

MINISTRY OF HEALTHCARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"

"Approved"

at the meeting of internal
medicine №1 department

Head of Department
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**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Current practice of internal medicine
<i>Content module</i>	Management of the patients with main symptoms and syndromes in gastroenterology clinic
<i>Study subject</i>	Management of the patients with dysphagia and heartburn Management of the patients with dyspepsia
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

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1. Relevance of the topic: Esophageal motility disorders are diseases attributable to esophageal neuromuscular dysfunction commonly associated with dysphagia, chest pain, or heartburn. The major entities are achalasia, diffuse esophageal spasm and GERD. Motility disorders can also be secondary to broader disease processes as is the case with pseudoachalasia, Chagas' disease, and scleroderma. Gastroesophageal reflux disease is prevalent in heartburn reasons. Most cases of heartburn occur because of excess acid reflux. Gastro-esophageal reflux disease (GERD) is a chronic and relapsing disease that results from the retrograde flow of gastric contents into the esophagus (gastro-esophageal reflux), oropharynx, and/or respiratory tract and causes troublesome symptoms or complications and/or mucosal injury. Functional dyspepsia (FD) is a medical condition that is characterized by one or more of the following symptoms: epigastric pain, epigastric burning, postprandial fullness, and early satiation that are unexplained after a routine clinical evaluation.

2. The main goal: To be able to choose and put into practice the approach to the patient with dysphagia, dyspepsia and heartburn, to determine tactics of treatment and prophylaxis.

Specific goals:

- To select the information indicating the presence of GERD, achalasia and FD in a patient from the data history;
- To create a scheme of diagnostic search;
- To identify the signs of GERD, achalasia and FD in an objective study of the patient (general examination, palpation, percussion, auscultation);
- To analyze and interpret the changes in the results of the laboratory and instrumental methods of investigation, depending on the course of the disease;
- To formulate and justify a preliminary diagnosis of GERD, achalasia and FD according to classification;
- To conduct differential diagnostics of diseases with the heartburn, dyspepsia, dysphagia;
- To develop a strategy of treatment depending on the disease and the existing complications;
- To provide medical care;
- To assess the patient's prognosis and to propose a plan of preventive actions;
- To apply deontological communication skills with patients.

3. Basic knowledge, abilities, skills (interdisciplinary integration)

Discipline	To know	To be able to
Anatomy	The structure of the gastrointestinal tract, blood supply, innervation	
Histology	The structure of the wall of the esophagus, stomach, intestines in health and disease	To interpret results of endoscopy with biopsy
Regional anatomy	Interposition of the gastrointestinal organs	
Physiology	Indicators of gastrointestinal tract function, its value	To determine the function of gastrointestinal organs
Morbid anatomy	Changes in the structure of gastrointestinal tract organs in pathology	
Radiology	Radiological changes at pathology of gastrointestinal organs	Analyze the radiological picture of the chest cavity and abdominal cavity

Prophylaxis	Diseases with such leading symptoms as heartburn, dyspepsia, dysphagia	Conduct an objective examination of the patient, analyze the clinical and laboratory results
Pharmacology	The mechanism of action, indications and contraindications for the PPI, H ₂ -blockers, antacids, prokinetics, antibiotics, antidiarrheal drugs	Prescribe the drugs of these groups

4. Do the tasks for independent work during preparation for classes.

4.1. The list of key terms, parameters, characteristics:

Term	Definition
Gastro-esophageal reflux disease	is a chronic and relapsing disease that results from the retrograde flow of gastric contents into the esophagus (gastro-esophageal reflux), oropharynx, and/or respiratory tract and causes troublesome symptoms or complications and/or mucosal injury.
Functional dyspepsia	is a medical condition that is characterized by one or more of the following symptoms: epigastric pain, epigastric burning, postprandial fullness, and early satiation that are unexplained after a routine clinical evaluation.
Inhibitors of proton pump (PPI)	group of drugs which suppress the production of stomach acid and work by inhibiting the molecule in the stomach glands that is responsible for acid secretion (the gastric acid pump).
H ₂ -blockers	group of which that interfere with acid production by blocking or antagonizing the actions of histamine.
Prokinetics	group of drugs which enhances gastrointestinal motility by increasing the frequency of contractions or making them stronger.
Dysphagia	difficulty with swallowing.
Odynophagia	refers to painful swallowing, typically resulting from mucosal ulceration within the oropharynx or esophagus.
Globus pharyngeus	is a foreign body sensation localized in the neck that does not interfere with swallowing and sometimes is relieved by swallowing.

4.2. Theoretical questions for the lesson:

1. Give the definitions of heartburn, dysphagia, dyspepsia, GERD, achalasia and FD.
2. Specify the risk factors for GERD, achalasia and FD.
3. The pathophysiological mechanisms of heartburn, dysphagia, dyspepsia.
4. Diagnostic criteria of GERD, achalasia and FD.

5. What are the endoscopic characteristics of GERD, achalasia?
6. Modern classification of GERD, achalasia and FD.
7. Specify the principles and features of GERD, achalasia and FD pharmacotherapy according to modern recommendations.
8. What lifestyle modifications should be recommended for patients with GERD, achalasia and FD?

4.3. Practical tasks that are performed in class:

1. IPP include:

- 1) Famotidine
- 2) Itoprid

3) Pantoprazole

- 4) Clarithromycin

2. H₂-blockers include:

1) Famotidine

- 2) Itoprid
- 3) Pantoprazole
- 4) Clarithromycin

3. Prokinetics include:

- 1) Famotidine

2) Itopride

- 3) Pantoprazole
- 4) Clarithromycin

4. Standard dosage of Omeprazole is:

- 1) 40 mg
- 2) 60 mg

3) 20 mg

- 4) 20-40 mg

5. Standard dosage of Pantoprazole is:

- 1) 20 mg
- 2) 60 mg

3) 40 mg

- 4) 20-40 mg

6. Standard dosage of Rabeprazole is:

- 1) 40 mg
- 2) 60 mg

3) 20 mg

- 4) 20-40 mg

7. Standard dosage of Esomeprazole is:

- 1) 20 mg
- 2) 60 mg

3) 40 mg

- 4) 20-40 mg

8. Standard dosage of Lansoprazole is:

- 1) 20 mg
- 2) 60 mg

3) 40 mg

4) **30 mg**

9. Atypical symptom of GERD is:

1) **chronic cough**

2) heartburn

3) chest pain

4) nausea

10. What are the types of FD?

1) typical, atypical

2) **postprandial distress syndrome, epigastric pain syndrome**

3) pain syndrome, dyspeptic syndrome

4) A, B, C and D types

TopicContent

MANAGEMENT OF THE PATIENTS WITH DYSPHAGIA

Dysphagia — difficulty with swallowing — refers to problems with the transit of food or liquid from the mouth to the hypopharynx or through the esophagus. Severe dysphagia can compromise nutrition, cause aspiration, and reduce quality of life. Additional terminology pertaining to swallowing dysfunction is as follows. **Aphagia** (inability to swallow) typically denotes complete esophageal obstruction, most commonly encountered in the acute setting of a food bolus or foreign body impaction. **Odynophagia** refers to painful swallowing, typically resulting from mucosal ulceration within the oropharynx or esophagus. It commonly is accompanied by dysphagia, but the converse is not true. **Globus pharyngeus** is a foreign body sensation localized in the neck that does not interfere with swallowing and sometimes is relieved by swallowing. Transfer dysphagia frequently results in nasal regurgitation and pulmonary aspiration during swallowing and is characteristic of oropharyngeal dysphagia. **Phagophobia** (fear of swallowing) and refusal to swallow may be psychogenic or related to anticipatory anxiety about food bolus obstruction, odynophagia, or aspiration.

Classification. Dysphagia can be subclassified both by location and by the circumstances in which it occurs. With respect to location, distinct considerations apply to oral, pharyngeal, or esophageal dysphagia. Normal transport of an ingested bolus depends on the consistency and size of the bolus, the caliber of the lumen, the integrity of peristaltic contraction, and deglutitive inhibition of both the upper esophageal sphincter (UES) and the lower esophageal sphincter (LES). Dysphagia caused by an oversized bolus or a narrow lumen is called structural dysphagia, whereas dysphagia due to abnormalities of peristalsis or impaired sphincter relaxation after swallowing is called propulsive or motor dysphagia.

Etiology and pathogenesis. More than one mechanism may be operative in a patient with dysphagia. Scleroderma commonly presents with absent peristalsis as well as a weakened LES that predisposes patients to peptic stricture formation. Likewise, radiation therapy for head and neck cancer may compound the functional deficits in the oropharyngeal swallow attributable to the tumor and cause cervical esophageal stenosis.

Oral and Pharyngeal (Oropharyngeal) Dysphagia. Oral-phase dysphagia is associated with poor bolus formation and control so that food has prolonged retention within the oral cavity and may seep out of the mouth. Drooling and difficulty in initiating swallowing are other characteristic signs. Poor bolus control also may lead to premature spillage of food into the hypopharynx with resultant aspiration into the trachea or regurgitation into the nasal cavity.

Pharyngeal-phase dysphagia is associated with retention of food in the pharynx due to

poor tongue or pharyngeal propulsion or obstruction at the UES. Signs and symptoms of concomitant hoarseness or cranial nerve dysfunction may be associated with oropharyngeal dysphagia. Oropharyngeal dysphagia may be due to neurologic, muscular, structural, iatrogenic, infectious, and metabolic causes. Iatrogenic, neurologic, and structural pathologies are most common. Iatrogenic causes include surgery and radiation, often in the setting of head and neck cancer. Neurogenic dysphagia resulting from cerebrovascular accidents, Parkinson's disease, and amyotrophic lateral sclerosis is a major source of morbidity related to aspiration and malnutrition. Medullary nuclei directly innervate the oropharynx. Lateralization of pharyngeal dysphagia implies either a structural pharyngeal lesion or a neurologic process that selectively targeted the ipsilateral brainstem nuclei or cranial nerve. Advances in functional brain imaging have elucidated an important role of the cerebral cortex in swallow function and dysphagia. Asymmetry in the cortical representation of the pharynx provides an explanation for the dysphagia that occurs as a consequence of unilateral cortical cerebrovascular accidents. Oropharyngeal structural lesions causing dysphagia include Zenker's diverticulum, cricopharyngeal bar, and neoplasia. Zenker's diverticulum typically is encountered in elderly patients, with an estimated prevalence between 1:1000 and 1:10,000. In addition to dysphagia, patients may present with regurgitation of particulate food debris, aspiration, and halitosis. The pathogenesis is related to stenosis of the cricopharyngeus that causes diminished opening of the UES and results in increased hypopharyngeal pressure during swallowing with development of a pulsion diverticulum immediately above the cricopharyngeus in a region of potential weakness known as Killian's dehiscence. A cricopharyngeal bar, appearing as a prominent indentation behind the lower third of the cricoid cartilage, is related to Zenker's diverticulum in that it involves limited distensibility of the cricopharyngeus and can lead to the formation of a Zenker's diverticulum. However, a cricopharyngeal bar is a common radiographic finding, and most patients with transient cricopharyngeal bars are asymptomatic, making it important to rule out alternative etiologies of dysphagia before treatment. Furthermore, cricopharyngeal bars may be secondary to other neuromuscular disorders.

Since the pharyngeal phase of swallowing occurs in less than a second, rapid-sequence fluoroscopy is necessary to evaluate for functional abnormalities. Adequate fluoroscopic examination requires that the patient be conscious and cooperative. The study incorporates recordings of swallow sequences during ingestion of food and liquids of varying consistencies. The pharynx is examined to detect bolus retention, regurgitation into the nose, or aspiration into the trachea. Timing and integrity of pharyngeal contraction and opening of the UES with a swallow are analyzed to assess both aspiration risk and the potential for swallow therapy. Structural abnormalities of the oropharynx, especially those which may require biopsies, also should be assessed by direct laryngoscopic examination.

Esophageal Dysphagia. The adult esophagus measures 18–26 cm in length and is anatomically divided into the cervical esophagus, extending from the pharyngoesophageal junction to the suprasternal notch, and the thoracic esophagus, which continues to the diaphragmatic hiatus. When distended, the esophageal lumen has internal dimensions of about 2 cm in the anteroposterior plane and 3 cm in the lateral plane. Solid food dysphagia becomes common when the lumen is narrowed to <13 mm but also can occur with larger diameters in the setting of poorly masticated food or motor dysfunction. Circumferential lesions are more likely to cause dysphagia than are lesions that involve only a partial circumference of the esophageal wall. The most common structural causes of dysphagia are Schatzki's rings, eosinophilic esophagitis, and peptic strictures. Dysphagia also occurs in the setting of gastroesophageal reflux disease without a stricture, perhaps on the basis of altered esophageal sensation, distensibility, or motor function.

Propulsive disorders leading to esophageal dysphagia result from abnormalities of peristalsis and/or deglutitive inhibition, potentially affecting the cervical or thoracic esophagus. Since striated muscle pathology usually involves both the oropharynx and the cervical esophagus, the clinical manifestations usually are dominated by oropharyngeal dysphagia.

Diseases affecting smooth muscle involve both the thoracic esophagus and the LES. A dominant manifestation of this, absent peristalsis, refers to either the complete absence of swallow-induced contraction or the presence of nonperistaltic, disordered contractions. Absent peristalsis and failure of deglutitive LES relaxation are the defining features of achalasia. In diffuse esophageal spasm (DES), LES function is normal, with the disordered motility restricted to the esophageal body. Absent peristalsis combined with severe weakness of the LES is a nonspecific pattern commonly found in patients with scleroderma.

Approach to the patient. The patient history is extremely valuable in making a presumptive diagnosis or at least substantially restricting the differential diagnoses in most patients. Key elements of the history are the localization of dysphagia, the circumstances in which dysphagia is experienced, other symptoms associated with dysphagia, and progression. Dysphagia that localizes to the suprasternal notch may indicate either an oropharyngeal or an esophageal etiology as distal dysphagia is referred proximally about 30% of the time. Dysphagia that localizes to the chest is esophageal in origin. Nasal regurgitation and tracheobronchial aspiration manifest by coughing with swallowing are hallmarks of oropharyngeal dysphagia. Severe cough with swallowing may also be a sign of a tracheoesophageal fistula. The presence of hoarseness may be another important diagnostic clue. When hoarseness precedes dysphagia, the primary lesion is usually laryngeal; hoarseness that occurs after the development of dysphagia may result from compromise of the recurrent laryngeal nerve by a malignancy. The type of food causing dysphagia is a crucial detail. Intermittent dysphagia that occurs only with solid food implies structural dysphagia, whereas constant dysphagia with both liquids and solids strongly suggests a motor abnormality. Two caveats to this pattern are that despite having a motor abnormality, patients with scleroderma generally develop mild dysphagia for solids only and, somewhat paradoxically, that patients with oropharyngeal dysphagia often have greater difficulty managing liquids than solids. Dysphagia that is progressive over the course of weeks to months raises concern for neoplasia. Episodic dysphagia to solids that is unchanged over years indicates a benign disease process such as a Schatzki's ring or eosinophilic esophagitis. Food impaction with a prolonged inability to pass an ingested bolus even with ingestion of liquid is typical of a structural dysphagia. Chest pain frequently accompanies dysphagia whether it is related to motor disorders, structural disorders, or reflux disease. A prolonged history of heartburn preceding the onset of dysphagia is suggestive of peptic stricture and, infrequently, esophageal adenocarcinoma. A history of prolonged nasogastric intubation, esophageal or head and neck surgery, ingestion of caustic agents or pills, previous radiation or chemotherapy, or associated mucocutaneous diseases may help isolate the cause of dysphagia. With accompanying odynophagia, which usually is indicative of ulceration, infectious or pill-induced esophagitis should be suspected. In patients with AIDS or other immunocompromised states, esophagitis due to opportunistic infections such as *Candida*, herpes simplex virus, or cytomegalovirus and to tumors such as Kaposi's sarcoma and lymphoma should be considered. A strong history of atopy increases concerns for eosinophilic esophagitis.

Physical examination is important in the evaluation of oral and pharyngeal dysphagia because dysphagia is usually only one of many manifestations of a more global disease process. Signs of bulbar or pseudobulbar palsy, including dysarthria, dysphonia, ptosis, tongue atrophy, and hyperactive jaw jerk, in addition to evidence of generalized neuromuscular disease, should be elicited. The neck should be examined for thyromegaly. A careful inspection of the mouth and pharynx should disclose lesions that may interfere with passage of food. Missing dentition can interfere with mastication and exacerbate an existing cause of dysphagia. Physical examination is less helpful in the evaluation of esophageal dysphagia as most relevant pathology is restricted to the esophagus. The notable exception is skin disease. Changes in the skin may suggest a diagnosis of scleroderma or mucocutaneous diseases such as pemphigoid, lichen planus and epidermolysis bullosa, all of which can involve the esophagus.

Although most instances of dysphagia are attributable to benign disease processes, dysphagia is also a cardinal symptom of several malignancies, making it an important symptom

to evaluate. Cancer may result in dysphagia due to intraluminal obstruction (esophageal or proximal gastric cancer, metastatic deposits), extrinsic compression (lymphoma, lung cancer), or paraneoplastic syndromes. Even when not attributable to malignancy, dysphagia is usually a manifestation of an identifiable and treatable disease entity, making its evaluation beneficial to the patient and gratifying to the practitioner. The specific diagnostic algorithm to pursue is guided by the details of the history.

If oral or pharyngeal dysphagia is suspected, a fluoroscopic swallow study, usually done by a swallow therapist, is the procedure of choice.

Otolaryngoscopic and neurologic evaluation also can be important, depending on the circumstances. For suspected esophageal dysphagia, upper endoscopy is the single most useful test. Endoscopy allows better visualization of mucosal lesions than does barium radiography and also allows one to obtain mucosal biopsies.

Endoscopic or histologic abnormalities are evident in the leading causes of esophageal dysphagia: Schatzki ring, gastroesophageal reflux disease and eosinophilic esophagitis. Furthermore, therapeutic intervention with esophageal dilation can be done as part of the procedure if it is deemed necessary. The emergence of eosinophilic esophagitis as a leading cause of dysphagia in both children and adults has led to the recommendation that esophageal mucosal biopsies be obtained routinely in the evaluation of unexplained dysphagia even if endoscopically identified esophageal mucosal lesions are absent. For cases of suspected esophageal motility disorders, endoscopy is still the appropriate initial evaluation as neoplastic and inflammatory conditions can secondarily produce patterns of either achalasia or esophageal spasm.

Esophageal manometry is done if dysphagia is not adequately explained by endoscopy or to confirm the diagnosis of a suspected esophageal motor disorder.

Barium radiography can provide useful adjunctive information in cases of subtle or complex esophageal strictures, prior esophageal surgery, esophageal diverticula, or paraesophageal herniation.

In specific cases, computed tomography (CT) examination and endoscopic ultrasonography may be useful.

Treatment. Treatment of dysphagia depends on both the locus and the specific etiology. Oropharyngeal dysphagia most commonly results from functional deficits caused by neurologic disorders. In such circumstances, the treatment focuses on utilizing postures or maneuvers devised to reduce pharyngeal residue and enhance airway protection learned under the direction of a trained swallow therapist. Aspiration risk may be reduced by altering the consistency of ingested food and liquid.

Dysphagia resulting from a cerebrovascular accident usually, but not always, spontaneously improves within the first few weeks after the event. More severe and persistent cases may require gastrostomy and enteral feeding. Patients with myasthenia gravis and polymyositis may respond to medical treatment of the primary neuromuscular disease.

Surgical intervention with cricopharyngeal myotomy is usually not helpful, with the exception of specific disorders such as the idiopathic cricopharyngeal bar, Zenker's diverticulum, and oculopharyngeal muscular dystrophy.

Chronic neurologic disorders such as Parkinson's disease and amyotrophic lateral sclerosis may manifest with severe oropharyngeal dysphagia. Feeding by a nasogastric tube or an endoscopically placed gastrostomy tube may be considered for nutritional support; however, these maneuvers do not provide protection against aspiration of salivary secretions or refluxed gastric contents. Treatment of esophageal dysphagia is covered in detail in Chap. 347.

The majority of causes of esophageal dysphagia are effectively managed by means of esophageal dilatation using bougie or balloon dilators.

Cancer and achalasia are often managed surgically, although endoscopic techniques are available for both palliation and primary therapy, respectively. Infectious etiologies respond to antimicrobial medications or treatment of the underlying immunosuppressive state.

Finally, eosinophilic esophagitis has emerged as an important cause of dysphagia that is amenable to treatment by elimination of dietary allergens or administration of swallowed, topically acting glucocorticoids.

Achalasia

Achalasia is a rare disease caused by loss of ganglion cells within the esophageal myenteric plexus with a population incidence of about 1:100,000 and usually presenting between age 25 and 60. The disease involves both excitatory (cholinergic) and inhibitory (nitric oxide) ganglionic neurons.

Functionally, inhibitory neurons mediate deglutitive lower esophageal sphincter (LES) relaxation and the sequential propagation of peristalsis. Their absence leads to impaired deglutitive LES relaxation and absent peristalsis.

Long-standing achalasia is characterized by progressive dilatation and sigmoid deformity of the esophagus with hypertrophy of the LES.

Clinical manifestations may include dysphagia, regurgitation, chest pain, and weight loss. Most patients report solid and liquid food dysphagia. Regurgitation occurs when food, fluid, and secretions are retained in the dilated esophagus. Patients with advanced achalasia are at risk for bronchitis, pneumonia, or lung abscess from chronic regurgitation and aspiration. Chest pain is frequent early in the course of achalasia, thought to result from esophageal spasm. Patients describe a squeezing, pressure-like retrosternal pain, sometimes radiating to the neck, arms, jaw, and back. Paradoxically, some patients complain of heartburn that may be a chest pain equivalent. Treatment of achalasia is less effective in relieving chest pain than it is in relieving dysphagia or regurgitation.

The differential diagnosis of achalasia includes DES, Chagas' disease, and pseudoachalasia. Chagas' disease is endemic in areas of central Brazil, Venezuela, and northern Argentina and spread by the bite of the reduviid (kissing) bug that transmits the protozoan, *Trypanosoma cruzi*. The chronic phase of the disease develops years after infection and results from destruction of autonomic ganglion cells throughout the body, including the heart, gut, urinary tract, and respiratory tract. Tumor infiltration, most commonly seen with carcinoma in the gastric fundus or distal esophagus, can mimic idiopathic achalasia. The resultant "pseudoachalasia" accounts for up to 5% of suspected cases and is more likely with advanced age, abrupt onset of symptoms (<1 year), and weight loss.

Hence, endoscopy is a necessary part of the evaluation of achalasia. When the clinical suspicion for pseudoachalasia is high and endoscopy nondiagnostic, computed tomography (CT) scanning or EUS may be of value. Rarely, pseudoachalasia can result from a paraneoplastic syndrome with circulating antineuronal antibodies. Achalasia is diagnosed by barium swallow x-ray and/or esophageal manometry; endoscopy has a relatively minor role other than to exclude pseudoachalasia. The barium swallow x-ray appearance is of a dilated esophagus with poor emptying, an air-fluid level, and tapering at the LES giving it a beak-like appearance. Occasionally, an epiphrenic diverticulum is observed. In long-standing achalasia, the esophagus may assume a sigmoid configuration. The diagnostic criteria for achalasia with esophageal manometry are impaired LES relaxation and absent peristalsis. High-resolution manometry has somewhat advanced this diagnosis; three subtypes of achalasia are differentiated based on the pattern of pressurization in the nonperistaltic esophagus. Because manometry identifies early disease before esophageal dilatation and food retention, it is the most sensitive diagnostic test.

There is no known way of preventing or reversing achalasia. Therapy is directed at reducing LES pressure so that gravity and esophageal pressurization promote esophageal emptying. Peristalsis rarely, if ever, recovers. However, in many instances, remnants of peristalsis masked by esophageal pressurization and dilatation prior to therapy are demonstrable

following effective treatment. LES pressure can be reduced by pharmacologic therapy, pneumatic balloon dilatation, or surgical myotomy.

Nitrates or calcium channel blockers are administered before eating, advising caution because of their effects on blood pressure. Sildenafil and alternative phosphodiesterase inhibitors effectively decrease LES pressure, but practicalities limit their clinical use in achalasia. Antacids, vitamins and preparations of bismuth can be prescribed as needed.

The only durable therapies for achalasia are pneumatic dilatation and Heller myotomy.

In untreated or inadequately treated achalasia, esophageal dilatation predisposes to stasis esophagitis. Prolonged stasis esophagitis is the likely explanation for the association between achalasia and esophageal squamous cell cancer. Tumors develop after years of achalasia, usually in the setting of a greatly dilated esophagus with the overall squamous cell cancer risk increased 17-fold compared to controls.

MANAGEMENT OF THE PATIENTS WITH HEARTBURN

Heartburn, a burning substernal sensation, is reported intermittently by at least 40% of the population. Classically, heartburn is felt to result from excess gastroesophageal reflux of acid. However, some cases exhibit normal esophageal acid exposure and may result from reflux of nonacidic material or heightened sensitivity of esophageal mucosal nerves.

Gastroesophageal reflux disease (GERD) is prevalent in heartburn reasons. Most cases of heartburn occur because of excess acid reflux, but reflux of nonacidic fluid produces similar symptoms. Alkaline reflux esophagitis produces GERD-like symptoms most often in patients who have had surgery for peptic ulcer disease. Ten percent of patients with heartburn exhibit normal esophageal acid exposure and no increase in nonacidic reflux (functional heartburn).

Gastroesophageal reflux disease

Gastro-esophageal reflux disease (GERD) is a chronic and relapsing disease that results from the retrograde flow of gastric contents into the esophagus (gastro-esophageal reflux), oropharynx, and/or respiratory tract and causes troublesome symptoms or complications and/or mucosal injury. GERD refers to the abnormal, not physiologic reflux.

Classification. Montreal Classification of GERD (2006): erosive reflux disease (with esophagitis), nonerosive reflux disease (is defined by the presence of troublesome reflux-associated symptoms and absence of mucosal breaks on endoscopy), Barrett's esophagus (with long segment or short segment). NB: Erythema is not reliable finding for diagnosis of reflux esophagitis!

LA Classification of Esophagitis by grade (1999):

Grade A	One (or more) mucosal break no longer than 5 mm that does not extend between the tops of two mucosal folds
Grade B	One (or more) mucosal break more than 5 mm long that does not extend between the tops of two mucosal folds
Grade C	One (or more) mucosal break that is continuous between the tops of two or more mucosal folds but which involve less than 75% of the circumference
Grade D	One (or more) mucosal break which involves at least 75% of the esophageal circumference

By symptoms: typical and atypical.

Epidemiology. It is estimated that GERD, defined as at least weekly heartburn or acid regurgitation, has a prevalence ranging from 10 to 20% in the Western world and less than 5% in Asia. The prevalence also tends to be higher in North America than Europe and higher in northern Europe than in southern Europe. The prevalence of heartburn is the highest in USA and Western Europe countries (20%) and the lowest is in China (2,5%).

Risk and etiological factors: obesity, pregnancy, hiatal hernia, tobacco abuse, alcohol consumption, overeating, taking medicines that decrease lower esophageal sphincter contractility (nitrates, calcium channel blockers, beta adrenergic agonists, papaverine, no-spa, anticholinergics, theophylline, morphine, meperidine, diazepam, and barbiturates etc.). A genetic component may also play a role in GERD exacerbation. Character of food can have an influence too, e.g. peppermint, coffee, fatty meal etc.

Pathogenesis. The esophagus is protected from the harmful effects of refluxed gastric contents by the antireflux barrier at the gastroesophageal junction, by esophageal clearance mechanisms, and by epithelial defensive factors. The antireflux barrier consists of the lower esophageal sphincter, crural diaphragm, phrenoesophageal ligament, and the angle of His, which causes an oblique entrance of the esophagus into the stomach. The attachment of the lower esophageal sphincter to the crural diaphragm results in increased pressure during inspiration and when intra-abdominal pressure increases. Disruption of normal defense mechanisms leads to pathologic amounts of reflux. Reflux of gastric contents from the stomach into the esophagus occurs in healthy individuals, but refluxed gastric contents are normally cleared in a two-step process: volume clearance by peristaltic function and neutralization of small amounts of residual acid by weakly alkaline swallowed saliva. In normal healthy individuals, physiologic reflux occurs primarily when the lower esophageal sphincter transiently relaxes in the absence of a swallow because of a vagally mediated reflex that is stimulated by gastric distention. Physiologic reflux is postprandial, short, asymptomatic and does not cause mucosal injuries of esophagus.

In GERD patients, transient relaxation of the lower esophageal sphincter or a low resting lower esophageal sphincter pressure can result in regurgitation, especially when intra-abdominal pressure is increased. A hiatal hernia, which results in spatial separation between the augmenting effects of the crural diaphragm and the lower esophageal sphincter, predisposes to reflux events by widening the opening of the gastroesophageal junction and decreasing the pressure of the lower esophageal sphincter. The result is an increased exposure of the esophagus to acid, with increased reflux events during transient physiologic relaxation of the lower esophageal sphincter. Hernias also act as a reservoir for gastric contents when normal esophageal clearance mechanisms result in trapping of fluids in the hernia sac; these contents can reflux into the esophagus when the lower esophageal sphincter relaxes during subsequent swallowing.

Obesity results in an increase in intragastric pressure, which increases the gastroesophageal pressure gradient and the frequency of transient lower esophageal sphincter relaxation, thereby predisposing gastric contents to migrate into the esophagus. In addition, obesity enhances the spatial separation of the crural diaphragm and the lower esophageal sphincter, thereby predisposing obese individuals to a hiatal hernia. The normal defense mechanisms based on peristalsis and saliva can also be impaired. Peristaltic dysfunction is associated with an increasing severity of esophagitis, and ineffective peristaltic clearance may occur when the amplitude of esophageal contractions is less than 30 mm Hg. Saliva production may be impaired by a variety of mechanisms, such as smoking and Sjögren's syndrome.

The esophageal mucosa contains several lines of defense. A pre-epithelial barrier is composed of a small unstirred water layer combined with bicarbonate from swallowed saliva and

from the secretions of submucosal glands. A second epithelial defense is composed of cell membranes and tight intercellular junctions, cellular and intercellular buffers, and cell membrane ion transporters. The postepithelial line of defense is composed of the blood supply to the esophagus. Acid and acidified pepsin in the refluxate are the key factors that damage the intercellular junctions, increase intracellular permeability, and dilate intercellular spaces. If sufficient quantities of refluxate diffuse into the intercellular spaces, cellular damage may occur. Signs and symptoms of GERD occur when defective epithelium comes into contact with refluxed acid, pepsin, or other noxious gastric contents.

In addition to the direct noxious effects of refluxed acid, pepsin, and bile, refluxed gastric juice stimulates esophageal epithelial cells to secrete chemokines that attract inflammatory cells into the esophagus, thereby damaging the esophageal mucosa.

Clinical features: The classic symptoms of GERD are heartburn and acid regurgitation; atypical symptoms include chest pain, dysphagia, and odynophagia.

Extraesophageal manifestations of reflux disease can include cough, laryngitis, asthma, and dental erosions, but these symptoms can be reliably attributed to reflux only if they are accompanied by classic signs and symptoms of reflux disease. Other proposed associations that are not clearly established include pharyngitis, sinusitis, otitis media, and idiopathic pulmonary fibrosis. When excessive gastric contents overwhelm the mucosal protective factors in the esophagus, esophagitis may be manifest as erosions or ulceration of the esophagus and may also lead to fibrosis with stricturing, columnar metaplasia (Barrett's esophagus) or esophageal adenocarcinoma. However, approximately two thirds of individuals with reflux symptoms have no evidence of esophageal damage by endoscopy.

Typical symptoms:

- 1) Heartburn
 - Retrosternal burning sensation
 - Most commonly post-prandial, nocturnal
 - Fatty foods, spicy foods, acidic foods
 - Relieved with antacids, water, milk
 - Worsened with recumbency
- 2) Acid Regurgitation
 - Perception of gastric content reflux in the mouth or hypopharynx
 - Taste: bitter, acidic

Atypical symptoms:

- 1) Atypical
 - Dysphagia, odynophagia
 - Nausea
 - Chest pain
 - Dyspepsia (non-severe upper abdominal discomfort)
 - Epigastric fullness, bloating
 - Frequent belching
 - Heartburn
- 2) Extraesophageal
 - Chronic cough, bronchospasm, pneumonia, fibrosis
 - Cardiac pain, arrhythmia
 - Hoarseness, laryngitis, pharyngitis, globus sensation, vocal cord dysfunction

- Stomatitis, dental erosions

RED FLAGS:

- Dysphagia (immediately assess for Barrett's Esophagus)
- Odynophagia (Assess for Esophageal Ulcer)
- Nausea/vomiting
- Melena
- Weight loss, anorexia
- Extended duration of symptoms with no response to PPIs
- Family history of peptic ulcer disease
- Symptom onset in the age more than 45 years, especially in males (high risk)
- High body temperature
- CBC changes (anemia, leukocytosis, etc.)

Diagnosis:

- History (patients' complaints, anamnesis)
- Physical examination
- Screening tests (CBC)
- Empiric trial (IPP, Alginates)
- Additional: endoscopy with biopsy, chromoendoscopy, manometry, pH testing, impedance.
- If necessary: bronchography, ultrasound diagnostic, Helicobacter pillory testing, ECG.

When GERD presents with typical signs and symptoms, such as heartburn or acid regurgitation, that are responsive to antisecretory therapy, no diagnostic evaluation is warranted.

Diagnostic endoscopy is warranted in individuals who fail to respond to therapy or have alarm symptoms or signs such as dysphagia, weight loss, anemia, gastrointestinal bleeding, or persistent heartburn. Endoscopy permits the detection of erosive esophagitis and complications such as a peptic stricture and Barrett's esophagus; mucosal biopsy, which is crucial in these settings, also excludes conditions that can mimic GERD, such as eosinophilic esophagitis. However, most patients have no mucosal damage seen on endoscopy, regardless of whether they are on or off antisecretory therapy.

Esophageal manometry is useful to exclude achalasia in patients with suggestive symptoms. Esophageal reflux testing by 24-hour transnasal pH monitoring, by 48-hour devices attached to the esophageal lumen, or by 24-hour combined impedance and pH monitoring, may be performed while patients are not on therapy to detect pathologic acid and nonacid reflux as well to correlate reflux events with atypical symptoms, especially in patients with normal endoscopies.

Barium radiography has no role in the diagnostic evaluation of patients with reflux disease.

Complications: esophageal strictures, Barrett's esophagus, ulcer of esophagus, bleeding, laryngitis, pharyngitis, sinusitis, adenocarcinoma, interstitial fibrosis, dental erosions (dental enamel loss).

Alternative diagnosis in GERD (Differential Diagnosis):

- Coronary artery disease (Ischemic heart disease)
- Gallstones
- Gastric/esophageal cancer
- Peptic ulcer disease
- Esophageal motility disorders
- Pill induced esophagitis

- Eosinophilic esophagitis
- Fungal or viral esophagitis
- Peptic stricture
- Metaplastic disease (Barrett's)
- Dysplastic disease (adenocarcinoma)

Treatment. General Measures:

- Avoidance of foods or beverages that may provoke symptoms, such as alcohol, coffee, spicy foods, fatty food, chocolate etc. and late meals (less than 2-3 hours before bedtime)
- Elevation of the head of the bed to 30 degrees for patients with nocturnal regurgitation or heartburn
- Weight loss should be part of any treatment program for obese patients
- Tobacco cessation.

Medications. Inhibition of gastric acid secretion is the cornerstone of the acute treatment of GERD, and proton pump inhibitors (PPIs) are superior to histamine (H₂)-receptor antagonists for both the healing of esophagitis and the control of symptoms. Once-daily standard dosage of PPIs 30-60 minutes prior to meal for 4-8 weeks (non-erosive form, Grade A, B) or 8-12 weeks (Grade C, D) is adequate. High dose (twice daily) is usually used for severe or refractory symptoms. Given the chronicity of reflux symptoms, long-term maintenance therapy with PPIs is typically required, with dosing titrated to the lowest dose necessary to control symptoms. Although PPIs are superior to H₂-receptor antagonists for long-term maintenance therapy as well as for short-term relief, H₂-receptor antagonists are useful in patients who are intolerant of PPIs, and can be used at bedtime to supplement PPIs in patients who have persistent symptoms.

PPIs suppress the production of stomach acid and work by inhibiting the molecule in the stomach glands that is responsible for acid secretion (the gastric acid pump). Once symptoms are controlled, people should receive the lowest effective dose of PPIs.

PPIs (standard dosage):

- Esomeprazole 40mg
- Lansoprazole 30mg
- Omeprazole 20mg
- Pantoprazole 40mg
- Rabeprazole 20mg
- Dexlansoprazole (long-acting form) 60mg

H₂-blockers interfere with acid production by blocking or antagonizing the actions of histamine. Histamine encourages acid secretion in the stomach. Famotidine is the most potent H₂ blocker.

Also antacid medications (e.g. Maalox) can be used. Antacids neutralize acids in the stomach, and are the drugs of choice for mild GERD symptoms. They may also stimulate the defensive systems in the stomach by increasing bicarbonate and mucus secretion. They should be prescribed 1-1.5 hours after meal 3-4 times a day for 14 days. The different brands all rely on various combinations of 3 basic ingredients: magnesium, calcium, or aluminum.

Magnesium salts are available in the form of magnesium carbonate, magnesium trisilicate, and most commonly, magnesium hydroxide (Milk of Magnesia). The major side effect of magnesium salts is diarrhea. Magnesium salts offered in combination products with aluminum (Maalox) balance the side effects of diarrhea and constipation.

Calcium carbonate is a potent and rapid-acting antacid. It can cause constipation. There have been rare cases of elevated levels of calcium in the blood (hypercalcemia) in people taking large doses of calcium carbonate for long periods of time. This condition can lead to kidney failure and is very dangerous. None of the other antacids has this potential side effect.

Aluminum salts (Almagel) are also available. The most common side effect of antacids containing aluminum salts is constipation. People who take large amounts of antacids that contain aluminum may also be at risk for calcium loss, which can lead to osteoporosis.

Prokinetic drugs help the stomach empty its contents more quickly and strengthen the esophageal sphincter. These are considered second-line access drugs due to side effects. The most widespread prokinetics are: Domperidone 10mg 3 times a day, Itoprid 50mg 3 times a day.

Antireflux surgery is an option for patients who have documented esophagitis and who are intolerant of PPIs or unresponsive to them. However, surgery has a number of serious complications that may affect quality of life, including dysphagia, vagal nerve injury, gas bloat syndrome, and diarrhea. There are inadequate data to support any of the many proposed endoscopic approaches to GERD at present.

MANAGEMENT OF THE PATIENTS WITH DYSPEPSIA

Functional dyspepsia

Functional dyspepsia (FD) is a medical condition that is characterized by one or more of the following symptoms: epigastric pain, epigastric burning, postprandial fullness, and early satiation that are unexplained after a routine clinical evaluation. Abdominal bloating and nausea also may be experienced, but they are less specific and are not considered cardinal symptoms of functional dyspepsia. Patients, who were not observed can be made a preliminary diagnosis of *uninvestigated dyspepsia*. Further they will be divided into 2 groups: those with an organic, systemic, or metabolic cause for the symptoms that can be identified by traditional diagnostic procedures where, if the disease improves or is eliminated, symptoms also improve or resolve (eg, peptic ulcer disease, malignancy, pancreaticobiliary disease, endocrine disorders, or medication use) and is described by the term *secondary dyspepsia*; those in whom no identifiable explanation for the symptoms can be identified by traditional diagnostic procedures that are exemplified under the “umbrella” term *functional dyspepsia*.

Classification. In the Rome IV criteria (2016), symptoms have been divided into postprandial distress syndrome and epigastric pain syndrome. The clinical utility of these subgroups is controversial because there is considerable overlap between them.

Epidemiology: FD is a common disorder, with an estimated prevalence of 10%-30% worldwide. Approximately 1 of 2 individuals with functional dyspepsia seeks health care for symptoms at some time in his or her life.

Risk factors: according to recent researches smoking is only marginally associated with dyspepsia and alcohol and coffee are not. Other data are controversial.

Pathogenesis. The pathobiology of functional dyspepsia is complex and multifactorial and not fully understood.

Gastrointestinal motor and sensory dysfunction, as well as impaired mucosal integrity, low-grade immune activation, and dysregulation of the gut-brain axis have all been implicated. Both central and peripheral mechanisms have been proposed. Although enhanced perception of gastric stimuli may be a key central mechanism, the roles of gastric acid, acute and chronic

gastric mucosal infections, and gastroduodenal dysmotility remain to be determined. Rome IV proposed next factors, that play a role in FD pathogenesis:

- delayed gastric emptying
- impaired gastric accommodation
- gastric and duodenal hypersensitivity to distention, acid, and other intraluminal stimuli
- *Helicobacter pylori* infection
- psychosocial factors.

The potential relation between these factors and dyspeptic symptoms remains unclear.

Diagnosis. Clinical examination. Dyspepsia can be suspected to be functional based on a clinical history consistent and the absence of alarm features, treatment of overlapping gastroesophageal reflux disease and *H. pylori*. The presence of anxiety, in particular symptom-related anxiety and comorbid IBS, increases the likelihood of functional dyspepsia.

The physical examination is generally normal, although epigastric tenderness may be present. In contrast to gastroparesis, a succussion splash is typically absent. Confirmation of the functional dyspepsia diagnosis requires a normal upper endoscopic examination. Evaluation for *H. pylori* should be performed by stool antigen, urea breath test, or gastric biopsy. If present, the infection should be eradicated, and then symptoms should be reassessed. Additional methods of laboratory and instrumental examination, besides upper endoscopy and HP testing, are: CBC, biochemical blood analysis, fecal occult blood test, ultrasound diagnostic of abdominal cavity, computer tomography.

Rome IV (2016) diagnostic criteria for functional dyspepsia (criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis):

1. One or more of the following:

Bothersome postprandial fullness/early satiation/epigastric pain/epigastric burning

2. No evidence of structural disease

(including at upper endoscopy) that is likely to explain the symptoms.

Rome IV (2016) diagnostic criteria for subgroups of patients with functional dyspepsia:

– *Postprandial Distress Syndrome*

Must include one or both of the following at least 3 days per week:

1. Bothersome postprandial fullness (severe enough to impact on usual activities)
2. Bothersome early satiation (severe enough not to finish a regular-size meal)

AND No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy).

Supportive remarks: postprandial epigastric pain or burning, epigastric bloating, excessive belching, and nausea can also be present; vomiting warrants consideration of another disorder; heartburn is not a dyspeptic symptom but may often coexist; symptoms that are relieved by evacuation of feces or gas should generally not be considered as part of dyspepsia.

– *Epigastric Pain Syndrome*

Must include one or both of the following at least 1 day per week:

1. Bothersome epigastric pain (severe enough to impact on usual activities)

2. Bothersome epigastric burning (severe enough to impact on usual activities) AND No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy).

Supportive remarks: pain may be induced by ingestion of a meal, relieved by ingestion of a meal, or may occur while fasting; postprandial epigastric bloating, belching, and nausea can also be present; persistent vomiting likely suggests another disorder; heartburn is not a dyspeptic symptom but may often coexist; the pain does not fulfill biliary pain criteria; symptoms that are relieved by evacuation of feces or gas generally should not be considered as part of dyspepsia.

Nonsteroidal anti-inflammatory medications, dietary supplements, and other prescription or over-the-counter medications can trigger dyspeptic symptoms. The patient's medication list should be reviewed, and nonessential treatments should be discontinued. A psychosocial history may reveal underlying stressors that contribute to symptoms.

Differential Diagnosis. Common organic causes of dyspepsia include peptic ulcer disease and gastroesophageal reflux disease. Delayed gastric emptying is present in a small number of patients with functional dyspepsia but is characteristic and more pronounced in patients with diabetic or idiopathic gastroparesis. Vomiting of undigested food is characteristic of these forms of gastroparesis, but not of dyspepsia. Gastric and esophageal cancers may also present with symptoms of dyspepsia but are much less common. Pancreaticobiliary disorders (including sphincter of Oddi dysfunction, chronic pancreatitis, or pancreatic cancer) also occasionally mimic dyspepsia.

Treatment. Reassurance, education, lifestyle, and dietary recommendations (more frequent, smaller meals and avoiding meals with high fat content) are frequently recommended to FD patients. Avoidance of nonsteroidal anti-inflammatory drugs, coffee, alcohol, and smoking is commonly recommended.

Eradication of *H. pylori* is recommended in patients with chronic dyspepsia and positive HP tests (according to Maastricht V).

Postprandial distress syndrome should be treated with prokinetics (cisapride and domperidone). Itopride is a novel prokinetic agent that works by antagonizing dopamine D2-receptors and inhibiting acetylcholinesterase, and has been shown to improve postprandial fullness and early satiety with a low rate of adverse reactions. Acotiamide is a novel compound with fundusrelaxing and gastroprokinetic properties, based on a procholinergic effect that improves dyspeptic symptoms over placebo.

In relieving epigastric pain syndrome proton pump inhibitors (PPIs) and Histamine₂-blockers (H₂-blockers) are recommended to use (PPIs and H₂-blockers were described in GERD chapter).

Psychotropic drugs, especially antidepressants, are often used as second-line drugs in functional gastrointestinal disorders. Often used levosulpiride, which also bears prokinetic properties; and often recruited in psychiatric rather than gastroenterological settings.

Psychological therapies are advocated as rescue therapy for FD symptoms that are severe and not responding to pharmacotherapy. Available controlled trials suggested clinical benefit, but lacked convincing evidence because of small sample sizes and poorly matched treatment groups.

Materials for self-control:

Situation tasks:

1. A 65-year-old patient complains of heartburn, sour eructation, burning, compressing retrosternal pain and pain along the esophagus rising during forward bending of body. The patient hasn't been examined, takes Almagel on his own initiative, claims to feel better after its taking. What is the preliminary diagnosis? What additional tests are necessary?
2. A patient of 37 years old complains of pain in epigastrium, more often during the sleep, or after emotional overload, heartburn. These symptoms amplify after eating. Objectively: a belly is painless on palpation, the liver and spleen are not enlarged. What is the preliminary diagnosis? What additional tests are necessary for the patient? The treatment plan?

Tests:

1. A 38 y.o. man complains of having occasional problems with swallowing of both hard and fluid food for many months. Sometimes he feels intense pain behind his breast bone, especially after hot drinks. There are asphyxia onsets at night. He has not put off weight. Objectively: his general condition is satisfactory, skin is of usual colour. Examination revealed no changes of gastrointestinal tract. X-ray picture of thorax organs presents esophagus dilatation with level of fluid in it. What is the preliminary diagnosis?

- A. Myasthenia
- B. Esophagus achalasia
- C. Cancer of esophagus
- D. Esophagus candidosis
- E. Gastroesophageal reflux

2. A patient suffering from gastroesophageal reflux has taken from time to time a certain drug that "reduces acidity" over 5 years. This drug was recommended by a pharmacist. The following side effects are observed: osteoporosis, muscle asthenia, indisposition. What drug has such following effects?

- A. Inhibitor of proton pump
- B. Aluminium-bearing antacid
- C. H₂-blocker
- D. Metoclopramide
- E. Gastrozepin

3. A 28-year-old male patient complains of regurgitation, cough and heartburn that occurs every day after a meal, when bending forward or lying down. These problems have been observed for 4 years. Objective status and laboratory values are normal. Upper endoscopy revealed esophagitis. What is the leading factor in the development of this disease?

- A. Helicobacter pylori infection
- B. Hypersecretion of hydrochloric acid
- C. Duodeno-gastric reflux
- D. Hypergastrinemia
- E. Failure of the inferior esophageal sphincter

4. A 49-year-old male patient complains of retrosternal pain, heartburn, weight loss of 8kg over the last year, constipation, weakness. The patient has been a smoker for 20 years, and has a 10-year history of Gastroesophageal reflux disease. The patient is asthenic, has dry skin. EGD revealed an ulcer in the lower third of the esophagus and esophageal stricture accompanied by edema, hyperemia and multiple erosions of the mucosa. What study is required for more accurate diagnosis?

- A. Fecal occult blood test
- B. X-ray examination of the esophagus
- C. Respiratory test for *Helicobacter pylori*
- D. pH-monitoring of the esophagus and the stomach
- E. Biopsy of the esophageal mucosa

5. The 48 years old patient complains of periodic pain in epigastrium, without irradiation, heartburn, which amplify after meals, migraine and sleeplessness. After reception of 20 mg of rabeprazole during first two days these symptoms disappeared. For what disease this clinical picture is typical?

- A. Functional dyspepsia
- B. Duodenal ulcer
- C. Type A chronic gastritis
- D. Chronic pancreatitis
- E. Chronic hepatitis

6. A 31-year-old male patient complains of periodic heartburning. Objectively: HR-70/min, AP- 125/75 mm Hg. Upper endoscopy confirms esophageal ulcer. Which of the given drugs will be a compulsory element of the treatment?

- A. Omeprazole
- B. Famotidine
- C. Pirenzepine
- D. Atropine
- E. Maalox

7. A patient complains of heartburn which gets stronger after overeating. Upper endoscopy shows ulcerative esophagitis. What group of medicines is the first line of therapy?

- A. Inhibitors of proton pump
- B. Prokinetics
- C. Probiotics
- D. Antibiotics
- E. Alginates

Correct answers for the situation tasks:

1. GERD. IPP test, upper endoscopy, pH-monitoring.
2. Functional dyspepsia, epigastric pain syndrome. US examination, upper endoscopy. Treatment e.g. Lansoprazole 30 mg 40 minutes before the breakfast, Famotidine 40 mg 30 minutes before the supper.

The answers for the tests:

1-B, 2-B, 3-E, 4-E, 5-A, 6-A, 7-A.

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