1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

A 40-year-old man comes to the emergency department because of right upper quadrant pain and fever. During the abdominal examination, the patient is instructed to breathe out while the examiner applies gentle pressure in the patient's right upper quadrant below the costal margin at the midclavicular line. The patient is then instructed to breathe in. At this point, he winces and abruptly stops his inhalation.

What is the function of the organ that likely needs to be removed?

A. Conjugation of bile

B. Production of bile

C. Production of coagulation factors

D. Secretion of digestive enzymes

E. Storage of bile

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is E. 88% chose this.

The patient is suffering from acute cholecystitis, which is supported by his clinical presentation and positive Murphy sign on abdominal exam. The inflamed gallbladder comes in contact with the examiner's hand on inspiration and elicits tenderness. Murphy sign is detected by first finding the exact location of the gallbladder with the use of ultrasound imaging and then eliciting maximal point tenderness beneath the ultrasound probe with the application of pressure to the gallbladder. This patient will likely undergo cholecystectomy. The primary function of the gallbladder is the storage and concentration of bile, though there are minimal changes in digestion following cholecystectomy.

Cholecystectomy Cholecystitis Gallbladder Ultrasound Medical ultrasound Bile Murphy's sign Digestion

A is not correct. 3% chose this.

Conjugation of bile occurs in the liver, where bile is conjugated with taurine or glycine to form bile salts, which are water-soluble and able to emulsify lipids. Since obstruction of cystic ducts by gallstones results in inflammation of the gallbladder in cholecystitis, removal of the liver is not necessary.

Cholecystitis Glycine Taurine Gallbladder Bile Liver Bile acid Gallstone Emulsion Lipid Inflammation Conjugated system

B is not correct. 5% chose this.

Bile is produced in liver hepatocytes, where primary bile acids are synthesized. They are then conjugated with taurine or glycine to form bile salts. In the gallbladder, bile is concentrated as solutes and water are reabsorbed.

Taurine Glycine Gallbladder Bile acid Liver Bile Hepatocyte Conjugated system

C is not correct. 2% chose this.

Production of coagulation factors occurs in the liver, not the gallbladder. Liver is not affected in the pathophysiology of cholecystitis and hence its removal is not surgically necessary.

Cholecystitis Gallbladder Liver Coagulation Pathophysiology

D is not correct. 2% chose this.

The pancreas is responsible for secreting enzymes, including trypsin, pancreatic lipase, pancreatic amylase, and chymotrypsin, to help with the digestion of carbohydrates, protein, and fat.

Chymotrypsin Trypsin Pancreatic lipase Amylase Pancreas Carbohydrate Lipase Protein Digestion Enzyme

Bottom Line:

The gallbladder is responsible for the storage of bile. A positive Murphy sign supports a diagnosis of acute cholecystitis, which will require removal of the gallbladder.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

of carbohydrates, protein, and fat.
Chymotrypsin Trypsin Pancreatic lipase Amylase Pancreas Carbohydrate Lipase Protein Digestion Enzyme

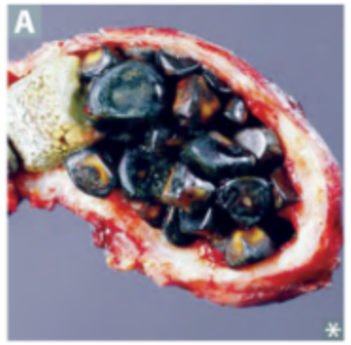
Bottom Line:

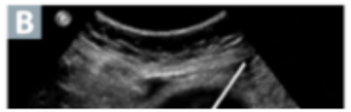
The gallbladder is responsible for the storage of bile. A positive Murphy sign supports a diagnosis of acute cholecystitis, which will require removal of the gallbladder.
Cholecystitis Gallbladder Cholecystectomy Bile Murphy's sign

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 379.1

Gallstones (cholelithiasis)





↑ cholesterol and/or bilirubin, ↓ bile salts, and gallbladder stasis all cause stones.

2 types of stones:

- Cholesterol stones (radiolucent with 10–20% opaque due to calcifications)—80% of stones. Associated with obesity, Crohn disease, advanced age, estrogen therapy, multiparity, rapid weight loss, Native American origin.
- Pigment stones A (black = radiopaque, Ca^{2+} bilirubinate, hemolysis; brown = radiolucent, infection). Associated with Crohn disease, chronic hemolysis, alcoholic cirrhosis,

Risk factors (4 F's):

1. Female
2. Fat
3. Fertile (pregnant)
4. Forty

Diagnose with ultrasound B. Treat with elective cholecystectomy if symptomatic.

Can cause fistula between gallbladder and GI tract → air in biliary tree (pneumobilia) → passage of gallstones into intestinal tract → obstruction of ileocecal valve (gallstone ileus).

- Altered mental status
- Shock (hypotension)

FA17 p 358.5

Bile

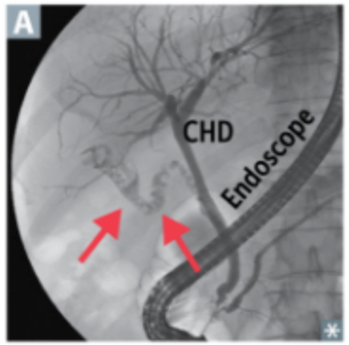
Composed of bile salts (bile acids conjugated to glycine or taurine, making them water soluble), phospholipids, cholesterol, bilirubin, water, and ions. Cholesterol 7α-hydroxylase catalyzes rate-limiting step of bile acid synthesis.

Functions:

- Digestion and absorption of lipids and fat-soluble vitamins
- Cholesterol excretion (body's 1° means of eliminating cholesterol)
- Antimicrobial activity (via membrane disruption)

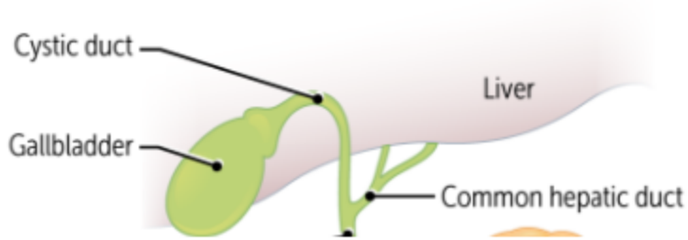
FA17 p 353.1

Biliary structures



Gallstones (filling defects in gallbladder and cystic duct, red arrows in A) that reach the confluence of the common bile and pancreatic ducts at the ampulla of Vater can block both the common bile and pancreatic ducts (double duct sign), causing both cholangitis and pancreatitis, respectively.

Tumors that arise in head of pancreas (usually ductal adenocarcinoma) can cause obstruction of common bile duct → enlarged gallbladder with painless jaundice (Courvoisier sign).



1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

A 3-week-old boy is brought to the emergency department by his parents after a 1-week history of nonbilious vomiting. His parents report that the child spits up after meals much more frequently than his older brother did at this age and that the vomiting sometimes seems forceful.

The section of the gastrointestinal tract affected in this condition typically plays a role in regulating which of the following parts of digestion?

A. Gastric acid production

B. Gastric emptying

C. Prevention of food backflow into the esophagus

D. Propulsion of food into the stomach

E. Swallowing

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is B. 63% chose this.

This patient presents with a history of classic pyloric stenosis, which involves the pyloric sphincter, which controls passage of gastric contents into the duodenal bulb. This condition is much more common in male than in female infants and usually develops between 3 and 5 weeks of age, and very rarely after 12 weeks. The patient presents with regurgitation and relentless projectile, nonbilious vomiting. On physical examination, visible peristalsis generally can be seen, and a mass (commonly described as "olive"-like) usually can be palpated in the epigastric region. The normal role of the pylorus is to limit the rate of gastric emptying. This is accomplished by contracting in response to each peristaltic wave.

Duodenal bulb Peristalsis Pylorus Pyloric sphincter Pyloric stenosis Duodenum Stenosis Epigastrium Regurgitation (digestion) Sphincter Vomiting Palpation Physical examination Stomach

A is not correct. 4% chose this.

Gastric acid is secreted by parietal cells located in the gastric glands of the body and fundus of the stomach. Gastrin-induced histamine release is the primary stimulus for acid production, but acetylcholine from the parasympathetic nervous system also plays a role. Acid production is inhibited by the hormone somatostatin. Dysfunctional acid production would lead to impaired ability to break up food particles but does not lead to the projectile vomiting.

Somatostatin Gastric acid Histamine Acetylcholine Parasympathetic nervous system Parietal cell Gastric glands Hormone Vomiting Fundus (stomach) Parietal lobe Stomach

C is not correct. 17% chose this.

The lower esophageal sphincter, located at the gastroesophageal junction, is responsible for preventing backflow of food and acid from the stomach into the esophagus. If this sphincter fails to constrict completely, the patient may experience acid reflux accompanied by an epigastric burning sensation. In infants, if the lower esophageal sphincter does not form properly, it is possible for stomach contents to back up into the esophagus and vomiting to occur; however, this action is not projectile, as seen in classic pyloric stenosis.

Esophagus Gastroesophageal junction Gastroesophageal reflux disease Lower esophageal sphincter Pyloric stenosis Stenosis Sphincter Epigastrium Vomiting Cardia Pylorus Esophageal cancer Stomach

D is not correct. 12% chose this.

The inner circular and outer longitudinal muscle layers of the esophagus aid in esophageal motility. The peristaltic contractions of the esophagus help propel food into the stomach. Dysregulation of this process can result in diffuse esophageal spasm, which can cause chest pain and dysphagia.

Dysphagia Esophagus Peristalsis Esophageal spasm Chest pain Motility Esophageal cancer Diffuse esophageal spasm Stomach Muscle

E is not correct. 4% chose this.

The process of swallowing is initiated when touch receptors near the pharyngeal opening are stimulated, sending a signal to the swallowing center in the medulla and lower pons. This center sends impulses to the muscles of the pharynx and esophagus, which then enact swallowing.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The inner circular and outer longitudinal muscle layers of the esophagus aid in esophageal motility. The peristaltic contractions of the esophagus help propel food into the stomach. Dysregulation of this process can result in diffuse esophageal spasm, which can cause chest pain and dysphagia.

Dysphagia Esophagus Peristalsis Esophageal spasm Chest pain Motility Esophageal cancer Diffuse esophageal spasm Stomach Muscle

E is not correct. 4% chose this.

The process of swallowing is initiated when touch receptors near the pharyngeal opening are stimulated, sending a signal to the swallowing center in the medulla and lower pons. This center sends impulses to the muscles of the pharynx and esophagus, which then enact swallowing.

Esophagus Pharynx Pons Medulla oblongata Somatosensory system Pharyngeal opening of auditory tube Receptor (biochemistry)

Bottom Line:

Pyloric stenosis in infants presents with regurgitation and relentless projectile, nonbilious vomiting. On physical examination, peristalsis generally can be seen, and a mass (commonly described as "olive"-like) usually can be palpated in the epigastric region.

Peristalsis Pyloric stenosis Stenosis Regurgitation (digestion) Vomiting Epigastrium Palpation Pylorus Physical examination Regurgitation (circulation)

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 345.1

Hypertrophic pyloric stenosis

Most common cause of gastric outlet obstruction in infants (1:600). Palpable olive-shaped mass in epigastric region, visible peristaltic waves, and nonbilious projectile vomiting at ~ 2–6 weeks old. More common in firstborn males; associated with exposure to macrolides. Results in hypokalemic hypochloremic metabolic alkalosis (2° to vomiting of gastric acid and subsequent volume contraction). Treatment is surgical incision (pyloromyotomy).

FA17 p 561.2

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

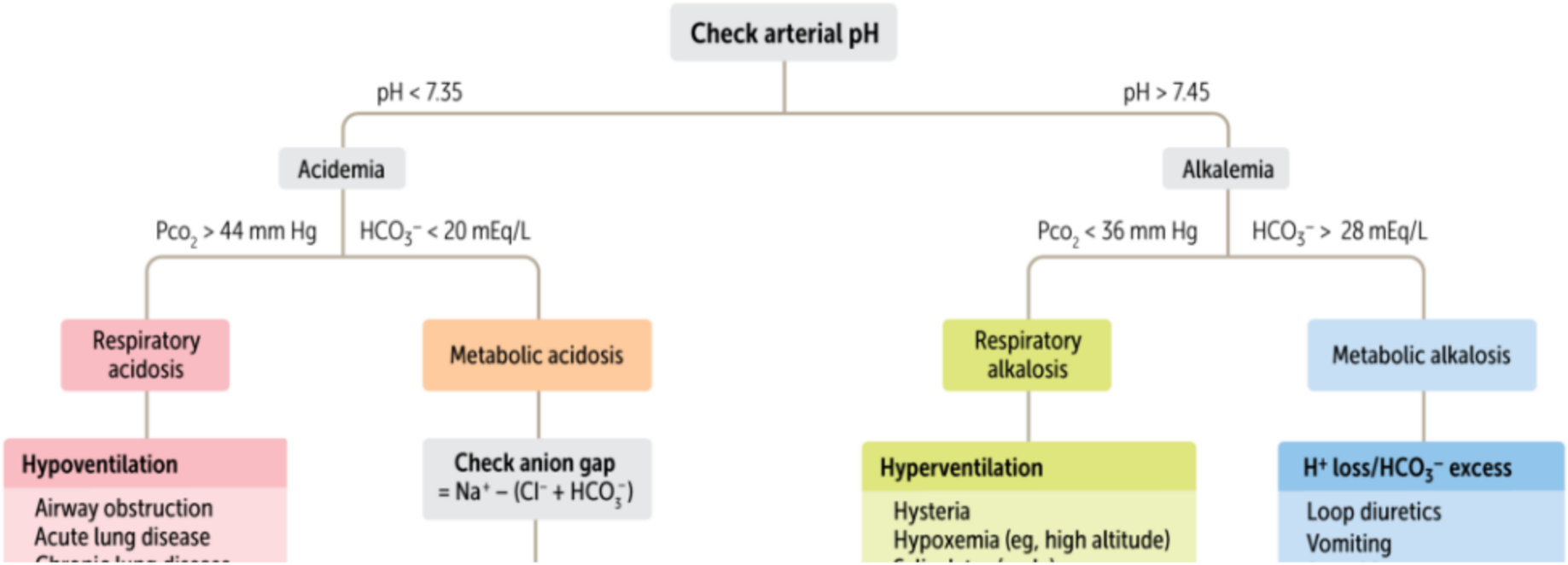
FA17 p 345.1

Hypertrophic pyloric stenosis

Most common cause of gastric outlet obstruction in infants (1:600). Palpable olive-shaped mass in epigastric region, visible peristaltic waves, and nonbilious projectile vomiting at ~ 2–6 weeks old. More common in firstborn males; associated with exposure to macrolides. Results in hypokalemic hypochloremic metabolic alkalosis (2° to vomiting of gastric acid and subsequent volume contraction). Treatment is surgical incision (pyloromyotomy).

FA17 p 561.2

Acidosis and alkalosis



1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

A 2-year-old boy is brought to the emergency department because of a fever of 40°C (104°F). He is making poor eye contact, displays limited interaction with his surroundings, and appears ill. Blood culture is positive for *Streptococcus pneumoniae*.

Which of the following are cells in the liver that would help to clear this organism?

A. Alveolar macrophages

B. Kupffer cells

C. Langerhans cells

D. Mesangial cells

E. Microglia

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is B. 84% chose this.

The child has bacteremia. Bacteremia, which is defined by presence of viable bacteria in the circulating blood, may result from simple daily activities such as tooth brushing or from more complicated causes such as indwelling catheters, dental procedures, intravenous drug use or infections. *Streptococcus pneumoniae* is a gram-positive, α-hemolytic diplococcus that may cause pneumonia, bacteremia, meningitis, and cellulitis. Bacteria in the bloodstream are typically destroyed by the reticuloendothelial system. Kupffer cells are reticuloendothelial cells found in the liver that would help to clear this organism from the bloodstream.

Streptococcus pneumoniae Bacteremia Meningitis Cellulitis Gram-positive bacteria Pneumonia Kupffer cell Streptococcus Intravenous therapy Bacteria Drug injection Liver Mononuclear phagocyte system Catheter Circulatory system Diplococcus

A is not correct. 6% chose this.

Alveolar macrophages are part of the reticuloendothelial system found in the lung. They are phagocytic cells and clear microorganisms from the circulation.

Alveolar macrophage Mononuclear phagocyte system Phagocyte Macrophage Phagocytosis Microorganism Pulmonary alveolus Lung

C is not correct. 5% chose this.

Langerhans cells are involved in antigen uptake in the skin and travel to lymph nodes to present antigen to T-lymphocytes.

Antigen T cell Langerhans cell Lymph node Lymph

D is not correct. 3% chose this.

Mesangial cells are part of the reticuloendothelial system found in the spleen and kidney. They are phagocytic cells and clear microorganisms from the circulation.

Spleen Phagocyte Mononuclear phagocyte system Phagocytosis Kidney Mesangial cell Microorganism

E is not correct. 2% chose this.

Microglia cells are part of the reticuloendothelial system found in the brain. They are phagocytic cells and clear microorganisms from the circulation.

Microglia Mononuclear phagocyte system Phagocyte Phagocytosis Microorganism Human brain Brain

Bottom Line:

In bacteremia, Kupffer cells (reticuloendothelial cells found in the liver) help to clear pathogens from the bloodstream.

Bacteremia Kupffer cell Liver Mononuclear phagocyte system Pathogen Circulatory system

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Bottom Line:

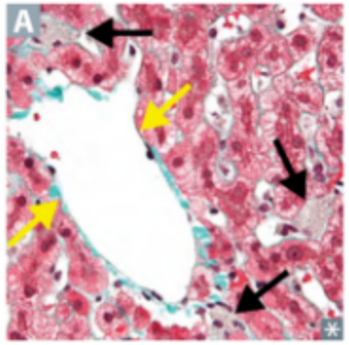
In bacteremia, Kupffer cells (reticuloendothelial cells found in the liver) help to clear pathogens from the bloodstream.

Bacteremia Kupffer cell Liver Mononuclear phagocyte system Pathogen Circulatory system

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 352.1

Liver tissue architecture



Apical surface of hepatocytes faces bile canaliculi. Basolateral surface faces sinusoids.

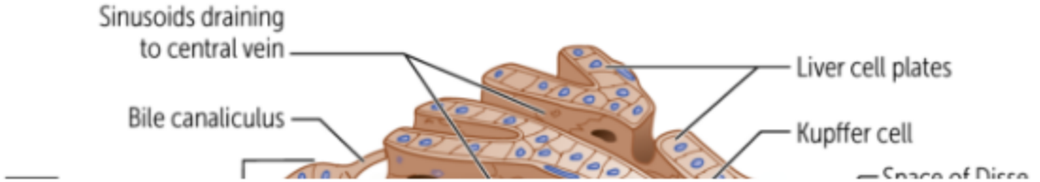
Kupffer cells, which are specialized macrophages, form the lining of these sinusoids (black arrows in A; 2 yellow arrows show hepatic venule).

Hepatic stellate (Ito) cells in space of Disse store vitamin A (when quiescent) and produce extracellular matrix (when activated).

Zone I—periportal zone:
- Affected 1st by viral hepatitis
- Ingested toxins (eg, cocaine)

Zone II—intermediate zone:
- Yellow fever

Zone III—pericentral vein (centrilobular) zone:
- Affected 1st by ischemia
- Contains cytochrome P-450 system
- Most sensitive to metabolic toxins
- Site of alcoholic hepatitis



1
2
3
▪ 4
▪ 5
▪ 6
▪ 7
▪ 8
▪ 9
▪ 10
▪ 11
▪ 12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

A 22-year-old college student presents to the emergency department with sudden-onset severe epigastric pain that radiates to the back. He has a history of heavy alcohol abuse.

The organ involved in this patient's pathology plays a key role in normal gastrointestinal physiology. Under normal physiologic conditions, the organ is stimulated by a hormone secreted by the duodenum in response to acidic chyme present in the lumen.

What effect does this hormone have when it stimulates the organ of interest?

A. Insulin secretion

B. Secretion of acid

C. Secretion of bicarbonate

D. Secretion of bile

E. Secretion of intrinsic factor

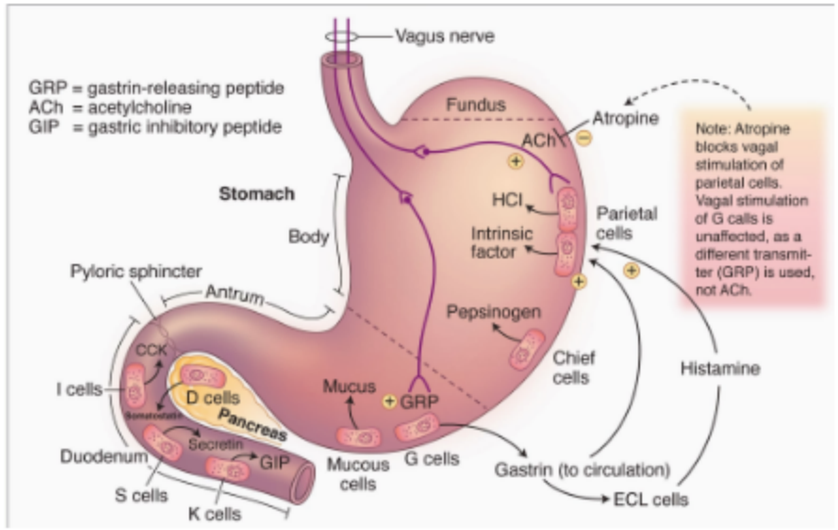
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is C. 73% chose this.

Secretin is released from duodenal S-cells when acidic chyme from the stomach is present in the duodenal lumen. Secretin acts on pancreatic duct cells to stimulate bicarbonate secretion. The diagram is an overview of the locations of GI secretory cells.

Chyme Secretin Pancreatic duct Secretin receptor Bicarbonate Duodenum Lumen (anatomy)

Acid Secretion Stomach



A is not correct. 8% chose this.

Insulin secretion is regulated by glucose levels in the blood. A rise in blood glucose results in glucose uptake into pancreatic β cells, facilitated by the GLUT-2 glucose transporter. Metabolism of glucose generates ATP, increasing the intracellular ATP/ADP ratio. This inhibits the activity of the ATP-sensitive K^+ channel on the β-cell membrane, leading to membrane depolarization and the influx of Ca^{2+} through voltage-dependent Ca^{2+} channels. Increased intracellular Ca^{2+} stimulates secretion of insulin.

Glucose transporter Glucose Depolarization Insulin signal transduction pathway Pulsatile insulin Adenosine triphosphate Insulin Blood sugar Metabolism

Intracellular Secretion

B is not correct. 3% chose this.

Parietal cells of the gastric fundus secrete acid in response to gastrin, histamine, and vagal acetylcholine release. Acid is not produced by the pancreas.

Gastric fundus Gastrin Histamine Acetylcholine Pancreas Parietal cell Vagus nerve Parietal lobe Fundus (stomach)

D is not correct. 14% chose this.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

D is not correct. 14% chose this.

Cholecystokinin (CCK) is produced and secreted by the I cells of the duodenum in response to small peptides and fatty acids from the stomach. CCK acts to stimulate contraction of the gallbladder while simultaneously relaxing the sphincter of Oddi. This results in bile secretion. CCK is not released in responsed to high acid levels and is therefore not correct.

Sphincter of Oddi Cholecystokinin Duodenum Gallbladder Bile Sphincter Fatty acid Peptide Cholecystokinin receptor Secretion Stomach

E is not correct. 2% chose this.

Intrinsic factor is produced by parietal cells of the gastric fundus and not by the pancreas. It is important for vitamin B_{12} absorbtion in the gut.

Intrinsic factor Gastric fundus Vitamin B12 Pancreas Parietal cell B vitamins Vitamin Fundus (stomach) Gastrointestinal tract

Bottom Line:

Secretin primarily acts on the pancreas to stimulate secretion of bicarbonate.

Secretin Pancreas Bicarbonate

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 357.1

Gastrointestinal secretory products

PRODUCT	SOURCE	ACTION	REGULATION	NOTES
Intrinsic factor	Parietal cells (stomach)	Vitamin B_{12}–binding protein (required for B_{12} uptake in terminal ileum)		Autoimmune destruction of parietal cells → chronic gastritis and pernicious anemia.
Gastric acid	Parietal cells	↓ stomach pH	↑ by histamine	

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FA17 p 357.1

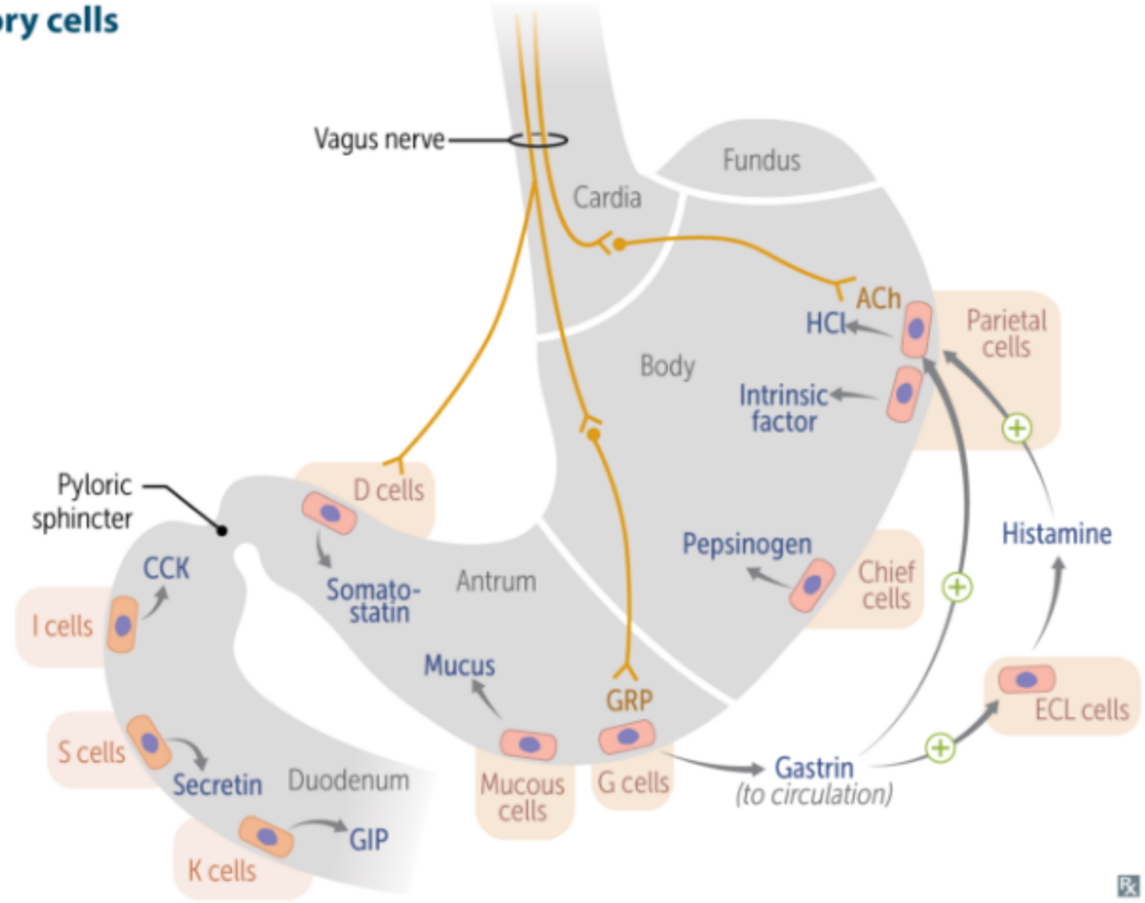
Gastrointestinal secretory products

PRODUCT	SOURCE	ACTION	REGULATION	NOTES
Intrinsic factor	Parietal cells (stomach)	Vitamin B_{12}–binding protein (required for B_{12} uptake in terminal ileum)		Autoimmune destruction of parietal cells → chronic gastritis and pernicious anemia.
Gastric acid	Parietal cells (stomach)	↓ stomach pH	↑ by histamine, ACh, gastrin ↓ by somatostatin, GIP, prostaglandin, secretin	
Pepsin	Chief cells (stomach)	Protein digestion	↑ by vagal stimulation, local acid	Pepsinogen (inactive) is converted to pepsin (active) in the presence of H^+.
Bicarbonate	Mucosal cells (stomach, duodenum, salivary glands, pancreas) and Brunner glands (duodenum)	Neutralizes acid	↑ by pancreatic and biliary secretion with secretin	Trapped in mucus that covers the gastric epithelium.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FA17 p 357.2

Locations of gastrointestinal secretory cells



Gastrin ↑ acid secretion primarily through its effects on enterochromaffin-like (ECL) cells (leading to histamine release) rather than through its direct effect on parietal cells.

1
2
3
4
▪ 5
▪ 6
▪ 7
▪ 8
▪ 9
▪ 10
▪ 11
▪ 12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

A 42-year-old mother of five comes to a primary care physician complaining of acute pain in her abdomen for 3 days that is most pronounced after meals. She is afebrile, her vital signs are stable, and her body mass index is 34 kg/m². On physical examination she has tenderness to palpation with voluntary guarding under the rib cage on the right side. Ultrasound confirms the diagnosis. The patient is counseled on surgical options as well as lifestyle modifications.

Which pair correctly matches the type of food the patient should avoid with the function of the hormone that is causing her abdominal pain?

A. High-carbohydrate foods; inhibits gastric acid secretion

B. High-carbohydrate foods; stimulates gastric acid secretion

C. High-fat foods; increases gastric motility

D. High-fat foods; stimulates gallbladder contraction

E. High-fat foods; stimulates pancreatic bicarbonate secretion

F. High-protein foods; inhibits gastric emptying

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is D. 85% chose this.

The patient has gallstones (cholelithiasis), as supported by her clinical presentation, ultrasound diagnosis, and risk factors (the "**4 F's**": **F**emale, **F**at, **F**ertile, and **F**orty years old or older). The gallbladder typically holds bile acids and lecithin, used to solubilize cholesterol and/or bilirubin. When these solubilizing substances are overwhelmed by high concentrations of either cholesterol and/or bilirubin, precipitants form and eventually make gallstones. Eating high-fat foods stimulates the release of cholecystokinin (CCK) by I cells of the duodenum and jejunum. CCK has multiple functions in the gastrointestinal tract including stimulation of gallbladder contraction. This can cause increased pain in gallstone sufferers. CCK also stimulates pancreatic enzyme secretion and inhibits gastric emptying.

Gallstone Bilirubin Jejunum Cholecystokinin Duodenum Gallbladder Lecithin Enzyme Human gastrointestinal tract Cholesterol Gastrointestinal tract Bile Bile acid Ultrasound Pancreas Medical ultrasound Secretion Stomach

A is not correct. 2% chose this.

Secretin, which inhibits gastric acid secretion, does not have a direct effect on the gallbladder like cholecystokinin does. It is stimulated by an increase in acid or fatty acids in the lumen of the duodenum, and is not stimulated by high-carbohydrate foods.

Secretin Cholecystokinin Duodenum Gastric acid Gallbladder Lumen (anatomy) Fatty acid Secretion

B is not correct. 4% chose this.

Gastrin does not directly affect the gallbladder. Its release is stimulated by gastric distention, amino acids, peptides, and the vagus nerve, so high-carbohydrate foods would not be expected to significantly stimulate its release.

Gastrin Gallbladder Vagus nerve Amino acid Peptide

C is not correct. 4% chose this.

Gastrin secretion by the G cells of the antrum of the stomach stimulates the secretion of stomach acids as well as increases gastric motility. Unlike cholecystokinin, gastrin does not have a direct effect on the gallbladder.

Cholecystokinin Gastrin Gallbladder G cell Pyloric antrum Antrum Secretion Stomach

E is not correct. 3% chose this.

High-fat foods are to be avoided in gallstone sufferers, but to prevent the release of cholecystokinin, not secretin. Secretin is stimulated by acid and fatty acids in the duodenum and is secreted by S cells of the duodenum. It stimulates pancreatic bicarbonate secretion to neutralize stomach acids and inhibits gastric acid secretion.

Gallstone Secretin Cholecystokinin Duodenum Gastric acid Bicarbonate Fatty acid Secretion Stomach

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

cholecystokinin, gastrin does not have a direct effect on the gallbladder.
Cholecystokinin Gastrin Gallbladder G cell Pyloric antrum Antrum Secretion Stomach

E is not correct. 3% chose this.

High-fat foods are to be avoided in gallstone sufferers, but to prevent the release of cholecystokinin, not secretin. Secretin is stimulated by acid and fatty acids in the duodenum and is secreted by S cells of the duodenum. It stimulates pancreatic bicarbonate secretion to neutralize stomach acids and inhibits gastric acid secretion.
Gallstone Secretin Cholecystokinin Duodenum Gastric acid Bicarbonate Fatty acid Secretion Stomach

F is not correct. 2% chose this.

High-fat, not high-protein, foods should be avoided to decrease cholecystokinin (CCK) release. Although amino acids also regulate CCK secretion, fatty foods have a greater effect on its release. CCK inhibits gastric emptying.
Cholecystokinin Amino acid Stomach CCK Secretion

Bottom Line:

Gallstone risk factors include the "**4 F**'s." Cholecystokinin, which stimulates gallbladder contraction, causes increased pain in patients with gallstones.
Gallstone Cholecystokinin Gallbladder

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 356.1

Gastrointestinal regulatory substances

REGULATORY SUBSTANCE	SOURCE	ACTION	REGULATION	NOTES
Gastrin	G cells (antrum of stomach, duodenum)	↑ gastric H^+ secretion ↑ growth of gastric mucosa ↑ gastric motility	↑ by stomach distention/ alkalinization,	↑ by chronic PPI use. ↑ in chronic atrophic gastritis (eg, *H pylori*).

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 356.1

Gastrointestinal regulatory substances

REGULATORY SUBSTANCE	SOURCE	ACTION	REGULATION	NOTES
Gastrin	G cells (antrum of stomach, duodenum)	↑ gastric H^+ secretion ↑ growth of gastric mucosa ↑ gastric motility	↑ by stomach distention/ alkalinization, amino acids, peptides, vagal stimulation via gastrin-releasing peptide (GRP) ↓ by pH < 1.5	↑ by chronic PPI use. ↑ in chronic atrophic gastritis (eg, *H pylori*). ↑↑ in Zollinger-Ellison syndrome (gastrinoma).
Somatostatin	D cells (pancreatic islets, GI mucosa)	↓ gastric acid and pepsinogen secretion ↓ pancreatic and small intestine fluid secretion ↓ gallbladder contraction ↓ insulin and glucagon release	↑ by acid ↓ by vagal stimulation	Inhibits secretion of various hormones (encourages somato-stasis). Octreotide is an analog used to treat acromegaly, carcinoid syndrome, and variceal bleeding.
Cholecystokinin	I cells (duodenum, jejunum)	↑ pancreatic secretion ↑ gallbladder contraction ↓ gastric emptying	↑ by fatty acids, amino acids	Acts on neural muscarinic pathways to cause pancreatic secretion.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Ghrelin	Stomach	↑ appetite	↑ in fasting state ↓ by food	↑ in Prader-Willi syndrome. ↓ after gastric bypass surgery.

FA17 p 358.1

Pancreatic secretions Isotonic fluid; low flow → high Cl^-, high flow → high HCO_3^-.

ENZYME	ROLE	NOTES
α-amylase	Starch digestion	Secreted in active form
Lipases	Fat digestion	
Proteases	Protein digestion	Includes trypsin, chymotrypsin, elastase, carboxypeptidases Secreted as proenzymes also known as zymogens
Trypsinogen	Converted to active enzyme trypsin → activation of other proenzymes and cleaving of additional trypsinogen molecules into active trypsin (positive feedback loop)	Converted to trypsin by enterokinase/enteropeptidase, a brush-border enzyme on duodenal and jejunal mucosa

FA17 p 379.1

Gallstones (cholelithiasis)



↑ cholesterol and/or bilirubin, ↓ bile salts, and gallbladder stasis all cause stones.

2 types of stones:

- Cholesterol stones (radiolucent with 10–20%

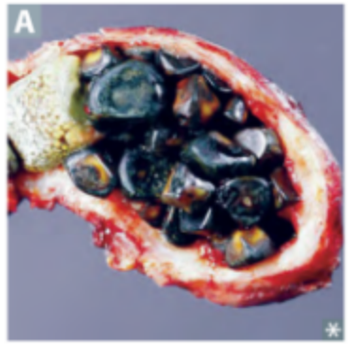
Risk factors (4 F's):

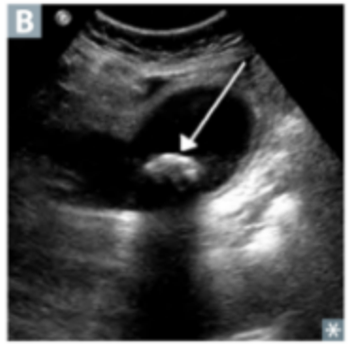
1. Female
2. Fat
3. Fertile (pregnant)

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FA17 p 379.1

Gallstones (cholelithiasis)





↑ cholesterol and/or bilirubin, ↓ bile salts, and gallbladder stasis all cause stones.

2 types of stones:

- Cholesterol stones (radiolucent with 10–20% opaque due to calcifications)—80% of stones. Associated with obesity, Crohn disease, advanced age, estrogen therapy, multiparity, rapid weight loss, Native American origin.
- Pigment stones A (black = radiopaque, Ca^{2+} bilirubinate, hemolysis; brown = radiolucent, infection). Associated with Crohn disease, chronic hemolysis, alcoholic cirrhosis, advanced age, biliary infections, total parenteral nutrition (TPN).

Most common complication is cholecystitis; can also cause acute pancreatitis, ascending cholangitis.

Risk factors (4 F's):

1. Female
2. Fat
3. Fertile (pregnant)
4. Forty

Diagnose with ultrasound B. Treat with elective cholecystectomy if symptomatic.

Can cause fistula between gallbladder and GI tract → air in biliary tree (pneumobilia) → passage of gallstones into intestinal tract → obstruction of ileocecal valve (gallstone ileus).

RELATED PATHOLOGIES	CHARACTERISTICS
Biliary colic	Associated with nausea/vomiting and dull RUQ pain. Neurohormonal activation (eg, by CCK after a fatty meal) triggers contraction of gallbladder, forcing stone into cystic duct. Labs are normal, ultrasound shows cholelithiasis.
Choledocholithiasis	Presence of gallstone(s) in common bile duct, often leading to elevated ALP, GGT, direct bilirubin,

1
2
3
4
5
• 6
• 7
• 8
• 9
• 10
• 11
• 12
• 13
• 14
• 15
• 16
• 17
• 18

A 34-year-old man comes to his physician complaining of worsening heartburn and reflux. He has been taking over-the-counter ranitidine but feels he needs a stronger prescription.

What is the mechanism of action of ranitidine?

A. Binds to the gastrin receptor on parietal cells

B. Binds to the M_3-receptor

C. Binds to the prostaglandin receptor

D. Binds to the somatostatin receptor

E. Inhibition of a G_s-coupled receptor

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is E. 56% chose this.

Ranitidine belongs to the class of drugs termed H_2-receptor antagonists. Other H_2-receptor antagonists include famotidine and cimetidine. They bind to H_2-receptors on parietal cells to block the receptors' interaction with histamine. This leads to an inhibition of G_s-coupled receptor, resulting in decreased downstream cAMP and acid production.

Cimetidine Ranitidine Famotidine Histamine Parietal cell Cyclic adenosine monophosphate

A is not correct. 20% chose this.

There is no known inhibitor for the gastrin receptor.

Gastrin Receptor (biochemistry)

B is not correct. 11% chose this.

Anticholinergics such as atropine bind to the M_3-receptor to block its interaction with acetylcholine, which stimulates acid secretion.

Atropine Acetylcholine Anticholinergic Secretion

C is not correct. 7% chose this.

Prostaglandins bind to the prostaglandin receptor on parietal cells and decrease acid secretion.

Prostaglandin receptor Prostaglandin Parietal cell Receptor (biochemistry) Secretion

D is not correct. 6% chose this.

There are no known inhibitors of the somatostatin receptor.

Somatostatin Somatostatin receptor Receptor (biochemistry)

Bottom Line:

Ranitidine is an H_2-blocker that reversibly blocks histamine H_2-receptors to decrease hydrogen ion secretion by parietal cells, and thereby relieves symptoms of heartburn and reflux.

Ranitidine Histamine Heartburn Parietal cell Gastroesophageal reflux disease Hydrogen Secretion

FIRST AID FIRST AID FACTS for year: 2017

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Bottom Line:

Ranitidine is an H_2-blocker that reversibly blocks histamine H_2-receptors to decrease hydrogen ion secretion by parietal cells, and thereby relieves symptoms of heartburn and reflux.

Ranitidine Histamine Heartburn Parietal cell Gastroesophageal reflux disease Hydrogen Secretion

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 381.2

H_2 blockers

Cimetidine, ranitidine, famotidine, nizatidine. Take H_2 blockers before you dine. Think "table for 2" to remember H_2.

MECHANISM	Reversible block of histamine H_2-receptors → ↓ H^+ secretion by parietal cells.
CLINICAL USE	Peptic ulcer, gastritis, mild esophageal reflux.
ADVERSE EFFECTS	Cimetidine is a potent inhibitor of cytochrome P-450 (multiple drug interactions); it also has antiandrogenic effects (prolactin release, gynecomastia, impotence, ↓ libido in males); can cross blood-brain barrier (confusion, dizziness, headaches) and placenta. Both cimetidine and ranitidine ↓ renal excretion of creatinine. Other H_2 blockers are relatively free of these effects.

FA17 p 357.1

Gastrointestinal secretory products

PRODUCT	SOURCE	ACTION	REGULATION	NOTES

ranitidine ↓ renal excretion of creatinine. Other H_2 blockers are relatively free of these effects.

FA17 p 357.1

Gastrointestinal secretory products

PRODUCT	SOURCE	ACTION	REGULATION	NOTES
Intrinsic factor	Parietal cells (stomach)	Vitamin B_{12}–binding protein (required for B_{12} uptake in terminal ileum)		Autoimmune destruction of parietal cells → chronic gastritis and pernicious anemia.
Gastric acid	Parietal cells (stomach)	↓ stomach pH	↑ by histamine, ACh, gastrin ↓ by somatostatin, GIP, prostaglandin, secretin	
Pepsin	Chief cells (stomach)	Protein digestion	↑ by vagal stimulation, local acid	Pepsinogen (inactive) is converted to pepsin (active) in the presence of H^+.
Bicarbonate	Mucosal cells (stomach, duodenum, salivary glands, pancreas) and	Neutralizes acid	↑ by pancreatic and biliary secretion with secretin	Trapped in mucus that covers the gastric epithelium.

1
2
3
4
5
6
▪ 7
▪ 8
▪ 9
▪ 10
▪ 11
▪ 12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

An 8-year-old boy presents to the emergency department because of 18 hours of severe vomiting. Arterial blood gas analysis reveals a pH of 7.48, a HCO_3^- level of 35 mEq/L, and a partial carbon dioxide pressure (PCO_2) of 48 mm Hg.

What is the type of acid-base disturbance occurring in this patient?

A. Metabolic alkalosis and metabolic acidosis

B. Metabolic alkalosis and respiratory acidosis

C. Metabolic alkalosis and respiratory alkalosis

D. Metabolic alkalosis with respiratory compensation

E. Respiratory alkalosis

F. Respiratory alkalosis with metabolic compensation

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is D. 65% chose this.

This patient is presenting with alkalosis (normal arterial pH is 7.35–7.45). The HCO_3^- level is substantially elevated (>11 mEq/L above normal), which suggests that the patient is experiencing metabolic alkalosis. This alkalosis is caused by the patient's recent history of severe vomiting. The vomiting causes a loss of hydrochloric acid from the gastrointestinal tract; this acid must be replaced, which triggers a respiratory compensation. Through hypoventilation, the PCO_2 rises and generates carbonic acid, which aids in lowering and normalizing the pH. This expected PCO_2 can be quantified with the formula (expected $PCO_2 = (0.7 \times HCO_3^-) + 20 \pm 5$). The patient's PCO_2 is 48, which is within the predicted range (39.5–49.5).

Hydrochloric acid Metabolic alkalosis Carbonic acid PH Respiratory compensation Hypoventilation Gastrointestinal tract Alkalosis Vomiting

Human gastrointestinal tract Metabolism

A is not correct. 3% chose this.

Vomiting typically induces a metabolic alkalosis due to a loss of H^+ from the stomach, leading to an increase in pH. This leaves an increased HCO_3^- concentration (generally >24 mEq/L) in the bloodstream; therefore, this patient does indeed have a metabolic alkalosis. However, some patients present with more than one condition causing an acid-base imbalance, which is known as a complicated, or mixed, condition. If a metabolic acidosis were occurring simultaneously (such as in ketoacidosis or diarrhea), the HCO_3^- level would be closer to normal because the two processes would have opposing effects on HCO_3^- levels and effectively cancel each other out. Therefore, there is no evidence of a concomitant metabolic acidosis in this vignette.

Metabolic alkalosis Metabolic acidosis Diarrhea Ketoacidosis Vomiting Alkalosis Acidosis Stomach Circulatory system Metabolism

B is not correct. 21% chose this.

This patient has a metabolic alkalosis, as evidenced by the increased pH with increased HCO_3^-level. PCO_2 is elevated as well, indicating that appropriate respiratory compensation is occurring. If a simultaneous respiratory acidosis were occurring, the PCO_2 would be even higher than anticipated, based on normal compensatory mechanisms. Therefore, there is no evidence of a concomitant respiratory acidosis in this vignette. Generally, PCO_2 should rise by 0.7 mm Hg for every 1-mEq/L increase in HCO_3^- level. So if CO_2 levels increase by more than that, a coexistent respiratory acidosis should be suspected.

Respiratory acidosis Metabolic alkalosis Respiratory compensation PH Alkalosis Acidosis Metabolism

C is not correct. 8% chose this.

This patient has an alkalosis, as evidenced by the elevated pH with increased HCO_3^-. PCO_2 is elevated as well, indicating that there is appropriate respiratory compensation occurring. If a simultaneous respiratory alkalosis were occurring, the PCO_2would be normal (meaning there is a failure of respiratory compensatory mechanisms) or it would be decreased. Therefore, there is no evidence of a concomitant respiratory alkalosis in this vignette. In an isolated metabolic alkalosis, respiratory compensation should occur. If it does not or if PCO_2 is low, a concurrent respiratory alkalosis should be suspected.

Respiratory alkalosis Metabolic alkalosis Respiratory compensation PH Alkalosis Metabolism Bicarbonate

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

C is not correct. 8% chose this.

This patient has an alkalosis, as evidenced by the elevated pH with increased HCO_3^-. PCO_2 is elevated as well, indicating that there is appropriate respiratory compensation occurring. If a simultaneous respiratory alkalosis were occurring, the PCO_2would be normal (meaning there is a failure of respiratory compensatory mechanisms) or it would be decreased. Therefore, there is no evidence of a concomitant respiratory alkalosis in this vignette. In an isolated metabolic alkalosis, respiratory compensation should occur. If it does not or if PCO_2 is low, a concurrent respiratory alkalosis should be suspected.

Respiratory alkalosis Metabolic alkalosis Respiratory compensation PH Alkalosis Metabolism Bicarbonate

E is not correct. 1% chose this.

Respiratory alkalosis results from increased respiration or hyperventilation, which increases the blood pH. However, the patient's PCO_2 is >40 mm Hg, suggesting that his symptoms are not caused by hyperventilation.

Respiratory alkalosis Hyperventilation Alkalosis Cellular respiration Respiration (physiology)

F is not correct. 2% chose this.

Respiratory alkalosis would result in the elevated pH seen in this patient, but would also cause a decreased PCO_2 of <40 mm Hg. Metabolic compensation occurs to lower the blood HCO_3^- levels by having the kidney excrete more HCO_3^-.

Respiratory alkalosis PH Alkalosis Kidney

Bottom Line:

Severe vomiting can cause metabolic alkalosis because of loss of hydrochloric acid from the gastrointestinal tract. This increase in pH will generally be counteracted by a decrease in respiratory rate, which will serve to decrease pH in the form of carbonic acid. A structured way to address acid-base questions is to look at the pH first to categorize the disorder as alkalosis or acidosis, and then to examine the PCO_2 levels to see if there is compensation.

Metabolic alkalosis Hydrochloric acid Carbonic acid PH Gastrointestinal tract Alkalosis Human gastrointestinal tract Vomiting Acidosis Respiratory rate Metabolism

FIRST AID FACTS for year: 2017

FA17 p 561.1

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FA17 p 561.1

Acid-base physiology

	pH	P_{CO_2}	$[HCO_3^-]$	COMPENSATORY RESPONSE
Metabolic acidosis	↓	↓	↓	Hyperventilation (immediate)
Metabolic alkalosis	↑	↑	↑	Hypoventilation (immediate)
Respiratory acidosis	↓	↑	↑	↑ renal $[HCO_3^-]$ reabsorption (delayed)
Respiratory alkalosis	↑	↓	↓	↓ renal $[HCO_3^-]$ reabsorption (delayed)

Key: ↑ ↓ = 1° disturbance; ↓ ↑ = compensatory response.

Henderson-Hasselbalch equation: $pH = 6.1 + \log \frac{[HCO_3^-]}{0.03\ P_{CO_2}}$

Predicted respiratory compensation for a simple metabolic acidosis can be calculated using the Winters formula. If measured P_{CO_2} > predicted P_{CO_2} → concomitant respiratory acidosis; if measured P_{CO_2} < predicted P_{CO_2} → concomitant respiratory alkalosis:

$$P_{CO_2} = 1.5\ [HCO_3^-] + 8 \pm 2$$

FA17 p 561.2

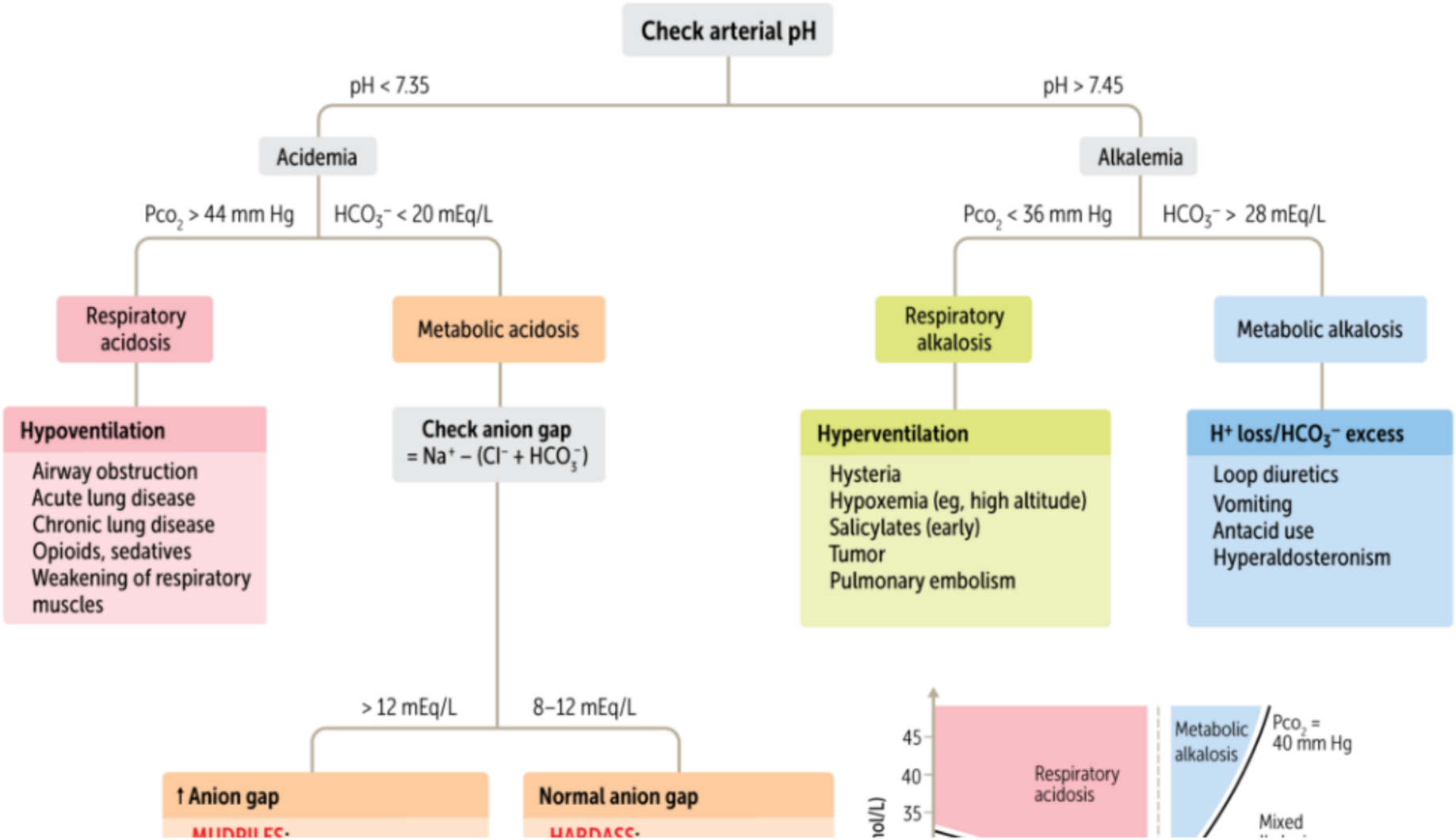
Acidosis and alkalosis



1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FA17 p 561.2

Acidosis and alkalosis



1
2
3
4
5
6
7
▪ 8
▪ 9
▪ 10
▪ 11
▪ 12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

A 10-year-old boy stayed home from school today because he is extremely nauseated and vomited six times overnight.

Choice	pH	Pco_2	HCO_3^-
A	<7.4	low	low
B	<7.4	high	high
C	>7.4	low	low
D	>7.4	low	high
E	>7.4	high	high

Which of the following set of laboratory values would most likely be found in this patient?

- A
- B
- C
- D
- E

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is E. 53% chose this.

Metabolic alkalosis is characterized by a pH >7.4 and high [HCO_3^-] due to loss of hydrogen ions in gastric acid, or a direct increase in [HCO_3^-]. Excessive vomiting causes a loss of hydrochloric acid from the stomach and is a common cause of metabolic alkalosis. In response to the alkalosis, the respiratory system compensates by decreasing ventilation, which increases the PCO_2, and lowers the pH. Other causes of metabolic alkalosis include diuretic or antacid use and hyperaldosteronism.

Notably, vomiting-induced metabolic alkalosis, even if severe enough to cause hypovolemia, is NOT a true contraction alkalosis. That term refers to the excessive loss of bicarbonate-poor fluid, such as in overdiuresis, that artificially increases the [HCO_3^-] by keeping a similar absolute amount of HCO_3^- in the serum while removing free water. Vomiting-induced metabolic alkalosis as in this case results from loss of hydrogen-rich fluid and is therefore a different pathophysiologic process and cannot be appropriately termed contraction alkalosis.

Antacid Metabolic alkalosis Hypovolemia Hydrochloric acid Diuretic Gastric acid PH Vomiting Alkalosis Hyperaldosteronism Blood plasma Stomach Metabolism Hydrogen Hydronium Respiratory system

A is not correct. 7% chose this.

This profile represents metabolic acidosis, which can be anion gap or non-anion gap. pH is low due to increased levels of H^+ or other acid. HCO_3^- is low because it is used to buffer excess H^+. PCO_2 is low as a result of respiratory compensation for the increased levels of acid. Causes of anion gap metabolic acidosis can be recalled using the mnemonic **MUDPILES** (**M**ethanol, **U**remia, **D**iabetic ketoacidosis, **P**araldehyde/phenformin, **I**ron/isoniazid, **L**actic acidosis, **E**thylene glycol, and **S**alicylates), and causes of non-anion gap metabolic acidosis include diarrhea, glue sniffing, renal tubular acidosis, and hyperchloremia.

Metabolic acidosis Anion gap Respiratory compensation Renal tubular acidosis Ethanol PH Diarrhea Acidosis Ketoacidosis Hyperchloremia Intoxicative inhalant Anion Mnemonic Metabolism Kidney

B is not correct. 5% chose this.

This profile represents respiratory acidosis, which is commonly caused by hypoventilation (chronic obstructive pulmonary disease, paralysis of respiratory muscles, opioids, or airway obstruction). The PCO_2 is high due to the decreased respiratory rate, which increases the level of acid in the blood, causing a low pH. The HCO_3^- will increase to compensate for the increased carbon dioxide/acid levels in the blood.

Respiratory acidosis Chronic obstructive pulmonary disease Hypoventilation Respiratory rate Acidosis Respiratory tract Opioid Airway obstruction Paralysis Respiratory disease Pulmonology

C is not correct. 12% chose this.

This profile represents respiratory alkalosis, most commonly caused by hyperventilation (anxiety, acute pain, or altitude). The PCO_2 is low due to the increased respiratory rate, which decreases the level of acid in the blood, causing a high pH. The HCO_3^- will decrease to compensate for decreased carbon dioxide/acid levels in the blood.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

low pH. The HCO_3^- will increase to compensate for the increased carbon dioxide/acid levels in the blood.

Respiratory acidosis Chronic obstructive pulmonary disease Hypoventilation Respiratory rate Acidosis Respiratory tract Opioid Airway obstruction Paralysis Respiratory disease Pulmonology

C is not correct. 12% chose this.

This profile represents respiratory alkalosis, most commonly caused by hyperventilation (anxiety, acute pain, or altitude). The PCO_2 is low due to the increased respiratory rate, which decreases the level of acid in the blood, causing a high pH. The HCO_3^- will decrease to compensate for decreased carbon dioxide/acid levels in the blood.

Respiratory alkalosis Hyperventilation Respiratory rate Alkalosis Anxiety

D is not correct. 23% chose this.

This profile represents mixed respiratory and metabolic alkalosis in which the pH is high due to decreased levels of CO_2 from increased respiratory rate as well as increased levels of HCO_3^-.

Metabolic alkalosis PH Respiratory rate Alkalosis Metabolism

Bottom Line:

Metabolic alkalosis, commonly due to excessive vomiting and hydrogen ion loss, is characterized by an increase in pH >7.4 and elevated plasma [HCO_3^-]. In response to the increased pH, the respiratory system compensates by decreasing ventilation and increasing the PCO_2.

Metabolic alkalosis Hydrogen ion PH Alkalosis Blood plasma Respiratory system Vomiting Ion Metabolism Hydrogen Ventilation (physiology)

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 561.2

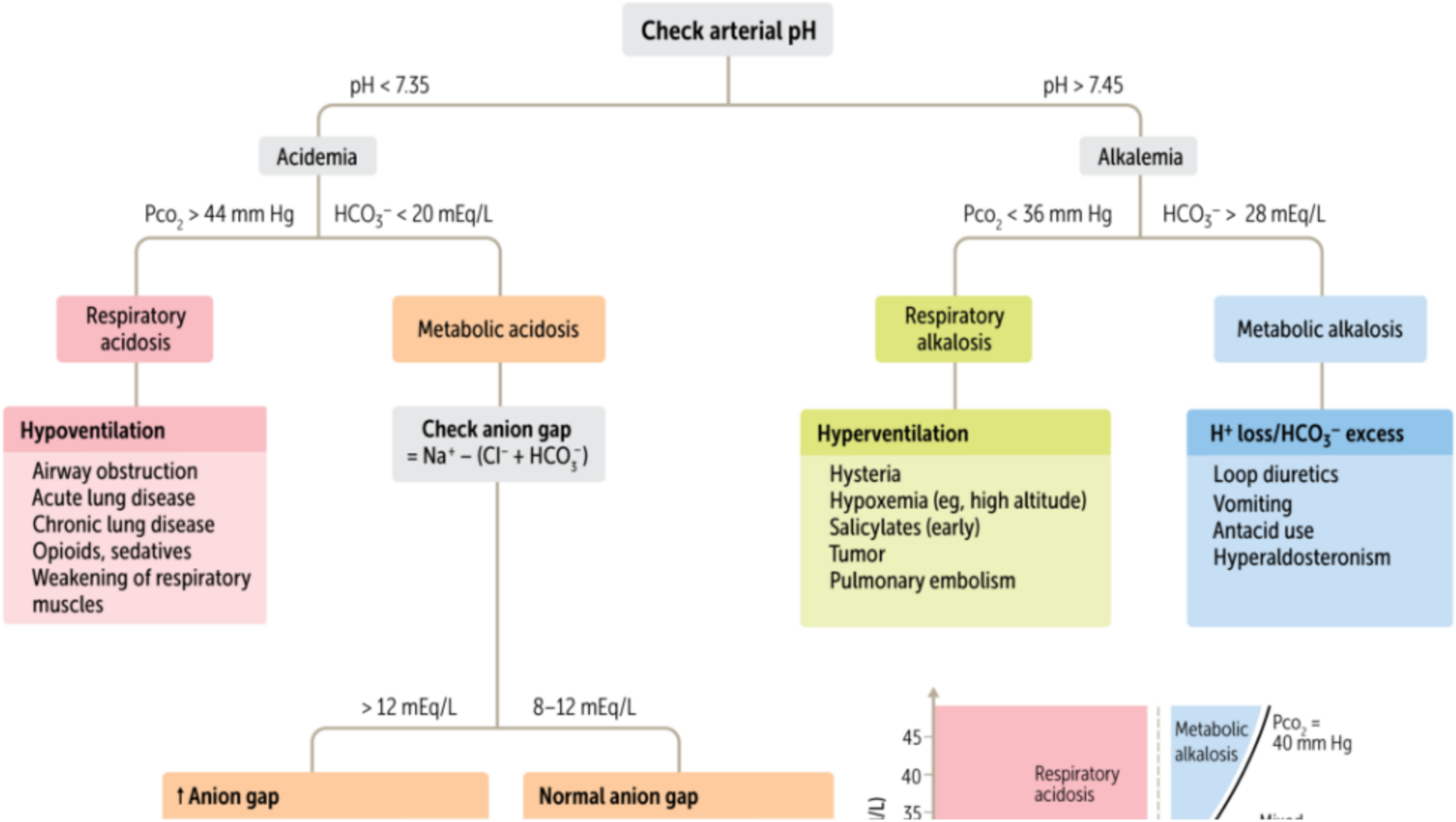
Acidosis and alkalosis

Check arterial pH

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

FA17 p 561.2

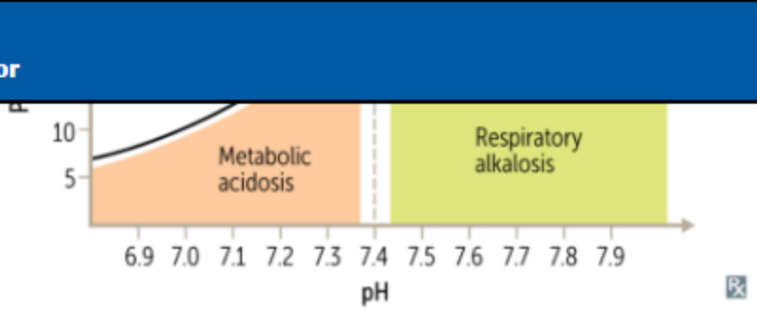
Acidosis and alkalosis



1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Lactic acidosis
Ethylene glycol (→→ oxalic acid)
Salicylates (late)

Spironolactone
Saline infusion



FA17 p 561.1

Acid-base physiology

	pH	Pco_2	$[HCO_3^-]$	COMPENSATORY RESPONSE
Metabolic acidosis	↓	↓	↓	Hyperventilation (immediate)
Metabolic alkalosis	↑	↑	↑	Hypoventilation (immediate)
Respiratory acidosis	↓	↑	↑	↑ renal $[HCO_3^-]$ reabsorption (delayed)
Respiratory alkalosis	↑	↓	↓	↓ renal $[HCO_3^-]$ reabsorption (delayed)

Key: ↑ ↓ = 1° disturbance; ↓ ↑ = compensatory response.

Henderson-Hasselbalch equation: $pH = 6.1 + \log \frac{[HCO_3^-]}{0.03\ Pco_2}$

Predicted respiratory compensation for a simple metabolic acidosis can be calculated using the Winters formula. If measured Pco_2 > predicted Pco_2 → concomitant respiratory acidosis; if measured Pco_2 < predicted Pco_2 → concomitant respiratory alkalosis:

$$Pco_2 = 1.5\ [HCO_3^-] + 8 \pm 2$$

1
2
3
4
5
6
7
8
▪ 9
▪ 10
▪ 11
▪ 12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

A 35-year-old man with decreased gastrointestinal motility is shown to have no slow waves on electric potential and force transducer recordings from gastrointestinal muscles over a period of 60 seconds.

Which of the following is the best description of slow waves in the gastrointestinal tract?

A. Contraction of skeletal muscle

B. Depolarization and repolarization of smooth muscle cells

C. Hyperpolarization of smooth muscle cells

D. Increase in secretions of smooth muscle cells

E. Relaxation of smooth muscle cells

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is B. 68% chose this.

Slow waves are precisely timed, rhythmic depolarizations and repolarizations of the muscularis propria of the stomach and intestines, independent of the presence or absence of stimulus. They move in an oral-to-anal direction. They occur in a frequency specific to each organ, with the stomach having the lowest frequency (3 cycles per minute) and the duodenum of the small intestine having the highest frequency (12 cycles per minute). They represent the basal electric rhythm of gastric and intestinal motility. Hormones and neurotransmitters released near the gastrointestinal smooth muscle cells can modulate the amplitude of the slow waves. Not all slow waves induce action potentials and stimulate smooth muscle contraction. Depending on the slow-wave amplitude and smooth muscle excitability, an action potential can be initiated if the hormones and neurotransmitters increase the slow-wave depolarization enough to reach threshold. Only when contractile events occur at the peak of cell depolarization during a slow wave does coordinated smooth muscle contraction occur in order to facilitate movement through the digestive tract.

Action potential Duodenum Muscular layer Depolarization Small intestine Neurotransmitter Muscle contraction Gastrointestinal tract Smooth muscle tissue Motility Hormone Stomach Human gastrointestinal tract Gastrointestinal physiology Muscle Slow-wave potential

A is not correct. 4% chose this.

When slow waves are accompanied by action potentials, smooth muscle contraction, not skeletal muscle contraction, occurs.

Skeletal muscle Muscle contraction Smooth muscle tissue Action potential Slow-wave potential Muscle

C is not correct. 11% chose this.

Slow waves cause depolarization (the potential becomes more positive, moving toward threshold), not hyperpolarization (the potential becomes more negative, moving further away from the threshold).

Hyperpolarization (biology) Depolarization Hyperpolarization (physics)

D is not correct. 4% chose this.

Slow waves do not play a role in secretions of smooth muscle cells.

Smooth muscle tissue Slow-wave potential Muscle

E is not correct. 13% chose this.

Relaxation of smooth muscle cells occurs after an action potential has been triggered to contract smooth muscles.

Action potential Smooth muscle tissue Muscle

Bottom Line:

Slow waves determine the frequency of contractions of the gastrointestinal tract. They are rhythmic depolarizations and repolarizations of the muscularis propria of the stomach and intestines and are independent of stimulus.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Relaxation of smooth muscle cells occurs after an action potential has been triggered to contract smooth muscles.
Action potential Smooth muscle tissue Muscle

Bottom Line:

Slow waves determine the frequency of contractions of the gastrointestinal tract. They are rhythmic depolarizations and repolarizations of the muscularis propria of the stomach and intestines and are independent of stimulus.
Human gastrointestinal tract Gastrointestinal tract Muscular layer Slow-wave potential Depolarization

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 347.1

Digestive tract anatomy

Layers of gut wall (inside to outside—MSMS):

- Mucosa—epithelium, lamina propria, muscularis mucosa
- Submucosa—includes Submucosal nerve plexus (Meissner), Secretes fluid
- Muscularis externa—includes Myenteric nerve plexus (Auerbach), Motility
- Serosa (when intraperitoneal), adventitia (when retroperitoneal)

Ulcers can extend into submucosa, inner or outer muscular layer. Erosions are in the mucosa only.

Frequencies of basal electric rhythm (slow waves):

- Stomach—3 waves/min
- Duodenum—12 waves/min
- Ileum—8–9 waves/min



1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

A 35-year-old woman with a history of rheumatoid arthritis is diagnosed with peptic ulcer disease. The doctor plans to prescribe her a medication that is known to be contraindicated in pregnancy. Before writing the prescription, he confirms that the patient is not pregnant and that she understands the risks were she to become pregnant while being treated.

What is the mechanism of action of the drug the doctor most likely prescribed?

A. Acts as a H_2-receptor agonist

B. Acts as a H_2-receptor antagonist

C. Acts as a prostaglandin agonist

D. Acts as a prostaglandin antagonist

E. Acts as a proton pump inhibitor

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is C. 55% chose this.

Misoprostol is the medication that was most likely prescribed. It is a prostaglandin E_1 analog and acts as an agonist at the prostaglandin receptors on parietal cells. This, in turn, results in increased mucus production and decreased acid secretion. Misoprostol may be used in patients with gastric ulcers induced by nonsteroidal anti-inflammatory (NSAID) use (this patient was likely taking chronic NSAIDs, given her history of rheumatoid arthritis) when the patient needs to continue NSAID use despite the gastrointestinal adverse effects. Another clinical use for misoprostol is in medically induced abortions, where it is used in combination with mifepristone, a progesterone antagonist.

Mifepristone Nonsteroidal anti-inflammatory drug Misoprostol Rheumatoid arthritis Prostaglandin Progesterone Peptic ulcer Receptor antagonist Agonist Mucus Anti-inflammatory Parietal cell Arthritis Pharmaceutical drug Structural analog Prostaglandin E Human gastrointestinal tract Gastrointestinal tract Receptor (biochemistry) Equine gastric ulcer syndrome

A is not correct. 3% chose this.

H_2-receptor agonists would not help reflux but in fact would worsen the symptoms.

Gastroesophageal reflux disease Reflux Agonist

B is not correct. 14% chose this.

H_2-receptor antagonists such as ranitidine, cimetidine, and famotidine inhibit acid secretion at the histamine receptor. Some H_2 blockers, such as cimetidine, cross the placenta, but are not explicitly contraindicated in pregnancy because they have been frequently used in pregnant patients without complications. However, ranitidine is preferred to cimetidine in pregnancy, as somes studies in animals and nonpregnant humans have raised concerns about feminization of the fetus with cimetidine use.

Cimetidine Famotidine Ranitidine Histamine Placenta Histamine receptor Secretion Contraindication Fetus Pregnancy

D is not correct. 17% chose this.

Misoprostol is a prostaglandin E_1 analog and acts as an agonist at prostaglandin receptors on parietal cells. It does not act as a prostaglandin antagonist.

Misoprostol Prostaglandin Agonist Parietal cell Receptor antagonist Structural analog Receptor (biochemistry) Prostaglandin E

E is not correct. 11% chose this.

Proton pump inhibitors (PPIs), such as omeprazole and lansoprazole, work to inhibit acid secretion. These drugs are a preferred treatment for peptic ulcer disease and may even be used to treat NSAID-induced ulcers. However, PPIs are not contraindicated in pregnancy.

Peptic ulcer Omeprazole Lansoprazole Proton-pump inhibitor Proton pump Proton Contraindication Pregnancy Secretion Ulcer

Bottom Line:

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

E is not correct. 11% chose this.

Proton pump inhibitors (PPIs), such as omeprazole and lansoprazole, work to inhibit acid secretion. These drugs are a preferred treatment for peptic ulcer disease and may even be used to treat NSAID-induced ulcers. However, PPIs are not contraindicated in pregnancy.

Peptic ulcer Omeprazole Lansoprazole Proton-pump inhibitor Proton pump Proton Contraindication Pregnancy Secretion Ulcer

Bottom Line:

In NSAID-induced peptic ulcer disease, misoprostol, a prostaglandin agonist, may be used. In these patients it inhibits acid secretion and protects the mucosa. Misoprostol is contraindicated in pregnant patients.

Peptic ulcer Misoprostol Prostaglandin Agonist Mucous membrane Contraindication

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 382.3

Misoprostol

MECHANISM	A PGE_1 analog. ↑ production and secretion of gastric mucous barrier, ↓ acid production.
CLINICAL USE	Prevention of NSAID-induced peptic ulcers (NSAIDs block PGE_1 production). Also used off-label for induction of labor (ripens cervix).
ADVERSE EFFECTS	Diarrhea. Contraindicated in women of childbearing potential (abortifacient).

FA17 p 381.1

Acid suppression therapy

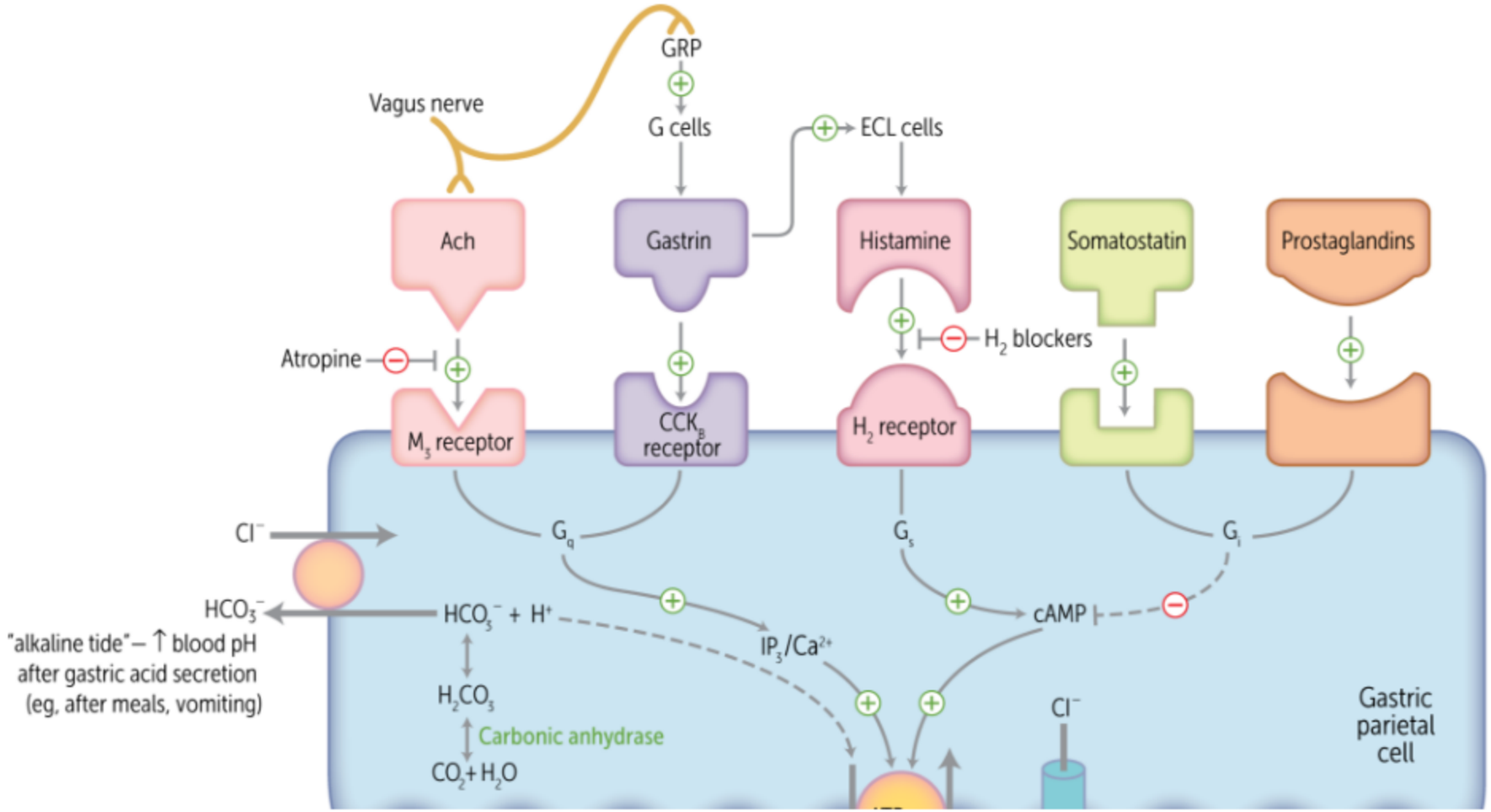
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

ADVERSE EFFECTS

Diarrhea. Contraindicated in women of childbearing potential (abortifacient).

FA17 p 381.1

Acid suppression therapy



Lock
Suspend
End Block

1
2
3
4
5
6
7
8
9
10
• 11
• 12
• 13
• 14
• 15
• 16
• 17
• 18

An overweight 37-year-old woman presents to her physician because of severe right upper quadrant abdominal pain. She has a low-grade fever. Laboratory tests show a serum total bilirubin level of 0.8 mg/dL, a serum aspartate aminotransferase level of 18 U/L, a serum alanine aminotransferase level of 20 U/L, and a leukocyte count of 15,000 cells/μL. Ultrasonography of the right upper quadrant is performed.

What is the ultrasound image likely to show?

A. Adhesions along the hepatic flexure of the large colon

B. Engorgement of the splenic vessels

C. Mass at the head of the pancreas

D. Obstruction of the common bile duct by a gallstone

E. Papillary sloughing of the right kidney

F. Wall thickening and inflammation of the gallbladder

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is F. 61% chose this.

This presentation is most consistent with acute cholecystitis, which usually presents in patients with the "**4 Fs**": **F**at, **F**emale, **F**ertile, and **F**orty years old. These patients usually have severe and steady right upper quadrant pain, low-grade fevers, leukocytosis, and slight elevations in transaminase levels. Elevations in bilirubin and alkaline phosphatase concentrations are not common in uncomplicated cholecystitis since biliary obstruction is limited to the gallbladder. The pain may radiate to the right upper shoulder or back. Murphy sign is considered present or positive if the patient has increased pain or suddenly halts inspiration due to pain (inspiratory arrest). Acute cholecystitis occurs when the cystic duct becomes acutely blocked by a gallstone, leading to irritation of the gallbladder mucosa and subsequent inflammation. Liver function tests are usually normal but may occasionally be slightly elevated. Senstitive findings include the presence of cholelithasis and a positive Murphy sign as confirmed by ultrasound. As shown in the image, signs of inflammation such as gallbladder wall thickening (>3 mm) are important secondary findings that confirm the diagnosis; the presence of gallstones supports the diagnosis, but is not diagnostic. Gallbladder wall distension and pericholecystic fluid are not highlighted in the image, but are also significant.

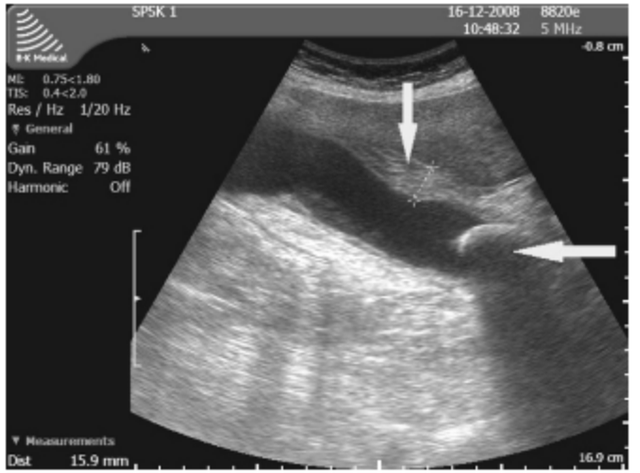


Image copyright © 2013 Cwik, et al.

It is important to remember that cholecystitis involves blockage of the cystic duct, whereas choledocolithiasis is an obstruction of the common bile duct (and would manifest with much more elevated liver function tests [LFTs]). Choledocolithiasis is especially dangerous because it often progresses to ascending cholangitis, which is an infection of the biliary tract.

Gallstone Cholecystitis Bilirubin Cystic duct Leukocytosis Alkaline phosphatase Gallbladder Liver function tests Common bile duct Ascending cholangitis

Quadrant (abdomen) Murphy's sign Liver Medical ultrasound Ultrasound Inflammation Bile Bile duct Biliary tract Mucous membrane Infection Transaminase

A is not correct. 5% chose this.

This would not be seen on ultrasound and does not correlate with the patient's clinical picture. Adhesions do not manifest with fever, hyperbilirubinemia, or elevations in liver function tests.

Liver function tests Medical ultrasound Hyperbilirubinemia Ultrasound Liver Adhesion (medicine) Fever

B is not correct. 2% chose this.

The spleen is located in the left upper quadrant of the abdomen, not the right, so it would not be seen on the ultrasound of this patient's right upper quadrant.

Spleen Medical ultrasound Quadrant (abdomen) Ultrasound Abdomen

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

This would not be seen on ultrasound and does not correlate with the patient's clinical picture. Adhesions do not manifest with fever, hyperbilirubinemia, or elevations in liver function tests.

Liver function tests Medical ultrasound Hyperbilirubinemia Ultrasound Liver Adhesion (medicine) Fever

B is not correct. 2% chose this.

The spleen is located in the left upper quadrant of the abdomen, not the right, so it would not be seen on the ultrasound of this patient's right upper quadrant.

Spleen Medical ultrasound Quadrant (abdomen) Ultrasound Abdomen

C is not correct. 3% chose this.

Although a mass at the head of the pancreas could be seen on right upper quadrant ultrasound, it does not fit the clinical picture of this patient, because an obstructive pancreatic head mass is more likely to result in painless jaundice. This patient has severe right upper quadrant pain more suggestive of gallbladder-related disease.

Jaundice Medical ultrasound Pancreas Quadrant (abdomen) Ultrasound Head of pancreas

D is not correct. 27% chose this.

Obstruction of the common bile duct by a gallstone is indicative of choledocolithiasis. If the common bile duct were to be obstructed, as in choledocolithiasis, one would see much higher serum bilirubin, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) as bile flows out of both the liver and gallbladder would be obstructed. Acute cholecystitis is a more likely diagnosis, in which the cystic duct is blocked, leading to inflammation and wall thickening in the gallbladder and only very mild elevations in liver function tests (LFTs), as is the case in this patient.

Gallstone Cholecystitis Bilirubin Cystic duct Alanine transaminase Aspartate transaminase Common bile duct Gallbladder Liver function tests Bile duct Bile Liver Blood plasma Inflammation Alanine Aspartic acid

E is not correct. 2% chose this.

Although the kidneys can be visualized with ultrasound, papillary sloughing would not be picked up by this imaging modality. Furthermore, this does not match the patient's clinical picture.

Ultrasound Medical ultrasound

Bottom Line:

In a patient with the "**4 Fs**" who presents with acute right upper quadrant pain in the abdomen and mildly elevated or normal LFTs, the most likely diagnosis is acute cholecystitis due to obstruction of the biliary duct system at the level of the cystic duct.

Bile duct Cholecystitis Cystic duct Quadrant (abdomen) Liver function tests Abdomen Bile

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Spleen Medical ultrasound Quadrant (abdomen) Ultrasound Abdomen

C is not correct. 3% chose this.

Although a mass at the head of the pancreas could be seen on right upper quadrant ultrasound, it does not fit the clinical picture of this patient, because an obstructive pancreatic head mass is more likely to result in painless jaundice. This patient has severe right upper quadrant pain more suggestive of gallbladder-related disease.

Jaundice Medical ultrasound Pancreas Quadrant (abdomen) Ultrasound Head of pancreas

D is not correct. 27% chose this.

Obstruction of the common bile duct by a gallstone is indicative of choledocolithiasis. If the common bile duct were to be obstructed, as in choledocolithiasis, one would see much higher serum bilirubin, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) as bile flows out of both the liver and gallbladder would be obstructed. Acute cholecystitis is a more likely diagnosis, in which the cystic duct is blocked, leading to inflammation and wall thickening in the gallbladder and only very mild elevations in liver function tests (LFTs), as is the case in this patient.

Gallstone Cholecystitis Bilirubin Cystic duct Alanine transaminase Aspartate transaminase Common bile duct Gallbladder Liver function tests Bile duct Bile Liver Blood plasma Inflammation Alanine Aspartic acid

E is not correct. 2% chose this.

Although the kidneys can be visualized with ultrasound, papillary sloughing would not be picked up by this imaging modality. Furthermore, this does not match the patient's clinical picture.

Ultrasound Medical ultrasound

Bottom Line:

In a patient with the "**4 Fs**" who presents with acute right upper quadrant pain in the abdomen and mildly elevated or normal LFTs, the most likely diagnosis is acute cholecystitis due to obstruction of the biliary duct system at the level of the cystic duct.

Bile duct Cholecystitis Cystic duct Quadrant (abdomen) Liver function tests Abdomen Bile

FIRST AID FACTS for year: 2017

1
2
3
4
5
6
7
8
9
10
11
▪ 12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

As a student, you are working in a laboratory over the summer that focuses on the biochemistry of the gastrointestinal (GI) tract. Your mentor wants to study the neural signaling processes and second-messenger pathways involved in the secretions of digestive enzymes by the salivary glands in response to food. In preparation, you review your first-year biochemistry notes on the role of digestive enzymes in the tract. You visualize a scenario where you consume a meal of mashed potatoes and you reflect on how the body would function in breaking down this meal for its nutrient value.

Which of the following is involved in the initial digestion of this macronutrient?

A. α-Amylase

B. Bile salts

C. Enterokinase

D. Gastric acid

E. Pepsin

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is A. 91% chose this.

α-Amylase (ptyalin) is the first enzyme involved in starch digestion, the dominant macronutrient of potatoes. This substance typically is secreted by the pancreas and the salivary glands.

Nutrient Enzyme Ptyalin Pancreas Digestion Starch Salivary gland

B is not correct. 1% chose this.

The liver produces bile salts, which the gallbladder stores and secretes. Bile salts are not involved in starch digestion but are critical for the digestion of fatty foods because they help emulsify lipids.

Gallbladder Bile acid Bile Liver Starch Lipid Emulsion Digestion Salt (chemistry)

C is not correct. 2% chose this.

The duodenum produces enterokinase, which converts trypsinogen, a pancreatic proenzyme, into its activated for trypsin. Trypsin is then able to digest proteins by cleaving peptide chains. Neither enterokinase nor trypsin is involved in starch digestion.

Zymogen Trypsinogen Duodenum Enteropeptidase Trypsin Digestion Peptide Starch

D is not correct. 3% chose this.

Parietal cells of the stomach secrete gastric acid and intrinsic factor; this decreases stomach pH and helps with vitamin B_{12} binding, respectively. The stomach mostly aids in the digestion of proteins. It is not involved in starch digestion.

Intrinsic factor Gastric acid Vitamin B12 PH B vitamins Parietal cell Digestion Starch Vitamin Protein Stomach

E is not correct. 3% chose this.

Chief cells of the stomach secrete pepsin, which aids in the digestion of protein, not starch. Potatoes are a starch-heavy food.

Pepsin Protein Chief cell Gastric chief cell Starch Parathyroid chief cell Digestion Stomach

Bottom Line:

Salivary glands secrete α-amylase (ptyalin), which begins starch digestion.

Ptyalin Salivary gland Digestion Starch

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Bottom Line:

Salivary glands secrete α-amylase (ptyalin), which begins starch digestion.
Ptyalin Salivary gland Digestion Starch

FIRST AID FACTS for year: 2017

FA17 p 358.1

Pancreatic secretions — Isotonic fluid; low flow → high Cl^-, high flow → high HCO_3^-.

ENZYME	ROLE	NOTES
α-amylase	Starch digestion	Secreted in active form
Lipases	Fat digestion	
Proteases	Protein digestion	Includes trypsin, chymotrypsin, elastase, carboxypeptidases Secreted as proenzymes also known as zymogens
Trypsinogen	Converted to active enzyme trypsin → activation of other proenzymes and cleaving of additional trypsinogen molecules into active trypsin (positive feedback loop)	Converted to trypsin by enterokinase/enteropeptidase, a brush-border enzyme on duodenal and jejunal mucosa

1
2
3
4
5
6
7
8
9
10
11
12
▪ 13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

A 43-year-old overweight woman presents to her doctor's office because of right upper quadrant abdominal pain. She has experienced similar episodes of this type of pain in the past and admits that it is worse after meals.

Increased secretion of which of the following is responsible for this patient's postprandial pain?

A. Cholecystokinin

B. Gastrin

C. Pepsin

D. Somatostatin

E. Vasoactive intestinal peptide

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is A. 71% chose this.

This is a classic presentation of cholelithiasis, or gallstones. Patients with cholelithiasis experience pain after meals as a result of the duodenal release of cholecystokinin (CCK), which causes the gallbladder to contract while the stone obstructs the cystic duct. CCK is stimulated by fatty acids and amino acids.

Gallstone Cholecystokinin Cystic duct Gallbladder Duodenum Fatty acid Amino acid

B is not correct. 19% chose this.

Gastrin is released by the G cells of the stomach in response to proteins or peptides in the stomach. Gastrin leads to increased secretion of gastric acid and low pH inhibits its secretion, leading to a negative feedback loop. However, in Zollinger-Ellison Syndrome (ZES), which is caused by a gastrin-secreting tumor in the pancreas, there is excessive gastric acid secretion in the stomach; these patients generally present with more severe symptoms such as pain in the esophagus, steatorrhea, wheezing or hematemesis. Gastrin does not have an effect on gallbladder contraction.

Hematemesis Gastrin Steatorrhea Esophagus Gallbladder Gastric acid Pancreas PH G cell Peptide Neoplasm Negative feedback Protein Secretion Stomach

C is not correct. 5% chose this.

Pepsin is a digestive protease released by chief cells in the stomach. Its precursor, pepsinogen, autocleaves in the acidic environment of the stomach. It is released under the influence gastrin and the influence of the vagus nerve. It does not cause gallbladder contraction.

Pepsin Gastrin Pepsinogen Vagus nerve Gallbladder Gastric chief cell Protease Chief cell Parathyroid chief cell Stomach

D is not correct. 3% chose this.

Somatostatin is released by the D cells of the duodenum, pyloric antrum, and pancreatic islets. It reduces smooth muscle contractions and inhibits the release of both insulin and glucagon from the pancreas. It does not cause gallbladder contraction.

Pyloric antrum Somatostatin Glucagon Duodenum Gallbladder Pancreas Pancreatic islets Delta cell Insulin Smooth muscle tissue Antrum Pylorus Muscle

E is not correct. 2% chose this.

Vasoactive intestinal peptide induces smooth muscle relaxation in the lower esophageal sphincter, stomach, gallbladder and stimulates secretion of water into pancreatic juice and bile. It also inhibits gastric acid secretion and absorption from the intestinal lumen. It causes relaxation rather than contraction of the gallbladder.

Pancreatic juice Vasoactive intestinal peptide Lower esophageal sphincter Gallbladder Gastric acid Lumen (anatomy) Gastrointestinal tract Bile

Smooth muscle tissue Human gastrointestinal tract Peptide Cardia Sphincter Muscle Secretion Stomach

Bottom Line:

Patients with gallstones experience pain after meals as a result of the duodenal release of cholecystokinin, which causes the gallbladder to contract while the stone obstructs the cystic duct.

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

Pancreatic juice Vasoactive intestinal peptide Lower esophageal sphincter Gallbladder Gastric acid Lumen (anatomy) Gastrointestinal tract Bile
Smooth muscle tissue Human gastrointestinal tract Peptide Cardia Sphincter Muscle Secretion Stomach

Bottom Line:

Patients with gallstones experience pain after meals as a result of the duodenal release of cholecystokinin, which causes the gallbladder to contract while the stone obstructs the cystic duct.
Cholecystokinin Cystic duct Gallbladder Duodenum Gallstone

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 356.1

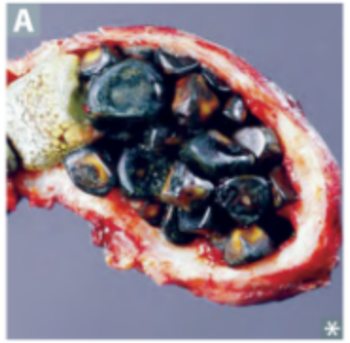
Gastrointestinal regulatory substances

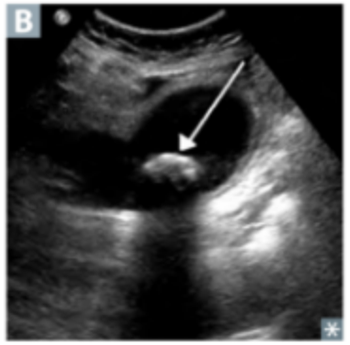
REGULATORY SUBSTANCE	SOURCE	ACTION	REGULATION	NOTES
Gastrin	G cells (antrum of stomach, duodenum)	↑ gastric H^+ secretion ↑ growth of gastric mucosa ↑ gastric motility	↑ by stomach distention/ alkalinization, amino acids, peptides, vagal stimulation via gastrin-releasing peptide (GRP) ↓ by pH < 1.5	↑ by chronic PPI use. ↑ in chronic atrophic gastritis (eg, *H pylori*). ↑↑ in Zollinger-Ellison syndrome (gastrinoma).
Somatostatin	D cells	↓ gastric acid and	↑ by acid	Inhibits secretion of various

		sphincter (LES)		
Ghrelin	Stomach	↑ appetite	↑ in fasting state ↓ by food	↑ in Prader-Willi syndrome. ↓ after gastric bypass surgery.

FA17 p 379.1

Gallstones (cholelithiasis)





↑ cholesterol and/or bilirubin, ↓ bile salts, and gallbladder stasis all cause stones.

2 types of stones:

- Cholesterol stones (radiolucent with 10–20% opaque due to calcifications)—80% of stones. Associated with obesity, Crohn disease, advanced age, estrogen therapy, multiparity, rapid weight loss, Native American origin.
- Pigment stones A (black = radiopaque, Ca^{2+} bilirubinate, hemolysis; brown = radiolucent, infection). Associated with Crohn disease, chronic hemolysis, alcoholic cirrhosis, advanced age, biliary infections, total parenteral nutrition (TPN).

Most common complication is cholecystitis; can also cause acute pancreatitis, ascending cholangitis.

Risk factors (4 F's):

1. Female
2. Fat
3. Fertile (pregnant)
4. Forty

Diagnose with ultrasound B. Treat with elective cholecystectomy if symptomatic.

Can cause fistula between gallbladder and GI tract → air in biliary tree (pneumobilia) → passage of gallstones into intestinal tract → obstruction of ileocecal valve (gallstone ileus).

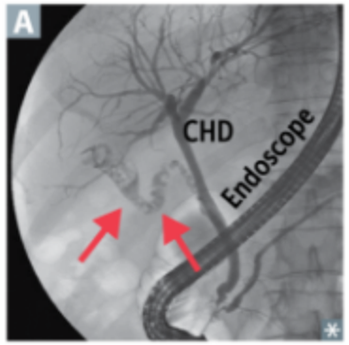
RELATED PATHOLOGIES	CHARACTERISTICS
Biliary colic	Associated with nausea/vomiting and dull RUQ pain. Neurohormonal activation (eg, by CCK after

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

- Shock (hypotension)

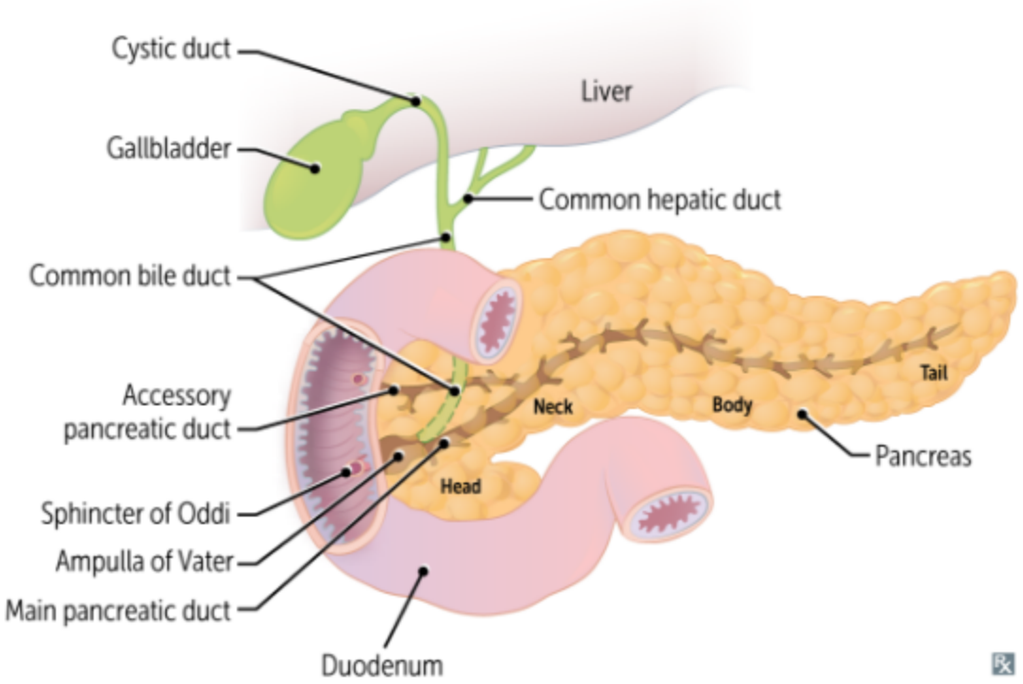
FA17 p 353.1

Biliary structures



Gallstones (filling defects in gallbladder and cystic duct, red arrows in A) that reach the confluence of the common bile and pancreatic ducts at the ampulla of Vater can block both the common bile and pancreatic ducts (double duct sign), causing both cholangitis and pancreatitis, respectively.

Tumors that arise in head of pancreas (usually ductal adenocarcinoma) can cause obstruction of common bile duct → enlarged gallbladder with painless jaundice (Courvoisier sign).



1
2
3
4
5
6
7
8
9
10
11
12
13
▪ 14
▪ 15
▪ 16
▪ 17
▪ 18

A variety of enzymes from the pancreas and small intestine are important to digestion and must undergo activation to become functional in the duodenum.

The loss of which enzyme would be most detrimental to the activation of other enzymes important for the digestive process?

A. Amylase

B. Chymotrypsinogen

C. Enterokinase

D. Lipase

E. Pepsin

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is C. 53% chose this.

Loss of enterokinase would prove to be most detrimental. Enterokinase is embedded in the intestinal mucosa and is responsible for activating trypsinogen into trypsin. The active trypsin then activates the rest of the pancreatic enzymes. Although enterokinase is not a pancreatic enzyme itself, it is most important for subsequent activation of enzymes from the pancreatic exocrine organ.

Trypsinogen Trypsin Enzyme Enteropeptidase Pancreas Exocrine gland Intestinal mucosa Digestive enzyme Mucous membrane

A is not correct. 4% chose this.

The loss of amylase from the pancreas would not be too detrimental, especially because it is also made in the salivary glands.

Amylase Pancreas Salivary gland

B is not correct. 27% chose this.

Chymotrypsinogen can be activated to chymotrypsin by trypsin. However, this process depends on the activation of trypsinogen to trypsin by enterokinase in the first place, so it is not as crucial to the entire process.

Chymotrypsinogen Trypsinogen Chymotrypsin Trypsin Enteropeptidase

D is not correct. 4% chose this.

Lipase does not help activate other enzymes. Deficiency in lipase may cause diarrhea, but would not be as detrimental as the total loss of enterokinase.

Enteropeptidase Lipase Diarrhea Enzyme

E is not correct. 12% chose this.

Although the digestion of proteins is indeed important, without enterokinase they would not even have a chance to be activated. Pepsin does not play a role in the activation of the other pancreatic enzymes.

Pepsin Enteropeptidase Digestion Digestive enzyme Enzyme Protein

Bottom Line:

Enterokinase activates trypsinogen into trypsin, which in turn activates the pancreatic enzymes.

Trypsinogen Trypsin Enteropeptidase Digestive enzyme Enzyme

FIRST AID for year: 2017

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

Bottom Line:

Enterokinase activates trypsinogen into trypsin, which in turn activates the pancreatic enzymes.
Trypsinogen Trypsin Enteropeptidase Digestive enzyme Enzyme

FIRST AID FOR year: 2017
FIRST AID FACTS

FA17 p 358.1

Pancreatic secretions Isotonic fluid; low flow → high Cl^-, high flow → high HCO_3^-.

ENZYME	ROLE	NOTES
α-amylase	Starch digestion	Secreted in active form
Lipases	Fat digestion	
Proteases	Protein digestion	Includes trypsin, chymotrypsin, elastase, carboxypeptidases Secreted as proenzymes also known as zymogens
Trypsinogen	Converted to active enzyme trypsin → activation of other proenzymes and cleaving of additional trypsinogen molecules into active trypsin (positive feedback loop)	Converted to trypsin by enterokinase/enteropeptidase, a brush-border enzyme on duodenal and jejunal mucosa

1
2
3
4
5
6
7
8
9
10
11
12
13
14
▪ 15
▪ 16
▪ 17
▪ 18

A 65-year-old woman presents to her doctor because of weakness, light-headedness, peripheral neuropathy, a recent 4.5-kg (10-lb) weight loss, and diarrhea. She has a past medical history of hypothyroidism. A complete blood cell count is significant for a hemoglobin level of 7.5 g/dL, a hematocrit level of 28%, and a mean corpuscular volume of 115 fL (normal: 80-100 fL).

This patient is most likely deficient in a substance produced by which of the following types of cells?

A. Chief cell

B. G cell

C. I cell

D. Mucous cell

E. Parietal cell

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is E. 80% chose this.

The vignette describes symptoms of anemia with some gastrointestinal effects, which should make one suspicious of pernicious anemia. Anemia is confirmed by the low hematocrit and hemoglobin levels, and the elevated mean corpuscular volume indicates that this is a macrocytic anemia. A common cause of macrocytic anemia is vitamin B_{12} and/or folate deficiency. This patient is also experiencing neurologic symptoms, and while both vitamin B_{12} and folate deficiencies cause macrocytic anemia, only vitamin B_{12} deficiency causes neurologic deficits. One cause of vitamin B_{12} deficiency is pernicious anemia. Pernicious anemia is more common in the elderly, those of northern European descent, and African-Americans. It is an autoimmune condition that can cause decreased production of intrinsic factor (which is required for vitamin B_{12} absorption) or reduced function of ileal IF-B_{12} receptors. Pernicious anemia is common in patients with other autoimmune conditions such as Hashimoto's thyroiditis and vitiligo. Intrinsic factor is produced in parietal cells of the stomach. This condition is treated with regular intramuscular injections of cobalamin (vitamin B_{12}).

Pernicious anemia Vitiligo Hashimoto's thyroiditis Macrocytic anemia Mean corpuscular volume Hematocrit Hemoglobin Intrinsic factor Folic acid Folate deficiency Vitamin B12 Anemia Cobalamin B vitamins Parietal cell Autoimmunity Autoimmune disease Ileum Gastrointestinal tract Vitamin Stomach Intramuscular injection Macrocytosis Human gastrointestinal tract

A is not correct. 7% chose this.

Chief cells of the stomach typically secrete pepsinogens. In the setting of low gastric pH, pepsinogen is converted to pepsin, which cleaves peptide bonds and aids in protein digestion.

Pepsin Pepsinogen PH Chief cell Protein Peptide Gastric chief cell Parathyroid chief cell Digestion Proteolysis Stomach

B is not correct. 4% chose this.

G cells located in the stomach typically secrete gastrin, which serves to increase gastric acid secretion, growth of gastric mucosa, and gastric motility.

Gastrin Gastric acid G cell Gastric mucosa Mucous membrane Motility Stomach

C is not correct. 6% chose this.

I cells are located in the duodenum and jejunum. These cells release cholecystokinin, an enzyme that causes increased pancreatic secretion, increased gall bladder contraction, and decreased gastric emptying.

Jejunum Cholecystokinin Duodenum Enzyme Gallbladder Urinary bladder Stomach

D is not correct. 3% chose this.

Mucous cells located in the stomach typically secrete mucus to lubricate the stomach and bicarbonate to protect the surface from the acidic environment.

Mucus Bicarbonate Acid Stomach

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

Jejunum Cholecystokinin Duodenum Enzyme Gallbladder Urinary bladder Stomach

D is not correct. 3% chose this.

Mucous cells located in the stomach typically secrete mucus to lubricate the stomach and bicarbonate to protect the surface from the acidic environment.

Mucus Bicarbonate Acid Stomach

Bottom Line:

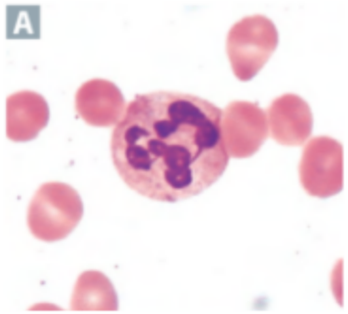
Pernicious anemia is an autoimmune condition that can cause decreased production of intrinsic factor by parietal cells in the stomach. Because intrinsic factor is required for the absorption of vitamin B_{12}, it can cause a macrocytic anemia.

Pernicious anemia Macrocytic anemia Intrinsic factor Vitamin B12 B vitamins Parietal cell Autoimmunity Macrocytosis Anemia Autoimmune disease Vitamin

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 398.1

Macrocytic (MCV > 100 fL) anemia

	DESCRIPTION	FINDINGS
Megaloblastic anemia A 	Impaired DNA synthesis → maturation of nucleus of precursor cells in bone marrow delayed relative to maturation of cytoplasm.	RBC macrocytosis, hypersegmented neutrophils A, glossitis.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

thumbs) in up to 50% of cases.

FA17 p 357.1

Gastrointestinal secretory products

PRODUCT	SOURCE	ACTION	REGULATION	NOTES
Intrinsic factor	Parietal cells (stomach)	Vitamin B_{12}–binding protein (required for B_{12} uptake in terminal ileum)		Autoimmune destruction of parietal cells → chronic gastritis and pernicious anemia.
Gastric acid	Parietal cells (stomach)	↓ stomach pH	↑ by histamine, ACh, gastrin ↓ by somatostatin, GIP, prostaglandin, secretin	
Pepsin	Chief cells (stomach)	Protein digestion	↑ by vagal stimulation, local acid	Pepsinogen (inactive) is converted to pepsin (active) in the presence of H^+.
Bicarbonate	Mucosal cells (stomach, duodenum, salivary glands, pancreas) and Brunner glands	Neutralizes acid	↑ by pancreatic and biliary secretion with secretin	Trapped in mucus that covers the gastric epithelium.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
▪ 16
▪ 17
▪ 18

A 53-year-old patient presents to the clinic because of lethargy, malaise, and peripheral edema. His pulse is 96/min. Although his abdomen is distended, the remainder of his body habitus appears cachectic. His chart reveals that he was recently involved in several motor vehicle collisions. Laboratory studies show:

WBC count: 12,000/mm³
Hemoglobin: 11 g/dL
Hematocrit: 35%
Aspartate aminotransferase: 120 U/L
Alanine aminotransferase: 50 U/L
Alkaline phosphatase: 80 U/L

What is the major physiologic mechanism for this patient's edema?

A. Constriction of arterioles

B. Decreased production of serum proteins

C. Increased permeability of capillaries

D. Increased permeability of glomeruli

E. Lymphatic blockage

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is B. 70% chose this.

The patient has a presentation that is consistent with alcoholic hepatitis. This is evidenced by a ratio of aspartate aminotransferase to alanine aminotransferase (AST:ALT ratio) >1.5 and a normal alkaline phosphatase level (elevation would be more suggestive of biliary obstruction). He also shows classic signs of metabolic encephalopathy, ascites, and edema. The mild anemia is the result of the suppressive effects of alcohol on the bone marrow. The major mechanism contributing to edema in this disease is the decreased production of serum proteins (eg, albumin) by the damaged liver. Therefore, the plasma oncotic pressure is decreased, allowing fluid to flow out of the capillaries.

Alcoholic hepatitis Aspartate transaminase Oncotic pressure Ascites Alanine transaminase Alkaline phosphatase Human serum albumin Edema Albumin Anemia Blood plasma Liver Hepatitis Capillary Alkalinity Bone marrow Alanine Aspartic acid Encephalopathy Alcoholism Serum (blood) Bile Alcohol Protein Metabolism Transaminase Bone

A is not correct. 5% chose this.

Arteriolar constriction would lead to decreased hydrostatic pressure in the capillaries downstream, resulting in decreased filtration of fluid into the interstitium. The constriction of venules, however, would be more likely to increase capillary hydrostatic pressure.

Hydrostatic pressure Capillary Arteriole Interstitial fluid Venule Starling equation Filtration Hydrostatics

C is not correct. 14% chose this.

Capillary permeability is usually caused by inflammation, and this may lead to edema. However, there is minimal inflammation in the peripheral vasculature in this case, as evidenced by the WBC count.

Vascular permeability Edema Capillary Inflammation Circulatory system Semipermeable membrane

D is not correct. 4% chose this.

The permeability of renal glomeruli is increased in diseases such as nephrotic syndrome, leading to the protein loss in the blood and a subsequent decrease in oncotic pressure. However, the kidneys are not the source of the problem in this case.

Nephrotic syndrome Glomerulus (kidney) Oncotic pressure Glomerulus Protein Semipermeable membrane Kidney

E is not correct. 7% chose this.

Lymphatic obstruction can cause edema, but it is not the underlying problem in this case.

Edema Lymphatic system Lymph Lymphangiectasia

Bottom Line:

Nephrotic syndrome Glomerulus (kidney) Oncotic pressure Glomerulus Protein Semipermeable membrane Kidney

E is not correct. 7% chose this.

Lymphatic obstruction can cause edema, but it is not the underlying problem in this case.

Edema Lymphatic system Lymph Lymphangiectasia

Bottom Line:

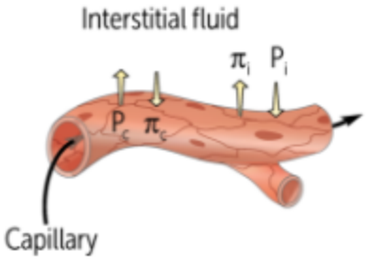
A decrease in oncotic pressure (as in liver failure or nephrotic syndrome), an increase in hydrostatic pressure (as in congestive heart failure), or a lymphatic obstruction can lead to edema. When evaluating a patient with edema, approach the patient in a systematic fashion to avoid missing the underlying problem.

Nephrotic syndrome Oncotic pressure Heart failure Congestive heart failure Edema Hydrostatic pressure Liver Liver failure Lymphangiectasia Lymphatic system Lymph

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 287.1

Capillary fluid exchange



Starling forces determine fluid movement through capillary membranes:

- P_c = capillary pressure—pushes fluid out of capillary
- P_i = interstitial fluid pressure—pushes fluid into capillary
- π_c = plasma colloid osmotic (oncotic) pressure—pulls fluid into capillary
- π_i = interstitial fluid colloid osmotic pressure—pulls fluid out of capillary

J_v = net fluid flow = $K_f [(P_c - P_i) - \varsigma(\pi_c - \pi_i)]$

K_f = capillary permeability to fluid

ς = reflection coefficient (measure of capillary permeability to protein)

Edema—excess fluid outflow into interstitium commonly caused by:

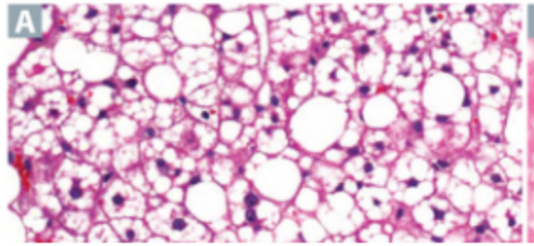
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

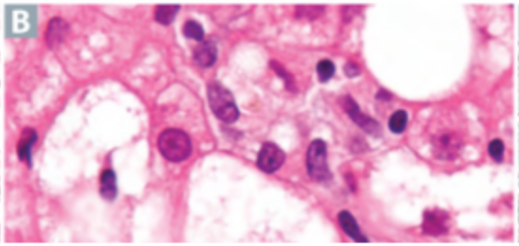
- ↑ capillary permeability (↑ K_f; eg, toxins, infections, burns)
- ↑ interstitial fluid colloid osmotic pressure (↑ π_i; eg, lymphatic blockage)

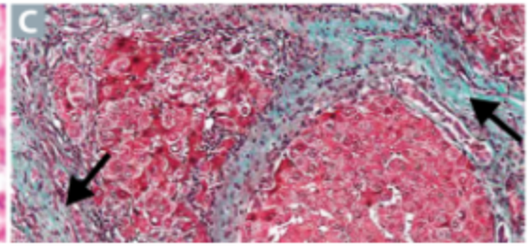
FA17 p 374.1

Alcoholic liver disease

Hepatic steatosis	Macrovesicular fatty change A that may be reversible with alcohol cessation.	
Alcoholic hepatitis	Requires sustained, long-term consumption. Swollen and necrotic hepatocytes with neutrophilic infiltration. Mallory bodies B (intracytoplasmic eosinophilic inclusions of damaged keratin filaments).	Make a toAST with alcohol: AST > ALT (ratio usually > 2:1).
Alcoholic cirrhosis	Final and usually irreversible form. Regenerative nodules surrounded by fibrous bands in response to chronic liver injury → portal hypertension and end-stage liver disease. Sclerosis around central vein (arrows in C) may be seen in early disease.	







1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
• 17
• 18

A 15-year-old boy presents to his pediatrician complaining that the right side of face and neck are swollen, just behind the angle of the jaw. Upon further questioning he also says he has produced more saliva lately than usual.

Compared with normal saliva, which of the following would be lower in concentration in this patient's saliva?

A. Amylase

B. Bicarbonate

C. Chloride

D. Potassium

E. Sodium

The correct answer is D. 34% chose this.

This vignette describes a boy with parotitis, which is classically caused by mumps (Typical presentation shown in the image. The incidence of this infection has decreased significantly since the advent of the MMR vaccine in 1967), but has a large range of causes, including autoimmune, bacterial, and idiopathic sources. In general, saliva secreted from duct acini starts out isotonic to plasma. As saliva travels, the excretory ducts and the intercalated ducts reabsorb Na^+ and Cl^- and secrete K^+ and HCO_3^-. At higher rates of flow, such as can be seen in the setting of parotitis, there is less time that saliva is in contact with the ductal epithelium, and less reabsorption of Na^+ and Cl^- and less secretion of K^+. As a result, salivary K^+ concentration decreases as flow rate increases. However, bicarbonate secretion is increased independently by the action of secretagogues. In addition, at lower salivary flows, saliva in the mouth tends to be hypotonic and slightly acidic compared with plasma. At higher flows, saliva is nearly isotonic to plasma and becomes more basic.

Parotitis Mumps Blood plasma Epithelium Autoimmune disease Vaccine MMR vaccine Excretion Idiopathy Saliva Bicarbonate Infection Acinus Autoimmunity Tonicity



Image courtesy of CDC/Barbar Rice

A is not correct. 20% chose this.

The concentration of amylase generally increases with the salivary flow rate.

Amylase

B is not correct. 18% chose this.

Bicarbonate (HCO_3^-) increases the pH and buffer capacity of saliva, especially during stimulation, in which secretagogues promote HCO_3^- secretion. The HCO_3^- concentration generally increases with salivary flow, resulting in a higher pH.

PH Bicarbonate Saliva Buffer solution

C is not correct. 16% chose this.

At higher rates of flow, there is less time that saliva is in contact with the ductal epithelium, which normally reabsorbs sodium chloride. Salivary chloride concentration increases as salivary flow rate increases.

Sodium chloride Sodium Epithelium Saliva

E is not correct. 12% chose this.

At higher rates of flow, there is less time that saliva is in contact with the ductal epithelium, which normally reabsorbs sodium chloride. Salivary sodium concentration increases as salivary flow rate increases

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

C is not correct. 16% chose this.

At higher rates of flow, there is less time that saliva is in contact with the ductal epithelium, which normally reabsorbs sodium chloride. Salivary chloride concentration increases as salivary flow rate increases.

Sodium chloride Sodium Epithelium Saliva

E is not correct. 12% chose this.

At higher rates of flow, there is less time that saliva is in contact with the ductal epithelium, which normally reabsorbs sodium chloride. Salivary sodium concentration increases as salivary flow rate increases.

Sodium chloride Sodium Epithelium Saliva

Bottom Line:

Saliva is isotonic at high flow rates and hypotonic at low flow rates as the ductal epithelium has more time to modify the secretion by reabsorbing sodium and chloride and secreting potassium. Bicarbonate secretion increases with salivary flow.

Saliva Tonicity Bicarbonate Sodium Epithelium Potassium Isotonicity Hypotonia Secretion

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 166.3

Mumps virus



A paramyxovirus that causes mumps, uncommon due to effectiveness of MMR vaccine.

Symptoms: Parotitis A, Orchitis (inflammation of testes), aseptic Meningitis, and Pancreatitis. Can cause sterility (especially after puberty).

Mumps makes your parotid glands and testes as big as POM-Poms.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
• 18

A 22-year-old woman presents with dyspnea upon exertion, weakness, and fatigue. On physical examination, she has hair loss, spoon-shaped fingernails, and pale mucous membranes. She has a history of heavy menstrual bleeding. Lab results reveal:

RBC count: 2 million/mm³
Mean corpuscular volume: 65 fL (normal: 80–100 fL)
Mean corpuscular hemoglobin: 24.0 pg (normal: 25.4–34.6 pg)
Ferritin: 6 ng/mL (normal: 12–150 ng/mL)
Red cell distribution width: 17% (normal: 11.5–14.5%).

Malabsorption in what part of the gastrointestinal tract will produce the type of anemia described in the stem?

A. Duodenum
B. Gastric fundus
C. Ileum
D. Jejunum
E. Sigmoid colon

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

The correct answer is A. 60% chose this.

One of the causes of microcytic anemia is iron deficiency. Iron is absorbed in the duodenum via two independent mechanisms. Heme-associated iron is taken up by a heme transporter in the luminal plasma membrane of the duodenal epithelial cell. Free ferric iron ions (Fe^{3+}) are converted to ferrous iron ions (Fe^{2+}) by a cytochrome B enzyme on the luminal plasma membrane of the duodenal epithelial cell. Ferrous iron ions are taken up by the divalent metal transporter channel, also in the luminal plasma membrane. Within the cell, iron is transferred to mucosal ferritin and then shuttled to transferrin in the plasma. Although some iron absorption may occur in the upper jejunum, most of it occurs in the duodenum. Thus, a deficiency in duodenal absorption will cause iron deficiency, which is the most common cause of microcytic anemia. Iron deficiency may lead to decreased mean corpuscular hemoglobin (MCH), which is the average amount of hemoglobin per red blood cell in a blood sample. Other common lab findings in iron deficiency anemia are an increased red cell distribution and low ferritin. See the table for further information on differentiating types of anemia.

	Iron Deficiency Anemia	Anemia of Chronic Disease	α-Thalassemia minor	β-Thalassemia minor
MCV	↓	↓ or Normal	↓	↓
RDW	↑	Normal	Normal	Normal
Serum Fe	↓	↓	Normal	Normal
TIBC	↑	↓	Normal	Normal
Ferritin	↓	↑ or Normal	Normal	Normal
Hemoglobin electrophoresis	Normal	Normal	Normal	↑HbA_2

Red blood cell Microcytic anemia Jejunum Ferritin Duodenum Iron-deficiency anemia Heme Transferrin Hemoglobin Enzyme Epithelium Anemia Iron

Iron deficiency Blood plasma Cell membrane Lumen (anatomy) Ferric Biological membrane Mean corpuscular hemoglobin Divalent Blood cell Iron(II) oxide

Cytochrome Ferrous

B is not correct. 5% chose this.

The gastric fundus contains parietal cells that secrete intrinsic factor, which is important in the absorption of vitamin B_{12}. No major nutrient absorption occurs in the gastric fundus. However, a deficiency in intrinsic factor will lead to malabsorption of vitamin B_{12} in the terminal ileum, which may lead to megaloblastic anemia from vitamin B_{12} deficiency.

Megaloblastic anemia Ileum Intrinsic factor Vitamin B12 Anemia Malabsorption B vitamins Parietal cell Gastric fundus Nutrient Vitamin Fundus (stomach)

C is not correct. 17% chose this.

The ileum is the major site of absorption of vitamin B_{12}, the fat-soluble vitamins (A, D, E, and K), fatty acids, and carbohydrates. Vitamin B_{12} deficiency causes megaloblastic, not microcytic, anemia.

Ileum Anemia B vitamins Vitamin B12 Carbohydrate Fatty acid Vitamin Microcytic anemia Lipophilicity

D is not correct. 16% chose this.

The jejunum is a major site of folate absorption. Decreased folate levels is another cause of megaloblastic anemia, in addition to vitamin B_{12} deficiency. To differentiate between folate and vitamin B_{12} deficiency, methylmalonic acid levels can be drawn, which will be elevated in vitamin B_{12} deficiency but normal in folate deficiency.

Megaloblastic anemia Jejunum Methylmalonic acid Folic acid Folate deficiency Vitamin B12 Anemia B vitamins Vitamin

causes megaloblastic, not microcytic, anemia.

Ileum Anemia B vitamins Vitamin B12 Carbohydrate Fatty acid Vitamin Microcytic anemia Lipophilicity

D is not correct. 16% chose this.

The jejunum is a major site of folate absorption. Decreased folate levels is another cause of megaloblastic anemia, in addition to vitamin B_{12} deficiency. To differentiate between folate and vitamin B_{12} deficiency, methylmalonic acid levels can be drawn, which will be elevated in vitamin B_{12} deficiency but normal in folate deficiency.

Megaloblastic anemia Jejunum Methylmalonic acid Folic acid Folate deficiency Vitamin B12 Anemia B vitamins Vitamin

E is not correct. 2% chose this.

The sigmoid colon is a site of water absorption. Malabsorption in the sigmoid colon will not affect RBC production.

Sigmoid colon Malabsorption Colon (anatomy)

Bottom Line:

Iron and calcium are predominately absorbed in the duodenum, and malabsorption in this segment of the gastrointestinal tract can lead to microcytic anemia.

Microcytic anemia Duodenum Anemia Malabsorption Human gastrointestinal tract Gastrointestinal tract Calcium Iron

FIRST AID FIRST AID FACTS for year: 2017

FA17 p 396.2

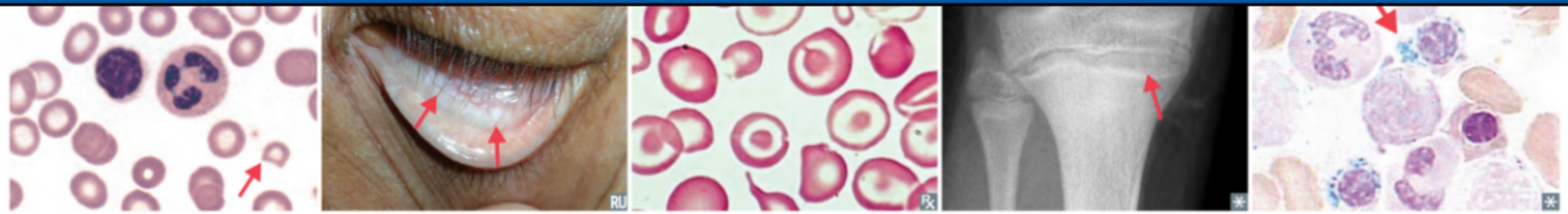
Microcytic (MCV < 80 fL), hypochromic anemia

Iron deficiency

↓ iron due to chronic bleeding (eg, GI loss, menorrhagia), malnutrition, absorption disorders, GI surgery (eg, gastrectomy), or ↑ demand (eg, pregnancy) → ↓ final step in heme synthesis.

Labs: ↓ iron, ↑ TIBC, ↓ ferritin, ↑ free erythrocyte protoporphyrin. Microcytosis and hypochromasia (central pallor)

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18



FA17 p 358.3

Vitamin/mineral absorption

Iron	Absorbed as Fe^{2+} in duodenum.	Iron Fist, Bro
Folate	Absorbed in small bowel.	Clinically relevant in patients with small bowel disease or after resection.
B_{12}	Absorbed in terminal ileum along with bile salts, requires intrinsic factor.	

FA17 p 358.5

Bile

Composed of bile salts (bile acids conjugated to glycine or taurine, making them water soluble), phospholipids, cholesterol, bilirubin, water, and ions. Cholesterol 7α-hydroxylase catalyzes rate-limiting step of bile acid synthesis.

Functions:

- Digestion and absorption of lipids and fat-soluble vitamins
- Cholesterol excretion (body's 1° means of eliminating cholesterol)
- Antimicrobial activity (via membrane disruption)