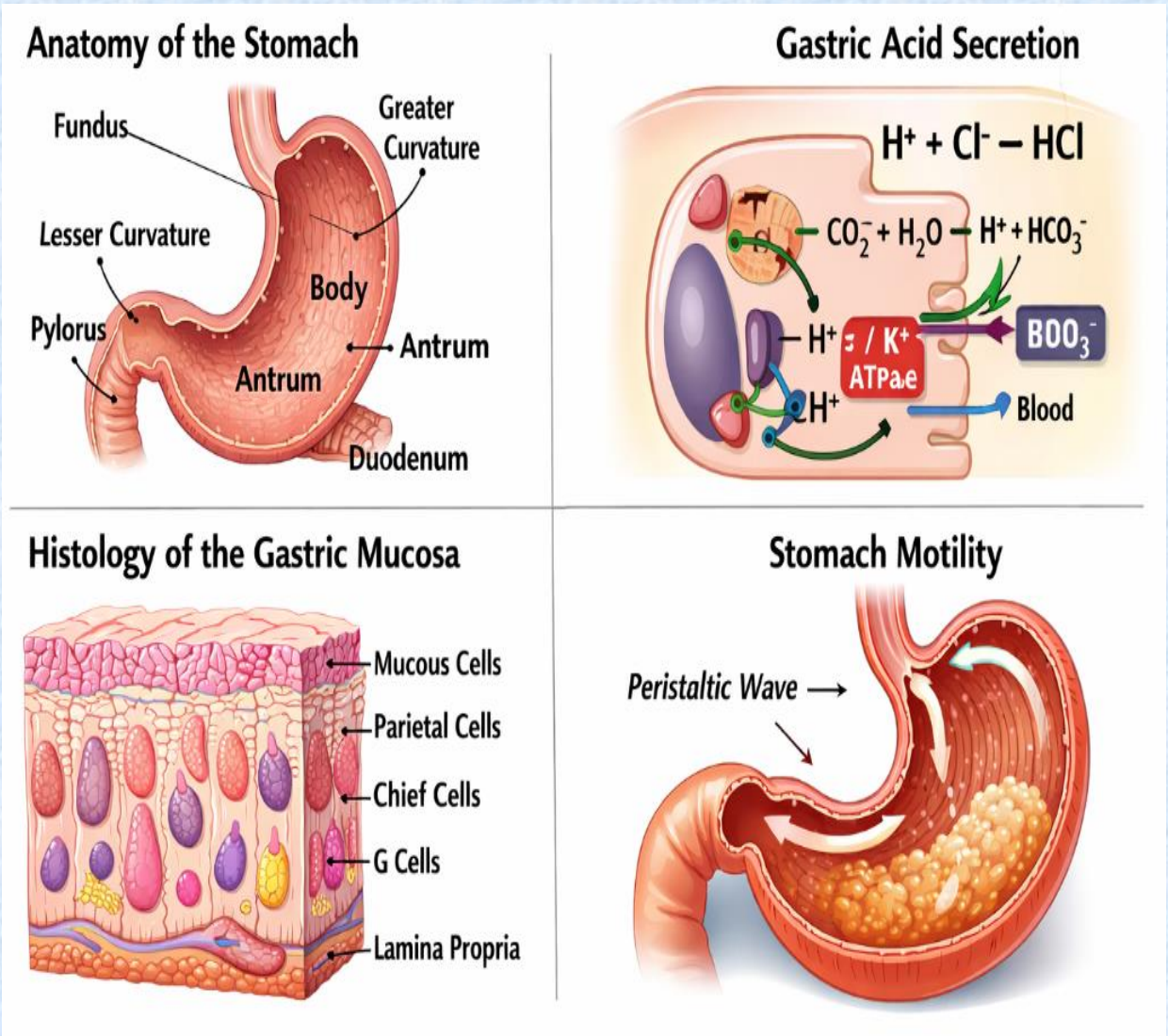
FERGANA MEDICAL INSTITUTE OF PUBLIC HEALTH

IBRAGIMOVA ZIYODA JALOLIDDINOVA

BASIC MORPHOFUNCTIONAL PRINCIPLES OF THE STOMACH



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This monograph was recommended for publication by the Scientific Council of the Fergana medical institute of public health in accordance with Resolution No. ___ of 2025.

CONTENT

Introduction

According to world statistics, approximately 7-14% of the adult population suffers from peptic ulcer disease. The prevalence of gastroesophageal reflux disease (GERD) among adults reaches 50%. Symptoms of the disease are observed equally often in men and women.

Extensive epidemiological studies in Western Europe and the USA show that from 40% to 81% of people constantly feel pain in the epigastric region, which is one of the main symptoms of chronic gastritis. Chronic gastritis is seasonal, and these diseases are exacerbated by weather changes. Typically, exacerbations of gastroduodenal diseases occur more often in spring and autumn.

As a result of the analysis of statistical data for the Republic of Uzbekistan, 56.6% of patients with gastrointestinal diseases are adults, 34.5% of patients are children under 14 years of age, and 8.9% are patients with all gastrointestinal diseases.

It is known that Helicobacter pylori infection is not detected in all patients with peptic ulcer disease, this group of patients accounts for 50–60% (gastric ulcer) and 50–85% (gastroduodenal ulcers). The persistence of Helicobacter pylori infection activates "aggression" factors and reduces "protective" factors, which facilitates the formation of ulcers.

This indicator increases dynamically, which occurs as a result of successful eradication therapy.

Purpose of the study:

To study the characteristics, course and treatment of chronic gastritis and peptic ulcer disease associated with and unrelated to Helicobacter pylori infection.

Objectives of the study:

1. To study the presence of Helicobacter pylori in patients with various forms of chronic gastritis.

2. To study the clinical features in patients with various forms of chronic gastritis.

3. To study the presence of Helicobacter pylori in patients with various forms of peptic ulcer disease.

4. To study the clinical features in patients with various forms of peptic ulcer disease in dynamics.

5. To develop an algorithm for monitoring and treating patients with chronic gastritis and peptic ulcer disease in order to prevent relapse of the disease:

Scientific novelty of the study.

Peptic ulcer disease has various localizations, and the pathogenetic and clinical aspects of the disease are similar to gastritis. At the same time, patients with Helicobacter pylori-associated peptic ulcer and chronic gastritis are characterized by inflammatory changes in the gastric mucosa and active "aggressive" factors, but a pronounced deficiency of "protective" factors leads to exacerbation of the disease.

Patients with peptic ulcer disease and chronic gastritis not associated with Helicobacter pylori infection are characterized by less pronounced inflammation in the gastric mucosa, low activity of "aggressive" factors and a significant deficiency of "protective" factors.

For the first time, the inclusion of antiviral drugs in the treatment complex helps to overcome the resistance of complex anti-ulcer therapy.

Long-term results up to 3 years were studied, in which the clinical course of the disease, relapses and complications were evaluated with Helicobacter pylori eradication. It was found that the frequency of relapses of Helicobacter pylori infection, chronic gastritis and peptic ulcer disease, with less severe symptoms, and less complicated exacerbations.

Methods and object of examination

During the research, a clinical and diagnostic complex was developed and implemented, which included the analysis of existing medical histories and the selection of new patients. Modern clinical and laboratory research methods (clinical,

morphological), serological, general laboratory tests (blood) and instrumental methods (gastroscopy, colonoscopy, ultrasound examination of internal organs) were used.

The methodology used identifies important indicators that allow choosing the most effective methods of anti-inflammatory and antiviral treatment of gastritis and peptic ulcer disease, as well as predicting the course of the disease and possible complications.

Scientific and practical significance of the study.

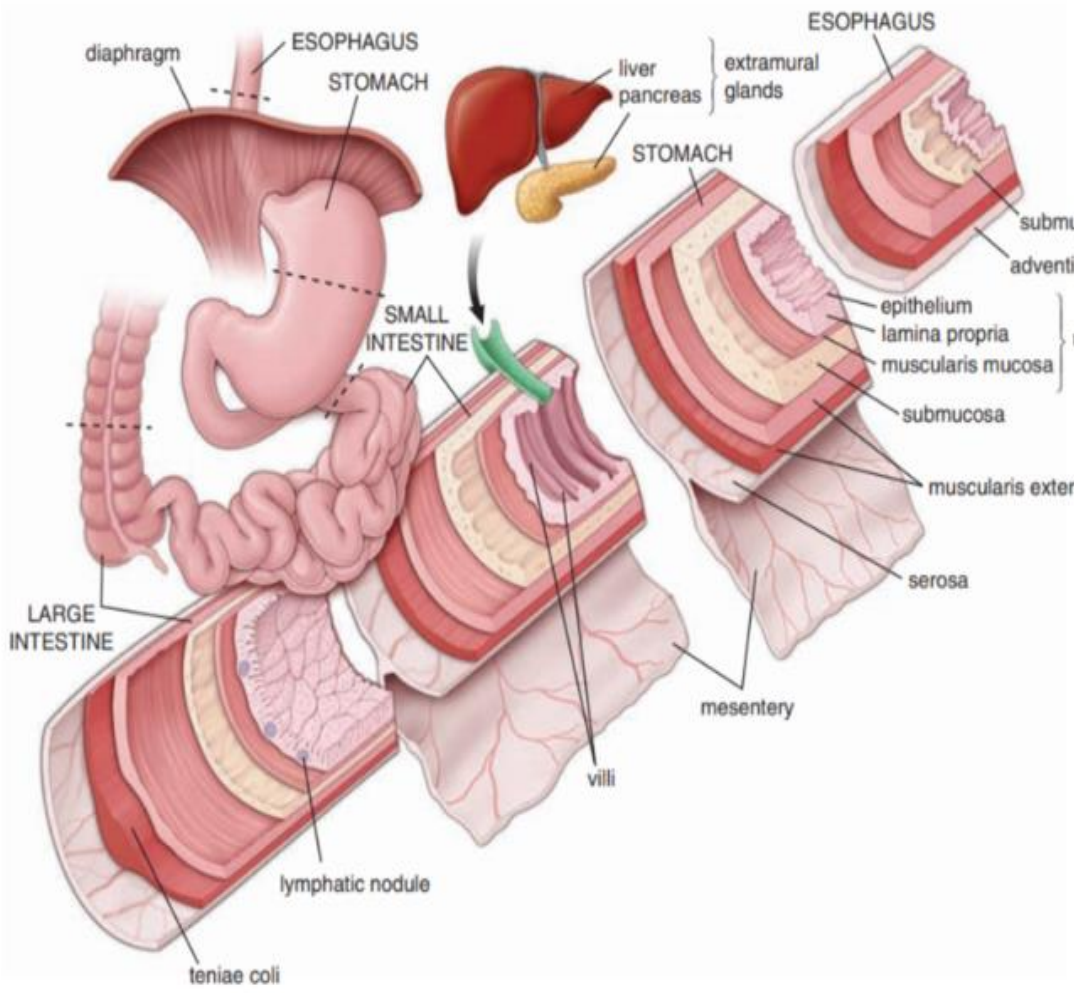
The main basis of this study was the study of the clinical course of gastritis and peptic ulcer disease, its recurrence and the development of complications, based on the work of scientists in our country and abroad. Despite the positive effect of modern treatment of these diseases, the disease continues to spread. The concept of various forms of chronic gastritis has been expanded, and they are considered a "risk factor" for peptic ulcer disease, and peptic ulcers not associated with Helicobacter pylori have been included.

CHAPTER I. Anatomy and histology of the stomach

1. Anatomy of the stomach

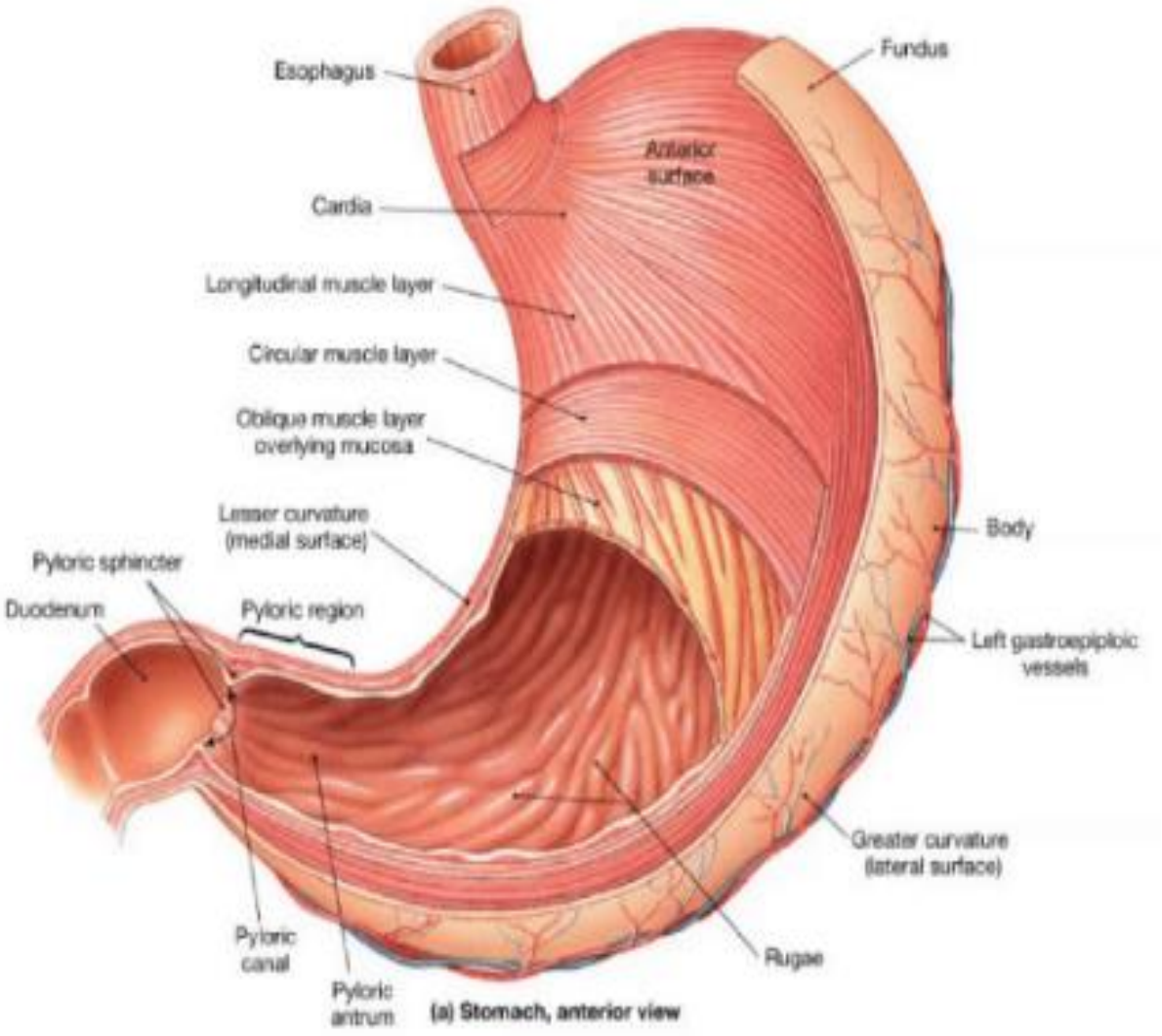
The stomach (ventriculus, gaster) is one of the central departments of the digestive system, located between the esophagus and the duodenum. It performs the functions of temporary storage of food, mechanical mixing and initiating chemical decomposition. This department is considered to be of practical and theoretical importance for medical students.

The stomach is located mainly in the epigastric and left subcostal areas, a large part of it can pass into the left hypochondrium, and a small part can pass a short distance from the midline to the right. From an anatomical-topographic point of view, the stomach is located in the upper part of the abdominal cavity , relatively close to the anterior abdominal wall, and from the back it is limited by the retroperitoneal organs through the bursa omentalis.



The upper part is located under the dome of the diaphragm and moves synchronously with the respiratory phases, and the lower part is in dynamic anatomical relationship with the transverse colon and its mesocolon. This relationship provides flexibility to changes in the volume of the stomach during the digestive process.

The posterior surface of the stomach is in close contact with the body of the pancreas, the left kidney, the adrenal gland, the splenic vessels, and the legs of the diaphragm through the area of the bursa omentalis , which in classical anatomy is described as the "place of the stomach bed". Therefore, inflammatory, ulcerative, or neoplastic processes of the stomach can spread directly or indirectly to these adjacent organs. For example, ulcers of the posterior wall of the stomach can penetrate the pancreas, which is clinically manifested by severe pancreatic pain.



The position and long axis of the stomach vary significantly depending on age, sex, body type (asthenic, normosthenic, hypersthenic type), intra-abdominal pressure, diaphragm height, and anatomical relationship with adjacent organs such as the liver, spleen, and pancreas. In asthenic body type, the stomach is often **ptotic** , lowered, and positioned in an elongated shape close to vertical, while in hypersthenics it is located higher and occupies a more transverse position. In normosthenics, the stomach is hook-shaped, representing the physiologically optimal position.

In children, the stomach is located relatively horizontally, its capacity is small, its walls are thinner, and the muscle layer is not sufficiently developed. With age, the stomach gradually moves to a more vertical position, the muscle layer strengthens, and its functional capacity increases. In old age, due to a decrease in muscle tone and an increase in connective tissue elements, gastric ptosis and delayed evacuation may occur.

The shape of the stomach is highly variable, and when empty it can be oblong, hooked, or tubular, and when full, its walls expand and take on a sac-like shape. The degree of filling with food, the physicochemical composition and volume of food, body position (standing, sitting, or lying), respiratory phases, abdominal muscle tone, and individual anatomical features directly affect the configuration and topography of the stomach. For example, liquid foods are quickly evacuated and cause less gastric distension, while solid and fatty foods cause gastric distension and increased peristalsis.

This topographic and morphological variability is the main theoretical and practical criterion for the correct assessment of the stomach in contrast radiography **,** CT/MRI , endoscopic examinations and surgical procedures. In radiography, the shape of the stomach, peristaltic waves, evacuation speed and degree of ptosis are determined; the distribution of the contrast medium provides important information about the functional state. CT and MRI examinations have an advantage in determining the spatial relationship of the stomach with adjacent organs (pancreas, liver, spleen, diaphragm), wall thickness and infiltrative processes. In endoscopy,

however, if the variability of anatomical landmarks is not taken into account, pathological foci may be misinterpreted.

In cases of gastric ptosis, diaphragmatic hernia, pancreatic diseases and pathology of adjacent organs, a thorough knowledge of the topography of the stomach is of decisive importance in differential diagnosis. Especially in surgical procedures (resection, vagotomy, laparoscopic interventions), taking into account the individual topographic features of the stomach reduces operational risks, prevents unnecessary or inappropriate surgical interventions, and significantly reduces the likelihood of postoperative complications.

2. Parts of the stomach

The stomach consists of the following main anatomical parts, each of which has its own morphological and functional significance:

The cardia is the anatomical zone connecting the esophagus to the stomach, which does not have a separate morphological sphincter from a structural point of view, but from a functional point of view it acts as the lower esophageal sphincter (LES). In this region, a powerful anti-reflux mechanism is formed as a result of the coordinated activity of the distal smooth muscle fibers of the esophagus, the circular muscle layer of the gastric wall, and the legs of the diaphragm (crura diaphragmatica). This mechanism limits the retrograde passage of the acidic environment of the stomach and proteolytic enzymes into the esophagus, protecting the epithelium of the upper part from damage.

Histologically, the cardiac area is dominated by mucous glands , with parietal cells being very rare or absent. This condition ensures the resistance of the cardiac epithelium to the aggressive effects of hydrochloric acid and pepsin. The mucus produced by the cardiac glands is alkaline in nature and forms a protective barrier.

Clinically, functional insufficiency of the cardiac region is one of the main pathogenetic factors in the development of gastroesophageal reflux disease (GERD) . Long-term reflux can lead to esophagitis, metaplasia processes and the formation of Barrett's esophagus , which increases the risk of developing esophageal

adenocarcinoma. Therefore, a thorough knowledge of the anatomical and histological features of the cardiac region is of great importance in gastroenterology and surgical practice.

The fundus is the upper, dome-shaped part of the stomach, usually located under the diaphragm and anatomically adjacent to the spleen on the left. The fundus is the highest point of the stomach, and its position is directly related to the height of the diaphragm and the phases of breathing. Anatomically, the fundus is located above the junction with the esophagus and plays an important role in the distribution of pressure within the stomach.

An air or gas bubble often accumulates in the fundus area, therefore, in X-ray examinations, it serves as the main landmark in assessing the functional state, peristalsis, and general shape of the stomach. The absence or change in the location of the gas bubble may indicate pathological conditions, including impaired gastric wall tone or pressure from adjacent organs.

Histologically, the fundus is rich in fundal (main) glands , which contain a large number of parietal and chief cells. Parietal cells synthesize hydrochloric acid and intrinsic factor, while chief cells produce pepsinogen. Therefore, the fundus is one of the most active zones of gastric juice secretion. The secretory activity of the fundus and body areas ensures the acidity of the internal environment of the stomach and is crucial for the initial breakdown of proteins.

Clinically, damage or functional changes in the fundus area can lead to decreased gastric secretion, impaired digestion, and reduced absorption of vitamin B12. Therefore, a thorough understanding of the anatomy and histology of the fundus is important in gastroenterological diagnosis and treatment.

The body (corpus) is the largest anatomical part of the stomach, it is the main working part of the stomach. It is in the body that intensive mechanical mixing of the food mass, the formation of peristaltic waves and the main stages of chemical breakdown of food particles take place. The muscle layers of the stomach wall are especially well developed in this area, ensuring the separation of the food mass into small fractions and its complete mixing with gastric juice.

The gastric glands are very densely located in the body, and their histological structure is similar to the fundal glands. Parietal cells actively synthesize hydrochloric acid and intrinsic factor, which creates the necessary conditions for maintaining optimal acidity of the gastric mucosa and the absorption of vitamin B12. Pepsinogen, produced by chief cells, is activated in an acidic environment and ensures the breakdown of proteins into polypeptides. Mucous cells, in turn, produce protective mucus, protecting the gastric wall from aggressive acid-enzyme effects.

Functionally, the body part plays a leading role in the secretory **,** motor and partly endocrine activity of the stomach. Violation of the secretory activity in this area can lead to states of hypochlorhydria or hyperacidity, which are clinically manifested by dyspeptic complaints, the development of ulcer disease or atrophic gastritis. Therefore, a thorough knowledge of the anatomical and histological features of the body part (corpus) is important in choosing clinical diagnostics and treatment tactics.

The pyloric part is the distal part of the stomach, which is anatomically and functionally the main zone regulating the passage of food mass from the stomach to the duodenum. This part consists of the antrum pyloricum and canalis pyloricus , each of which performs a specific morphological and physiological function.

The pyloric antrum is a relatively large area where the food mass is actively mixed and the grinding process reaches its final stage. Histologically, the antrum is dominated by mucous glands and contains a large number of gastrin-producing G-cells . The hormone gastrin increases the secretion of gastric juice, stimulates the activity of parietal cells, and increases gastric peristalsis. At the same time, the pyloric area also contains D-cells that produce somatostatin, which act as a regulator that inhibits gastrin secretion.

The pyloric canal is a narrow passage that leads to the pyloric sphincter. A strongly developed circular muscle layer located in the pylorus area forms the pyloric sphincter. This sphincter ensures the gradual passage of food from the stomach to the duodenum in small portions, prevents the regurgitation of intestinal contents into the stomach, and maintains the integrity of gastrointestinal transit.

Clinically, the pyloric region is of particular importance: in children, hypertrophy of the pyloric muscles can lead to congenital pyloric stenosis , which is manifested by vomiting and impaired food passage. In adults, ulcers, cicatricial strictures and tumors of the pyloric region can disrupt the evacuation function of the stomach and cause clinically severe dyspeptic conditions. Therefore, a thorough knowledge of the anatomical and histological features of the pyloric region is of great importance in gastroenterology and surgical practice.

3. Curves and surfaces

The stomach has two main curvatures anatomically, which are important anatomical landmarks in the external configuration of the stomach, the location of blood vessels and lymphatic vessels, as well as in surgical and endoscopic procedures. Along the curvatures are located the vascular-nerve bundles of the stomach, and these areas are considered sensitive zones for pathological processes.

The lesser curvature (curvatura minor) is located along the upper and right edge of the stomach, through which the lesser omentum (omentum minus) is attached. The a. gastrica sinistra and a. gastrica dextra pass through this area , forming anastomoses with each other. The mucous membrane in the area of the lesser curvature is relatively thinner, and due to certain features of the blood supply, this area is more sensitive to the aggressive effects of gastric juice. Clinically, gastric ulcers, chronic gastritis foci, and adenocarcinomas are often localized precisely in the area of the lesser curvature, which indicates the important diagnostic value of this zone.

The greater curvature (curvatura major) runs along the lower and left edge of the stomach and is attached to it by the greater omentum **(**omentum majus). Along this area , the a. gastroepiploica sinistra and a. gastroepiploica dextra , as well as the short gastric vessels, are located. The greater curvature provides the extensibility of the stomach wall and plays an important role in the mechanical mixing of food mass and the formation of peristaltic waves. The mechanisms of limiting inflammatory processes and preventing the spread of infection are implemented through the greater omentum.

The stomach has anterior (facies anterior) and posterior (facies posterior) surfaces, which are in close topographic relationship with adjacent organs. The anterior surface is mainly in contact with the diaphragm and liver, and the posterior surface is associated with the spleen, pancreas, left kidney and transverse colon. These anatomical relationships explain the mechanism of spread of pathological conditions to adjacent organs during inflammation, tumor processes and perforations and are of great practical importance in surgical interventions.

4. Blood supply and innervation

The stomach is supplied with arterial blood mainly through the branches of the truncus coeliacus , a system adapted to meet the high metabolic, secretory and regenerative needs of the stomach. The arterial blood supply includes a. gastrica sinistra **,** a. gastrica dextra **,** a. gastroepiploica sinistra and dextra , as well as aa. gastricae breves (short gastric arteries) . These vessels are located along the lesser and greater curvatures, forming numerous arterial anastomoses . These anastomoses reduce the risk of ischemic damage to various parts of the stomach wall and ensure the stability of blood flow during the digestive process. Especially in surgical procedures (subtotal or total gastrectomy, distal resection), the preservation of these anastomoses is crucial to ensure the viability of the residual part of the stomach.

The arterial blood supply of the stomach is functionally closely related to the secretory zones , and the blood flow is relatively stronger in the fundus and body areas due to the dense concentration of parietal cells. In cases of stress, hypovolemia, or shock, perfusion disorders are observed in these areas, which can lead to the development of stress ulcers and hemorrhagic gastritis.

Venous blood is mainly drained into the v. gastricae sinistra et dextra **,** v. gastroepiploicae into the v. portae hepatis system. This ensures that substances, drugs and toxins absorbed through the stomach are primarily metabolized in the liver. Clinically, in cases of portal hypertension, portal gastropathy and upper gastrointestinal bleeding may develop as a result of the expansion of the venous plexuses in the lower part of the stomach and esophagus. These conditions are life-threatening and require an urgent endoscopic and surgical approach.

The innervation of the stomach occurs through a complex integration **of** the central, autonomic, and enteric nervous systems . Parasympathetic innervation via the vagus nerve enhances gastric gland secretion, peristaltic activity, and coordinated opening and closing of the pyloric sphincter. The bulk of the vagus nerve impulses are transmitted through the enteric nervous system, providing automatic adaptation of the stomach through local reflexes.

Sympathetic nerve fibers (mainly through the truncus sympathicus and plexus coeliacus**)** control vascular tone, transmit pain impulses to the central nervous system, and inhibit gastric secretion and motility in stress situations. When the physiological balance between the parasympathetic and sympathetic systems is disturbed, functional dyspepsia, spasms, hyperacidity, or evacuation disorders occur.

Clinically and practically, the nerve supply of the stomach forms the pathophysiological basis of surgical methods such as selective and truncal vagotomy**.** Also, the integrated and coordinated functioning of the vascular and nervous systems ensures the flexible, efficient and physiological functioning of the stomach, and is of crucial importance in understanding the mechanisms of clinical symptoms and pathological processes.

2. Histology of the stomach

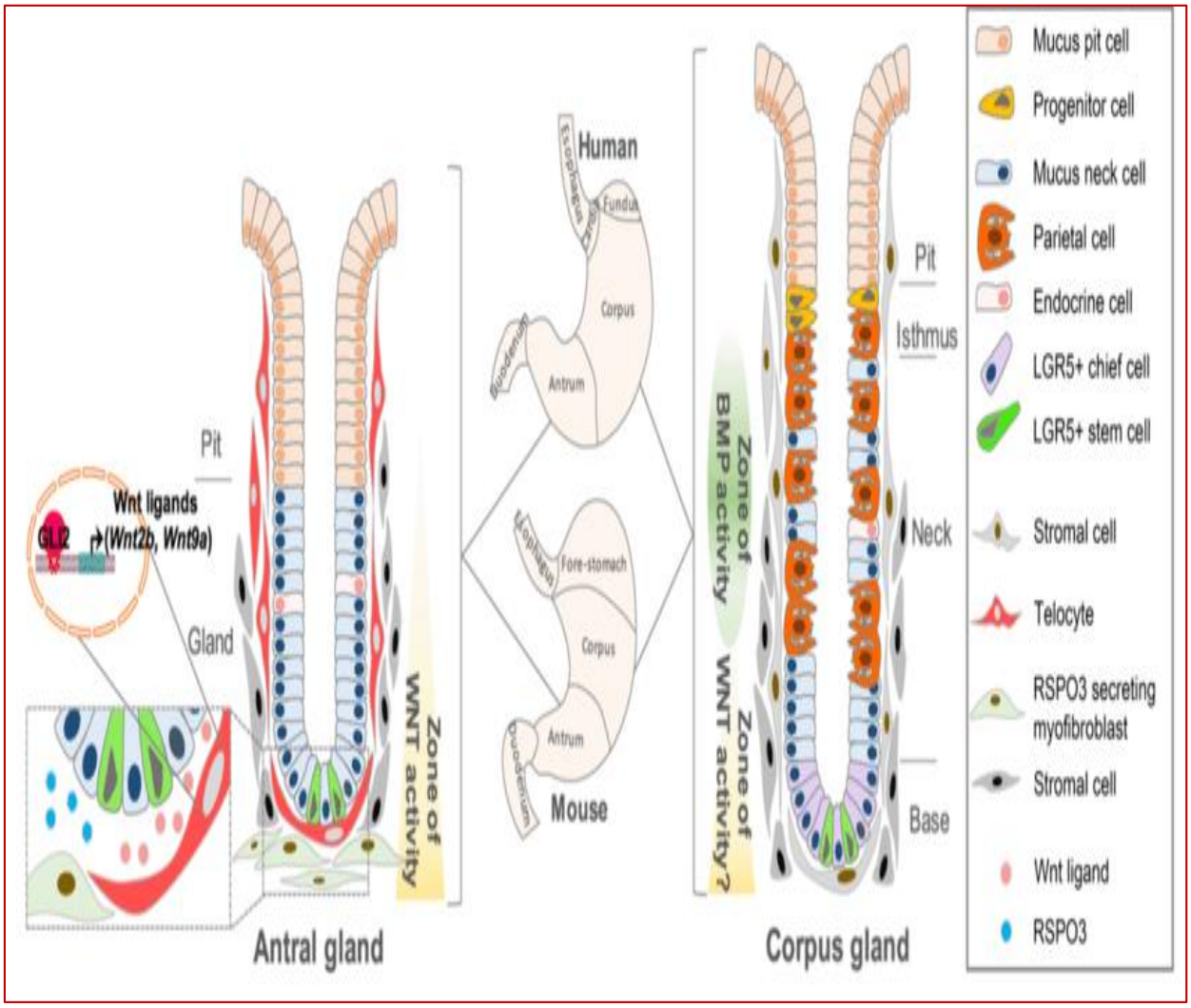
The stomach wall has a complex functional structure, consisting of four main layers . Each layer plays an important role in ensuring the secretory, motor, protective and regulatory functions of the stomach.

1. Mucous membrane (tunica mucosa)

The mucosa is the most important, biologically active and clinically significant layer of the stomach wall . It is this layer that is at the heart of the secretory, protective and regulatory functions of the stomach, and most pathological processes begin in this layer first. The mucosa consists of the following components:

a single-layered columnar epithelium , which is composed of highly specialized secretory cells. The epithelial cells continuously produce mucus, which is rich in mucin glycoproteins , water, and bicarbonate ions, and forms a mucus–

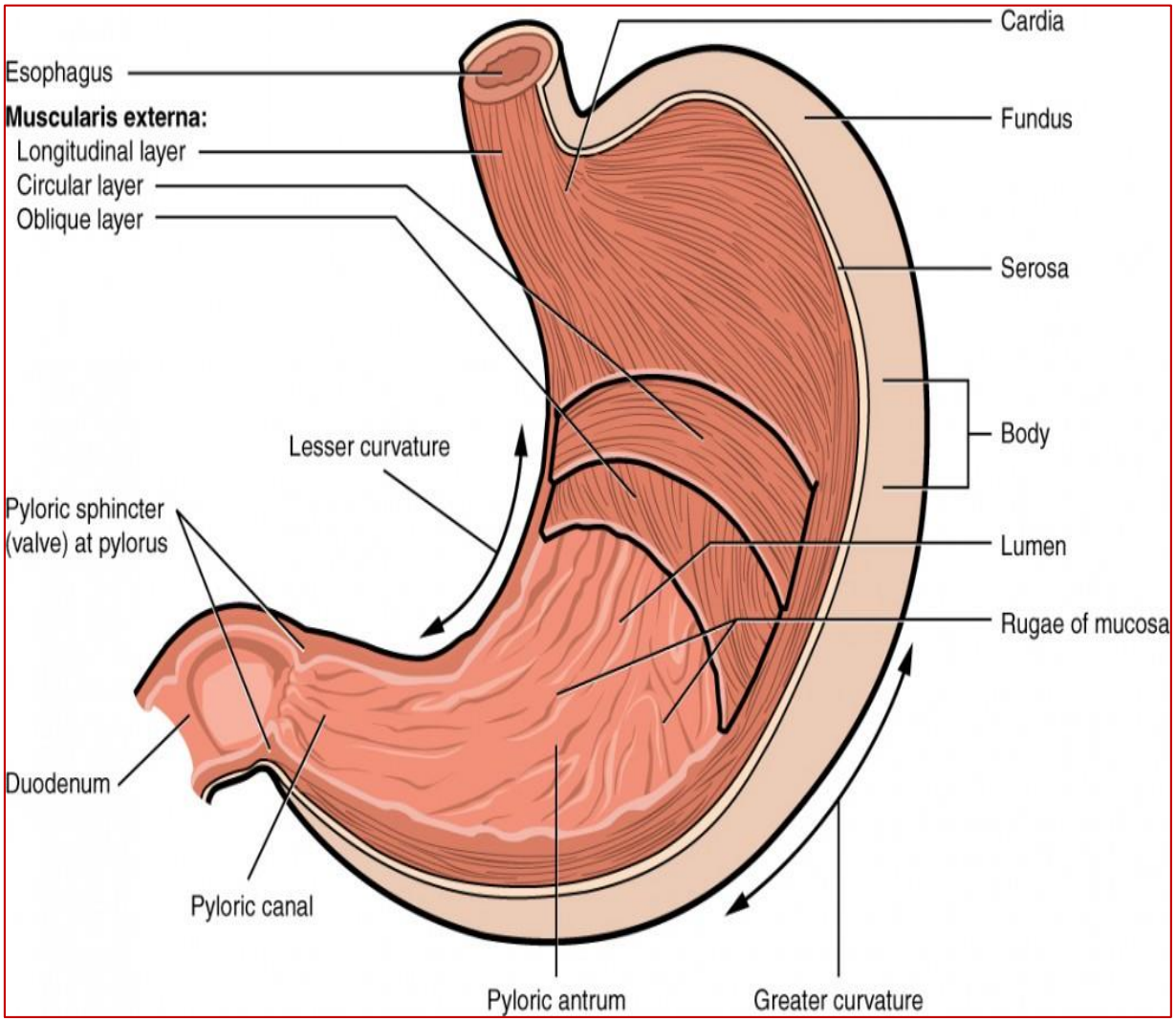
bicarbonate protective barrier against the strongly acidic environment of gastric juice **.** This barrier maintains a relatively neutral pH (≈6–7) on the epithelial surface and protects the epithelial cells from the cytotoxic and proteolytic effects of hydrochloric acid and pepsin.



Tight junctions between epithelial cells limit the back diffusion of hydrogen ions and maintain the integrity of the gastric mucosa. In addition, epithelial cells participate in the synthesis of prostaglandins, which increase mucus and bicarbonate secretion, and improve local blood flow.

The rapid renewal of epithelial cells (regeneration) is achieved by their dividing neck cells, and the epithelium is usually completely renewed within 3–5 days. This high regenerative capacity increases the resistance of the mucosa to mechanical, chemical, and bacterial damage. Disruption of the regeneration process

(e.g., due to Helicobacter pylori infection, NSAIDs, or ischemia) leads to the destruction of the epithelial barrier, erosion, and ulceration.



Lamina propria is a loose fibrous connective tissue that forms the main supporting and functional environment of the mucosa. This layer contains gastric glands, a dense capillary vascular network, lymphatic vessels, nerve fibers, and many immunocompetent cells - plasma cells, T and B lymphocytes, macrophages, dendritic cells . Lamina propria provides trophic support to the mucosal epithelium, supports metabolism, and creates the microenvironment necessary for epithelial regeneration.

This layer is considered the central link of the local immune defense system**,** providing mucosal immunity through plasma cells that produce secretory IgA. At the level of the lamina propria, antigen presentation, cytokine and chemokine release occur, and these processes determine the intensity and duration of the inflammatory

response. In Helicobacter pylori infection, it is within the lamina propria that lymphocytic infiltration, follicle formation, and morphological changes characteristic of chronic active gastritis develop. In autoimmune gastritis, immune cells are directed against parietal cells, which is accompanied by progressive inflammation and glandular atrophy at the level of the lamina propria. Therefore, a deep understanding of the structure and cellular composition of the lamina propria is important in understanding the types of gastritis, immune-pathogenetic processes, and the mechanisms of development of gastric tumors.

The muscularis mucosae is a thin but functionally important layer of smooth muscle fibers that clearly separates the mucosa from the submucosa. This muscularis mucosae provides independent and localized movements of the mucosa **,** acting relatively independently of the general peristalsis of the gastric wall.

Rhythmic and subtle contractions of the muscularis mucosae facilitate the active release of gastric glandular secretions through the glandular ducts, prevent stagnation of secretions within the gland, and ensure their even distribution over the epithelial surface **.** In addition, this muscular layer plays an important role in the formation and maintenance of the mucosal folds (plicae gastricae), thereby expanding the functional area of the mucosal surface.

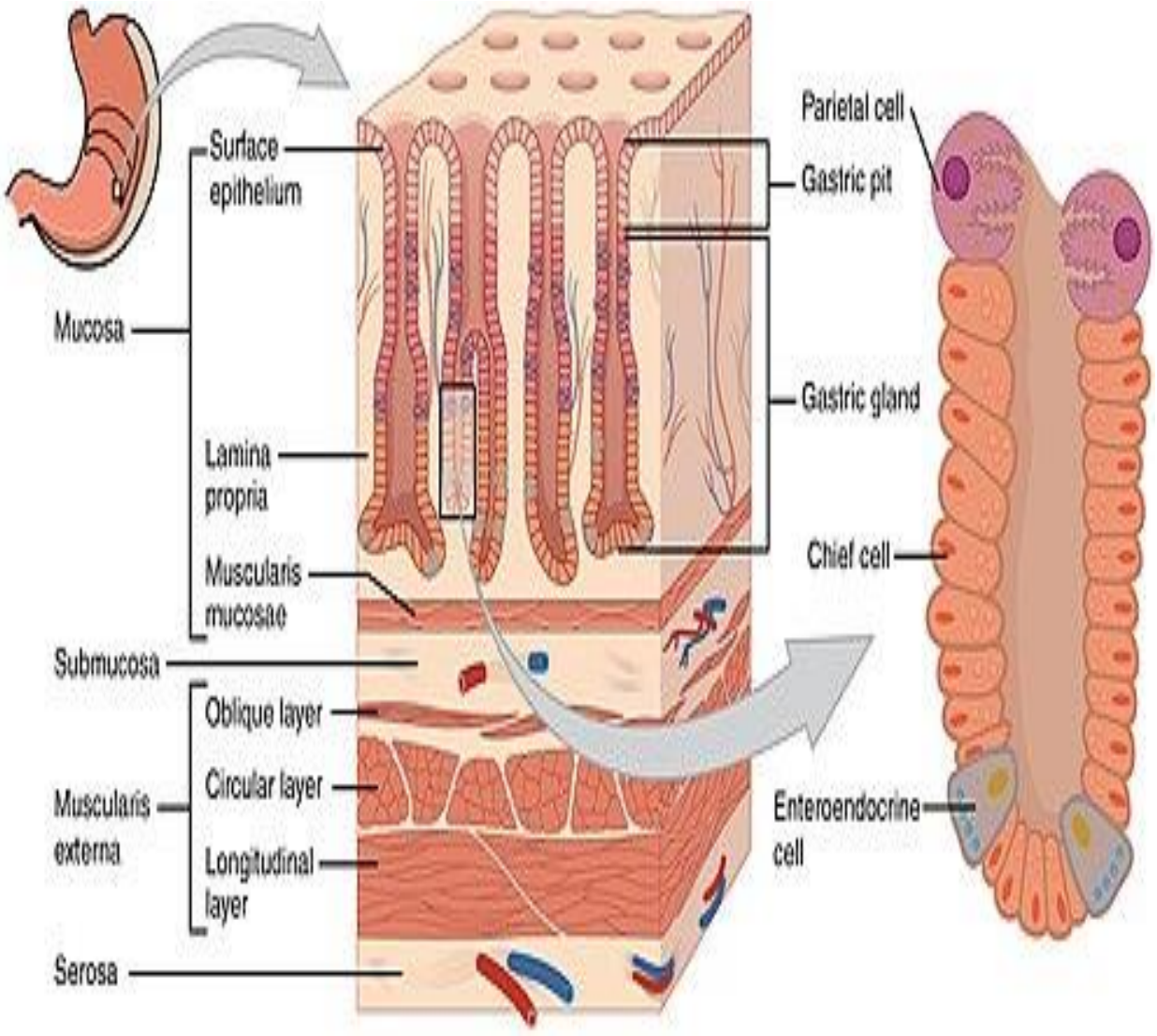
Histologically, the muscularis mucosae is inextricably linked to the lamina propria, and hyperplasia or destruction of this layer can be observed in inflammatory processes (gastritis). Disruption of the integrity of the muscularis mucosae leads to impaired mucosal drainage, accumulation of glandular secretions, and the spread of inflammatory processes to deeper layers.

Clinically and practically, the muscularis mucosae is a diagnostic boundary layer: in tumor processes, the passage of cells through this layer indicates the transition of the disease to the invasive stage. Therefore, the determination of the preservation or destruction of the muscularis mucosae in the evaluation of biopsy materials is crucial in differentiating between early carcinoma, intramucosal and submucosal invasion stages.

Clinically, mucosal damage is the main morphological substrate of acute and chronic gastritis**,** erosion, gastric ulcer, as well as atrophic and metaplastic processes**.** Long-term structural changes in the mucosa can create the basis for the development of gastric adenocarcinoma. Therefore, a thorough knowledge of the anatomical and histological structure of the mucosa is crucial for clinical diagnosis, endoscopic evaluation and selection of treatment strategies.

2. Gastric glands and cells

The glands located in the gastric mucosa are distinguished by their location and cellular composition (cardiac, fundal, and pyloric glands). These glands form the morphological basis of the secretory and endocrine functions of the stomach, and their cellular composition is strictly specialized. Each cell type has its own ultrastructure, enzymatic activity, and clinical significance:



Chief cells are highly specialized secretory cells with a strongly basophilic cytoplasm , located mainly in the basal part of the glands . They are characterized by a developed rough endoplasmic reticulum , a well-formed Golgi apparatus , and numerous zymogen granules , which indicate their adaptation to intensive protein synthesis and enzyme production.

Pepsinogen, synthesized by chief cells , is a physiologically inactive proenzyme that is converted to the active enzyme pepsin under the influence of the acidic environment of the stomach (pH < 3) . Pepsin breaks down proteins into polypeptides and oligopeptides, thus providing the initial stage of protein digestion in the stomach. In addition, chief cells also produce a small amount of gastric lipase , which plays an important role in the initial breakdown of fats, especially in infants.

In histological preparations, chief cells are recognized by their basal location, strongly basophilic cytoplasm, and relatively low nuclei. They are smaller and more densely packed than parietal cells.

Clinically, the decrease in the activity or atrophy of chief cells is manifested by protein maldigestion , flatulence, a feeling of heaviness and dyspeptic complaints. In atrophic gastritis and long-term inflammatory processes, a decrease in the number of chief cells leads to a decrease in the enzymatic activity of gastric juice. Therefore, a thorough knowledge of the structure and function of chief cells is important in the clinical assessment of gastritis pathogenesis, digestive disorders and gastric secretory insufficiency.

Parietal cells (oxyntic cells) are highly specialized secretory cells of large, pyramidal shape, located mainly in the fundus and body regions of the gastric glands. They are characterized by eosinophilic cytoplasm **,** numerous mitochondria**,** a developed tubulovesicular system , and a complex membrane apparatus, which indicates their adaptation to high-energy secretory activity.

Hydrochloric acid (HCl), synthesized by parietal cells, is secreted into the gastric cavity by a special H^+ /K^+ -ATPase (proton pump) . This process occurs with the participation of the enzyme carbonic anhydrase and is characterized by the active secretion of hydrogen ions. HCl provides the acidity of gastric juice, plays a

crucial role in the conversion of pepsinogen to pepsin, denaturation of proteins, and bactericidal protection against microorganisms.

Parietal cells also produce intrinsic factor (Castle factor) . This glycoprotein protects vitamin B12 from proteolytic cleavage in the stomach and duodenum and ensures its absorption through special receptors in the terminal ileum . Intrinsic factor deficiency leads to malabsorption of vitamin B12, impaired DNA synthesis, and the development of megaloblastic hemopoiesis .

In histological preparations, parietal cells are distinguished by their large size, pale eosinophilic cytoplasm, and centrally located nuclei. They are larger than chief cells and are more common in the neck and middle of the gland.

Clinically, autoimmune damage to parietal cells (autoimmune gastritis) or atrophic processes lead to a sharp decrease in the secretion of HCl and intrinsic factor. As a result, hypochlorhydria or achlorhydria , digestive disorders, changes in intestinal microflora, pernicious (megaloblastic) anemia and an increased risk of developing gastric adenocarcinoma are observed. Therefore, a deep understanding of the structure, secretion mechanisms and pathology of parietal cells is of great importance in the practice of gastroenterology, hematology and oncology.

Mucous cells are highly differentiated secretory cells located in the neck and superficial epithelium of the gastric glands. They produce a neutral to alkaline mucus , which contains mucin glycoproteins, water, and bicarbonate ions, forming a stable protective barrier in the gastric mucosa.

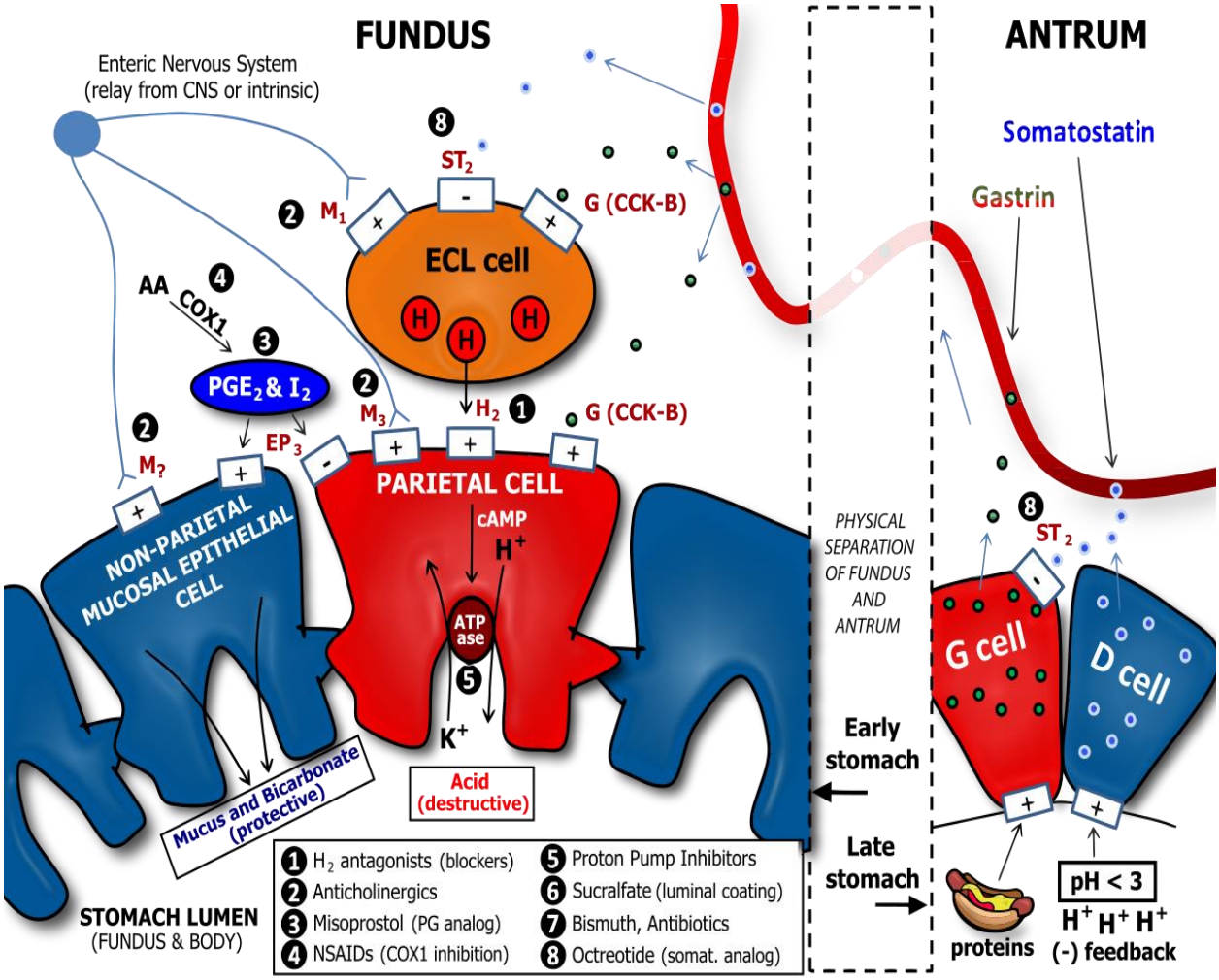
The mucus produced acts as a mechanical, chemical and biological protective layer on the surface of the epithelium **,** limiting the autolytic action of hydrochloric acid and pepsin, and also partially blocking the adhesion of microorganisms to the epithelium. The activity of mucous cells is closely related to prostaglandins, which enhance mucus secretion and improve the blood supply to the mucosa.

In histological preparations, mucous cells are characterized by pale cytoplasm, basal location of the nucleus, and mucus-filled vacuoles. Clinically, decreased function of mucous cells or impaired mucus quality significantly increases

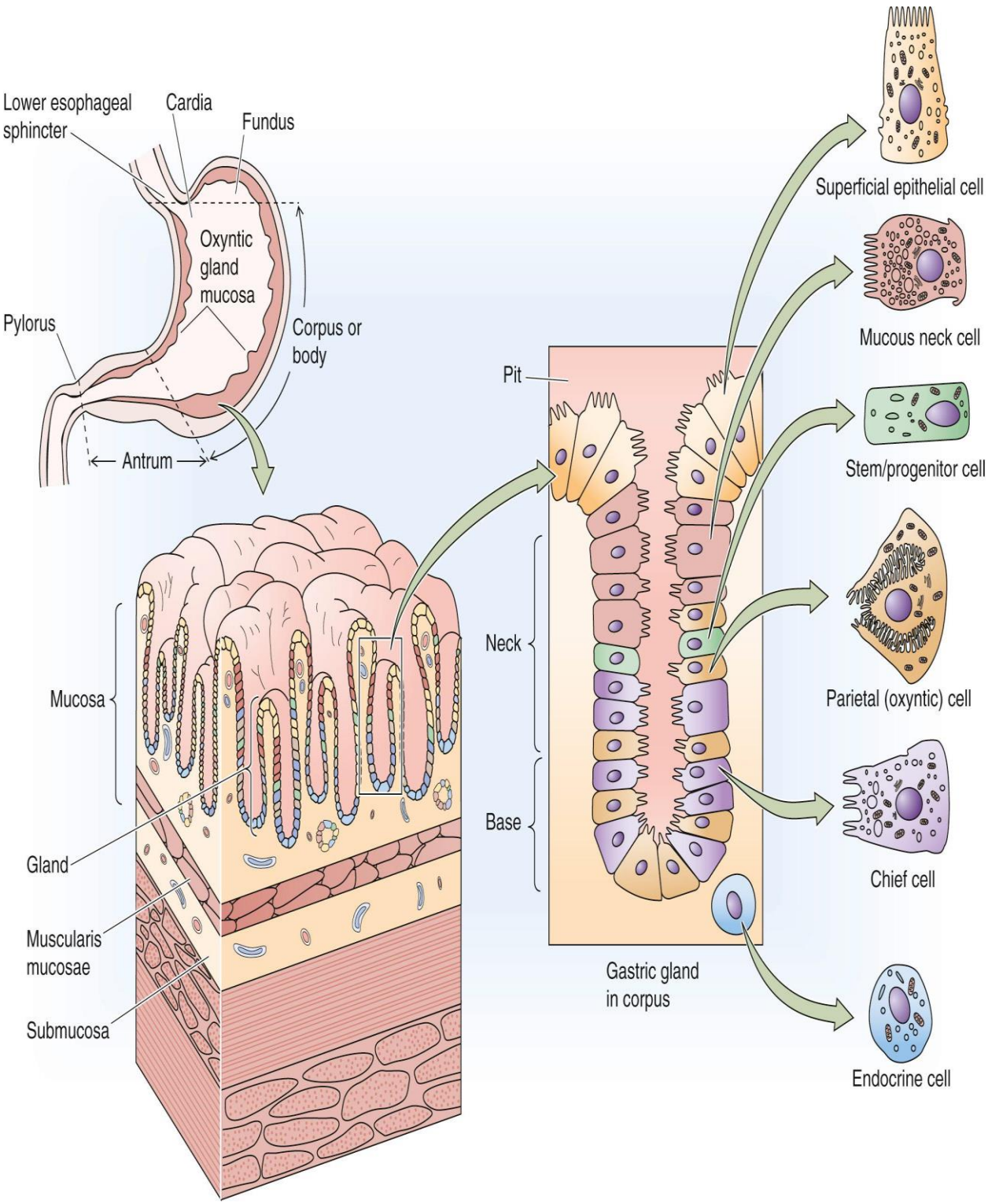
the susceptibility to the development of erosion, gastric ulcer, NSAID --induced gastritis, and stress ulcers **.** Therefore, a thorough understanding of the protective mechanisms of mucous cells is important in understanding the pathogenesis of gastric mucosal pathologies.

Enteroendocrine cells are specialized cells that are part of the diffuse endocrine system (DNES) located in the gastric mucosa , which are few in number but extremely functionally active. They are mainly located in the basal part of the glands and secrete hormones from the basal side , thereby finely regulating gastric activity in a paracrine, endocrine and neuroendocrine manner. These cells constitute the central regulatory link of gastric secretion, motility and mucosal trophism.

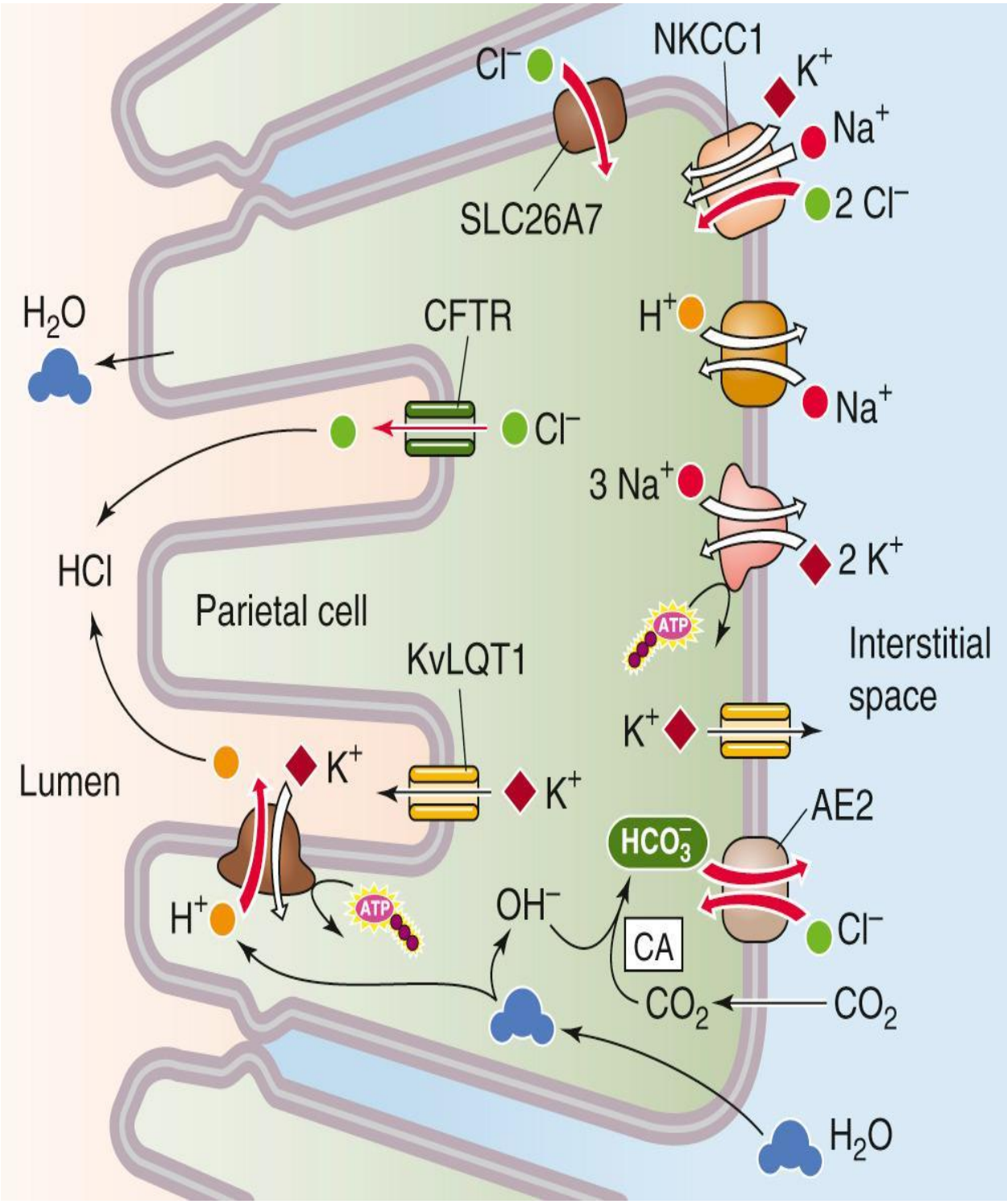
G cells (mainly located in the pyloric antrum) are highly specialized enteroendocrine cells that synthesize the hormone gastrin . Gastrin secretion is regulated by the pH of the gastric cavity, the protein content of the diet, vagus nerve stimulation, and local paracrine mechanisms.



Gastrin stimulates parietal cells directly and **i**ndirectly through ECL cells , increasing the secretion of hydrochloric acid. At the same time, gastrin activates the peristalsis of the gastric muscle layer, coordinates the evacuation process in the pyloric region, and supports the proliferation and regeneration of the mucous glands. As a trophic hormone, gastrin plays an important role in the physiological renewal of glandular cells in the fundus and body regions.

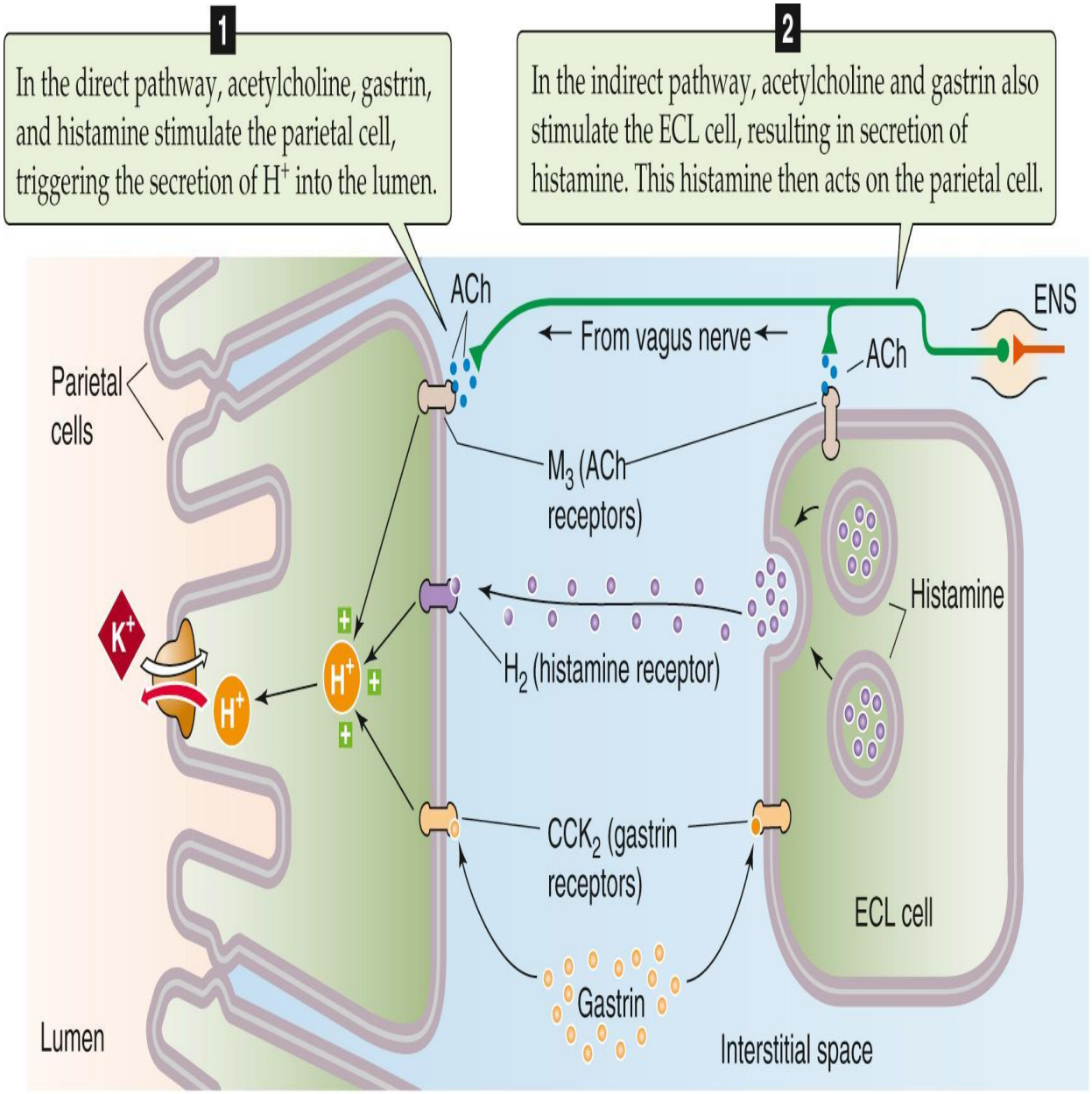


Increased G-cell activity leads to **hypergastrinemia** , which is the pathogenetic basis of severe clinical conditions such as hyperacidity, gastric and duodenal ulcers, and **Zollinger–Ellison syndrome** . Conversely, decreased G-cell activity can be manifested by decreased gastric secretion and impaired digestion. Therefore, a thorough understanding of the hormonal regulation of G-cells is important for the understanding and treatment of gastroenterological diseases.



ECL cells (enterochromaffin-like cells) are located mainly in the fundus and body regions and produce histamine . Histamine is one of the most potent stimulants of HCl secretion through H2 receptors on parietal cells. Therefore, the activity of ECL cells constitutes one of the main regulatory mechanisms of acid secretion, and the clinical effects of H2 blockers and proton pump inhibitors are directly related to this chain.

D-cells It produces somatostatin **,** which inhibits the secretion of gastrin and histamine, thereby maintaining gastric acidity within physiological limits. D-cells are important as an "inhibitory system" of gastric secretory activity, and when their activity decreases, the risk of hyperacidity and ulcer development increases.



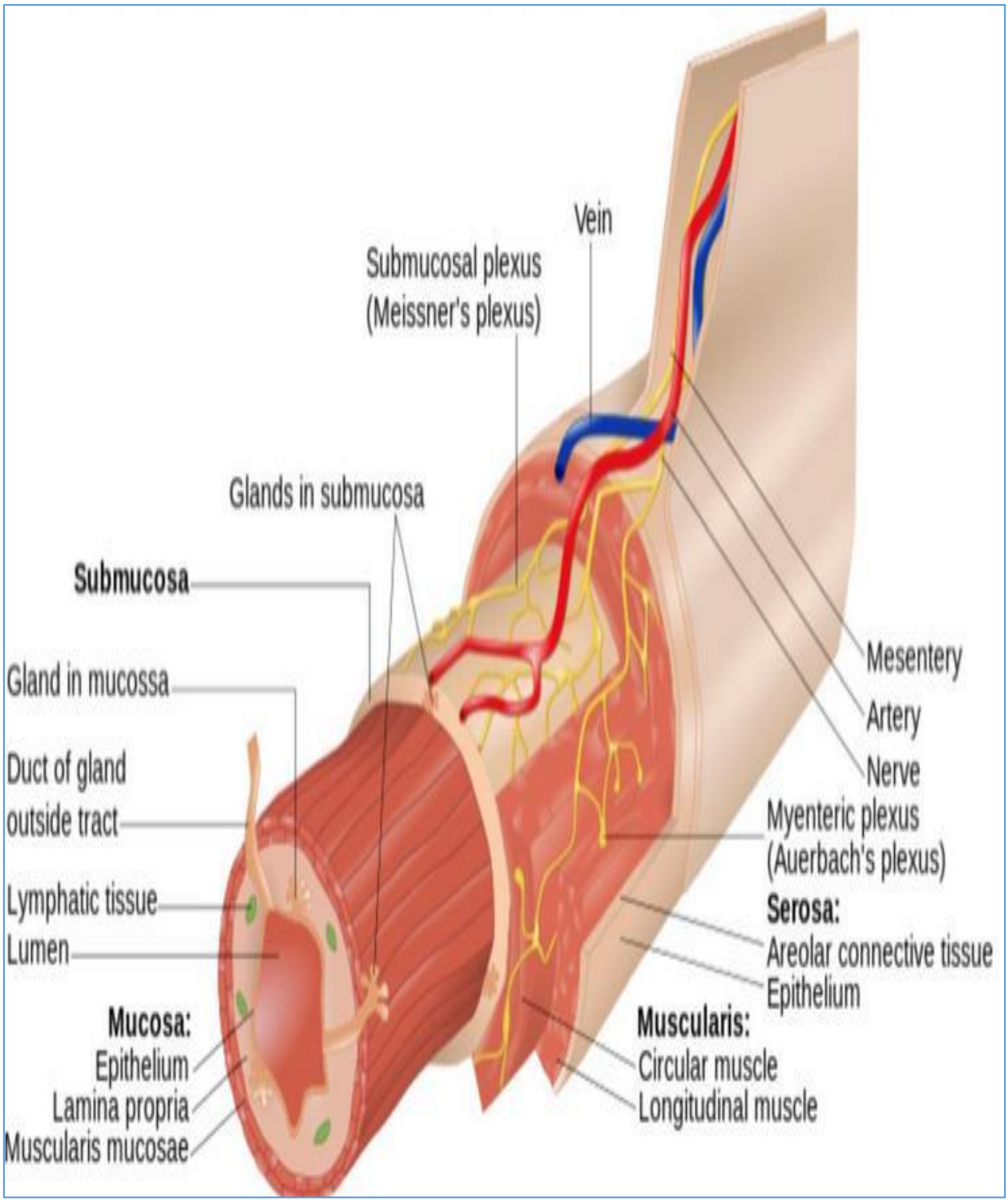
In histological preparations, enteroendocrine cells are poorly visible with simple stains, but they are clearly identified using immunohistochemical methods (chromogranin A, synaptophysin). Clinically, proliferation or hormonal imbalance of these cells can lead to the development of Zollinger–Ellison syndrome **,** neuroendocrine tumors (carcinoids) , and functional dyspepsia. Therefore, a deep understanding of the structure and functional regulation of enteroendocrine cells is of great importance in the practice of gastroenterology, endocrinology, and oncology.

3. Submucosa

The submucosa is a dense and elastic connective tissue that provides mechanical strength to the stomach wall and acts as a functional shock absorber between the mucosa and the muscularis mucosa . This layer ensures an even distribution of forces during the processes of stretching and contracting the stomach wall without deforming. contains large arterial, venous, and lymphatic vessels that meet the high metabolic needs of the mucosa. It is through this layer that oxygen and nutrients are delivered to the mucosa and metabolic products are removed. The rich network of lymphatic vessels plays an important role in local immune control and the limitation of inflammatory processes.

Meissner's (submucosal) nerve bundle, located in the submucosa, is an important component of the enteric nervous system. This nerve bundle reflexively controls the secretion of gastric glands, the trophism of the mucosa, capillary blood flow, and mucus production. Normal activity of Meissner's bundle is necessary for the rapid renewal of the mucosal epithelium, the stability of the mucus-bicarbonate barrier, and the maintenance of local defense mechanisms.

a high degree of elasticity of the stomach wall , allowing the mucosa and muscular layer to move freely relative to each other during filling with food. This property protects against a sharp increase in intragastric pressure and ensures the physiological course of peristalsis.



Clinically, the submucosa is of great diagnostic and prognostic importance . Submucosal edema, hemorrhages, and fibrotic changes are observed in cases of inflammation and portal hypertension. In neoplastic processes, the penetration of tumor cells into the submucosa is an important criterion for determining the stage of the disease. Accurate assessment of the mucosa-submucosa boundary during

endoscopic examination and biopsy is crucial in differentiating early carcinoma from invasive processes.

4. Muscular layer (tunica muscularis)

The muscular layer of the stomach is one of the most morphologically and functionally complex muscle structures in the digestive system. It consists of three layers and forms the main structural basis for the motor, evacuation and mixing functions of the stomach. Such a complex structure of the muscular layer is necessary for the effective mechanical processing of food mass and its preparation for passage into the intestine.

The internal oblique muscle layer is a unique layer that is unique to the stomach. These muscle fibers are arranged in different directions and provide intensive mechanical mixing, grinding and turning of the food mass. The internal oblique layer creates strong local pressure zones in the stomach and plays an important role in achieving a uniform consistency of the chyme. Clinically, when the sufficient activity of this layer is disrupted, incomplete mixing of food and dyspeptic complaints occur.

The middle circular muscle layer is the main generator of peristaltic waves. Rhythmic contractions of this layer move the food mass distally along the stomach. It is this muscle layer that thickens significantly in the pylorus area, forming a powerful pyloric sphincter **.** The pyloric sphincter tightly controls the amount of food passing from the stomach into the duodenum and prevents the regurgitation of intestinal contents into the stomach. When this mechanism is disrupted, reflux-duodenitis or evacuation insufficiency may develop.

The outer longitudinal muscle layer - provides the general contraction, extension and sliding movements of the stomach. This layer is important in maintaining the shape and position of the stomach, as well as in the transmission of peristaltic waves from top to bottom. The longitudinal muscle fibers work in coordination with the circular layer and ensure the continuous movement of the food mass.

the coordinated, reflex and rhythmic activity of these three muscle layers, the food mass is completely mechanically processed, brought to the state of chyme and then prepared for passage into the duodenum. Functional or morphological disorders of the muscle layer can lead to clinical conditions such as gastric atony, spasms, pylorospasm, and slowing of the evacuation process **.** Therefore, a deep knowledge of the anatomy and physiology of the muscle layer of the stomach is of great importance in gastroenterology and surgical practice.

5. Serous layer (tunica serosa)

The peritoneum is covered by the visceral layer of the peritoneum, which is composed of mesothelium cells . The serous fluid secreted by the mesothelium cells ensures the free and physiological movement of the stomach without friction with adjacent organs (liver, diaphragm, spleen, and transverse colon). This fluid, in addition to reducing friction, acts as a shock absorber, softening mechanical shocks between the organs in the abdominal cavity.

Mesothelial cells are not limited to mechanical protection, but also act as a biologically active layer : they regulate fluid and electrolyte metabolism, modulate immune responses and inflammatory processes by secreting cytokines and growth factors . In pathological conditions (perforation, infection, tumors), it is mesothelial cells that play an important role in the initiation of the peritoneal reaction, enhancing the release of inflammatory mediators.

The serous layer maintains the anatomical position of the stomach as an intraperitoneal organ , provides adaptation to changes in pressure in the abdominal cavity, and maintains the functional harmony of adjacent organs during gastric movements. Clinically, the serous layer determines the direction of spread of pathological processes (perforation, ulcer, tumors) into the abdominal cavity. When the stomach wall is perforated, peritonitis develops precisely through the serous layer, and peritoneal dissemination of tumors is also directly related to this layer. Therefore, a deep understanding of the structure and function of the serous layer is of great importance in surgical, oncological, and emergency medical practice.

CHAPTER II. Etiology, pathogenesis, treatment and prevention methods of gastritis

1. Etiology and pathogenesis of gastritis

The digestive system occupies a significant place in the general morbidity of the population. Also, these diseases are characterized by a tendency to relapse. Therefore, they usually last a long time. The number of diseases with gastritis and peptic ulcer disease, liver cirrhosis, cholecystitis, pancreatitis, tumors of the stomach and intestines is increasing day by day. This leads to a relatively high incidence of temporary or long-term disability among the population, and sometimes even death of patients.

Etiology and pathogenesis.

Gastritis is a polyetiological disease characterized by inflammation of the gastric mucosa, which occurs under the influence of a number of external and internal negative factors. Gastritis is also characterized not only by the occurrence of varying degrees of pathomorphological changes (atrophic or hypertrophic) and functional changes in the gastric mucosa and its glandular structures, but also by the spread of the pathological process to other layers of the stomach.

The following exogenous (eating disorders - quantity and quality of food, eating a lot in the evening, alcohol abuse, excessive use of spicy spices in cooking, toxicoinfections caused by non-compliance with sanitary and hygienic rules in the storage of food products and ingredients, the effects of salmonella, shigella, staphylococcus, etc., allergies to certain foods - eggs, spices, fruits, raspberries, strawberries, etc., the effects of drugs - acetylsalicylic acid, corticosteroids, pyrazolone derivatives, antibiotics, cardiac glycosides, etc.) and endogenous factors (infection - influenza, measles, autointoxication - kidney and liver failure, tissue breakdown in the body - burns, frostbite, radiation exposure, etc.) are of great importance in its origin.

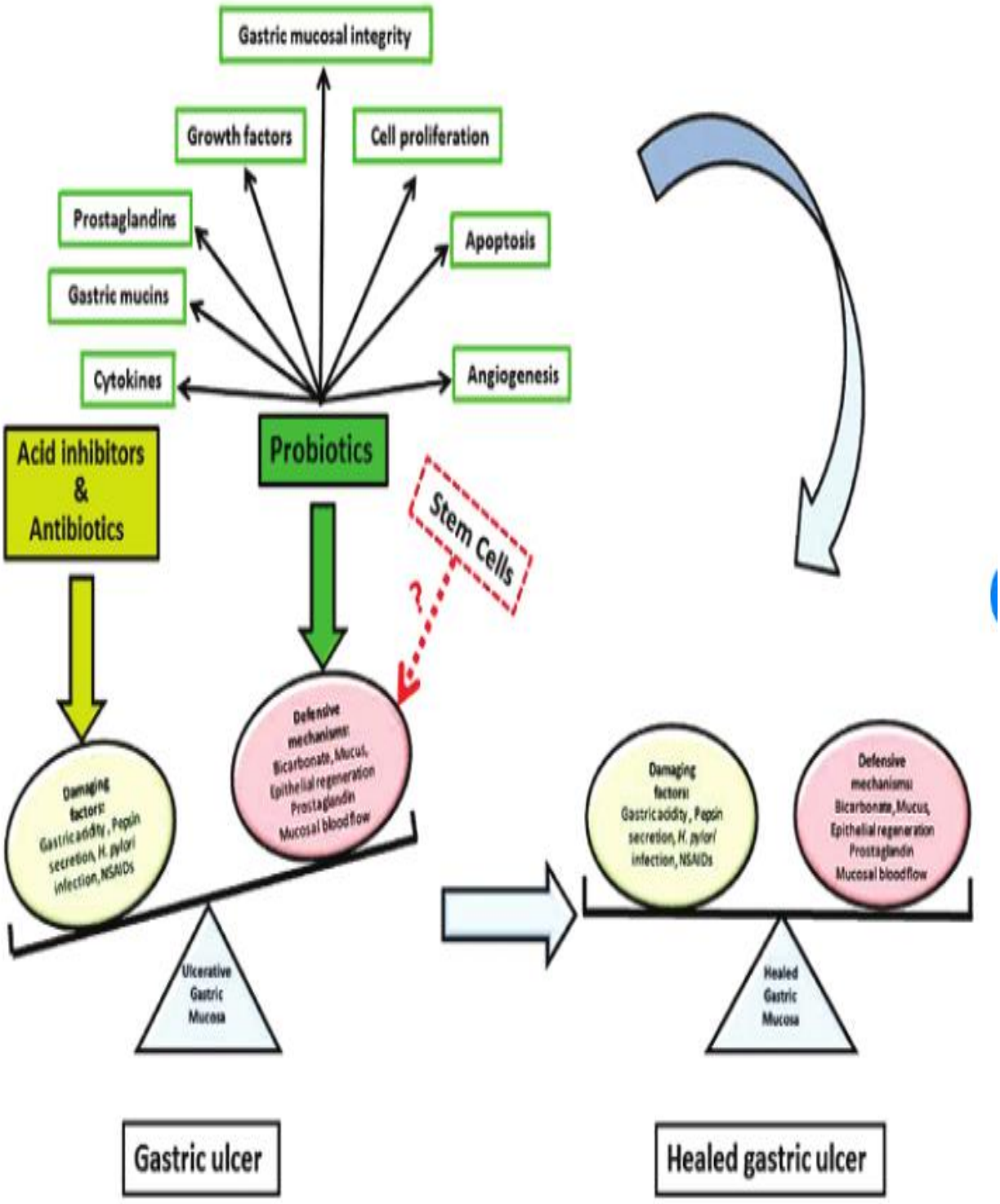
In a healthy person, the epithelium of the gastric mucosal wall undergoes rapid regeneration due to bicarbonate secretion and blood supply (Table 1).

Table 1. Differences between healthy and inflamed gastric mucosa

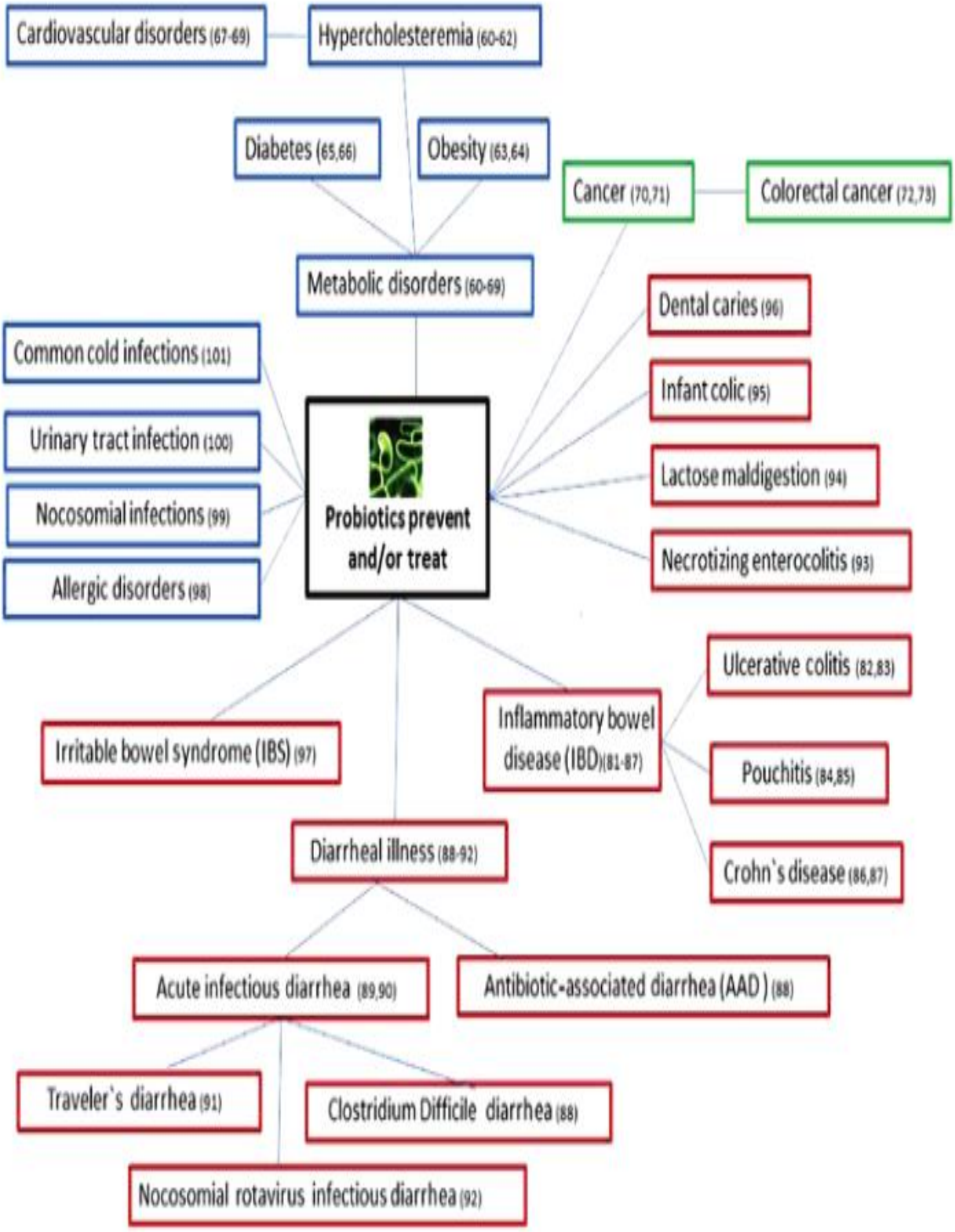
Characters/Characteristics	Healthy gastric mucosa	Inflamed stomach lining (gastritis)
Appearance (endoscopic)	Smooth, pink, uniform	Red, swollen, scaly, or eroded
Microscopic view	Normal glands, minimal inflammation	Inflammatory cells (neutrophils, lymphocytes), glandular atrophy
Secretion	Produces a normal acidic environment and mucus	Secretory activity is impaired, there may be a decrease or increase in acidity
Signs of injury	No.	Ulcers, erosions, microcracks are possible
Protein production	Normal	Sometimes it is disrupted (for example, in Menetrier's disease)
Recovery	Fast and efficient	If inflammation persists, recovery will be slower.
Symptoms of infection	No.	Often associated with H. pylori
Cancer risk	Very low	The risk increases if chronic and atrophic

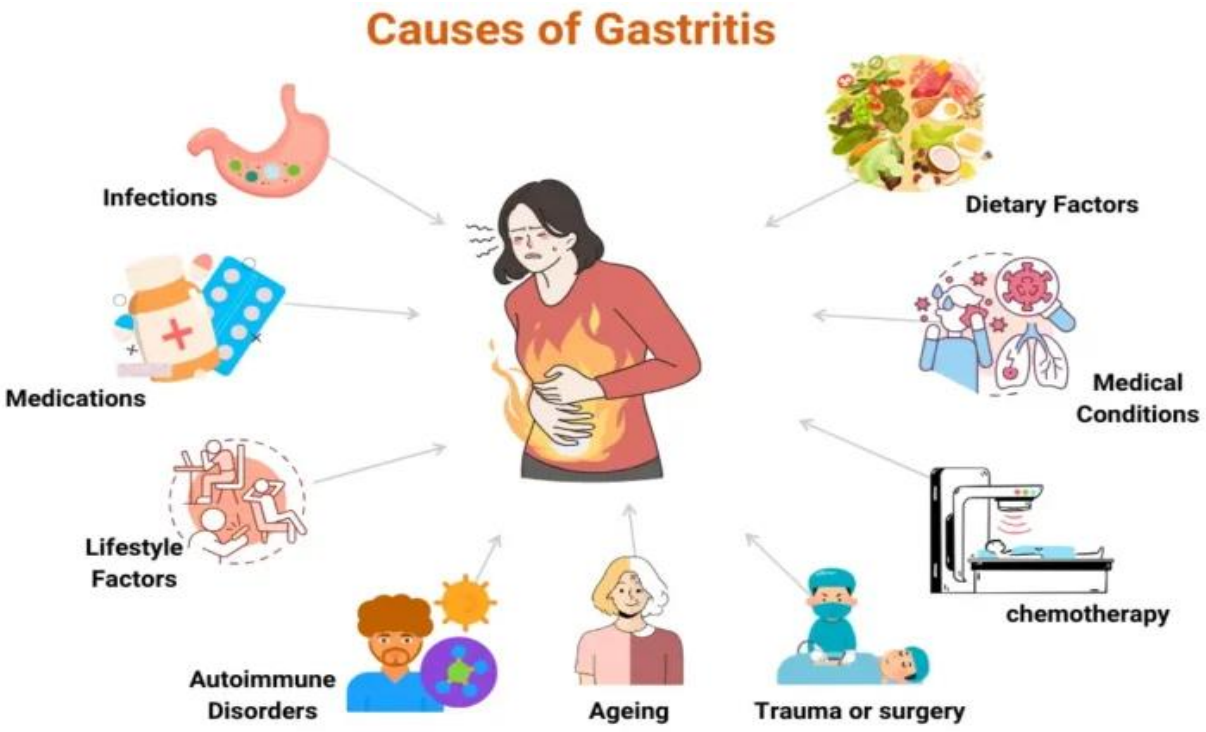
Helicobacter pylori belongs to the group of gram-negative bacteria, has a spiral and twisted - S-shaped. They are intensely colored and have 4-6 tails on one side of their body, which allows them to move freely in gastric juice and attach to its mucous membrane. Helicobacter pylori Vibrios secrete a number of substances that have a direct cytotoxic effect on the mucous membrane. As a result,

inflammation is observed in it, and later the pathological process spreads to the deeper parts of the gastric mucosa, leading to atrophy of the glandular apparatus. Thus, Helicobacter pylori In the late stages of gastritis caused by Vibrio, chronic atrophic multifocal pangastritis develops, and its clinical manifestations are sharply different from those of primary gastritis caused by Vibrio.



It should be noted that the gastric mucosa is infected with Helicobacter pylori. Infection with H. pylori not only causes the two types of gastritis mentioned above, but also causes acute Helicobacter gastritis, giant hypertrophic chronic gastritis (Menetrie's disease), and peptic ulcers.





2. Types of gastritis and their clinical picture

According to statistics, approximately 80-90% of people experience at least one episode of the disease during their lifetime. In old age, up to 70-90% of people suffer from various forms of gastritis.

According to the World Health Organization, 50% of the world's population suffers from chronic gastritis. Gastritis accounts for approximately 35% of digestive system diseases, and 80-85% of stomach diseases.

In professional medicine, other classifications of gastritis are distinguished, including those depending on the type of pathogenesis:

1. Autoimmune gastritis (type A);
2. Exogenous gastritis (type B) caused by *Helicobacter pylori;*
3. Mixed gastritis (type A + B);
4. Gastritis (type C) induced by NYQP, chemicals, and bile;
5. Special forms of gastritis;
6. Gastritis that occurs against the background of decreased and increased secretion of hydrochloric acid;
7. Other forms of morphological and functional manifestations of gastritis.

In addition, there are the following types of gastritis depending on the damage to the gastric mucosa (Figure 1):

1. Superficial (catarrhal): inflammation of only the mucous membrane.
2. Erosive: erosions on the mucosa.
3. Atrophic: shrinkage of the mucosa.
4. Phlegmon: Inflammation of all layers of the stomach, often due to ulcers or cancer.

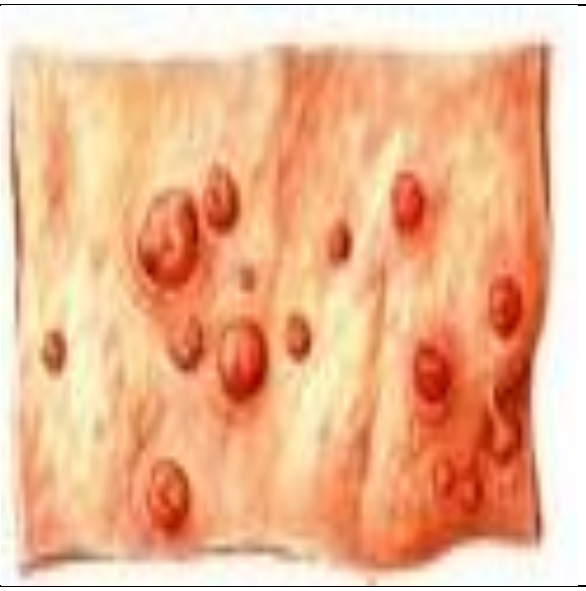
<table>
<tr><td colspan="2">Types of gastritis according to the damage to the gastric mucosa</td></tr>
<tr><td>  </td><td>  </td></tr>
<tr><td>Atrophic gastritis</td><td>Phlegmonous gastritis</td></tr>
<tr><td>  </td><td>  </td></tr>
<tr><td>Erosive gastritis</td><td>Polyposis gastritis</td></tr>
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Figure 1. *Types of gastritis according to the damage to the gastric mucosa*

Two types are distinguished according to the course of the disease:

1. sharp

2. chronic gastritis.

The most modern and complete classification of gastritis is the etiological classification proposed in the Kyoto global consensus in 2015. Its necessity is due to the development of highly accurate endoscopic diagnostic methods for gastritis, as well as the desire to develop a classification of gastritis based on a general principle, taking into account the currently achieved theoretical knowledge (UDD 1, UUR A).

Kyoto etiological classification of gastritis

► Gastritis associated with *H. pylori.* ► Gastritis induced by drugs. ► Autoimmune gastritis. ► Stress-related gastritis. ► Specific forms of gastritis: a. Allergic gastritis. b. Gastritis due to biliary reflux. c. Lymphocytic gastritis. d. Menetrie's disease. e. Eosinophilic gastritis.

► Infectious gastritis:

1. Gastric phlegmon. 2. Bacterial gastritis:
   a. Called with *H. pylori* ;
   b. Enterococci.
3. Mycobacterial gastritis:
   Related to tuberculosis;
   b. Non-tuberculosis (non-tuberculosis).
4. Second stage syphilitic gastritis.
5. Viral gastritis:
   a. cytomegalovirus;

b. Enterovirus.

6. Fungal gastritis (caused by fungus):
   a. Mucormycosis;
   b. Gastric candidiasis;

c. Gastric histoplasmosis.

7. Parasitic gastritis:

a. Gastric hypoxia;

b. Gastric cryptosporidiosis;

c. Gastric strongyloidiasis.

► Gastritis associated with other diseases:

1. Crohn's disease. 2. Sarcoidosis. 3. Vasculitis.

► Gastritis due to external factors:

1. Alcohol-related gastritis. 2. Radiation gastritis. 3. Chemical gastritis.

► Gastritis of unknown etiology, but characterized by certain endoscopic or pathological signs:

1. Superficial gastritis:

a. Sharp;

b. Chronic.

2. Acute hemorrhagic gastritis.

3. Chronic atrophic gastritis:

a. Light;

b. Average ;

c. Heavy.

4. Metaplastic gastritis.

5. Granulematous gastritis. 6. Hypertrophic gastritis.

► Other gastritis:

• Chronic gastritis not classified elsewhere.

3. Acute gastritis.

The pathogenesis of the disease is inextricably linked to the factors that cause it and develops in two different forms (Figure 2).

In exogenous acute gastritis, the etiological factor directly affects the gastric mucosa, causing its inflammation, and secondary changes are added later.

Acute exogenous and endogenous and depending on the changes in the gastric mucosa, its superficial (simple), erosive, and corrosive types are distinguished.

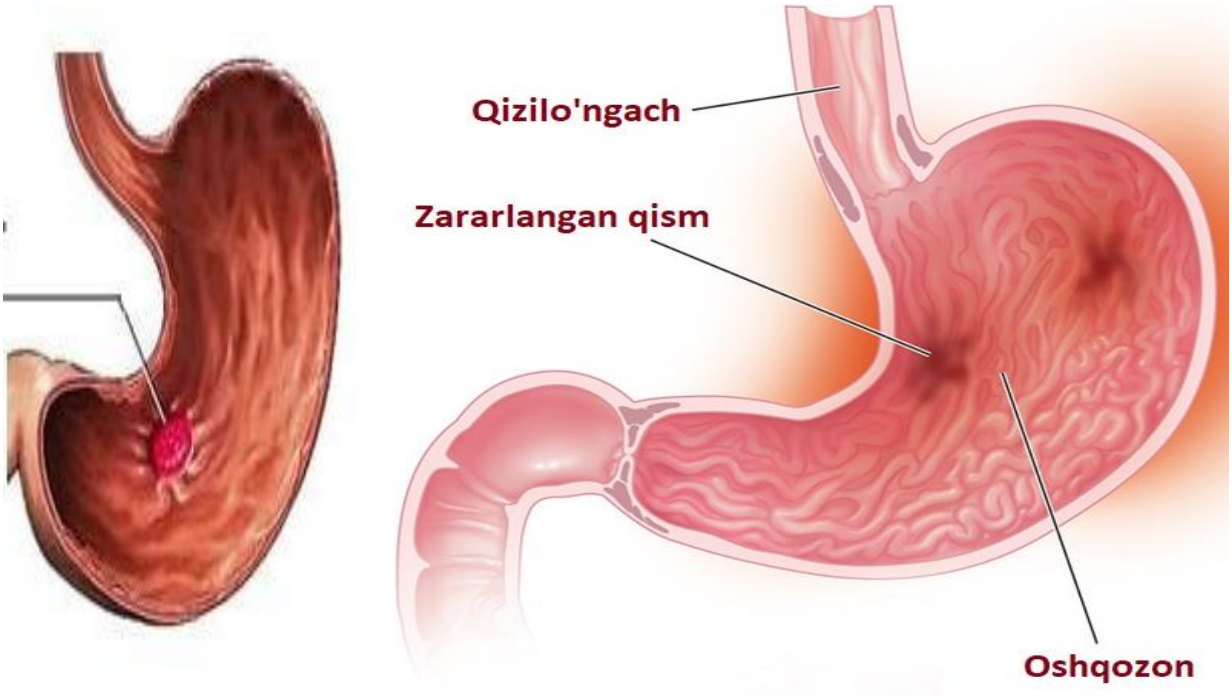


Figure 2. *Acute gastritis.*

In endogenous acute gastritis, the causative agent gradually causes changes in the gastric mucosa by hematogenous means, and the patient's condition is dominated by symptoms of general intoxication. In some cases, both pathogenetic mechanisms are actively involved in the development of the disease.

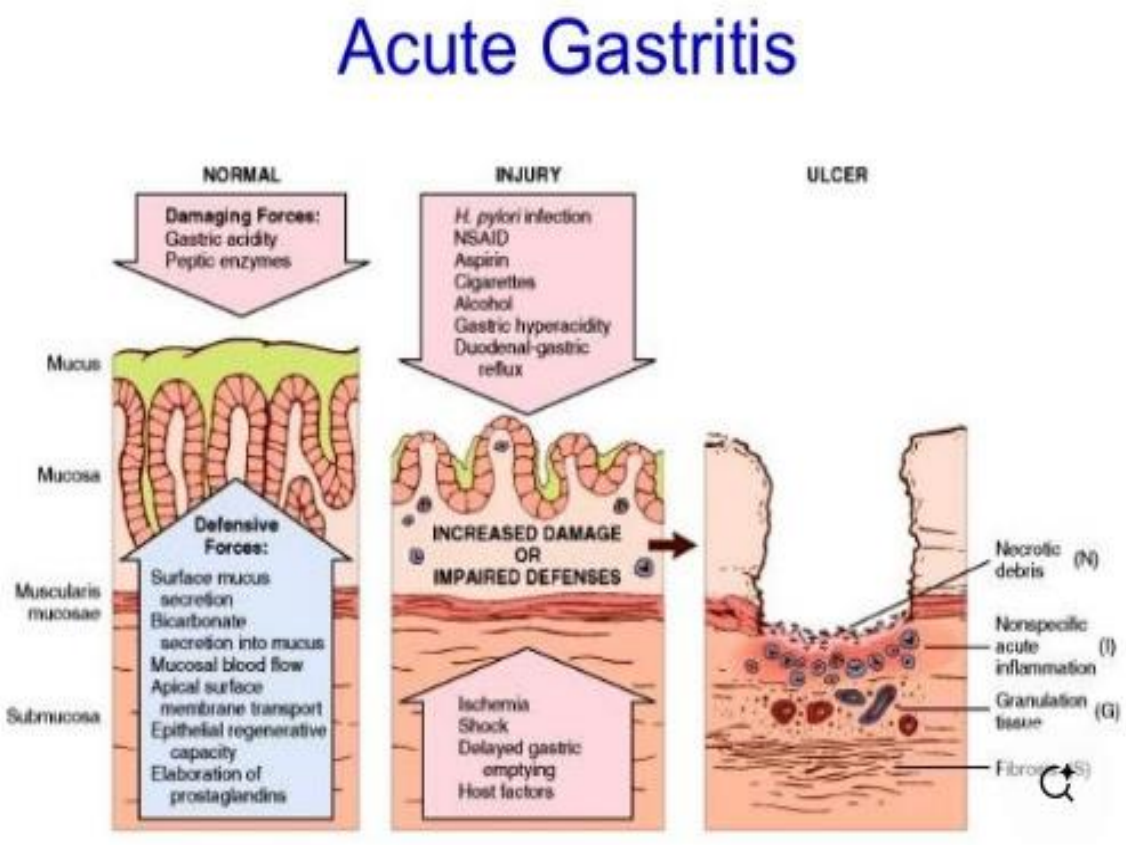
Clinical picture. The clinical manifestations of acute gastritis depend on its severity and clinical course. In acute exogenous superficial (simple) gastritis, patients experience nausea, vomiting of mucus and undigested food, sometimes with bile, a feeling of indigestion in the epigastric region, abdominal pain, an unpleasant taste in the mouth, general weakness, pale skin, tongue coating, in some severe cases,

a decrease in blood pressure, and signs of enteritis are observed. In most cases, the disease lasts an average of 5-6 days.

In the development of acute corrosive gastritis, the effect of high concentrations of acid or alkali on the gastric mucosa is important. As a result, necrosis of the mucosa is observed. In this form of acute gastritis, patients experience severe pain in the mouth, esophagus and epigastric region, mucous and bloody vomiting, in severe cases, numbness and later symptoms of "acute abdomen".

Acute gastritis It develops due to inflammation of the stomach lining, often caused by the bacterium Helicobacter pylori. Its causes are as follows:

- Food poisoning infections (salmonellosis).
- Overeating.
- Alcohol abuse and smoking.
- Exposure of the mucous membrane to drugs (analgesics, cytostatics).
- Food allergies.
- Stress, fatigue, chronic insomnia.



4. Chronic gastritis

Chronic gastritis is a chronic disease characterized by inflammation and dystrophic changes in the gastric mucosa, which leads to impaired physiological regeneration processes and glandular atrophy, as well as changes in its motor, secretory, and incretin activity (Figure 3).

One of the most common diseases of the digestive system is chronic gastritis, which can occur both as an independent disease and as a concomitant disease. Chronic gastritis is a common disease among the population and occurs more often (50-80%) in adults.

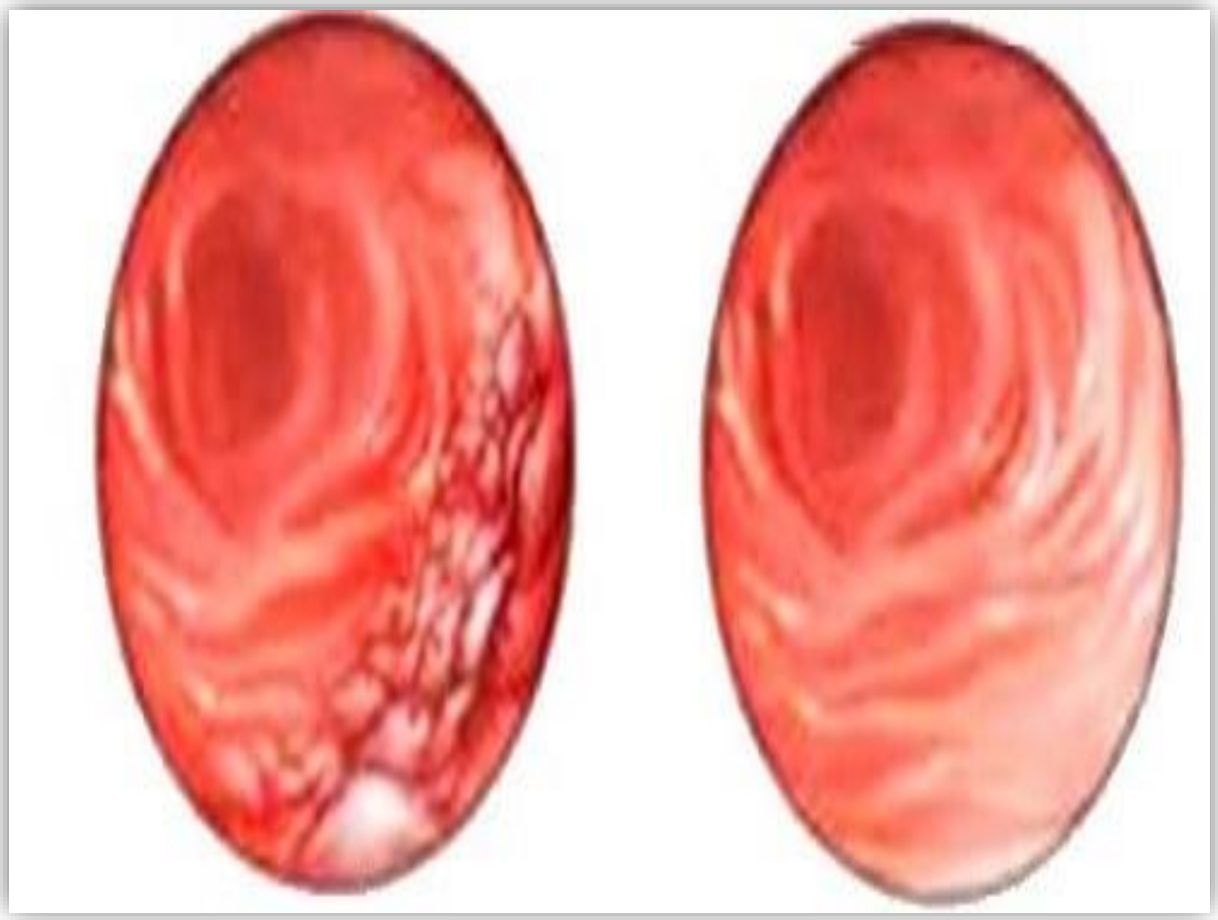


Figure 3. *Normal gastric mucosa in chronic gastritis mucous membrane*

The autoimmune type of gastritis accounts for approximately 9%, the Helicobacter pylori-associated type for 70%, sycopathic pangastritis for 12%, reflux gastritis for 5%, lymphocytic type for 2%, and the rest for another 2% (Figure 4).

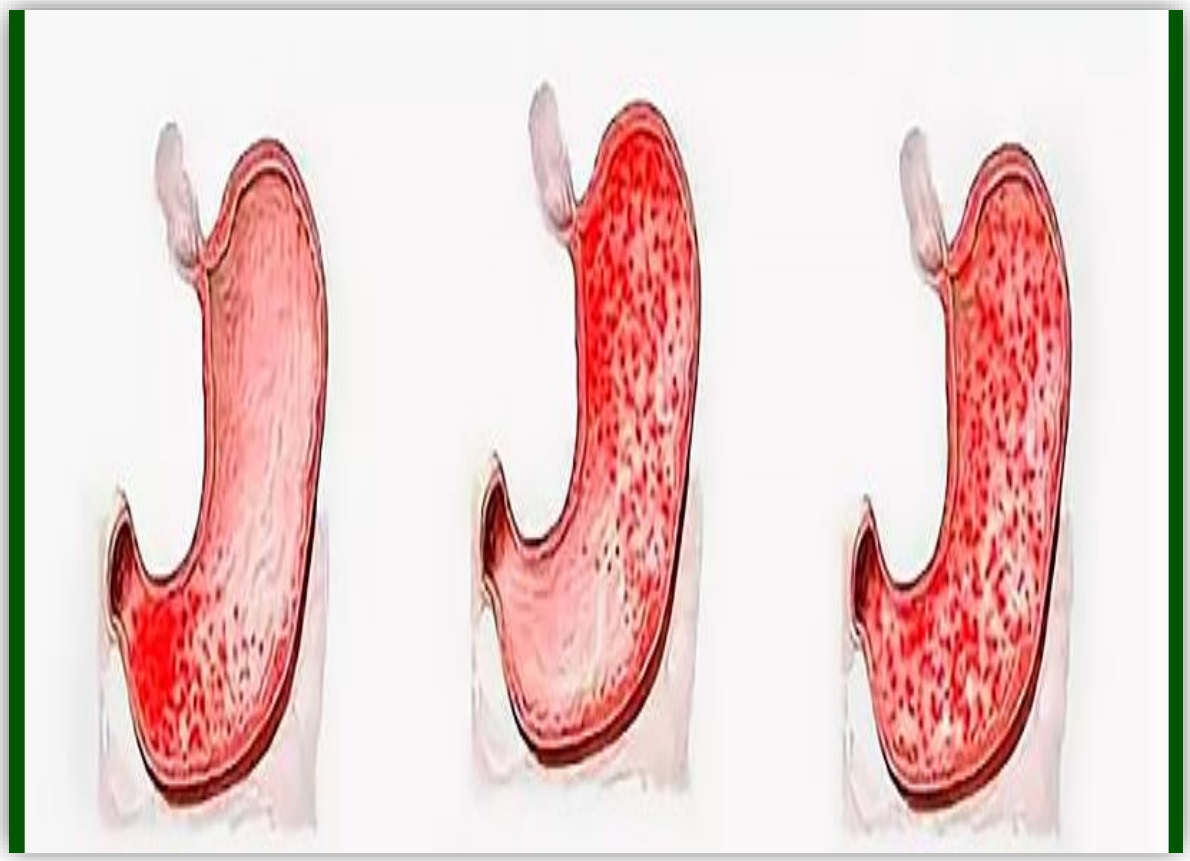


Figure 4 *. Gastritis type A Gastritis type B Multifocal gastritis (pangastritis)*

Chronic gastritis is a polyetiological disease, the following factors are important in its etiology:

1. Dietary causes - violation of the order and quality of food intake (long intervals between meals, frequent eating without chewing it well, direct negative effects of hot liquids and solid foods on the gastric mucosa, abuse of spicy spices and alcohol, smoking).

2. Helicobacter pylori - confirmed in numerous bacteriological, histological and immunological studies, is one of the important factors causing dystrophic-inflammatory processes in the stomach. This is confirmed by the disappearance of symptoms of the disease and the occurrence of remission as a result of the

elimination of the pathogen with drugs. Infection source sick forbidden or house animals is considered .

3. Due to endogenous, autoimmune and hereditary determination, antibodies are formed against gastric mucosal cells (autoimmune gastritis) . This is due to the histological affinity of HLA, Bg, DR3, DR4 antigens to gastric mucosal cells.

4. It is also caused by reflux of bile into the gastric mucosa and conditions such as gastroenterostomies, vagotomy, cholecystectomy, and duodenal obstruction following gastric resection.

5. The gastric mucosa is also damaged by prolonged and improper use of medications and certain agricultural products (glucocorticosteroids, acids, rauwolfia, etc.).

6. Hypovitaminosis

7. Chronic infectious diseases , metabolic diseases, endogenous intoxications

Other diseases of the gastrointestinal tract, such as chronic pancreatitis and chronic cholecystitis, which have a reflex effect on the gastric mucosa.

Catarrhal gastritis is associated with malnutrition and mild food poisoning. Fibrinous and necrotic gastritis usually occur as a result of poisoning with heavy metals, saturated acids and hydroxide salts. Phlegmonous gastritis is associated with mechanical injury to the stomach wall. Chronic reactive gastritis, as noted above, is often accompanied by gastroduodenal reflux and is accompanied by erosions and hemorrhages.

In 1990, at the 9th International Congress of Gastroenterologists (Sydney, Australia), a new classification of gastritis, called the "Sydney System", was adopted, according to which the disease is divided into 3 main groups:

1. Acute (neutrophilic infiltration only);

2. Chronic (active phase - lymphoplasmacytic infiltration accompanied by granulocytes);

3. Special forms (granulomatosis (in Crohn's disease, sarcoidosis and tuberculosis), giant hypertrophic - Menetrie's disease, eosinophilic (in bronchial asthma, eczema and atopic dermatitis), lymphocytic, reactive-reflux gastritis).

Currently, the classification of chronic gastritis based on the "Sydney system" adopted in Houston in 1996 is used in practice (Table 2).

Table 2. Classification of chronic gastritis (Houston 1996)

Types of gastritis	**Etiological factors**	**Synonyms (according to the previous Sydney classification)**
Non-atrophic	Helicobacter pylori and other factors	-superficial ; -chronic antral gastritis - type B - hypersecretory gastritis
Atrophic-autoimmune	Immune mechanisms	A gastritis ; Diffuse gastritis of the stomach; Gastritis of the gastric body is accompanied by vitamin B12 deficiency anemia and decreased secretion.
Atrophic-multifocal	Helicobacter pylori, eating disorders .	Mixed gastritis A and B
Gastritis caused by chemical exposure	Chemical exposure factors – Bile (duodenogastric reflux) fluid, NYQV, glucocorticosteroids, etc.	Type C reactive gastritis; reactive reflux gastritis
Radiation	Light damage	
Lymphocyte	Idiopathic, immune mechanisms, gluten, Helicobacter pylori	Gastritis occurring with celiac disease

Granulomatosis	Crohn's disease, sarcoidosis, Wegener's granulomatosis	Borderline granulomatosis
Eosinophilic	Idiopathic, food allergies, and other allergens	Allergic
Infections	Bacteria (infections other than Helicobacter pylori), fungi, parasites	
Giant hypertrophy	Menetrie's disease	

Clinical picture. The clinical manifestations of chronic gastritis depend on the etiological factor that caused it and the histological changes in the gastric mucosa.

Chronic non-atrophic gastritis (type B) is most often found among young people. The disease is most often manifested by pain, indigestion, and sometimes asthenoneurotic syndrome. The pain is characterized by various symptoms, sometimes acute attacks. The cause of the appearance is associated with the consumption of spicy, fried, fatty foods or their poor chewing, and can last up to 1-1.5 hours and disappear on its own. The pain is most often localized below the sphenoid or in the pyloroduodenal region, sometimes observed on an empty stomach. In almost all cases, the pain syndrome can be eliminated by administering antispasmodics.

Along with pain, patients are also bothered by dyspeptic symptoms (feelings of heaviness and fullness in the stomach, heartburn, sour belching, and, in rare cases, nausea and vomiting).

Asthenoneurotic syndrome is characterized by symptoms such as high levels of emotionality, rapid mood swings, and rapid fatigue.

Although the results of objective examination of patients are nonspecific and have limited diagnostic value, some of the data obtained allow us to distinguish chronic gastritis from gastric and duodenal ulcers, their cancer, and functional dyspeptic changes.

during acute colic-like pains in the stomach, patients take a forced position. When examining their tongue, the root area is covered with a white or yellowish-white coating. When palpating the abdomen, localized pain (sometimes quite intense) is detected in the epigastric and pyloroduodenal areas, unlike peptic ulcer disease, tension of the muscles of its anterior wall is not detected.

During deep sliding palpation, pain symptoms are noted due to the presence of spastic contractions in various parts of the colon (sigmoid, ascending, descending, and sigmoid colon), which indicates the presence of colonic impaction syndrome.

Abdominal auscultation reveals normal or slightly increased intestinal contractions. The size of the liver and spleen is usually unchanged in patients.

Some patients have symptoms of asthenoneurotic syndrome, such as skin dermographism, arterial hypotension, pulse rate variability, cold and wet palms, and increased salivation, which are clearly visible and persistent during objective examination. They indirectly indicate dysfunction of the autonomic nervous system, in particular, the predominance of the tone of the parasympathetic system.

Hypersecretion of gastric juice is noted in 60% of patients, and in 30% of cases, its normal level is noted.

In chronic autoimmune gastritis (fundal type A), atrophic processes occur early as a result of damage to the mucous membrane of the fundus and body of the stomach (Fig. 5). This weakens gastric acidity and pepsin secretion. The disease is more common in middle-aged and elderly people. Patients experience symptoms such as heaviness in the upper abdomen after eating, a feeling of fullness in the stomach, belching with food and air, and an unpleasant taste in the mouth. They usually have decreased appetite, flatulence, and abdominal swelling. On general examination, an atrophic - "lacquered tongue" is observed, and during exacerbations it is covered with a white coating.

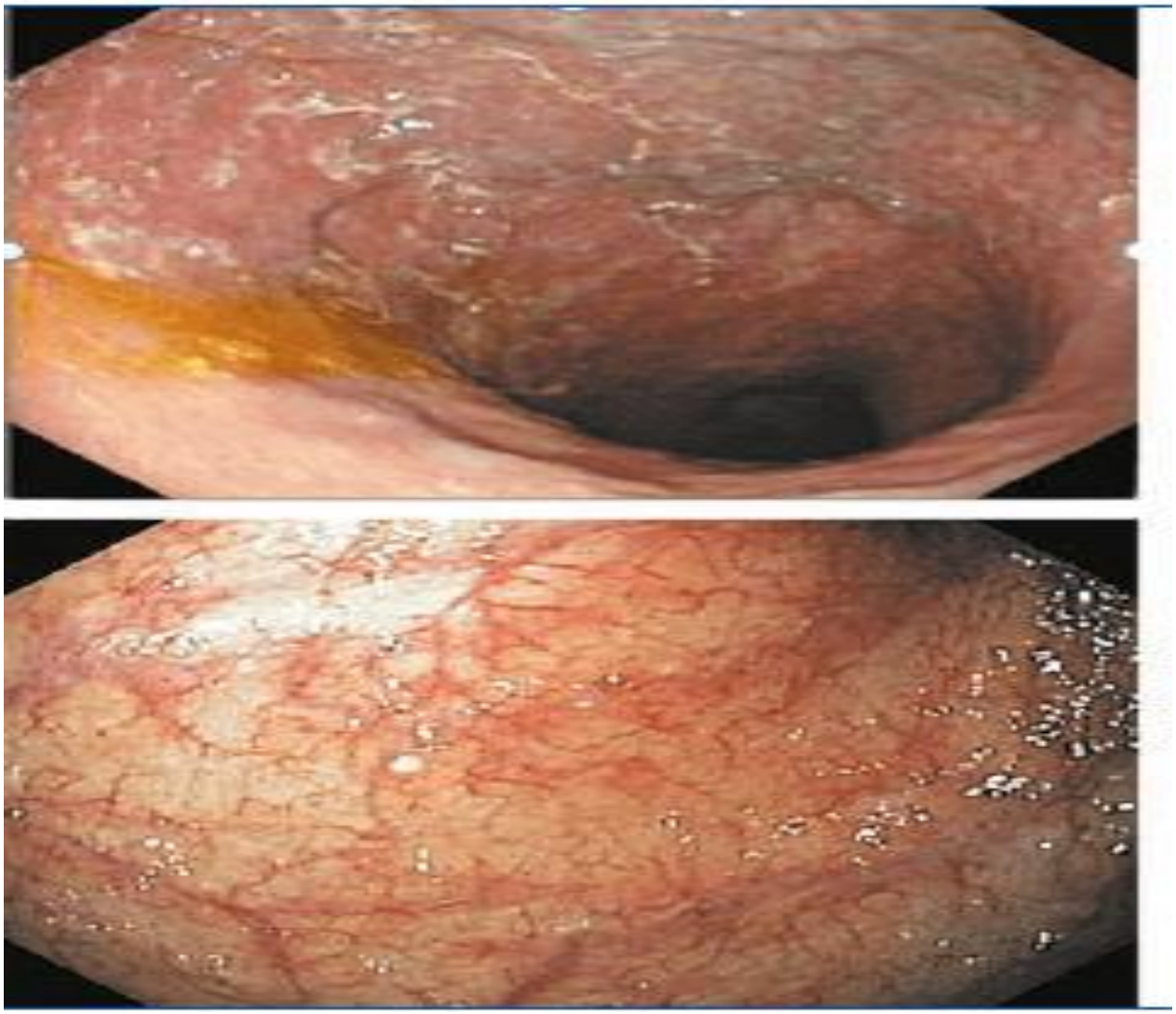


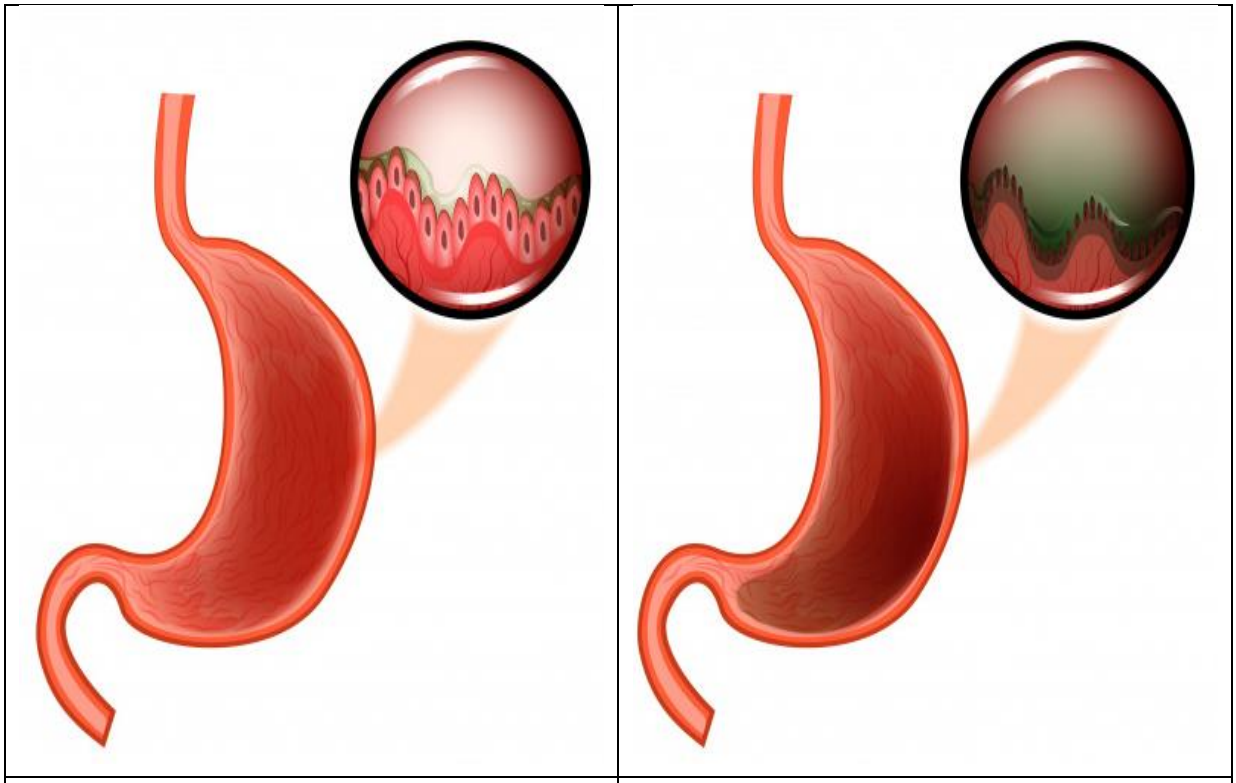
Figure 5. *Appearance of the gastric mucosa in chronic autoimmune gastritis.*

As the disease progresses, atrophic processes in the gastric mucosa and secretory insufficiency intensify, patients develop a tendency to diarrhea and a decrease in body weight is observed. Due to the deficiency of intrinsic factor of Castle, vitamin B_{12} deficiency anemia develops, which causes weakness, rapid fatigue, and later dizziness, shortness of breath and tachycardia are added. Sensitivity in the tongue (paresthesia) is impaired.

Due to secretory insufficiency in the stomach and pancreas and accelerated motility of food particles, absorption and pre-digestive processes in the small intestine are impaired, leading to symptoms of polyhypovitaminosis and decreased resistance to infections in some patients (mostly in the elderly).

On physical examination, patients are usually thin, with pale skin and visible mucous membranes, dry skin, brittle nails, and cracks in the corners of the mouth. Abdominal examination reveals epigastric pain that radiates to the lower border of the stomach, at rest, and rumbling and pain on palpation of the large intestine.

Atrophic multifocal or idiopathic pangastritis (types A and B). Most patients with this type of gastritis have a history of long-term irregular eating habits (more salty, spicy, and rough) and low consumption of fruits and vegetables (Figure 6).



healthy stomach lining	atrophic gastritis in the stomach damaged mucous membrane

Figure 6. *Atrophic gastritis.*

Also, in patients with gastritis of type A and B, infected with Helicobacter pylori bacteria and suffering from antral gastritis, after 15-20 years, pangastritis occurs and is clinically manifested as a result of an antral expansion of these bacteria from the mucosal epithelium of the antral part of the stomach. The disease occurs more often among the middle-aged and elderly population and is asymptomatic in the early stages. However, in most cases, patients complain of pain in the epigastric

region, dyspeptic changes (feeling of heaviness in the stomach, belching, nausea, a "metallic" taste in the mouth, decreased appetite, etc.), intestinal dyspepsia (flatulence, diarrhea) and weight loss. The predominant atrophic processes in the gastric mucosa and the weakening of the secretory activity of its glands cause a change in the pain syndrome.

Pangastritis is characterized by the appearance of long-lasting and dull pain during or shortly after eating. Patients complain of a feeling of heaviness and fullness in the stomach area even after eating a small amount of food. In other words, patients are more bothered by dyspeptic changes. They have complaints such as loss of appetite, belching with food and air, metallic taste in the mouth, nausea, rumbling and heaviness in the stomach, and changes in bowel movements. In most cases, astheno-neurotic syndrome occurs - increased weakness, fatigue, irritability, and a tendency to depression.

During the general examination, weight loss, pale skin, cracks in the corners of the mouth are detected. The tongue is usually covered with a white coating, the abdomen is distended due to pronounced flatulence. On palpation, a vague and diffuse pain is noted in the epigastric region. The gastric border is shifted downward. On palpation of the large intestine, rumbling is often heard due to the presence of a large amount of air and fluid retention.

of reactive reflux gastritis (type C) is associated with damage to the gastric and duodenal mucosa (Fig. 7). It most often occurs due to gastric resection, vagotomy, cholecystectomy, pyloric insufficiency, and chronic intestinal permeability disorders.

Patients with lesions of the pyloric part of the stomach experience pain and dyspeptic changes. Pain in the epigastric and pyloric regions sometimes (in the presence of erosions in the mucous membrane of the pyloric part) becomes intense and sharp.

They appear 1–1.5 hours after eating or taking nonspecific anti-inflammatory drugs and quickly disappear with the help of antispasmodics.

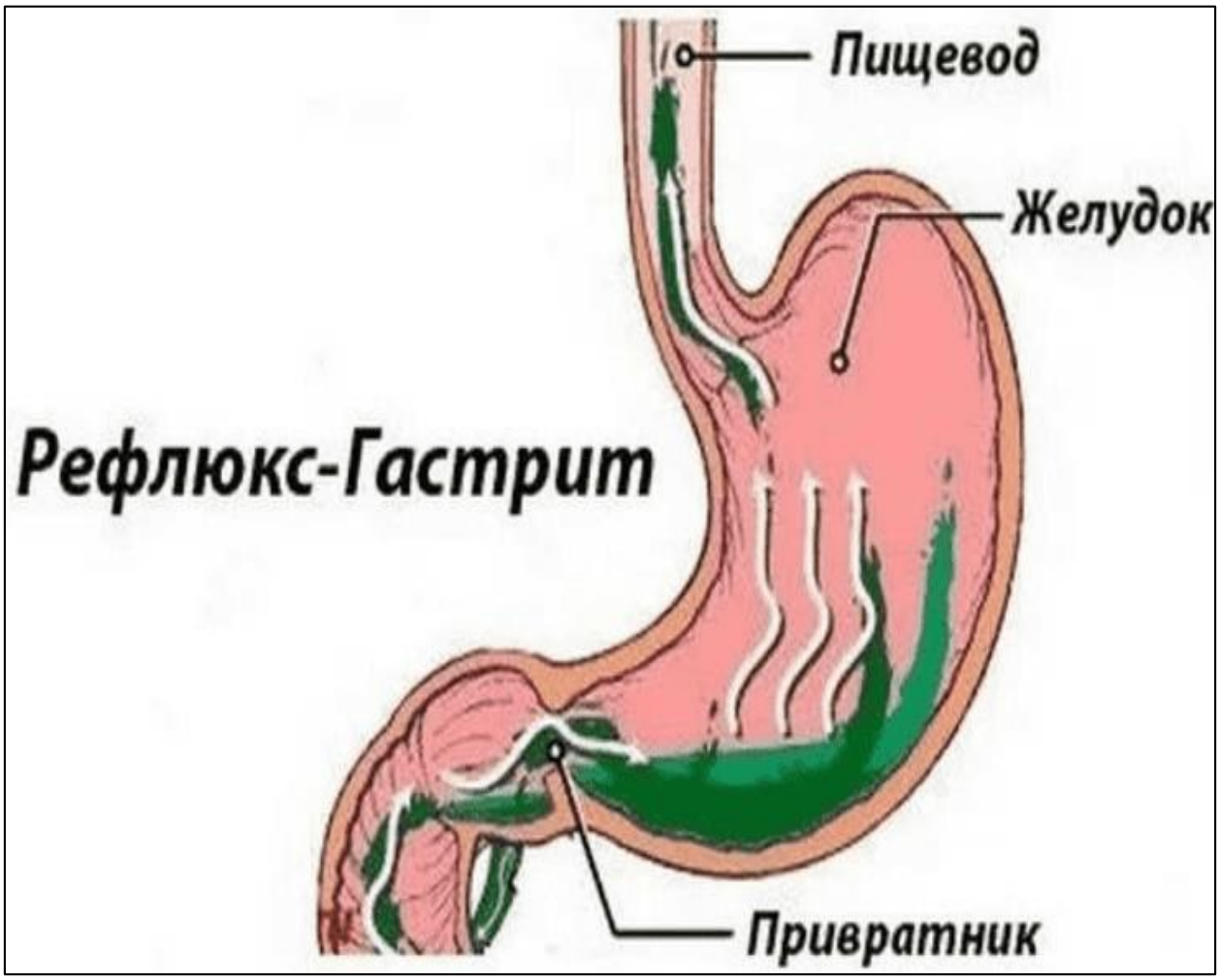


Figure 7. *Reactive-reflux gastritis (type C)*

This indicates that they are spastic in nature. Some patients experience intractable vomiting of bile, a bitter taste in the mouth, weight loss, and signs of anemia.

reactive gastritis, the damage to the pyloric mucosa is erosive, and sometimes they may experience coffee-colored vomiting or black tarry stools (melena) due to recurrent bleeding. Over time, patients develop chronic iron deficiency anemia.

lymphocytic gastritis, erosions in the gastric mucosa and pronounced lymphocytic infiltration of its epithelium are detected. In a healthy person, there are 3-5 lymphocytes per 100 epitheliocytes. In histological examination of a special

plate of the gastric mucosa obtained using gastrofibroscopy, this ratio is at least 30 to 100 and more in lymphocytic gastritis. These changes are also observed in gluten enteropathy in most cases.

Giant hypertrophic gastritis – Menetrie's disease (giant fold gastritis) is rare in practice and is more often associated with allergic conditions. Patients have a tendency to have protein in the blood (50-55 g/l), a decrease in the secretion of gastric juice, and a decrease in secretory activity. They complain of cramping pains in the upper abdomen after eating and a feeling of heaviness in the epigastric region (Fig. 8).

	**Healthy gastric mucosa**
	**Menetrie's disease -** rare, chronic stomach disease mainly gastric mucosal hyperplasia (growth) and protein-losing gastropathy is described by. - **Endoscopy** : Very large and abnormal folds are visible - **Biopsy** : Foveolar cell hyperplasia, deep glandular atrophy - **Laboratory tests** : Low albumin levels in the blood (a sign of protein loss)

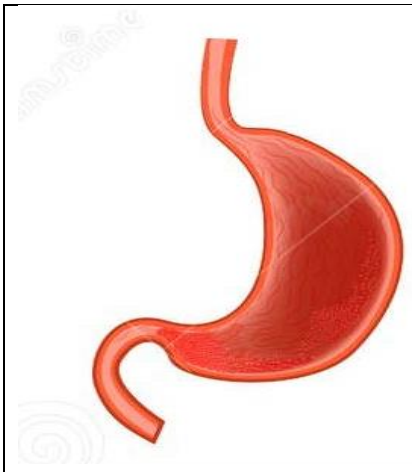
	**Granular form of hypertrophic gastritis** This is a chronic inflammatory disease of the gastric mucosa, characterized by swelling (hypertrophy) of the mucosa and the formation of small nodules (granules). This form is called hypertrophic gastritis. It is a relatively mild but long-lasting type.
	**Nodular hypertrophic gastritis** is a disease characterized by inflammation and thickening of the gastric mucosa, with wart-like or nodular growths and changes on its surface. It is one of the rare, chronic forms.
	**Polypous gastritis is a form of** chronic inflammatory disease characterized by numerous polyp-like growths or protrusions in the gastric mucosa.

Figure 8. *Giant hypertrophic gastritis*

In some cases, vomiting, diarrhea are observed, and a decrease in appetite, sometimes leading to anorexia. Body weight can decrease by 10-20 kg. In 25-40% of cases, peripheral edema is observed, associated with decreased secretion of gastric juice and a decrease in blood proteins. Approximately 10% of patients with hypertrophic gastritis develop gastric carcinoma.

5. Diagnosis of gastritis

The following tests are performed to diagnose gastritis:

1. Ultrasound examination of the stomach.
2. Esophagogastroduodenoscopy (examination of the stomach using a gastroscope).
3. Gastric mucosal biopsy.
4. Gastric secretion test.
5. Clinical blood analysis.
6. Abdominal radiography.

Examination using esophagogastroduodenoscopy is crucial in the diagnosis of gastritis. It allows not only to study anatomical changes in the gastric mucosa, but also to determine its functional state, and, if indicated, to take a biopsy and sometimes to determine the etiology of the disease.

In recent years, gastrofibroscopy has also been used to perform certain procedures - in particular, to administer medications to the affected area, stop bleeding, treat with low-intensity laser beams, remove foreign bodies, perform polypectomy, and evaluate the effectiveness of treatment.

As mentioned above, esophagogastrofibroscopy is widely used in the diagnosis of diseases of the gastrointestinal tract, especially gastritis. However, there are a number of contraindications to its conduct, which are listed below, and the treating physician should be well aware of them.

1. Significant narrowing of the esophagus;
2. The presence of pathological processes in the thoracic cavity (tumor, mediastinitis, aortic aneurysm);
3. Pronounced kyphoscoliosis;
4. High-grade heart and respiratory failure;
5. Acute circulatory disorders in the brain;
6. Unstable angina, acute myocardial infarction, unstable angina class III-IV.

In type B gastritis, endoscopy reveals areas of diffuse and patchy hyperemia and edema in the antrum of the stomach. In some cases, erosion of the mucosa is usually observed in areas of obvious inflammation, and sometimes in the body of the stomach. Also, in some patients, submucosal hemorrhages, hyperplasia of the

folds, slowing of the movement of food from the stomach, spasm of its antrum and pyloric cup are detected.

In chronic pangastritis, endoscopy reveals smoothing of the folds of the body and antral part of the stomach, thinning of the mucosa, pronounced vascular pattern, and erosions in its body. Morphological examination of the mucosa reveals areas of atrophy, mononuclear infiltration, intestinal metaplasia, and dysplasia. This type of chronic gastritis has a higher incidence of polyps and gastric cancer than others.

In giant cell gastritis, giant folds resembling brain tumors and a large amount of mucus can be seen in the stomach (Menetrie's disease). The mucosa is easily mobile, and areas of eroded hemorrhage are noticeable. If intestinal metaplasia is observed during histological examination, patients should be re-examined every 6-12 months with a biopsy by EFGDS. The pit is predominantly mucous, has an enlarged and uneven appearance, and sometimes cysts of various sizes are detected. There is an absolute and relative decrease in the number of chief and repair cells of the mucosa and the appearance of mucous-producing cells in their place.

In EGFDS, granulomas containing multinucleated cells (epithelioid) are detected in the gastric mucosal lamina propria of patients with granulomatous gastritis.

Type C gastritis is characterized by focal hyperemia, swelling of the mucosa, yellow-tinged gastric juice, a slight opening of the pyloric valve, and in some cases, reflux of intestinal contents into the stomach.

The severity of the pathological process is assessed based on the following 5 morphological changes detected in a biopsy taken from the gastric mucosa:

- Degree of inflammation;
- Inflammatory activity;
- Area of atrophy in the mucosa;
- The presence of intestinal metaplasia;
- The degree of infection of the mucous membrane with Helicobacter pylori.

In practical medicine, in addition to esophagogastroscopy, X-ray examination of the stomach and duodenum is performed using several of the following methods to diagnose chronic gastritis:

- General abdominal radiography;
- Examination of the stomach using artificial contrast by giving the patient an aqueous solution of barium sulfate;
- Gastric pneumography (filling it with gas);
- Relaxation duodenography (injection of barium into the intestine by inducing hypotension using pharmacological drugs);

Among the listed examinations, the most widely used in practice is the examination of the stomach and duodenum with the use of an aqueous solution of barium sulfate, after which the patient drinks it. Although it is possible to assess the condition of the entire gastrointestinal tract, including the small and large intestines, detailed information is obtained about the appearance of the mucous membranes of the esophagus, stomach and duodenum. Changes in the surface of the gastric mucosa, impaired peristalsis and tone, and organ deformation are X-ray signs of chronic gastritis. Usually, thickening of the mucous membrane folds is observed. In the late stages of the disease, more often in atrophic gastritis, a coarse-cellular and uneven pattern of the mucous membrane is noticeable. When the antral part of the stomach is damaged (type V antral Helicobacter gastritis or type AV pangastritis), rigidity and thickening of this area, as well as impaired peristalsis, and in some cases even deformation, are observed. Also, a large amount of fluid accumulation in the stomach lining (hypersecretion) is one of the X-ray signs characteristic of gastritis.

The results of histological analysis of a biopsy obtained using gastrofibroscopy are in many cases crucial in confirming the diagnosis of chronic gastritis, as it is the only method for morphological diagnosis of the disease.

Helicobacter pylori detection

There are several methods for determining the degree of its distribution in the gastric mucosa:

- Cytology ;
- Urease test;
- Histological;
- Immunological.

The first three of them are widely used in practice.

most eroded part of the mucosa) or duodenum of the stomach using endoscopy is dried, stained with silver, and examined under a microscope to determine the level of microbial contamination (based on the number of *Helicobacter pylori counted*) (Figure 9) .

Figure 9. *Appearance of Helicobacter pylori*

There are three levels of *Helicobacter pylori infection:*

1. **Light (+)** – up to 20 microbes are present in the field of view;
2. **Moderate (++)** – there are 20 to 40 microbes in the field of view;
3. **High (+++)** – more than 40 microbes are present in the field of view.

Urease test It is an express method for the detection of *Helicobacter pylori* . The standard "campi-sinama" consists of a gel-carrier consisting of chamomile

(urea), sodium azide and phenol-roth solution. Phenol-roth is used as a pH indicator and changes its color from yellow to raspberry when the medium becomes alkaline.

Typically, *Helicobacter pylori* hydrolyzes urea (urea) with the help of the urease enzyme, forming ammonia, which leads to an increase in pH. As a result, the appearance of a raspberry color in the medium indicates the presence of *Helicobacter pylori microbes* in biopsy samples. Based on the time of color change of the sample, the degree of damage to the mucosa by the microbe is assessed:

1. The appearance of a raspberry color in the smear within an hour after the examination indicates a high level of infection of the mucous membrane with *Helicobacter pylori;*

2. The appearance of a raspberry color in the urine within two to three hours after the test indicates a moderate infection;

3. The appearance of a raspberry color in the urine within one day (24 hours) after the test indicates a mild infection;

4. The appearance of a raspberry color after a day or two means that the test is negative . after biopsy of the gastric mucosa. Therefore, this test is widely used in practice.

Histological examination can detect not only morphological changes in the gastric and duodenal mucosa, but also *Helicobacter pylori by staining using the Romanovsky-Giemza method.*

In recent years, a number of new histological methods have been used, including **immunocytochemical** or **DNA hybridization** methods based on monoclonal antibodies. These methods significantly increase the sensitivity and specificity of morphological detection of *Helicobacter pylori* , and also allow the identification of different strains of the bacteria. In addition, these methods are also used to determine the cause of mucosal re-infection after effective anti- *Helicobacter* therapy.

Serological testing is based on the detection of specific antibodies in the serum of a patient infected with *Helicobacter pylori, which are formed* after 3-4 weeks. High antibody titers can be detected even when the disease is in clinical

remission. For this purpose, various methods are used, including the assessment of antibodies of the IgG and IgA classes in serum by immunoenzyme analysis .

Helicobacter pylori is highly reliable and is often used for screening , ie to determine the level of infection in various population groups.

Microbiological examination allows the cultivation of *Helicobacter pylori* and the determination of its sensitivity to antibiotics. This allows the selection of appropriate therapy for the treatment of chronic gastritis, duodenitis, and ulcer disease associated with *Helicobacter pylori.*

Helicobacter pylori using radionuclides (breath test) — has been widely used in practice in recent years. This test is characterized by high sensitivity in monitoring the effectiveness of anti-Helicobacter treatment and detecting microorganisms. In it, the patient *ingests* a solution of urea labeled with the C14 radioactive isotope and the amount of CO_2 in the exhaled breath is determined. If *Helicobacter pylori* is present in the stomach, it breaks down urea under the influence of the urease enzyme and produces CO_2 .

is considered positive if, one hour after taking the urea, the level of radioactivity in the exhaled air is greater than 2% of the amount taken.

Diagnosing chronic gastritis and applying appropriate treatment measures, it is important to assess the activity of gastric juice production. The following methods are used to determine it:

- **Fractional examination of gastric secretion using a thin probe** - this method is used to study the acidity of basal and stimulated gastric juice, its enzyme production activity, pH level, and the physical and chemical properties of gastric contents.
- ***Examination using intragastric pH-metry*** .

testing the secretion of gastric juice using a thin probe with pentagastrin, histamine and insulin has been one of the main methods. However, currently, intragastric pH-metry is considered a highly informative and perfect method. Since it is the most physiological method, it does not affect the functioning of the stomach and does not cause pathological reflexes.

Fractional probing method. This method is performed using a thin probe with a diameter of 4–5 mm and a length of 100–150 cm. The end of the probe, which is inserted into the stomach, is closed, and there are holes on the sides. 3–5 days before the examination with a probe, patients are prohibited from taking antacids , H_2 -histamine receptor blockers and other antisecretory agents. The procedure is performed in the morning, after breakfast, after a 12-hour fast. The patient should be seated in a chair, with his back resting on his backrest and his head slightly tilted.

the patient's neck and chest, and a special container (tray) is given to catch the saliva flowing into the mouth. The sterilized probe is held with the right hand at a distance of 15 cm from the end that will be inserted into the stomach, and the left hand holds the free part of the probe. The closed end of the probe is placed on the root of the tongue and slowly moved towards the palate. At the same time, the patient should take a deep breath and make active swallowing movements. Through this, the probe is slowly moved into the esophagus. If a cough occurs during the insertion of the probe, it should be removed immediately. Otherwise, the probe may fall into the larynx or trachea.

The depth of insertion of the probe into the stomach (L) is calculated as follows:

L (cm) = patient height – 100

in most cases, in older people, the depth of insertion of the probe corresponds to its second mark (70–75 cm above the closed end). In this case, the probe is in the pyloric cup area, which allows for complete removal of gastric contents.

Gastric juice is a product produced by its glands and mucous membrane. Therefore, testing gastric juice (ie, assessing secretory function) is one of the main auxiliary methods, allowing to assess the activity, morphological state, and evacuator activity of the gastric mucosa.

studying the secretion at different stages of the secretory cycle over a long period of time not only qualitatively, but also quantitatively assessing the composition of the juice and collecting detailed information about the gastric

mucosa. Examination of the secretion of juice from the stomach should be carried out in accordance with the established goals and objectives.

Once inserted into the stomach, a thin probe in most cases does not provoke a gag reflex and can remain there for 1.5–2.0 hours, or even more. This allows for prolonged gastric juice aspiration, observation and study of its secretory activity at certain intervals. A syringe is attached to one end of the probe protruding from the oral cavity, and gastric juice is aspirated.

Verification steps

Patients are prepared for the gastric juice test as follows: The day before, a light dinner is eaten no later than 8:00 PM . On the morning of the test, there is no breakfast, and it is also forbidden to take liquids and medications. Smoking is not allowed.

Basal secretion	In all cases, the intensity of secretion of juice from the glands during the first hour of examination of the stomach with a probe is determined during functional rest, that is, during the period of digestive intermission.
	For this purpose, the juice is usually sucked on an empty stomach and taken four times every 15 minutes. This is called basal secretion.
The stomach of healthy people usually holds 50 ml of fluid on an empty stomach, and rarely more.	
Basal secretion per hour is 30-150 ml (average 50 ml).	
Stimulation of gastric secretion	After four portions of basal secretion are taken (ie after 60 minutes), to study the secretory activity of the gastric mucosa during digestion, patients are given a stimulant drug (histamine, histalog, pentagastrin, etc.) subcutaneously instead of food, and the secretion of juice is increased.
	Enhancing gastric juice secretion with medications allows for the production of juice suitable for laboratory analysis.

After stimulation, gastric juice is collected at 15-minute intervals for one hour, and each portion is collected in separate containers and the amount is determined.

Information about the rate of evacuation (ie motor function) of gastric juice is obtained from the volume of gastric juice 25-30 minutes after stimulation. Normally, this figure is on average 75 ml.

The sum of the last four portions indicates the amount of gastric juice secreted over an hour. In a healthy person, this figure is on average 60 ml during intermittent aspiration, and 1.5-2 times more during continuous aspiration.

When considering the portions of gastric juice taken, the following are taken into account:

- Color;
- Consistency;
- The presence or absence of additional compounds;
- Smell.

Normally, gastric juice is almost colorless. The presence of bile (when it enters the stomach from the duodenum) gives it a yellow or green color, the presence of blood gives it a red or often black-brown color, and the presence of a large amount of blood is an indication to immediately stop the probe.

In a healthy person, the consistency of the mucus is liquid. The more mucus it contains, the more viscous and stretchy it is, sometimes this property is so strong that it cannot be separated from its main component for examination.

A large amount of mucus indicates gastritis. In addition to the above, the mixture may also contain remnants of food eaten the day before. This indicates a violation of the emptying function of the stomach.

Chemical tests. After the external characteristics of the sap are described, its chemical composition is examined.

Each serving contains:

- free hydrochloric acid;

- total acidity;
- bound hydrochloric acid;
- lactic acid;
- maximum acidity of the portion - pepsin content.

The acidity of gastric juice is determined by titrating it with a 0.1 mmol/l sodium hydroxide (NaOH) solution in the presence of an indicator. In most cases, the acidity level is determined by the amount of NaOH in milliliters required to neutralize 100 ml of juice. Recently, the amount of hydrochloric acid has been expressed in milligrams or milliequivalents. Titration is carried out by adding 2 drops of indicator to 5 or 10 ml of juice. A 0.5% solution of dimethylaminoazobenzene in alcohol and a 1% solution of phenolphthalein in alcohol (recently, a reddish phenol solution has often been used) are used as indicators. Due to the presence of hydrochloric acid, after the addition of dimethylaminoazobenzene, the gastric juice turns red. Titration of the juice with a NaOH solution is carried out by shaking the beaker regularly until a pink-orange color appears, that is, until the free HCl is neutralized. The amount of NaOH used for titration is multiplied by 20 and the concentration of free HCl in mmol/liter is found. Then the titration is continued. At this time, the gastric juice turns yellow due to staining with dimethylaminoazobenzene and then red again (due to staining with phenolphthalein). This corresponds to the complete neutralization of all acidic valences of the gastric juice. From the beginning of the process until the gastric juice turns red, the amount of NaOH used for titration is multiplied by 20 and the total acidity is determined in mmol/liter

Even in the gastric juice of a healthy person, there are small amounts of proteins (pepsin, gastromucoprotein): in gastritis, bleeding ulcers, and gastric cancer, the amount of protein and bound hydrochloric acid in the stomach increases. It is determined by titrating a separate portion of gastric juice (5 ml) in the presence of any free acid, which turns yellow in the presence of any free acid, and turns purple when neutralized. The amount of free hydrochloric acid is determined by subtracting

the amount of NaOH consumed in the titration with alizarin per milliliter (multiplied by 20) from the total acidity indicator.

The standard indicator of acidity, which has been used for many years, has recently been revised. Thus, in healthy people, free hydrochloric acid is absent or does not exceed 10–20 TB on an empty stomach at breakfast. The normal acidity after a light breakfast given for testing is 20–40 tb for free hydrochloric acid and 40–60 tb for total acidity.

Numerous studies conducted among healthy people have shown that only 50% of them have the acidity level corresponding to the indicated figures, and the remaining 50% have it lower or higher, depending on their constitutional features. Even so, when the total acidity indicator is below 20 tb - it should be considered a hypoacid state, and when it is above 100 tb - a hyperacid state. The determination of the complete absence of hydrochloric acid is of important diagnostic importance. The absence of free hydrochloric acid in the gastric juice after the introduction of histamine in a maximum dose is called histamine-refractory achlorhydria and indicates the presence of atrophic processes in the gastric mucosa.

The acidity indicator (acid concentration) does not provide complete information about the acid-forming activity of the stomach. To get a complete picture of acid production, it is necessary to calculate the debit hour of hydrochloric acid - the indicator of hydrochloric acid secretion (the amount of acid produced by the stomach in one hour). To calculate the debit hour, it is necessary to multiply the indicator of the acid concentration in the gastric juice by the volume of secretion for one hour and divide by the resulting number relative to the acid concentration: by 100 if the acid concentration is indicated in mg%, and by 1000 if in mEq/l.

If the acidity index is multiplied by 3.65, the acid concentration in the titrated unit of acidity can be written as mg%, since the titration unit of the weight-by-weight index is 3.65 mg of hydrochloric acid or 0.1 mEq in 100 ml of juice.

So, for example, if the acidity is 60 tb, the hydrochloric acid can be expressed as 60 mmol/l or 60 mEq/l or (3.65 • 60) mg%.

Not all patients are recommended to swallow a probe (contraindication: gastric tumor, esophageal stenosis, aortic aneurysm, etc.) or not all those who are recommended can swallow it. For many years, ways to determine acidity without a probe have been sought. In 1905, Sali proposed a simple method, which consisted of the following: the patient is offered to swallow a small and thin rubber bag tied to a catgut thread and containing 0.1 g of methylene blue. After that, the patient has lunch as usual. If there is hydrochloric acid in the stomach, the catgut dissolves and is digested, and the methylene blue dissolves in the stomach and after a certain time the urine turns blue.

In the last decade, a number of tests based on the use of ion-exchange resins have been proposed. A pill made from this resin is added to a specific substance (quinine, azure-1 dye, etc.), which, under the influence of hydrochloric acid in the stomach, is separated from the main product and excreted in the urine. This method provides complete information about the presence or absence of hydrochloric acid in the stomach, but does not determine its quantity. It can be used only in patients with normal kidney function.

Currently, a radiotelemetric method (endoradiosonography) is used to determine the acidity (or more precisely, pH) of gastric juice.

The second important aspect of studying gastric juice is determining its digestive properties, mainly in terms of the degree of protein digestion.

A simple method for determining the peptic activity of the juice was proposed by Mettom in 1899. A thin glass tube filled with denatured (especially) egg white is lowered into a test tube with gastric juice (slightly acidified, if it does not contain free hydrochloric acid) and placed in a thermostat. After a day, the height of the tube free of protein is measured (in mm). When the amount of pepsin is normal, the sum of the lengths of the free areas on both sides of the tube should be 6-12 mm.

Currently, the adapted VNTugolukov method, which gives more accurate results, is widely used. 2% dry plasma is placed in one of the two centrifuge tubes (the lower part is divided into small, clear divisions) and the gastric juice to be tested,

diluted in a ratio of 1:100, is poured into the other of the tubes, and the previously well-boiled juice is poured into the other. Both tubes are left in a thermostat for 20 hours. After that, a solution of trichloroacetic acid is poured into both tubes and, after thorough mixing, centrifuged. The digestive properties of the gastric juice are assessed by the decrease in the amount of precipitated protein. The obtained indicators can be compared with the results of various experiments conducted with different solutions of similar pure dry pepsin, and the amount of pepsin in the gastric juice can be expressed in milligrams.

To determine the activity of the stomach to produce pepsinogen without using a probe, an attempt is made to determine the amount of pepsinogen in the urine (uro-pepsinogen). According to available data, not all pepsinogen is secreted in the stomach, part of it (about 1%) enters the blood and is excreted in the urine, which indicates that it is produced in the stomach. Uropepsinogen, like pepsin in gastric juice, is also determined by the method of curdling milk or by the method of VN Tugolukov.

The detection of lactic acid in gastric juice is also of diagnostic importance. It appears in the stomach only during the vital activity of lactic acid-producing bacilli that vegetate (proliferate) in the absence of hydrochloric acid, or in malignant tumors in which the glycolysis process in gastric cells, which leads to the formation of lactic acid, proceeds anaerobically. This change is not considered pathognomonic for tumors, but careful additional examinations are required to rule it out.

One of the methods for determining lactic acid is the Uffelman reaction. 1-2% phenol solution is poured into 2/3 of the test tube and 2-3 drops of 10% ferric chloride solution are added. The reagent turns dark purple. Tilt the test tube and slowly pour 2-3 drops of gastric juice along its wall. In the presence of lactic acid, the drops of juice that fall to the bottom of the test tube turn bright yellow with ferric-lactic acid.

6.Treatment of gastritis methods and prevention

Treatment for gastritis includes:

1. Adherence to a special diet.
2. Preparations to normalize acidity in the stomach.
3. Means for restoring the gastric mucosa.
4. Antibacterial therapy .

Treatment of patients is carried out without drugs and with the help of drugs. ***Treatment without drugs.*** Patients are advised to eat warm food, the composition of which is ground before cooking, chewing thoroughly 5-6 times a day. Patients with chronic non-atrophic gastritis are excluded from the diet of salty, smoked, fatty, fried and spicy foods that have an aggressive effect on the gastric mucosa, and alcoholic beverages are also prohibited.

A, in addition to reducing mechanical effects on the stomach, boiled meat and vegetable soups, lean fish, a sufficient amount of vegetables and fruits, juices, and coffee are prescribed, which enhance its secretory activity. It is also advisable to widely use spices in cooking.

Drug treatment - is carried out taking into account the type of chronic gastritis. For the complex treatment of gastritis of types B, A B and C, a number of antisecretory drugs are used, which are divided into 5 main groups:

1. ***Antacids*** - neutralize hydrochloric acid and adsorb pepsin. Aluminum - containing antacids, in addition to the above effects, have a cytoprotective effect, enhance the synthesis of glycoproteins and accelerate reparative processes. Absorbable antacids include sodium bicarbonate, magnesium oxide, etc. In recent years, poorly absorbed (non-systemic) antacids have been used in practice. ***Aluminum*** phosphate , aluminum-magnesium antacids - Maalox, Phosphalugel , Almagel , etc. are representatives of this group of drugs. It is advisable to take antacids 3-4 times a day, 1-1.5 hours after meals. Some doctors recommend taking non-systemic antacids before bedtime. However, this approach is questionable, and the acid-neutralizing properties of the drug may not be manifested;

2. ***Non-selective m-cholinoblockers*** (atropine, platifillin, methacin) - have weak antisecretory properties. When prescribing them, some patients may experience side effects such as tachycardia, constipation, urinary disorders, and increased intraocular pressure;

3. ***Selective M-cholinoblockers*** pirenzepine, gastrozepine, etc. block the fundal cells of the gastric mucosa and are prescribed to be taken 50 mg twice a day, half an hour before meals.

4. ***H_2 histamine receptor blockers.*** There are several generations of them, the first of which is cimetidine, the second is ranitidine, the third is famotidine, quamatel, and the fourth is nizatidine;

5. ***Proton pump inhibitors*** - have a highly selective effect, blocking the ability of the gastric mucosa to produce acid. Currently, there are 4 generations of them. 1st generation - omeprazole 20-40 mg, 2nd - pantoprazole 20-40 mg, 3rd - rabeprazole 20 mg, esomeprazole 4th - lansoprazole 30-60 mg are prescribed per day.

6. ***Encapsulating agents*** , including cytoprotectors - sucralfate, denol, etc., isolecithin, pepsin bind bile acids and retain prostaglandin in the stomach for a long time, increasing mucus production. They are taken 1 g 4 times a day (3 times 1 hour before meals and 1 time before bedtime) for 2-4 weeks.

In ***idiopathic pangastritis (type*** A B), it is forbidden to follow a long-term diet, that is, to eat smoked, fatty meats, spicy, salty and spicy foods. It is recommended to take drugs that enhance reparative processes and have an anti-inflammatory effect, such as sucralfate (venter), bismuth trinitrate (denol), sofalcone, misoprostol (cytotec) 1 g 3 times a day 1.5-2.0 hours after eating. Also, prokinetics, spasmolytics, vitamins A , E and ascorbic acid are prescribed from time to time. Patients should take chamomile, mint, dandelion tinctures, plantaglucid granules for a long time. Patients with idiopathic pangastritis should undergo a gastrofibroscopy examination once a year to exclude malignancy.

In reflux gastritis (type C), drugs that promote faster gastric emptying are prescribed - metoclopramide, loperamide, domperidone, bromopride (viaben), peritol. They are usually taken 1/2-1 tablet 3 times a day before meals. It is also recommended to take sucralfate, sofalcon 1 g 3 times a day 1.5-2.0 hours after meals. Sulpiride (eglonil, dogmatil) is prescribed 0.05 g during the day and at night, and antacids (almagel, phosphalugel, malox, ulcosane) are prescribed according to the indications.

patients are diagnosed ***with H. pylori-associated chronic gastritis,*** treatment measures should be aimed at eliminating clinical symptoms, reducing gastric secretory activity, and eradicating the bacteria. For this purpose, the "M aa strix IV consensus" algorithm, adopted in 2011, is currently used. According to it, the patient is prescribed proton pump inhibitors (omeprazole 40 mg per day, pantoprazole, rabeprazole or others) for 4-6 weeks, amoxicillin (2 g per day) and clarithromycin (500 mg twice a day) for 10-14 days. Instead of moxifloxacin , metronidazole tablets (500 mg twice a day) can be used. Levofloxacin, furazolidone, rifaximin, rifabutin are also recommended as reserve antibiotics after sensitivity is determined. In addition to the above, if the acidity of gastric juice is high, antisecretory agents (antacids, selective and non-selective M-cholinoblockers, H_2 histamine receptor blockers, and enveloping agents - cytoprotectors) are used.

type A gastritis , natural gastric juice (one tablespoon with ½ cup of water during meals, an acidin - pepsin tablet dissolved in ½ cup of water before meals), anti-inflammatory agents that enhance the acid production activity of the stomach (1 tablespoon of bargizub - a decoction of zubturum leaves, taken 3 times a day before meals or plantoglucide 0.5-1.0 g 3 times a day before meals), drugs that restore pancreatic enzyme deficiency (abomin, festal, digestive, panzinorm, mezim forte, creon 1 tablet per day during meals), drugs that improve microcirculation and reparative processes and mucosal nutrition (actovegin, solcoseryl 5.0 ml intramuscularly or intravenously for 10-15 days, methyluracil 0.5 g 3 times a day, vitamins, including folic and ascorbic acids), are recommended.

To eliminate pain syndrome in patients, M-cholinolytics (gastrocepin 25-50 mg 2 times a day), selective spasmolytics (mebeverine-dyuspatalin 200 mg 2-3 times a day) are prescribed.

Prevention.

Primary and secondary prevention of the disease includes adherence to a diet, avoidance of foods and beverages (primarily alcohol) that have mechanical, chemical, or thermal side effects, ie, irritating and negatively affecting the gastric mucosa, adherence to personal hygiene rules, oral hygiene, etc. These include eliminating harmful effects associated with the profession, and quitting tobacco and secondhand smoke.

No. 1 for gastritis excludes the following:

1. Bread.
2. Soup.
3. Cornmeal and peach porridge, legumes.
4. Fatty fish and meat varieties.
5. Canned goods.
6. Sour milk products.
7. Carbonated drinks, tea, coffee, alcohol.
8. Chocolate and ice cream.

CHAPTER III. Etiology, pathogenesis, treatment and prevention methods of peptic ulcer disease

1. Etiology and pathogenesis of peptic ulcer disease

Peptic ulcer disease is a chronic, relapsing disease with alternating periods of exacerbation and remission, the morphological basis of which is the formation of ulcers in the mucous membrane.

Gastric and duodenal ulcers are very common among internal diseases. According to world statistics, approximately 10% of the elderly population suffers from gastric and duodenal ulcers.

According to data provided by the World Health Organization (WHO), peptic ulcer disease (PUD) is a global epidemic and one of the most pressing challenges for healthcare systems.

Global prevalence in 2019 : The global prevalence of gastric cancer was estimated at 8.09 million cases, an increase of 25.82% compared to 1990.

Age-adjusted prevalence rate : 99.40 cases per 100,000 people in 2019, a decrease from 1990.

The prevalence rate was higher in men than in women, but the difference was decreasing.

South Asia : In 2019, the age-adjusted prevalence rate in this region was 156.62 cases per 100,000 population, the highest. The highest prevalence rate in the world was 330.32 cases per 100,000 population.

From 1990 to 2019: There has been a significant decrease in the incidence and mortality rates of peptic ulcers. However, there has been a slight increase in prevalence and mortality rates over the past 15 years.

According to data obtained in recent years, the incidence of peptic ulcer disease has increased by 25-30%. Duodenal ulcer disease is 4 times more common than gastric ulcer. Gastric ulcer is observed slightly more often in men than in women.

Etiology and pathogenesis: the origin of gastric and duodenal ulcers is polyetiological, in which hereditary, psychological, alimentary, mechanical, Helicobacter pylori bacteria, harmful habits and uncontrolled long-term use of

certain drugs play an important role. Under the influence of these etiological factors, ulcer disease develops as a result of an imbalance between aggressive and protective factors.

gastric and duodenal ulcers are due not only to the prevalence of these digestive system diseases (occurring in 7–10% of the adult population), but also to the lack of sufficiently reliable treatment methods that would minimize the likelihood of recurrence of the disease.

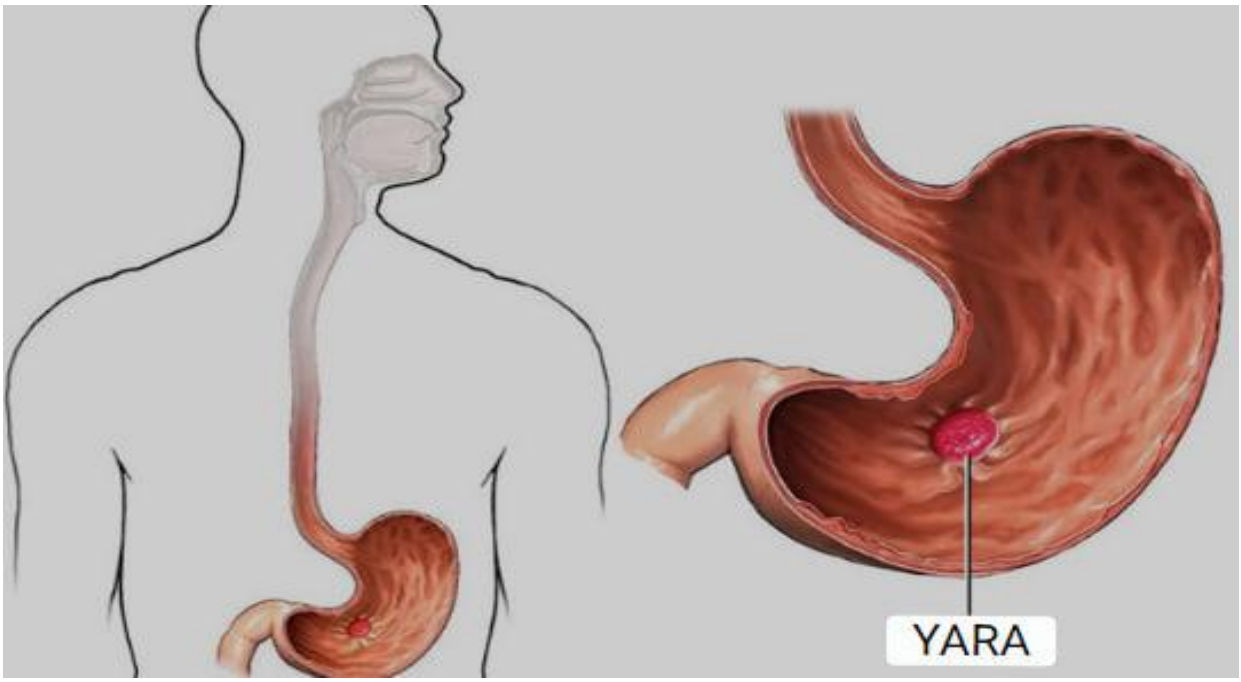


Figure 10. *Stomach ulcer*

Peptic ulcer disease is a chronic disease, the main morphological manifestation of which is the formation of recurrent ulcers in the stomach or duodenum. It is important to note that medicine has always strictly distinguished between peptic ulcer disease and symptomatic ulcers.

Symptomatic ulcers are ulcers that occur in the gastroduodenal mucosa against the background of various diseases and conditions. Such ulcers are observed in endocrine system pathologies (parathyroid adenoma, Zollinger-Ellison syndrome), in stressful situations, in acute or chronic circulatory disorders, in allergies, as well as in the use of non-steroidal anti-inflammatory drugs (NSAIDs).

Risk factors for peptic ulcer disease:

- Increased stomach acidity
- Blood type I (0)
- Tuxedo
- Acceptance of YQNV
- Psychological stress
- Hereditary predisposition BLA-B5, B14, B15-antigen

Gastric and duodenal ulcer is a complex pathological process, the basis of which is the inflammatory reaction of the body. This reaction is manifested by the formation of local damage to the mucous membrane of the upper parts of the gastrointestinal tract. This condition occurs as a response to a violation of the endogenous balance between local "protective" and "aggressive" factors in the gastroduodenal area.

"Aggressive" factors include:

1. increased secretory capacity of the stomach - that is, an increase in the number of chief and parietal cells that produce hydrochloric acid and pepsin,
2. An altered response of glandular cells to nervous and humoral stimuli, leading to excessive acid production and sometimes disproportionate hypergastrinemia.
3. Also, rapid evacuation of acid-filled mass from the stomach into the duodenum (which causes "acid shock"), bile acids, alcohol, nicotine, drugs (especially nonsteroidal anti-inflammatory drugs and glucocorticoids), and Helicobacter pylori infection also have an aggressive effect.

The etiological role of bacteria in the development of peptic ulcers has been suggested for a long time. Spirochetes were first described in the stomachs of animals in 1893, and in the 1940s, these microorganisms were identified in the stomachs of people suffering from ulcers and stomach cancer.

It was only in 1983 that the pathogenetic link between bacterial infection and peptic ulcer disease was scientifically confirmed.

- H. pylori infection is detected in 70-90% of patients with peptic ulcers.
- without H. pylori are usually associated with long-term use of nonsteroidal anti-inflammatory drugs (eg, aspirin, ibuprofen) or stress.

Australian researchers Robin Warren and Barry Marshall discovered spiral-shaped bacteria in the stomachs of patients with chronic gastritis and peptic ulcers and were able to grow them in culture. Initially, these bacteria were considered to belong to the Campylobacter genus, but they were later classified as a separate, new genus and have been known worldwide since 1989 as Helicobacter pylori (H. pylori).

H. pylori is a gram-negative, microaerophilic (requires little oxygen) bacterium that has a curved or spiral shape and numerous cilia.

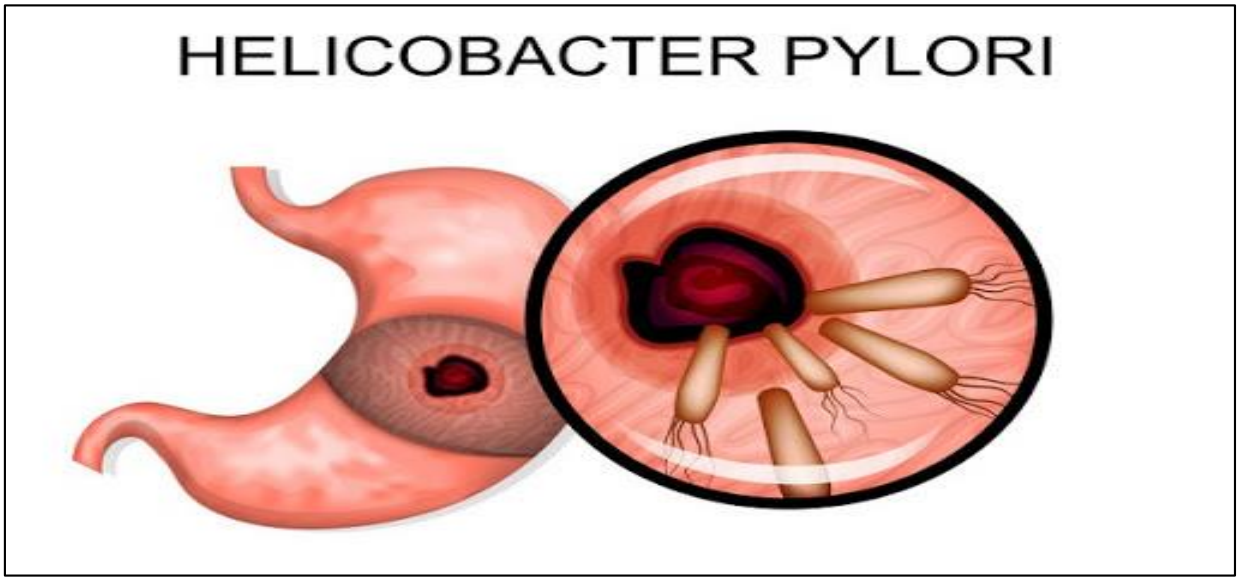


Figure 11. *Image of Helicobacter pylori causing stomach ulcers.*

It is mainly located deep in the stomach wall and on the surface of epithelial cells, mainly under the mucous membrane. Despite this very unusual environment, H. pylori does not face competition from other microorganisms.

Helicobacter pylori is a conditionally pathogenic bacterium. In certain cases, it can be considered a symbiont, since this bacterium is resistant to the aggressive environment of the stomach (pH in its habitat is ≈ 7) and, probably, appeared in the human body during the formation of the microenvironment of organs and systems. However, with changes in the external environment, new strains of H. pylori can acquire pathogenic properties.

Virulence (pathogenicity)

Currently, the following virulence factors of H. pylori are known, which allow the bacterium to establish itself in the human body and persist for a long time:

- Its spiral shape and the presence of cilia provide mobility and help it move within the mucous layer.
- The presence of adaptive enzymes
- Adhesive ability (adhesion to epithelial cells)
- Ability to suppress the immune system

The mobility of the bacterium allows it to move inside and live under the mucous membrane. To protect itself from the aggressive effects of gastric juice, H. pylori produces the enzyme urease, which breaks down urea to form ammonia, thereby neutralizing hydrogen ions in the gastric environment. Urease is produced in the cytoplasm and on the outer surface of the bacterium.

H. pylori urease is a toxin for the gastric epithelium because it increases inflammation in the mucosa. This occurs by activating monocytes and neutrophils, increasing cytokine secretion, and generating oxygen and nitrogen radicals. Nitric oxide and reactive oxygen radicals are key factors protecting the mucosa. If nitric oxide synthase is blocked, blood flow to the mucosa decreases and cell apoptosis (self-destruction) develops.

Apoptosis is a process that ensures tissue homeostasis, and its excessive activation disrupts the physiological and reparative regeneration of the mucosa.

Studies conducted in recent years confirm that Helicobacter pylori is most likely the main cause of chronic gastritis and stomach ulcers.

The study of H. pylori as an etiological factor in chronic gastritis has made it possible to further clarify the multifactorial pathogenesis of peptic ulcer disease.

When H. pylori is detected, morphological signs of gastritis can always be seen (this is infiltration with neutrophils in a specific layer of the epithelium and mucosa, as well as mononuclear infiltration, which indicates the activity of gastritis). Once the infection is eliminated, these morphological signs disappear.

Thus, chronic nonatrophic gastritis is completely cured after eradication of H. pylori. The etiological role of H. pylori in chronic gastritis makes it an important factor in the pathogenesis of ulcer disease.

inflammatory infiltrate cells play an important role in the damage to the gastric mucosa. When H. pylori adheres to epithelial cells, they produce a number of cytokines, primarily interleukin-8. Leukocytes migrate from the blood vessels to the site of inflammation. Activated macrophages secrete interferon-gamma and tumor necrosis factor (TNF), which recruit other cells involved in the inflammatory response. Reactive oxygen species metabolites of neutrophils damage the gastric epithelium. As a result, the mucosa becomes more sensitive to the aggressive effects of the acid-peptic factor.

In recent years, some molecular mechanisms have been revealed that are associated with the presence of bacteria and impair the process of repair (reparative regeneration) of the gastric epithelium.

Therefore, H. pylori infection slows the healing of gastroduodenal ulcers .

It has been shown that H. pylori is closely associated with factors of aggressiveness in ulcer disease . It may act directly or indirectly through cytokines secreted by monocytes and lymphocytes in the inflammatory infiltrate. This may lead to the activation of gastrin-producing G-cells and somatostatin-producing D-cells. leads to a disruption of the balance between them. They play an important role in controlling the activity of parietal cells .

Hypergastrinemia (excessive production of the hormone gastrin) causes parietal cell proliferation and increased acid production in the stomach. Eradication of H. pylori significantly reduces serum gastrin levels and acid production.

New insights into the pathogenesis of ulcer disease have been gained by studying the effect of Helicobacter pylori infection on the cytoprotective (cellular defense) capacity of the gastroduodenal zone . When this protection is impaired, the main pathogenetic mechanisms leading to ulcer formation are activated, for example:

- reduced energy supply for trophic (tissue-nourishing) processes,

- slowing down cell regeneration,
- Factors that contribute to the disruption of tissue protective mechanisms and other wound formation.

In ulcer disease, various metabolic disorders are observed that determine the activity of reparative processes in the gastroduodenal zone , including:

- nucleic acid imbalance,
- disruption of oxidation-reduction processes,
- severe trophic disorders,
- disorders at various stages of protein metabolism.

play an important role in the etiology and pathogenesis of ulcer disease, which gives grounds for classifying this disease as a psychosomatic disease. This explains the specificity of the disease and its various manifestations.

Autonomic nervous system imbalance - Parasympathetic in stressful situations and sympathetic **The balance** between the systems is disrupted, which leads to excessive secretion of stomach acid.

Disruption of the protective mechanisms of the mucous membrane - Under the influence of cortisol and adrenaline, the synthesis of prostaglandins decreases, which weakens the protective function of the mucous membrane.

Circulatory disorders - Stress reduces blood circulation in the stomach lining, which increases the susceptibility to ulcer formation.

Weakened immune system - Stress reduces the immune response, which contributes to the activation of Helicobacter pylori infection.

Psychological and personal factors, especially stress **,** anxiety , and Personal characteristics play a decisive role in the development of gastric and duodenal ulcers. These factors affect not only the onset of the disease, but also its recurrence **and** severity.

the onset of ulcer disease, the patient develops a "psychological attitude" towards it, that is, a reduced tolerance for psychological trauma. This leads to the formation of a psychosomatic cycle (a situation in which inflammation and mental state reinforce each other).

play an important role in the occurrence of ulcer disease, then among the causes of its exacerbation, various disorders of regulatory mechanisms play a major role. These disorders occur at levels ranging from the cerebral cortex to complex nervous, hormonal and humoral (blood-borne) regulatory mechanisms. Ultimately, this disrupts the balance between factors that protect the gastric and duodenal mucosa and factors that damage it .

Protective factors include:

- mucus secretion,
- prostaglandins (biologically active substances),
- epithelial cell regeneration,
- adequate blood supply to the mucosa.

In the 1960s, H. Davenport introduced the concept of the "protective mucosal barrier of the stomach ." This is the first-level defense system against aggressive factors. It includes :

- gastric juice,
- alkaline bicarbonate secretion,
- normal blood circulation (microcirculation),
- cell renewal,
- synthesis of certain prostaglandins.

In recent years, much attention has been paid to the function of the mucosal bicarbonate barrier, which acts as a single system at the epithelial cell level , helping to understand the true balance between aggressive and protective factors.

healthy people, protective factors always prevail over harmful factors, which helps to maintain the structure of the mucous membrane under the influence of external and internal factors.

However, when the pH of gastric juice drops to 1.5 , the mucus-bicarbonate barrier loses its protective role. Gastric juice is a special substance consisting of glycoproteins and mucins that protects the underlying epithelial cells from harmful effects. It is produced by epithelial cells (in the stomach and duodenum) and contains sulfated glycoproteins and surface-active phospholipids.

Bicarbonates are produced by epithelial cells through intracellular metabolic processes and secreted from the apical (top) surface of the cell membrane.

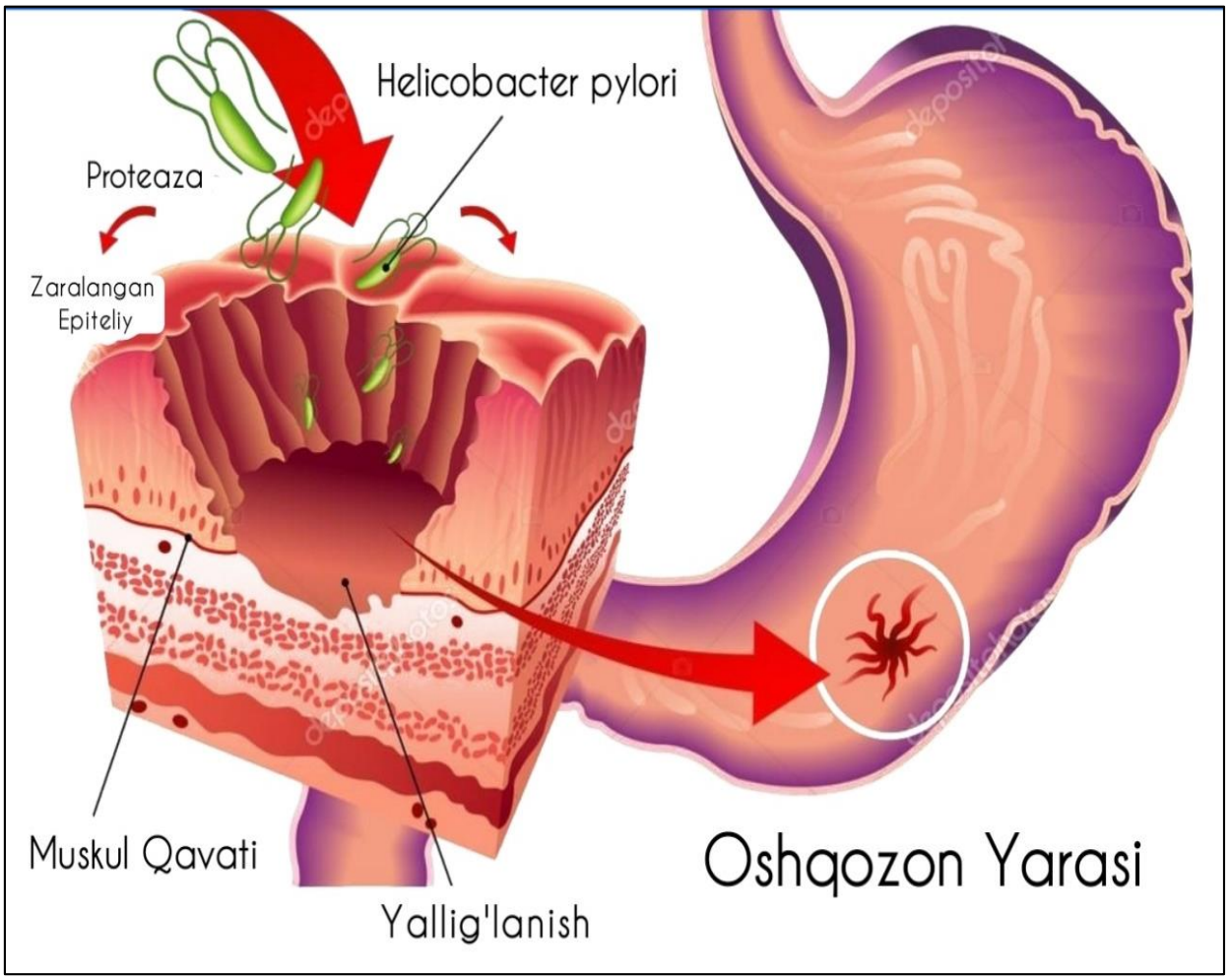


Figure 12. Damage to the gastric mucosa from Helicobacter pylori infection

The next line of defense is the impermeable membrane of the epithelial cells. These are the apical membranes of the cells, which are covered by the stomach and prevent the passage of acid back into the cells from the stomach cavity . The position of the apical cells and their interconnections are sufficient to maintain a pH of 7 inside the cells.

The third line of defense is the blood microcirculation. It supplies cells with water, oxygen, nutrients, and buffers. Without these substances, cells cannot perform the secretion process. Capillaries also perform the function of draining H^+ ions back into the body.

A separate group from a clinical point of view are patients with low gastric acidity (hyposecretory). In them, the main role in the disease is not played by acid-peptic factors. In such patients, microcirculation disorders of the gastrointestinal tract are detected, which:

- duration of illness,
- inflammation and tissue damage,
- the severity of complications,
- directly related to the strength of the pain syndrome.

2. Classification of peptic ulcer disease and its clinical picture

In recent years, significant advances have been made in the field of diagnosis and treatment of peptic ulcer disease, and numerous studies have been significantly expanded.

Classification

According to the modern international classification:

- Stomach ulcer
- Duodenal ulcer
- Gastrointestinal ulcers

Also known as primary (or true) ulcer disease oath secondary (symptomatic) ulcers there is.

Symptomatic gastroduodenal ulcers:

- Stress-related wounds (myocardial infarction, sepsis, surgery)
- Wounds that call for medicine
- Ulcers associated with endocrine diseases
- Wounds associated with circulatory disorders and hypoxemia
- Toxic wounds
- Ulcers that occur in liver, pancreas, and blood diseases

duodenal ulcers, compiled by AL Grebnev and AA Sheptulin in 1995 :

According to etiology and pathogenesis:

1. Primary wound disease:

a. *Associated with Helicobacter pylori (HP)*

b. *Not related to HP*

According to the location of the wounds:

1. Stomach ulcers:

a. Cardiac and subcardiac departments

b. Stomach body

c. Antral section

d. Pyloric section

2. Gastroduodenal ulcers (occurring in both the stomach and duodenum)

By size:

- Small (less than 0.5 cm)
- Medium (0.6–1.9 cm)
- Large (2.0–3.0 cm)
- Giant (larger than 3.0 cm)

According to the stages of the disease :

- Sharpening (sharpening)
- Remission (quiet period)

According to the nature of the passage:

- Mild: rare (once every 2–3 years)
- Average: rape every year
- Severe : 2–3 times a year or more often

CLINICAL PICTURE OF THE DISEASE:

I. COMPLAINTS:

1. PAIN SYNDROME – main symptom:

- Related to food intake
- in the epigastric (stomach) area and behind the breastbone (cardiac or posterior wall ulcers)
- **Early pain** : 15-60 minutes after **eating**
- **Evening pain** : 1.5–3 hours after **eating**
- **Hunger or night pain:** typical of duodenal and pyloric ulcers

- Pain is relieved by food, antacids, antispasmodics, or vomiting
- Pain worsens in spring and autumn

of pain (radiation) :

- Lumbar, between the scapula - in postbulbar wounds
- Heart, left scapula, thoracic spine – in cardiac and subcardiac wounds
- Waist, right hypospadias, interscapular region – duodenal ulcer

Pain type:

- Stretching, aching, burning, tightening , piercing, spreading (in complications)

Pain mechanisms:

- Increased intragastric pressure (sphincter spasm, muscle tone)
- Spasm of the duodeno-gastric zone
- Baroreceptor irritation
- Increased secretion
- Microcirculation disorder

Types of pain:

- **Visceral** : associated with food, disappears after meals or antacids
- **Spasmodic** : on an empty stomach and at night, after meals or from heat
- **Somatic (inflammatory)** : persistent pain, due to perivisceritis

2. DYSPEPTIC SYNDROME:

- **Burning sensation in the stomach** : weakness of the lower esophageal sphincter, increased tone of the pyloric muscles
- **Vomiting** : air, food, sour, bitter or foul-smelling
- **Nausea**
- **Vomiting** : at the peak of pain, at different intervals (depending on the department), often with sour or previously eaten food; mixed with bile (duodeno-gastric reflux); coffee-colored (presence of blood)

3. Irritable bowel syndrome:

- Pain throughout the intestines

- Constipation
- Loose stools (melena - gastrointestinal bleeding)

4. Astheno-vegetative syndrome:

- Weight loss, decreased performance, insomnia, irritability

II. HISTORY:

- Identification of hereditary diseases
- Constitutional type
- Risk factors and underlying diseases
- Duration of the disease, previous treatments

III. PHYSICAL EXAMINATION:

1. Symptoms of autonomic dysfunction: Excessive outside sweating , flushing / back white dermographism , dyshidrosis , skin whitening

2. Pain when pressing on the hand :

- In the epigastric region, right upper quadrant, around the umbilicus, pyloroduodenal zone
- Diffuse pain - complications
- Mendeleev's sign is positive

3. Diagnosis of peptic ulcer disease

Laboratory and instrumental examinations:

1. **EGDS (esophagogastroduodenoscopy)** (Fig. 13) **and biopsy**

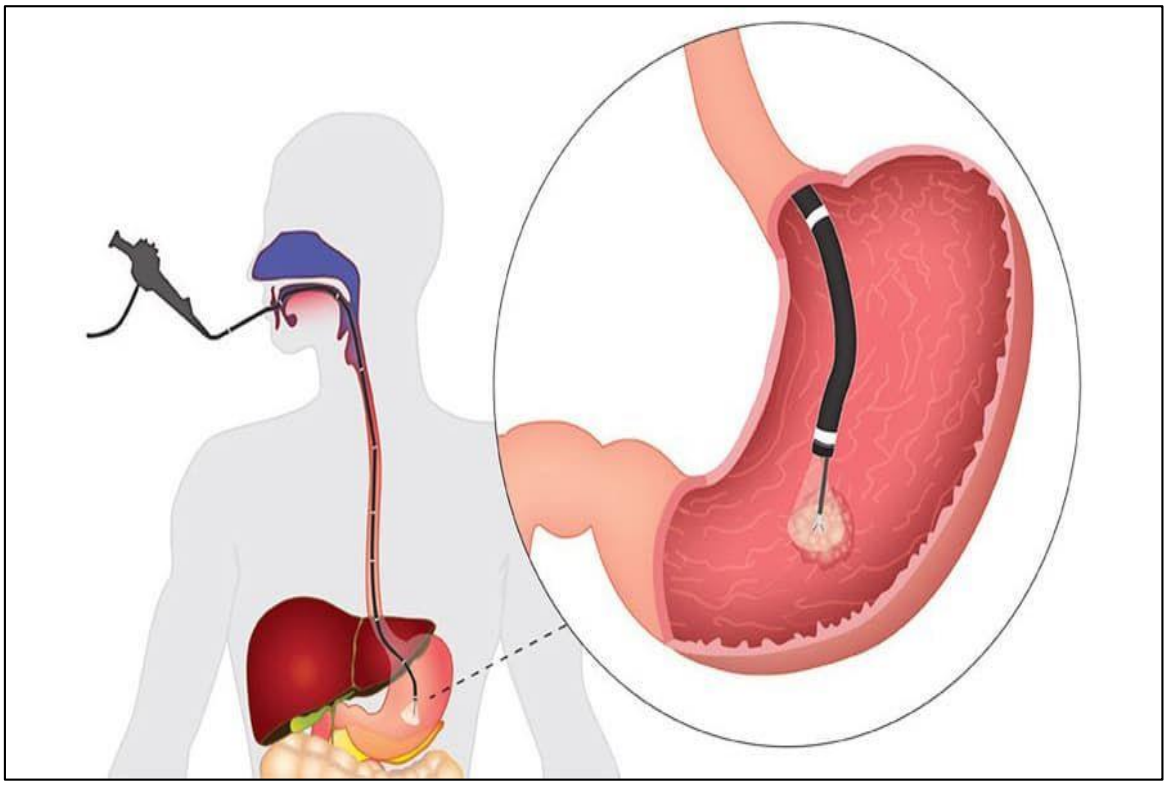


Figure 13. Examination of the stomach through a probe

2. **H. pylori diagnosis** (Table 3) **:** Breath urease test, PCR test of stool or blood, biopsy histology, urease test on biopsy

Table 3. Diagnostic methods for H. pylori:

Methods that do not require EGDS	**Methods requiring EGDS**
Urease breath test (with C_{13}), PCR on stool, serum antibodies	Rapid urease test, biopsy histology, PCR on biopsy

3. **X-ray** ("finger-like" entry, barium retention for more than 6 hours, local pain during examination, spasm, gastric tumors, intestinal deformation)

4. **pH-metry** (Table 4) **-** a method for studying gastric secretion (at the beginning and after stimulation), measuring the level of acidity (pH) in the stomach. **It is** a diagnostic method based on the principle of gastric acid secretion .

Table 4

pH - metric	
Purpose	- Determining the level of acidity in the stomach - Assessment of hyperacidity (excess acid) or normal status
Method type	- **Intragastric pH-metry** (only inside the stomach) - **24-hour pH-metry** (continuous monitoring)
Transfer method	A thin probe is inserted through the nose into the stomach; real-time or daily pH readings are recorded
Norm	The pH of the stomach is normally between **1.5–2.5.**
The result of a stomach ulcer	- Often **pH < 2.0** (hyperacidity) - In rare cases, the pH may be normal
Diagnostic value	- Assess response to proton pump inhibitors or antacid therapy -Determining acid reflux at night
Advantages	- Noninvasive, painless, accurate - Dynamic control is available (around the clock)
Disadvantages	- The probe may cause discomfort in some patients - Requires expensive equipment

5. **Biochemical analysis of blood** - serum iron, total iron binding capacity of plasma
6. **Gregersen's reaction** - detection of occult blood in feces
7. **X-ray contrast duodenography**
8. **Blood general analysis** (Table 5)

Table 5

Indicator	Norm	Changes in stomach ulcers
Hematocrit (indicator)	Men: 40-54%, Women: 37-47%	It may decrease because the hematocrit decreases as a result of bleeding.
Total blood volume	4.0-5.5*10^6/mm³ (I)	Bleeding from a wound can cause a decrease in blood volume or the development of anemia.
Leukocytes	4.0-9.0 x 10^9/l	It may increase if there is bleeding or an inflammatory process indicating infection.
Erythrocytes	I: 4.5–5.9 x 10^12/L	It may decrease if the stomach ulcer is severe and bleeding occurs.
Hemoglobin	I: 130–175 g/L	May decrease, especially in cases of bleeding (hypovolemia or anemia).
Platelets	150-400 x 10^9/L	After bleeding, platelet counts may increase as the body responds to blood loss.
Reticulocytes	0.5-2.5%	It may increase with the severity of blood loss, as new red blood cells begin to be produced.
ESR (Erythrocyte Sedimentation Rate)	I: 2-10 mm/hour, Women: 3-15 mm/hour	It may increase if there is inflammation or infection.

9. **Serum gastrin determination** – in case of suspected gastrinoma
10. **Radionuclide gastroscintigraphy** – assessment of the rate of evacuation of food from the stomach

Table 6. Complications of peptic ulcer disease and its clinical picture

Complications	Surface speed, %	Clinical picture
Bleeding	10–15	• Vomiting blood • Melena (black stools) • Signs of acute blood loss
Perforation	6–20 (*?)	• Typical symptoms: "stab-like" pain in the epigastric region • Signs of pneumoperitoneum, then peritonitis
Penetration	15 (*?)	• Clinical picture – depends on how deep the wound has penetrated and which organ is affected
Pyloric and duodenal stenosis	6–15 (*?)	• As stenosis decompensation worsens: vomiting (with food eaten that day), • Belching with a foul odor, weight loss

Note: *The frequency of these complications has not been adequately studied in epidemiological studies in recent years.*

4. Treatment and prevention of stomach ulcer disease

In modern times, the prevention and treatment of diseases is a socio-economic and medical complex of society. Through measures aimed at preserving and strengthening human health, it is possible to increase the compensatory and adaptive abilities of the body and eliminate the causes that lead to the recurrence of the disease.

The following factors should be considered in the formation of peptic ulcer disease:

1. Clinical variant - the manifestation of the disease.
2. Whether or not it is associated with Helicobacter pylori.
3. The form of the disease is either newly diagnosed or recurrent.
4. The location of the ulcer – pyloric region, antral region, etc.

5. The stage of the disease is remission or exacerbation.
6. The course of the disease is mild, moderate, or severe.
7. The presence of problems - complications or their absence.

Example of a diagnosis: Type A - gastric ulcer, newly diagnosed, associated with Helicobacter pylori, pyloric ulcer, stage of exacerbation, moderate severity, uncomplicated.

General treatment principles:

1. aggravating factors: smoking, alcohol, strong tea and coffee, stopping taking nonsteroidal anti-inflammatory drugs, pyrazolone tablets, corticosteroids and other drugs.
2. Normalization of work and rest regime.
3. Diet therapy: Diet according to Table #1 (Pevzner).
4. Physiotherapeutic treatment methods used in the uncomplicated course of the disease and the absence of signs of occult bleeding, when the period of exacerbation subsides:

- Paraffin applications
- Acupuncture reflexology
- Laser therapy
- Magnetotherapy
- Hyperbaric oxygenation

In the stomach Summarizing the importance of aggressive factors in ulcer formation, the main task of treatment during the exacerbation of gastric ulcer is currently to suppress aggressive factors. This task is being successfully solved with the help of drugs (histamine H2-receptor blockers, non - absorbable antacids, anticholinergics).

Follow the principles of mechanical, thermal, and chemical preservation.

I. Eradication of Helicobacter pylori infection: Treatment is provided to patients with peptic ulcer disease during exacerbation or remission, if the test for H. pylori

infection is positive , and to all patients with complications of peptic ulcer disease after their condition has stabilized.

1. Stage 1 eradication therapy (7 days): Goals in the treatment of Helicobacter infection:

- eradication of Helicobacter pylori from the gastric and duodenal mucosa;
- Stopping (suppressing) active inflammation in the gastric and duodenal mucosa;
- Ensuring the healing of wounds and erosions ;
- Preventing the development of complications and complications , including lymphoma and stomach cancer.

a. Proton pump inhibitor:

Lansoprazole 30 mg 2 times a day.

Omeprazole 20 mg 2 times a day.

Pantoprazole 40 mg 2 times a day.

Rabeprazole 20 mg 2 times a day.

Esomeprazole 20 mg 2 times a day.

Ranitidine bismuth citrate 400 mg 2 times a day.

b. Antibiotics :

A moxicillin 1000 mg 2 times a day.

Clarithromycin 500 mg 2 times a day.

Metronidazole 400 mg 2 times a day.

Tinidazole 500 mg 2 times a day.

Control of eradication effectiveness is carried out at least 4 weeks after the end of anti-helicobacter therapy. If treatment is ineffective **,** the following is prescribed:

2. Second stage eradication therapy (7 days):

A proton pump inhibitor (or ranitidine) is taken in a standard dose 2 times a day and includes the following medications:

- Lansoprazole;
- Omeprazole;
- Pantoprazole;

- Rabeprazole;
- Esomeprazole;
- Ranitidine bismuth citrate.

In addition to these, the following are prescribed.

Bismuth salts:

Bismuth subcitrate in colloidal form 120 mg 4 times a day. Antibiotics are prescribed in addition to these.

Antibiotics :

Metronidazole 400 mg 2 times a day.

Tetracycline 500 mg 4 times a day.

II. Alternative therapy:

Proton pump inhibitor:

Omeprazole 20 mg 2 times a day.

H2 - receptor blockers:

Ranitidine 150 mg 2 times a day.

Famotidine 20 mg 2 times a day.

Treatment duration:

Peptic ulcer disease usually heals within 4-8 weeks.

Supportive therapy:

Halve the dose of the drug and maintain this dose for 1 week.

III. Symptomatic remedies:

Antacids :

Rutacid 1-2 tablets 3-4 times a day and at night.

Sucralfate 1 g 4 times a day or 2 g 2 times a day.

Note: It is recommended to take antacids before a test for Helicobacter pylori infection, as they do not affect the results .

The goal of treatment (in case of Helicobacter pylori infection):

Removal of Helicobacter pylori from the gastroduodenal mucosa.

Reducing active inflammation of the gastric mucosa.

Preventing recurrence and the development of complications.

The goal of treatment (in the absence of Helicobacter pylori infection): To reduce symptoms and improve the patient's condition.

Antisecretory drugs:

Ranitidine 300 mg once daily in the evening.

Famotidine 40 mg once daily in the evening.

Omeprazole 20 mg at 2-3 pm.

Antacids :

Maalox, Phosphalugel, Almagel , Gel, etc.

Cytoprotectors:

Sucralfate (Venter) 4 g daily or 2 g twice daily

Surgical treatment:

Indications for surgical treatment are absolute and relative, and are divided into:

Absolute guidelines:

- Wound perforation;
- Severe or recurrent gastritis and duodenal bleeding;
- Pyloroduodenal stenosis and deformations of the hard mucous membrane of the stomach, accompanied by impaired evacuation function.

Relative indications:

- Frequent recurrent ulcers that respond poorly to conservative treatment;
- Wounds that do not heal for a long time despite conservative treatment, with severe clinical symptoms (pain, vomiting, occult bleeding);
- Recurrent bleeding in history;
- proper treatment for 4-6 months;
- Recurrence after repair of a previous perforation wound;
- Ulcers caused by high acidity;

- Social constraints (lack of funds or patient's desire to take regular medications).

Note: If 3-4 hospital treatments over 4-8 weeks, despite the selection of the right treatment, do not lead to recovery or long-term remission (5-8 years), a surgical treatment strategy should be considered, as this is necessary to avoid exposing the patient to the development of dangerous complications.

Types of surgical treatment:

1. **Vagotomy** — cutting the main and branches of the vagus nerve:

a) Bilateral main vagotomy (cutting the main branches of the vagus nerve and their minor branches at a distance of at least 6 cm around the stomach);

b) Pyloroplasty or duodenoplasty with primary vagotomy (low postoperative mortality rate - less than 1%, recurrence rate 8-10%); Haeneke-Mikulich or Finney methods;

c) Selective proximal vagotomy (cutting the small branches of the Latarje nerve, which innervates the acid-producing cells located in the body and fundus of the stomach, cutting the distal branches of the Latarje nerve can lead to post-resection syndromes), the goal is partial denervation of the acid-producing zone of the stomach - the body and fundus, preserving the branch of the vagus nerve, can be combined with pyloroplasty;

d) Anterectomy with vagotomy and Billrot-1, Billrot-2 or Ru anastomoses (used to treat duodenal ulcers).

2. **Gastric resection:**

- Distal (removal of the distal part of the stomach);
- Proximal.

3. **Gastroectomy;**
4. **Surgical treatment aimed at treating wound complications:**

- Continuous gastrotomy of the stomach in case of bleeding and repair of the defect;
- Closure of the perforation hole with a double-layer suture or the Opel-Polikarpov method.

Prognosis and outcomes of ulcer disease:

- The prognosis is good in uncomplicated forms, worsens in frequently recurring forms, and is severe if complications are present.
- Successful eradication of Helicobacter pylori infection radically changes the course of the disease and prevents relapses.

Prevention of wound disease:

- Eliminate psycho-emotional stress, reduce chronic intoxications (smoking, alcohol);
- Normalization of work and rest regime;
- Rational nutrition;
- Active drug treatment against Helicobacter pylori infection;
- Supportive treatment with antisecretory and adjuvant drugs.

CHAPTER IV. Research methods and their results

1. Research materials and methods

Although available statistical data on the prevalence of chronic gastritis and peptic ulcer disease in the Republic of Uzbekistan are limited, existing studies indicate that this disease is widespread.

Helicobacter pylori infection (74.9%) indicates the widespread nature of this disease.

To address the objectives, 346 patients and 17 healthy individuals (control group) were studied between 2021 and 2024.

Of these 346 patients, 140 suffered from chronic gastritis and peptic ulcer disease, who underwent dynamic observation after various treatment regimens for peptic ulcer disease: 80 underwent eradication therapy, and 6 underwent anti-ulcer therapy.

Also, 162 patients with chronic gastritis and gastric ulcer were under dynamic observation for up to 3 years (chronic gastritis was observed to develop into duodenal ulcer, and gastric ulcer was observed to develop into duodenal ulcer after eradication).

2. Research results and discussion

Of the 346 patients, 217 were diagnosed with gastrointestinal ulcers: 60 had duodenal ulcers, 11 had esophageal ulcers, 51 had gastric ulcers, and 6 had transitory gastric-duodenal ulcers. The remaining 129 patients were diagnosed with chronic gastritis as a result of esophagogastroduodenoscopy. Morphological changes in the mucosa were confirmed in 100% of all 129 patients with chronic gastritis: chronic nonatrophic gastritis in 54 patients and chronic atrophic gastritis in 74 patients, and only 1 patient had unchanged mucosa.

The control group consisted of 13 men and 4 women aged 20–35 years, who, as a result of a thorough clinical and instrumental examination, did not reveal any pathology in their gastrointestinal tract. They had no history of either acute or chronic diseases of internal organs.

The purpose of esophagogastroduodenoscopy was to determine the condition of the mucosa, the localization and size of the ulcer, and to obtain diagnostic biopsies (from healthy tissue surrounding the ulcer).

Including: 2 biopsies from the lesser curvature of the body of the stomach, 2 from the lesser curvature of the lower part of the stomach (2–3 cm from the pylorus), and in rare cases, 1 biopsy from the duodenum.

eosin for histological examination, and the degree of Helicobacter pylori infection was determined. Helicobacter pylori infection was confirmed by morphological examination using the "gold standard", that is, biopsy samples specially stained with Giemsa stain.

acute peptic ulcer disease received eradication and antiulcer therapy. Depending on the presence of Helicobacter pylori infection, patients were divided into groups: 80 patients with Helicobacter pylori-associated ulcers, 12 patients with non-Helicobacter pylori-associated ulcers, and 7 patients with difficult-to-heal gastric and duodenal ulcers, both associated and unrelated to Helicobacter pylori.

diagnosed on the basis of endoscopic and morphological examinations were studied. In order to study the characteristics and prognosis of chronic gastritis, as well as the presence or absence of *Helicobacter pylori* infection in the gastric mucosa, these 512 patients with chronic gastritis were divided into two groups:

1. *Helicobacter pylori* infection (n = 91)
2. Chronic gastritis not associated with *Helicobacter pylori* infection (n=38)

Eradication therapy is indicated for patients with various forms of Helicobacter pylori-associated peptic ulcer disease; the combination of drugs, their doses, and duration of treatment are determined based on the degree of infection, the course of the disease, and the patient's age.

with non-Helicobacter pylori-associated disease, antiulcer drugs should be selected based on the level of gastric acid production. A complex of diagnostic and prophylactic measures has been developed for dynamic monitoring of patients with gastritis and peptic ulcer disease after successful Helicobacter pylori eradication.

CONCLUSION

of Helicobacter pylori in the gastric mucosa is accompanied by severe acute and chronic inflammation (in which the amount *of Helicobacter pylori* and the degree of inflammation are directly proportional to each other), hypertrophy of gastroduodenal G-cells and preservation of normal somatostatin are observed; in the absence of *Helicobacter pylori* infection, inflammation in the gastric mucosa is not severe, the number of gastroduodenal G-cells is normal, and the level of somatostatin is low. An inverse correlation was found between the duration of *Helicobacter pylori infection and the herpesviruses in the* stomach in patients with chronic gastritis. The distinction of the main etiopathogenetic types of chronic gastritis allows us to determine its diagnosis, treatment methods and prognosis based on a modern approach.

Morpho-functional and clinical assessment of the main forms of gastric and duodenal ulcers, depending on the presence or absence of Helicobacter pylori infection in the gastric mucosa, was studied. 217 patients were examined. *They* were diagnosed with UTI based on clinical, endoscopic and morphological examinations: 60 of them had duodenal ulcers, 11 had esophageal ulcers, 51 had gastric ulcers and 6 had transitory gastric-duodenal ulcers.

When considering the etiopathogenesis in patients with chronic gastritis (70.7% chronic gastritis associated with *Helicobacter pylori* infection , 29.3% chronic gastritis not associated with *Helicobacter pylori* infection), it was found that 16.9% of patients had autoimmune chronic gastritis, 35% of patients had antral chronic gastritis associated with *Helicobacter pylori infection* , 34% of patients had chronic gastritis, and 14.1% of patients had antral chronic gastritis not associated with *Helicobacter pylori.* infection .

As a result of monitoring patients with peptic ulcer disease, relapse of the disease was detected after successful eradication of *Helicobacter pylori infection.* According to clinical signs - in 50% of cases, according to endoscopic data - in 60% of cases (of which 15% of cases were ulcers, 45% - gastric erosion). Relapse of peptic ulcer disease caused changes in the clinical picture: in 83% of patients the

disease continued with mild symptoms or without symptoms. *Helicobacter pylori* Recurrence of infection was detected in 45% of patients.

Thus, the results of the study showed that the presence of HP infection in the gastric mucosa allowed us to identify similarities and differences between the "aggressive" and "protective" factors in the clinical course of chronic gastritis and ulcer diseases of various localization (gastric ulcer, transitory gastroduodenal ulcer , duodenal ulcer). At the same time, the absence of *Helicobacter pylori* infection in the gastric mucosa also indicates similarities and differences between the "aggressive" and "protective" factors in the clinical course. However, clinically, similar symptoms are observed in 13% of patients with chronic gastritis associated with *Helicobacter pylori and in 8% of patients with chronic gastritis not associated with Helicobacter pylori* . A number of authors included 22 patients in the group of chronic gastritis, who were considered to be at high risk of developing ulcer disease.

PRACTICAL RECOMMENDATIONS

The standard guidelines for medical care for patients with chronic gastritis should include: *Helicobacter pylori in the blood fluid* It is necessary to determine the levels of antibodies against the infection, as well as prescribe appropriate differential treatment.

Helicobacter pylori Patients with chronic gastritis associated with infection should undergo a mandatory morphological examination, and if atrophy of the gastric mucosa is detected, eradication treatment should be performed.

The high activity and rapid progression of the atrophic process of the gastric mucosa in patients with autoimmune chronic gastritis require the detection of the presence of the herpes virus in the gastric mucosa using immunogastrochemical tests. If the virus is detected, antiviral treatment is necessary.

Patients with peptic ulcer disease of various localization should be tested for *Helicobacter pylori. If the test result is positive, eradication treatment should be carried out. The combination of drugs, their doses and duration of treatment are determined depending on the degree of Helicobacter pylori* infection, the course of the disease and the age of the patient; if *the Helicobacter pylori* test result is negative, it is necessary to select an antiulcer drug, which depends on the initial level of acid production.

Helicobacter pylori Patients with peptic ulcer disease associated with Helicobacter pylori infection should be monitored annually for 5 years after successful eradication therapy. If *Helicobacter pylori* infection is detected, re-eradication therapy with second- or third-line drugs is appropriate. If *Helicobacter pylori* If the infection test is negative, treatment with a proton pump inhibitor at a standard dose for one month is necessary, which is carried out during the expected seasonal inflammatory period. Subsequently, gastroscopy should be performed only if indicated.

Case of topics

Case 1 (Ulcer malignization and an "indeterminate biopsy")

A 49-year-old man presents with gradually progressive, persistent epigastric pain over the past year, not clearly related to food intake, marked loss of appetite, and a 7-kg weight loss over 6 months. He has been smoking for more than 20 years and has been regularly taking NSAIDs without medical supervision. Physical examination reveals pallor and tenderness on deep palpation in the epigastric region. Upper GI endoscopy shows an ulcer in the body of the stomach with irregular, infiltrated margins, a firm base, and contact bleeding. The initial biopsy is reported as "indeterminate (dysplasia or suspicious for malignancy)."

Tasks:

- **Diagnosis:** Justify the most likely diagnosis (gastric ulcer suspicious for malignant transformation).
- **Differential diagnosis:** Ulcerated gastric carcinoma, benign gastric ulcer, MALT lymphoma, gastric involvement of Crohn's disease, NSAID-related deep erosion.
- **Pathogenesis:** Chronic mucosal injury (NSAIDs → reduced prostaglandins), smoking-related microcirculatory impairment, possible *Helicobacter pylori*–associated inflammation → atrophy → dysplasia → malignant transformation.
- **Management:** Urgent repeat endoscopy with 6–8 deep biopsies, endoscopic ultrasound to assess wall layers, abdominal CT to exclude metastases, *H. pylori* testing; urgent oncogastroenterology referral if bleeding or obstruction is present.
- **Prognosis:** Favorable if detected early; poor if diagnosed late due to lymphatic and hematogenous metastases.

Case 2 (PPI-refractory dyspepsia after two eradication attempts)

A 32-year-old woman reports recurrent burning and stabbing epigastric pain, heartburn, acid regurgitation, and postprandial discomfort for 3 years. Symptoms improve temporarily with treatment but never resolve. She underwent standard triple *H. pylori* eradication therapy twice in the past 2 years. Intragastric pH monitoring shows hyperacidity. Symptoms worsen during stress.

Tasks:

- **Diagnosis:** PPI-refractory dyspepsia (consider resistant *H. pylori*, hyperacid gastritis, suspected Zollinger–Ellison syndrome, overlap with GERD).
- **Differential diagnosis:** GERD, functional dyspepsia, bile reflux gastritis, gastrinoma (ZES), drug- or alcohol-related gastritis, IBS overlap.
- **Pathogenesis:** Eradication failure (antibiotic resistance, poor adherence, reinfection), persistent acid injury, visceral hypersensitivity.
- **Management:** Confirm *H. pylori* status (^{13}C breath test or stool antigen), antibiotic susceptibility testing and quadruple therapy if needed, optimize PPI dosing, repeat endoscopy; measure fasting gastrin if hyperacidity persists.
- **Prognosis:** Good with individualized therapy; uncontrolled disease increases ulcer and bleeding risk.

Case 3 (Upper GI bleeding with risk of hypovolemic shock)

A 58-year-old man is admitted urgently with severe weakness, dizziness, palpitations, and melena over the last 24 hours. He has a 5-year history of gastric ulcer disease and recently used NSAIDs and antiplatelet agents. BP is 90/60 mmHg, HR 110/min, skin is pale and clammy. Hemoglobin is 85 g/L.

Tasks:

- **Diagnosis:** Active upper GI bleeding from a gastric ulcer.
- **Differential diagnosis:** Hemorrhagic erosive gastritis, esophageal or gastric variceal bleeding, Mallory–Weiss syndrome, bleeding gastric tumor.
- **Pathogenesis:** Erosion of a vessel at the ulcer base with acid-peptic aggression and impaired hemostasis.
- **Management:** ABC approach, large-bore IV access, fluids and blood products as indicated, high-dose IV PPI, urgent therapeutic endoscopy (injection, clipping, thermal coagulation); embolization or surgery if endoscopic therapy fails.
- **Prognosis:** Favorable with early hemostasis; mortality increases with rebleeding, age, comorbidities, or delayed care.

Case 4 (Intestinal metaplasia with active inflammation)

A 41-year-old man has had dyspeptic symptoms for over 5 years. Recently, epigastric burning, postprandial pain, and early satiety have worsened. He has a history of incompletely treated *H. pylori* gastritis. Endoscopy shows antral hyperemia and mucosal fragility. Biopsy reveals active inflammation with intestinal metaplasia.

Tasks:

- **Diagnosis:** Chronic active *H. pylori*–associated gastritis complicated by intestinal metaplasia.
- **Differential diagnosis:** Autoimmune atrophic gastritis, chemical (bile reflux) gastritis, long-term drug-induced gastritis, early neoplasia.
- **Pathogenesis:** Persistent *H. pylori* infection → chronic inflammation → epithelial damage → intestinal metaplasia → increased cancer risk.
- **Management:** Complete *H. pylori* eradication, endoscopic surveillance based on OLGA/OLGIM staging, lifestyle modification.
- **Prognosis:** Depends on extent of metaplasia; risk of gastric adenocarcinoma if not monitored.

Case 5 (Autoimmune atrophic gastritis with vitamin B12 deficiency)

A 66-year-old woman with a 10-year history of atrophic gastritis presents with fatigue, dizziness, memory impairment, and paresthesias. Labs show megaloblastic anemia. Epigastric pressure-like pain has recently intensified.

Tasks:

- **Diagnosis:** Autoimmune atrophic gastritis complicated by pernicious anemia.
- **Differential diagnosis:** *H. pylori*–related atrophic gastritis, gastric malignancy, peptic ulcer disease, bile reflux, ischemic heart disease.
- **Pathogenesis:** Autoantibodies to parietal cells and intrinsic factor → achlorhydria and impaired B12 absorption → hematologic and neurologic manifestations; increased risk of carcinoid and adenocarcinoma.

- **Management:** Lifelong vitamin B12 replacement, regular endoscopic surveillance, screening for other autoimmune diseases.
- **Prognosis:** Good with early diagnosis and follow-up; untreated cases risk neurologic damage and cancer.

Case 6 (Stress-related erosive gastritis)

A 27-year-old man develops acute epigastric pain, nausea, and repeated vomiting after 72 hours of severe emotional stress and sleep deprivation. Endoscopy reveals multiple superficial erosions with petechial bleeding.

Tasks:

- **Diagnosis:** Acute stress-related erosive gastritis.
- **Differential diagnosis:** NSAID-induced gastritis, acute *H. pylori* gastritis, GERD, acute pancreatitis, food poisoning.
- **Pathogenesis:** Stress → sympathetic activation → splanchnic hypoperfusion → mucosal ischemia → erosions and bleeding.
- **Management:** High-dose PPI, mucosal protectants, antiemetics, fluid therapy; inpatient monitoring if bleeding risk is high.
- **Prognosis:** Favorable with early treatment; recurrent stress increases bleeding risk.

Case 7 (Cicatricial pyloric stenosis)

A 54-year-old man with an 8-year history of recurrent gastric ulcer presents with postprandial fullness, repeated vomiting of stale food, weight loss, and dehydration. Endoscopy shows severe pyloric narrowing with scarring.

Tasks:

- **Diagnosis:** Cicatricial pyloric stenosis secondary to peptic ulcer disease.
- **Differential diagnosis:** Malignant gastric outlet obstruction, duodenal ulcer stenosis, functional gastroparesis.
- **Pathogenesis:** Chronic ulceration → fibrosis and scarring → gastric outlet obstruction → vomiting and metabolic alkalosis.

- **Management:** Correct fluids and electrolytes, gastric decompression, consider endoscopic balloon dilation; surgery if unsuccessful.
- **Prognosis:** Good with timely intervention; delayed treatment leads to severe metabolic disturbances.

Case 8 (Deep ulcer with nocturnal pain)

A 38-year-old woman reports nocturnal and fasting epigastric burning pain relieved temporarily by food or milk. Endoscopy shows a small but deep ulcer near the lesser curvature with hyperacidity.

Tasks:

- **Diagnosis:** Hyperacid peptic gastric ulcer.
- **Differential diagnosis:** Duodenal ulcer, GERD, functional dyspepsia, mild acute pancreatitis, bile reflux gastritis.
- **Pathogenesis:** Acid-peptic aggression exceeding mucosal defense; role of *H. pylori*, smoking, and stress.
- **Management:** *H. pylori* testing and eradication, appropriate PPI therapy, risk-factor modification; repeat endoscopy to confirm healing.
- **Prognosis:** Favorable with adherence; noncompliance increases bleeding and perforation risk.

Case 9 (Perforation with misleading pain relief)

A 61-year-old man experiences sudden severe “knife-like” epigastric pain followed by apparent pain relief but rapid clinical deterioration. Examination reveals a rigid abdomen. Imaging shows free subdiaphragmatic air.

Tasks:

- **Diagnosis:** Perforated peptic ulcer with generalized peritonitis.
- **Differential diagnosis:** Acute pancreatitis, perforated appendicitis, mesenteric ischemia, acute cholecystitis, bowel perforation.
- **Pathogenesis:** Ulcer penetration through serosa → leakage of gastric contents → chemical then bacterial peritonitis; transient pain relief due to phase transition.

- **Management:** Emergency surgery, broad-spectrum antibiotics, aggressive resuscitation, ICU care.
- **Prognosis:** Time-dependent; early surgery improves survival.

Case 10 (H. pylori–negative hypoacid atrophic gastritis)

A 35-year-old woman has long-standing dyspepsia with early satiety and bloating. Endoscopy shows pale, thinned mucosa; pH-metry reveals hypoacidity; *H. pylori* tests are negative. Biopsy confirms glandular atrophy.

Tasks:

- **Diagnosis:** Hypoacid atrophic gastritis.
- **Differential diagnosis:** Autoimmune gastritis, bile reflux gastritis, long-term PPI-related atrophy, endocrine disorders.
- **Pathogenesis:** Glandular atrophy → reduced acid and enzyme secretion → impaired digestion and dysbiosis.
- **Management:** Assess B12 and iron, autoantibody testing, symptomatic therapy, scheduled surveillance.
- **Prognosis:** Progressive atrophy increases precancerous risk.

Case 11 (Alcohol-related erosive gastritis)

A 59-year-old man with long-term alcohol use presents with epigastric pain, heartburn, and occasional hematemesis. Endoscopy shows multiple erosions with hemorrhagic foci.

Tasks:

- **Diagnosis:** Chronic alcohol-related erosive gastritis.
- **Differential diagnosis:** NSAID gastritis, *H. pylori* gastritis, portal hypertensive gastropathy, peptic ulcer disease.
- **Pathogenesis:** Direct ethanol toxicity → disruption of mucosal barrier → microcirculatory impairment → erosions and bleeding.
- **Management:** Complete alcohol cessation, PPI therapy, mucosal protectants, inpatient care if bleeding risk is high.

- **Prognosis:** Continued alcohol use markedly increases bleeding and ulcer risk.

Case 12 (Recurrent ulcer due to improper PPI use)

A 44-year-old man treated for gastric ulcer develops recurrent epigastric pain within one year. History reveals incorrect PPI dosing and intake with meals.

Tasks:

- **Diagnosis:** Recurrent gastric ulcer.
- **Differential diagnosis:** *H. pylori* reinfection, NSAID use, smoking, gastrinoma.
- **Pathogenesis:** Inadequate acid suppression → impaired healing → relapse.
- **Management:** Correct PPI regimen, reassess *H. pylori*, eliminate risk factors, follow-up endoscopy.
- **Prognosis:** High recurrence without preventive measures.

Case 13 (High-grade dysplasia with refusal of intervention)

A 70-year-old woman with chronic gastritis is diagnosed with high-grade dysplasia but refuses endoscopic or surgical treatment.

Tasks:

- **Diagnosis:** High-grade gastric epithelial dysplasia.
- **Differential diagnosis:** Early gastric carcinoma, reactive atypia.
- **Pathogenesis:** Chronic inflammation and metaplasia leading to dysplasia.
- **Management:** Informed consent discussion, offer alternative endoscopic options, close surveillance.
- **Prognosis:** High risk of malignant transformation without treatment.

Case 14 (*H. pylori* positive, normal acidity, lymphoplasmacytic infiltration)

A 33-year-old man is incidentally found to be *H. pylori* positive with normal acid secretion. Biopsy shows lymphoplasmacytic infiltration.

Tasks:

- **Diagnosis:** Chronic active *H. pylori*–associated gastritis.
- **Differential diagnosis:** Autoimmune gastritis, early MALT lymphoma.

- **Pathogenesis:** Local immune response to *H. pylori* antigens.
- **Management:** Eradication therapy, confirm cure, endoscopic follow-up.
- **Prognosis:** Regression with treatment; progression risk if untreated.

Case 15 (Change in ulcer characteristics and treatment resistance)

A 46-year-old man with a 6-year history of gastric ulcer develops constant, nocturnal pain unresponsive to therapy. Endoscopy shows hardened ulcer margins and an irregular base.

Tasks:

- **Diagnosis:** Gastric ulcer suspicious for malignization.
- **Differential diagnosis:** Penetrating ulcer, resistant *H. pylori* infection, continued NSAID exposure.
- **Pathogenesis:** Chronic ulceration → persistent inflammation → cellular atypia → malignant transformation.
- **Management:** Immediate multiple biopsies, EUS/CT, oncologic consultation.
- **Prognosis:** Significantly improved with early detection; poor in advanced stages.

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