Syndrome of functional dyspepsia and/or chronic gastritis?

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Function is inconceivable without structure, while structure is pointless without function.
V. H. Vasilenko

The syndrome of functional (gastric, gastroduo- disorders syndrome — FGDS) — is a functional denal dyspepsia — SFD (Functional gastroduodenal symptom, which, according to the latest revision of the Rome criteria (III, 2006), is characterized by the appearance of epigastric pain syndrome (epigastralgia) and dyspeptic manifestations, induced by food intake and localized in the epigastric ares, closer to the midline [45, 53].

Brief history. The term "dyspepsia" is derived from the Greek words dys (violation) and peptein (digestion) — "indigestion." More precisely, dyspepsia is number of clinical symptoms reflecting dysfunction of various organs of the digestive system. We must distinguish mainly gastric or gastroduodenal, dyspepsia; ileal and colonic, biliary, hepatic and pancreatic dyspepsia.

The emergence of the term (and concept) “syndrome of functional dyspepsia” (SFD) is described in the following way. In 1980, Multinational Working Teams to Develop Diagnostic Criteria for Functional Gastrointestinal Disorders was created. In 1984, W. Thompson introduced the first definition SFD, describing it as a "chronic recurrent, often associated with food intake, discomfort in the epigastric region, which forces suspected peptic ulcer disease, but this statement is not proved" [7, 67]. In 1988, at the World Congress of Gastroenterology in Rome a permanent Working Team Committee for functional disorders of the gastrointestinal tract was established, chaired by D. Drossman, and its part — Subcommittee on functional disorders of the stomach and duodenum led by N. Talley [38]. The first recommendations of this Committee for the diagnosis and treatment of functional gastrointestinal disorders appeared in 1994 as "Rome criteria I" [52]. In 1999 they published "Rome criteria II", and in 2006 — "Rome criteria III" [14, 44, 49]. Each revised definition, diagnostic criteria, the number and name of the clinical forms (variants), terminology and principles of treatment of SFD.

At various times, as the SFD synonyms use the term "irritable stomach", "essential and/or idiopathic dyspepsia", "endoscopically negative dyspepsia", "inorganic dyspepsia", "non-ulcer dyspepsia," "pseudo-ulcerous syndrome" et al., but in the end eventually settled on the term "functional dyspepsia syndrome" [7, 25, 54].

Thus, according to the proposed version, the story begins studying the SFD since 1980. We are ready to challenge this view and argue their position.
One of the giants of the domestic medicine V.P. Obraztsov (1849-1920) in his monograph, published posthumously by surviving manuscripts of his closest student N.D. Strazhesko in 1924 titled "Diseases of the stomach, intestines and peritoneum" [11] highlighted the chapter "Functional dyspepsia of stomach" (pp. 64-69), which gives the following definition of this syndrome: "Under the name of functional or nervous stomach dyspepsia we mean dyspepsia, based on which we have no certain structural changes of the stomach". And further: "The nervous functional dyspepsia along with dyspeptic phenomena observed in organic suffering of stomach — catarrh (chronic gastritis — Y.Ts.), ulcers, tumors, stomach extensions (gastroparesis — Y.Ts.) — exists in a large mass patients with dyspeptic complaints, the stomach which neither clinical nor random post-mortem studies clearly no changes, we do not find. This dyspepsia has to be attributed to the functional defects of the stomach and its function disorder — motor, sensory, etc." [11].

It is easy to make sure that the V.P. Obraztsov anticipated almost all the fundamental provisions of author-composer Rome criteria: definition of the nature of the process as a functional disorder, its considerable prevalence, the need to distinguish between organic and

functional dyspepsia, the value in its pathogenesis of disorders in the nervous system regulation, motor dysfunction and hypersensitivity [31].

Also in 1924, regardless of V.P. Obraztsov, «Der Reizmagen» ("irritated stomach") was published by German authors K. Westphal and W. Kuckuck [74]. It was observed that patients with irritable stomach, despite the presence of symptoms that resemble an ulcer, a thorough X-ray, endoscopy, functional (determining the secretory function of the stomach — Y.Ts.) and histological examination did not reveal any ulcers or gastritis. As a clinical and pathogenetic variant of "irritable stomach", these authors suggested to distinguish between hyper- and hypoergic forms depending on the predominance of the parasympathetic or sympathetic tone of the autonomic nervous system [74].

This excursion into the past indicates that the presence of SFD, its clinical manifestations and mechanisms of development were discussed long before the recommendations of the "Rome Criteria", and in the basic provisions of "Rome criteria" There is nothing new.

Prevalence of SFD is claimed is very high, especially in the developed countries of Europe and North America, where diarrheal gastroduodenal disorders occur in 30-40% of the population, especially at a young age and is more common in women than in men [7, 14, 35, 39]. To the doctor, however, refers only every fourth or fifth [38], which suggests that the symptoms of SFD is not too burdensome for the patient and in most cases did not significantly affect the quality of life (QOL). According to most authors who have studied the problem of SFD, often still found "functional" rather than "organic dyspepsia" — at a ratio of 50-70% and 30-50% [7, 31, 38, 39].

The essence of syndrome of functional dyspepsia, its etiology and pathogenesis. According to the definition given in the Rome criteria I-III, SFD **is a** functional symptom related to gastroduodenal area, which eliminates any organic, systemic and/or metabolic disease processes that can explain the existing symptoms [44, 49, 52].

Etiology of SFD to date is not certain. Not casually refer to it sometimes use the term "essential, or idiopathic dyspepsia" [56]. B. Cash [43] believes that the basis of SFD are not sufficiently adapted to the psychological reaction to stress influences, and SFD is considering how the psychosomatic process.

Even in 1992 opinions were expressed that the SFD can develop as a result of somatization, anxiety and depressive disorders [48, 62, 68]. These assumptions are confirmed by the effect of psychotherapy, hypnotherapy and destination of psychotropic drugs. Patients with SFD noted more pronounced than in the controls, the level of anxiety, depressive symptoms, neurotic and hypochondriac reactions [40]. Research etiological role of psycho-emotional disorders in SFD is not enough.

In the pathogenesis of SFD certainly proved the leading role of disorders of motor and evacuation function of the stomach and incoordination of gastric and duodenal motility.

Among the specific mechanisms of gastroduodenal dyspeptic disorders in SFD must name ccomodation stomach (40-50% of patients), which implies the absence of a relaxation of the proximal stomach after meals and adequate increase in the volume of the stomach. This leads to an increase in intragastric pressure, which leads to the appearance of pain and feeling of fullness in the epigastrium after eating.

Another manifestation of gastric motility disorders when SFD is dysrhythmia, which is characterized by a decrease (bradygastria) or (much less likely) increase (tachygastria) peristaltic activity of the stomach (at a rate of about 3 imp/min). This type of motility disorders of the stomach flows from stasis of its contents, and nausea (sometimes), vomiting, and epigastric fullness (from 23-59%).

Another form of movement disorders is discoordination (dyssynchronism) motor function of the stomach and duodenum. Normally, for each gastric contraction there are 4 duodenal contraction (12 contractions per minute).

Thus, when SFD prevail dyskinesia, hypokinesia until gastroparesis delayed gastric emptying in the duodenum (50% of patients). There is a chance of duodenal reflux. These data

were obtained electrogastrogram (4-channel electrogastrograph Digitrapper-EGG, USA) and standard gastric scintigraphy with radionuclide labeled food load determining rate and tempo evacuation of food from the stomach into the duodenum [15, 32].

Another factor in the pathogenesis of putative SFD is a visceral hypersensitivity of gastric mucosa (GM), its baro-, chemoreceptors to mechanical and conventional food stimuli, as well as reducing the threshold of sensitivity to stretching (in 50-90% of patients), but to prove this situation is extremely difficult. They allege that hypersensitivity coolant causes the appearance of pain, belching, and sometimes vomiting after meals (from 33-61%).

It is necessary to discuss the fundamental question about the alleged role of infection Helicobacter pylori (HP) in the SFD development. As you know, the Maastricht consensus on the diagnosis and treatment of HP-associated diseases follows a policy of total destruction on the HP (test and treat strategy — to identify and destroy). Most of the evidence of studies carried out mainly by foreign authors, does not confirm the existence of a definite connection between the HP-infection and SFD. It was found that the colonization of HP coolant is not accompanied by the appearance of any clinical symptoms, including dyspepsia: clinical relevance in HP-infection is completely absent [31]. Thus, dyspeptic syndrome can be caused by coolant HP colonization.

Studies by N. Talley [42] could not find evidence to support the role of HP-infection in the development of the SFD. F. Froehlich et al. [55] provided further evidence of the absence of any connection between the presence of HP in the stomach and the development of SFD. They believe that HP should not be a target for therapeutic intervention during SFD as HP eradication does not reduce the clinical symptoms (pain, gastric dyspepsia) and improve the quality of life of patients. R. Loffeld et al. [50] deny role HP, including their cytotoxic (CagA-positive) strains in SFD and etiology of need of eradication of these patients (HP) therapy.

P. Moayyedi et al. [73] conducted a thorough analysis of the multi-systematic reviews (Annals of Internal Medicine, and the updated data register-controlled studies of Cochrane), covering a large number of randomized controlled trials on the role of NO in the development of the SFD, which was published until 2002. The conclusion of these authors is clear: between the SFD and the HP infection is not a natural connection.

And other authors came to similar conclusions [46, 60, 69, 72]. The final line under the issue discussed spent L. Laine et al. [61] meta-analysis by the most evidence from randomized controlled trials. The authors concluded that the development of the SFD is not related to HP infection, so there is no reason to recommend the eradication of HP in patients with SFD, which is also ineffective.

Thus, the discussion on this topic can be considered exhausted. Summarizing the results of the discussion of this topic, P. Moayyedi et al. [46] sarcastically noted that decision-makers about HP eradication in patients with SFD, be prepared to pay for this treatment yourself. With regard to assumptions about the role of family history in the pathogenesis of SFD, the research evidence could not be found on this issue.

Clinical picture. The manifestations are diverse SFD. It is curious and instructive to trace the "terminological creativity" author-composer Rome criteria I-III.

Initially (Rome criteria-I, 1994), the drafters of the authors proposed to distinguish four clinical variants of SFD: ulcerous-like (with predominant pain syndrome); dyskinetic (with a predominance of dyspeptic symptoms) reflux-like (with the dominance of the symptom of heartburn) and nonspecific (with vague complaints which do not fit in any of the first three options). It is unclear on what principle is based division of clinical variants: ulcerous-like — in the presence of pain (complaints), dyskinetic — on dysmotility (function), nonspecific — atypical, hardly classified signs.

Back in I950, I. M. Pound [27] used the term " ulcerous-like option" when referring to a form of chronic gastritis (CG). In his proposals appeared and "tumor", and "dyspeptic" forms of CG. Thus, he wanted to emphasize that there is a painful form of CG (gastritis dolorosa), and the CG with a predominance of dyspeptic complaints, and CG that resembles the clinical

manifestations of gastric cancer. In our time, the drafters of the Rome criteria and their followers claim that CG is always asymptomatic (?!) [14, 25, 44], that does not correspond to reality.

Prominent gastroenterologist of those years, O.L. Gordon [2] objected to the division of CG on ulcerous-like and tumour-like, but only because of ethical considerations: impressionable, hypochondriac patients, these terms can cause anxiety, even panic for fear that CG may they transform into cancer and gastric ulcer. In clinical epidemiology is a medical term mental impact on patients has been called "the effect of the label" [26].

SFD as a synonym for a long time appeared the term "non-ulcer dyspepsia» (non-ulcer dyspepsia), which is widely used abroad and in our country [25, 35, 40, 42, 46, 55,61, 67, 69]. The term is clearly bad. For example, there is a real "monster terminology" if necessary to designate ulcerous-like version of SFD "ulcerous-like form of non-ulcer dyspepsia" (!). Furthermore, this allows the possibility of the term when SFD various organic diseases of the stomach, with the exception of peptic ulcer disease [29, 31].

Failures and the term "non-specific form of SFD": essentially all clinical variants of SFD are nonspecific. We are staunch opponents of the terms starting with the denial — the particle "not" (non-ulcer, nonspecific, and others.): They unwittingly raise the question: "If it is not ulcerative, what is it?". An analogue of the term "non-ulcer dyspepsia" is not SFD, but "peptic disease without ulcer" or "latent peptic ulcer disease" [29, 31].

In contrast to the Rome criteria-I in Rome criteria-II remained only 3 of 4 clinical variants of SFD: deleted "reflux-like option" with the dominant symptom of acid reflux, which decided to move to "gastroesophageal reflux disease" (GERD) without endoscopic signs of reflux oesophagitis — in endoscopically negative form of GERD [49].

The Rome III criteria included only 2 clinical variants of SFD: deleted "non-specific option." The "ulcerous-like option" of SFD was renamed into "painful option" (epigastric pain syndrome — EPS), and the "dyskinetic option" (meal-induced dyspeptic syndrome — MIDS). In this case somehow it suggested another, an alternative name for it (postprandial distress-syndrome — PDS), which we believe is unfortunate and unnecessary.

As is known, the terms "stress" and "distress" were at one time offered by H. Selye [19], so we turn to the source. By definition, H. Selye, "stress (stress) is a non-specific response of the body to any claim brought against him. Stress is is the sum of all non-specific phenomena (including damage and defense), stress is not just a nervous tension..; stress should not be avoided..; stress is the aroma and taste of life..; complete freedom from stress is death. Distress is a "malicious or bad stress; it is a grief, distress, exhaustion, malaise" [19]. The question arises, how does dyspeptic version of SFD to distress? Why this term to refer to recommend it dyspeptic version of SFD, rather than, for example, a painful option? Clear answer to these questions is no.

According to the Rome criteria III, painful option SFD (EPS) is diagnosed on the basis of the following criteria:

- recurrent pain and/or burning sensation in the epigastrium, usually of moderate intensity, with a frequency of at least 1 time per week;
- absence of generalized pain and/or pain, localized in other parts of the abdomen (except epigastric), or chest and a lack of persistent heartburn;
- epigastric pain not disappear after passing flatus and bowel movement;
- absence of symptoms of dysfunction of the gallbladder and sphincter of Oddi.

The note states that the pain may be burning in nature, but should not be subject to the retrosternal and epigastric pain, which usually occurs after meals, can sometimes appear empty stomach. In addition, pain SFD version (EPS) may in some cases be combined with dyspeptic version (MIDS). Dyspeptic SFD version (MIDS) is characterized by the following features:

- emergence of disturbing feeling of fullness in the epigastrium after taking the usual amount of food (the former name of "discomfort"), at least several times a week. Replacement of concise and exact term "discomfort" in the verbose description of the patient's sensations we feel inadequate justified;

- feeling of early satiety (fullness), preventing the reception of normal volume of food that appears several times a week. In addition, swelling in the upper abdomen and/or nausea and excessive burping after a meal may arise. It is noted that dyspeptic version of SFD (MIDS) in some cases may be associated with pain (TDS).

In addition, Rome III criteria twice reduced the time criterion in the diagnosis of SFD: gastroduodenal dyspepsia symptoms should be observed for 12 weeks over the past 6 months (instead of 12 months as previously). It is easy to notice the uncertainty, "fragility" of diagnostic criteria of clinical variants of SFD, which also can be combined with each other. The criteria for diagnosis of SFD also include symptoms that are designed to help in the differential diagnosis of SFD and irritable bowel syndrome (IBS) and GERD, but their value is impaired indicating the possibility of the combined flow of SFD and IBS (in 12-30% of cases), and GERD (22-24%) [3, 23], which extremely complicates diagnostics of SFD.

The most important condition of SFD diagnostics is its distinction with the "organic dyspepsia". Functional dyspepsia occurs in about 2 times more likely than organic — 65 and 35% of cases [36].

Rome criteria numerate the main diseases of the upper digestive tract, which can cause "organic dyspepsia": peptic ulcer (PU); gastric cancer (GC); GERD and its complications, including Barrett's esophagus and adenocarcinoma of the esophagus; chronic cholecystitis, including calculous one; chronic pancreatitis.

Noteworthy is the fact that in this list for some reason not included CG — the most common organic disease of the digestive system. It is surprising and even bewildering.

Trying to explain the reason for exclusion from the list of CG organic diseases, contrary to the diagnosis of SFD, usually refer to the possibility of a long latency flow CG and the fact if the diagnosis of chronic hepatitis — this is a purely morphological rather than clinical diagnosis.

It is impossible to accept this. The latent CG course really occurs in about 50% of cases. But for a long period of latency observed in other diseases, including gastrointestinal: with cholelithiasis, gastric cancer and others. Clinicians are well aware that upon CG are possible pain, and dyspepsia.

Thus, in non-atrophic antral CG intragastric pH monitoring records often hyperacidity and electrogastrography — hyper- and dyskinesia of the stomach with a predominance of pain, upon fundal atrophic CG — hypoacidity up to achlorhydria, hypo- and dyskinesia of the stomach with the prevalence of dyspeptic symptoms (belching, nausea, decreased appetite, heaviness in the pit of the stomach after a meal, unpleasant taste in the mouth, and others.) [33]. These findings are confirmed by other authors who have studied this issue [28, 34].

Thus, we can confidently assert that the CG is not only morphological, but also the clinical concept. Uncertainty, ambiguity item author-composer Rome criteria for CG has spawned the emergence of such a freak of terminology such as "CG with functional dyspepsia syndrome", in which two mutually combined pathological process: CG and SFD, organic and functional dyspepsia [12].

In Japan, where some authors also abuse such a diagnosis, there was a landmark publication [57]. Group of authors for 10 years have seen more than 1 thousand of patients with various gastroduodenal diseases associated with HP infection. By the end of the observation period are diagnosed with the development of gastric cancer in 2.2% of patients with hyperplastic gastric polyps, 3.4% of patients with gastric ulcer and 3.7% of patients with SFD (?!); at PU of duodenum were observed incidence of gastric cancer. Based on these data, these authors suggested the inclusion of patients with SFD in the group of patients with precancerous diseases (?!).

It is obvious that as a functional SFD syndrome having no morphological substrate, it is not able to evolve GC. The development of GC in a small proportion of the observed patients with SFD in fact due to the presence in them of atrophic forms of CG, which is hidden behind the "facade" (diagnosis) SFD combined with CG [57]. This is an example for how paradoxical erroneous conclusions can arrive at overlapping diagnoses SFD and CG [29].

Pajares H. Garcia [13] believes that the CG is an independent disease (nosological form) having its etiology, pathogenesis, clinical and histopathologic and SFD — a functional clinical syndrome that has no morphological substrate.

It may be added that the SFD as a functional syndrome usually is not progressing and has a favorable course and outcome. At the same time CG is a progressive organic process occurring with increasing atrophy of the glandular epithelium and secretory insufficiency up to the gastric ahilii with increased risk of gastric cancer [29]. That is why the combination of diagnoses SFD and HG invalid because thereby violated the very foundation on which is based the doctrine of functional disorders of the gastrointestinal tract, and the distinction is meaningless functional and organic dyspepsia [29].

Another important issue to be discussed: SFD is not a nosological and syndromal diagnosis that is treated as final. Some domestic authors categorically state: "SFD is a separate disease unit, and is encoded in the International Classification of Diseases, 10th revision (ICD-10, 1995) by code K30" [41]. Both of these assertions are at least controversial. Recently, foreign publications can be traced very negative trend: the substitution of nosological forms of syndromic diagnosis — unjustified "nosologization" of syndromes.

Leading domestic pathologists D.S. Sarkisov [17] and V.V. Serov [22] believed that "syndromic diagnosis is a diagnosis of ignorance", stressing: "syndrome is pathogenetic concept and nosology — etiological one". An outstanding clinician and scientist V.H. Vasilenko [1] was also an opponent of syndromic diagnosis. "Syndromic diagnosis — he thought — leads us away from the essence of disease" [1]. Another major clinician and scientist I.A. Kassirsky [5] pointed out: "Some are trying to hide their inability to understand the true nosology for syndromic diagnosis".

Syndrome is an intermediate stage in the nosological diagnosis. V.V Serov [22] rightly observed: "Syndromes of about 1.5 thousand, and disease entities, pathogenesis of which is ensured by these syndromes, more than 20 thousand". In some cases, under the guise of the syndromic diagnosis of hidden danger, but until that time, organic compensated up to pathological processes of cancer.

Not confirmed by the assertion that the SFD is reflected in the ICD-10. Under the codes K30, to which the authors refer to the above-mentioned [41], appears indigestion, but without specifying: functional or organic; special mention of the SFD is not in contrast to the CMB, which is presented in the ICD-10, three cipher: K58.0 (IBS with diarrhea), K59.0 (constipation) and K58.9 (painful form IBS). Another controversial issue that requires discussion: "Is it possible to recognize the existence of a purely functional diseases and syndromes?"

The division of diseases and syndromes in the functional and organic adopted in clinical medicine, is justified protest by the pathologists who defend the unity of structure and function, and consider all the so-called functional diseases and syndromes of structural and functional. D.S. Sarkisov stated: "It is always possible to find morphological changes corresponding to changes in a subtle and dynamic functions" [17, 18]. On the indissoluble unity of structure and function, V.H. Vasilenko said in his bright form and precise within the meaning of the aphorism, rendered as an epigraph to this article [20]. Pathomorphologists prove that with the help of electron microscopy, histochemistry, molecular biology and genetics the so-called functional diseases and syndromes are usually defined structural changes of cell membranes, the nuclear and cytoplasmic organelles receptor system, etc. [18, 21].

Differences between the thin structural changes, detectable under the so-called functional diseases and syndromes, with the help of ultra-modern diagnostic methods (electron microscopy, histochemistry, etc.), And gross morphological changes in the structure of organs and tissues, determined by light microscopy, consists, in our opinion, in reversibility and irreversibility of the first second, which are also prone to progression.

The diagnosis of SFD is a diagnosis of exclusion. We need differential diagnosis of SFD and all diseases of organic nature, which may cause "organic dyspepsia", including CG.

A careful history should give the doctor important information about the way of life of the patient, presence and frequency of stress situations, psycho-emotional status of patients, bad habits (smoking, alcoholism, drug addiction), food habits and preferences, diet, abuse of strong coffee, especially before going to bed, and m. n. It is important to establish whether the patient does not receive pharmacological agents that have irritative and damaging effect on the coolant (nonsteroidal antiinflammatory drugs, etc.).

For the differential diagnosis of SFD and organic diseases that can cause the appearance of "organic dyspepsia", using all the modern arsenal of instrumental and laboratory diagnostics (esophagogastroduodenoscopy, ultrasound, computed tomography, etc.), including biopsy and morphological examination of gastric biopsies, total and biochemical blood tests, daily intragastric pH-metry, electrogastrography, gastroscintigraphy; determination of gastric mucosa colonization by HP (non-invasive and invasive methods), and others. It is advisable to evaluate the QOL of patients with SFD with a special SF-36 and the index dyspepsia Nepean [51].

Rome criteria II and III offered a list of symptoms, which (if any) completely exclude the diagnosis of SFD called “alarm syndromes”, or “red flags syndromes”. The most important of them is that this is a symptom of progressive dysphagia; haehmatemesis and bloody feces (meteena); fever; unmotivated weight loss; anemia, leukocytosis, elevated erythrocyte sedimentation rate; pathological changes in the biochemical analysis of blood; the appearance of the first symptoms of dyspepsia after 45-50 years [14, 44, 49]. Have any of these symptoms exclude the diagnosis of SFD.

Treatment. Patients with SFD must have comprehensive and individualized treatment. The most important element of treatment is to streamline the way of life (lifestyle modification): sleep and wakefulness, work and rest, giving up smoking and drinking alcohol. It should be possible, avoid taking drugs, are irritating to the stomach, as well as stress situations, conflicts and quarrels. In this regard, expressed the view that the need for education of patients (education), stress relief (reassurance) [39]. Patients with SFD need psychological support.

Medical nutrition in SFD has not been developed. In view of the pathogenesis of this syndrome should be advised to split meals in small portions with the exception of mechanical coarse varieties of food, sharp, irritating foods, different spices, refried, marinated, smoked and salty foods. It is important to take into account the patient's personal experience in relation to tolerance of certain foods and dishes. Pharmacotherapy should set realistic treatment goals [39, 59].

In connection with the expected value of the psycho-emotional and psychosocial factors and related anxiety-depressive and personality disorders in the development of the SFD, it is recommended to use psycho- and hypnotherapy [25, 30, 39, 43, 62]. E. Calvert et al. [63] used hypnotherapy in the treatment of 126 patients with SFD for 16 weeks; long-term results were followed for 56 weeks. According to long-term results of clinical symptoms, improvement was observed in 73% of patients (versus 43% for pharmacotherapy; $p < 0.05$), and improved quality of life — 44% (vs. 20%; $p < 0.05$). After treatment, patients less likely to seek medical attention and take less medication, and that has provided some economic benefits.

From psychotropics most commonly used antidepressants balanced effect and a bicyclic structure: paroxetine (paxil) with antidepressant and anti-anxiety effect at a dose of 20 mg/day. Another drug in this group — citalopram (tsipramil) is used at a dose of 20-40 mg/day for a long time. From anxiolytic (antianxiety agents) — benzodiazepine derivatives positive effect was achieved when using grandaxinum (tofizopam) 50-100 mg 1-3 times a day for 2 weeks; the drug has a positive effect on psychovegetative syndrome, it eliminates anxiety and promotes correction of functional disorders [14, 16, 24, 30, 71]. Due to the side effects of anxiolytics is prescribed for a short course.

Upon pain form of SFD doctors recommend the appointment of proton pump inhibitors: omeprazole (20 mg 2 times a day), or rabeprazole (10-20 mg 2 times a day within 3-4 weeks). Their effectiveness in SFD, however, only an average of 39.5% (placebo 30%) [23, 29, 38, 39, 40, 41, 47]. The use of proton pump inhibitors, in our opinion, is not sufficiently substantiated.

Occasionally these patients are prescribed antacids (maalox suspension, phosphalugel, etc.) with questionable effect. For the impact on the main pathogenetic factor of SFD — a violation of the motor-evacuation function of the stomach — use modern prokinetics enhancing and normalizing gastric motility. High evaluation for the treatment of SFD deserves a new prokinetic itopride hydrochloride (Ganaton), has a double effect — antidopaminergic and anticholinesterase; it was also an analgesic. The drug is administered at a dose of 100 mg 3 times daily for 8 weeks. The analgesic effect is achieved in 59-64% of patients (placebo 41%, $p < 0.05$), feeling of heaviness and overflow epigastric disappear in 73% (placebo 63%, $p = 0.04$), quality of life improved by 18 ± 21, 9 points (placebo 13,2 ± 19,4 points; $p = 0.02$). The effect was evaluated on a 6-point scale (Lidsovsky questionnaire dyspepsia — Leeds dyspepsia questionnaire — LDQ) [1, 7, 37].

In dyspeptic variant SFD justified the appointment prokinetics — domperidone (Motilium) and metoclopramide (Cerucalum), which act as antagonists of dopamine receptors in the chemoreceptor trigger zone (10 mg 3 times daily for 3-4 weeks) [10, 23, 29, 31, 39].

Special attention should be paid to trimebutin (debridat, trimedat) — an antagonist of opiate receptors, acting on the motor system enkephalinergic regulation. Having an affinity for the regulators of excitation and inhibition of motor, trimebutine has modulating (normalizing) effect as in hyper- and hypodyskinesis of stomach and duodenum (100-200 mg 3 times daily for 3-4 weeks).

Of the alternative methods of treatment SFD, STW 5-II received a positive assessment, made of medicinal herbs (extract pods hot pepper, color masterbatch grass, peppermint leaves, caraway field, licorice root and lemon balm). In the treatment of 120 patients with SFD for three consecutive four-week courses noted the disappearance of clinical symptoms after the first 4 weeks 43.4% (3.3% placebo, $p < 0.01$) [70].

Compilers of Rome criteria-I-III, as well as their followers, referring to the indications of the Maastricht consensus, recommend eradication of HP in patients with SFD, while recognizing its dubious efficacy [10, 12, 14, 25, 44, 49, 52]. We are convinced that the eradication of HP in no way justified in SFD as:

• HP is not involved in the pathogenesis of SFD and (especially) it is not an etiological factor [7, 13];

• HP is detected in the stomach of patients with SFD in 46- 64.8% of cases, which is less than the general population (85-91%) [7, 14];

• HP after successful eradication already at 6 months at 38.7% of the patients symptoms recur SFD with no HP in the stomach [6, 7];

• frequency and severity of pain and dyspeptic syndromes in SFD infected and not infected with HP do not differ [7, 41];

• SFD elimination of symptoms after eradication of HP is less than 25%, which does not differ from the placebo effect (30%) [8];

• undue HP eradication upon SFD promotes selection of resistant to cytotoxic therapy and HP strains, improves colonization of stomach by fungi of the genus Candida (from 16.5 to 33%) [4, 8, 9, 29];

• HP eradication in the stomach in patients with SFD contributes to frequent GERD and its severe complications [58, 65].

Only a few publications are trying to prove the feasibility of eradication of HP with the SFD, but in a strange way in each of them the lead author is head of the "Maastricht" group P. Malfertheiner [54].

Concluding the review of the problem of SFD, we can say that the vagueness and inconsistency of many provisions of the Rome criteria I-III of the definitions and diagnostic criteria for SFD, its demands differentiation with IBS and GERD, while recognizing the possibility of a combined course, the selection of clinical forms of SFD and justification their differences and at the same time an indication of the fact that their symptoms can occur in both clinical variants, terminological mess — all this undermines the credibility of the doctrine of the SFD. And the need to use many modern methods of instrumental and laboratory examination for

differential diagnosis and differentiation of functional and organic dyspepsia extremely complicated and expensive process [29, 31, 38, 39]. Finally, the fact that the authors-compilers Rome criteria is subject to overlapping diagnoses SFD and CG, functional and organic dyspepsia, it destroys the foundation on which rests the doctrine of functional gastroduodenal syndromes.

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Syndrome of functional dyspepsia and/or chronic gastritis?

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Key words: functional dyspepsia, chronic gastritis, diagnostics, treatment, efficacy

Definition and prevalence of syndrome of functional dyspepsia (SFD) are discussed along with the brief history of the problem, priority works of V. P. Obraztsov and other authors, current views of SFD etiology and pathogenesis with reference to the role of H.pylori infections. Clinical variants of SFD, their diagnostic criteria and principals of differential diagnostics are described. Special attention is given to the debatable relationship between CFD and chronic gastritis. Nosological and syndromal diagnostics, structure-function relationship, goals and methods of SFD treatment and other matters of dispute are considered.