Ministry of Health of Ukraine

Kharkiv National Medical University

Inquiry of the patients with the diseases of digestive organs. Examination and superficial palpation of the abdomen.

Methodical instructions for students

Рекомендовано
Ученым советомХНМУ
Протокол №__от_____2017 г.

Kharkiv
KhNMU
2017

Inquiry of the patients with the diseases of digestive organs. Examination and superficial palpation of the abdomen: Method. instr. for students / Authors. T.V. Ashcheulova, O.M. Kovalyova, G.V.Kozhemiaka – Kharkiv: KhNMU, 2017. – 42 p.

Authors: T.V. Ashcheulova
O.M. Kovalyova,
G.V. Kozhemiaka

1. INQUIRY

Complaints of the patients with the diseases of digestive organs are various, and depend on which part of the alimentary tract is affected (Tab.1.1). The most common complaints are:

- Dysphagia
- Heartburn (pyrosis)
- Regurgitation, cructio, ructus
- Nausea
- Vomiting
- Abdominal pain
- Diarrhea
- Constipation
- Gain and loss in weight
- Gastrointestinal bleeding

Tab. 1. 1. Overview of approach to patients with common gastrointestinal disorders.

Site of disorder	Common symptoms	Possible physical signs
Esophagus	Dysphagia Odynophagia Heartburn, chest pain Hematemesis/melena	
Stomach	Nausea and vomiting Epigastric pain Hematemesis/melena Early satiety	Distension Tenderness Succusion splash Mass
Pancreas	Pain Weight loss Diarrhea Steatorrhea	Mass Jaundice
Duodenum	Pain Nausea/ vomiting Hematemesis	Tenderness Altered bowel sounds Distension Mass
Jejunum	Pain Diarrhea	Altered bowel sounds Distension Mass

Ileum	Pain Diarrhea	Altered bowel sounds Distension Mass
Colon	Diarrhea Pain Blood	Tenderness Mass Distension
Rectum	Pain urgency Hematochezia Pruritus	Tenderness

Dysphagia is defined as a sensation of "sticking" or obstruction of the passage of food through the mouth, pharynx, or esophagus. It should be distinguished from other symptoms related to swallowing.

Aphagia signifies complete esophageal obstruction, which is usually due to bolus impaction and represents a medical emergency.

Difficulty in initiating a swallow occurs in disorders of the voluntary phase of swallowing. However, once initiated, swallowing is completed normally.

Odynophagia means painful swallowing. Frequently, odynophagia and dysphagia occur together.

Globus pharyngeous is the sensation of a lump lodged in the throat. However, no difficulty is encountered when swallowing is performed.

Misdirection of food, resulting in nasal regurgitation and laryngeal and pulmonary aspiration of food during swallowing, is characteristic of oropharyngeal dysphagia.

Phagophobia, meaning fear of swallowing, and *refusal to swallow* may occur in hysteria, rabies, tetanus, and pharyngeal paralysis due to fear of aspiration. Painful inflammatory lesions that cause odynophagia also may cause refusal to swallow.

Some patients may feel the food as it goes down the esophagus. This esophageal sensitivity is not associated with the food sticking or obstruction, however. Similarly, the *feeling of fullness in the epigastrium* that occurs after a meal or after swallowing air should not be confused with dysphagia,

The normal transport of an ingested bolus through the swallowing passage depends on the size of the ingested bolus; the luminal diameter of the swallowing passage; the peristalsis contraction; and deglutitive inhibition, including normal relaxation of upper and lower esophageal

sphincters during swallowing. Dysphagia caused by a large bolus or luminal narrowing is called *organic (or mechanical) dysphagia,* whereas dysphagia due to incoordination or weakness of peristaltic contractions or to impaired deglutitive inhibition is called *functional (or motor) dysphagia* (Tab. 1.2.1).

Tab. 1.2.1. Types of dysphagia

Dysphagia

Functional	Organic
- muscular spasm, caused by reflex disorders in innervation of the esophageal muscles, or by neurosis;	- cancer; - cicatricial stenosis of the esophagus; - presence of the foreign body; - compression from outside by an aortic aneurism, goiter, mediastinal tumor, pleuritis exudativa;
- occurs in paroxysms, difficulty in swallowing of the liquid food is typical, while the solid food passes easily.	- develops gradually and progresses up to the complete delay of not only solid, but also liquid food.

Tab. 1.2.2. Dysphagia

Process and Problem	Timing	Factors that Aggravate	Factors that Relieve	Associated Symptoms and Conditions
Transfer Dysphagia, *due to motor disorders affecting the pharyngeal muscles*	Acute or gradual onset and a variable course, depending on the underlying disorder	Attempts to start the swallowing process		Aspiration into the lungs or regurgitation into the nose with attempts to swallow. Neurologic evidence of stroke, bulbar palsy, or

				other neuromuscular condition
Esophageal Dysphagia **Mechanical Narrowing**				
• *Mucosal rings and webs*	Intermittent	Solid foods	Regurgitation of the bolus of food	Usually none
• *Esophageal stricture*	Intermittent, may become slowly progressive	Solid foods	Regurgitation of the bolus of food	A long history of heartburn and regurgitation
• *Esophageal cancer*	May be intermittent at first; progressive over month	Solid foods, with progression to liquids	Regurgitation of the bolus of food	Pain in the chest and back and weight loss, especially late in the course of illness
Motor disorders				
• Diffuse esophageal spasm	Intermittent	Solids or liquids	Maneuvers described below; sometimes nitroglycerine	Chest pain that mimics angina pectoris or myocardial infarction and lasts minutes to hours; possibly heartburn
• Scleroderma	Intermittent, may progress slowly	Solids or liquids	Repeated swallowing, movements such as	Heartburn. Other manifestatio

• Achalasia	Intermittent, may progress	Solids or liquids	straightening the back, raising the arms, or a Valsalva maneuver (straining down against a closed glottis)	n of scleroderma Regurgitatio n. Often at night when lying down, with nocturnal cough; possibly chest pain precipitated by eating

Heartburn (pyrosis) is a specific burning sensation behind the sternum associated with regurgitation of gastric contents into the inferior portion of the esophagus. Occasional heartburn is common in normal persons, but frequent and severe heartburn is generally a manifestation of esophageal dysfunction. Heartburn may result from abnormal motor activity or distension of the esophagus, sensitivity of the esophageal mucosa to refluxed acid or bile, or esophageal mucosal inflammation (esophagitis). Heartburn is most often associated with gastroesophageal reflux. In this setting, heartburn typically occurs after a large meal, with stooping or bending, or when the patient is supine. It may be accompanied by the spontaneous appearance in the mouth of fluid, which may be salty ("water brash"), sour (gastric contents), or bitter and green or yellow (bile). Heartburn may arise following the ingestion of certain foods (e.g., citrus fruit juices) or drugs (alcohol and aspirin). Heartburn arises also in various diseases of the alimentary tract with hyperacidity – gastritis, peptic ulcer disease, cholecystitis, hiatus hernia, and in pregnancy.

Regurgitation (crutio, ructus) is return of the part of swallowed food into the mouth due to backward movement of esophagus and stomach with open cardia without contraction of diaphragm and abdominal muscles (Tab1.3).

Tab. 1.3. Regurgitation

	By air *(eructation)*	By food or by gas *(regurgitatio)*	
Cause	Due to air swallowing (aerophagy) during the fast meals and is heard at a distance	Motor dysfunction of the stomach with increased formation of gas due to abnormal Fermentation or Putrefaction	
Odour	Odouless	Odouless or smell of sour, or bitter oil, which is due to the presence of organic acids	Odour of rotten eggs (hydrogen sulphide), that indicates intensive degradation of proteins
Disease	Psychoneurosis Fast meals	Usually associated with hypersecretion of gastric juice and occurs during pain attacks in ulcer. Can occur in normal or insufficient secretion of the stomach in failure of the cardia, when the stomach contents are regurgitated into the esophagus	Pylorus stenosis with great distension of the stomach and significant congestion in it

Nausea is reflex act associated with irritation of the vagus nerve. Nausea denotes the feeling of an imminent desire to vomit, usually referred to the throat or epigastrium. The mechanism of nausea is unknown. Nausea often precedes or accompanies vomiting. Often accompanying severe nausea is evidence of altered autonomic (especially parasympathetic) activity, such as skin pallor, increased perspiration, hypersalivation, defecation, and, occasionally, hypotension

and bradycardia (vasovagal syndrome); anorexia is also usually present. It is usually associated with diminished functional activity of the stomach (hypotonicity, hypoperistalsis, and hyposecretion) and altered small-intestinal motility (hypertonicity and reversed peristalsis of the duodenum).

The most common causes of nausea are listed in Table 1.4.

Tab. 1.4. Causes of nausea.

Nausea *can be due to*

Gastric diseases	**Other causes**
- Acute gastritis - Chronic gastritis - Cancer of the stomach -	- Pregnancy - Renal failure - Deranged cerebral circulation
Arises after meal	There is no relation with meal

Vomiting (emesis) refers to the forceful oral expulsion of gastric contents. Nausea, retching, and hypersalivation frequently precede the act of vomiting, which is highly integrated sequence of involuntary visceral and somatic motor events. The stomach plays a

relatively passive pole in the vomiting process, the major ejection force being provided by the abdominal musculature. With relaxation of the gastric fundus and gastroesophageal sphincter, a sharp increase in intraabdominal pressure is brought about by forceful contraction of the diaphragm and abdominal wall muscles. This, together with concomitant annular contraction of the gastric pylorus, results in the expulsion of gastric contents into the esophagus. Increased intrathoracic pressure results in the further movement of esophageal contents into the mouth. Reversal of the normal direction of esophageal peristalsis may play a role in this process. Reflex elevation of the soft palate during the vomiting at prevents the entry of the expelled material into the nasopharynx, whereas reflex closure of the glottis and inhibition of respiration help to prevent pulmonary aspiration.

Vomiting mechanism. The act of vomiting is under the control of two functionally distinct medullary centers: **the vomiting center** in the dorsal portion of the lateral reticular formation and the **chemoreceptor**

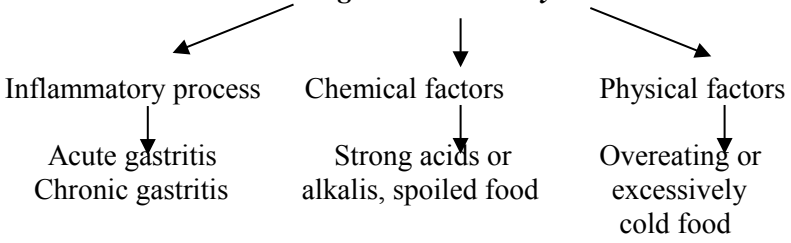
trigger zone in the area postrema of the floor of the fourth ventricle. The vomiting center controls and integrates the actual act of emesis. It receives afferent stimuli from the gastrointestinal tract and others part of the body, from higher brainstem and cortical centers, especially the labyrinthine apparatus, and from the chemoreceptor trigger zone. The important efferent pathways in vomiting are the phrenic nerves (to the diaphragm), the spinal nerves (to the intercostals and abdominal musculature), and visceral efferent fibers in the vagus nerve (to the larynx, pharynx, esophagus, and stomach). The vomiting center is located near other medullary centers regulating respiratory, vasomotor, and autonomic functions that may be involved in the act of vomiting.

Vomiting is common manifestations of many organic and functional disorders (Tab. 1.5).

Tab.1.5. Vomiting etiology

Central	Peripheral (of visceral etiology, reflex)	Hematogenic and toxic
Nervous • Encephalitis • Meningitis • Cerebral tumor • Stroke Sudden blood pressure elevation	•Peptic ulcer disease •Gastric tumor •Diseases of bile ducts •Pancreatitis •Myocardial infarction •Renal colic	• Liver failure • Renal failure • Diabetes mellitus

Vomiting of gastric etiology is caused by stimulation of receptors in the gastric mucosa by:



Vomiting can also be caused by difficult evacuation of the stomach due to spasms or stenosed pylorus.

If the patient complains of vomiting you should ask about the time when the vomiting arises: before or after meals, possible

connection with pain, the amount and character of the vomiting material. Vomiting may attend acute gastritis, exacerbation of chronic gastritis, peptic ulcer disease, spasm and organic stenosis of pulorus, and cancer of stomach.

Morning vomiting on a fasting stomach is characteristic

of chronic gastritis, especially in alcoholics.
The vomited material contain much mucus and traces of blood

of pyloric stenosi
The vomited material contain food taken a day or two before, has odour of rotten eggs, the amount is from 3 to 4 liters

Vomiting occurs after meals

10-15 minutes

Ulcer or cancer of cardiac part of the stomach
Acute gastritis

2-3 hours

Ulcer or cancer of the stomach body

4-6 hours

Ulcer of the pulorus duodenum

The **odour** of the vomit is usually acid, but it can be fetid due to putrefactive processes in the stomach, or even faecal in presence of a faecal fistula between the stomach and the transverse colon.

The **reaction** of vomited material may be various:

Acid		*Alkaline*
due to the presence of hydrochloric acid in hyperchlorhydia	***Neutral*** in achilia	due to the presence of ammonia compounds in pyloric stenosis, renal hypofunction, in regurgitation of the duodenal contents into the stomach

Abdominal pain is important and the leading symptom in diseases of digestive system.

You should ask the patient about:

1. *location* of the pain (epigastric region, right or left
2. hypochondrium, umbilical region, etc)
3. *character* of the pain (periodical or paroxysmal (at certain time of the day); permanent or seasonal (in spring or autumn); intensity (dull, stabbing, etc)
4. *connection with meals* (fasting pain, after meal: **early** – occurring 30-40 min after meals;
   late – 90-120 min after meals;
   nocturnal, hunger pain, which is abated after taking food.)
5. *radiation* of the pain
6. *relieving factors* (vomiting, taking food or soda, spasmolytics, warmly)
7. possible *connection* between *pain* and *physical strain* (weight lifting, traffic jolting)

Tab. 1.6.1. Abdominal pain

Problem	Process	Location	Quality
1	2	3	4
Peptic Ulcer and Dyspepsia *(These disorders*	Peptic ulcer refers to a demonstrable ulcer, usually in the duodenum or stomach. Dyspepsia	Epigastric, may radiate to the back	Variable: gnawing, burning, boring, aching,

cannot be reliably differentiated by symptoms and signs.)	causes similar symptoms but no ulceration.		pressing, or hungerlike
Cancer of the Stomach	A malignant neoplasm	Epigastric	Variable

Tab. 1.6.2. Abdominal pain

Timing	Factors That May Aggravate	Factors That May Relieve	Associated Symptoms and Setting
5	6	7	8
Intermittent. Duodenal ulcer is more likely than gastric ulcer or dyspepsia to cause pain that (1) wakes the patient at night, and (2) occurs intermittently over a few weeks, then disappears for month, and then recurs	Variable	Food and antacids may bring relief, but not necessarily in any of these disorders and least commonly in gastric ulcer	Nausea, vomiting, belching, bloating; heartburn (more common in duodenal ulcer); weight loss (more common in gastric ulcer). Dyspepsia is more common in the young (20-29 yr), gastric ulcer in the older (over 50 yr), and duodenal ulcer in those from 30-60 yr.
The history of pain is typically shorter than in peptic ulcer. The pain is persistent and slowly progressive.	Often food	Not relived by food or antacids	Anorexia, nausea, easy satiety, weight loss, and sometimes bleeding. Most common in ages 50-70

Tab. 1.6.3. Abdominal pain

1	2	3	4
Acute Pancreatitis	An acute inflammation of the pancreas	Epigastric, may radiate to the back or other parts of the abdomen; may be poorly localized	Usually steady
Chronic Pancreatitis	Fibrosis of the pancreas secondary to recurrent inflammation	Epigastric, radiating through to the back	Steady, deep
Cancer of the Pancreas	A malignant neoplasm	Epigastric and in either upper quadrant; often radiate to the back	Steady, deep
Biliary Colic	Sudden obstruction of the cystic duct or common bile duct by a gallstone	Epigastric or right upper quadrant; may radiate to the right scapula and shoulder	Steady, aching: not colicky

Tab. 1.6.4. Abdominal pain

5	6	7	8
Acute onset, persistent pain	Lying supine	Leaning forward with trunk flexed	Nausea, vomiting, abdominal distension, fever. Often history of previous attacks and of alcohol abuse or gallstones
Chronic or recurrent course	Alcohol, heavy or fatty meals	Possibly leaning forward with	Symptoms of decreased pancreatic function may appear:

		trunk flexed; often intractable	diarrhea with fatty stools (steatorrhea) and diabetes mellitus
Persistent pain; relentlessly progressive illness		Possibly leaning forward with trunk flexed; often intractable	Anorexia, nausea, vomiting, weight loss, and jaundice. Emotional symptoms, including depression
Rapid onset over a few minutes, lasts one to several hours and subsides gradually. Often recurrent			Anorexia, nausea, vomiting, restlessness

Tab. 1.6.5. Abdominal pain

1	2	3	4
Acute Cholecystitis	Inflammation of the gallbladder, usually from obstruction of the cystic duct by a gallstone	Right upper quadrant or upper abdominal; may radiate to the right scapular area	Steady, aching
Acute Diverticulitis	Acute inflammation of a colonic diverticulum, a saclike mucosal outpouching through the colonic muscle	Left lower quadrant	May be cramping at first, but becomes steady
Acute Appendicitis	Acute inflammation of the appendix with distension or obstruction	1. Poorly localized *periumbilical pain*, followed usually by	1. Mild but increasing, possibly cramping 2. Steady

		2. *Right lower quadrant pain*	and more severe

Tab. 1.6.6. Abdominal pain

5	6	7	8
Gradual onset; course longer than in biliary colic	Jarring, deep breathing		Anorexia, nausea, vomiting, and fever
Often a gradual onset			Fever, constipation. There may be initial brief diarrhea
1. Lasts roughly 4-6 ht 2. Depends on intervention	1. 2. Movement or cough.	1. 2. If it subsides temporarily, suspect perforation of the appendix	Anorexia, nausea, possibly vomiting, which typically follow the onset of pain; low fever

Tab. 1.6.7. Abdominal pain

1	2	3	4
Acute Mechanical Intestinal Obstruction	Obstruction of the bowel lumen, most commonly caused by (1) adhesions or hernials (small bowels), or (2) cancer or diverticulitis (colon)	1. *Small bowel*: periumbilical or upper abdominal 2. *Colon*: lower abdominal or generalized	1. Cramping 2. Cramping
Acute Arterial Occlusion	Blocked blood supply to the bowel and	May be periumbilical at first, then	Cramping at first, then steady

	mesentery from thrombosis or embolus	diffuse	

Tab. 1.6.8. Abdominal pain

5	6	7	8
1. Paroxysmal; may decrease as bowel mobility is impaired 2. Paroxysmal, though typically milder			1. Vomiting of bile and mucus (high bstruction) or fecal material (low obstruction). Obstipation develops. 2. obstipation early. Vomiting late if at all. Prior symptoms ofunderlying cause.
Usually abrupt in onset, then persistent			Vomiting, diarrhea (sometimes bloody), constipation, shock

Diarrhea is formally defined as an increase in daily stool weight above 200 g. Typically, the patients also may describe an abnormal increase in stool liquidity and frequency. Diarrhea is considered *acute* when lasting less than 7 to 14 days and *chronic* when lasting more than 2 to 3 weeks.

Acute diarrhea. The most common causes of acute diarrhea are infectious agents.

Acute diarrhea also may be caused by ingested drugs or toxins, the administration of hemotherapy, resumption of enteral feeding following a prolonged fast, fecal impaction (overflow diarrhea), or particular situations, such as marathon running.

Virtually any medication can cause diarrhea, and a careful drug history should be obtained in any patient with acute diarrhea. Other ingested toxins also must be considered, including organophosphate insecticides, mushrooms, arsenic, and even caffeine. Acute diverticulitis occasionally may present with diarrhea accompanied by fever and abdominal pain. In patients with acute bloody diarrhea, diagnostic considerations may include superior mesenteric arterial or venous thrombosis, ischemic or drug-induced colitis, or idiopathic inflammatory bowel disease (ulcerative colitis or Crohn's disease). In the elderly patient with acute colitis, differentiating an ischemic cause from enterohemorrhagic *E. coli* O157:H7 may difficult because both diseases may be associated with submucosal hemorrhage that presents as "thumbprinting" on a plain abdominal radiograph.

Chronic diarrhea. Diarrhea that persists for weeks or month, whether constant or intermittent, requires evaluation. Although in the majority of cases the cause will prove to be irritable bowel syndrome, diarrhea may represent a manifestation of an underlying serious illness, and a careful search for disease should be taken.

Chronic diarrhea can be categorized pathophysiologically as inflammatory, osmotic (malabsorption), secretory, due to intestinal dysmotility, or factitious (Tab.1.7.1).

Tab. 1.7.1. Classification of chronic diarrhea

Mechanism	Clinical features	Examples
INFLAMMATORY		
Mucosal and submucosal inflammation Damaged epithelium In some cases impaired intestinal absorption and excessive secretion	Fever, abdominal pain, blood and/or leukocytes in stool	Ulcerative colitis Crohn's disease Radiation enterocolitis Eosinophilic gastroenteritis Infections associated with AIDS
OSMOTIC		
Nonabsorbed or nondigested intraluminal solute	Improvement of diarrhea with fasting Bulky, greasy, foul-	Pancreatic insufficiency Bacterial overgrowth Celiac sprue Lactase deficiency

	smelling stools; weight loss Nutrient deficiencies Osmotic gap in fecal water	Whipple's disease Abetalipoproteinemia Short bowel syndrome
SECRETORY		
Excessive secretion of electrolytes	Watery diarrhea, persists with fasting Dehydration Other systemic effects of hormones Absence of osmotic gap in fecal water	Carcinoid syndrome Zollinger-Ellison syndrome Vasoactive intestinal peptide-secretory pancreatic adenomas Medullary carcinoma of thyroid Villous adenoma of rectum Microscopic colitis Cholerrheic diarrhea
ALTERED INTESTINAL MOTILITY		
Rapid transit In some cases associated bacterial overgrowth	Alternating diarrhea and constipation Neurologic symptoms; bladder involvement	Irritable bowel syndrome Fecal impaction Neurologic diseases
FACTITIOUS		
Self-induced	Usually women Watery diarrhea with hypokalemia, Weakness, edema	Laxative abuse

Inflammatory diarrhea are characterized by the presence of fever, abdominal tenderness, and blood or leukocytes in the stool, with inflammatory lesions on intestinal mucosal biopsy. In some cases, hypoalbuminemia, hypoglobulinemia, and protein-losing enteropathy may be present. In addition to inflammation, the mechanism of diarrhea may also be malabsorption or increased intestinal secretion.

Osmotic diarrhea occurs when an orally ingested solute is not fully absorbed in the small intestine and thereby exerts an osmotic force that draws fluid into the intestinal lumen. The increased luminal fluid volume overwhelms the capacity of the colon for reabsorption. The nonabsorbed solute can be a maldigested or malabsorbed nutrient or drug. Clinical symptoms are usually recognized because of the malanbsorption of fat (steatorrhea) or carbohydrates. Protein or amino acid malabsorption (azotorrhea) is generally nor recognized clinically unless it is severe enough to cause malnutrition or the consequences of a specific deficiency in an amino acid.

Secretory diarrhea is characterized by a large volume of fecal output caused by abnormal fluid and electrolyte transport not necessarily related to the ingestion of food. Therefore, diarrhea usually persists with fasting. The term watery diarrhea is often used synonymously with secretory diarrhea. Because there is no malabsorbed solute, fecal osmolality in secretory diarrheas can be accounted for by normal ionic constituents with no fecal osmotic gap.

Altered intestinal motility. Diarrhea may be associated with disorders that affect intestinal motility. The most common of these is irritable bowel syndrome, in which diarrhea typically alternates with constipation and is associated with abdominal pain, the passage of mucus, and a sense of incomplete evacuation. Diarrhea may occasionally occur paradoxically as a result of fecal impaction or an obstructing tumor with the overflow of liquid colonic contents around the impacted stool or obstruction. A variety of neurologic diseases also may be associated with diarrhea because of altered autonomic control of bowel function. Profuse watery diarrhea, often with incontinence, may be seen in the patients with type 1 diabetes and is often associated with severe neuropathy, nephropathy, and retinopathy. Additional contributing factors may include bacterial overgrowth secondary to intestinal dysmotility, pancreas exocrine insufficiency, or rarely, celiac sprue.

Factitious diarrhea is self-induced by the patient and may result from intestinal infection, the addition of water or urine to the stool, or self-medication with laxatives. Patients are predominantly women with severe chronic watery diarrhea, abdominal pain, nausea and vomiting, weight loss, peripheral edema, and weakness resulting from hypokalemia. The diagnosis of factitious diarrhea should be

suspected in a patient with a history of psychiatric disease or multiple previous negative evaluations for diarrhea.

Constipation is a common complaint in clinical practice because of wide range of normal bowel habits, constipation is difficult to define precisely. Most persons have at least three bowel movements per week, and constipation has been defined as a frequency of defecation of less than three times per week. However, stool frequency alone is not a sufficient criterion to use, because many constipated patients describe a normal frequency of defecation but subjective complaints of excessive straining, hard stools, lower abdominal fullness, and a sense of incomplete evacuation. Thus a combination of objective and subjective criteria must be used to define constipation.

Pathophysiologically, constipation generally results from disordered colonic transit or anorectal function as a result of a primary motility disturbance, certain drugs, or in association with a large number of systemic diseases that affect the gastrointestinal tract.

In the patient presenting with the recent onset of constipation, an obstructing lesion of the colon should be sought. In addition to a colonic neoplasm, other causes of colonic obstruction include strictures due to colonic ischemia, diverticular disease, or inflammatory bowel disease; foreign bodies', or anal strictures. Anal sphincter spasm due to painful hemorrhoids or fissures also may inhibit the desire to evacuate.

In the absence of an obstructing lesion of the colon, disturbed colonic motility may mimic colonic obstruction. Disruption of parasympathetic innervation to the colon as a result of injury or lesions of the lumbosacral spine or sacral nerves may produce constipation with hypomotility, colonic dilatation, decreased rectal tone and sensation, and impaired defecation. In patients with multiple sclerosis, constipation may be associated with neurogenic dysfunction of other organs. Similarly, constipation may be associated with lesions of the central nervous system caused by parkinsonism or a cerebrovascular accident.

Drugs that may lead to constipation include those with anticholinergic properties, such as antidepressants and antipsychotics, codeine and other narcotic analgesics, aluminum- or calcium-containing antacids, sucralfate, iron supplements, and calcium channel blockers. In patients with certain endocrinopathies such as hypothyroidism and

diabetes mellitus, constipation is generally mild and responsive to therapy. Rarely, life-threatening megacolon occurs in patients with myxedema. Constipation is common during pregnancy, presumably as a result of altered progesterone and estrogen levels, which decrease intestinal transit. Collagen vascular diseases may be associated with constipation, which may be a particularly prominent feature of progressive systemic sclerosis, in which delayed intestinal transit results from atrophy and fibrosis of colonic smooth muscle.

Gain and loss in weight

In normal persons weight is stable over long periods because food intake is matched to energy expenditure by neural activity in the hypothalamus that provides signals to eat or to stop eating. Because the system is usually effective, either weight gain or weight loss may bring a patient to the physician. No history is complete without ascertaining whether weight has been gained or lost and, if so, how much. In general, a change of 5 percent of body weight or 5 kg is considered significant. However, a 5-kg weight loss may not be of importance in a 130-kfl man who is trying to lose weight, while a 2-kg weight loss may well be worrisome in a person weighing 40 kg,

Weight gain is less likely to have a pathotogic cause than weight loss. In most cases it is a consequence of overeating and inadequate exercise, and the diagnosis is usually simple obesity. Occasionally obesity may be the consequence of hypothyroidism, Cushing's syndrome, or hypothalamic disease such as craniopharyngioma. Workup for pathologic causes of weight gain is rarely indicated unless accompanying signs suggest an underlying cause. Sometimes weight gain is not fat but fluid, in which case the primary problems to be considered are congestive heart failure, renal failure, or cirrhosis with ascrtes.

Although rarely caused by disease, obesity predisposes to disease, especially diabetes mellitus but also to gall stones, degenerative joint disease, hyperlipidemia. atherosclerosis, hypertension, sleep apnea, and perhaps cancer.

Weight loss. In all studies there are patients who lose weight without discernible cause, but significant involuntary weight loss is usually a marker of serious disease. Even if no disease is found on initial evaluation, it should not be assumed that weight loss is idiopathic. The

patient should be followed at regular intervals with careful repeat examination since occult illness causing weight loss may not become manifest for long periods.

The only common causes of increased metabolism are hyperthyroidism, pheochromocytoma, and major exercise programs. Loss of ingested energy generally is due to either diabetes mellitus with glycosuria or intestinal malabsorption with steatorrhea. Chronic pancreatitis in alcoholics is the most common cause of steatorrhea, but malabsorption can occur with intestinal lymphoma, celiac sprue, islet cell tumors (such as somatostatinomas or gastrinomas), radiation injury, biliary tract obstruction, inflammatory bowel disease, and a variety of other disorders.

Under most circumstances the diagnosis of the cause of weight loss is not difficult and is revealed by history, physical examination, and routine laboratory screening.

Gastrointestinal bleeding.

Hematomesis is defined as the vomiting of blood, and ***melena*** as the passage of stools rendered black and tarry by the presence of blood. These clinical manifestations of gastrointestinal hemorrhage suggest a proximal source of bleeding. The color of vomited blood depends on the concentration of hydrochloric add in the stomach and the duration of its contact with the blood. Thus, if vomiting occurs shortly after the onset of bleeding, the vomitus appears red, and later the appearance win be dark red, brown, or black. Precipitated blood clots and acid-degraded blood in the vomitus will produce a characteristic "coffee grounds" appearance when vomited. Hematemesis usually indicates bleeding proximal to the ligament of Treitz. because blood entering the fit tract below the duodenum rarely enters the stomach.

Approximately 60 mL of blood is required to produce a single black stool; acute blood loss greater than this may produce melena for as long as 7 days. After the stool color returns to normal, tests for occult blood may remain positive for over a week. The black color of melena results from contact of the blood with hydrochloric acid to produce hematin. Such stools are tarry ("sticky") and have a characteristic odor. This tarry consistency is in contrast to black or dark gray stools occurring after the ingestion of iron, bismuth, or licorice. Gastrointestinal bleeding, even if detected only by positive tests for

occult blood, indicates potentially serious disease and must be further investigated.

Hematochezia, the passage of red blood per rectum, generally signifies bleeding from a source distal to the ligament of Treitz. However, brisk proximal bleeding can cause hematochezia due to rapid transit.

The clinical manifestations of gastrointestinal bleeding depend on the extent and rate of hemorrhage and the presence of coincidental diseases. Blood loss of less than 500 mL is rarely associated with systemic signs; exceptions include bleeding in the elderly or in the anemic patient in whom smaller amounts of blood loss may produce hemodynamic alterations. Rapid hemorrhage of greater volume results in decreased venous return to the heart, decreased cardiac output, and increased peripheral resistance due to reflex vasoconstriction. Orthostatic hypotension greater than a change of 10 mmHg usually indicates a 20 percent or greater reduction in blood volume. Concomitant symptoms may include lightheadedness, syncope, nausea, sweating, and thirst. When blood loss is 25 to 40 percent of blood volume, shock frequently ensues with pronounced tachycardia and hypotension. Pallor is prominent, and the skin is cool. However, in the presence of beta-adrenergic and calcium channel blockers, these clinical signs may be blunted.

In the setting of rapid hemorrhage, the initial hematocrit may not accurately reflect the magnitude of blood loss, since equilibration with extravascular fluid and hemodilution often require over 8 h. Common laboratory findings include mild leukocytosis and thrombocytosis which develop within 6 h after the onset of bleeding. The blood urea nitrogen (BUN) may be elevated out of proportion to the creatinine, particularly in upper gastrointestinal bleeding, due to breakdown of blood proteins to urea by intestinal bacteria as well as mild reduction in the glomerular filtration rate.

Occult bleeding, detected by card test for hemoglobin peroxidase, is an important means of finding colorectal neoplasia at earlier, potentially curable stages. Testing is advocated for patients over age 50 as a part of the yearly checkup. Multiple stools should be tested (usually two samples from three stools), and if any sample is positive, additional studies should be performed. A positive result can be due to physiologic blood loss, dietary peroxidases, undercooked meat, or any

cause of upper or lower gastrointestinal bleeding. The daily ingestion of over 500 mg of vitamin C may result in a false-negative test. To limit the confounding variables, patients should be tested on a high-fiber and low-meat diet with no ingestion of nonsteroidal anti-inflammatory agents (NSAIDs) or vitamin C, although the daily low dose of aspirin (80 to 325 mg) taken to prevent cardiovascular disease generally does not lead to false-positive results.

Upper gastrointestinal bleeding. A careful history and physical examination of the oropharynx and nasal cavity should serve to exclude epistaxis or swallowed blood as a source of hematemesis or melena.
The most common causes of upper gastrointestinal hemorrhage are:

- Erosive or hemorrhagic gastropathy
- Duodenal ulcer
- Gastric ulcer
- Mallory-Weiss tear
- Varices or portal hypertensive gastropathy
- Arteriovenous malformations

Lower gastrointestinal bleeding

Tab. 1.8.1. Common causes of acute lower gastrointestinal bleeding (in order of frequency)

Under age 55	Over age 55
Anorectal disease (hemorrhoids, fissures) Colitis (inflammatory bowel disease) Diverticulosis Polyps, cancer (hyperplastic, hamartomas) Angiodysplasia	Anorectal disease (hemorrhoids, fissures) Diverticulosis Angiodysplasia Polyps, cancer Enterocolitic (ischemic, infectious, inflammatory bowel disease, radiation)

Tab. 1.8.2. BLACK and BLOODY STOOLS

Problem	Selected Causes	Associated Symptoms

		and Setting
Melena Melena refers to the passage of black, tarry (sticky and shiny) stools. Tests for occult blood are positive. Melena signifies the loss of a least 60 ml of blood into the gastrointestinal tract (less in infants and children), usually from the esophagus, stomach, or duodenum. Less commonly, when intestinal transit is slow, the blood may originate in the jejunum, ileum, or ascending colon. In infants, melena may result from swallowing blood during the birth process.	Peptic ulcer Gastritis or stress ulcers Esophageal or gastric varices Reflux esophagitis Mallory-Weiss tear, a muscular tear in the esophagus due to retching and vomiting	Often, but not necessary, a history of epigastric pain. Recent ingestion of alcohol, aspirin, or other anti-inflammatory drugs; recent bodily trauma, severe burns, surgery, or increased intracranial pressure. Cirrhosis of the liver or other cause of portal hypertension. History of heartburn. Retching, vomiting, often recent ingestion of alcohol.

Black, Nonsticky Stools Black stools may result from other causes and then usually give negative results when tested for occult blood. (Ingestion of iron or other substances, however, may cause a positive test result in the absence of blood.) These stools have no pathologic significance.	Ingestion of iron, bismuth salts as in Pepto-Bismol, licorice, or even commercial chocolate cookies	

Red Blood in the Stools Red blood usually originates in the colon, rectum, or anus, and much less frequently in the jejunum or ileum. Upper gastrointestinal hemorrhage, however, may also cause red stools. The amount of blood lost is then usually large (more than a liter). Transit time through the intestinal tract is accordingly rapid, giving insufficient time for the blood to turn black.	Cancer of the colon Benign polyps Diverticula of the colon Inflammatory conditions of the colon and rectum Ulcerative colitis Infectious dysenteries Proctitis in men or women who have had frequent anal intercourse Ischemic colitis Hemorrhoids Anal fissure	Often a change in bowel habits Often no other symptoms Often no other symptoms Diarrhea Diarrhea Rectal urgency, tenesmus Lower abdominal pain and sometimes fever or shock in persons over age 50 years Blood on the toilet paper or on the surface of the stool, or dripping into the toilet Blood on the toilet paper or on the surface of the stool; anal pain
Reddish but Nonbloody Stools	The ingestion of beets	Pink urine, which usually precedes the reddish stool

2. INSPECTION OF THE ABDOMEN

Starting from your usual standing position at the right side of the bed, inspect the abdomen. When looking at the contour of the abdomen and watching for peristalsis, it is helpful to sit or bend down so that you can view the abdomen tangentially.

Note (see Tab.2.1.1):

Tab. 2.1.1. Inspection of the abdomen

Shape of the abdomen	- round - oval - spherical - pendulous - flatten ("frog belly") - fell
Size of the abdomen	- doesn't enlarged - enlarged
Symmetry of the abdomen	- symmetrical - asymmetrical
Participation of the anterior abdominal wall in the breathing act	- takes part - doesn't take part - lags (to indicate location)
Umbilicus position	- somewhat retracted - smoothed - protruded
Expression of the subcutaneous veins	- invisible - pronounced - in a form of Medusa head
Scars	- absent - present (to indicate location, size, color, primary or secondary healing etc)
Eruption	- absent - present (to describe elements)
Scratches	- absent - present
Visible pulsation	- absent - present (to indicate location)

Visible peristalsis	- absent - present (to indicate)
Telangioectasia	- absent - present

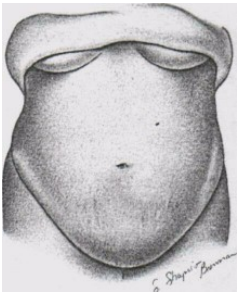
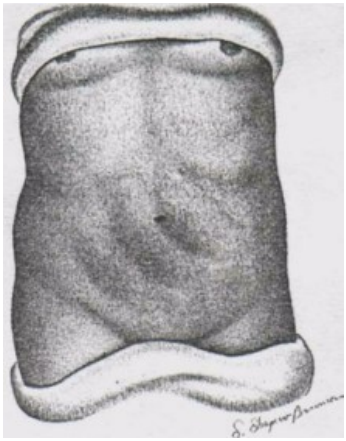
In **norm** the abdomen is of a oval shape, doesn't enlarged in size, symmetrical, anterior abdominal wall take part in the breathing act, umbilicus is retracted, pronounced venous network, scars, eruption, telangioectasia, scratches, visible pulsation and peristalsis are absent.

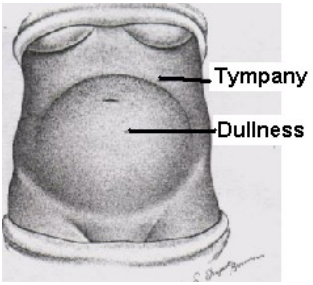
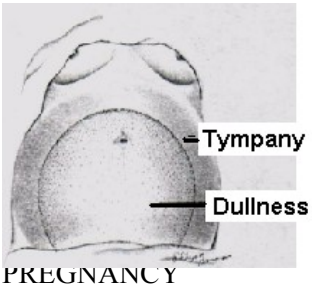
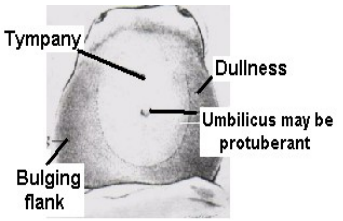
The most common causes of protuberant abdomen are pregnancy obesity, meteorism, and ascitis (Tab.2.1.2, Tab. 2.1.3).

Tab. 2.1.2. Differential signs of meteorism and ascitis

Sign	Meteorism	Ascitis
Skin	Doesn't change	"Rice-paper" (thin, shining)
Umbilicus	Retracted	Smoothed, or protruded
Fluctuation symptom	Absent	Present
Percussion	Tympanic sound	Dull sound in sloping sites

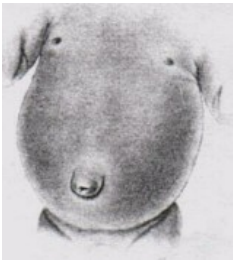
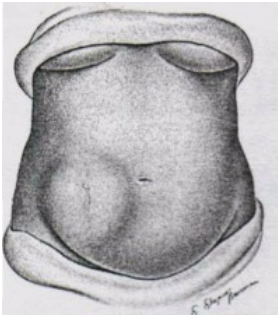
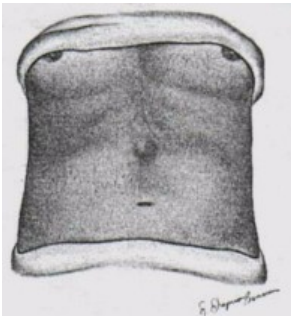
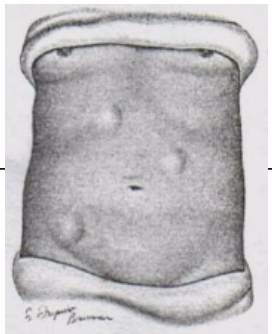
Tab. 2.1.3. Protuberant abdomens

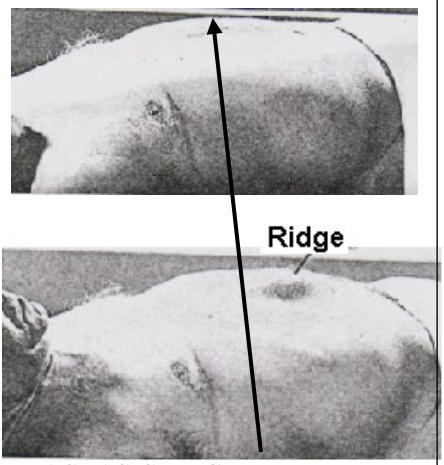
 FAT	Fat is the most common cause of a protuberant abdomen and is associated with generalized obesity. The abdominal wall is thick. Fat in the mesentery and omentum also contributes to abdominal size. The umbilicus may appear sunken. The percussion note is normal. An apron of fatty tissue may extend below the inguinal ligaments. Lift it to look for inflammation in the skin fold or even for a hidden hernia.
GAZ 	Gaseous distension may be localized, as shown, or generalized. It causes a tympanitic percussion note. Increased intestinal gas production due to certain foods may cause mild distension. More serious are intestinal obstruction and adynamic (paralytic) ileus. Note the location of the distension. Distension becomes more marked in colonic than in small bowel obstruction.

 TUMOR	A large, solid tumor, usually rising out of the pelvis, is dull to percussion. Air-filled bowel is displaced to the periphery. Causes include ovarian tumors and uterine myomata. Occasionally, a markedly distended bladder may be mistaken for such a tumor.
 PREGNANCY	Pregnancy is a common cause of a pelvic “tumor”. Listen for the fetal heart.
 **ASCITIC FLUID**	Ascitic fluid seeks the lowest point in the abdomen, producing bulging flanks that are dull to percussion. The umbilicus may protrude. Turn the patient onto one side to detect the shift in position of the fluid level (shifting dullness).

Localized bulges in the abdominal wall include ventral hernias (defects in the wall through which tissue protrudes) and subcutaneous tumors such as lipomas. The more common ventral hernias are umbilical, incisional, and epigastric. Rectus diastasis is also sometimes so classified. Hernias and a rectus diastasis usually become re evident when the patient raises head and shoulders from a supine position (Tab.2.2.4).

Tab.2.2.4. Localized bulges in the abdominal cavity

 UMBILICAL HERNIA	Umbilical hernias protrude through a defective umbilical ring. They are most common in infants but also occur in adults. In infants, but not in adults, they are usually close spontaneously within a year or two.
 INCISIONAL HERNIA	An incisional hernia protrudes through an operative scar. By palpation, note the length and width of the defect in the abdominal wall. A small defect, through which a large hernia has passed, has a greater risk of complications than a large defect.
 EPIGASTRIC HERNIA	An epigastric hernia is a small midline protrusion through a defect in the linea alba, somewhere between the xiphoid process and umbilicus. With the patient's head and shoulders raised (or with the patient standing), look for it, and run your finger pad down the linea alba to feel it.
	

LIPOMA	Lipomas are common, benign, fatty tumors usually located in the subcutaneous tissues almost anywhere in the body, including the abdominal wall. Small or large, they are usually soft and often lobulated. When your finger presses down on the edge of a lipoma, the tumor typically slips out from under it.
 DIASTASIS RECTI	A rectus diastasis is a separation of the two rectus abdominis muscles, through which abdominal contents buldge to form a midline ridge when the patient raises head and shoulders. Repeated pregnancies, obesity, and chronic lung disease may predispose to it. It has no clinical consequences.

3. PALPATION OF THE ABDOMEN

Palpation of the abdomen is the main method of physical examination of the abdominal organs along with X-ray examination.

This method was first proposed be French physician Glenard. Later the Russian physicians Obraztsov and Strazhesko developed this method.

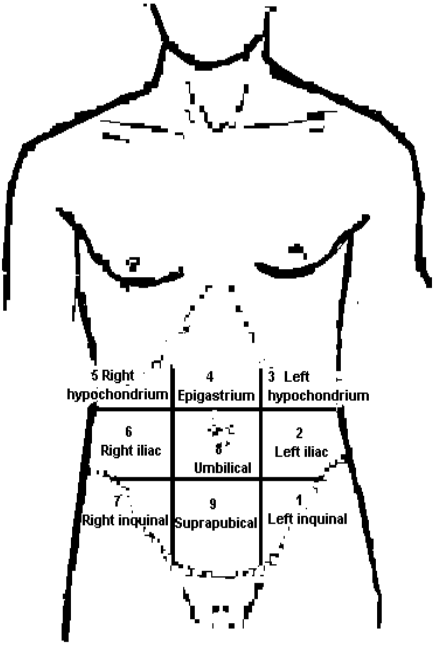
Technique. For a good abdominal examination you need god light, a relaxed patient, and full exposure of the abdomen from above the xiphoid process to the symhysis pubis. The groins should be visible, although the genitalia should be kept draped. The patient should not have a full bladder. Make the patient comfortable in a supine position, the bed should not be too soft, with a small firm pillow for the head. The patient should relax in his bed, his legs should be stretched. The patient should keep arms at the sides or folded across the chest. This position ensures relaxation of the abdominal muscles. Although patients commonly put their arms over their heads, this move should be discourages because it stretches and tightens the abdominal wall and makes palpation difficult. The ambient temperature should be comfortable for the patient, and the hands of the doctor should be warm and dry. Rubbing your hands together or running hot water over them may help to warm them. Before palpation, ask the patient to point to any areas of pain, and examine painful or tender areas last. Monitor your examination by watching the patient's face for signs of discomfort.

Surface, penetrative, and deep palpation are distinguished.

Superficial tentative oriental palpation of the abdomen

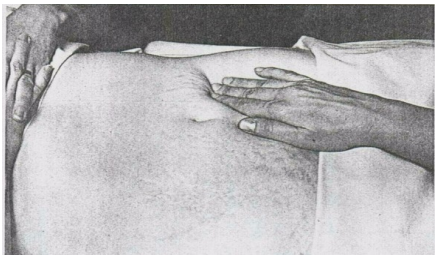
You should examine abdomen by the tips of the fingers, which should be slightly flexed, very tentative and careful to prevent muscular defense. Approach slowly and avoid quick, unexpected movements. Surface palpation conducts in topographic regions of the abdomen (Fig. 1).

Fig. 1. Topographic regions of the abdomen



Keeping your hand and forearm on a horizontal plane, with fingers together and flat on the abdominal surface, palpate the abdomen with a light, gentle, dipping motion. When moving your hand from place to place, raise it just off the skin. Moving smoothly, feel in all quadrants (Fig.2).

Fig.2. Superficial palpation of the abdomen



Superficial light palpation is helpful in identifying muscular resistance, abdominal tenderness, diastesis recti, and fluctuation symptom (Tab.2.3.1).

Tab. 3.1.1. **Superficial palpation of the abdomen**	
Resistance of the anterior abdominal wall	- soft - muscular defense - rigid
Tenderness of the abdomen	- painless - painful (to indicate location)
Diastasis recti	- absent - present
Fluctuation symptom	- negative - positive

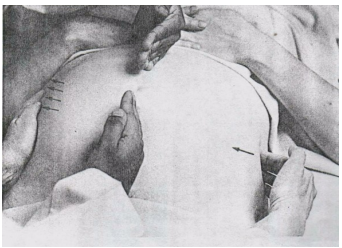
Identify increased resistance to your hand or any area of tenderness. If *resistance of the anterior abdominal wall* is present, try to distinguish voluntary guarding from involuntary muscular spasm. To do this try all relaxing method, feel relaxation of abdominal muscles that normally accompanies exhalation, ask the patient to mouth-breath with jaw dropped open. Voluntary guarding usually decreases with these maneuvers. Involuntary rigidity (muscular spasm) typically persists despite these maneuvers. It indicates peritoneal inflammation.

If *abdominal tenderness* is present, you should indicate topographic region of the painful areas.

If *diastasis recti* is present you will feel separation of the two rectus abdominis muscles when you move your hand along midline.

Fluctuation symptom. While you tap one flank sharply with your fingertips, feel on the opposite flank for an impulse transmitted through the fluid. You can also ask the patient or an assistant to press the edges of both hands firmly down the midline of the abdomen. This pressure helps to stop the transmission of a wave through fat (Fig.3). An easily palpable impulse suggests ascites.

Fig.3. Fluctuation symptom



Norm. In superficial palpation the abdomen is soft, painless, diastasis recti is absent, fluctuation symptom is negative.

Penetrative palpation of the abdomen

Penetrative palpation includes revelation of tenderness points.

Penetrative palpation of the abdomen:

Gall bladder point (Ker point) - painless / painful

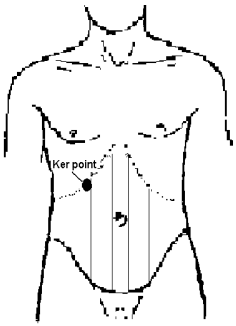
Appendix point (McBurne point) - painless / painful

Site of the duodenum bulb projection painless / painful

Shchetkin-Blumberg symptom - negative /positive (to indicate location

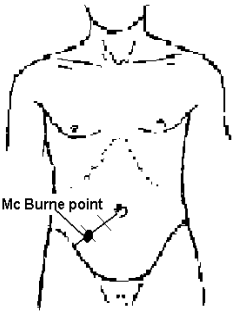
Ker point - projection point of the gall bladder - places in a point of intersection of the right costal arch with the lateral edge of the right rectus abdominal muscle

Fig. 4. Projection point of the gall bladder (Ker point)



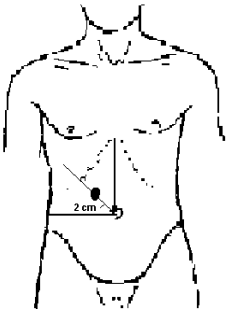
McBurne point – projection point of the vermiform process – places at the border of median and outer third of the right umbilico-iliacae line

Fig. 5. Appendix projection point (Mc Burne point)



Duodenum bulb projection point places 2 cm from the umbilicus on the bisector of right angle formed by anterior median line and line drawing through the umbilicus

Fig.6. Duodenum bulb projection point



Shchetkin-Blumberg symptom – indicates irritation of the peritoneum. Local pressure causes strong pain, which becomes more severe when the pressure is released.

Abdominal pain and tenderness, especially when associated with muscular spasm, suggest inflammation of the parietal peritoneum. Localize it as accurately as possible. First, even before palpation, ask the patient to cough and determine where the cough produced pain. Thus guided, palpate gently with one finger to map the tender area. Pain produced by light percussion has similar localizing value. These gentle maneuvers may be all you need to establish an area of peritoneal inflammation. If not, look for rebound tenderness. Press your fingers in firmly and slowly, and them quickly withdraw them. Watch and listen to the patient for sign of pain. Ask the patient to compare which hurt more, the pressing or letting go, and to show you exactly where it hurt. Pain induced or increased by quick withdrawal constitutes rebound tenderness. It results from the rapid movement of inflamed peritoneum.

Norm. In penetrative palpation projection points of the gall bladder, duodenum bulb, and appendix are painless. Shchetkin-Blumberg symptom is negativ

Test control.

1. The patient, 38 years has arrived with complaints to difficulty of swallowing of firm food, vomiting, decrease in body weight. In the anamnesis - a poisoning with a alkali. Inspection: pallor skin, an exhaustion. At superficial palpation the abdomen is soft and painless.

What organ defeat it is possible to think of?
A. Stomach
B. Pancreas
C. Oesophagus
D. Intestines
E. Liver
2. The patient, 33 years complaints to a heartburn, a pain in epigastrium that arises right after food, tarry [currant jelly] stool during 2 days, fainting fit, weakness. In the anamnesis - a stomach ulcer. Inspection - pallor skin. What complication is it possible to think of?
A. Perforation
B. Penetration
C. Malignization
D. A bleeding
E. Pilorostenosis
3. Patient P. 60 years is disturbed heavy sense epigastral site, with disgust for meat food, vomiting by the food eaten on the eve, decrease in body weight. In the anamnesis - a stomach. Inspection: - pallor skin, the expressed growing thin, above left clavicle a dense lymph nod is palpate. Detonation of abdomen wall in epigastral site is determined. At palpation in epigastral site it is more than stomach to the left of a median line, palpable formation in the size 3x4 cm. Your previous diagnosis?
A Pilorostenosis
B. Bleeding.
C. Stomach cancer
D. Atrophic gastritis
E. Ulcer
4. The patient, 42 years, complains of dyspnea, increase of abdomen. In the anamnesis - abusing alcohol. Abdomen inspection – is increased, umbilicus is protruding by formation of a hernia, - behind the umbilicus « the head of a jellyfish ». Your diagnostic assumptions?
A. Flatting
B. Obesity
S. Tumor
D. Ascitis
E. Cyst

5. The patient, 48 years, complains of weight in right hypochondrium, increase abdomen. During 10 years suffers on chronic persistent hepatitis. At abdomen inspection in vertical position -is loose-hanging, umbilicus is protruding a little. In horizontal position detonation of lateral departments abdomen is marked. Your diagnostic assumptions?
A. Flatting
B. Obesity
S. Tumor
D. Ascitis
E. Cyst
6. The patient, 70 years has arrived in clinic with complaints on sharp knife-like pain in the top of the abdomen that has appear after rise heavy. In the anamnesis - a stomach ulcer during 4 years. Inspection. Position of the patient is forced - lays with the pressed to a breast legs, features are aggravated, pale skin, covered sticky then. Superficial palpation: the poured pressur of abdomen wall muscles, sharp painess in epigastral part is marked. What pathology is it possible to think of?
A. Stomach ulcer exacerbation
B. Ulcer perforation
C.Acute cholecystitis
D. Peritonitis
E. Bleeding
7. The patient, 35 years complains of a pain in epigastrium, that appears in 30 minutes after food, a heartburn, decrease of appetite, tarry [currant jelly] stool. The anamnesis. 4 year of stomach ulcer. The beginning of disease connects with stress, an aggravation during the autumn-spring period. Inspection tongue is covered by white patch near root. Superficial palpation: moderate plainness in epigastral part. Your diagnostic assumptions?
A. Ulcer penetration
B. Ulcer perforation
C. Ulcer malignization
D. Peritonitis
E. Bleeding
8. The patient, 70 years, has arrived in clinic with complaints to a constant pain and sensation of spreading in paraumbilical site that amplify after reception even a small amount of food. The simplification comes after vomiting. In the anamnesis - stomach ulcer. Last

aggravation about three months ago. Inspection – skin is dry, the patient of lowered feed, visible peristaltics of stomach in the form of deep waves which go from left hypochondrium to right is determined. Your diagnosis?
A. Flatting
B. Perforation
C. Pilirostenosis
D. Ascitis
E. Tumor of stomach
9. The patient, 19 years, complains on colicky [cramping] pain that arises after fat food and attend by heartburn, an eructation sour. Objectively: tongue is densely imposed white patch. At palpation – moderate painness in epigastrium. Your diagnosis?
A. Atrophy gastritis
B. Stomach ulcer
C. Calculous cholecystitis
D. Chronic gastritis
E. Pilorostenosis
10. Patient, 35 years, complains of a pain in epigastrium that arises shortly after food, faintness. An eructation, stool instability. Diseases developed gradually, first attributes has been appears about three years ago. Inspection: patient is satisfactory fatness, tongue is imposed white patch, crude with reflections teeth on edges. Moderate palpatory tenderness is defined at epigastric region. Your previous diagnosis?
A. Acute gastritis
B. Chronic cholecystitis
C. Stomach ulcer
D. Chronic pancreatitis
E. Chronic gastritis

Keys: 1C, 2D, 3C, 4D, 5D, 6B, 7E, 8C, 9D, 10E.

Methodical instructions

Inquiry of the patients with the diseases of digestive organs. Examination and superficial palpation of the abdomen.

Methodical instructions for students

Authors: T.V. Ashcheulova
O. M. Kovalyova
G.V. Kozhemiaka

Chief Editor Ashcheulova T.V.

Редактор_____
Корректор_____
Компьютерная верстка_____

Редакционно-издательский отдел
Пр. Науки, г. Харьков, 4, ХНМУ, 61022