PEPTIC ULCER DISEASE

I LEVEL

1. Which consequences are typical for the hyperchlorhydria and increased secretory function of gastric glands:

1. predisposition to the constipation
2. increase in pepsin activity
3. relaxation of the pylorus
4. increase in stomach motility
5. decrease in the gut motility

2. Constipation (difficult evacuation of feces) seems to be characteristic of:

1. B-1 avitaminosis
2. poor nutrition
3. low activity of stomach juice
4. low content of fibres in uptaken food
5. low content of Ca^{++} and K^{+} in uptaken food
6. hyperactivity of stomach juice

3. Predisposition to the atonic constipation can be revealed at:

1. vitamin B_1 deficiency
2. poor nutrition
3. decreased acidity of gastric juice
4. cellulose deficiency in the food
5. deficiency of K^{+} - and Ca^{2+} salts in the food

4. Which of the following are characteristics of increased secretion of gastric juice:

1. rapid gastric emptying
2. increased amount of gastric juice with pH<2,0 on the empty stomach
3. absence of the pepsin activity
4. long spasm of the pylorus
5. heartburn, acidy belching

5. Which of the following are characteristics of decreased secretion of gastric juice:

1. rapid gastric emptying
2. increased amount of gastric juice with pH<2,0 on the empty stomach
3. absence of the pepsin activity
4. long spasm of the pylorus
5. heartburn, acidy belching

6. Which of the following take part in the pathogenesis of a heartburn:

1. gaping cardiac sphincter (relaxed lower esophageal sphincter)
2. gastro-esophageal reflux
3. spasm and antiperistalsis of esophagus
4. decreased acidity of gastric juice
5. increased amount of organic acids in stomach

7. Which of the following take part in the pathogenesis of a belching:

1. fermentation and rotting in the stomach
2. increased stomach volume or pressure
3. cardiospasm
4. spasm of pyloric sphincter
5. reflex contraction of stomach and diaphragm muscles

8. Which of the following are consequences of bad food mastication:

1. decrease in reflex gastric juice secretion
2. increase in reflex pancreatic juice secretion
3. increase in pancreatic juice secretion
4. mechanical injury of esophageal, gastric mucous
5. delayed digestion in stomach

9. High activity of gut peristalsis seems to be a result of:

1. achilia
2. acholia
3. low vagus tone
4. increased exitability of gut wall receptors
5. enteritis
6. low content of fibres in uptaken food

10. Meteorism may lead to:

1. high reflectory diuresis
2. reflectory diuresis inhibition
3. changeable arterial pressure
4. drop in venous pressure
5. dyspnea

11. Steatorrhea and creatorrhea after uptake of food rich of fat and meet may be an evidence of:

1. pancreatic achilia **(**absence of pancreatic juice secretion)
2. stomach juice hypersecretion
3. stomach achilia
4. acholia

12. The following substances take part in gut autointoxication:

1. hydrogen sulphide
2. skatol
3. histamine
4. serotonin
5. putrescin
6. cadaverin

13. In the pathogenesis of the dumping syndrome the essential role are playing:

1. polyuria
2. rapid gastric emptying
3. secretion of epinephrine, serotonin, formation of bradykinin
4. hypoglycemia, then hyperglycemia
5. intestinal distension and increase in permeability of mesenterical vessels
6. decrease in blood volume

14. Disturbances in primary digestion are preferential in:

1. obstructive jaundice
2. chronic pancreatitis
3. lactase deficiency
4. duodenitis
5. gluten enteropathy

15. Disturbances in secondary digestion are preferential in:

1. obstructive jaundice
2. lactase deficiency
3. gluten enteropathy
4. chronic pancreatitis
5. duodenitis

16. Which of the following are consequences of acholia:

1. absence of the bile in the duodenum
2. decreased pH in the duodenum
3. disturbance of fat emulcification
4. disturbance of fatty acids absorption
5. steatorrhea

17. Point to the consequences of acholia:

1. lack of bile in duodenum
2. low pH in duodenum
3. low lipase activity
4. meteorism
5. anemia

18. Which of the following are consequences of acholia:

1. absence of the bile in the duodenum
2. absence of lipase
3. decrease in lipase activity
4. rapid fat splitting
5. increase in fermentation and rotting

19. Point to the consequences of acholia:

1. fat stool
2. A,D,E,K avitaminosis
3. disturbances in fat emulsion formation
4. low secretion of stomach juice
5. steatorrhea
6. creatorrhea

20. Point to the consequences of pancreatic achilia:

1. lack of bile in duodenum
2. low pH in duodenum
3. lack of lipase
4. disturbances in fat splitting
5. steatorrhea
6. increased fermentation and purification in the gut

21. Which of the following are consequences of pancreatic achilia:

1. absence of bile in duodenum
2. absence of lipase
3. disturbance of fat emulcification
4. steatorrhea
5. creatorrhea

22. Clinical features of gut digestion disturbances are revealed in pancreas destruction of:

1. 25%-30% 2. 50% 3. 70 %4. 95%

23.Membrane digestion can be violate at:

1. liver and pancreas diseases with disturbance of gut digestion
2. resection of 25% of intestine
3. disturbance of structure and ultrastructure of jejunum
4. disturbance of enzymes on intestinal surface
5. disturbance of motility and excretory function of intestine

24. Preferential possible causes of impaired secondary digestion are:

1. of 25% jejunum resection
2. lack of enzymes in gut epithelium
3. disbacteriosis
4. gut inflammation
5. acute pancreatitis

25. Disturbances in carbohydrate balance may reveal themselves in form:

1. vomiting
2. meteorism
3. burning
4. colic
5. pain
6. diarrhea

26. Secondary digestion can be violate at:

1. severe liver and pancreas diseases
2. of 50% intestine resection
3. disturbance of motility and excretory function
4. disturbance of enzymes on intestinal surface

27. Malabsorption syndrome can be revealed with:

1. meteorism
2. constipation
3. diarrhea
4. loss of weigth
5. hypoproteinemia

28. Malabsorption syndrome includes the following signs:

1. meteorism
2. diarrhea
3. steatorrhea
4. decreased blood ammonia ions
5. hyperproteinemia

29. Malabsorption syndrome includes:

1. constipation
2. loss of weight
3. vit. A,D,E,K deficiency
4. hypercalcemia
5 meteorism.

30. Malabsorption syndrome can be revealed with:

1. meteorism
2. constipation
3. decreased in NH^3 in the blood
4. steatorrhea
5. vit A,D,E,K deficiency

31. Absorption of nutrients in small intestine becomes impaired after resection of:

1. 25% of gut 2. 50% of gut 3. 75% of gut

32. Absorption of nutrients can be violate at:

1. inflammation caused by infection
2. inflammation caused by endotoxins (during uremia)
3. resection of 25% of the intestine
4. atrophic processes in intestine mucous

33. Which of the following are consequences of "high" resection of intestine:

1. Fe deficiency in organism
2. vit B_{12} deficiency in organism
3. folic acid deficiency in organism
4. deficit of liposoluble vitamins A,D,E,K in the organism
5. steatorrhea

34. Which of the following are consequences of "high" resection of intestine:

1. folic acid deficiency in organism
2. vit B_{12} deficiency in organism
3. decrease in absorption of bile acids
4. malabsorption
5. diarrhea

35. The consequences of "high" resection of small intestine are:

1. iron deficiency
2. vit.B-12 deficiency
3. steatorrhea
4. diarrhea
5. folate deficiency

36. The consequences of low resection of small intestine are:

1. iron deficiency
2. folate deficiency
3. steatorrhea
4. diarrhea
5. vit. A,D,E,K deficiency
6. decreased bile salts absorption

37. Which of the following are consequences of "low" resection of intestine:

1. Fe deficiency in organism
2. vit B_{12} deficiency in organism
3. folic acid deficiency in organism
4. deficit of liposoluble vitamins A,D,E,K in the organism
5. steatorrhea

38. Which of the following are consequences of "low" resection of intestine:

1. deficit of folic acid in the organism
2. deficit of vit B_{12} in the organism
3. decrease in absorption of bile acids
4. malabsorption
5. diarrhea

39. High Ca++ level in enterocytes may lead to:

1. increased absorption of Na+,Cl-and water
2. decreased absorption of Na+,Cl- and water
3. increased secretion of Na+, Cl- and water
4. decreased secretion of Na+,Cl- and water

40. Low Ca++ concentration in enterocytes may result in:

1. increased Na+,Cl- and water absorption
2. decreased Na+,Cl- and water absorption
3. decreased secretion of Na+,Cl- and water
4. increased secretion of Na+,Cl- and water

41. Disbacteriosis is accompanied by the following bacterial shifts in small intestine:

1. increased total content of microbes
2. eschericia coli, clebciellas, lactobacillus, and enterococci prevalure
3. loss of bifidbacteria
4. escherichia, staphilococci,streptococci, clebciellas, proteus, and yeast prevalure
5. increased content of bifidobacteria
6. decreased total content of bacteria

42. Disbacteriosis is accompanied by the following bacterial shifts in large intestine:

1. increased total content of microbes
2. escherichia coli, clebciellas,lactobacillus, and enterococci prevalure
3. loss of bifidobacteria
4. escherichia,staphilococci,clebciellas, streptococci, proteus, and yeast prevalure
5. increased content of bifidobacteria
6. decreased total content of bacteria

43. The basic mechanisms of disturbances in digestion during disbacteriosis are:

1. significant pH shift in the gut
2. accumulation of toxic substances in the gut lumen
3. destruction of digestive enzymes
4. decreased gut wall permeability
5. accelerated reparation of gut epithelium
6. bacterial competition for life-important substance

44. Which of the following approvals are true?

1. histamine increases HCl secretion by acting on H_1-receptors
2. histamine increases HCl secretion by acting on H_2-receptors
3. irritation of vagus inhibits secretion of HCl in the stomach
4. irritation of vagus stimulates secretion of HCl in the stomach

45. Which of the following approvals are true?

1. Glucocorticoids stimulate gastric juice secretion
2. Glucocorticoids stimulate mucus formation in the stomach
3. Glucocorticoids improve regeneration of gastric mucous layer
4. Glucocorticoids disturb motor-evacuation function of stomach

46. Which of the following approvals are true?

1. Cholinoblockers inhibit secretion of HCl in the stomach
2. Blockers of H_2 – histamine receptors stimulate secretion of HCl in the stomach
3. Aspirin promote development of ulcer disease due to blocking of cyclooxygenase
4. Aspirin inhibits development of ulcer disease due to inhibition prostaglandin secretion in the stomach
5. Beer and dry wine stimulate secretion of HCl in the stomach

47. Point out what from the following can play an impotent role in pathogenesis of stress-ulcers:

1. increased exitability of hypothalamic centers
2. low vagus activity
3. high tone of sympathetic nervous system
4. increased level of ACTH and glucocorticoids in the blood

48. Point out what from the following can play an impotent role in pathogenesis of stress-ulcers:

1. increased level of ACTH and glucocorticoids in the blood
2. increased activity of acid-peptic factor
3. stimulation of mucus formation in the stomach
4. duodeno-gastric reflux

49. Which of the following are characteristics (markers) of the mucus condition:

1. pepsinogen
2. H^+ ions
3. glycoproteins
4. gastromucoproteine
5. sialic acids
6. N-acetylneuraminic acid (NANA)

50. Which of the following can facilitate the peptic ulceration:

1. decreasing in gastric motor function
2. decreasing in duodenal motor function
3. duodeno-gastric reflux
4. gastro-esophagal reflux
5. duodenostasis

51. Point out the typical consequences of duodeno-gastric reflux:

1. increased formation of mucus in the stomach
2. weakening of mucus barrier
3. injury of epithelial cells
4. metaplasia of gastric epithelium
5. increased risk of stomach malignancy
6. bacterial overgrowth

52. Which are characteristics of Zollinger-Ellison syndrome?

1. hyperplasia of D-cells in pyloric antrum
2. hyperplasia of G-cells in pyloric antrum
3. gastrinsecreting pancreatic tumor
4. gastrinsecreting duodenal tumor
5. hyperplasia of enterochromaffin-like cells

53. Point out the pathogenetic factors of gastric "aspirin" ulcers:

1. decreased synthesis of prostaglandins E
2. increase in mucus formation
3. increase in H^+ - formation in deep layers in mucous membrane

54. Point out pathogenetic factors of dumping syndrome:

1. rapid evacuation of food from stomach into jejunum
2. hyperirritation of receptors of gut wall
3. elaboration of serotonin and VIP
4. decreasing in the blood epinephrine and bradykinin
5. vasodilation and the increase of mesenteric vascular permeability
6. increase in total blood volume

II level

1. List the reflexogenic areas which irritation can lead to the vomiting.
   1…..2…..3…..4…..5…..6…
2. Enumerate the stomach function that can be associated with digestive disorders.
   1…2…3…4…5…
3. Write the causes of putrefaction and fermentation in the gut at acholia.
   1…..2…..
4. Write the forms of ileus.
   1….2….3….
5. List the main manifestations of the gut excretory function disorders.
   1……2….
6. List the main syndromes typical for bacteria overgrowth (disbacteriosis).
   1…2….3….
7. Write the main principles of peptic ulcer treatment.
   1….2…..3…..4….5…..6….
8. Enumerate the protective antiulcerous factors of the mucous in GIT.
   1…2…3…4…5…6…
9. List the external factors with ulcerous effect.
   1…2…3…4…
10. List the internal factors with ulcerous effect.
    1…2…3…4…
11. Write the signs of hereditary predisposition to the peptic ulcer disease.
    1….2….3….4….5..
12. List the examples of secondary symptomatic peptic ulcers.
    1…2…3…4…
13. Write the main protective properties of GIT mucus.
    1…2…3…4…5..
14. Write the main protective antiulcerous factors of prostaglandins E.
    1…2…3…4…
15. List the main pathogenic factors of Helycobacter pilori.
    1…2…3…4…5…
16. Enumerate the main medicine which have ulcerous effect.
    1…2…3…4…
17. Enumerate the main alimentary (food) factors that can facilitate peptic ulcer disease. 1…2…3…4…5…