Persistent delusions of modern gastroenterology

Y. S. Tsimmerman

Perm State Medical University n. a. E. A. Vagner, Perm, Russia

Everyone can err, but to persevere in their errors can only fool

Cicero (106 — 43 BC)

Thousands of paths lead to confusion, to the truth — only one

Jean-Jacques Rousseau (1712 — 1778)

Key words: gastroenterology, delusions, Helicobacter pylori, functional gastrointestinal disorders, syndromic diagnosis, placebo, terminology

Constant in modern gastroenterology observed, as we see it, the two erroneous tendencies: 1. ineradicable committed to the perception of attitudes and ideas prevailing in foreign scientific publications, considering them as something not to be questioned and criticized as "ultimate truth" and 2. the tendency to blindly follow "fashionable" research areas (trends), many current gastroenterological problems.

Outstanding Russian pathologist VV. Serov (1924 — 2007) in 1992 stated: "Some of the fundamental provisions of our scientists considered" under hypnosis "different authors abroad" [20]. A renowned cardiologist EI. Chazov (born 1929) stated, "The best judge of science is time, the history of medicine puts everything in its place — the present and the superficial — "fashionable" [44]

So, in our view, overly exaggerated the role of Helicobacter pylori (Hp) in the development of gastroduodenal inflectional diseases. Facts are ignored about proliferating new authentic facts to refute the lead role of these bacteria in the genesis of common gastrointestinal diseases, such as chronic gastritis (CG), peptic PU (PU) and gastric cancer each year (GC).

It is a concept fundamental objections, declaring the possibility of the existence of functional gastrointestinal disorders (functional gastrointestinal disorders) without corresponding to the preceding structural changes (syndrome of functional gastroduodenal dyspepsia — SFD; irritable bowel syndrome), allegedly without morphological equivalent.

We consider it inadmissible to constantly practiced use of foreign researchers placebo ("dummy") in clinical trials of new pharmacological agents not getting the free consent of patients for these studies, it is desirable — in written form.

Unacceptable for the clinician and the substitution of nosological diagnosis — syndromic diagnosis, widely spread over abroad (SFD and IBS), and giving the latter the status of the final diagnosis.

Cause rejection of numerous "consensus" (conciliation meeting) and compiled on the basis of their recommendations on the diagnosis and treatment because they are contrary to the foundations of nym principles of EBM (evidence-based medicine). In particular, it can be called, "Maastricht consensus -1 — 4" for the testimony of the sacred to the eradication of Hp with Hp-associated gastroduodenal diseases, methods of diagnosis of Hp infection and its eradication therapy; "Rome Consensus (criteria)-I-IV», regulating diagnosis and treatment of the so-called (TN.) Functional gastrointestinal disorders (SFD and IBS); "Kyoto

consensus", regarded principles etiologic classification and morphological criteria for chronic ha Street (hCG), and others.

Their authors, compilers do not reckon with the fact that it ignores important elements of knowledge as a clinical logic of thinking, analysis and synthesis to clinical facts; reduced physician competence, his humanity, the ability to perfection Vat his art of healing. The doctor becomes a simple tech skim performer set of recommendations, which is contrary to the fundamental principles of medical practice and.

Even in the XIX century, the famous German philosopher Ludwig Fejerbach (1804 — 1872) stated: "Only he who thinks, is free and self-sufficient."

There are other errors, debatable and controversial problems of gastroenterology, requiring objective considerations and solutions.

Progress in science discussions is absolutely necessary. We are impressed by the statement ancient Greek historian Herodotus, who lived in the V century BC, which stated: "If it is not expressed opposing views, then do not choose from the best."

I. We must start with the most urgent problems — the role of Hp infection in the development of gastroduodenal diseases such as chronic hepatitis, PU and gastric cancer.

As is known, Hp — very common gram-negative microaerophilic, spiral-shaped bacteria that are infected 35 — 50% of the population of Europe and North America, and 90 — 95% of people in developing countries in Asia, Africa and Latin America — on average, 60% of the world population.

Hp — a non-invasive microorganism life activity is limited to the gastric mucosa (GM): supraepithelial mucus, the outer surface of a single layer of cylindrical epithelium ray stomach (between the fibers), and (in part) intracellular dimensional space.

It is important to note the following Hp infection:

1. Most people infected with Hp (70%) — often is healthy bacillicarriers— lifelong.

2. The clinical consequences of Hp vital functions in the human stomach in the form of various gastroduodenal diseases are observed in only 1% of them [9].

3. If coolant Hp colonization clinical symptoms (clinical relevance) completely absent [50, 54].

These facts were the basis for the famous Russian microbiologist SV. Sidorenko make the following judgment: "Widespread Hp-infection in persons with no signs of pathology — this is a powerful argument that refutes a leading role Helicobacter pylori in the development of gastroduodenal diseases" [21].

4. Supporters of the concept of the primacy of Hp with gastroduodenal pathology allege that in the stomach of these bacteria are not tons of competitors, and in case of detection of other microorganisms announce their transient microflora, unable to colonize the stomach.

At the same time, we conducted (in collaboration with microbiologists) study of gastric microbiota adapted modern methods of microbiological research refutes this claim.

Thus, when hCG in 82.1 ± 7.2% in the antrum cases revealed the presence of 105 species of microorganisms colonizing ing coolant, including a microbial associations — In 55.7%, etc. When in use, is not dominated by Hp, and streptococci (52,5 ± 4,4%), staphylococci (23,0 ± 2,1%), Enterobacteriaceae (9,8 ± 2,8%), Corynebacterium (9,8 ± 3,0%) and fungi of the genus Candida (19,7 ± 1,7%). Hp were detected in 18 ± 3,6%, and always in association with another microbiota [30, 38]. Highlighted in the microflora of coolant had usually adhesiveness and (in some cases significant) invasiveness (unlike Hp) and pathogenic properties (45,5

± 6,7%), including urease activity (27,3 ± 0, 6%). This was the basis to consider nye identified microorganisms in the stomach is not transient, and mucosal micro flora (M-microflora) [38] that can lead to the development of infectious-inflammatory process in the stomach (hCG).

At PU, as we have established, periulcerous zone 93 species colonize microorganisms that were found in 90.5% of patients; prevailed among them streptococci (57,1 ± 3,1%), Helicobacter pylori (52,4 ± 3,0%), fungi of the genus Candida (40,5 ± 1,5%), Enterobacteriaceae (9,5 ± 3 0%), Corynebacterium (7,1 ± 2,3%) et al., most often in the form of microbial associations (69.4%). Signs of pathogenic STI set at 56,4 ± 6,7% of them [30, 38].

Famous scientists, gastroenterologists M. J. Braser and S. Falkov (USA) also suggests that "the addition Hp, stomach exhibit numerous other bacterial species" [59]. Confirm data and domestic gastroenteritis logs obtained by us [45].

Thus, in the stomach in patients with chronic hepatitis and PU do not gelikobakterioza determined and dysbiosis gastroduodenal zone, with a significant portion dedicated microbiota has pathogenicity bubbled properties and may contribute to the development of chronic hepatitis and PU, along with Hp, and (very likely) and without their participation [31, 43].

The views of the supporters of the concept of the decisive role of Hp infection in PU development most clearly reflected in the numerous postulates DY Graham (USA): "No Hp — no PU"; "Hp — pathogen that is the main cause of PU "; "PU should be regarded as alocal manifestation cial bacteria infection (Hp)»; "Eradication of Hp leads to full treatment of PU"; "Duodenal PU — infectious process, two antimicrobial therapy treats the cause, not a structural defect "; "PU goes down in history" and others. [66].

It should immediately be said that none of the postulates DY Graham did not find its confirmation.

Thus, despite the efforts undertaken by s, it failed to prove the etiologic role of Hp at PU. For recognition of Hp, like any other organism, must be the cause of the disease is known to its compliance with three requirements (conditions) P. Koch ("triad" by Koch), however, Hp, an etiological factor PU does not correspond to the two of them.

The first requirement states: "microagent must always be detected in the patient" (in this case at PU). However, as it turned out, a large proportion of cases PU develops without all of the COG on the participation of Hp, — it Hp-negative forms of PU in which to establish the presence of Hp in the stomach (using 2 — 3 methods of diagnostics) failed [3, 13, 60 68, 72].

Hp-negative forms PU identified with a frequency of 20 — 30% to 50% as in the stomach and when duodenal venous defect localization [3, 13, 60, 68, 72]. In the US, the frequency of PU, not associated with Hp, varies from 39 to 52% (Schubert et al.; Sprung et al.), And in Austria Lee reaches 45% (Henry et al.) [1, 9].

Thus, the postulate DY Graham "No Hp — no PU" was misleading, and (at the suggestion of the famous Dutch gastroenterologist GNJ Tytgat) was replaced by a new "No Hp — no Hp-associated PU", recognizes the existence of Hp-negative forms of PU [74].

The second requirement (condition) "triad" Koch "is worded as follows:" Microbe-pathogen (Hp), highlighted in pure culture, when introduced into the body susceptible to it man should cause his development of the same for the disease (PU)".

To prove the etiologic role of Hp at PU is one of the "discoverers" Hp BJ Marshall introduced his stomach concentrated suspension of pure culture of Hp (109 microbial bodies). After 7 — 10 days he developed typical clinical symptom s acute gastritis, not PU, which soon disappeared without ka cal any consequences.

Repeated experiments with self-infection culture Hp gave the same result [70].

Outstanding pathologist our country IV. Davydovsky (1887 — 1968) argued that "one-uniqueness factor can never be the whole reason — it is only a necessary part of the reasons for the same is not always the most important" and "the reason that does not work, does not have reason" [7].

Thus, to confirm the etiological role of Hp failed at PU. And postulates DY Graham: «H — Pathogenic microorganism, which is the main cause of PU "and others, but PU etiology remains unknown.

The same conclusion was reached in the long run and one of the Naib Lee consecutive Storo CENI etiological significance Hp at PU — LI. Aruin (gastroenterologist, morphology), "PU is not a "classic" infection: one Hp infection is not sufficient for its occurrence. Any attempt to detect any one or etiological patogenetiche lish factor (critical unit) responsible for the development of the PU, is doomed to failure ". [2]

Already mentioned the American gastroenterologist MJ Blaser said that "the development of PU associated with Hp, — it is not a natural outcome of the interaction between man and microbe, and the result is a random" imbalance "between Hp and his" master ",carrying with it certain, only his inherent signs (genetic predisposition to PU; special structure and reactivity of the immune system — Ya.Ts).. Only in case of accidental "coincidence" to con the differentiated features of man and microbe may occur conditions for the development of PU. In other cases, the person remains healthy bacillicarriers or he develops chronic hepatitis, "[57].

Neither confirmed postulate DY Graham, that "eradication of Hp leads to a complete cure PU", whereby "PU goes down in history."

Recently, one of the most staunch followers concept of the leading role in the etiology Hp PU in our country (IV. Maiev) in an article on modern trends in the study Research Institute of the stomach and duodenum, and was forced to admit: "In spite of the already many years of active struggle... with Hp-infection, the prevalence of PU in our country, and in most countries is not reduced. Consistently high (at 10%) remains in the frequency of its formidable s complicated (perforation, penetration, bleeding, etc.). "[4].

Consequently, the active eradication therapy, aimed at the destruction of Hp tion and healing PU, which was conducted for 20 years, was fruitless!

Infectious concept of the origin of PU (H p) can not explain why the PU develops in only 12 — 15% of infected Hp people and ulcerative defect localized in the stomach or duodenum, spontaneously rumen without any treatment within 3 — 5 weeks, in spite of the continuing contamination of the stomach H p; why against Hp-infections occur regularly recurrent PU, alternating with remission; why at PU, as a rule, it formed a single ulcerative defect, rather than multiple erosive and ulcerative lesions?

Outstanding therapist EH. Tareev (1895 — 1986) with chagrin it stated: "Infectious concept is a permanent" magnet "that distracts doctors and scientists from major non-infectious agents". [14]

We believe idiopathic PU gastroenterology and not an infectious disease [25, 42].

No less complex and controversial issue of the relationship GC with Hp infection. According to P. With rrea (USA) — one of the most authority GOVERNMENTAL scholars of RJ — RJ Development — A multi-

stage and multi-factor process (a multistep and multifactorial process), races drawn in time [63]. The etiology of gastric cancer has not yet been established. In its development are involved: genetic factors (hereditarily burdened), physical (ionizing radiation) and multiple chemical carcinogens. International Agency for Research on Cancer — IARC, which is a WHO department, identifying Hp to class I carcinogen. However, evidence of a carcinogenic effect Hp is not enough.

1. In the presence of Hp-infection in the stomach gastric cancer by developing discharges a only 1% of cases, and among Indian and African populations, where infection of the population Hp reaches 90% or more, RJ diagnostic ruyut much less than that ofpeople in Europe and North America, whose colonization Hp gastric not exceed 35 — 50%.

2. It was found that, based on epidemiological evidence, while Nali Chii-Hp-infection in the stomach, develops only the distal (piloroantralny) AJ (the corner of the stomach), and proximal (cardia) gastric cancer is not associated with Hp-infection. Moreover, were represented Lena strong the proof of the fact that the presence of Hp in the gastric antrum, especially their of CagA-positive strains, somehow prevents the development of cardiac gastric cancer, gastroesophageal reflux disease (GERD), Barrett's esophagus (precancer) and adenocarcinoma of esophagus performing protective role [61, 67, 71].

3. Hp is not directly involved in the development of gastric cancer, do not produce mutagenic and carcinogenic substances [22] and should not be considered as a carcinogen. Perhaps, Hp is kokantserogenami are, but and it needs no proof.

4. The assertion that there is a sequence (phasing) in the development of gastric cancer: non-atrophic CG — Atrophic CG — Intestinal metaplasia — Epithelial dysplasia — RJ [63] has recently been refuted. So, on your conferring European SRI Study Group Hp-infection (EHSG), proho flown to the city of Ljubljana (2012), there were two reports represented Lena evidence that RJ can develop in the absence of atrophic process in the stomach: M. Varbanova e t al. It showed that RJ does not correlate with the severity of atrophy andintestinal metapla sion coolant for OLGA system, a M. Leia et al. We found that in most cases of gastric cancer occurs with normal levels of pepsinogen-1 and -2 in the blood that indicate the absence of the process and atrophic in GM [49].

Thus, atrophic hCG as RJ development step, apparently, is not mandatory.

5. In 10% of cases of gastric cancer develops without the participation of Hp: in patients with autoimmune atrophic fundic chronic hepatitis (type A), which in 40% of cases associated with megaloblastic anemia, as well as in cases of genetic instability and the presence of gene errors.

6. The role of Hp in the development of gastric cancer could only confirm the epidemiological studies, in connection with which F. Roccas (Th. Rokkas) believed that "the relationship between Hp infection and the subsequent development of gastric cancer remains unclear epidemiological paradox" [16].

By the way, Russia's leading surgeons, gastroenterologists (AF. Chernousov et al.) Do not recognize the value of Hp-infection in developing TII RJ [32].

7. Compilers "Maastricht Consensus" (MK -1 — 4, 1996 — 2010) at essentially monopolized the right to define indications for eradication of Hp and method of eradication treatment, transportation of the voice of the strategy on the total elimination of Hp (test and treat strategy: detect and destroy!) that scientifically unproven and practically feasible.

This strategy corresponds to another one postulate DY Graham, threatening on the sound and the erroneous on the content of "Good Hp — only dead H". The practical implementation of this strategy was the main cause of the rise from year to year Resistance Hp to eradication therapy, which has to Stigl critical level, and also the emergence of (selection) cytotoxic strains of Hp, which acquired the "island of pathogenicity» (pathogenecity-associated island — of PAI), located on site of the chromosomal DNA, which is believed appeared at later stages of their evolution in the result of the horizontal transfer of some other microbe [9, 21]. The "island of pathogenicity" were SET on the claimed existence of genes cytotoxicity: cagA, vacA, iceA, babA. Just as after the detection of an "island of pathogenicity" had been made numerous attempts to identify cytotoxic strains of Hp, responsible for the development of specific gastroduodenal disorders (PU, gastric cancer and others.), But all they were vanities governmental "ulcerogenic", "carcinogenic", and etc. strains Hp not su exists.

The authoritative American gastroenterologist MJ Blaser schi thaws that "between a man and Hp there is a kind of homeo- stasis: as long as they do not attempt to destroy, they do not harm your "owner" (man) [58]; "In Depending on the circumstances, Hp mo gut conduct yourself as a commensal or even both symbionts, as a component of the normal microflora of the stomach, but in certain circumstances can act and in a pathogen" [1, 55].

8. unreasonably, without sufficient scientific evidence were expanded indications for eradication of Hp for the expense of patients with GERD, UDF and NSAID-gastritis ("gastropathy") and even healthy bacteria carriers ("at the request of the patient"), laid dec ix issue of eradication of Hp on people not having medical education, that is unacceptable. Arbitrarily setting a low threshold effectiveness of eradication therapy (80%), allowing for "survival" to 20% Hp, the authors-compilers "MK" we have created the conditions for the races about lence of resistant strains of bacteria and selection of cytotoxic strains.

Only in the last review of "MK -4" (Florence, 2010) were the last, made the forced recognition:

1. "When SFD eradication of Hp is a complete and long-term elimination of symptoms from the 1st of 12 patients" (that is, 8.3% — I. D.). And in the "Rome Criteria-III" stated that the use of placebo when UDF effectively in the 20 — 40%!

2. "Hp does not influence on the severity and frequency of symptoms and the effectiveness ciency of treatment with GERD, and epidemiological studies de monstriruyut negative correlation between propagation tion Hp and the development of GERD and adenocarcinoma of the esophagus" ; that is, eradication of Hp drive IT to frequent GERD and adenocarcinoma pi schevoda !

3. « Hp-infection does not eliminate the risk of PU formation in gastroduodenal zone when receiving NSAIDs."

4. "So far, not enough evidence about the association of Hp with other (non-gastric) diseases, including heart and neurological."

After the eradication of Hp improve functional capabilities of the body of the stomach, but how much is due to regression of atrophy, it remains controversial. Convincing evidence that eradication of Hp leads to regression of intestinal metaplasia, is not obtained [28, 65].

While on the forced recognition in the "MK -4" it retained the recommendation to carry out eradication of Hp at SFD, GERD and NSAID-gastritis ("gastropathy") [65].

In one of the recent publications of MJ Blaser and S. Falkow deliv-Zali very reasonable idea: after eradication of Hp can not exclude the fact that the vacant "niche" (stomach) colonize other bacteria, which are due to the selection will be more virulent than Hp [59].

All often become published research in which proves that after eradication of Hp is marked acceleration of not only GERD, esophageal Barrett (precancer), and adenocarcinoma of the esophagus, but and bronchial asthma, obesity and skin allergic diseases, especially frequent in children [62, 71].

These studies began to base it for performances MJ Blaser on one of the last gastroenterological weeks in the US (2014) to report, eloquently titled " of Helicobacter pylori: friend or foe ?" And make the judgment that "there is a certain balance between the negative and positive action Hp on the foreheads of the century ". [56]

We believe the award of the Nobel Prize for Medicine "discoverers» of Helicobacter pylori the BJ a Marshall and JR Warren mistake of the Nobel Committee, which occurred and previously [25].

In its time, the Nobel committee has not said the Nobel Prize of outstanding scientists like Hans Selye, who discovered the existence of the general adaptation syndrome, explaining the essence of the biological concepts (phenomenon) of stress and distress ; AM Ugolev, proved the existence of the Priest n full-time (of the membrane, contact) digestion in the thin intestine and disclosed the its physiological mechanism, and also put forward the concept (theory) functional units ; Christian Bernard, the first suc but carried out the transplant patient donor of heart and...

On the background of these outstanding achievements, became a landmark in Isto Rhee medicine, the discovery in the stomach of Helicobacter pylori — a non-invasive low-virulent bacteria, vital activity of which is limited to the gastric compartment, it looks more than modest Soba Thieme [26].

II. The concept of the existence of functional gastrointestinal disorders (Functional Gastrointestinal Disorders) — syndrome of functional gastroduodenal dyspepsia (SFD) and the syndrome of irritable bowel (IBS), which occur without morphological Sanchez who substrate, was proposed in 1988 g. on World con gress gastroenterologists in grams. Rome (Italy). Then the same was created constantly operating committee (Working Team Committee) for their study under the chairmanship of DA Drossman (US).

In 1994 for the first time published recommendations of the Committee on the diagnosis, clinical manifestations and treatment of functional gastrointestinal disorders who received notoriety as the "Rome Criteria" (Kazakhstan). In 2016 g. It was presented already the 4th, a revised version of the "RC" [64].

From the very beginning of the emergence of the concept of leading Russian pathologists DS. Sarkisov (1924 — 2000) and VV. Serov made with her uncompromising criticism, defending the unity of the structure and function. DS. Sarkisov, in particular, wrote: "It is always possible to find a morph of the logic changes, the corresponding thin and Dynamic nym change functions" [18]. They had proved that with the help of electronic microscopy, histochemistry, molecular biology and genetics at all TN. functional diseases and syndromes can place the presence of changes in cell membranes, nuclear and cytoplasmic organelles, the receptor apparatus and etc.

Even long before the publication of "Kazakhstan -1" an outstanding clinician and scientist of our country in the.X. Vasilenko (1897 — 1987) wrote: "In order to understand the disease classification of diseases should be based not only on the identified morphological changes in the body, but and on the belief that violations of the structure are detected earlier than clinical and functional manifestations of the disease" [5].

Well known and its shining on the forms e and maintenance FSA-ism "function without structure is unthinkable, and the structure without function is meaningless". [19]

In different years, we in their publications advocated the position that when the functional disorders of the digestive tract has always can be found their equivalent [33, 34, 35, 36, 37].

In 1998 g. in the framework of the annual Russian Gastroenterological Week was held a "round table" dedicated to the SRK, a transcript of which in 1999 g. was published in the Journal of [8]. Concluding the debate, the Head (VT. Ivashkin) said literally the following: "DS. Sarkisov, a pathologist distinguishes philosophical approach, and its subtle notes retain the freshness of thought in for many years. I think that the morphological basis of the syndrome of irritable bowel early or on s the bottom of the pop up" [8]. This event happened in 2015. In the journal article, with the participation of Russian Gastroenterology VT. Ivashkina was Confirm Suppress: "In the present time, all the more attention is paid to the study of the NIJ inflammatory changes in the mucous intestine and at IBS, again, developing as a result of violations of the barrier function of the gut and cytokine imbalance... New data on the pathogenesis of IBS Zuko-uniformly lead to the inclusion in the scheme treatment of IBS drugs, an incense- inflammatory activity (glucocorticoid preparations of 5-ASA and others.)" [15].

It has been found that it is the inflammatory process in the intestine and immune dysregulation lie at the basis of not only postinfectious forms IBS, but and IBS without prior infection process. The presence of inflammatory percent ECCA in the gut with IBS is confirmed not only by morphological studies, but and increase the content of faecal calprotectin — calcium-binding protein, which indicates to the active of inflammation -inflammatory process in the gut [48, 69, 73].

Furthermore the first, in patients with IBS set elevated level of pro-inflammatory cytokines in blood (IL -1b, IL -6, IL -8 TNF) [48].

The "RC-I-III -compilers authors insisted on the need for a differential diagnosis between UDF, IBS and various organic diseases s stomach and intestines. Bed and « the PK-IV of» (2016) are the same (presumably to "save face") state, if the term "functional" does not mean the absence of organic (morphological) changes in the stomach and intestine (?).

When SFD is known, enumerable organic gastroduodenal disease excluding this diagnosis do not mention CG, arguing that hCG — it is a morphological diagnosis, with which always no clinical symptoms, and at the appearance of dyspeptic and painful syndromes believe diagnosed "CG a syndrome of functional dyspepsia ", uniting in odes Mr. diagnosis of an organic process in the stomach (CG) with functional syndrome (SFD) [64].

Gastroenterologists in for many years studying the problem of chronic hepatitis, are convinced that, in at least in 50% of cases of chronic hepatitis occurs with pain syndrome (gastritis dolorosa), arising in connection with hyperdyskinesis (antral non-atrophic CG), as well as a dyspeptic phenomena (atrophic fundic or total hCG) [24, 41, 46, 47].

In connection with the exposition, we believe that the concept of the possibility of the existence of functional gastrointestinal races disorders without structural equivalents, exhausted themselves and suffered a complete fiasco. Therefore, the publication of "Kazakhstan -IV" (2016) is excessive.

III. In the last 20 years there was a vicious practice: instead of nosological diagnosis began to be used (following for foreign gastroenterologists) syndromic diagnoses (SFD and IBS), to torye positioned as the final. With "nosological" syndromes agree, of course, impossible.

The outstanding Russian clinician MP. Konchalovsky (1875) wrote: "In the diagnosis we distinguish three stages: the first stage — the syndromic diagnosis, the second stage — pathogenetic diagnosis and, finally, the third stage — nosological (causative) dia prognosis" [11]. It is obvious that the syndromic diagnosis — it is only an intermediate stage on the way to the nosological diagnosis. V.H. Vasilenko was confident in the fact that "syndromic diagnoses gone us from the essence of the disease" [5]. Even more categorical was another famous scientist — IA. Kassirsky (1898 — 1971): "Some are trying to direct thief in their inability to understand in real nosology for syndromic diagnosis" [10]. And such a reputable pathologist as DS. Sarkisov believed that "syndromic diagnosis — is a diagnosis of ignorance" [17].

Failure to nosological principle in the diagnosis of somatic diseases leads to a decrease in the importance of on notions as the etiology, pathogenesis, typical clinical symptoms and outcomes of a particular disease, its repeatability (repeatability of the bridge), stereotype.

Renowned pathologist VV. Serov emphasized: "syndrome — the notion of pathogenetic and nosology — etiological. Syndromes of about 1500, and nosological units, the pathogenesis of which been ensured vides these syndromes, more than 20 thousand" [19].

Not less flawed attempts to replace the etiology of a specific disease, "factors of risk" (RF), or "predictors Bo 's disease" that contributes to the "blurring" the leading role of the etiology in the origin of disease, as a long list of risk factors is not can replace etiology s disease [12].

IV. We consider it unacceptable to use during clinical trials of new drugs means placebo («dummy") that became traditional for foreign researchers, since it violates ethical norms of medical activity and is inhumane: the patient receiving placebo, did not suspect that it is not treated, but only mimic treatment. Besides that, with the evidentiary point of view is much more convincing to establish that a new medical preparation governmental means rather similar, already established preparations and not "dummies".

Founder of Clinical Pharmacology in our country, a well-known clinician B.E. Votchal (1895 — 1971) believed that "when the allocation of the control group of patients receiving placebo ("dummy"), we are deceiving the patient, — do not treat it, just do kind of what we treat" [6].

It should be noted that the "Helsinki Declaration of the World Medical Association", which regulates the procedure of clinical trials of new medicinal products, requires compliance sleduyushih conditions:

1. The potential efficacy, risk and inconvenience (side effects and others. — Ya.Ts.) A new method of treatment compared with the benefits of the best of the existing therapeutic agents.

2. In all studies, with the participation of the people the patient has to be an appropriate way inform esc of view, methods of research, the possible risks and inconveniences. The doctor conducting the EC to follow, should receive from the party informed consent with these things, preferably in written form. [23]

Comments as they say, are unnecessary. But not less than indicated in the declaration requirements are constantly ignored abroad governmental researchers.

V. In the published journal articles, oral presentations (reports) often have to face with the sloppiness of style, inability to logically correct to build the phrase to find the point words to express their thoughts, with the unfortunate terminology (e.g., "dysfunctional disorders' biliary tract, — this is a tautology) [52].

V.H. Vasilenko was convinced that "the exact terminology (cleritas defitionis) characterizes the level of science and of course optionally go for mutual understanding" and "the lack of precise terminology ogy is unworthy of science" [5].

So, for example, we consider illiterate use of the term "ethiopathogenesis". This terminology "hybrid" (or "centaur") abused no roofing to practical doctors, but and scientists in their publications [40]. The term "etiology" as it is known, — this is the doctrine of the reason of illness, of its essence, and the term "pathogenesis" reflects the internal mechanism of the pathological process. In these terms different from Dr. Yerzhan, and combine them into one term can not. Academic "Encyclopedic Dictionary of Medical Terms" (under the editorship. BV. Petrovsky) certifies term etiopathogenesis as a "bankrupt term, the use of which contributes to the confusion of concepts prich and us, and the investigation into the pathology" [51].

Is unacceptable, and the term "-patiya" (enteropathy, colopathy, NSAID-gastropathy, and others.), Because it does not reflect the nature of the pathological process (inflammation, tumor and the like.). About the term "-patiya" clearly expressed the famous pas pathology VV. Serov: "The term "-patiya " — is a haven of ignorance and misunderstanding". [20]

Examples of the use of unsuccessful and erroneous medical cal terms the great multitude, but we restrict ourselves to only two given examples. Interested yuschihsya problems medical tion of terminology, we refer to our publications previous years [27, 29, 39, 40].

Concluding the article about the errors of modern gastroenterology, I want to refer to the words of wonderful and wise man, an outstanding clinician and is learned th V.H. Vasilenko, who had an encyclopedic knowledge and philosophical warehouse mind: "That, that is not subjected to questioning and criticism, early considered reliable and proven" [5].

References:

1. Авраменко А. А. Хеликобактериоз / А. А. Авраменко, А. И. Гоженко. — Николаев, 2007.
2. Аруин Л. И. Helicobacter pylori в этиологии и патогенезе язвенной болезни / Л. И. Аруин // Материлы 7й сессии Российской группы по изучению Helicobacter pylori. — Н. Новгород, 1998. — С. 6–9.
3. Баранская Е. К. Язвенная болезнь и инфекция Helicobacter pylori / Е. К. Баранская // Русск. мед. журн. — 2000. — № 1. — С. 8–14.
4. Важные практические результаты и современные тенденции в изучении заболеваний желудка и двенадцатиперстной кишки / К. В. Маев, А. А. Самсонов, Н. Г. Андреев, Д. Н. Андреев // Российск. журн. гастроэнтерол., гепатол. и колопроктол. — 2012. — № 4. — С. 17–27.
5. Василенко В. Х. Введение в клинику внутренних болезней / В. Х. Василенко. — М., 1985.
6. Вотчал Б. Е. Очерки клинической фармакологии / Б. Е. Вотчал. — М., 1965.
7. Давыдовский И. В. Проблема причинности в медицине. Этиология / И. В. Давыдовский. — М., 1962.
8. Диагностика и лечение синдрома раздраженной кишки. Материалы «круглого стола» // Российск. журн. гастроэнтерол., гепа¬тол. и колопроктол. — 1999. — № 2. — С. 61–71.
9. Исаков В. А. Хеликобактериоз / В. А. Исаков, И. В. Домарадский. — М., 2003.
10. Кассирский И. А. О врачевании (проблемы и раздумья) / И. А. Кассирский. — М., 1970.

11. Кончаловский М. П. Избранные труды: «О клинических синдромах в научно-исследовательской работе в клиниках и больницах» / М. П. Кончаловский. — М., 1961. — С. 52–61.
12. Лещинский Л. А. «Факторы риска» (критика понятия и предложения к классификации) / Л. А. Лещинский, А. С. Димов // Клин. мед. — 1982. — № 8.
13. Минушкин О. Н. Современный взгляд на эрадикацию Helicobacter pylori / О. Н. Минушкин, О. В. Аронова // Практикующий врач. — 2002. — № 1. — С. 52–54.
14. Мухин Н. А. Некоторые клинические аспекты проблемы этиологии внутренних болезней / Н. А. Мухин // Клин. мед. — 2000. — № 8. — С. 7–11.
15. Полуэктова Е. А. Применение комбинированного препарата альверина цитрата и симетикона в лечении больных с синдромом раздраженного кишечника / Е. А. Полуэктова, С. Ю. Кучумова, В. Т. Ивашкин // Российск. журн. гастроэнтерол., гепатол. и колопроктол. — 2012. — № 4. — С. 38–47.
16. Роккас Ф. Инфекция Helicobacter pylori как фак¬тор риска рака желудка: современные доказательства / Ф. Роккас // Российск. журн. гастроэнтерол., гепатол. и колопроктол. — 2012. — № 3. — С. 66–70.
17. Саркисов Д. С. Некоторые особенности развития медико- биологических наук в последние столетия / Д. С. Саркисов // Клин. мед. — 2002. — № 7. — С. 4–8.
18. Саркисов Д. С. Следует, наконец, отказаться от понятия «функциональная болезнь», «функциональная патология» / Д. С. Саркисов // Клин. мед. — 1998. — № 3. — С. 64–65.
19. Серов В. В. Общая патология — теория клинической медицины / В. В. Серов // Клин. мед. — 1998. — № 10. — С. 4–6.
20. Серов В. В. Общепатологические подходы познания болезни / В. В. Серов. — Саратов, 1992.
21. Сидоренко С. В. Диагностика и лечение инфекций, вызываемых Helicobacter pylori / С. В. Сидоренко // Инфекции в амбулаторной практике. — М., 2002. — С. 125–140.
22. Сиппонен П. Гастрит — атрофический гастрит — кишечная метаплазия — рак желудка: обратима ли эта последовательность? / П. Сиппонен, К. Сеппола // Российск. журн. гастроэнтерол., гепатол. и колопроктол. — 1999. — № 2. — С. 30–35.
23. Хельсинкская декларация Всемирной медицинской ассоциации // Клин. фармакол. и тер. — 1997. — № 6 (3). — С. 63–64.
24. Хронический гастрит / Л. И. Аруин, П. Я. Григорьев, В. А. Исаков, Э. П. Яковенко. — Амстердам, 1993.
25. Циммерман Я. С. Антибактериальная терапия и ее влияние на эндоэкологическую систему «макроорганизм — эндосимбионтные бактерии» (на примере Helicobacter pylori-ассоциированных заболеваний) / Я. С. Циммерман // Клин. фармакол. и тер. — 2015. — № 24 (2). — С. 5–12.
26. Циммерман Я. С. Гастродуоденальные заболевания и Helicobacter pylori-инфекция: общее обозрение проблемы / Я. С. Циммерман // Клин. мед. — 2009. — № 5. — С. 9–15.
27. Циммерман Я. С. «Западноевропеизмы» и их место в современной русской медицинской терминологии, другие спорные терминологические проблемы / Я. С. Циммерман // Клин. мед. — 2000. — № 1. — С. 59–63.
28. Циммерман Я. С. «Маастрихтский консенсус-4»: основные положения и комментарии к ним / Я. С. Циммерман // Клин. мед. — 2012. — № 9. — С. 28–34.

29. Циммерман Я. С. Медицинские термины и их связь с искусством и литературой / Я. С. Циммерман // Клин. мед. — 1998. — № 1. — С. 67–70.
30. Циммерман Я. С. Микрофлора слизистой оболочки желудка, ее свойства и роль в развитии острого и хронического гастрита / Я. С. Циммерман, Ю. А. Захарова, В. Е. Ведерников // Клин. мед. — 2012. — № 11. — С. 41–46.
31. Циммерман Я. С. Проблема хронического гастрита / Я. С. Циммерман // Клин. мед. — 2008. — № 5. — С. 13–21.
32. Циммерман Я. С. Рак желудка: современный взгляд на проблему / Я. С. Циммерман // Вестн. хирургич. гастроэнтерол. — 2011. — № 2. — С. 77–88.
33. Циммерман Я. С. «Римские критерии-III» и синдром функциональной (гастродуоденальной) диспепсии / Я. С. Циммерман // Клин. мед. — 2008. — № 4. — С. 59–66.
34. Циммерман Я. С. Синдром раздраженной кишки: какова его истинная сущность? / Я. С. Циммерман // Клин. мед. — 2014. — № 7. — С. 19–29.
35. Циммерман Я. С. Синдром раздраженной кишки: современные представления, спорные и нерешенные вопросы / Экспер. и клин. гастроэнтерол. — 2003. — № 6. — С. 124–132.
36. Циммерман Я. С. Синдром функциональной (неязвенной) диспепсии: современные представления, спорные и нерешенные вопросы / Я. С. Циммерман // Клин. мед. — 2004. — № 5. — С. 16–22.
37. Циммерман Я. С. Синдром функциональной диспепсии и/или хронический гастрит? / Я. С. Циммерман // Клин. мед. — 2013. — № 3. — С. 8–15.
38. Циммерман Я. С. Сравнительная оценка диагностических тестов определения Helicobacter pylori и спектр мукозной микрофлоры желудка при гастрите и язвенной болезни / Я. С. Циммерман, Ю. А. Захарова, В. Е. Ведерников // Клин. мед. — 2013. — № 4. — С. 42–48.
39. Циммерман Я. С. Терминологические проблемы в кардиологии и других разделах медицины / Я. С. Циммерман // Клин. мед. — 1998. — № 3. — С. 58–62.
40. Циммерман Я. С. Терминологические проблемы гастроэнтерологии / Я. С. Циммерман // Российск. журн. гастроэнтерол., гепатол и колопроктол. — 1996. — № 4. — С. 6–10.
41. Циммерман Я. С. Хронический гастрит и язвенная болезнь / Я. С. Циммерман. — Пермь, 2000.
42. Циммерман Я. С. Этиология, патогенез и лечение язвенной болезни, ассоциированной с Helicobacter pylori-инфекцией: состояние проблемы и перспективы / Я. С. Циммерман // Клин. мед. — 2006. — № 3. — С. 9–19.
43. Циммерман Я. С. Язвенная болезнь: актуальные проблемы этиологии, патогенеза, дифференцированного лечения / Я. С. Циммерман // Клин. мед. — 2012; 8: 11-18.
44. Чазов Е. И. Прекрасная цель / Е. И. Чазов // Журн. Знамя. — 1984. — № 3. — С. 175.
45. Чернин В. В. Место Helicobacter pylori в нормомикробиозе и дисбактериозе мукозной микрофлоры эзофагогастродуоденальной зоны, развитии язвенной болезни, хронического гастрита и эзофагита / В. В. Чернин, В. М. Червинец, С. Н. Базлов // Экспер. и клин. гастроэнтерол. — 2016. — № 2. — С. 79–86.
46. Чернин В. В. Хронический гастрит / В. В. Чернин. — Тверь, 2006.

47. Шептулин А. А. Механизмы возникновения и современные принципы лечения диспепсических расстройств у больных хроническим гастритом / А. А. Шептулин // Российск. журн. гастроэнтерол., гепатол. и колопроктол. — 1999. — № 4. — С. 30–34.
48. Шептулин А. А. Новое в этиологии и патогенезе синдрома раздраженного кишечника / А. А. Шептулин, М. Л. Визе-Хрипунова // Клин. мед. — 2016. — № 2. — С. 92–96.
49. Шептулин А. А. Обсуждение докладов рабочего совещания Европейской группы по изучению инфекции Helicobacter pylori (Любляна, 2012) / А. А. Шептулин // Российск. журн. гастроэнтерол., гепатол. и колопр¬октол. — 2013. — № 3. — С. 85–88.
50. Щербаков П. Л. Эпидемиология инфекции Helicobacter pylori / П. Л. Щербаков // Российск. журн. гастроэнтерол., гепатол. и колопроктол. — 1999. — № 2. — С. 8–11.
51. Энциклопедический словарь медицинских терминов в 3х т. / Под ред. Б. В. Петровского. — М., 1984. — № 3. — С. 337.
52. Этиопатогенез повреждения слизистой оболочки двенадцатиперстной кишки при хроническом панкреатите / Л. В. Винокурова, В. К. Дроздов, Е. В. Ткаченко [и др.] // Вестн. клуба панкреатол. — 2010. — № 1. — С. 44–46.
53. Blaser M. J. Cost of commensalism (state of the art lecture) / M. J. Blaser // 6th United European Gastroenterology Week. Abstract on disk. — 1997.
54. Blaser M. J. Ecology of Helicobacter pylori in the human stomach / M. J. Blaser // J. Clin. Invest. — 1997. — Vol. 100, No 4. — P. 759–762.
55. Blaser M. J. Helicobacter pylori are indigenous to the human stomach: duodenal ulceration is due to changes in gastric microecology in the modern era / M. J. Blaser // Gut. — 1998. — Vol. 43. — P. 721–727.
56. Blaser M. J. Helicobacter pylori: balance and inbalance / M. J. Blaser // Eur. J. Gastroenterol., hepatol. — 1998. — Vol. 10. — P. 15–18.
57. Blaser M. J. Helicobacter pylori and gastric disease / M. J. Blaser // Brit. Med. J. — 1998. — Vol. 316. — P. 1507–1510.
58. Blaser M. J. Hypothesis: the changing relationships of Helicobacter pylori and humans: implications for health and disease / M. J. Blaser // J. Infect. Dis. — 1999. — Vol. 179, No 6. — P. 1523–1530.
59. Blaser M. J. Исчезающая микробиота / Пер. с англ. / M. J. Blaser, S. Falkow // Клинич. фармакол. и тер. — 2014. — Vol. 4. — P. 7-16.
60. Bytzer R. Helicobacter pylori-negative duodenal ulcers: prevalence, clinical characteristics and prognosis: results from a randomized trial with 2-year fallow-up / R. Bytzer, P. S. Taglbjaerd // Am. J. Gastroenterol. — 2001. —Vol. 96. — P. 1409–1415.
61. CagA-positive strains of Helicobacter pylori may protect against Barrett's esophagus / M. F. Vaezi, G. W. Falk, R. M. Peek [et al.] // Am. J. Gastroenterol. — 2000. — Vol. 95. — P. 2206–2211.
62. Chen Y. Inverse associated of Helicobacter pylori with asthma and allergies / Y. Chen, M. J. Blaser // Arch. Intern. Med. — 2007. — Vol. 167. — P. 821–827.
63. Correa P. Human gastric cancerogenesis: a multistep and multifactorial process. First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention / P. Correa // Cancer Res. — 1992. — Vol. 52. — P. 6735–6740.

64. Drossman D. A. The functional gastrointestinal disorders and the Rome-III process / D. A. Drossman // Gastroenterology. — 2006. — Vol. 130, No 5. — P. 1377–1390.
65. European Study group Management of Helicobacter pylori infection — the Maastricht-4 Florence Consensus Report / P. Malfentheiner, F. Megraud, C. O'Morain [et al.] // Gut. — 2010. — Vol. 61, No 5. — P. 646–664.
66. Graham D. Y. Campylobacter pylori and peptic PU disease / Gastroenterology. — 1989. — Vol. 96. — P. 615–625.
67. Helicobacter pylori infection and risk of cardia cancer and non-cardia gastric cancer. A nested case-control study / S. Hansen, K. K. Melby, S. Aase [et al.] // Scand. J. Gasrtoenterol. — 1999. — Vol. 34. — P. 353–360.
68. Laine L. Has the impact of Helicobacter Pylori therapy on PU recurrence in the United State been overstated? A meta-analysis of rigorously disignet trials / L. Laine, R. Hopkins, L. Gerardi // Am. J. Gastroenterol. — 1998. — Vol. 93, No 9. — P. 1409–1415.
69. Lashner B. A. Clinical research advances in ulcerative colitis / B. A. Lashner // Medscape Conference Coverage based on selected sessions at the 66th Annual Scientific Meeting. — Las Vegas, 2001.
70. Morris A. Ingestion of Campylobacter pyloridis cause gastritis and raised fasting gastric pH / A. Morris, J. Nickolson // Am. J. Gastroenterol. — 1987. — Vol. 82, No 3. — P. 192–199.
71. Peel A. M. Helicobacter pylori and gastrointestinal tract adenocarcinomas / A. M. Peel, M. Y. Blaser // Nature Rev. Cancer. — 2002. — Vol. 2. — P. 28–37.
72. Prevalence and risk factors of Helicobacter pylori — negative peptic PU: a multicenter study / G. Moucci, R. di Battisa, C. Abbati [et al.] // Gastroenterol. — 2000. — Vol. 31. — P. 42–47.
73. Tibbie J. Faecal calprotectin and faecal mysterious blood is tested in diagnosis colorectal carcinoma and adenoma / J. Tibbie // Gut. — 2001. — Vol. 49. — P. 402–408.
74. Tytgat G. N. J. No Helicobacter pylori, no Helicobacter pylori-associated peptic PU disease / G. N. J. Tytgat // Aliment. Pharmacol. Ther. — 1995. — Vol. 9, Suppl. 1. — P. 39–42.

Persistent delusions of modern gastroenterology

Y. S. Tsimmerman

Perm State Medical University n. a. E. A. Vagner, Perm, Russia

Key words: gastroenterology, delusions, Helicobacter pylori, functional gastrointestinal disorders, syndromic diagnosis, placebo, terminology

The author shares his thoughts about exaggerating the role of infection with Helicobacter pylori in etiology and pathogenesis of chronic gastritis, peptic PU disease, gastric cancer. Arguments against the common concept of functional gastrointestinal disorders, formulation of syndromic rather than nosological diagnosis are represented. Particular attention is paid to the moral and ethical aspects of the use of placebo in clinical trials. The shortcomings and errors of the terms that are used in modern gastroenterology are analyzed in detail.