publishing articles using various diagnostic equipment. In such cases, doctors are forced to join the mainstream, resulting in the perception of universal support. Articles without the use of recommended equipment are not accepted for publication as low-level work. This gave rise to the idea of general support for the new gastroenterology, which led to a false conclusion about its scientific value.

Analysis of this observation exemplifies the absurd conclusions drawn from equipment based on unscientific principles. The use of equipment and misconceptions about the pathophysiology of GERD led to delays in pathogenetic treatment for many years and disease progression. Analysis of clinical symptoms and radiographic examination revealed that gastroesophageal reflux developed long ago, possibly in childhood, as a mild form with few significant symptoms. It developed because of hydrochloric acid hypersecretion, as evidenced by the diagnosis of antral gastritis, which was classified as superficial during the use of PPIs. In most cases, hypersecretion occurs due to lactose intolerance. Undegraded lactose causes the release of histamine from intestinal mast cells. This histamine leads to increased hydrochloric acid secretion in the stomach [19]. Extra-esophageal presentation of severe gastroesophageal reflux disease. Gastritis. Pharyngitis. Partial unilateral vocal cord paralysis, cholesteatoma (LT), Eustachian tube dysfunction probably represents a single chain of pathogenesis.

I've provided key references to scientific studies. They do not include reviews or consensus results, which lack scientific standing. Some statements are not cited because they are well-known and widely cited. These are most easily found on my website: http://www.anorectalmalformations.com.

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