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CHAPTER 1

THE ETIOLOGY AND PATHOGENESIS OF PEPTIC ULCER

(P.106-149).

1.4. “Causticus alkalotic exspuition” theory – a fresh approach to the role of Helicobacter pylori in the etiology and pathogenesis of peptic ulcer.

1.4.1 Basic contradictions of the traditional views on the etiology and pathogenesis of peptic ulcer.

However, not all authors acknowledge the role of HP in peptic ulcer etiology, their impugments come down to the following points: 1) peptic ulcer develops only in 1 from 8 contaminated; 2) organ – preserving operations in the better part of monitoring lead to ulcer elimination, not having a significant impact on gastric mucosa and duodenum; 3) administration of modern high powered antisecretory drugs (H_2 – histamine antagonists and other) produces a significant ulcer-healing outcome, despite the maintenance of HP in gastric mucosa and duodenum.; 4) men predominantly suffer from peptic ulcer, while the density of gastric mucosa and duodenum colonization is not connected with the gender; 5) HP is typically disclosed in older persons, while the peptic ulcer is a prerogative of young people; 6) PU has a polycyclic progress of a disease, so that ulcerations are healing because of treatment, without treatment and even contrary to treatment activity, despite the persistence of HP in gastric mucosa and duodenum; 7) hyper secretion of hydrochloric acid and pepsin can cause the ulceration in an absence of HP.

To this we can add, that the contagious conception of peptic ulcer etiology can't explain the solitariness of ulcerous defect, alternation of relapses and remissions, seasonality of recrudescence and other processes. Some researchers even deny the connection of HP with abnormal changes of gastric mucosa, assigning HP a part to saprophytes or consecutive infection.

However, the following facts acknowledge the role of HP – infection in ulcer development: 1) frequent HP contamination of ulcerous persons; 2) While long-term follow-up for HP-contaminated patients ulcer develops on a more frequent basis in a group of HP-contaminated, then in a group of non-contaminated; 3) while the eradication of HP is observed a significant frequency reduction of ulcer relapses – from 60-100 to less than 15%.

Beyond that, monitoring shows, that even patients with duodenal ulcer have different measures of acid secretions, staying in a great percent of the time within a standard values (Grossman M.I., 1980). Most of the apparently healthy people have the same high level of acid secretion, as those who suffer from the peptic ulcer. (Guerre J. et. al., 1981). Also, it was found, that apparently healthy people have a continuous acid secretion and high level of hyper secretion for 61,5% of the time of day, and a considerable part of patients during the night have a level, close to neutral one (Leia U.I., 1987). So that, the facts of continuous acid secretion and night hyper secretion should be considered in peptic ulcer pathogenesis in an ambivalent manner.

Peptic ulcer can develop upon any type and degree of acid forming functions, as well as upon hyper anacidity(Baron J. H., 1982). Terms of ulcer cicatrisation don't depend on the degree of hyper secretion, it doesn't affect on recrudescence acuity, and in a remission period the level of secretion doesn't change and stays permanent, the same as at the moment of recrudescence [34, 121].

Hyper secretion level of hydrochloric acid and ulcer localization are not interlinking. For example, high secretion is considered to be an indicant, peculiar to duodenal ulcer, but it is noticed while gastric ulcer (30-40% of patients), in the same breath, multiple ulcers can be present in case of normal function of gastric secretion [34, 84, 121]. Therefore, taking into account quoted hereinabove contradictions, it can be confirmed, that hydrochloric acid hyper secretion doesn't constitute the

sufficient ground for peptic ulcer contraction [34, 121]. It is also affirmed by our researches of acidity level of peptic ulcer patients with a various ulcerous defect allocation (table 17) [110].

Table 17. Peptic ulcer patients degree of incidence frequency with various ulcerous defects allocation depending on acidity level (n=323).

Ulcerous defect allocation	Hyperacidity high-grade		Hyperacidity moderate		Normal acidity		Hypoacidity moderate		Hypoacidity high-grade	
	figure	%	figure	%	figure	%	figure	%	figure	%
1. Duodenum	55	19,3	103	36,2	61	21,4	42	14,7	24	8,4
2. Gaster	3	12,5	3	12,5	7	29,2	6	25	5	20,8
3. Anastomosis	-	-	1	7,2	3	21,4	3	21,4	7	50

Beyond that, this contradictions affects on peptic ulcer clinical implications understanding. For example, such a typical symptom, like pyrosis, is interpreted, as an evidence of hyperacidity, that gives a doctor a rise to prescribe preparations of antisecretory group while the detection during the endoscopy of ulcerous defect, even not detected instrumentally the level of patient's gastric secretion. Our researches don't confirm this opinion Table 18) [34, 42].

Table 18. Quotient of contamination degree with *Helicobacter pylori* of gastric mucosa and the gastric fluid acidity level before and after therapy of chronic gastritis type B patients with the symptom "pyrosis" (n=62).

Phase of pathological process	Acidity level of the gastric fluid (UE) M ± M	The gastric mucosa seeding level / (+) M ± M	
		Antrum	Gastric body
Active (before treatment phase)	11,3 ± 0,14	2,8 ± 0,13	2,4 ± 0,13
Remission	19,4 ± 0,13	1,4 ± 0,13	1,2 ± 0,13

How it is seen from this spreadsheet, after the therapy, when the level of gastric mucosa contamination with HP infection decreases, the level of acidity increases. If before the therapy the level of gastric fluid acidity on conversion to CU on an average correlated to selective hyperacidity, then after course of treatment it is correlated to moderate near-total hyperacidity, wherein the pyrosis subsided in the proximity of 58 patients (93,5%), whose level of contamination with HP – infection authentically decreased. 4 patients (6,5%), whose pyrosis didn't disappear after the treatment, the degree of HP – infection contamination stayed the same; this patients had a level of gastric fluid acidity, which correlated to normal acidity and moderate hypoacidity. It has given the occasion to draw a conclusion,

that such symptom as pyrosis is one manifestation of sufficient mass of active form of HP – infection vital activity and demands a deeper insight.

It is considered, that HP destroys the gastric mucosa blanket, creating a “leaking roof” effect, forming favorable conditions for hydrogen ions back diffusion, a well-known factor of ulceration, and facilitating the access for hydrochloric acid and pepsin to epithelial cells [34, 449]. More comprehensively, the staging of changing initiation induced by HP can be represented in the following way.

1. HP appears in gaster, penetrates under the layer of mucus into the depths of fosses in the antral pyloric zone, where the amount of pH is close to neutral, cause the adhesion on cells, and shedding toxins and NH_4^+ of bacteria destroy them. Then, HP can penetrate to intercellular space and even inside parietal cells, interrupting their vital activity.

2. To contamination in an intact gaster undergo predominantly zones with a high leaching function, i.e. antral pyloric, pyloric epithelium, heterotoped in duodenum, i.e. zones, where in the production of bicarbonates the leading role belongs to the vascular link.

3. Immunogenesis in the mucous coat of the stomach in reply to penetrated HP is confined in appearance of plasma cells, that synthesize immunoglobulin of various classes, impacting on epithelium structure, and also in appearance infiltration by the polymorphonuclear neutrophils its own plate of mucous tunic (i.e. forming the form pleine of type B chronic gastritis).

4. Neutrophils release a great amount of leukotriene C4 (LTC_4), which considered to be a part of so called slow-reacting substance of anaphylaxis and have a number of important properties, resembling the histamine's one, including apparent influence on vascular tone, that causes a peracute angiospasm, the result of which is trophism violation: decrease of incoming O_2 to the mucous membrane, nutrients and bicarbonates. Under the influence of gamma-glutamyl transpeptidase, which in a great amount evolves HP, LTC_4 changes into leukotriene D4, dominating on the activity its precursor in decades and hundred times.

5. The trophism violation leads to inefficiency of “mucous – bicarbonate barrier, which even in conditions of normal secretion of HCl creates the best conditions for the mucous tunic attack with the aggressive contents and retrodiffusion H^+ , as well as for the deterioration of physiological reparative regeneration, and this leads to the formation of erosions and ulcers.

6. Long-term persistence of HP in the mucous tunic leads to a permanent activation of Immunogenesis in it, leading to prolonged trophic violation and permanent damage of epithelium, the consequences of what is an activation of proliferation processes and their gradual predominance on the processes of epithelium cells differentiation. This means the progression of type B chronic gastritis and leads to a slow, deficient ulcer cicatrisation, i.e. to the chronicity of the process [34, 121].

A great importance of peptic ulcer development is attached not only to the interrelations of the factors of aggression and gastroduodenal zone protection, but also the stress. However, the peptic ulcer develops not in all people with increased acidity and all affected by stress. Apparently, individual characteristic of the organism imports, depending on which, the impact of negative environmental factor is realized or not in an ulcer process. The number of authors consider the peptic ulcer, as the disease of adaptation, and the process and the level of human adaptation can be stipulated due to psycho-functional peculiarities, and a condition of energy metabolism. F.Z. Meyerson (1986) as a fundamental link of adaptation to the environment considers the activation of mitochondrion and the increase of capacity of oxidative ATP resynthesis. The impacts of environmental factors (especially unfavorable and intense) lead to ATP deficiency and glycolysis activation [34, 375].

Apart from the entire peptic ulcer is gastric ulcer (GU). Some researchers believe that the GU is an independent disease with a typical clinical course, special etiological factors and pathogenetic mechanisms, which significantly differ from those of duodenal ulcer and peptic ulcer

gastroenteroanastomosis. In numerous studies of gastroenterologists, psychologists and surgeons were held studies to find out the cause of ulceration, created experimental gastric ulcer models. Nevertheless, many questions of etiology and pathogenesis is not resolved, there is no generally accepted concept of the formation of gastric ulcers [84, 438, 571].

In gastric ulcer, gastritis is defined in the antrum and in the body of stomach. In the end, appears the atrophy and metaplasia with prevalence of intestinal cells - intestinal metaplasia. The atrophy begins in the antrum, and then its foci are found on the anterior and posterior wall of the gastric body. Gradually they are increased in size, they merge and that leads to progressive zone reduction, which secretes the acid,, and the boundary between the pyloric glands removes in the proximal direction [34, 277].

At the same time, HP infection is detected in 70 - 100% of gastric ulcer patients [267, 389, 537]. The frequency of HP infection was significantly higher in gastric I-type (58.3%) (ulcers, localized more proximal (above) to the angle of the stomach, without clearly detectable pathology of the duodenum, pyloric and prepiloric zone) and type III (62, 5%) (prepiloric ulcers, including in combination with pathology of duodenum or ulcer of more proximal location), than for gastric ulcer of type II (26.7%) (ulcers, that are more proximal to the angle of the stomach in combination of ulcer or cicatricial ulcerous deformation of the duodenum or the pylorus), and while the second type of ulcer was found a significantly more manifested duodenogastric reflux [332, 389]. There is an inverse relationship between intensity duodenogastric reflux and frequency and intensity of HP contamination, depending on the degree of acid inhibition [389, 613, 627]. According to other facts, with duodenogastric reflux HP was detected in 91% of patients, in the absence of duodenogastric reflux - in 41% of patients, after the 6 months eradication and monitoring was found a significant reduction in the severity of duodenogastric reflux [34, 389, 550].

If the use of pharmaceuticals helped to improve the healing of duodenal ulcers and increase the frequency of persistent remission, the conservative treatment of gastric ulcer is usually insufficiently effective. Conservative methods of treatment of such patients facilitated an increase of frequency occurrence of giant and **carcinomatous degenerated** ulcers, often in advanced stages, which became a prompt to consider the gastric ulcer a disease, which demands an operative therapy [34, 84, 130].

Thus, we can say with full confidence, that today there is no any theory that could fully explain all the details and explain the contradictions that baffle more than one generation of gastroenterologists. The importance of this problem has given us the right to put forward a new theory of the ulceration mechanism – the theory of "caustic alkali expuition", which is an offspring of 15-year study of the problem, and is based on data, obtained from the 4009 comprehensive examination of patients with chronic *Helicobacter pylori* infection, of which more than 1/3 had erosive and ulcerous lesions of gastro-duodenal zone of varied localization and were in different stages of the pathological process development.

1.4.2 "Alpha" and "Omega" interaction of macro organism - the human body - and microorganism - *Helicobacter pylori*

Before proceeding to statement of the new theory ideas and erosive lesions and ulceration mechanisms, it is necessary to highlight what is the basis - the "alpha" and "omega" - the interaction of macro organism (the human body), and the microorganism (*Helicobacter pylori*).

1. "ALPHA"

The acid-peptic factor ↔ *Helicobacter pylori* ↔ The immune system.

(Avramenko – Gozhenko “balance”, 2007)

In this "balance" the permanent role is reserved for the distribution of power inputs of HP infection on 3 directions:

1st direction -inputs on HP infection vital activity and,primarily on mitosis [20, 34];

2nd direction - the inputs on fight with the human body's immune system. As is well-known, for combating the HP, neutrophils secrete a large amounts of free radicals, aimed to destroy HP infection, in retaliation, bacterium forms its anti-oxidant system, allocating a large amount of scavenger enzyme and superoxide anion scavenger [20, 34, 115, 336];

3rd direction – the inputs on a change of pH environment around themselves in order to: a) create the optimum pH differences - 1.4 - between the internal pH environment of bacteria and pH of gastric juice, which creates the conditions for the electrochemical reactions to replenish HP's energy reserves [34, 274]; b) neutralization of the negative effect of pepsin on HP-infection (at pH environment above 4, type I pepsin (optimum action 1.8 - 2), type II pepsin (optimum action 3.2 - 3.6) is not activated). HP allocates urease enzyme for this, which decompose urea into ammonia and water. Ammonia water, which is formed in this case, is a caustic (ammonia spirit), neutralize hydrochloric acid of the gastric juice (is produced a neutral ammonium chloride and water), leading to increase of pH environment [20, 34, 207, 451].

In this situation, the 1st and 3rd (a) direction are essential for replication of bacterial species, at

the same time the inputs of the 2nd and 3rd (b) directions are required for survival in hostile environment of the stomach, which until 1983 year was considered sterile [20, 34, 303].

As a result of this interaction between macro- and microorganism an uneasy equilibrium is formed(pathological homeostasis),and when it is violated, the development of a pathological process in the gastrointestinal tract takes place. Let's consider the options of this disorder:

1. **The acid-peptic factor ↔ Helicobacter pylori ↔ The immune system.**

In case of a saved pathological homeostasis HP spends maximum of energy on a constant struggle with acid-peptic factor and immunity, the rest energy - on the minimum level of mitosis, necessary to the survival of HP infection in the stomach. This situation is peculiar to the latent forms of chronic gastritis type B.

2. **Helicobacter pylori ↔ The immune system.** **The acid-peptic factor** ↗

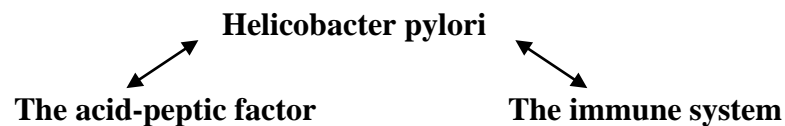
In case of acidity reduction, caused by both physiological peculiarities of the gastrointestinal tract (to 7-12 years, children's acidity is lower than the adults one) [20, 34, 167, 207], as well as the influence from the outside: the use of inhibitors of gastric secretion [34, 275, 403, 416], stress, smoking [4, 20], ingestion of bronchial mucus, in case of bronchopulmonary system and nasopharynx pathology [20, 251],duodenogastral reflux [48,] the level of energy demands shifts to struggle the immune system and the reproduction consumptions.

3. **The acid-peptic factor ↔ Helicobacter pylori** ↘ **The immune system**

In case of immune defense level reduction, caused both by a physiological peculiarities of immune development (up to 7 years, children's level of immune protection is lower than the adults one [34, 167]; in the puberty period (adolescence), in the involution period of sexual function, during women's pregnancy and lactogenesis - immunosuppression due to hormonal changes) [13, 30, 40], if

there are chronic pathologies of various organs and systems, predominantly, pathologies of the endocrine system and organs, affiliated with the system of immune defense (chronic disease of tonsils, inflammation of appendicitis crest) (the theory of a single immune and endocrine system) [37, 105] as well as due to external influences: stress (immunosuppression) [34, 105, 242, 265, 360], adverse environmental impacts (water, food, air, radiation background) [6, 10, 92, 164, 175, 176, 189, 325, 355, 385], deconditioning syndrome [11, 91, 361, 372], operational intervention (in the first place - on the organs of endocrine and immune systems) [37] the level of energy demands shifts towards the struggle with the acid-peptic factor and the reproduction consumptions.

4 .



By reducing the level of immune protection and the level of acidity of gastric juice, which causes were recited in the 2nd and 3rd paragraphs, there is a minimum of energy demands to fight the immunity and the acid-peptic factor, and the maximum - for reproduction of HP infection, which causes the most favorable conditions for the rapid growth of the bacterial mass, and under certain conditions, a transformation of quantity into a new qualitative level of pathology - the transformation of chronic gastritis type B in erosive and ulcerative process in the gastro-duodenal zone [20, 34, 108]. It is critical to underscore that stress is a universal background, that creates the best conditions for the vital activity of HP infection, as well as the factor, responsible for the mechanism formation of erosive and ulcerative lesions pyloric bulbar zone, that will be substantiated while outlining the new theory of the formation mechanism of erosive and ulcerative lesions of gastro-duodenal zone - the theory of "caustic alkali exspuition."

2. "OMEGA"

A second key factor in understanding the relationship of the human body and HP infection is the understanding of chronic gastritis type B stages and the effect of this the staging on: 1) the topography of the dispersal of HP infection; 2) concentration of HP infection; 3) the level of acidity of gastric juice, taking into account the fact that HP infection refers to the etiological factor of the second type (clinical manifestations develop upon the availability of a certain level of bacterial mass; decrease of HP-infection bacterization level by the gastric mucosa, even without complete removal of the agent, leads to clinical recovery) [18, 20, 21, 34].

1st stage – the stage of "primary installation"

This stage is peculiar to children under 7 - 10 years of age, when the immune system is not yet fully formed, and the acidity level is lower than in the adult organism. In this situation, the most "comfortable" zone for the primary insertion an antrum, where the pyloric glands, secreting bicarbonates, create an alkaline environment, convenient enough to form the primary site of HP infection [34, 167, 303]. The initial insertion of HP infection is often asymptomatic, that forms a latent form of chronic gastritis type B [34, 109, 167]. However, in a situation, when there is an active reproduction of HP infection in the antrum, its influence on the level of hydrochloric acid secretion, and, hence, on the whole acid-peptic factor, increases dramatically. Irritating G-cells, HP increases the level of the gastrin-releasing factor, and, hence, the level of gastric acidity (hyperacidity). In this situation, the

extension of HP infection in the body of the stomach is severely limited due to the fact, that HP itself creates "acid barrier" [34]. Schematically, this stage can be represented as follows (Figure 26).

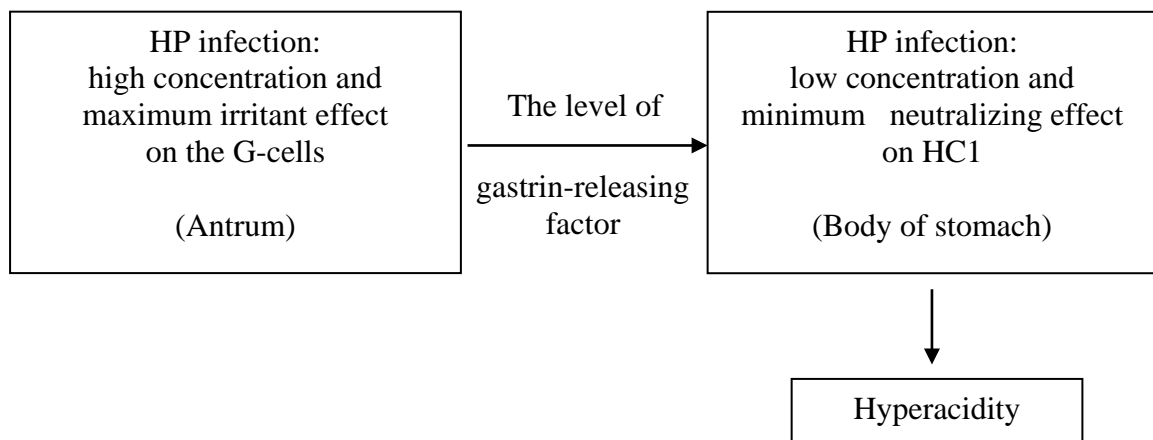


Figure 26. Scheme of the stage of "primary installation"

This stage is the most favorable for the eradication: HP-infection "sandwiched" in a small space in a tight place - in the mucosa antrum. HP deleting leads to vanishing of causes, that irritated G-cells, leading to reduction of gastrin-releasing factor level and stabilization of acidity on the level, typical for normal acidity, what is confirmed by the results of our studies.

2nd stage - the stage of "pangastritis" formation

This stage, according to our research, begins from the adolescence (11- 14 years) and lasts till 40-45 years. Formation of pangastritis is conditional upon the sharp destabilization of the hormonal and immune system at puberty, as well as a change in the social status of individuals, namely: an access to an independent life and participation in various social groupments: study in organized groups (specialized secondary and higher educational institutions, and so on) [17, 34]; for men – defense work, often not in places of primary residence, that significantly increases the psychological stress and generates deconditioning syndrome [34, 35, 361]. The women - hormonal changes, coming from pregnancy and lactation, as well as the stresses connected to child care, which also leads to immunosuppression and gastritis type B activation [14]. Also, the process of marriage, which requires a deep psychoemotional alteration, related to the change of life, and with the formation of a sense of responsibility to the family, as well as relationships with family members; divorce [34, 185, 607]. Also an important role in the formation of pangastritis plays the social activity of individuals (specificity of work activities), social life and political situation in the country [34, 39, 185, 411]. In this situation, there is a retrograde colonization of HP infection from the antrum into the body of the stomach, that is accompanied by a decrease of the annoying effects on G-cells by HP due to reduction of bacteria concentration in the antrum and amplification of HP infection neutralizing impact on the hydrochloric acid, due to increase of bacteria concentration in the body of the stomach (a state of pathological equilibrium). This step can be schematically represented as follows (Figure 27).

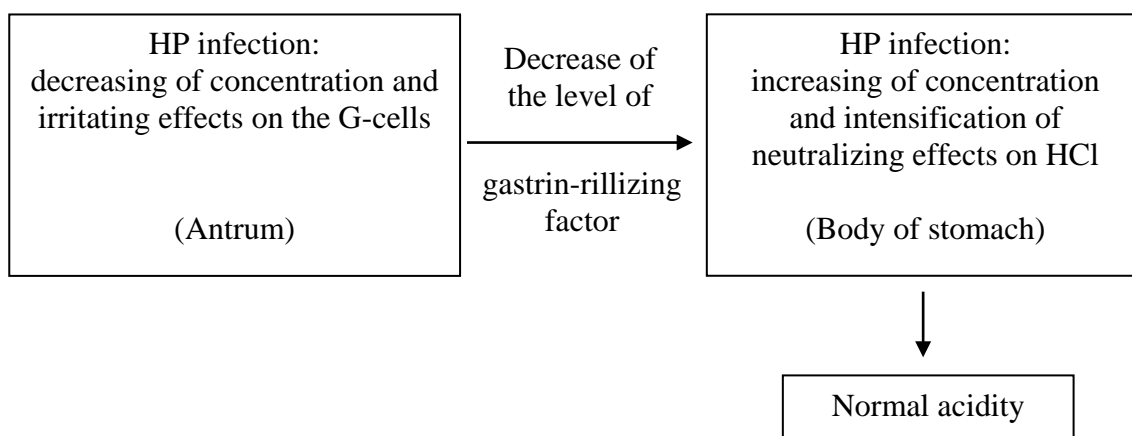


Figure 27. Scheme of the stage of "pangastritis" formation

3rd stage - the stage of the initial changes in the morphology of the gastric mucosa glands and the formation of functional hypoacidity (anacidity)

This stage is developed after 45 years and can last up to 60 years, when the HP infection maximally moves in the gastric body, and has the most neutralizing impact on HCl, and in the antrum it is detected or at a minimum concentration in active form or in the form of coccus of the type II, which are ready to be released in the intestine, or a mixed variant is possible [20, 34]. In such a stage begins morphological change of antral mucosa: histological examination often reveals atrophy, metaplasia and dysplasia in differing degrees of severity, which is confirmed by the results of our research. Schematically, this stage can be represented as follows (Figure 28).

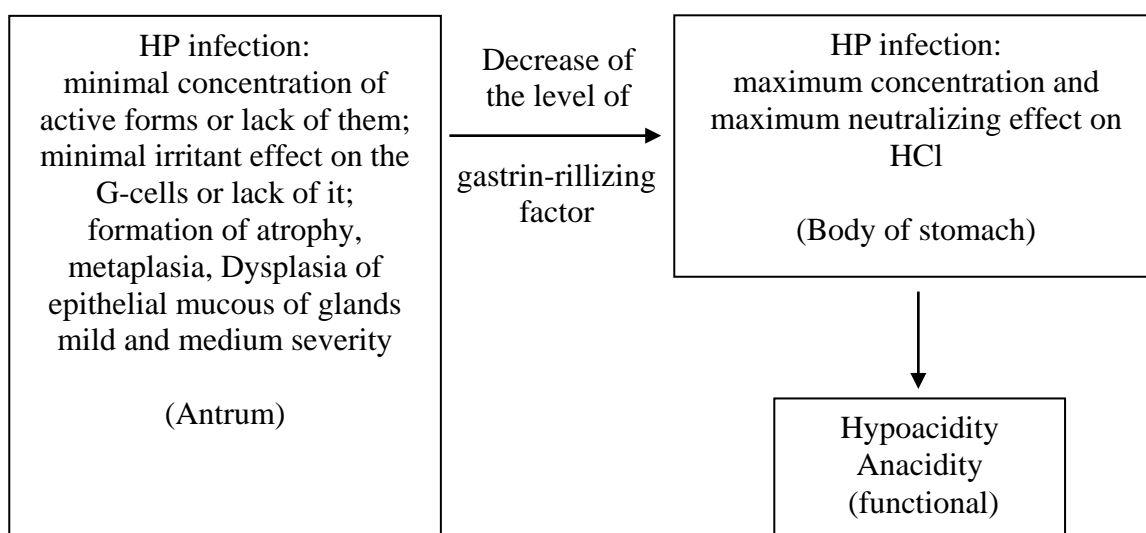


Figure 28. Scheme of the stage of the initial changes in the morphology of the gastric mucosa glands and the formation of functional hypoacidity (anacidity).

The second and the third stages are reversible: after successful eradication the acidity level is restored; mild and medium severity atrophy, metaplasia, and dysplasia of mucosa glands epithelium undergo the involution, which is confirmed by the results of our researches, and by the results of studies by others [20].

For the second and the third stages the presence of intracellular forms of HP infection is definitive, when HP is blocking the function of parietal cell ab intra. [34] These conditions can be exposed to management with a help of eradication by our scheme [161]. This is even more significant, considering the fact that the presence of HP near the parietal cell nucleus, especially during mitosis, may increase the risk of mutation, and under compromised immunity can lead to the development of gastric cancer [24, 34].

4th stage - the stage of the formation of a total atrophy of the gastric mucosa glands.

This stage is marked in its classical form after 65 years, when the atrophy of the mucosa glands epithelium of both the antrum and corpus has been observed, which is accompanied by severe organic hypoacidity or anacidity[202, 350]. At this stage, the active forms of HP infection are rarely detected, only detected at low concentrations, or not detected at all, and only the presence of coccus of the second type are noticed, that have, in our view, transient etiology. This confirms the view, that for the normal vital activity of the active forms of HP infection a fully functional mucosa of gastric type is required [34, 66]. Schematically, this stage can be represented as follows (Figure 29).

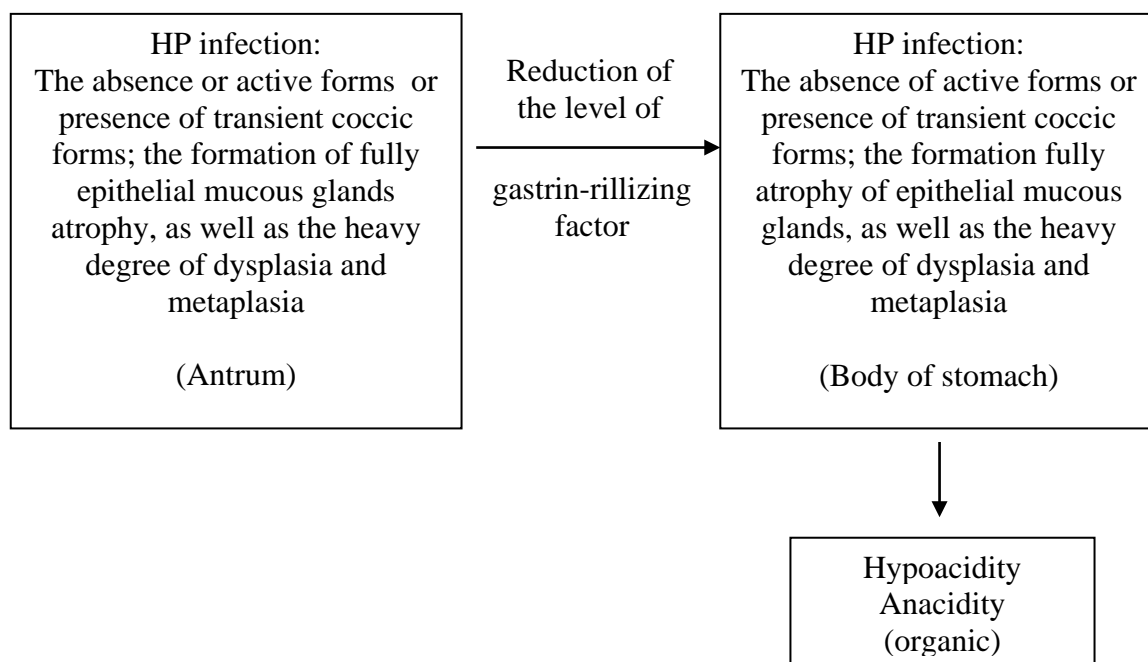


Figure 29. Scheme of the stage of the formation of a total atrophy of the gastric mucosa glands.

This step is not reversible; severe atrophy, metaplasia, and dysplasia of the mucosa epithelium refer to a precancerous state that necessitates early recognition and appropriate eradication of HP infection before the progression of abovementioned pathological changes [34, 54, 167, 303, 350].

The occurrence of staging progression of the chronic gastritis type B requires a detailed individual examination of each patient in order to obtain full information, what is possible by carrying out complex examination of patients we have proposed [301].

It is critical to underscore that there is no clear age-related gradation of chronic gastritis type B progression, because this process is influenced by both - external factors (ecology habitat, work style, participation in social groups on exit to the independent life, family relationships, atmosphere in a country where the patient lives) and internal factors (psychoemotional state of the patient, social habits - alcohol, smoking, drug use), which requires an individual approach to diagnosis and treatment, what

most completely satisfy the schemes of complex examination and treatment we proposed, that are based on deep knowledge of the pathophysiology of chronic helicobacteriosis and, also, on rational pharmacotherapy, based on a knowledge of the drugs medical claims, that are used in the eradication schemes, and taking into account the properties of the HP infection itself [12, 20, 34, 115, 336, 347, 360, 428, 467, 661].

1.4.3 The main theses of the "caustic alkali exspuition" theory.

Theory of "caustic alkali exspuition" (Avramenko A.A., Gozhenko A.I, 2007).

Before expressing the statements of the new theory of ulceration, it is necessary to place greater focus on the contradictions in the views on the etiology and pathogenesis, which exist in the world gastroenterology. First of all, the arguments of scientists who do not recognize the role of HP in the ulcer etiology. Their impugments come down to the following:

- 1) peptic ulcer develops only 1 in 8 contaminated;
- 2) Organ – preserving operations, because of peptic ulcer, don't have a significant impact on gastric mucosa and duodenum oecizing with HP infection, but in the better part of monitoring lead to ulcer elimination;
- 3) A significant ulcer-healing outcome is produced by the administration of modern high powered antisecretory drugs (H2 – histamine antagonists and other), despite the maintenance of HP in gastric mucosa and duodenum;
- 4) Men preferentially suffer from peptic ulcer, while the density of gastric mucosa and duodenum colonization is not connected with the gender;
- 5) PU has a polycyclic progress of a disease, so that ulcerations are healing because of treatment, without treatment and even contrary to treatment activity, despite the persistence of HP in gastric mucosa and duodenum;
- 6) Hyper secretion of hydrochloric acid and pepsin can cause the ulceration in an absence of HP [328, 389];
- 7) Some researchers even deny the connection of HP with abnormal changes of gastric mucosa, assigning HP a part to saprophytes or consecutive infection[267, 389, 537], as the contagious conception of peptic ulcer etiology can't explain the solitariness of ulcerous defect, alternation of relapses and remissions, seasonality of recrudescences and other processes [389]. However, the following facts acknowledge the role of HP – infection in ulcer development:

- 1) frequent HP contamination of ulcerous persons;
- 2) while long-term follow-up for HP-contaminated patients ulcer develops on a more frequent basis in a group of HP-contaminated, then in a group of non-contaminated;
- 3) by the eradication of HP a significant frequency reduction of ulcer relapses is observed – from 60-100 to less than 15% [389,668].

Confirmed facts remain unexplained:

- 1) Peptic ulcer can develop in any type and degree of acid-forming function, including the hypoachlorhydria. Ulcer cicatrization timescales don't depend on degree of hypersecretion, it does not affect the nature of the exacerbations, and upon the occurrence of remission secretion level does not change and remains constant, the same as at the time of exacerbations [34, 121].
- 2) the formation of mediogastric ulcers come against the background of normal acidity and hypoachlorhydria, and this the average size of ulcerous defects exceed the average size of ulcers of pyloric bulbar zone in 2-3 times [342].

3) typical localization of mediogastric ulcers is middle - upper third of the gastric body along the lesser curvature of stomach, while after gastric secretion the gastric juice is concentrated in the gastric fundus along greater curvature of stomach [342].

4) contradictions in the understanding of the ulceration etiology and pathogenesis affect the conception of the clinical implications of PU. For example, such typical symptom as pyrosis, which is interpreted as an implication of hyperacidity, has been noted at any level of acidity [34, 42].

Taking into account a number of contradictions in the understanding of the ulceration etiology and pathogenesis, which cannot be explained only from the position of the imbalance between the factors of aggression and defense, interplaying at the local level, the researchers of this problem are bound to acknowledge, that the shroud of mystery, that hides, in the words of J. Cruveilhier, the main reasons and mechanisms of PU progression, is not completely cleared up, and up to date [403].

Thus, summarizing all the abovementioned, it is possible to reach an unconsoling conclusion: modern gastroenterology, at the views on the etiology and pathogenesis of PU, as well as the role of the chronic gastritis type B in this process, is in a dead end, and the reasons for this, in our opinion, started from a postulate, that was put forward in 1910 by Schwartz: "No acid - no ulcer" and was interpreted as formerly, and up to now, as "Acid is the cause of the ulcerous defect". Discovery of HP infection has put forward a new postulate "Without HP-associated gastritis there is no ulcer", however, the therapeutic regimens continue to include in addition to anti-helicobacter drugs, drugs that suppress the production of HCl - Proton pump inhibitors [34, 167, 303]. The trouble is that each postulate was put forward on a standalone basis, resulting in a monodirectional, and therefore, not a complete vision of the problem. We will try to show what the problem is, converting and combining these postulates into a single formulation.

First of all, in the first postulate we replace the word "acid" on the phrase "acidic medium of the gaster," which is more correct, and the second postulate will be replaced by the expression "the gastric mucosa, where HP infection inhabits" and combine both postulates into a new wording: "Without acidic medium of the gaster, on the gastric mucosa of which, HP infection inhabits, there is no ulcer". Now we simplify this expression and obtain the following wording "Without acidic medium of the gaster, where HP infection inhabits, there is no ulcer". Interchanging words, we will receive the final wording "Without HP infection, that inhabits in acidic medium of the gaster, there is no ulcer", that does not contradict with any knowledge about the habitat or topographic zone of gastro-intestinal tract, where active forms of HP are developing.

The importance of this formulation is that it has two interpretations:

1) In reference to the gastric mucosa are aggressive both - medium of the gaster, and the aggressor that inhabits there - HP;

2) In reference to the gastric mucosa HP is mainly aggressive, and acidic medium of the gaster is only its habitat, which is aggressive to Helicobacter infection.

The second interpretation of the wording we have received is valid in all respects, because it explains why ulcerous defects are developing at any level of acidity: **ulcerative defects are forming at any level of acidity, because acid-peptic factor does not damage the mucosa of the gastroduodenal zone!**

Why is this statement correct? This statement is correct, because the acid-peptic factor and the factors that protect the mucosa from its damaging effect, laid into us genetically and work, what has long ago been proven, simultaneously: once the HCl level increases, then, immediately, the level of bicarbonate increases [34 121, 257]. The organism has more than one level of protection: bicarbonates, gastric mucin, prostaglandins, well-developed circulatory system, the rapid regeneration of the gastric epithelium, which is comparable to a computer program with 5 levels of protection: while, at least, one level of protection is operating, the programme won't be out of order. However, when the depletion of the protective forces of the mucosa approaches, i.e. occurs the atrophy, in parallel occurs the atrophy of the glands, that produce aggression factor - the acid-peptic factor [34, 202, 350], but the lesions will still occur, especially while gastric ulcer in the elderly.

In addition, recent histologists studies cast doubt on the view that the leading element of the pathogenesis of peptic ulcer is an imbalance between acid-peptic aggression of gastric juice and protective elements of gastric mucosa, that leads to the predominance of aggressive factors, the pride of

place among which goes to the active hydrogen ions (AHI), secreted by parietal cells. Authors point out, that a common concept, that mucus and bicarbonate are the most important factors for protecting the gastric mucosa and the duodenal mucous membrane due to the lack of ways to get a real picture of AHI, was reflected only in a theoretical scheme, that includes the mechanisms of secretion of AHI and protection factors of mucous membrane from their damaging effect [369, 675]. When painting the gastric mucosa with nitroblue tetrazolium, which under the influence of AHI turns to dark navy formazan crystals, it turns out that AHI are located diffusely and evenly all over depth of the fundus of the stomach mucous membrane. Even relatively large, compared to other places, AHI accumulation, observed in the zone of the epithelial cells joints, does not cause damage to the mucous membrane cells. The gastric mucosa is colored areas in zones of localization not only parietal, but also superficial cells, which secrete mucus and bicarbonates [369].

But the most interesting, is that the color intensity of pyloric mucous membrane is not inferior to that the same in other parts of the gaster, although at current views mucous membrane of this part of the gaster should not have been painted so intense, it is evidence that AHI are placed diffusely and evenly over the entire thickness of mucous membrane, without disturbing its integrity [369].

In addition, accusing the acid-peptic factor in deterioration of the mucosa of the gastroduodenal zone, no one bothered to compare the picture of the damage, that occurs at peptic ulcer, with the data of three sciences - biochemistry, toxicology and forensic medicine, which describe - how mucosal lesion must look like, when it is in contact with acid . Upon contact with the acid on the mucous membrane forms coagulative (dry) necrosis - necrosis of protein tissue, which is characterized by a dense formation[16, 145]. According to forensic medicine, in the area of the contact with acid, irritation, inflammation, combustion, tissue destruction appears. Necrotic tissues constitute rather compact crusts, slightly raised above the rest of the unaffected tissue and surrounded by areas of inflammation. Under the influence of H^+ hemoglobin breakdown is taking place: due to its produced derivatives (haematoporphyrin, ferrihaemoglobin, acid hematin)its tissues gain on dark brown or brownish-black color, what practically can be only confirmed amid gastrointestinal hemorrhage [16, 145].

Depending on the type of disturbing factor in the situation, hemoglobin breakdown doesn't take place, crust has its indicative color. At this rate, the crust, that forms after the combustion caused by nitric acid, has an indicative yellow color; after the combustion with sulfuric acid - brownish-black color, **and after the combustion caused by hydrochloric acid the affected mucosa is characterized by slate-gray color (grey-black color, with a cyanotic tincture), and the mucosa is affected over a large field [16, 83, 414]**. The folds of the gastric mucosa become dry, brittle, or smooth, so that the mucous membrane is flattened, it becomes thin, dense, but the perforation of the gastrointestinal wall because of the lesion of the mucous membrane with hydrochloric acid doesn't take place. Regenerative process also has its own peculiar properties: tissue heals under a crust, which exfoliates, and until the mucous membrane is not completely restored, the crust won't entirely desquamate from the lesion location [16, 83, 414].

We believe that the injury pattern of mucosa due to exogenous penetration of hydrochloric acid is genuine for the damaging action of HCl, because the mucosal immunity is not currently activated, and there comes the perficient imbalance between aggression and protective factors. But in practice, while the endoscopy of peptic ulcer patients, entirely different picture is determined.

How do the verum ulcers with localization in different zones of the digestive tract while peptic ulcer look like? The answer to this question is given by images (Figure 30, 31, 32, 33).



Figure 30. Duodenal bulb ulcer in an active phase

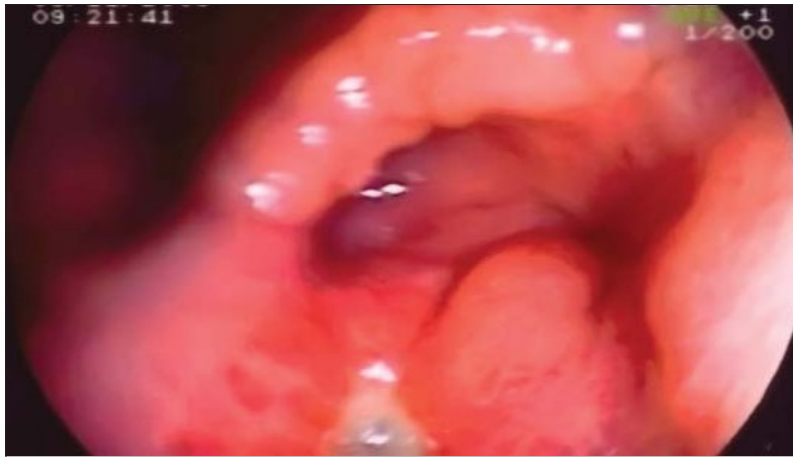


Figure 31. Antrum ulcer (prepyloric zone) in an active stage



Figure 32. Corporeal gastric ulcer across the lesser curvature of stomach in the middle third in an active stage.



Figure 33. Necrosis, covering the ulcerous defect in an active stage.

As it can be seen from these photographs, as well as from personal 15-year-old term of service as an endoscopist, a true gastrointestinal ulcer has a completely different view: ulcerous defect is local; fibrin and necrosis, which cover the defect, are white or gray-yellow color (depending on the stage of healing of the defect) [56]; often, apparent inflammatory elevated border on the periphery has been noted, creating a crater picture and necrosis elevation is never tracked above the healthy tissue [342]; Necrosis has a quaggy character in 100% of cases, which allowed us to strip it easily, while the biochemical studies on the identification of damaging factor.

The cicatrization of the ulcerous defect at PU also has a definitive dynamics: initially, in the first phase defect has the picture that was described above. After 7-14 days water thesaurismosis on the edges begins to dissipate, the ulcer d begins to flatten in depth, which leads to a relative ulcer auxesis. In parallel, cleaning of the fundus from necrosis is in progress and the acentric rim of granulation tissue emerges. On the 21 - 24 day, according to cicatrization the damaged surface breaks to small separate areas (partial epithelialization stage, or stage of "confluent" erosions). Further, depending on the condition of the tissue and the influence of the range of both, endogenous and exogenous factors, impurity spot is healing in three types: in mucous type, in the form of "white" rumen (thin delicate rumen) or in the form of a "red" rumen (rugged thick scar) [342].

What is more, affection with the hydrochloric acid comes to a dense wall of muscle layer and does not outstep, what is characterised by unusual occurrences of perforations, although, perforation is not a rare phenomena in PU pathogenesis [16, 34]. Also, upon completion of the ulcer and type B chronic gastritis treatment with the usage of PPI or without them, after successful eradication has been noted the "return phenomenon" - increase of gastric acid production (after PPI - at least 8 weeks), which, however, does not lead to new ulceration [21, 317, 488, 496]. Moreover, during experimental work on animals for the obtainment of ulcerated mucosa of the small intestine, the last is irrigated with hydrochloric acid at a concentration of 80-130 millimoles per liter [117], while according to our studies the concentration of free hydrochloric acid in the gastric juice that reaches 80 millimoles per liter and higher is a very rare phenomenon. We detected only 3 cases out of the 321 researches (0.9%): 1 case (0.3%) (the concentration of HCl - 81 millimoles per liter) - patient with chronic helicobacteriosis without erosive and ulcerative lesions of gastroduodenal zone in the active phase ; 1 case (0.3%) (HCl concentration - 89 millimoles per liter) - patient with chronic helicobacteriosis with ulcerative lesions of the pylorus in the active phase; 1 case (0,3%) (HCl concentration - 80 millimoles per liter) - patient with chronic helicobacteriosis with ulcerative lesions of the duodenal bulb in the initial stage of epithelialization, that says more about the exceptions to the rule, than on the regularities.

Thus, putting two and two together, we can reasonably omit accusation against acid-peptic factor, as a damage factor, take into account its affinity for necrotic tissue and its role in ulcer abrasion from necrosis, which even more emphasizes the discrepancy in views on the role of acid-peptic, as primary and basic factor of aggression (it turns out that the acid-peptic factor first destroys the mucosa and then upregulates its cicatrization?) [134].

Now the explicable question arises: "If it is not an acid-peptic factor, what factor is the factor of the damage?" To answer this question we must seek assistance in some writings in biochemistry, toxicology and forensic medicine. Loose necrosis of gray and white color, deep affections with apparent perifocal inflammation, tendency to deep affections down to the perforation is peculiar to lesions of another class of chemicals - caustic alkalis. Alkalis go with the help of hydroxyl ions (OH⁻ ions). Unlike acids, alkalis, reacting on protein with large masses of hydroxyl ion, cause its swelling, then, melting and dilution with a formation of alkali albuminates, that are freely soluble in water. Due to the dissolving action, alkalis can easily penetrate into tissue depth, forming a thick layer of liquefactive (colliquative) necrosis, which is characterized by looseness that what determines a greater depth of damage and prolonged healing period. Caustic alkalis easily dissolve not only the mucosa, but the muscle tissue, which often leads to organ perforation [16, 83, 145]

Which caustic alkali can be formed in the gastric cavity and damage the tissue? The only substance that is formed in the gastric cavity and is in an aqueous solution refers to the caustic alkalis, is the ammonia (in contact with the moisture of the gas mixture of the gastric cavity, in our view, forms ammonium hydroxide - ammonia spirit, that distinguish particularly irritating and deep cauterizing action, by long-term effect of which, occurs the mucosal detachments in the form of bubbles and the subsequent development of necrosis) [16, 145]. As we know, all strains of HP-infection are characterized by one common thing - occurrence of urease enzyme, which degrades urea to ammonia that goes to neutralize the acid-peptic factor of the hydrochloric acid. However, not the entire ammonia goes to neutralization: the part of ammonia accumulates in the gastric lumen ("residual" ammonia-RA). The higher the concentration and dispersal area of HP infection on the gastric mucosa, the higher will be the concentration of RA in the gas mixture of gastric cavity [52].

it is important to emphasize that the assumption of the possible role of ammonia as a factor of damage were put forward earlier [515], and, most interestingly, it was confirmed in experimental studies by Japanese scientists. They studied the effect of urea and products of its enzymatic hydrolysis on the gastric mucosa of rats. They determined that the urea concentration of 0.25 - 2.5 mg / ml did not cause pathological changes in the stomach of the test animals. After administration by oral gavage under the urethane anesthesia 100 - 1000 action units of urease, ammonia concentration increased rapidly, at the same time pointed out the negative effect of urease on motor function of the stomach and gastric blood flow. The most important thing is that, what was found by them: increasing the concentration of ammonia causes necrotic lesions in the gastric mucosa, in other words, it has been experimentally proven that the products of urea decomposition with the participation of urease of HP have a direct destructive effect on the gastric mucosa and can be involved in the pathogenesis of gastritis and ulcer! [335].

1.4.4 Horizontal and vertical mechanism of ulcerous defects formation of different localization.

But what is the mechanism that leads to the appearance in humans gastric cavity highly concentrated solution of ammonium hydroxide - ammonia spirit, and the role it plays in the formation of ulcerous defects of various localization at PU? To answer this question, we must seek assistance in some writings in physics and aerodynamics, which are cross functional and suitable for both cars and for living system [135, 343, 380, 409].

RA concentration in the gastric cavity will depend on two factors: the amount of its income in a gastric cavity and the amount of its removal from the gastric cavity. Adding the concept of the gastric cavity as a container with a certain volume, which has two channels to remove RA-pyloric sphincter cavity with the transition to duodenum and cardiac sphincter cavity with the transition to the esophagus to our armoury, it is possible to draw an analogy with the math problem of school course about the pool and the two tubes with one difference: pipes serve to remove the contents of the basin, while the surface of the pool wall or partly (in case HP infection is localized only on the mucosa of antrum or corpus) or fully (if pangastritis) produce the content (in this case, ammonia).

RA number of entering the gastric cavity, as mentioned above, depends on the area of resettlement and concentration of HP infection on the mucosa, that is, the number of bacteria- bacterial mass. Number of remote RA will depend on the diameter of the "pipe" - pyloric and cardiac sphincters [135].

According to our data, the main role in this process need to be given to pyloric sphincter, because all the examined patients had a well-closed cardiac sphincter without evidently visual hiatus, while the diameter of the pyloric canal and the tonicity of its walls was in marked contrast by groups (on the role of the cardiac sphincter in the process of removing ammonia will be discussed below).

According to the data of our researches it was found out, that in forming duodenal bulb ulcer, diameter of pyloric canal on the average equal to 0.73 ± 0.02 cm, and the tonicity of the pyloric sphincter - 8.29 ± 0.53 s., while as in forming of pyloric and pyloric bulbar ulcers, diameter of the pyloric canal was 0.36 ± 0.11 cm and for prepyloric ulcers - 0.1 ± 0.12 cm and the tonicity of the pyloric sphincter - 11.2 ± 0.61 seconds.

This tendency is quite understandable from the viewpoint of aerohydrodynamics laws and anatomy of the area. With the gas passage from the volume cavity (body of stomach) through the segment, with a gradual decrease of the cross-section area (antrum (shape - truncated cone) in a narrow space, that have a certain diameter (pyloric canal), the so-called Borda nozzle is formed, which is characterized by an increase in gas density and the formation of the firm gas efflux [409]. However, the final place of the formation of the largest jet head of gas and its concentration is observed at the contact point of the gas jet with an obstacle on his way [380]. In this situation, such obstacle a wall of the duodenal bulb, where concentrated ammonia, that reacted with H₂O, forms an alkaline drop of ammonium hydroxide that leads to the formation of the ulcerous defect. This statement is confirmed by the studies of Prof. Y. Reshetilov, about high humidity in the gastric cavity (85-96%) and duodenum (98%), as well as data on the operation of ammonia refrigeration machines, the limit of tolerance of moisture content in ammonia - no more than 0.2%, because of a large amount of moisture index forms great number of exhibited all the chemical properties of ammonium hydroxide, which corrodes the zinc and copper alloys [103, 157, 520].

While reducing the nozzle diameter, the concentration of ammonia and H₂O can increase in the channel, which can lead to the formation of alkali and the formation of a defect or in the channel or at its outlet (pyloric bulbar and pyloric ulcers). Under the firm spasm of the pyloric sphincter ammonia and H₂O concentrate and form ammonium hydroxide alkaline drop in front of him (prepyloric ulcers taking into account the fact that the antrum is shaped like conoid).

Formation of erosive lesions in the duodenal bulb also has its own peculiarities. Minor in size erosions (up to 0.4 cm in diameter) are formed at the tightly compressed pyloric sphincter, but at lower RA concentration in the antrum than in duodenal ulcer, that stipulate the formation of the final concentration of ammonia and H₂O only in the outflow tract of the pyloric sphincter and occurs the effect of "injection" (under high pressure line droplets of ammonium hydroxide splash across the bulb mucous membrane, inflicting only superficial damage by brief contact of influencing factor, which is confirmed by endoscopic examination). Formation of large erosions (0.5 cm and more) has another mechanism. In this situation, pylorus is not closed, but is opened as far as possible (average diameter - 0.94 ± 0.06), and the increase in the concentration of ammonia and H₂O occur in anatomically narrow place –the bulb outflow tract. Here, the contact of damaging factor is longer-term than under the formation of small erosions, but RA concentration in the gastric cavity is smaller than at PU, that stipulate shallow, though large in size injuries of mucosa with ammonium hydroxide, which is confirmed by endoscopic examination.

How creates elevated pressure and therefore a high concentration of ammonia in pyloric bulbar zone? According to the anatomy and physiology of gastrointestinal tract, the major role in the removal of the contents of the gastric cavity performs peristaltic wave.

Immediately, after eating there is a relaxation of the stomach fundus (process is called accommodation); as a result the stomach is able to take a fairly large amount of food. Subsequent stirring of food with gastric juice and chymus moving occur with the help of peristaltic contractions of the stomach wall. Wave is born in the field of cardiac pacemaker. Hence, muscle contraction spread at a rate of 10-40 cm / s, gradually increasing to the pyloric [383, 515]. There are three types of waves: the first type - low amplitude wave, duration 5-20 seconds; the second type - the higher amplitude wave, duration of 12-60 seconds. This waves keep the tonicity of the stomach and conduce the slow mixing of the chymus, directly adjacent to the wall, with the gastric juice. As far as the food digest in the stomach there is a third type - a special peristaltic wave

that reaches such an increase of intraventricular pressure, at which the pyloric sphincter opens and a portion of the chymus enters the duodenum. Such a wave is called peristaltic wave of propulsive nature, or propulsive wave or systolic contraction of the pylorus. These waves occur in the cardia region, but the greatest contraction is observed in the pylorus; the frequency of these waves – up to 6 or 7 in 1 min. Due to this wave, pressure in the region of the pylorus increases up to 20-30 cm of water column and becomes higher in the duodenum, while in the fundic of the stomach gastric pressure under summation of slow tonic contraction of the stomach muscles and rapid phase contractions, extending only 10 - 15 s., achieves 45-65 cm of water column [59, 383]. Evacuation speed depends on the consistency and quality of food composition. So the evacuation of liquid food begins almost immediately after its reception; dense foods stored in the stomach up to 4 - 6 hours, while carbohydrates and proteins leave the stomach rapidly than fats. An important role in the evacuation of stomach contents plays the so-called antra-duodenal coordination - synchronous pyloric relaxation while contracting the antrum [60].

Because of stress, a violation of antra-duodenal coordination (increase of the pyloric sphincter tonicity) takes place, increases peristalsis (hyperkinetic disorder), so that, the speed and peristaltic wave depth rise sharply, creating a situation that could be called as the effect of "piston" [60, 150, 228, 403]. If this occurs, the pressure sharply increases and the densification of ammonia and H₂O in prepyloric zone arises, that entails the formation of the above-mentioned damage mechanisms of pyloric bulbar zone. However, there is also an exogenous way of gastric pressure increase - sharp pressure on the stomach from the outside ("bellows" effect) associated with: 1) a sharp tension of anterior abdominal wall muscles (physical labour, exercise); 2) during back massage; 3) stroke (accidental or intentional) in the abdominal region 4) laparoscopic operations (sharp increase of intra-abdominal pressure due to the administration of carbon dioxide by 12-14 mm Hg (16.3 - 19 cm of water column) [154, 429], that confirmed by our studies. In these cases, according to Pascal's law, pressure increases not only in the abdominal cavity, but in all hollow organs located in it, including the gaster [248, 457]. There are also mixed embodiments. All these features can be grouped into elements of "horizontal" mechanism of formation damage factor.

According to our studies with pyloric canal diameter from 0.1 to 0.4 cm and an increased tonicity of the pyloric contactor, at a concentration of "residual ammonia" in the antrum from 7.1 to 9.5 mmol/L and by increasing gastric pressure with physical load of 45 to 100 cm of water column, the conditions for forming the "injection" phenomenon occur, when increasing the concentration of ammonia from 90 to 300 mmol/L leads to the formation of droplets of concentrated alkali - ammonium hydroxide, which is formed, already at the outlet of the pyloric canal and under high pressure sprayed on the mucosa, forming multiple, but not a deep damage, known as "erosion of duodenal bulb."

Graphically, this process can be represented as follows (fig.34).

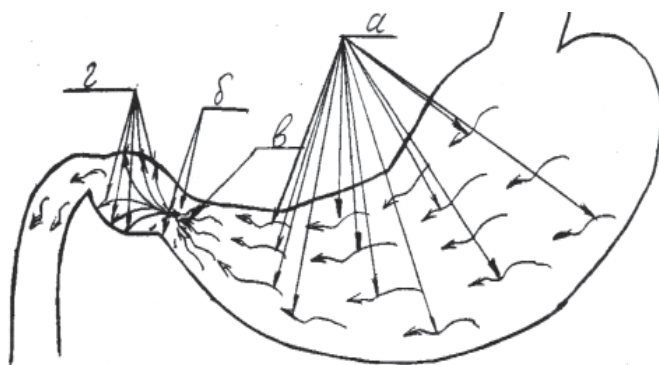


Figure 34. The mechanism of the formation of erosive lesions of the duodenal bulb in patients with chronic helicobacteriosis:

- a) flow direction of "residual" ammonia in the gastric cavity;
- b) pyloric sphincter in spastic condition;
- c) the place of the maximum concentration of "residual" ammonia (prepyloric zone of the antrum);
- z) places of erosive lesions of the duodenal bulb mucosa ("spread" across the entire mucosal area).

When the diameter of the pyloric canal is from 0.8 to 2.0 cm, and the absence of increased tonicity of the pyloric contactor, at a concentration of "residual" ammonia in the antrum from 6.8 to 11.1 mmol /L and by increasing gastric pressure during physical load from 47 up to 89 cm of water column, creating such conditions, when increasing the concentration of ammonia from 99 to 272 mmol /L leads to the formation of droplets of concentrated alkali - ammonium hydroxide in anatomically tight spot - in the bulb outflow department, where mucosal damage are tangential in nature and achieve small-sized area ulcers, however, do not differ from the usual erosion ("erosive and ulcerative bulbitis ") for the depth of the damage.

Graphically, this process can be represented as follows (Figure 35).

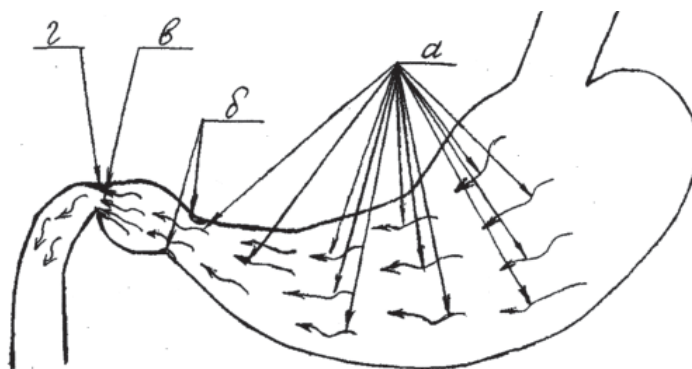


Figure 35. The mechanism of formation of erosive and ulcerative lesions of the duodenal bulb in patients with chronic helicobacteriosis;

a) flow direction "residual" of ammonia in the gastric cavity;

b) the pyloric sphincter in gaping condition;

c) the place of the maximum concentration of "residual" ammonia (output of duodenal bulb);

z) the place of erosive and ulcerative lesions of the duodenal bulb mucosa ((output of duodenal bulb).

When the diameter of pyloric canal is from 0.4 to 0.9 cm and an increased tonicity of the pyloric contactor, at a concentration "residual" ammonia in the antrum from 10.6 to 12.9 mmol /L and by increasing gastric pressure during physical load from 76 to 100 cm water column, creating such conditions, when increasing the concentration of ammonia from 88.1 to 146.6 mmol / L leads to formation in local place of the duodenal bulb mucosa drops of concentrated alkali - ammonium hydroxide, which lead to damage of the mucous wall of the bulb, known as "peptic duodenal ulcer." Formation of "kissing" ulcers of duodenal bulb depends on the shape of the pyloric canal and bulb, which have an oval skylight, due to both individual anatomical features and the result of abnormal deformation (cicatricial deformity).

Graphically, this process can be represented as follows (Figure 36).

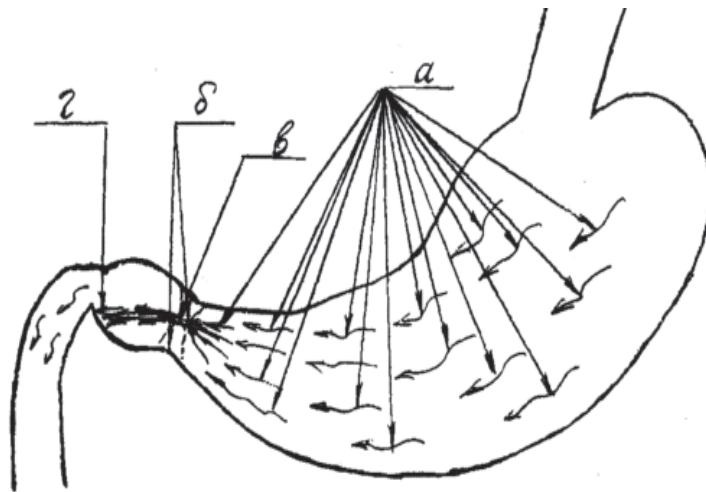


Figure 36. The mechanism of formation of ulcerative lesions of the duodenal bulb in patients with chronic helicobacteriosis;
 a) flow direction "residual" of ammonia in the gastric cavity;
 б) the pyloric sphincter in half-bent state;
 в) the place of the maximum concentration of "residual" ammonia (pyloric canal) in the form of sustainable dense flow;
 з) the place of ulcerative lesions of the duodenal bulb tissues (Any bulb department 12 duodenal ulcer).

When the diameter of pyloric channel is from 0,1 to 0,6 cm and there is a significant spasm of pyloric sphincter, at a concentration in the cavity of the stomach "residual" of ammonia in the gastric cavity from 9.6 to 12.9 mmol /L and at increasing of intragastric pressure due to physical activity from 72 to 100 cm of water column, such conditions are created, when ammonia concentration rise from 76,7 to 300 mmol /L leads to the formation of concentrated alkali droplets - ammonium hydroxide in prepyloric zone, in the pyloric canal and in the junction of the pyloric canal in the duodenal bulb, where the mucosa defects are formed, known as "prepyloric ulcer" and pyloric bulbar ulcer. "

Graphically, this process can be represented as follows (Figure 37).

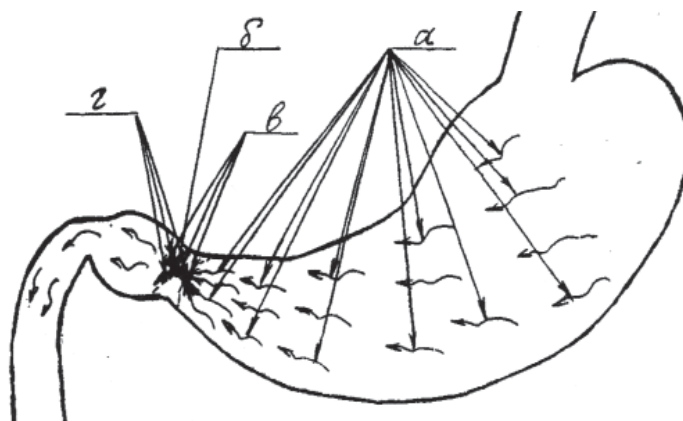


Figure 37 The mechanism of formation of ulcerative lesions of in the prepyloric zone of the antrum, in the pyloric canal and pyloric bulbar zone in patients with chronic helicobacteriosis:
 a) flow direction "residual" of ammonia in the gastric cavity;
 б) the pyloric sphincter in spastic state;
 в) the place of the maximum concentration of "residual" (the prepyloric zone of the antrum, the pyloric canal and pyloric bulbar zone);
 з) the place of ulcerative lesions " (the prepyloric zone of the antrum, the pyloric canal and pyloric bulbar zone).

How the mechanism of gastric body ulcers is formed (mediogastric ulcers)? This mechanism has its features, so that it can be called "vertical" mechanism. In the analysis of the concentration, the topography of the settlement and the position of HP towards the parietal cell (extracellular or intracellular) clears up one peculiarity: in the lesser curvature of the gastric body (zones of ulcers localization) HP is detected at a high concentration - (+++) - (++++), but either partially or completely located intracellularly in parietal cells, thus either partially or completely blocking the production of hydrochloric acid in this zone. At the same time under ulcerative defect on the mucosa over greater curvature of the body of stomach HP is also determined at a very high concentration - (++++), but all the bacterial mass is located on parietal cells of the mucous surfaces.

In the antrum HP active forms, capable of the production of ammonia in the active phase of the pathological process or absent (to our knowledge - in 36.4% of cases), or are at a much smaller concentration than in the body of the stomach (according to our data, in 5 - 45 times). This peculiarity of the HP position on the gastric mucosa determines a powerful stream of RA, which is formed in the gastric mucosa at the greater curvature, and rushes from the bottom upwards to the lesser curvature, exposing the mucosa of this zone, deprived of acid protection, threat of alkali damage. However, this is not enough: the final pressure increase and the concentration of ammonia and H₂O in the zone of future damage, resulting in the formation of ammonium hydroxide, appears during physical activity (according to our data in all patients in 100% of cases). This is stipulated due to the peculiarities of the contraction of muscles of the anterior abdominal wall: they contract from the bottom upwards, passing the direction of contraction on the stomach (from the bottom to the lesser curvature) and sharply increasing the movement of RA to the zone of the future damage, that is most often found in the middle third of the body of the stomach (to 50.4%), while the incidence of cardial and subcardial ulcers of the stomach ranges from 0.3 to 18.4% [59, 62, 383, 418, 419]. Also a definite role in this process play peculiarities of vascularisation of the lesser curvature of the stomach of the body: the portion lesser curvature (2,3 ± 0,3 cm) supplies with blood only with a small secondary branches of large gastric arteries, which stipulates relative hypoxia, which intensifies with age under the development of atherosclerosis, and improves pattern for intracellular penetration of HP (HP- microaerophil), which leads to the neutralization of the acid protection, and, therefore, facilitates to formation of ulcerous defect [31, 96, 217, 356]. Therefore, ulcerous defect of this zone are formed not so often, taking into account the specificity of the formation of this mechanism, but can have a size of 2-3 times more than in pyloric bulbar area, as the rigid directing nozzle is missing, when forming mediogastric ulcers are formed [342]. In addition, in a combustion with caustic alkali, necrosis boundary goes far beyond the direct action of alkali, which also explains the large size of mediogastric ulcers and a significant (p <0,001) mismatch between the size of ulcers in the duodenal bulb (average dimensions 1,19 ± 0,04 cm) and diameter of pyloric canal (average dimensions 0,73 ± 0,02 cm) in the active stage [16].

Thus, according to our data, in a vertical formation of "alkaline drops" ammonium hydroxide in the stomach major role play:

a) extracellular active forms of HP infection with a high concentration - (++++), which are located on the mucous membrane in the body of the stomach along the greater curvature and create a powerful stream of "residual" of ammonia at a concentration from 11.8 to 23.2 mmol /L, directed from the greater curvature of the gastric body to the direction of the lesser curvature;

b) intracellular active forms of HP infection with a high concentration - (++++), which are located in the parietal cells of the mucous membrane in the body of the stomach along lesser curvature and block the secretion of hydrochloric acid, which leads to the disappearance of the acid protection of this zone;

c) a low concentration of active forms of HP or their absence on the mucosa of the antrum along the greater and lesser curvature behind gaping pyloric sphincter (from 0.8 to 2.0 cm);

d) increasing intragastric pressure during physical activity from 79 to 100 cm of water column, which, eventually, concentrates ammonia from 89.3 to 134 mmol /L in the lesser curvature of the gastric body, which is devoid of the acid protection, what facilitate the formation of a concentrated alkali - ammonium hydroxide and the mucous membrane damage, known as the "mediogastric ulcer."

Graphically, formation of mediogastric ulcers in "vertical" mechanism can be represented as follows (Figure 38).

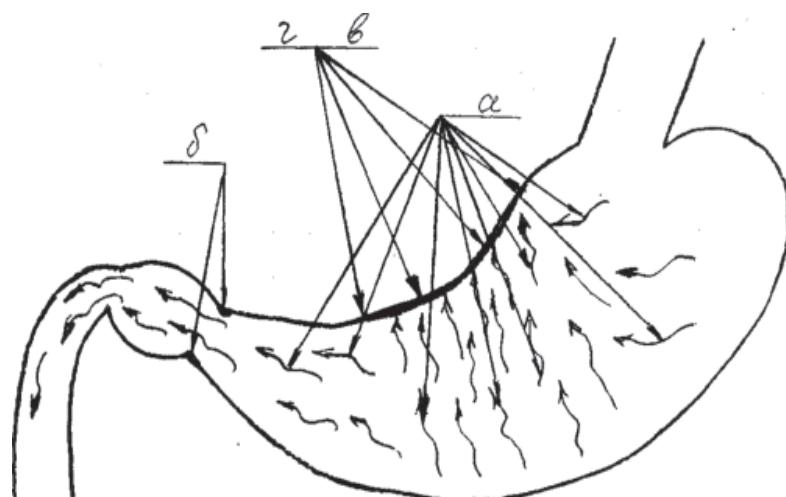


Figure 38. Mechanism of formation of mediogastric ulcerative lesions:
a) flow direction of "residual" of ammonia in the gastric cavity;
b) the pyloric sphincter in gaping condition;
c) the place of the maximum concentration of "residual" ammonia (middle third- upper third of the gastric body on the lesser curvature);
z) places of ulcerative lesions (tissues of middle third - the upper third of the body of the stomach on the lesser curvature).

In the absence or insufficient distinct manifestation of any elements that form the mechanisms that lead to erosive and ulcerative lesions of gastroduodenal zone, the activation of the pathological process can take place in the form of an exacerbation of chronic gastroduodenitis without destructive manifestations. Graphically, it looks as follows (Figure 39).

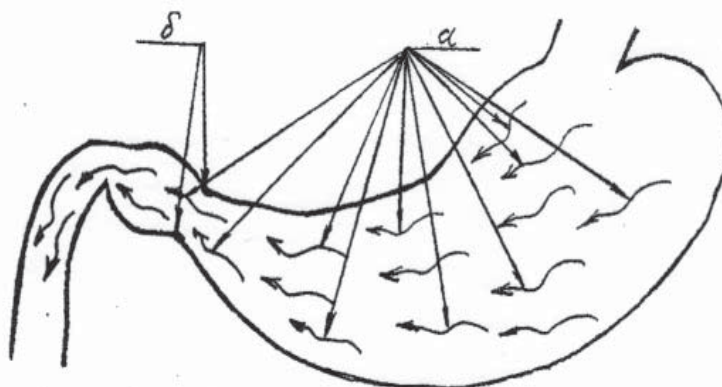


Figure 39. Flow directions of "residual" ammonia in patients with chronic helicobacteriosis without erosive and ulcerative lesions of gastroduodenal zone in the attack phase:
a) flow direction of "residual" ammonia in the gastric cavity;
b) pyloric sphincter in gaping state.

Our findings do not contradict, but complement the opinion of the majority of scientists who, recognizing nosological unity of GU and DU, believe that it is appropriate to distinguish at least 2 clinical pathogenetic forms of PU: pyloroduodenal and mediogastric. The reasons for this approach are significant differences in the clinical picture, the character of the motor-evacuation and secretory disorders of the stomach and duodenum, the directionality of vegetative and psycho-emotional disorders, and certain peculiarities of the mechanism of ulceration [112, 196, 398, 403]. Several studies

have shown that the pathogenetic importance of severity of aggression factors and state of gastric mucosa protective factors is unequal at different localization of ulcerous damages. Thus, according to most scientists, at the local level, in pyloroduodenal ulcers are essential factors of aggression, while in mediogastric - the weakening of protective mechanisms, first of all – mucous-bicarbonate barrier, the local immunological imbalance, microcirculatory failure, regenerative dysfunction, etc. [149, 403]. Here, our views diverge from the orthodox opinion, as in the charge of this opinion puts the role of acid-peptic factor, as a factor of aggression which fades out. From our point of view, changes in the level of acid-peptic factor and protection mechanisms is only a reflection of staging of type B chronic gastritis, which is characterized by topographical movement of HP as a key supplier of the etiological factor - RA, what was mentioned earlier in this chapter, and therefore, a change in the topography of a possible ulcerous defect [23].

Differences between the main clinical-pathogenic forms of PU touch on the functional state of the main divisions of the autonomic nervous system (ANS). In pyloroduodenal ulcer prevalent the tonicity of the parasympathetic division of the ANS, which affects the tonicity of the pyloric and cardiac sphincters (increases), strengthen gastrointestinal motility, there is an accelerated and spasmodic evacuation of its acidic content in the duodenum. At PU of mediogastric localization prevails sympathicotonia, the tonicity and the stomach motility is usually reduced, and the evacuation out of the stomach often decelerated. In addition, there is often insufficiency of pyloric closing function, which creates prerequisites for the development of duodenogastric reflux [350, 398, 403]. The data we obtained, fit into the above statements about pyloroduodenal ulcer, except the most important thing - that is evacuated from the stomach. According to the laws of aerodynamics, in the presence of the cavity, that conformed to the shape of stomach, fluid (gastric juice) and gas (gas mixture of the gastric cavity the gas), the gas will always be the first to be evacuated (according to our theory - concentrated ammonia) [380]. With regard mediogastric ulcers, then there is no contradictions at all: delayed evacuation, on the basis of the above statements of the formation of "vertical" mechanism, that helps to create a sustainable stream of ammonia with a greater curvature in the direction of the lesser curvature of the gastric body; "Dehiscence" of sphincter does not stipulate the increase of the concentration of ammonia at the outlet of the stomach.

The statements of ulceration mechanisms, outlined in our theory of the "caustic alkali exspuition", have been fully confirmed by the experimental work carried out by Japanese scientists at the Mongolian jerboas: HP infection was put animals on the gastric mucosa of animals, and later, ulcerative lesions developed there in vivo [167]. The fact, that for Japanese scientists became an artistic achievement, has quite logical explanation in terms of the mechanisms of ulceration in human-beings that were above-mentioned by us.

One of the main differences between jerboas and rats is locomotion: if rats move on 4 legs in a horizontal position of the body, then jerboa jumps, keeping the corpus more in a vertical position, i.e., repel with a help of powerful hind legs off the ground, at which time sharply straining muscles of the anterior abdominal wall. Sharp pressure on the stomach while moving, creates the effect of "bellows" that, in the presence of RA, as a sequence of HP infection presence on the gastric mucosa, in the stomach cavity of animals, leads to the ulceration.

In recent years a number of scientists have expressed the view of the possibility of the ulceration without HP infection (number of HP-negative PU patients according to various estimates ranging from 13 to 30%)[126, 127, 612, 619]. How true is this belief? And here, the theory of "caustic alkali exspuition" has "legs". But first it is necessary to exclude the possibility of errors during testing: first it is necessary to clarify how patients were tested, because the same method at the same stage of development of the pathological process may be true, while the other is not; secondly, it is necessary to test in parallel at least in two different ways; and thirdly, to test for topographic zones (especially the mucous membrane of lesser curvature of the gastric body in the middle-upper third, where is the "saving oasis" for HP - infection). Such requirements are in exact accordance with our patients' survey method [301].

When analyzing the results of the initial examination of 4 groups with erosive and ulcerative lesions of gastroduodenal zone of the digestive tract, in the active phase of the pathological process (150 of the 321 (46.7%)), it was found that HP in the active form was absent: in 1st topographic zone - in 22

cases (14.7%), once in 2 topographical areas - in 17 cases (11.3%), once in 3 topographical areas - in 13 cases (8.7%), once in 4 topographic areas in 11 cases (7.3%) (1 case - a group with ulcerative lesions of the duodenal bulb, 10 cases - in the group with erosive lesions of the duodenal bulb). However, a careful history-taking of patients with a total absence of reactive HP on gastric mucosa showed that per calendar day before the examination 3 patients out of 11 (27.3%) were taking pancreatic enzymes, 1 patient (9, 1%) - an antibiotic, 1 patient (9.1%) - bismuth preparation (Gastro-Norm), 1 patient (9.1%) – drug of PPI group (omez); 1 patient (9.1%) had a bilious vomiting the day before, in 3 patients (27.3%) while the endoscopic examination of the stomach cavity was detected the bile, i.e. in 10 patients out of 11 (90.9%) were present factors, facilitating to the rapid transition of HP infection active forms to inactive[12]. Only in the first case (9.1%), these factors were absent; however, the patient noted persistent opilation.

Whence in the gastric cavity, in this case, ammonia came from, resulting in damage to the mucous membrane? To answer this question, you need to understand one important thing: **HP is a leading supplier of ammonia, but not the only one!** As is known, a number of microorganisms that live in the upper respiratory tract and nasopharynx and in the large intestine have the urease activity [34]. In activation of the microorganisms that live in the nose and throat, and upper respiratory tract, there is an inflammatory process, and saturated with ammonia mucus is often swallowed by the patients, especially at night, as evidenced by our long-term experience and the results of our studies [5, 251, 252]. From our point of view, once the ammonia is in the stomach cavity with mucus under the influence of the human body temperature, it evaporates and concentrates in the cavity of the stomach, creating a cluster of gas, either alone or in conjunction with RA in the presence of HP (in 118 (36.8%) patients; aggravation in the stomach was preceded by flu and acute respiratory diseases, as well as exacerbation of chronic bronchitis, asthma and pneumonia).

The important role in this process plays such addiction, as smoking. Reasoning about correctness of the judgment is shown in Table 19.

Table 19. Main toxic and tumorigenic substances contained in vaporous phase of freshly generated tobacco smoke from the cigarette

Substance	Concentration in one cigarette	Biological effect
1. carbonic oxide	10 - 23 mg	T
2. acetic aldehyde	0,5 - 1,2 mg	CS
3. nitric oxide	50 - 600 mcg	T
4. hydrocyanic acid	150 - 300 mcg	CS, T
5. ammonia NH ₃	50 - 170 mcg	T
6. acrylic aldehyde	50 - 100 mcg	CS
7. benzol	20 - 50 mcg	HC
8. formaldehyde	5 - 100 mcg	C
9. 2-nitropropane	0,2 - 2,2 mcg	C
10. hydrazine	24 - 43 ng	C
11. urethan	20 - 38 ng	C
12. vinyl chloride	1,3 - 1,6 ng	HC

T - toxic substance; CS - cytotoxin substance; HC - human carcinogen; C - carcinogen.

As can be seen from the table the concentration of ammonia from the firing of the cigarettes took 5th place out of 12 ,formed in this, gases [156]. Subsequently forms the same insertion mechanism of

ammonia into the gastric cavity, as in diseases of the nasopharynx and respiratory system. Bronchial mucus in the stomach is bad by the fact that having an alkaline environment, it alkalizes the gastric material, creating a good background for the development of HP infection [3]. That's why, in our view, chance of new PU recurrences is always higher for smokers, than non-smokers.

The presence of inflammation in the large intestine may also affect the penetration of ammonia in the stomach. During adequate nutrition and normal operation of digestive tract in the intestines is always about 200 ml of various gases: N₂ (11-90%), O₂ (0-11%), CO₂ (3-54%), H₂S (0-30%), CH₄ (0-56%), NH₃ (1-8%), H₂ (1-10%). Just a day in the intestine produces about 20 liters of gas. Among them exudes from the intestine through the rectum from 200 to 2000 ml, and the major part - 90-98%, reabsorb through the intestinal wall into the blood and excreted through the lungs with the expired air [138]. It is known that inflammatory bowel disease is often accompanied by active flatulence - bloating, combined with diarrhea, and constipation [34, 430]. Taking cue from our education on behavior in public places (to release gases at strangers is a bad taste), patients have to restrain themselves. What to do with this gas mixture? As mentioned above, some of it, is absorbed by the intestines, partially comes naturally, and partly, from our point of view, because of the constant gas exchange between the stomach and intestines - in the cavity of the stomach, and then through the esophagus when you exhale - the external environment, that is often accompanied by bad breath. Thus, there is always a possibility that ammonia can reach high concentrations, once in the stomach in this way. Later in the process of formation of erosive and ulcerative lesions of gastroduodenal zone plug in the mechanisms outlined above.

Admission to the stomach cavity of ammonia could be, in our view, connected with liver and kidney disease (hepatogenous ulcers, ulceration in chronic renal insufficiency). In the pathology of the liver when it cannot neutralize the ammonia which is formed during the digestion, the gas, entering the arterial blood flow, then diffuses through the gastric mucosa into the lumen [321]. Further, the ulceration mechanism, described above, is possible. The same happens with chronic kidney pathology, when the function of waste evacuator takes the gastrointestinal mucosa. The stomach affects the most commonly and early in chronic renal insufficiency, which defines with the term "uremic gastropathy". With increased excretion of nitrogenous products in the stomach cavity, increases the formation of ammonia due to the urea splitting, which, in our view, forms an increased ammonia concentration in the gas mixture of the stomach cavity (RA, formed by the decomposition of urea by Helicobacter infection), with subsequent inclusion of ulceration mechanisms [283, 320, 383].

Thus, the ulceration mechanism though it forms, final outcome of it, in our view, is the damage to the mucous of gastroduodenal zone of the digestive tract with caustic alkali - concentrated ammonium hydroxide, which is confirmed by biochemical studies of necrotic masses: in the acute phase of ulcerous process reveals the prevalence of ammonia, while in ulcers, at the stage of primary healing process in necrosis already prevails the presence of hydrochloric acid. These results are expectable, as in the acute phase with a stored ulceration mechanism; damage area is constantly exposed to damaging factor. Subsequently, some of the elements of damage mechanism disappear for various reasons: changing the diameter of the pyloric sphincter (it becomes bigger and, therefore, density of ammonia and H₂O becomes less, so antispasmodic drugs should be assigned to a group of drugs of the pathogenetic action, and immediately incorporated into antiulcer therapy scheme) reduces speed and depth peristaltic waves, that is most often associated with the termination of stress situations in which was the patient, as well as the limitation of physical activity by the patient, and going to a sparing diet, which does not stimulate acid production, and therefore does not cause HP to ammonia production and accordingly, RA. The important role plays the development in 7-14 days from the beginning of the exacerbation of reactive pancreatitis, accompanied by duodenogastric reflux, reducing the concentration of the active form of HP, and accordingly, RA [36, 48]. An important role plays antihelicobacter therapy, which reduces the concentration of HP [34]. In this situation, the prevailing factor of influence on the damage zone becomes acid-peptic factor that begins to clean the wound from necrosis. With this, from our point of view, connects longer-term epithelialization of mediogastric ulcers: acid-peptic factor cleans the wound badly due to the low level of acidity, which results in low level of pepsin and gastricsin transition from the inactive to the active form [207, 350].

In the control group, where during the examination has been detected the type B chronic gastritis, there was no comprehensive set of elements for the formation of a "horizontal" and "vertical"

mechanism of ulceration, although in 12 patients (15%) ulcers were previously found in the duodenal bulb, in 2 (2.5%) – ulcers of the output sector of the stomach, and in 2 patients (2.5%) of the control group, who had previously identified mediogastric ulcers, preserved topography of HP settling on the mucous, but changed job profile (decreased physical activity); in 7 patients (8.8%) were determined erosive lesions duodenal bulb. In the group with ulcerative lesions of the stomach outlet in 13 patients (30.2%) also determined ulcerative lesions of the duodenum. In 3 patients with mediogastric ulcers previously determined ulcer of the duodenum, but at an earlier age (an average of $32,0 \pm 7,18$ years).

Thus, summarizing all the above, we can reasonably put forward a new thesis in understanding the etiology and pathogenesis of ulcer formation: "Without ammonia, there is no ulcer", which is the "cornerstone" of created new theory – theory of alkali damage of the gastric mucosa of and duodenum with ammonium hydroxide ("caustic alkali exspuition"), the essence of which was described above.

1.4.5 Reasons, that conducting the formation of ulceration mechanisms

Analyzing the causes preceding the exacerbation of chronic gastritis type B and participating in the formation of erosive and ulcerative lesions of the gastrointestinal tract of various departments, the following main terms can be emphasized:

1) Transferred acute respiratory diseases or exacerbation of chronic diseases of respiratory system and throat. In this situation, a large number of bronchial mucus is swallowed by the patient, which leads to alkalization of gastric materials. In addition, due to the activation of viral and bacterial flora of respiratory system and throat deteriorates the general immunity of the body. Reducing the level of immunity and shift the pH environment of the stomach to the alkaline side create the most favorable conditions for the breeding of HP infection and contribute to the formation of ulceration mechanism, which is confirmed by our studies [219, 251, 252].

2) Acute illness or exacerbation of chronic diseases of other organs and systems. Any disease leads to immunological changes in the organism. Often, in the treatment of various diseases uses antibacterial drugs, NSAIDs, and in particular pathologies- hormones. These drugs can have a negative impact on the gastric mucosa, causing its injury, worsening the regeneration of existing damage, but also cause immunosuppression [74, 104, 186, 187, 333, 334, 450, 452, 465, 479, 481, 491, 561, 565, 572, 574, 596, 616, 633, 634, 635, 639, 646, 650, 653]. This creates favorable conditions for the breeding of HP infection and promotes the formation of ulceration mechanism, which is confirmed by our research.

3) Psycho-emotional breakdowns (home, work-related or simultaneous, related to surgery, chronic nervous breakdown related to the illness or death of loved ones). Any psycho-emotional stress - it is, in many respects, immunosuppression, long-term psycho-emotional stress is a functional exhaustion of the parietal cells [34, 105, 242.265, 350, 360]. The combination of both the negative effects of stress creates the most comfortable conditions for the breeding of HP infection and promotes the formation of ulceration mechanism, which is confirmed by our research. In this case, the probability of the formation of intracellular stores as the basis for future exacerbation of the pathological process that leads to deterioration of eradication.

4) Disadaptation (a trip to another city, region, country). Change of climate, water, food, social environment requires adaptation to the new conditions.

Environmental factors that affect the human immune reactivity are divided into 3 main groups: abiotic, biotic and anthropogenic - socioeconomic. These factors can damage the immune system directly or indirectly. Direct action carried out directly on lymphoid organs, phagocytosed cell, lymphocytes and their derivatives. It is manifested by decreased activity of phagocytosis, hyperplasia of the lymphoid organs, decrease in number of immune cells, a decrease of intensity of immunoblasts and plasma cells formation, a violation of cooperative T, B and A cells, decrease synthesis of Ig. Indirect action effects through chromosomal damage, the effect on the endocrine and nervous system, regulating the immune response [361].

Due to exposure of cold climate adaptive restructuring of the gastric mucosa function is characterized by a decrease of its secretory activity, due to the energy savings for satisfying the requirements of the body in enhance heat production. Transition of the gastric mucosa to a more

economical way of activity is achieved by reducing the work of the proton pump (as a demanding mechanism) on the way of regulating the activity of gastric enzymes, as well as synthesizing and isolating enzymes, active at higher pH values, which, along with the deterioration of the immune system, improves the conditions for the growth of HP infection, which is confirmed by our studies [11, 91, 200, 372].

5) Contact with nitro-dyes, varnish, etc. (steam inhalation). Inhalation of toxic substances leads to the fact that, avoiding hepatic barrier, these substances cause intoxication, and thus adversely affect the immunity, creating comfortable conditions for the growth of HP infection, promoting the formation of ulceration mechanism, which is confirmed by our studies [83, 143, 338, 374].

6) The error in the diet (spicy, salty, fried, sour, fatty foods, foods with nitrates: small radishes, cucumbers, tomatoes, watermelons, melons, fermented, pickled, canned vegetables, alcohol). Admission of spicy, salt, fried food stimulates the secretion of gastric acid and acid food lowers the pH of the stomach, and therefore provoke the production of ammonia; fatty foods increases the load on the pancreas that can cause pancreatitis and aggravate the disease state, which is confirmed by our studies [129, 393].

Nitrates, trapped in the body with food and water, metabolize in a much more toxic nitrites, which are regarded as precursors of carcinogens -N-nitroso compounds, which provokes the poisoning of the body, and such nitrated manure as urea decomposes by HP infection to ammonia that strengthens and enhances intoxication, from our point of view, the level of etiological factor of ulceration - RA, which is confirmed by our studies [6, 7, 83, 143, 192, 395, 406].

Application in food fermented, pickled, canned vegetables can also have an impact on the disease process. While pickling, marinating, canning of vegetables identify a significant (60-70%) reduction in the level of nitrates in vegetables, which is connected with their transition into brine, and with the flow of the recovery processes in the chain of $\text{NO}_3 - \text{NO}_2 - \text{NH}_3$ [406]. Furthermore, while conservation acetic and acetylsalicylic acids are often used. With the use of such vegetables ammonia enters the stomach from the brine, and the acidification of the stomach environment with the aforementioned acids, triggers HP on production of ammonia, which increases the level of RA, and thus creates the preconditions for erosive and ulcerative lesions of gastroduodenal zone, which is confirmed by our researches.

Admission of alcohol, namely beer, stimulates the secretion of gastric juice, which results in a response by the HP infection by way of ammonia production, as evidenced by our studies [291].

7) Physical exercise. Physical activity is accompanied by a contraction of anterior abdominal wall muscles and increase of intra-abdominal pressure and, consequently, an increase of intragastric pressure and the formation of the "bellows" effect - an element of the ulceration mechanism, which is confirmed by our researches.

8) The pressure on the stomach from the outside: conducting massages (deep back massage), striking in the area of the anterior abdominal wall. Massage, as well as a strike in the anterior abdominal wall, provokes the effect of "bellows", but without the participation of the patient, but the consequences will be the same as during physical activity, as evidenced by our researches.

9) Bad habits (smoking). Smoking, as was stated above, is the supplier of the etiological factor – ammonia, and bronchial mucus that is swallowed by the smoking patient, alkaline stomach environment, improving conditions for the habitat and breeding of HP infection, as evidenced by our researches [3, 156].

CHAPTER 2

CLINICAL ISSUES OF PEPTIC ULCER MANIFESTATION

(P.149-156)

Clinical manifestations of peptic ulcer are multifaceted: their variability is related to age, sex, general condition of organism of the patient, limitation of disease, frequency of exacerbation, localization of the ulcer defect, the presence of complications.

1)Pain. The leading symptom of peptic ulcer disease is pain, which is characterized by periodicity within 24 hours, seasonality (spring-autumn period), the presence of bright gaps - absence of relapses for several years (Troitskii triad) [133].

The ulcer pain, predominantly, are localized in epigastria, but depending on the localization, ulcer may focus on different areas. Gastric ulcer patients feel them predominantly in the centre or on the left side of the median line, with the prepyloric, pyloric and duodenal ulcer – in the pyloroduodenal zone itself [261].

For high stomach ulcers (forestomach) early pain is specific. It occurs immediately after eating, especially spicy and hot; sometimes nagging, oppressive, arching pain which localizes under the spur or in the left hypochondrium. During the ulcer localization in areas of the body and the bottom of the stomach (mediogastric ulcers) pain occurs within 20-30 minutes after eating, occasionally at night. Pain reaches special intensity within the localization of ulcer in the pyloric canal, it occurs through 40 min-1 hour after taking meal. While localization of ulcer in duodenal bulb ulcer or stomach division antrum pain often occurs on an empty stomach (a hungry ache), at night and after 1.5-2 hours after taking meal (late pain) [133].

However, neither localization nor temporal rhythm of pain is not for peptic ulcer's disease pathognomonic. For example, late pains occur in chronic pancreatitis, chronic enteritis, and mostly night - in pancreatic cancer. There is only one exception to this - hungry pain, i.e. calming down immediately after eating. It is exactly they who are really pathognomonic for ulcer, because neither is not observed in any other disease. Irradiation is not always observed and more peculiar penetrating ulcer. Localization has also a value. For example, the posterior wall ulcer duodenal bulb radiating primarily in the right half of the lumbar region, subcardial ulcers is in the heart area, and located at the corner of stomach-upper.

Ulcer pain mechanism has not yet been fully revealed. According to some researchers, it is possible to identify two groups of factors. First goes down to the fact that pains are eased or eliminated by impacts which reduce the degree of local irritation of ulcer by the hydrochloric acid, which is confirmed by pain relief by taking meal; drugs with neutralizing or inhibitory effect on hydrochloric acid and even dilution of it by drinking water. The second group of factors is a motor response on ulcer's irritation, which partly has defensive focus. So, duodenal ulcer is often accompanied by pylorospasm, which in some way block the way to it the acid content of the stomach, and also hypermobility of the duodenum, whereby release of acid in the lower sections of the duodenum is fastened. These seemingly suitable protective reaction may nevertheless serve as a second factor in the genesis of ulcer pain. Reinforced reduction and spasms of gastroduodenal muscles, coinciding with the area damaged can cause spastic character. The resulting from compression of the vessels spasmed muscle tissue ischemia can matter. Finally, you cannot withdraw from the account and increase due to spasms intra-luminal pressures in the stomach or duodenum, which leads to irritation of the baroreceptors in the field of pathologically changed tissues. The basis to say this is plain an analgetic effect of antispasmodic agents, especially when applying the holinolytic activity [261]. From our point of view, on the basis of the theory of "caustic alkali sputum" irritation factor of the ulcer defect is really present, but it is not how much muriatic acid, which during ulcer formation may have different, even very low concentrations of [121] as ammonium hydroxide is a caustic alkali. As it was mentioned above, alkalis, unlike acids, by melting and liquefying the slimy and submucosal layer easily penetrate deep into tissues, until the muscular layer, irritating meissner's and auerbach's plexuses, but such ulcers should have greater depth. According to our observations in the vast majority of cases the pain

is due to the presence of reactive pancreatitis, which is 7-14 days from the activation of the pathological process in the stomach and occurs due to compression of the fatterpachin corpuscula, which are found in the tissues of the pancreas, in the development of edema tissue itself, as well as with increasing introduct pressure [15, 36]. Proof of this is that the majority of patients with painful syndrome during the endoscopy examination of ulcer defects, which are in the initial stages, incomplete or complete epithelization. As far as pain control while lowering the concentration of hydrochloric acid in any way here it is necessary to consider the reactions of *H. pylori* to changes in pH medium of its habitat: while raising pH medium *Helicobacter* infection will less produce ammonia, and when applying Protonenpumpenhemmer will turn in the inactive form, which generally produces no ammonia, which further leads to a decrease in the concentration of ammonium hydroxide and reduce irritating effect on ulcers [12]. The second group of factors determined, from our point of view, a violation of antroduodenal coordination stressor situation that increases the concentration of ammonium hydroxide in spasm zone of gatekeeper [60]. The use of antispasmodic agents reduces tone pyloric bagasse and increases the diameter of the pyloric canal, which leads to redice of ammonium hydroxide concentration, hence, and its irritation action on the ulcerative defect.

2)Heartburn. According to various authors, heartburn is the most common and early symptom of peptic ulcer disease. Heartburn can be preceded by the formation of ulcers, sometimes ahead of the appearance of pain for several years. With their emergency heartburn usually subsides, and because of that some clinicians see in it peculiar equivalent of pain. Later, hunger, night heartburns are distinguished. Mechanism of heartburn occurrence is connected not only with high acidity of gastric juice, but also with upper gastroesophageal reflux disease, which is caused by a decrease in cardiac tone of pulp, so heartburn, even painful, may be with low acidity gastric juice [133, 261].

On the basis of our data, the emergency of the symptom of heartburn does not depend on the level of acidity of gastric juice but from concentration and the resettlement area of HP infection on gastric mucosa and, accordingly, the concentration of RA that requires pH measuring by a sick person before prescribing treatment [42, 226, 243, 350]. Considering the fact that in the area of cardiac stomach zone and cardiac pulp also formed the Bord nozzle, therefore, here too exist increased concentration of ammonia (Cardial pulp is a higher pressure zone (approximately 10.5 mmHg) than in the bottom of the stomach) [85, 409]. After cardiac orifice goes the expansion of the lumen of the esophagus that, from our point of view, makes **erosive-ulcerous** defeat of mucous membrane of esophagus precisely in its lower third-in the zone of greatest concentration of ammonia, and therefore, ammonium hydroxide [71, 436]. Regardless of whether or not this mucous of the zone damage will be formed or not, the increased concentration of hydroxyl ammonium will assist the irritating effect on the nerve endings of mucous membrane, which has a structure of pavement multilayer epithelium and nerve endings are on its surface, which results from our point of view, a sense of stinging [71, 569]. As a fact the same mechanism is formed as with D of PU) pyloric and prepyloric ulcers, but only in the opposite direction, along with it gas, according to the laws of aerodynamics, always will break first in relation to gastric juice [135, 380, 409]. This interpretation of the symptom of heartburn, from our point of view, will force researchers to look differently on the etiology and pathogenesis of diseases such as gastroesophageal reflux disease (GERD).

Evidence of this is our research data: 25 patients (31.3%) groups with lesions of bulbs 12 duodenal ulcer have never experienced the feeling of heartburn, and 25 (31.3%) heartburn disappears in average 3.86 ± 0.87 months before the exacerbation; 15 patients (18.8%) of the group with ulcerous lesions of bulbs 12 duodenal ulcer have never experienced the feeling of heartburn and 65 (81.2%) heartburn disappears in average 3.97 ± 1.57 months before the exacerbation; patients (27.9%) groups with ulcerous lesions of stomach output never experienced the feeling of heartburn, and 19 (44.2%) heartburn disappear in an average 3.75 ± 0.74 months before the exacerbation; 11 patients (30.6%) ulcerous lesions of the mediogastral group of the stomach have never experienced the feeling of heartburn, and 15 (41.7%) heartburn disappear for an average of 9.60 ± 6.18 months before the exacerbation; 38 patients (46.3%) groups without erosive-ulcerous defeats of gastroduodenal zones of **digestive tract** never experienced the feeling of heartburn, and 16 (19.5%) heartburn disappear in average 3.22 ± 0.82 months before the aggravation that gave us the right to call this phenomenon as a

symptom of "lost," which can be interpreted as the beginning of formation of a horizontal mechanism erosive-ulcerative losses of pilorobulbic zone.

3)Belching. Belching belongs to nonspecific symptoms and is found almost in all gastroduodenal diseases and even in their absence. When localizing an ulcer in the duodenum burp belching does not belong to the usual complaints. It occurs more often in patients with mediogastric ulcer and can be both empty and acidic [261].

This symptom, from our point of view, also reflects the protective property of **digestive tract** as for the regulation of gastric cavity pressure and not only. When excess gas due to how life HP (NH₃, H₂S, CO₂), and violation of the normal digestion not only in the stomach, but also in the gut (especially for stool retention), gas mixture breaks through the Cardial pulp into the esophagus, however, broader disclosure of Cardia, that is not accompanied by increases in the concentration of ammonia in this place, and therefore lack of heartburn. It reaffirms the long-standing way of dealing with heartburn – patients use soda. Contact of soda with hydrochloric acid gastric juice neutralizing reaction occurs, which is accompanied by large amounts of CO₂. The increase in the concentration of CO₂ in the gas mixture cavity of the stomach dramatically increases the pressure that leads to wider disclosure of cardiac orifice and rapid discharge of gas mixture, which includes ammonia, an external sphere that at the time reduces the concentration of ammonia. Presence in the stomach cavity gas mixture gas with an unpleasant smell (ammonia, hydrogen sulfide, etc.) in various proportions forms the olfactory peculiarities of belching.

4)Nausea, vomiting. These symptoms, according to different authors, are associated with painful syndrome: vomiting usually occurs at an altitude of pain and brings the patient, which is often causes it itself, relief. Vomiting is often a sign of evacuative - motor function of stomach while pyloric stenosis; in such cases, the vomit contains the remains of food eaten before. A symptom of bleeding is bloody vomiting - hematemesis [133].

In the absence of stenosis, nausea and vomiting, from our point of view, could be the extent of such reasons:

a) with formed reactive pancreatitis, biliary reflux is often noticed. Bile, irritating the gastric mucosa, provokes nausea, which may end up with vomiting [36, 106]; b) when inhaling air flow coming through the throat into the trachea, and then into the lungs, in the area of the exit of the esophagus into the throat, according to the laws of aerohydrodynamics, a negative pressure zone, which causes drawing it effect. When this gas mixture, which includes ammonia, from the stomach through the esophagus, bypassing the hepatic barrier gets into the lungs (bronchial-pulmonary system plays a role in this case ejector-"Jet pump"), and then-in the total blood flow [135]. In view of the fact that ammonia is neurotoxic, increasing its concentration in the blood has a negative impact on the central nervous system that causes the central nausea and vomiting [82, 321]. This conclusion is confirmed by our research: after eradication, where the level of RA declined, nausea and vomiting in patients stopped [52].

b) considering the fact that ammonia has a strong irritant effect, possible strong irritation of the gastric mucosa with subsequent nausea and vomiting, which brings relief due to a temporary decrease in the concentration of ammonia in the stomach cavity [145].

5)Sense of gravity in epigastria. This phenomenon is, according to our view, is a manifestation of the high pressure caused by intragastric increased concentration of gases, metabolic products of HP infection, as well as adjustment disorder (decrease or absence of relaxation fundic division of stomach) [60]. This situation explains the phenomenon and as a symptom of "fast (early) saturation" (determination of feeling of saturation with a small amount of food).

While accomodation disorder the stomach will be content with practically constant volume. Pressure in the cavity fundic division of stomach with pathology will be total: 65 cm water pillar (the maximum normal) + pressure on the lining of the stomach due to gas mixture that pathology would more concentrated because of the vital functions of HP infection (CO₂, H₂S, OA). According to the law of Archimedes is a small amount of food (volume one of food bolus -5-15 cm³, patients noted the onset of a symptom of early saturation after 3-7 sips, i.e. on average received into the stomach 30-70 cm³), displacing the same volume of gas mixture of the stomach, increases the concentration of gas

and therefore increases pressure gas mixture on the lining of the stomach. Stretching of the stomach walls mimics the phenomenon of saturation [207, 343]. This is confirmed by the data of our study.

6) Flatulence Abdominal distension has, from our point of view, a double mechanism:

a) gas mixture (NH₃, H₂S, CO₂) producing HP peristaltic wave moves from the stomach to the intestine;

b) with CG (chronic gastritis) of type B disbiosis of the *intestines is* very often formed, from our point of view, as the atmosphere in the intestines, which creates a reset from the stomach and gas mixture which is more suitable for pathogenic intestinal flora and violation of HP infection started the cascade of proteolytic digestion in the stomach [34, 138, 401]. Especially flatulence is expressed with III degree of disbiosis, i.e. contamination of the small intestine, where bacteria from the colon, find a significant number of non fully splitted substrates of meal, which, in turn, contribute to increased reproduction gas-forming bacteria. If the intestinal contents prevail carbohydrates then develops appropriate breeding, fermentation bacteria and formation of CO₂. If the quota has been increased in the diet of proteins, it contributes to the development of putrefactive bacteria which produce NH₃, H₂S, indole, skatole [138].

7) Abnormality of stool (constipation, diarrhea). Violation of the stool is the result of disbiosis, which develops due to reasons stated above [34, 138, 401] as well as in violation of exocrinous function of the pancreas while jet pancreatitis, which is actually always formed while *exacerbation of peptic ulcer* [36].

8) Changes of palatability of oral cavity (dryness, bitterness, metal flavour, hypersalivacion). Dry, taste of metal, from our point of view, is a manifestation of the *caustic* of toxic gas mixtures, which includes ammonia, on the mucosa of the oral cavity and its receptors, as well as one of the manifestations of intoxication [83, 145]. A sense of bitterness in the morning is due to the fact that in horizontal position with duodenogastric reflux it is easier to get into the stomach and from there into the esophagus and pharynx, which results the formation of this feeling. Hypersalivation is caused from our point of view:

a) irritating effect on salivary glands of mouth of ammonia, which is a part of the gas mixture which enters the oral cavity when you **exhale** from the stomach through the esophagus [145];

b) at hipoacidity, from our point of view, as the compensatory delivery mechanism of H⁺ in the stomach cavity, considering the fact that normally it is produced and ingested up to 2000 ml of saliva, and when hypersalivation - even more, with this pH of saliva is shifting for more acidic side. At the same time patients after eating feeling acid in your mouth [59, 207];

c) with the number of poisonings, when the mechanism of origin of hypersalivation may not be associated with a lesion of digestive organs directly (e.g., nitrogen fertilizer components) [374].

9) Furred tongue (yellow, grey, white fur). From our point of view, yellow fur is due to mucosal tongue contact with bile; grey and white fur - contact tongue mucous with toxic gas mixture, which is concentrated in the mouth, getting there when you exhale from the stomach through the esophagus [83].

10) Common manifestations of intoxication (weakness, lethargy, fatigue, etc.). Such manifestaions are due to intoxication, associated with increases in the concentrations of OA, which, according the laws of air hydrodynamics, when inspiration is drawn from the stomach through the esophagus into the lungs [135]. Chemically active and very volatile ammonia impacts on lungs structure (alveolar membrane), breaking their barrier and transport function. Large area suction surface of lungs (more than 100 m²), the small thickness of the alveolar membranes (1 μm) and intense blood flow ensures rapid development of toxicity. OA, absorbing through the lungs, gets into the small circle of blood circulation, and then bypassing the barriers of the liver in a large circle to the organs and tissues [145]. Toxicity of RA, the incoming in gas mixture in cavity of the stomach, exploding more and so that the gas mixture is heated up to "constantly" + 36,6°C -temperature of the human body-and has high humidity [145]. In addition, the toxicity of ammonia also affects the Ph: the pH sphere above, the toxic effect of ammonia is stronger, which is especially important in the thinking of the development level of HCG type in [143].

CHAPTER 6

COMPLETE PHYSICAL EXAMINATION AS A METHOD OF DETERMINATION OF DEVELOPMENT SPECIFICITY OF THE PATHOLOGICAL PROCESSES.

(P.189-195)

6.1 Methodology of a complete physical examination of patients with peptic ulcer.

The choice of drugs and rational schemes, which are necessary to treat patients, depends on the stage and phase of development of the underlying disease - type B chronic gastritis, one manifestation of which is the erosive and ulcerative lesions of gastroduodenal zone of various localization. Unfortunately, imposed worldwide generic treatment concept that can be traced in all 3 of the Maastricht Consensuses deprives doctors of incapability of rational and independent thought and trims his work down to the automatic assignment schemes excluding the patient's personality that resembles a child's game - "guessing". What are the recommendations for the use of schemes: first, apply the scheme of the first line, and then, if there isn't an effect - a scheme of the second line, and then, if the scheme does not give effect, to apply an individual approach. Logically, treatment should begin immediately; taking into account the patient's personality that will make antiulcer therapy rational and qualitative, as well as reduce the number of side effects from the drugs. From our point of view, the Maastricht consensuses recommendations are designed to increase sales of drugs produced by firms - sponsors, and, above all, the PPI, which are basic in all schemes. However, the use of PPI without taking into account the individuality of stages of the pathological process in the patient may result from the game - "guessing" in another game, known as the "Russian Roulette", i.e. lead to a situation where the prescription of the PPI will be a decisive factor in the development oncological disease in the stomach and cause the death of the patient. [24] The most amazing thing is that the Third Maastricht Consensuses itself indicates the possibility of the development of atrophy (precancerous state) and malignification of gastric mucosa during inhibition of gastric secretion, thus proving its bankruptcy of ability [642]. In this situation, only the individual approach to the patient will give an opportunity to solve this difficult problem, and this approach is based on a preliminary patient's complete physical examination, which we have developed and introduced into practice 5 years ago (during this time comprehensively examined more than 4000 patients).

Comprehensive survey involves the performance of incremental intragastric pH measurement, esophagogastroduodenoscopy and biopsy sampling for histological studies and double testing on HP infection (urease test and microscopic examination by Giemsa stained touch smear) according to our procedure [301]. The survey is conducted in the following order: first carries out a pH measurement, and then - endoscopy biopsy for histological examination of mucosa and testing on HP infection. Testing is done in the morning on an empty stomach, 12-14 hours after the last meal.

1. To study the level of gastric acidity in the different phases and stages of type B chronic gastritis development such a device is used - an acidity indicator of the stomach (AIS) - 2. AIS allows, with a help of the original pH- microsampling PE-pH-2 (diameter 2.0 mm), portable high-speed microelectronic devices, to detect intraluminal pH of the digestive tract, such as intragastric pH - the acidity of the stomach (Fig. 43).

Intragastric pH measurement (basal topographic pH measurement for gastric extension) carried out by the method of Chernobrovogo V.N. (1989.), which is a consequence of the original approach to the study of acid-forming function of the stomach (Copyright certificate №1388800 USSR copyright certificate number 1399677 USSR) [407].

Intragastric pH monitoring using AIS is held in the morning (8.00 - 9.00), fasting, in 12-14 hours after the last meal. For proper evaluation of the basal (baseline) stomach acidity excluded prior medicine admission was excluded, and for 3 - 4 hours to the study - smoking and drinking liquids. On the experimental subject's skin of the back surface of the lower third of the forearm a drape, moistened with a saturated solution of potassium chloride, is laid, and on it outer auxiliary silver chloride electrode, which is fixed to the arm with an elastic bandage. The experimental subject holds between the teeth blocking device - rubber-dam, through a channel of which, saturated in warm water, pH- microsampling (the position of the experimental subject - in the seating position) at a depth of 40 cm from the front edge of incisors, that ensures the presence of the first (distal) pH olive area near the esophagogastric

junction and has registered, as a rule, neutral or weakly alkaline environment (pH 7.0 - 7.5), less often weak acid medium (pH 5.0 - 6.9). After registration of pH at a depth of 40 cm probe is inserted deep on 20 cm, defining pH every 1 cm (15 - 20 sec), which provides in average human height the penetration of pH olive on entire length of the stomach from its input to the output. Catheterization ends with the slow stretching of the catheter with the registration of pH every 1 cm to the original depth of insertion of the probe - 40 cm.

While registering and evaluating the results of express-method pH measurement of the amount of pH of the stomach extension, are allocated in accordance with the six - 0 - 5 (in ascending order of acid-forming function) - functional pH interval (pH FI) basal gastric pH-grams: pH 7.0 -7.5 (anacidity-FI pH 0); pH 3,6-6,9 (apparent hypoacidity - FI pH1); pH 2.3-3.5 (hypoacidity moderate - FI pH = 2); pH 1.6-2.2 (normal acidity - FI pH = 3); pH 1.3-1.5 (hyperacidity moderate - FI pH 4); pH 0.9-1.2 (apparent hyperacidity - FI, pH 5).

The formulation of the conclusion is based on taking into account the data of the table, at that stand out: 1) the FI of the pH, coincided with the experimental subject's maximum level of acidity; 2) the FI of the pH, which accounts for the largest number of measuring points of pH on the stomach extension - the dominant FI pH;

3) FI pH on the number of points is divided:

up to 5 points - minimum;

from 6 to 10 points - selective;

from 11 to 15 points - absolute;

16 to 19 points - subtotal;

20 points - total.

2. After carrying out incremental intragastric pH measurement to determine the state of organs of the upper section of the digestive tract, esophagogastroduodenoscopy (EGD) by the standard technique is carried out [342].

3. During the EGD performs biopsic material sampling for histological examination of mucosa and double testing on HP infection from 5 topographic zones of upper gastrointestinal tract: from the duodenum bulbs, from the middle third of the antrum (65-70 cm from the incisors) and the body of the stomach (50-55 cm from the incisors) for large and lesser curvature, at that the distance between biopsy samples from each topographic zone must not exceed 0.5 cm. Sampling locations are recorded in the log of comprehensive survey on the distance from the incisors in reference to a digital layout of endoscope sheathing. Biopsic material sampling is produced from the edge of ulcers and erosions in the localization of them in the duodenum bulb, and in their absence - from the site of the inflammation of the same bulb. In each topographic zone first biopsy sample is taken at the site of apparent inflammation, the other one - under visual control on the place of the first biopsy material sampling (total - 3-4 biopsy samples from each topographic zone). 1-2 biopsy samples from each zone were used for histological examination of mucosa, 1 biopsy sample - for urease test, 1 biopsy sample - for the preparation and microscopy of Giemsa stained touch smears [301]. In the presence of ulcers in the stomach, regardless of localization, biopsy material sampling increases from 5 to 10 biopsies from the edges of ulcerous defects. Histological examination of biopsy materials is carried out by common method, taking into account the recent classification [34, 111].

4. Test on an urease activity is carried out by our modification, which increases the quality of the test with relating to common technique. A solution for the test is made daily: to 10.0 ml of distilled water, which is in the centrifuge tube, is added 8-10 particles of the indicator (phenol red) and 0.01 g of tetracycline hydrochloride to suppress the bacterial flora, except HP, and the solution is thoroughly mixed and put in a thermostat at + 37 ° C. Prior to testing in 5 centrifuge tubes are placed 15 mg of laboratory urea and 0.5 ml of basic solution is added. In the tubes with a resulting solution are added the biopsy samples of the mucosa from each topographical zones and the tubes are incubated in a thermostat at + 37° C for 24 hours. The test is considered to be positive when the color of the solution changes from chrome primordial to light magenta. Regarding the time of occurrence of a positive response, concentration of the active form of HP infection in the mucous is calculated: from 1 to 10 minutes - (++++); from 11 to 45 minutes - (+++); from 46 minutes to 1 hour and 30 minutes - (++) ; 1h from 31 minutes to 24 hours - (+); absence of reaction for 24 hours - (-).

Microscopy of stained touch smears is carried out by our modification that improves the quality of smears relatively to the common method, especially in determining the presence of HP mitosis. Preparation of touch smears is carried out in the following manner: biopsy sample of mucosa is smeared on biopsy slide, pretreated with 96% ethanol, and dried in a thermostat at + 37° C for 1 hour. Then, touch smear is colored with aqueous-alcoholic solution (1: 1) of methylene blue for 0.5 - 1 minute, thoroughly rinsed with distilled water and dried in a thermostat for 1 hour, and then, microscopy of smears, using immersion system, is held. Calculation of the concentration of both active and HP coccoid forms in the field of view is carried out according to generally accepted criteria: 1 to 20 - (); from 21 to 50 - (++) ; from 51 to 100 - (+++) ; of 101 or more - (++++).

6.2 Evaluation of the data, obtained during the comprehensive survey of patients with peptic ulcer.

Carrying out incremental intragastric pH measurement allows to estimate the condition of the gastric mucosa secretory function and creating a primary picture of the possible stage of development of the pathological process. [20]

Implementation of EGD allows to estimate the condition of the upper abdomen; reveal the presence of erosive and ulcerative lesions and masses, as well as to diagnose the sequelae of ulcerative process - bleeding, perforation, penetration and stenosis [342]. Furthermore, during the EGD in the cavity of the stomach availability factors are determined, which affect the readings of intragastric pH measurement - the presence of duodenogastric reflux and bronchial mucus (bronchial mucus and bile alkalize environment in the stomach cavity) [4, 5, 48]. Histological studies of 5 topographical zones increase the percentage of detection of mucosal changes, as atrophy, metaplasia and dysplasia, and gastric cancer in an early form [301].

During the dual testing for Hp infection both tests are carried out in parallel, not only for the purpose of determining the presence, concentration, form of HP and its settlement on the topography of the mucous, but also to determine the location of HP infection - the extracellular or intracellular. Our method of determining the intracellular location of HP is based on the properties of HP knowledge - namely, its ability to penetrate the parietal cell and block the synthesis of HCl [34]. When bacteria are in the cell, it does not react with the reagent during the urease test, as between bacteria and the reagent is wall of parietal cell, which leads to a change in time of the reaction: if the HP-infection is completely extracellular, a positive response of urease test coincides with the true concentration of HP, which is confirmed by microscopy of touch smears; If HP is partially in the cell, and partly - outside of the cell, the onset of a positive urease test reaction will be different from the true concentration of HP - it will be more; in a situation where the entire HP-infection is in the parietal cells, the urease test will be negative in 24 hours. One of the manifestations of HP intracellular location is hypoachlorhydria, has been noted during the incremental pH measurement.

Urease test in 24 hours will be negative if there are HP inactive forms, which are detected in microscopy of touch smears, but hypoachlorhydria is not detected. Hypoachlorhydria, in the absence of intracellular HP, is detected in the case of complete atrophy of the parietal cell, which is confirmed by histological studies.

Thus, when carrying out a comprehensive survey, the most important thing is achieved - getting an individual picture of the pathological process, what in the future will determine an individual choice of drugs and treatment regimens.

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