Duodenal motility in health and disease. Review

Anatomy. Anatomy. The duodenum is divided into 4 parts. The duodenal bulb about 2 cm long, is the first part of the duodenum and is slightly dilated. It is at the vertebral level of L1. It has a bulb shape only on radiographs when filled with chyme from the stomach. The first part of the duodenum is the most common location of ulcers since it is where the acidic chyme meets the duodenal mucosa before mixing with the alkaline secretions of the duodenum.

The second part of the duodenum begins at the superior duodenal flexure. It goes inferior to the lower border of vertebral body L3, before making a sharp turn medially into the inferior duodenal flexure, the end of the descending part. The second part of the duodenum contains the major duodenal papilla through which the pancreatic duct and common bile duct conduct pancreatic juice and bile. The junction between the embryological foregut and midgut lies just below the major duodenal papilla.

The third part, or horizontal part or inferior part of the duodenum is 10~12 cm in length. It begins at the inferior duodenal flexure and passes transversely to the left, passing in front of the inferior vena cava, abdominal aorta, and the vertebral column. The superior mesenteric artery and vein are anterior to the third part of the duodenum.

The fourth part, or ascending part, of the duodenum passes upward, joining with the jejunum at the duodenojejunal flexure. The fourth part of the duodenum is at the vertebral level L3.

Under microscopy, the duodenum has a villous mucosa, which distinct from the mucosa of the pylorus. In the mucosa, there is Brunner's glands, which secrete mucus and bicarbonate to neutralize stomach acids.

Duodenal function. The duodenum is largely responsible for the breakdown of food in the small intestine, using enzymes. It also regulates the rate of emptying

of the stomach via hormonal pathways. Secretin and cholecystokinin are released from cells in the duodenal epithelium in response to acidic and fatty stimuli present there. These cause the liver and gall bladder to release bile, and the pancreas to release bicarbonate and digestive enzymes such as trypsin, lipase, and amylase into the duodenum as they are needed. In lactose-tolerant people in the villous mucosa is lactase, which breaks down lactose into glucose and galactose (Wikipedia).

In the literature, there is almost no information about the function of the duodenum to protect the small intestine from the aggressive effects of hydrochloric acid. The only sphincter of the duodenum is considered the sphincter of Oddi. Other sphincters are mentioned only in the historical aspect, as something not deserving of serious attention. This can be seen from the following phrase: " The "sphincters" of the duodenum are mentioned and evaluated" [1], where the quotes in the word "sphincters" define the authors' attitude to this issue. This is because all intestinal sphincters do not have clear anatomical (and histological) boundaries. For example, the lower esophageal sphincter is described as a thickening of the circular layer without clear boundaries between the esophagus and the stomach. All sphincters of the intestine are characterized by a thickening of the circular muscles, contraction according to a specific program, and clear size during contraction.

Function of the duodenal bulb and post bulbar sphincter (PBS). During anatomical examination and during surgery, the duodenal bulb does not differ in shape from other parts of the small intestine. Often it is slightly wider than the 2nd part of the duodenum **(Figure 1. a)**. On an x-ray examination, during the entry of chyme from the stomach, it acquires the shape of a bulb because of the contraction of the post bulbar sphincter **(Figure 1.b)**. When the bulb is filled with chyme, the pressure in it rises, which leads to reflex closure of the pyloric sphincter. At this time the base of the bulb is expanded, which confirms the rise

in pressure in the bulb. This reaction of the pyloric sphincter conform to the Bayliss-Starling gut law and is characteristic for all sphincter zones: an increase in pressure in the stomach causes an increase in the tone of the LES; an increase in pressure in the rectum causes an increase in the tone of the rectosigmoid sphincter, etc. [2,3].

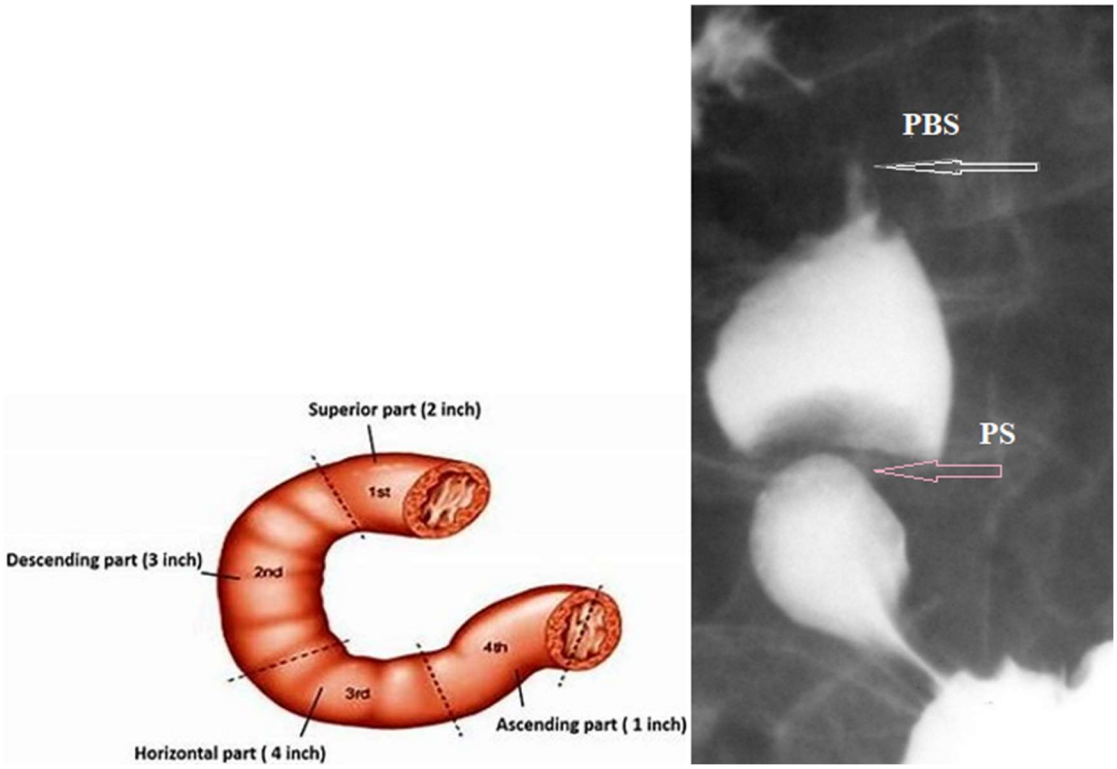

Figure 1. (a) Diagram of an empty duodenum (b) Radiograph of the gastroduodenal junction. The duodenal bulb is triangular because of the contraction of the post bulbar sphincter (white arrow - PBS). The complete filling of the bulb led to an increase in pressure in it, which caused a contraction of the pyloric sphincter (pink arrow -PS).

The PBS function provides a portioned evacuation of the chyme from the stomach. Portions equal to the capacity of the duodenal bulb. This x-ray picture is observed in all studies regardless of barium pH since the bolus volume is the factor in the PS response. The length of the PBS could not be determined because the evacuation of the bulb occurs quickly and without delay.

In 1906, Ochsner during gallbladder and stomach operations drew attention to the expanded part of the duodenum with gas to a point just below the entrance of common duct, while below this it was contracted. On the anatomical material, he found a narrowing in the third part of the duodenum. It was found that the dilatation of the upper portion of the duodenum was most commonly present in patients suffering from chronic cholecystitis with sand or gallstones in the gallbladder. He concluded about the presence of a sphincter at this place whose physiological function would consist of providing for a means of retaining the chyme in the upper portion of the duodenum sufficiently long to provide for a thorough mixing with bile and pancreatic fluid. He assumed that this sphincter in the case of inflammation in this vicinity can lead to pathological conditions [4]. Since then, this sphincter has been called Ochsner's sphincter. In the second part of the duodenum, the sphincter is localized, which in the literature is called the Kapanji's sphincter [5].

Ochsner sphincter and Kapanji sphincter.

As shown in an article by Ochsner over 100 years ago, the sphincter described by him reacts to hydrochloric acid. Therefore, when studying with barium without hydrochloric acid, the sphincter does not contract and it cannot be detected in patients without duodenal pathology. In the inflammatory process, the tone of the sphincters of the duodenum increases. In such cases, you can record the contraction of the sphincters and measure their length (**Figures 2**).

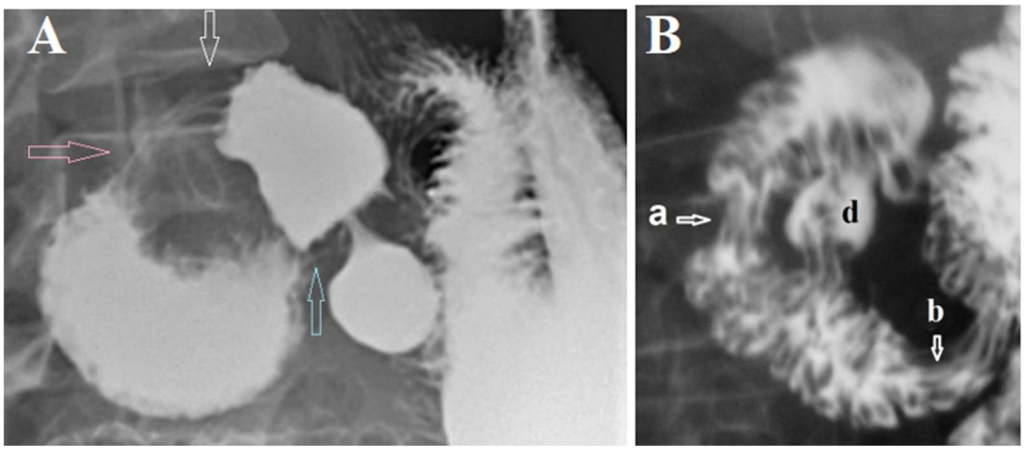

Figure 2. Radiographs of elderly patients with duodenitis and duodenal dyskinesia. **(A)** The duodenal bulb is expanded and deformed. Despite the large volume of contents, it continues to receive chyme, since the bulb has not reached its maximum capacity. This indicates a high tone of PBS (white arrow). A sharp expansion of the intestine is determined between the Kapanji sphincter (pink arrow) and the Ochsner sphincter (blue arrow). This expansion is due to the high pressure that occurs when both sphincters contract; **(B)** The duodenum was emptied, but the barium remained in deep folds because the barium-filled bowel was very wide. Two zones of contraction with longitudinal folds are visible: (a) the Kapanji sphincter, and (b) the Ochsner sphincter. The juxtapupillary diverticulum is located between them. These diverticula result from the extrusion of the mucosa between the muscular fibers. Thus, this diverticulum is evidence of high pressure that occurs during contraction between Kapanji and Ochsner sphincters [6].

In the article by Levin et al, X-ray examinations were carried out in 8 patients with the addition of 3 grams of vitamin C to 200 ml of barium. Such a slight decrease in pH, nevertheless, contributed to the contraction of the Ochsner and Kapanji sphincters (Figure 3), which confirms the opinion of Ochsner about the reaction of sphincters to hydrochloric acid. In a retrospective analysis of radiographs, the sphincter Kapandji was detected and measured in 16 cases. He was in the 2nd part of the duodenum at 2-3 cm from the PBS. Its length ranged from 1 to 3 cm (2.05± 0.09 cm). The Ochsner sphincter was found on 20 x-rays. It was in the third part of the duodenum in the L-3 projection. Its length ranged from 2 to 4.2 cm (3.2 ± 0.15 cm). In all cases, these sphincters were in the same place, which excludes the possibility of registering peristaltic contraction [6].

Sphincter Oddi. The sphincter of Oddi (SO) is a muscular valve responsible for controlling the flow of bile and pancreatic secretions through the ampulla of Vater into the second part of the duodenum. It is composed of three layers of concentric smooth muscle that surrounds the common bile duct, the main pancreatic duct, and the ampulla of Vater. The papilla of Vater includes the SO and its overlying mucosa. The SO serves to regulate the flow of bile and pancreatic juices as well as to prevent the reflux of duodenal contents into the pancreatobiliary system.

CO is the most studied sphincter of the duodenum. Its length during contrasting (Figure 3) and, as a high pressure zone between the common bile duct and the duodenum, is approximately 1 cm [7,8]. Its electrical parameters and relationships with hormones are known, etc. [9]. However, "The mechanism of dysfunction (SO) remains uncertain, but disruption of neural pathways involved in sphincter function seems likely" [10].

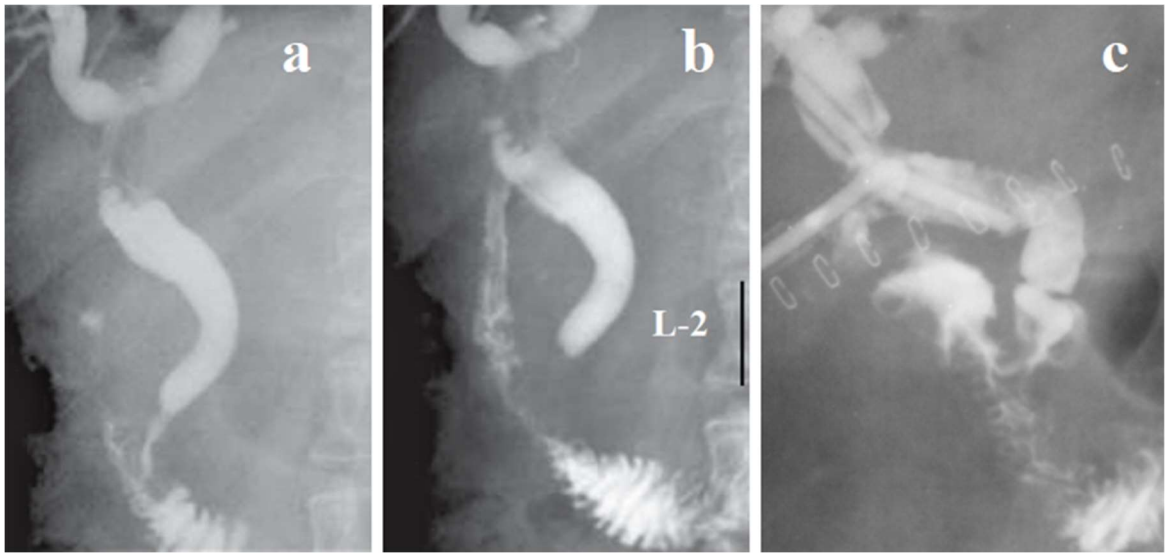

Figure 3. (a-b) Endoscopic retrograde cholangiopancreatography (ERCP) in a patient with obstructive jaundice. It is known that the true height of L-1 is 2.2 cm. However, it turned out to be impossible to accurately determine the height L-1 on the radiograph. We measured the L-2 height, assuming it to be 2.3 cm. The length of the non-contrasting gap between the common duct and the duodenum is approximately 1.3 cm. This is the contracted SO. (c) A significant expansion of the common bile duct is combined with a shortening of SO. Its upper part looks like an ampulla.

In one article on SO normal anatomy describes a completely different picture. "Cholangiography can visualize the movement of the sphincter peristalsis. First the upper part, the sphincter choledochus, opens from above downwards, the contrast enters the ampulla. Then the sphincter choledochus contacts, again from above downwards, isolating a small portion of contrast in the ampulla. The distal sphincter opens, and the systolic volume falls into the duodenum (opening phase). Thereafter the sphincter contracts again, this time from below upwards - an antiperistalsis movement. First the distal sphincter is closed, flowed by the

sphincter choledochus. When the contraction is complete, no contrast is seen in the intramural part and the contracted muscle produces a convex stop of the contrast in the lower common duct (closing phase) "[11].

Firstly, SO refers to anatomical sphincters, but all anatomical sphincters (upper esophageal, lower esophageal, pyloric, and internal anal sphincter) open without peristalsis. Secondly, cholangiography is not produced in healthy people. The authors describe a severe form of SO dyskinesia, suggesting, like many other researchers, that motor function is the same in healthy and patients. Ampullar expansion of the common duct occurs in the case of significant expansion of the duct when its weak wall during peristalsis is unable to create a threshold pressure for SO opening. The ampoule can create this pressure because the contraction force above it is greater than at the base. The same error is typical for the description of the norm of the esophageal-gastric junction, where patients with phrenic ampulla are considered healthy [12]. Third, some authors consider that a normal OS function is "Allow retrograde filling of the gallbladder" [13]. However, there is no physiological meaning in this, which is not typical for the physiology of living organisms. We need to learn perfection from Nature. It is obvious that retrograde motion in the common duct is evidence of SO dyskinesia.

Duodenojejunal sphincter (DLS). An anatomical study by Shafik et al found the thickened circular muscle and narrowing at duodenojejunal junction (DJJ) from 0.75 to 0.9 cm in length [14]. A high-pressure zone of 1.6 ± 0.04 cm length was detected at the DJJ [15]. Of 112 patients who complained of epigastric distention and discomfort after meals, they encountered nine patients in whom the DJJ did not open on duodenal contraction [16]. Based on these studies, they hypothesized the presence of a sphincter in the DJJ, the dyssynergia of which can cause pathological symptoms.

I. The normal duodenal motility

Each 3-5 peristaltic wave of the stomach forms a closed cavity in the antrum. The volume of this antral chamber is approximately equal to the capacity of the duodenal bulb. During the contraction of the antral chamber (antral systole), pressure rises in it, which causes the pyloric sphincter to open, and a certain portion of chyme is injected into the duodenum. During the filling of the duodenal bulb, the post bulbar sphincter contracts. When the bulb is completely filled with a bolus, the pressure in it rises, which leads to the closure of the pyloric sphincter, after which the PBS relaxes and a bolus penetrates the 2nd part of the duodenum up to the Ochsner sphincter.

Hydrochloric acid causes a contraction of the Ochsner sphincter, which prevents the penetration of aggressive chyme into the jejunum. The bolus is discarded retrograde to the Kapangi sphincter. At this time, secretin and cholecystokinin are released from cells in the duodenal epithelium in response to acidic and fatty stimuli present there. These cause the liver and gall bladder to release bile, and the pancreas to release bicarbonate and digestive enzymes such as trypsin, lipase, and amylase into the duodenum as they are needed. The contraction of the Ochsner and Kapangi sphincters, which is described as a pendulum-like movement, mixes an acidic chyme with bile and pancreatic juice. When the pH of the chyme increases to a certain level, the Ochsner sphincter relaxes, allowing already the less aggressive bolus to enter the jejunum. Duodenojejunal sphincter prevents the backflow of chyme from the jejunum into the duodenum.

It is amazing how the duodenum functions expedient. This principle of expediency must be borne in mind when searching for still unknown facts of the physiology of living organisms.

II. Pathological physiology of some diseases of the duodenum.

In modern literature, there is no generally accepted understanding of the etiology and pathogenesis of sphincter Oddi dyskinesia (SOD). The most common phrase is that the cause of SOD is not known. There were offer speculative hypotheses that do not have any scientific confirmation: 1) A paradoxical response to endogenous hormones due to dysfunctional neurologic pathways may cause sphincter Oddi dyskinesia; 2) Sphincter of Oddi dyskinesia may lie at the hormonal and neurotransmitter level leading to sphincter of Oddi hypertension [13].

It is known that at SOD the basal pressure of SO can be higher or lower than normal. The pathological indicators are the basal pressure > 40 mm Hg, the frequency of contraction is more than 8 per minute and the proportion of retrograde peristalsis is more than 50% compared to the antegrad[17]. These characteristics indicated organic stenosis and served as the basis for sphincterotomy. But evaluation of long-term results showed that in many cases the source of symptoms was not related to SO. At the same time, the procedure itself sometimes led to severe complications [18].

Zhang et al, when examining 45 patients who underwent cholecystectomy with a T-shaped drainage in the common bile duct, revealed duodenobiliary reflux in 16 (36%) patients. The radioactive marker was found in the biliary tract 2 hours after ingestion. In most of them, a manometric examination revealed a decrease in the motor function of SO [19]. The literature shows the role of juxtapapillary duodenal diverticulum in the formation of gallbladder stones and other pancreaticobiliary diseases [20,21]. Both in this and in other articles, the condition was associated with SO pathology without any connection with the condition of the duodenum [13,17,18,19].

To the pathological physiology of pancreatobiliar diseases.

Analysis of the literature convincingly proves the correctness of the assumption expressed by Ochsner more than 100 years ago. There is every reason to believe that the hypersecretion of hydrochloric acid causes an increase in the tone of the Ochsner and Kapanji sphincters. Therefore, in the segment of the intestine between these sphincters, where SO is located, a high pressure arises, which leads to the reflux of acidic duodenal contents through SO. This leads to an increase in pressure in the biliary tract to their expansion, to chemical inflammation, stagnation of bile, and the formation of gallstones, and causes cholecystitis and pancreatitis when the microflora penetrates with bile. Thus, SOD occurs whenever the duodenal chyme crosses the sphincter barrier. It should be noted that until now, only the advanced degree of damage to the SO function has been diagnosed.

Dyskinesia of the duodenum.

It is a well-known fact that most duodenal ulcers are in the first part, even though the duodenal bulb mucous is better than all other parts protected from the aggressive effects of hydrochloric acid. This can be explained by impaired sphincters function. The increase in PBS tone results in the stagnation of the sour bolus in the bulb. If because of the duodenal expansion the tone of the Kapanji sphincter is reduced, then during the contraction of the Ochsner sphincter, the bolus re-enters the bulb. Both options contribute to the appearance of ulcers of the duodenal bulb (**Figure 4**).

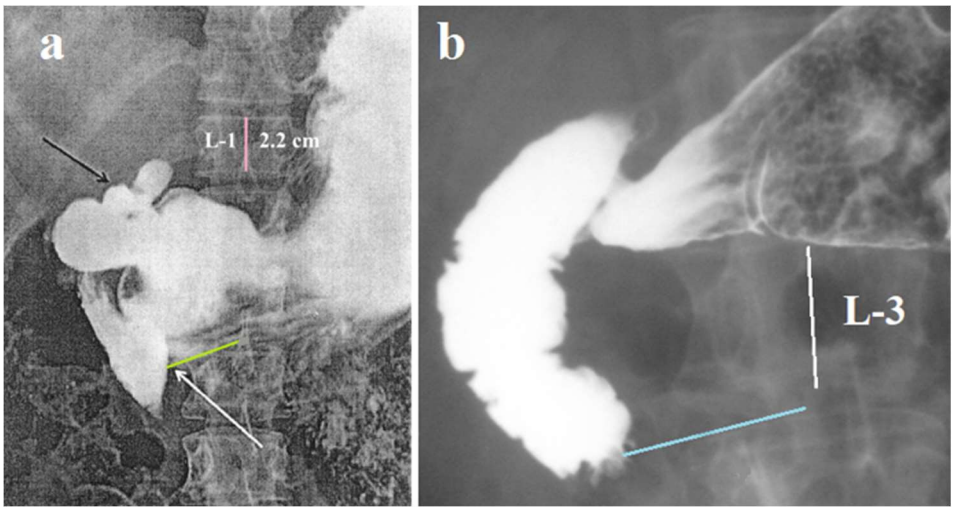

Figure 4. Radiographs of patients with duodenal dyskinesia. **(a)** Case described as superior mesenteric artery syndrome. The black arrow shows an ulcer in the duodenal bulb. White arrow shows the beginning of the narrowing in the third part of the duodenum, which is regarded as compressing the duodenum in the aorto-mesenteric angle. The pink line corresponds to the height of the 1st lumbar vertebra, which in adults is 2.2 cm. Consequently, the beginning of the narrowing of the duodenum is at a distance of 3 cm from the aorta. **(b)** There is zone of contraction of the duodenum , which begins 3.2 cm from the midline of the vertebra. The other sphincters (pyloric, post-bulbar, and Kapanji) have not contracted.

An analysis of the literature on superior mesenteric artery syndrome (SMAS) showed that the narrowing zone, which is interpreted as compression of the duodenum between the aorta and the superior mesenteric artery, starts at 3.30 ± 0.15 cm from the midline of the vertebra, where the aortomesenteric angle located. In position and length, it corresponds to the contracted sphincter of Ochsner (3.20 ± 0.15 cm) and has nothing to do with the aortomesenteric angle [22]. It always occurs with hypersecretion of gastric juice and therefore, at least in chronic course, accompanied by GERD [23]. Thus, the cause of SMAS is Ochsner sphincter dyskinesia.

Neri et al using the color Doppler studies detected reduced aortomesenteric angle <25° in 29 of the 950 patients with dyspepsia and/or abdominal pain who, according to their assessment, had SMAS during hypotonic duodenography with barium [24]. The diagnosis of SMAS was established solely on the basis of

abrupt bowel contraction distal to the dilated segment. Since then, detection of an aortomesenteric angle <25 ° has been considered the gold standard for diagnosing SMAS.

It is known that SMAS is one of the forms of duodenal obstruction. Any violation of patency is accompanied by the expansion of the stomach and duodenum. The main clinical symptom is recurrent vomiting. These symptoms are not mentioned in the article by Neri et al. On the presented X-ray there is no expansion of the stomach and duodenum. The narrowing in the 3rd part of the duodenum begins to the right of the vertebra and cannot be associated with the aortomesenteric angle **(Figure 5.a***)*.

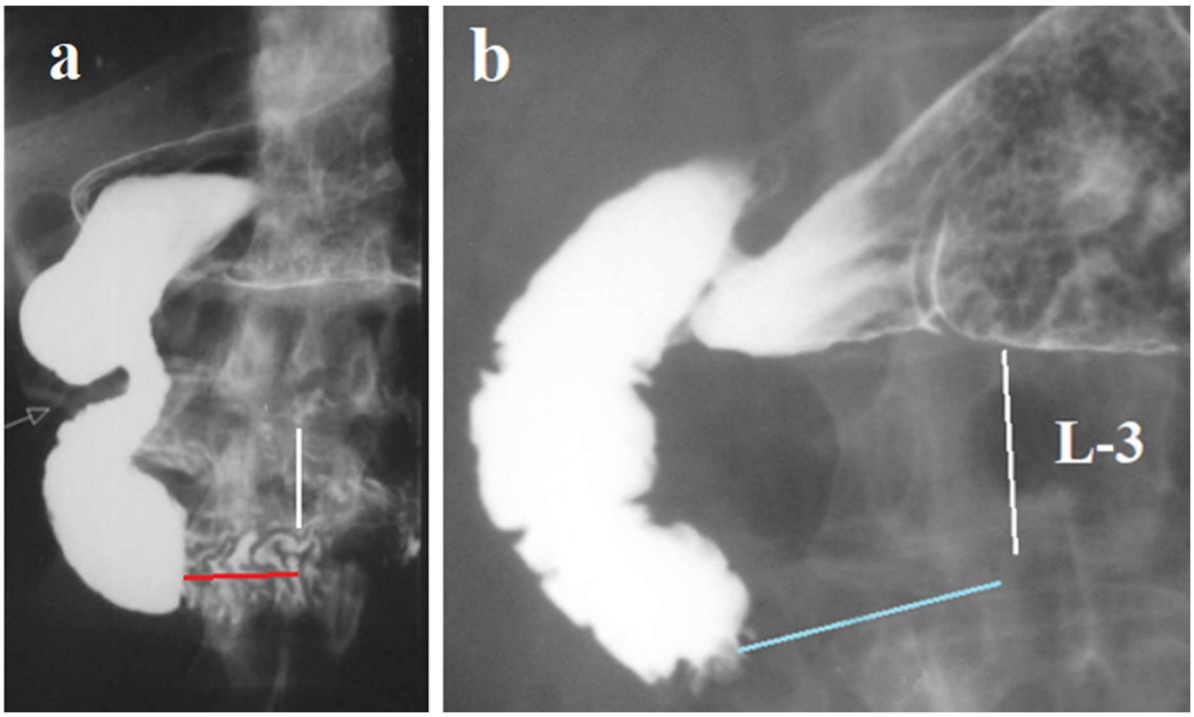

Figure 5. (a) Radiograph from the article by Neri et al. This is the only radiographs, which was supposed to prove the diagnosis of SMAS. Despite hypotonic duodenography the stomach and duodenum are not dilated. The constriction, about 2.5 cm long, which starts to the right of the vertebra, is caused by the contraction of the Ochsner sphincter. The white arrow shows the Kapanji sphincter. Obviously, this radiograph does not differ from the radiograph of our patient (b), who, unlike the patient Neri et al, did not undergo surgery.

Currently, it is believed that the violation of patency in the 3rd part of the duodenum is due to compression of the gut in the aortomesenteric angle. It is assumed that in healthy individuals' fatty tissue in this corner pushes the SMA away from the aorta, which increases the aortomesenteric angle and makes room for the duodenum. Conditions accompanied by the loss of fatty tissue lead to a decrease in the angle and the duodenum is compressed between the vessels. An analysis of the literature revealed the following facts that contradict the accepted concept of the pathogenesis of the SMAS. 1) low body weight is not a determining factor in the pathogenesis of the SMAS, since 23.7% [25] to 50% [26] of the patients have a normal BMI. 2) In third world countries, there are hundreds of millions of people with low BMI but the SMAS frequency does not increase. 3) Bhagirath Desai et al. did a prospective study of 100 patients who had undergone a CT scan for various other complaints. A strong positive correlation was found between BMI and the angle between the aorta and SMA. With BMI increase, the angle also increases. In 25% of patients, these rates were less than the norm, which indicates that the angle cannot serve as the gold standard for the diagnosis of SMAS [27]. Obviously, the authors of the article diagnosed SMAS in patients with severe duodenal dyskinesia with low weight. Due to a misdiagnosis, they falsely concluded that an aortomesenteric angle < 25 º is undeniable evidence of SMAS. In order to convince readers of the reliability of their conclusion, the authors referred to articles allegedly with the same results: "The aortomesenteric angle is normally 25–60 ° [2, 3, 6, 7, 10-12] and the mean aortomesenteric distance of 10-28 mm [1-3, 6, 7, 10-12]" [24]. However, none of these references published studies on the measurement of normal aortomesenteric angle and the distance between these vessels. Errors in science are possible, but a lie is inadmissible.

An article by Neri et al opened a pandora's box. Many articles have appeared in which duodenojejunostomy was performed in patients with Ochsner sphincter

dyskinesia, even without attempts at conservative treatment. An example is the article by Kirby et al, which describes four patients with esophagitis. They were operated on only because their aortomesenteric angle was < 28 º (why not 25º?) [28] (**Figure 6**).

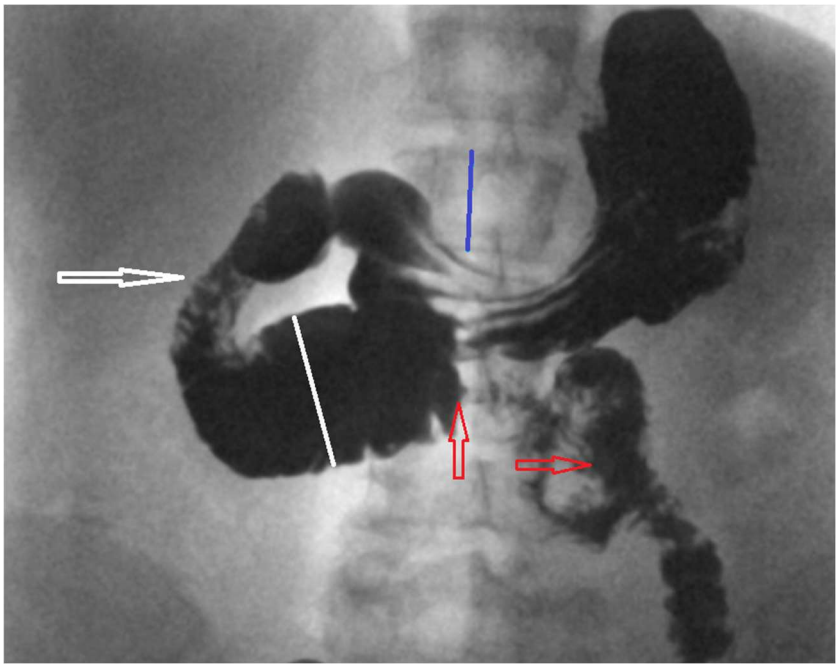

Figure 6. Rotated x-ray from article by Kirby et al. True height L-1 (blue line) is 2.2 cm. The stomach is not dilated. The width of the duodenum (white line) between the Kapanji sphincter (white arrow) and the Ochsner sphincter (between the red arrows 3.5 cm long) is 3.7 cm. Evacuation of barium into the jejunum began.

In this observation, there are no signs of duodenal obstruction. The extended duodenum between the contracting sphincters produces mixing of the chyme with bile and pancreatic juice. This is a typical picture of Ochsner sphincter dyskinesia, described more than 100 years ago. The combination of duodenal dyskinesia with esophagitis is not accidental. These concomitant problems are caused by the same etiological factor - hydrochloric acid hypersecretion. It is not surprising that the description of the clinical picture of GERD, gastritis, gastroparesis, duodenitis, and duodenal dyskinesia contains the same symptoms. And we do not know for certain the origin of each of the symptoms.

With a light hand, Neri et al, seriously ill patients with duodenal dyskinesia are operate only because they are thin and therefore have a narrow aortomesenteric angle. Dyskinesia of the Ochsner sphincter is always accompanied by the expansion of the proximal duodenum, because of which the PBS and Kapanji sphincter stretch and do not function. In such conditions, after duodenojejunostomy, a large volume of acid chyme from the stomach immediately enters the jejunum, i.e. a situation arises that should have been stopped by all sphincters of the duodenum. This situation is called dumping syndrome. “Early dumping occurs within 1 h after eating, when rapid emptying of food into the small intestine triggers rapid fluid shifts into the intestinal lumen and release of gastrointestinal hormones, resulting in gastrointestinal and vasomotor symptoms. Late dumping occurs 1-3 h after carbohydrate ingestion, caused by an incretin-driven hyperinsulinemic response resulting in hypoglycemia” [29]. It is also necessary to consider the damaging effect of hydrochloric acid on the intestinal mucosa. “If dietary approaches are unsuccessful, somatostatin analogues should be considered in patients with dumping syndrome and impaired quality of life. Surgical re-intervention or continuous enteral feeding may be necessary for treatment-refractory dumping syndrome, but outcomes are variable” [29].

"Superior mesenteric artery syndrome" is a severe form of Ochsner sphincter dyskinesia, which is characterized by symptoms of duodenal obstruction (vomiting, expansion of the stomach and duodenum, absence, or scanty evacuation from the duodenum). Conservative pathogenetic treatment of Ochsner sphincter dyskinesia [23] may be effective in most cases.

Functional dyspepsia (FD). One of the difficulties in dealing with Functional gastrointestinal disorders (FGIDs) is that there are no biochemical markers or structural abnormalities that can be used to objectively diagnose or monitor the

progression of these disorders. Diagnoses are based on medical history and physical examination. The Rome criteria for FGIDs have been developed by working committees of the Rome Foundation through literature review and a consensus process [30]. Disturbances of gastric and duodenal motor function such as gastroparesis and functional dyspepsia are not differentiated on clinical symptoms. For example, of all patients with idiopathic gastroparesis, 86% met the criteria for functional dyspepsia [31,32]. Analysis of the literature indicates that "Functional dyspepsia» are not diagnoses, since, firstly, there is no data on the etiology, pathological physiology, and pathogenesis of this condition. Secondly, there are no specific symptoms, since the early satiety, postprandial fullness, nausea, vomiting, belching, and bloating are typical for GERD, gastritis, duodenitis, and ulcerative lesions of the stomach and duodenum. If, as Talley et al argue, "gastroesophageal reflux disease and irritable bowel syndrome are part of the functional dyspepsia spectrum" [33], then the same can be said about gastritis and duodenitis.

From the article Oustamanolakis and Tack you can understand how the idea of FD came about. In cases where the cause of dyspepsia has been diagnosed (peptic ulcer, gastroesophageal reflux disease, gastric or esophageal cancer, pancreatic or biliary disorders, intolerance to food or drugs, and other infectious or systemic diseases), the authors denote the term "organic dyspepsia. In those cases, where the diagnosis was not established, they were designated as functional dyspepsia. Surprisingly, the authors classify genetic predisposition, infection from Helicobacter pylori, or other organisms as pathogenetic factors of FD [34]. It follows from this that gastritis and duodenitis caused by Helicobacter pylori or other organisms is FD, and when a patient develops an ulcer on the background of treatment with acid-suppressive drugs, eradication of H. pylori, prokinetic agents, fundus-relaxing drugs, antidepressants, and psychological interventions, it will already be organic dyspepsia.

From the analysis of the literature, it is obvious that the Roma IV criteria, which are adopted by voting, are not related to science. Научные достижения не могут быть анонимными. Who called abdominal pain in children with GERD "functional abdominal pain disorder" on the grounds that it does not match the typical triad of symptoms (chest pain, regurgitation, heartburn) and/or if the patient a pH <4 does not exceed 6 -10%? Constipation in children can be called functional constipation, but if it does not indicate which function is impaired, then this name has no meaning [30] (Figure 7).

Table 2 of 4

Table 2. Functional gastrointestinal disorders: children and adolescents.

H1. Functional nausea and vomiting disorders
H1a. Cyclic vomiting syndrome
H1b. Functional nausea and functional vomiting
H1c. Rumination syndrome
H1d. Aerophagia
H2. Functional abdominal pain disorders
H2a. Functional dyspepsia
H2b. Irritable bowel syndrome
H2c. Abdominal migraine
H2d. Functional abdominal pain – not otherwise specified
H3. Functional defecation disorders
H3a. Functional constipation
H3b. Nonretentive fecal incontinence

Figure 7. Table from article Koppen et al [30].

Of course, the term FD can be used by a physician when he sends a patient to a gastroenterological examination if the empirically prescribed treatment has not

improved the patient's condition. The examination of the patient should establish a diagnosis.

Analysis of the literature indicates that the cause of FD is the hypersecretion of hydrochloric acid. Therefore, the earliest disease that causes pathological symptoms is GERD. Unfortunately, modern ideas about the pathogenesis of GERD and the principles of examination reveal only very severe forms of GERD. Endoscopic examination without histology does not reveal esophagitis, but if there are no gross signs of esophagitis, histological examination is not recommended. Having a so-called hiatus hernia is mistakenly thought to be predisposing to GERD, when in fact it is evidence of GERD. pH-metry diagnoses only very severe forms of GERD [35]. This gives rise to the concept of FD, which is "classified by the Rome criteria as disorders of brain-gut interaction without structural alteration" [36]. As shown earlier, the hypersecretion of hydrochloric acid causes irritation and an inflammatory reaction in the esophagus [35], in the stomach [37], duodenum, and in the biliary tract. Dyskinesia of these organs is evidence of this effect.

Conclusions

1. X-ray examination of the digestive tract is physiological, since its results are clear, obvious and do not use foreign structures that can change the results, i.e. serve as artifacts.

2. In the duodenum 6 sphincters function (pyloric, postbulbar, Kapanji, Oddi, Ochsner and duodenojejunal), which provide a portioned evacuation of the bolus from the stomach and protect the small intestine from the damaging effects of hydrochloric acid.

3. Hypersecretion of hydrochloric acid leads to dysfunction of the duodenal sphincters (dyskinesia), irritation, and inflammation of all upper parts of the digestive tract, which manifests itself by different periods of ontogenesis with esophagitis, gastritis, duodenitis, and can be complicated by ulcerative lesions, and by malignant process.

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