DDSEP Chapter 1: Question 1

Which of the following would be expected to be associated with a decrease in gastric acid secretion?

A. Chronic gastric outlet obstruction  
B. Systemic mastocytosis  
C. Misoprostol  
D. Ingestion of proteins  
E. Increased intracranial pressure

The recommended response is C.

In the physiology of gastric acid secretion, parietal cell function is stimulated by histamine, acetylcholine, and gastrin and is inhibited by prostaglandins and somatostatin. Acetylcholine is released from nerve endings as a result of vagal nerve stimulation. In conditions of increased intracranial pressure, there is increased vagal activity. Systemic mastocytosis is associated with increased systemic release of histamine and is associated with gastric acid hypersecretion. Misoprostol is a synthetic, prostaglandin PGE₂ analog. Gastric acid secretion is stimulated by the presence of food in the stomach, especially proteins. Gastric distention, which can occur with chronic gastric outlet obstructions, stimulates modest levels of gastric acid secretion through gastrin release via neural reflex pathways.
A 65-year-old woman has a prior history of hospitalization for UGI bleeding from a duodenal ulcer. Which one of the following therapies is not useful for preventing recurrent ulcer hemorrhage?

A. Long-term maintenance therapy with full dose H₂RA or daily PPI
B. H. pylori eradication
C. Discontinuation of NSAID intake
D. Ulcer surgery
E. Bland diet

The recommended response is E.

One study showed that in a patient with a prior documented ulcer hemorrhage, there was a 36% incidence of rebleeding during a follow-up period of about 61 weeks if the patient was not taking maintenance medical therapy. Recurrent ulcer hemorrhage can be decreased by using maintenance full dose H₂RA (i.e., ranitidine 300 mg or famotidine 40 mg hs) or daily PPIs. H. pylori eradication has also been shown to prevent recurrent ulcer hemorrhage. Cessation of NSAID intake is also important in preventing recurrent hemorrhage. Ulcer surgery is required in fewer than 5% of patients with UGI bleeding if endoscopic coagulation is used in high-risk patients. Ulcer surgery for continued or recurrent hemorrhage is appropriate to prevent exsanguination and recurrent ulceration and hemorrhage. Dietary modifications have not been shown to decrease ulcer recurrence or hemorrhage.


Chapter 1: Training in Acid-Peptic Disease

DDSEP Chapter 5: Question 5

An important risk factor for peptic ulcer hemorrhage includes:

A. Gastric acid hypersecretion
B. Corticosteroid use
C. Cigarette smoking
D. Non-steroidal anti-inflammatory drug use
E. Ethanol consumption

The recommended response is D.

NSAID ingestion has been clearly linked to peptic ulcer bleeding (Choice D). In CURE studies aspirin or NSAID ingestion within two weeks of ulcer hemorrhage was identified as the main risk factor in 53% of duodenal ulcers and 61% gastric ulcer patients. Gastric acid hypersecretion is not essential for ulcer hemorrhage. No significant differences in secretory parameters such as basal, peak, or meal-stimulated acid output and parietal cell sensitivity, have been noted among ulcer patients with and without bleeding. Corticosteroid use alone is not associated with an increased frequency of ulcer hemorrhage. However, concomitant steroid use with NSAIDs increases the chance of UGI bleeding by 10-fold compared to NSAID use alone. Neither cigarette smoking nor ethanol consumption are associated with increased ulcer hemorrhage.

Chapter 2: Training in Biliary Tract Diseases and Pancreatic Disorders

DDSEP Chapter 1: Question 2

Which of the following conditions is most likely to be associated with decreased serum concentrations of Vitamin B$_{12}$?

A. Pancreatic exocrine insufficiency
B. A 10-day course of oral antibiotics
C. Excess secretion of intrinsic factor
D. Stimulation of parietal cell function
E. Multiple endocrine neoplasia type-I (MEN-I) syndrome

The recommended response is A.

Intrinsic factor, a glycoprotein whose primary role is the facilitation of cobalamin (Vitamin B$_{12}$) absorption, is secreted by the parietal cell under the same stimulatory conditions as is hydrochloric acid. Cobalamin, when liberated from food (protein) by acid and pepsin, initially combines preferentially with R proteins present in saliva. In the alkaline environment of the duodenum where R proteins are hydrolyzed by pancreatic enzymes, cobalamin preferentially binds to intrinsic factor. Failure to absorb cobalamin can occur with intrinsic factor deficiency, pancreatic exocrine insufficiency, small bowel bacterial overgrowth (usually in the presence of achlorhydria), or ileal disease. MEN-I is an autosomal dominant condition that includes hyperparathyroidism, gastrinoma (ZE syndrome) or other islet cell tumors, and pituitary tumors. Thus, the increased parietal cell secretion in ZE syndrome would be expected to be associated with increased intrinsic factor secretion.
Chapter 2: Training in Biliary Tract Diseases and Pancreatic Disorders

DDSEP Chapter 7: Question 1

Serum cholecystokinin levels increase during a meal and mediate pancreatic secretion. Which of the following is true about this cholecystokinin effect in humans?

a) It is mediated by cholecystokinin receptors on the pancreatic acinar cell
b) It is mediated by enhancing pancreatic blood flow
c) It can be largely blocked by the muscarinic antagonist, atropine
d) It can be largely blocked by serotonin antagonists
e) It stimulates secretion of fluid and electrolytes more than protein

The recommended response is C.

There are two cholecystokinin (CCK) receptors, CCK_1 (previously CCK_A) and CCK_2 (previously CCK_B). The CCK_1 receptor has a much higher affinity for CCK than the CCK_2 receptor and mediates the secretory responses of the acinar cell to CCK. CCK antagonists have been shown to reduce the meal-stimulated pancreatic response, particularly that of proteins more than fluid and electrolytes. When atropine is given with the meal, pancreatic secretory responses are prominently diminished. Surprisingly, it was found that when the pancreas is stimulated by physiologic concentrations of CCK, atropine also inhibited most pancreatic secretion. Further, whether the CCK_1 is present on the human pancreatic acinar cell remains unclear. Thus, in humans it appears that CCK primarily stimulates the pancreas by acting on neural pathways and not directly on the pancreatic acinar cell.

Chapter 2: Training in Biliary Tract Diseases and Pancreatic Disorders

DDSEP Chapter 8: Question 1

The pathogenesis of cholesterol gallstone formation typically includes

A. bile containing excess triglycerides, cholesterol crystals, and hypermotility of the gallbladder
B. simultaneous release of cholecystokinin (CCK) and somatostatin
C. lack of endogenous prostaglandins and antinucleation factors coupled with relaxation of the sphincter of Oddi
D. bile supersaturated with cholesterol, stasis of bile within the gallbladder, and nucleation of cholesterol to form crystals
E. a period of rapid weight gain and calcium deficiency

The recommended response is D.

The three major mechanisms leading to gallstone formation are increased secretion of cholesterol into bile, decreased gallbladder motility, and formation of cholesterol crystals (Hyperlink to Cholesterol Gallstone Pathogenesis section). Patients with cholesterol gallstones characteristically have bile that is supersaturated with cholesterol as predicted from model solutions of cholesterol, bile acid, and phospholipids\(^3\). Excess amounts of bile triglycerides have not been observed in gallstone formation. Altered gallbladder motility promotes gallstone formation by bile stasis. Gallbladder contractility is enhanced by cholecystokinin (CCK) release in response to the fat content of ingested food. The hormone somatostatin, however, has been shown to inhibit gallbladder contractility which aids in gallstone formation. Both the absence of antinucleation factors in bile and sphincter of Oddi relaxation contribute to the solubilization of cholesterol which prevents nidus formation. During rapid weight loss, bile lithogenicity is augmented by the decreased synthesis of bile acids and impaired gallbladder motility which increases the risk of gallstone formation. Calcium deficiency has not been implicated as a direct cause of gallstone formation.

Mucus protects the gastroduodenal mucosal surface through all of the following mechanisms except:

A. Trapping of bicarbonate (HCO$_3^-$) in the unstirred layer  
B. Increasing gastroduodenal mucosal blood flow  
C. Preventing pepsin from injuring epithelial cells  
D. Containing protective chemicals such as lipids and trefoil peptides  
E. Neutralizing hydrogen ion with negatively charged glycoproteins

The recommended response is B.

Mucus protects the gastroduodenal surface by (1) trapping HCO$_3^-$ in the unstirred layer, (2) preventing pepsin and gastric lipase from reaching and attacking the lining epithelial cells, (3) lubricating the gastric lining to prevent abrasions from coarse food particles, (4) neutralizing H$^+$ with negatively charged glycoproteins and peptides of mucus, and (5) trapping ingested bacteria. Mucus also contains protective chemicals that prevent injury to the epithelium, such as lipids (in which H$^+$ and pepsin are insoluble) and trefoil peptides (which maintain mucosal integrity). Mucus does not affect gastroduodenal mucosal blood flow.
Chapter 3: Training in Cellular and Molecular Physiology

DDSEP Chapter 3: Question 2

A 20-year-old man presented to the Emergency Room with severe weakness. He gave a history of loose stools since childhood. Admission laboratory data included: serum sodium 134 mmol/L, serum potassium 2.1 mmol/L, serum chloride 85 mmol/L, serum bicarbonate 45 mmol/L and creatinine 3.5 mg/dL. He received 6 liters of normal saline with potassium intravenously over the first 24 hours in the hospital and serum electrolyte concentrations normalized. Spot stool sample revealed $[\text{Na}^+]$ of 80 mmol/L, $[\text{K}^+]$ of 50 mmol/L, and $[\text{Cl}^-]$ of 95 mmol/L.

His condition is most likely due to a mutation of what gene?

A. APC (adenomatous polyposis coli)
B. DRA (down-regulated in adenoma)
C. CFTR (cystic fibrosis transmembrane regulator)
D. MEN-1 (multiple endocrine neoplasia-type 1)
E. NBC1 (Na$^+$-bicarbonate cotransporter-1)

The recommended response is B.

This patient with congenital diarrhea with alkalosis suffers from chloridorrhea, also known as congenital chloride diarrhea. The condition is characterized by watery diarrhea present from birth, elevated serum bicarbonate concentration, and very high stool chloride concentration (>90 mmol/L). It is due to a mutation in the DRA (down-regulated in adenoma) gene, also known as SLC26A3 (Answer B). This gene is one member of a family of solute-linked carrier (SLC) molecules that are involved with the transport of chloride, bicarbonate, sulfate, iodide, oxalate, formate, hydroxyl and fructose across plasma membranes. SLC26A3 is an electroneutral chloride–bicarbonate exchanger that is present in the ileum and colon. Mutations in this gene make chloride poorly absorbable in the distal intestine and result in retention of fluid within the lumen in proportion to the unabsorbed ions. Diarrhea can be reduced by reducing the number of chloride ions in the lumen by inhibiting gastric HCl secretion with a proton-pump inhibitor or by stimulating an alternative pathway for chloride absorption by administering butyrate.

The APC gene (Answer A) is mutated in patients with familial polyposis syndrome and has nothing to do with chloride transport. CFTR (Answer C) is a chloride channel in the mucosa that is active in mucosal chloride secretion; mutations in its gene are responsible for cystic fibrosis. MEN-1 (Answer D) is the gene that is mutated in multiple endocrine neoplasia, type 1. NBC1 (Answer E) is a sodium–bicarbonate cotransporter in the proximal tubule; mutations of its gene are associated with renal tubular acidosis with ocular abnormalities.

References:
Chapter 3: Training in Cellular and Molecular Physiology

DDSEP Chapter 4: Question 18

The morphine was stopped. The next morning, the patient in 5-18 had an abdominal radiograph. There was colonic gas down to the rectum, dilatation of the entire colon and a cecal diameter of 16 cm. The total white count is $8.3 \times 10^9/L$ with normal differential. The physical examination findings are unchanged. The orthopedic surgeons would like the patient confined to bed, preferably on his back. The most appropriate next management is:

A. Transfer the patient to the endoscopy unit for immediate colonoscopic decompression and placement of a large bore red rubber catheter
B. Emergency endoscopy on the orthopedic ward, keeping the patient on his back while doing an unprepared colonoscopic decompression and placement of soft tube for decompression over the next 24 hours
C. Cardiac and blood pressure monitoring with 1.0 mg neostigmine i.v.
D. Percutaneous cecostomy in the radiology suite
E. A cocktail of antibiotics and probiotics to change the colonic flora

The recommended response is C.

A trial of neostigmine is indicated before any decompressive procedure. The patient should also have daily abdominal examination, radiograph and white count to assess progress and identify any signs of peritoneal irritation or viscus perforation. Monitoring is essential, especially in the elderly, as the anticholinesterase effect of neostigmine can result in bradycardia. If the patient becomes hypotensive, atropine should be used to reverse the effect of neostigmine.

Chapter 4: Training in Endoscopy

DDSEP Chapter 1: Question 4

Which of the following infectious agents is the least likely cause of gastric ulceration?

A. Cryptosporidium
B. Helicobacter pylori
C. Cytomegalovirus
D. Treponema pallidum
E. Herpes simplex virus-type 1

The recommended response is A.

Infectious causes of ulcer disease include viral etiologies such as herpes simplex-type 1 and cytomegalovirus (CMV). These should be suspected in immunocompromised or post-transplant patients. However, CMV-induced ulcers have also been observed in immunocompetent patients. The proof that syphilis causes gastric ulcers comes from the demonstration of the organism in the gastric mucosa, appropriate serology, and evidence that lesions regress after antibiotic therapy. Gastric syphilis is caused by Treponema pallidum, an organism that can be viewed in gastric biopsies by silver staining and by fluorescent antibody techniques. Helicobacter pylori is the most common cause of gastric and duodenal ulcers.
Chapter 4: Training in Endoscopy

DDSEP Chapter 2: Question 4

A 44 year old man from Vietnam who has been in the U.S. for 13 years presents with epigastric and right sided abdominal pain for several months, with a 30 pound weight loss and watery diarrhea, but no fever, chills or night sweats. Physical exam is normal. Three stool ova and parasite exams are negative. A CT scan of the abdomen shows 3 circumferential areas of bowel wall thickening with fat standing in the right colon and adenopathy. At colonoscopy, (see figure 1) there is also a small segment of nodular inflammation in the transverse colon. The most likely diagnosis is:

A. Tuberculosis
B. MAC
C. Lymphoma
D. Schistosomiasis
E. Crohn’s disease

The recommended response is A.

The ulcer at the ileocecal valve is very suggestive of tuberculosis. The segmental nature of the abnormalities and the right sided location strongly favor diagnosis of tuberculosis if this is an infectious ideology while the Crohn’s disease would be a similar presentation in a patient raised in the U.S.. Crohn’s is less likely given this patient’s ethnic background. Without any evidence for HIV mycobacterium avian complex (MAC) and lymphoma are far less likely and are unlikely to present with colonic ulcers such as seen here. Schistosomiasis is not endemic in Vietnam.


Figure 1
Chapter 4: Training in Endoscopy

DDSEP Chapter 3: Question 22

A 36-year-old woman undergoes EGD for diarrhea, weight loss, and abdominal bloating. The endoscopic view of the descending duodenum is shown in Figure 8. Which of the following test results is most likely to be abnormal?

A. Eosinophil count  
B. Serum ferritin  
C. Stool test for ova and parasite  
D. Gastric emptying nuclear scan  
E. Amylase and lipase

The recommended response is B.

The endoscopic view shows mucosal pallor and scalloping of the small intestinal mucosa in the second portion of the duodenum, highly suggestive of celiac disease. These changes have been shown to correlate with degree of villous atrophy. However, the endoscopic appearance of normal duodenal mucosa cannot be used to rule out celiac disease. The use of magnifying endoscopes can enhance the ability to identify villous atrophy, but offer no advantage compared to the gold standard of mucosal biopsy.

Iron deficiency anemia is one of the most common clinical manifestations of celiac disease because iron is absorbed in the proximal duodenum. Thus, an abnormally low serum ferritin level is a common laboratory finding in celiac disease. Eosinophilia is not associated with celiac disease, but is associated with eosinophilic gastroenteritis, crohn’s disease, and parasitic infestations. Stool examination for ova and parasite is useful when the infestation is suspected. However, the endoscopic view in the case is not suggestive of any infection. Gastric emptying nuclear scan and pancreatic enzymes are normal in patients with celiac disease.


Chapter 5: Training in Ethics, Medical Economics, and System-Based Practice

DDSEP Chapter 2: Question 27

How many deaths occur each year in the United States due to food poisoning?

A. 500  
B. 1,000  
C. 5,000  
D. 3,000  
E. 10,000

The recommended response is C.

There are 5,000 deaths per year due to food poisoning in the United States.

CDC net
An Afro-American 13-year-old child is seen because of recurrent abdominal pain (RAP). He started to complain of vague mid-abdominal pain almost 2 years before; initially the episodes were mild, occurred about once every other week, and lasted a few minutes. Subsequently, however, they have appeared more frequently and their intensity may have increased. Currently, he has sharp pain at least twice a week, vaguely localized in the mid-abdominal area, rated 6 to 7 on a pain scale of 1 to 10, presenting in no clear association with any event, but sometimes following a meal. No nausea, vomiting, and/or constipation are reported. However, he does occasionally present loose stools and in 2 occasions they may have been blood-tinged. He reports no major change in appetite, but for the fear that eating may trigger the pain, his food intake has decreased, and his mother thinks he may have lost 2 to 3 kg in the past few months. No contributory elements are found in the past medical history. In review of systems, there has been in the past couple of years some concern about his growth in stature, and parents are planning a visit to an endocrinologist. No family history of GI disorder is reported. Both parents are tall. On physical examination, the boy appears thin; his weight is just below the 3rd percentile and his height is in the 10th percentile. The abdomen is full, with some tenderness at palpation of the mid and lower quadrants; there are no masses. His Tanner stage is II.

Which initial investigations are likely to provide the key for the diagnosis?

A. None, as this boy evidently is affected by functional RAP
B. An EGD
C. A plain X-ray film of the abdomen
D. Lab work: CBC with differential, ESR, CRP, ASCA, and p-ANCA titers, iron panel, albumin
E. Lab work: amylase, lipase, liver function tests (LFTs)

The recommended response is D.

Assessing a child for RAP is a daunting task. Functional causes are known to be very common, occurring in approximately 15% of school-age children and teenagers, and they may have protean manifestations. There are, however, elements in this patient’s history that seem to indicate an organic origin: his stunted growth, his weight loss, the presence of occasional bloody, loose stools, and the objective finding of abdominal tenderness, typically absent in functional RAP. Thus, performing no investigations would be a poor choice. An EGD is definitely an option, however, the patient does not present symptoms or signs clearly pointing to an upper GI involvement and, thus, the procedure is unlikely to provide support for a diagnosis at this initial stage. Even less informative would be a plain film of the abdomen, unlikely to disclose any abnormalities responsible for RAP. Pancreatitits would have presented with a more acute course, would not have caused any blood in the stools, and the location of the pain would have been different. In addition, liver diseases do not cause RAP. Thus, to obtain the tests in option E is clearly not a valid option. Rather, this young teenager with somewhat inadequate skeletal
growth and weight loss, RAP, and occasional bloody and loose stools may well be affected by Crohn’s disease. It is in fact known that changes in linear growth and in weight gain can precede, often by years, any obvious GI signs/symptoms, to the point that it is not infrequent that children with CD are first evaluated by endocrinologists.
Chapter 5: Training in Ethics, Medical Economics, and System-Based Practice

DDSEP Chapter 12: Question 13

Which of the following family histories would prompt a 35 y.o. female to obtain a colonoscopy at age 35?

A. mother with colorectal cancer, age 65

B. father with adenoma, age 45

C. grandmother with colorectal cancer, age 45

D. grandfather with colorectal cancer, age 60

E. grandmother with adenoma, age 50, and a maternal uncle with adenoma, age 55

The recommended response is B.

Screening for colorectal cancer is recommended at age 40, or 10 years younger than the earliest diagnosis in the family, whichever is earliest, when a first degree relative has an adenoma or cancer diagnosed at < 60 years of age, or two first-degree relatives diagnosed with colorectal cancer at any age.

Chapter 6: Training in Geriatric Gastroenterology

DDSEP Chapter 3: Question 3

A 75-year-old woman presents for evaluation of chronic diarrhea and weight loss. She has had loose stools and excessive flatus for six months. During that time her weight has decreased from 110 pounds to 90 pounds (height 67 inches). Diarrhea mainly occurs after meals, but sometimes wakes her from sleep. Stools are especially malodorous and are of varying consistency, but rarely formed. She occasionally notes oil on the surface of the water in the commode. There is no blood in the stool.

Twenty years ago she had an antrectomy and vagotomy for a bleeding prepyloric ulcer. She had intermittent heartburn and was taking omeprazole for suspected reflux disease. She did not have diabetes, heart, or liver disease.

Physical examination showed a thin elderly woman in no distress. Abdomen was slightly distended and the percussion note was tympanitic. Bowel sounds were active. Anal sphincter tone was reduced and squeeze was weak. Stool was brown and fecal occult blood test was negative.

Laboratory tests revealed: hemoglobin 11.5 g/dL, MCV 105 fL, total protein 5.0 g/dL, albumin 2.9 g/dL, serum vitamin B\textsubscript{12} level 100 pmol/L, and serum folate 18 ng/mL.

Her diarrhea is most likely due to:
A. Vitamin B\textsubscript{12} deficiency
B. Post-vagotomy dumping syndrome
C. Superior mesenteric artery syndrome
D. Small bowel bacterial overgrowth
E. Chronic pancreatitis

The recommended response is D.

Diarrhea in the elderly can have many causes. Since so many patients are on multiple medications, drug-induced diarrhea must be considered. She has been taking omeprazole, and diarrhea is a common side-effect of all proton-pump inhibitors. She has had previous gastric surgery which is also a risk factor for diarrhea. Post-gastrectomy diarrhea can be due to anatomical changes produced by the surgery or to complications of the surgery. Anatomical changes can produce dumping syndrome due to unregulated gastric emptying (Answer B), but symptoms typically begin soon after surgery and not years later. B\textsubscript{12} deficiency (Answer A) takes some time to develop after surgery due to relatively large body stores and the time it takes to develop atrophic gastritis post-operatively. Although this patient has documented B\textsubscript{12} deficiency, by itself B\textsubscript{12} deficiency is an unlikely cause for chronic diarrhea. Other problems that can develop over time include gastrocolic fistula and small bowel bacterial overgrowth.

Small bowel bacterial overgrowth (Answer D) is a relatively common cause for diarrhea and weight loss in the elderly. Factors associated with an increased likelihood of a positive glucose breath hydrogen test include increasing age, low serum vitamin B\textsubscript{12} level, low serum albumin concentration, previous partial gastrectomy, previous right hemicolectomy, the presence of small bowel diverticula, and use of a proton pump inhibitor. Elderly patients with chronic diarrhea and any of these risk factors should be considered to have small bowel bacterial
overgrowth until proven otherwise. Small bowel bacterial overgrowth may occur without any of these factors and may be due to motility disorders in the small bowel.

Superior mesenteric artery syndrome (Answer C) occurs when weight loss leads to reduction of the mesenteric fat pad and obstruction of the duodenum by the superior mesenteric artery. This produces vomiting and abdominal pain, but is not a cause of chronic diarrhea. Chronic pancreatitis (Answer E) can cause steatorrhea and weight loss, but she had no particular predisposing factors for this condition.

References:
Chapter 6: Training in Geriatric Gastroenterology

DDSEP Chapter 4: Question 17

A 65-year-old male who weighs 300 lb and underwent total hip arthroplasty for DJD of the right hip 3 days ago, complains of significant bloating, failure to pass gas, and failure to have a bowel movement for 5 days. He has discomfort but denies crampy abdominal pain. His last suit trousers were 43 inch waist. He is taking no antihypertensive medications; the surgery was performed under spinal anesthesia, and he is presently slowly tapering morphine analgesia. The abdomen is markedly distended with a circumference of 50 inches. Bowel sounds are absent.

The most likely diagnosis is:

A. Colon cancer
B. Diverticular stricture
C. **Acute colonic pseudo-obstruction**
D. Opiate induced constipation
E. Peritonitis

The recommended response is C.

While opiates are contributing to the diagnosis, the most likely diagnosis is a multifactorial acute colonic pseudo-obstruction, a known complication after orthopedic procedures including those performed under spinal anesthesia. However, treatment should start with avoidance or stopping agents that interfere with gastrointestinal motility.

Chapter 6: Training in Geriatric Gastroenterology

DDSEP Chapter 4: Question 19

A 72-year-old male has lost 15 kg in the last 6 months, without dieting. He has chronic nausea of 3 months’ duration, aggravated after meals. He gives a 30-pack per year history of smoking. Hematology and chemistry group are unremarkable, except for serum albumen of 3.0 g/dL with no proteinuria. Chest x-ray is reported negative. CT abdomen is negative. The most likely diagnosis is:

A. Idiopathic gastroparesis
B. Chronic idiopathic intestinal pseudo-obstruction
C. Amyloidosis
D. Paraneoplastic pseudo-obstruction
E. Nonulcer dyspepsia

The recommended response is D.

The heavy smoking and significant weight loss are worrisome features suggestive of paraneoplastic pseudo-obstruction. The most likely underlying malignancy is small cell lung cancer. Rarer associations are carcinoid and ovarian tumors. A blood test (antineuronal nuclear antibody) can be used to screen for the paraneoplastic syndrome. Mediastinal CT may be necessary, as the small cell lung cancer may not be visible on a chest radiograph.

A 50 year old man is seen in the office for routine evaluation. He is asymptomatic. He acknowledges drinking one pint of liquor per day for many years. Physical examination reveals marked hepatomegaly, without splenomegaly. Liver enzymes include AST 65 and ALT 25 (normal for each less than 50). CBC, coagulation studies, bilirubin and albumin are normal. If this patient were to undergo liver biopsy, which of the following findings is MOST LIKELY to be present:

A. Microvesicular steatosis  
B. **Macroversicular steatosis**  
C. Macronodular cirrhosis  
D. Mallory’s hyaline  
E. Ballooning degeneration of hepatocytes with polymorphonuclear infiltrate

The recommended response is B.

Macrovesicular steatosis is virtually universal following heavy alcohol use. It is usually asymptomatic but commonly causes hepatomegaly. Microvesicular steatosis is characteristic of disorders of mitochondrial fatty acid oxidation and is a rare manifestation of alcoholic liver disease. Findings of alcoholic hepatitis, such as Mallory’s hyaline, cellular ballooning and polymorphonuclear infiltrate, occur in about 20% of heavy drinkers. Similarly only about 20% of chronic heavy users of alcohol develop cirrhosis, typically micronodular. It should be noted that absence of findings to suggest alcoholic hepatitis or cirrhosis (leukocytosis, jaundice, splenomegaly, ascites, etc.) does not exclude these possibilities, as they are often insidious and subclinical.

Chapter 7: Training in Hepatology

DDSEP Chapter 10: Question 1

A 19 year old Caucasian male is referred to you because he recently developed acute HBV. At the time of diagnosis he was HB surface antigen positive, anti-HB core IgM positive, was icteric with a bilirubin of 6.5 mg/dl and had a serum ALT of 550 IU/l. He has been monitored by his primary care physician for the past 4 months. Serum ALT and bilirubin normalized 2 months ago and HB surface antigen has become undetectable. However, he remains anti-HB surface negative. Which of the following statements is not true of this patient?

A. He is likely to be in the window phase and will eventually develop anti-HB surface with time.
B. Anti-E is likely to be positive.
C. HBV DNA is likely to be negative.
D. **He is likely to develop chronic hepatitis B with a mutant form of the virus.**
E. He is likely to be anti-HB core positive.

The recommended response is D.

This patient has acute HBV and appears to be in the window phase of the natural history of this disease. The time required for anti-HB surface to appear following the resolution of HB surface antigen may take weeks to many months. Since this patient has already normalized serum ALT and bilirubin and has lost HB surface antigen it is very likely he is resolving this infection and does not have chronic HBV with a mutated form of this virus.

DDSEP Chapter 11: Question 16

In a cirrhotic patient with acute hepatic encephalopathy, which of the following tests will not be useful:

A. Diagnostic paracentesis  
B. Stool guaiac  
C. Toxicology screen  
D. BUN, creatinine  
E. Ammonia levels

The recommended response is E.

HE is the neuropsychiatric manifestation of cirrhosis. The diagnosis of HE is clinical and is based on history and physical examination findings. Even though ammonia, a toxin normally removed by the liver, plays a key role in the pathogenesis of hepatic encephalopathy, blood ammonia levels are not useful in the diagnosis and management of HE because they are unreliable and correlate poorly with its stage. The mainstay of therapy of acute HE involves the identification and treatment of the precipitating factor (present in over 80% of cases). Precipitant factors that need to be investigated in any patient with acute encephalopathy are infections (such as spontaneous bacterial peritonitis), prerenal azotemia, electrolyte disturbances, gastrointestinal bleeding, and use of narcotics and sedatives.

A 25 year-old female with a 5-year history of stable Crohn’s colitis presents with fatigue. She denies bowel irregularity, abdominal pain, fever, melena, or rectal bleeding. Her labs are remarkable for Hct 32 with MCV 108. Which of the following is most likely the cause of her anemia?

A. occult GI blood loss  
B. budesonide use  
C. mesalamine use  
D. sulfasalazine use  
E. heavy menses

The recommended response is D.

This medication is an inhibitor of folate absorption. Chronic use may result in folate deficiency causing megaloblastic anemia. Mesalamine does not affect folate absorption. Budesonide use is not associated with anemia. Occult GI bleeding or excessive menstrual blood loss may lead to iron deficiency (microcytic) anemia.

Chapter 8: Training in Inflammation and Enteric Infectious Disease

DDSEP Chapter 2: Question 1

Following a one week vacation in Mexico, a 22 year-old student presents with 3 days of watery diarrhea, some nausea, no vomiting or fever. She has abdominal cramping relieved by bowel movements but no other abdominal pain. Symptoms began a day before returning home, and have continued for the past two days. On exam, she is afebrile and well hydrated. Abdominal exam shows mild left lower quadrant tenderness, with no guarding or rebound.

What is the most appropriate management of this patient?

A. Stool culture
B. Empiric fluoroquinolones
C. Abdominal films
D. No therapy
E. Metronidazole

The recommended response is D.

This clinical scenario is most consistent with traveler’s diarrhea and the most common pathogen is Enterotoxigenic E. coli. In the absences of fever or blood in the stools there is no indication for empiric antibiotic. The history is much more compatible with an acute bacterial infection rather than a parasite. Thus, metronidazole would not be indicated. Given absence of fever and blood in the stool, a stool culture is likely to be of low yield, nor is abdominal x-ray indicated. The correct answer is to do nothing. If the patient does not get better then consideration could be given to stool culture and sending stools for ova and parasites.

Chapter 9: Training in Malignancy

DDSEP Chapter 1: Question 5

A 64-year-old patient with dyspepsia had an upper endoscopy that revealed a gastric ulcer. After histologic assessment of endoscopic biopsies, adenocarcinoma and *H. pylori* infection were diagnosed. Endoscopic ultrasound indicated that the gastric adenocarcinoma was limited to the mucosa but regional lymph nodes were suspicious for malignancy. Abdominal CT scanning confirmed suspicious lymph nodes adjacent to the stomach, but there was no evidence of malignancy in other areas of the abdomen. Which of the following is the most appropriate initial treatment?

A. Subtotal gastrectomy

B. Proton pump inhibitor, amoxicillin, and clarithromycin for 10 days

C. Proton pump inhibitor, metronidazole, clarithromycin, and tetracycline for 14 days

D. Radiation therapy

E. Chemotherapy

The recommended response is A.

The prognosis of gastric adenocarcinoma is primarily related to depth of tumor penetration through the gastric wall, irrespective of the extent of nodal involvement. Early gastric cancer is limited to the gastric mucosa or submucosa, as is seen in this case. Advanced gastric cancer extends into the muscular layers of the stomach and has a worse prognosis. The best curative therapy for early gastric cancer is partial or subtotal gastric resection. Chemotherapy and radiation therapy are much less effective than surgical resection. *H. pylori* therapies are not of benefit in reversing malignancy in patients with established gastric cancer. However, in early gastric cancer patients who have undergone partial gastric resections, *H. pylori* therapies may reduce the frequency of subsequent cancers. Further, in patients with MALToma, eradication of *H. pylori* leads to regression in approximately 80% of cases.


Chapter 9: Training in Malignancy

DDSEP Chapter 4: Question 22

A 38-year-old man reports with longstanding constipation and marked abdominal distension. He reports that his father and 2 of 4 elder siblings have had their thyroids removed for a problem that runs in the family. On examination, blood pressure is 230/120 and, after he gets over anxiety associated with being in the doctor's office, the pressure is measured at 140/90. The abdomen is grossly distended, tympanic and non-tender. The most likely diagnosis is:

A. Familial goiter, hypothyroidism and constipation

B. Medullary carcinoma of thyroid and megacolon

C. Multiple endocrine neoplasia type IIA

D. **Multiple endocrine neoplasia type IIB**

E. Familial thyrotoxicosis

The recommended response is D.

The patient has MEN IIB and should have colon evaluation, serum calcitonin, urinary metanephrines, ultrasound of thyroid and adrenal glands since 100% of patients develop medullary carcinoma of the thyroid and about 60% develop phaeochromocytome.
Chapter 9: Training in Malignancy

DDSEP Chapter 12: Question 3

A 70 year-old white man presents to you with long-standing history of GERD, which is poorly controlled. He weighs 220lbs and drinks heavily. His father died of “cancer of the stomach” at age 68. He intermittently takes a proton pump inhibitor. His last endoscopy revealed long segment Barrett’s with indeterminate dysplasia. The best course of action to prevent adenocarcinoma of the esophagus is to

A. Start on a standing dose PPI  
B. Refer for fundoplication  
C. **Counsel to lose weight**  
D. Counsel to stop drinking  
E. Start on an NSAID

The recommended response is C.

Obesity appears to be one of the most important risk factors associated with esophageal cancer. Also, life style changes have very little if any adverse effects. Controlling acid secretion has not been shown to decrease development of esophageal cancer, nor has fundoplication, possibly as neither approach guarantees cessation of reflux. Alcohol is not a risk factor for adenocarcinoma, but this patient would benefit from alcohol cessation to decrease his risk of developing squamous cell cancer of the esophagus. NSAIDs are currently not recommended as a chemopreventive measure, but may be advocated in the near future for high-risk patients with dysplasia.

Chapter 10: Training in Motility and Functional Illnesses

DDSEP Chapter 3: Question 19

All of the following drugs frequently are associated with the development of constipation EXCEPT:

A. Amitriptyline
B. Amlodipine
C. Amoxicillin
D. Aluminum hydroxide
E. Antihistamines

The recommended response is C.

Drugs are a common cause of constipation and it is essential to go over a complete list of all prescription, over-the-counter, and herbal preparations that a patient is taking. Anticholinergic drugs and drugs with anticholinergic side-effects like the tricyclic antidepressants frequently produce constipation (Answer A). Calcium channel blockers are another category of drugs that may cause constipation (Answer B). Aluminum and calcium salts also tend to be constipating (Answer D). Antihistamines (both sedating and non-sedating) may cause constipation (Answer E). Not listed but probably the most problematic are narcotics. These agents routinely cause constipation at doses used to produce analgesia. Stimulant laxatives such as senna may be needed to maintain regular bowel movements.

Reference:
Chapter 10: Training in Motility and Functional Illnesses

DDSEP Chapter 2: Question 15

A 30 year old woman has had alternating diarrhea and constipation for the past ten years. Physical exam is normal. Stool for enteric pathogens is negative, but a stool exam for ova and parasites shows rare Blastocystis hominis (fewer than 5 organisms).

A. Treat with metronidazole  
B. Resend stool culture  
C. Test well water  
D. No treatment  
E. Treat family members

The recommended response is D.

Blastocystis hominis can be seen in the stools. While there is debate as to whether or not this is a pathogen, most feel that it is not pathogenic. However, if a patient is immunosuppressed or Blastocystis hominis is present in large numbers, it might be reasonable to give a therapeutic trial to see if diarrhea resolves, but just as reasonable would be no treatment at all.

The nerve network situated between the longitudinal and circular layers of the muscularis propria is named:

A. Meissner's plexus  
B. Laimer's plexus  
C. Auerbach's plexus  
D. The triangular plexus  
E. The brachial plexus

The recommended response is C.

Auerbach's plexus, also known as the myenteric plexus. Meissner's plexus is the submucosal plexus; the others are not enteric structures.
Supplementation with which of the following may improve outcomes in infectious diarrhea in children?

A. Zinc
B. Calcium
C. Magnesium
D. Selenium
E. Iron

The recommended response is A.

There is evidence in children that zinc supplementation improves outcomes in chronic diarrhea. Zinc deficiency has been shown to cause diarrhea.

Chapter 11: Training in Nutrition

DDSEP Chapter 12: Question 15

Which of the following statements is false?

A. Age is a risk factor for developing colorectal adenomas and cancer
B. Family history is a risk factor for developing colorectal adenomas and cancer
C. Blacks in the U.S. have a higher proportion of cancers under the age of 50
D. High serum selenium is a risk factor for developing colorectal adenomas and cancer
E. Crohn’s colitis is a risk factor for developing colorectal adenomas and cancer

The recommended response is D.

Low serum selenium, not high, is associated with the development of colorectal cancer. Age, family and personal history of polyps and colorectal cancer, and ethnicity (particularly blacks and Ashkenazi Jews with the I1307K mutation) can be associated with a higher risk for colorectal cancer.


Chapter 11: Training in Nutrition

DDSEP Chapter 13: Question 1

A 30 year-old man had resection of 120 cm of ileum following a gunshot wound to the abdomen. He is least likely to become deficient in which of the following nutrients:

A. Iron  
B. Vitamin D  
C. Vitamin A  
D. Vitamin B\textsubscript{12}  
E. Magnesium

The recommended response is A.

Vitamin B\textsubscript{12} is absorbed in the ileum, and resection of 120 cm will result in vitamin B\textsubscript{12} malabsorption. The ileum is the site of bile salt malabsorption, and resection of 100 cm would result in bile salt deficiency, steatorrhea, and fat soluble vitamin (A, D, E, K) malabsorption. Magnesium will bind to malabsorbed fatty acids and be lost in the stool. Iron is absorbed mainly in the duodenum, and would not be affected by an ileal resection.

A 45-year-old white male business executive sees you because of classic heartburn and acid regurgitation. An upper endoscopy revealed a displaced squamocolumnar junction. Eight large capacity biopsies were obtained and were read by your pathologist as containing columnar epithelium with pseudogoblet cells that stain negative by Alcian blue staining. You have reviewed the slides with the pathologist and tell the patient which of the following?

A. Biopsies confirm the diagnosis of Barrett’s esophagus and repeat surveillance endoscopy in 1 to 2 years
B. Biopsies confirm the diagnosis of Barrett’s esophagus and repeat surveillance endoscopy in 2 to 3 years
C. Request that the pathologist obtain cytokeratin stains to differentiate the columnar epithelium originating from the stomach or the esophagus
D. Suggest mucosal ablation with bipolar electrocoagulation to eliminate the columnar epithelium
E. Inform the patient that biopsies do not confirm the diagnosis of Barrett’s esophagus, however, since this could represent sampling error you would like to confirm the finding by repeating the biopsy.

The recommended response is E.

This patient represents a common dilemma in Barrett’s esophagus, namely is it present or not? The diagnosis of Barrett’s esophagus is based on two components: displacement of the squamocolumnar junction above the level of the esophagogastric junction as described in this patient and the histologic finding of specialized intestinal metaplasia defined by goblet cells in biopsies obtained from this segment. Some pathologists will still commonly refer to gastric fundic and cardia type epithelium, as found in this patient as “compatible with Barrett’s esophagus”. Thus, while this patient had a displaced squamocolumnar junction, intestinal metaplasia was not encountered. These biopsies demonstrate instead columnar epithelium with pseudogoblet cells characterized by distended gastric surface foveolar-type cells that stain for PAS but do not contain Alcian blue positive acid mucins. However, since the true specialized intestinal metaplasia could have been missed by sampling error, confirmation of the finding by repeat biopsy should be considered. Ablation therapy at present has no role in Barrett’s esophagus without dysplasia. Unfortunately, immunohistochemical cytokeratin staining patterns of biopsies obtained from questionable areas of Barrett’s esophagus do not reliably distinguish between intestinal metaplasia of the cardia and the esophagus.

Chapter 12: Training in Pathology

DDSEP Chapter 2: Question 8

A 19 year old college student passes a large thin white object (see Figure 3). He has noted mild watery diarrhea and a 10 pound weight loss. He had traveled extensively in Russia the previous summer, and enjoys sushi.

Which of the following is most likely?

A. Ascaris  
B. Taenia solum  
C. Taenia saginatum  
D. Diphyllobothrium latum  
E. Hymenolopsis nana

The recommended response is D.

The picture shows a tapeworm. Ascaris can be seen grossly in the stools but looks different. While any of the tapeworms are possible, the history of eating sushi strongly suggests the freshwater fish tapeworm D. latum associated with fish and could be seen with salmon sushi as salmon migrate in a fresh water phase. The appropriate treatment is Invermectin.


Figure 3
Chapter 12: Training in Pathology

DDSEP Chapter 3: Question 24

A 35-year-old woman complains of intermittent episodes of hematochezia with bowel movements. She also notes tenesmus, mild rectal pain, and the need to strain during bowel movement. She occasionally needs to manually assist her defecation. Colonoscopy is performed and the endoscopic view of rectum is shown in Figure 10. Which of the following is not the pathology finding of this condition?

A. Obliteration of the lamina propria by fibromuscular proliferation
B. Cryptitis and crypt abscess
C. Thickened muscularis mucosae
D. Branched and distorted crypts
E. Collagen infiltration of the lamina propria

The recommended response is B.

The endoscopic figure shows a solitary rectal ulcer. Solitary rectal ulcer syndrome is a spectrum of clinicopathological abnormalities which can affect both adults and children. The pathogenesis remains obscure, but seems to involve abnormal contraction of the pelvic floor and rectal prolapse, resulting in rectal mucosal ischemia. The clinical characteristics of this syndrome include rectal bleeding, mucus in the stool, tenesmus, and straining to defecate. Patients often instrument their own rectum because of a sensation of incomplete evacuation. Endoscopic appearance varies from erythema (18%) to ulceration (57%) or polypoid lesions (28%). These lesions are usually found on the anterior rectal wall and could be single or multiple, measuring between 0.5 and 5.0 cm in diameter. This condition is rare and may be misdiagnosed as inflammatory bowel disease or cancer. Defecography or proctography may be useful to evaluate pelvic floor dysfunction. Evidence of rectal prolapse, either external or internal, with or without abnormal perineal descent on evacuation may be detected. Biopsy specimens show obliteration of the lamina propria by fibromuscular proliferation, thickened muscularis mucosae, branched and distorted crypts, and collagen infiltration of the lamina propria. Treatment should be aimed at restoring a normal pattern of defecation pattern. The patient should be instructed to avoid excessive straining and to regulate defecation habits by biofeedback behavioral retraining and fiber supplements. Modest osmotic laxatives can be considered. Topical steroids and 5-ASA enemas are not effective. Sucralfate enemas and local applications of human fibrin sealant have provided some improvement in small case series. If these local measures do not resolve the problem, surgical treatment may be considered.


A 5-year-old boy presents with a history of constipation. His mother reports that in the past 2 years, the child—who had been properly toilet-trained around age 2—had started to have less frequent defecations. The stools have been typically large, hard, and passed approximately every 3 to 4 days and with “a lot of straining.” In a couple of instances, streaks of fresh blood have been seen around stools. She informs you that several remedies such as fruit juices, prunes, and mineral oil have failed. The only notable finding in his past medical history is the need for many formula changes during his first year of life, as mom says he was “lactose intolerant.” This appears to have regressed after age 13 months and he now drinks milk without problems. The physical examination is unremarkable, except for some stools palpated in the rectal vault, slightly hemato-negative.

In addition to functional constipation, what would be a likely diagnosis for this patient’s symptoms?

A. Hirschsprung’s disease (HD)
B. Lactose intolerance
C. Cow’s milk protein allergy
D. Hypothyroidism
E. Ectopic, anteriorly displaced anus

The recommended response is C.

The history and physical both rule out HD; there is no malnutrition, history starts late (is neonatal in more than 90% of HD), stools are large and round (they are string-like in HD), and are present in the rectal vault. Likewise, an anteriorly-displaced anus is easily ruled out by the physical examination; this often-overlooked disorder typically causes constipation from an early age, as the angle between the rectum and the anal canal becomes too narrow for stools to be expelled normally. Although hypothyroidism can lead to constipation, for this problem to cause it other signs would have to be present; at this age in particular, short stature and slowed development. Lactose intolerance is rarely present at this young age and would lead to the opposite clinical manifestation of diarrhea. Cow’s milk protein allergy (CMPA), on the other hand, is a relatively recently recognized cause of constipation. Allergic symptoms may appear during infancy with GI manifestations such as vomiting and diarrhea (a condition that commonly leads to multiple formula changes and is often erroneously referred to as “lactose intolerance”), which can subside. However, a condition of proctitis may occur and continue in the following years with minimal overt symptomatology, essentially causing painful defecations and, hence, a voluntary retention of stools that is the ultimate reason for constipation. The response to withdrawal of milk and derivatives from the diet is typically dramatic.
An 8-year-old Caucasian boy is brought to his primary care pediatrician with a 6-month history of recurrent abdominal pain. The episodes occur 3 to 4 times per week and consist of “twisting” pain, in the mid- and lower abdominal quadrants. The episodes are occasionally associated with loose stools; otherwise, there are no additional gastrointestinal (GI) or systemic complaints. According to the mother, the boy’s appetite has diminished and there may have been some weight loss, but this is not documented. The mother also thinks that the child has not grown in 2 years. The past medical history is not contributory; he had been breast-fed for the first 12 months of life. There is no family history of any significant GI disease. The patient has a 13-year-old brother who had been diagnosed with type 1 diabetes mellitus 2 years earlier.

Which of the following tests is recommended?

A. 24-hr. pH-probe monitoring
B. Colonoscopy
C. Serum antitissue transglutaminase
D. Serum antigliadin antibodies
E. Serum H. pylori antibody titer

The recommended response is C.

This boy has celiac disease. This condition affects almost 1% of Caucasians and can present at any age following introduction of gluten. Presentation with recurrent abdominal pain, though not its most common presenting symptom, is well described. Diarrhea, reduced appetite, and weight loss are additional symptoms. In addition, there is a first-degree relative with type 1 diabetes, a condition well known to be associated with a high incidence of celiac disease. The best test to screen for celiac disease is the measurement of serum antitissue transglutaminase: the test is reported to have a sensitivity of almost 100%, with a somewhat lower specificity. Adding serum IgA antibodies to detect the occasional patient with IgA deficiency (about 3% of all celiac disease patients) can be useful. Antigliadin antibodies are much less specific and they should be omitted from the tests used in screening. There are no indications for colonoscopy in this child at this stage, as the procedure is not likely to show significant causes of recurrent abdominal pain in the absence of signs or symptoms of colonic involvement. H. pylori gastritis is poorly correlated with the symptoms described; furthermore, serum H. pylori antibody titer is a relatively poor test for this infection, with a low sensitivity. Finally, the clinical presentation does not suggest gastroesophageal reflux disease (GERD), making the indication for a pH probe not necessary.
Chapter 13: Training in Pediatric Gastroenterology

DDSEP Chapter 14: Question 2

A 16-year-old child known to have celiac disease since age 4 (diagnosed after a 2-year history of chronic diarrhea and weight loss) is seen as an outpatient 3 years after his last appointment (the patient had not kept the expected annual appointments). His mother believes that he is not adhering strictly to the diet, although he would not admit it. The patient displays a defiant attitude and denies any dietetic transgression. He does say that when on just 1 or 2 occasions in the past years he may have eaten some gluten-containing food, this hasn't bothered him at all, to the point he is not sure he has the disorder. Mom insists that he appears more tired, something he vehemently denies. Physical exam shows a normal teenage boy, not malnourished.

What is the best course of action?

A. Surmise that the patient may indeed have been ingesting substantial amounts of gluten and that he must have grown out of his gluten intolerance, as he has evidently not shown any of the symptoms he presented when first diagnosed, and his physical examination is within normal limits
B. Tell the patient that there is no such thing as transient celiac disease and he must stay on a diet as originally recommended
C. Plan a repeat esophagastroduodenoscopy (EGD) with duodenal biopsy to verify if he is relapsed
D. Obtain serologic evidence of recurrence by checking anti-Endomysium antibodies (EMA) and, if results are positive, try to reinforce the need to stay on a diet
E. Obtain blood work for CBC, serum iron, and antigliadin antibodies

The recommended response is E.

Teenagers are well known to often resist compliance to pharmacologic and/or dietetic treatments. This boy had celiac disease and should have been seen at regular yearly intervals to monitor compliance and any medical/nutritional issues. Celiac disease is permanent, so there is no chance that he has outgrown it, although even long-term "apparent health" has been described in celiac patients who were not on a gluten-free diet; however, to simply state that he needs to stay on a diet without further action is unlikely to result in any change in compliance. A repeat EGD might show signs of relapse; however, this is not necessarily the case, as relapse of duodenal morphological changes can occur as late as 2 years in a minority of celiac patients eating gluten. Thus, to propose the biopsy directly would not be a good choice. Although EMA
are highly specific, they have less sensitivity and are not the most reliable tests to judge minor dietary indiscretions. On the contrary, anti-gliadin antibodies are much less specific for celiac disease, but—being anti-food proteins antibodies unlike the EMA autoimmune antibodies—they are promptly responsive to the introduction of gluten. Thus, a well-followed gluten-free diet should result in almost undetectable serum values of anti-gliadin antibodies, both IgA and IgG. Furthermore, if the patient is indeed reacting to gluten ingestion and is easily tired as mother reports, checking him for possible iron-deficiency anemia is indicated, and may well serve to enforce the dietetic compliance.
A 52-year-old white male presents for evaluation of sudden onset of abdominal pain and shoulder pain. His past medical history is notable for a history of coronary artery disease, hypertension, gallstones, and osteoarthritis. Medications include an ACE inhibitor, a beta-blocker, aspirin and ibuprofen. Abdominal examination is remarkable for hypoactive bowel sounds, a rigid abdomen and generalized rebound tenderness. Rectal exam is normal and fecal occult blood testing is negative. In evaluation of his shoulder pain, a portable CXR was taken and reveals free air. What is the next most appropriate step in the management of this patient?

A. Consult general surgery
B. Gastric lavage with saline to assess for GI bleeding
C. Abdominal CT scan
D. MRI
E. *Helicobacter pylori* antibody testing

The recommended response is A.

The chest X-ray is consistent with a perforated viscus. There is a past history of peptic ulcer disease in up to 75% of patients with perforated viscus. The acute onset of severe abdominal pain is usually the first symptom of a perforated duodenal or gastric ulcer. Elderly patients taking NSAIDs are particularly susceptible to complications of peptic ulcer disease such as perforation and most patients with perforated ulcers will require operations. While an abdominal CT scan is a more sensitive test for the detection of free abdominal air than routine X-rays, in this case, an abdominal CT scan is not necessary to make the diagnosis of a perforated viscus. Since it has already been established that this patient has a perforation, instillation of fluids through a NG tube for gastric lavage would not be prudent and would likely lead to peritoneal contamination. While it would be helpful to know whether *Helicobacter pylori* contributed to pathogenesis of ulceration, the more immediate concern is to repair the perforation. Surgical exploration is the preferred approach for most patients presenting with pneumoperitoneum; however, some patients may be poor surgical candidates because of co-morbid diseases. In such patients, medical therapy may be successful. Such patients may include those with perforation of greater than 24 hours in whom a water-soluble upper GI contrast study reveals the perforation to be sealed off or completely contained. Medical therapy would consist of a nasogastric tube with intermittent suction, intravenous H₂-blockers or proton pump inhibitors and antibiotics.
Chapter 14: Training in Radiology

DDSEP Chapter 2: Question 6

A 40 year old woman hospitalized after a car accident undergoes surgery to stabilize a spinal injury. She develops a spinal abscess and requires long term antibiotics with metronidazole and moxiflaxacin and is discharged to a chronic care facility. A month later she develops acute abdominal distension, she does not have diarrhea and has had no bowel movement for 3 days. On evaluation in the ER she has hypotension fever, and respiratory compromise. A WBC count is 36,000. CT scan shows marked colon wall thickening (Figure 2). A rapid stool test for *C. difficile* is positive for toxin A.

What is the most appropriate next step?

A. Colonoscopy with decompression  
B. **Exploratory laparotomy and colectomy**  
C. NG tube and IV antibiotics  
D. Urgent left hemicolecctiony  
E. Antibiotics per rectum

The recommended response is B.

The patient already is presenting with significant evidence of toxic colon and imminent perforation. The most appropriate step is exploratory laparotomy and colectomy. Colonoscopy is not needed for diagnosis as a rapid stool test for toxin A was positive and may also be dangerous. NG tube and antibiotics are unlikely to be helpful in a patient so sick. Antibiotics per rectum are unlikely to be helpful when the patient is this sick. Left hemicolecctiony would not adequately remove the diseased colon. A total colectomy with ileostomy should be done.

Chapter 14: Training in Radiology

DDSEP Chapter 4: Question 25

A 45-year-old woman presents for an evaluation of progressive dysphagia for 2 years. She complains of symptoms with both solids and liquids. Occasionally, she develops regurgitation of undigested foods 10-15 minutes after meals. Figure 12A shows the endoscopic view of the distal esophagus. Figure 12B shows the finding of barium esophagogram. Which of the following is the most likely diagnosis?

A. Schatzski ring

B. Adenocarcinoma of the esophagogastric junction

C. **Achalasia**

D. CREST syndrome

E. Hiatal hernia with gastroesophageal reflux disease

The recommended response is C.

The endoscopic figure shows a “rosette” appearance of the lower esophageal sphincter that remains closed with air insufflation. However, the endoscope can easily traverse with gentle pressure allowing examination of the stomach. The barium esophagogram shows the dilated esophagus ends in a so-called pointed bird’s beak that represents the nonrelaxing lower esophageal sphincter. These features are consistent with achalasia. The disease affects both sexes equally and can occur at any age. Onset is usually in the third to fifth decades. The duration of symptoms at presentation averages 2 years. Dysphagia is almost uniformly the predominant symptom, with solids in nearly all patients and with liquids in at least two thirds. The combination of dysphagia for both liquids and solids has some utility in suggesting achalasia over obstructive strictures or tumors. The severity of dysphagia fluctuates and often accompanies chest pain, regurgitation, weight loss, nocturnal coughing spells, and bronchopulmonary complications. The diagnosis is suspected from a compatible clinical history and confirmed by typical findings in barium esophagogram, EGD, and manometry.


Chapter 15: Training in Research

NO QUESTIONS
A 62 year old male with a 30 year history of alcohol abuse presents with jaundice and pruritus. His bilirubin is elevated to 6 mg/dl and transaminases are 2.5 times the upper limit of normal. Abdominal CT scanning revealed calcification in the head of the pancreas but no mass. ERCP revealed multiple strictures in the main pancreatic duct and a moderately high grade 2 cm stricture in the intrapancreatic portion of the common bile duct. No malignancy was detected on brushings and biopsies of the stricture as well as by EUS guided FNA. The CEA and CA 19-9 levels were normal. The patient has no constitutional signs of malignancy.

Which of the following is the most effective way of managing the common bile duct stricture.

A. ERCP with balloon dilation and placement of several plastic stents across the stricture.
B. **Roux-en-Y Choledochojjunostomy**
C. Whipple procedure
D. Pancreatic enzyme supplementation and Octreotide
E. ERCP with metal stent placement

The recommended response is B.

ERCP with serial dilations and stenting is an effective therapy for benign bile duct strictures not related to chronic pancreatitis. In chronic pancreatitis only 25-50% respond. This poor response is presumably because of the differing pathogenesis between biliary strictures associated with chronic pancreatitis and post-operative strictures. In chronic pancreatitis, fibrotic tissue and calcification in the pancreatic head surrounds the bile duct; in the latter, only the duct wall is fibrotic. ERCP with metal stenting has been attempted; however, universal stent occlusion due to epithelial hyperplasia limits this approach. A Whipple procedure for this situation would be unreasonable. Pancreatic enzyme supplementation and Octreotide would be unlikely to confer significant benefit. A biliary enteric bypass is the appropriate management in this patient.

Chapter 16: Training in Surgery

DDSEP Chapter 3: Question 15

A 46-year-old woman presents to your office because of refractory constipation present since childhood. She has marked infrequency of defecation when not using laxatives or enemas, but has no problem expelling stool that is in the rectum. She has tried various laxatives (milk of magnesia, polyethylene glycol, lactulose, bisacodyl, senna) which have proven ineffective, even when given in very large doses. In addition, she has taken cisapride, tegaserod, misoprostol, colchicine, and bethanechol with some transient success, but ultimate failure. She was currently keeping her bowels open with large volume tap water enemas twice a week. She had no abdominal pain of note and was not depressed.

Physical examination showed no evidence of systemic illness. Her abdomen was soft and not distended. Bowel sounds were active. No fecal masses were palpable. Rectal examination showed normal anal sphincter tone. No stool was present in the rectum. The rectal outlet opened appropriately when simulating defecation during digital examination.

Since medications had not worked, she is interested in surgery for her problem.

The procedure of choice for her problem would be:

A. Subtotal colectomy with ileorectal anastomosis
B. Subtotal colectomy with ileoanal anastomosis
C. Segmental resection of the rectosigmoid colon
D. Pelvic floor reconstruction
E. Anal sphincterotomy

The recommended response is A.

Patients with refractory constipation sometimes are referred for consideration of surgical approaches to their problem. Surgery can improve a desperate situation, but patient selection is the key to success. Patients who have pain as a major complaint, who have depression, or who have unrealistic expectations about the outcome of surgery are poor prospects. Surgery can improve bowel frequency and reduce the time and bother required to keep one’s bowels open, but it does not necessarily reduce pain, ease depression or improve interpersonal relationships. Patients who have significant depression or pain often have more problems—particularly bowel obstruction—post-operatively than those who don’t have these comorbidities.

The procedure of choice for severe slow transit constipation is subtotal colectomy with ileorectal anastomosis (Answer A). Ileostomy is an alternative, but generally less satisfactory option. Subtotal colectomy with ileoanal anastomosis (Answer B) is a technically more challenging procedure that is usually not needed for this indication; technical failure requiring resection of the ileal pouch results in mandatory creation of an end-ileostomy. Lesser resections, such as segmental resection of the sigmoid colon, almost never are successful regardless of how “redundant” the sigmoid colon appears to be; the problem with severe slow transit constipation seems to be a pancolonic process. Her history is not consistent with functional outlet obstruction or dyssynergia and operations on her pelvic floor (Answer D) or anal sphincter (Answer E) are not going to be helpful.

References:
A 65-year-old man with a history of gastroesophageal reflux disease and large hiatal hernia underwent a laparoscopic Nissen fundoplication 5 years ago. His symptoms completely resolved after surgery. He presents with recurrent symptoms of heartburn and regurgitation for 2 months. These symptoms have been partially controlled with oral administration of proton pump inhibitors. The retroflexed view of gastroesophageal junction is shown in Figure 24. Which of the following is the most likely diagnosis?

A. Slipped Nissen fundoplication
B. Paraesophageal hernia
C. Recurrent hiatal hernia
D. Partial disruption of Nissen fundoplication
E. Intact Nissen fundoplication

The recommended response is D.

The endoscopic figure shows the retroflexed view of gastroesophageal junction. There appears to be the absence of anterior and posterior grooves and visible suture material in the gastric fundus suggesting partial disruption of the Nissen fundoplication. Reappearance of gastroesophageal reflux symptoms after a successful antireflux surgery warrants further investigations. The most important investigation is the careful examination of gastroesophageal junction during EGD. Several causes of recurrence can be diagnosed, including a slipped Nissen fundoplication, total or partial disruption of the Nissen fundoplication, and disruption of the crural repair resulting in recurrent hiatal hernia (herniation of the intact fundoplication into the chest cavity). Though EGD is more sensitive than barium contrast radiography, paraesophageal hernia and anatomical relationships among organs may be better appreciated by upper gastrointestinal series. In addition, esophageal pH monitoring study can document the pattern, frequency, and duration of acid reflux, and can help to establish that post-operative symptoms are secondary to acid reflux.


You are asked for an opinion by the family physician of a 25-year old female with a chronic unexplained cough. A chest X-ray was negative and she has failed to respond to twice daily PPI therapy given for 16 weeks. A dual 24-hour pH test performed by another gastroenterologist revealed no increase in acid exposure in the proximal or distal esophagus while on therapy. She has never had any heartburn or acid regurgitation.

What would be the best next step?

A. Upper endoscopy  
B. Barium esophagram  
C. Surgical referral for failure of medical therapy  
D. Addition of a nocturnal dose of a H$_2$-receptor antagonist  
E. Appropriate referral to evaluate for asthma with a methacholine challenge test and sinus disease with a CT of the sinuses

The recommended response is E.

Chronic unexplained cough is one of the extraesophageal manifestations of GERD. Just as in patients with typical symptoms of GERD, there is no diagnostic gold standard for GERD in these patients. Upper endoscopy has a low diagnostic yield in patients with extraesophageal manifestations of GERD and would be of little use in this patient. The same is true for a barium esophagram. Antireflux surgery as an empiric trial is tempting to consider in patients who fail medical therapy. However, unlike the setting of classic GERD where the short-term response rate is approximately 90%, the response for atypical symptoms is closer to 50% unless there is documented acid exposure and a prior response to a PPI. This patient has not had a response to PPI therapy. Nocturnal acid breakthrough, is defined as pH <4 for at least one hour in the overnight period despite twice daily PPI therapy. While a night-time dose of a H$_2$-receptor antagonist may correct nocturnal acid breakthrough when administered acutely, chronic use of a H$_2$-receptor antagonist is associated with tolerance and thus may not reliably decrease intragastric pH when administered on a chronic basis. Furthermore, nocturnal gastric acid breakthrough is a pharmacologic observation that has never been demonstrated to have clinical significance. There is no evidence of reflux in this patient and the failure to respond to PPI therapy predicts a suboptimal response to antireflux surgery.

Patients with a clinical profile highly suggestive of silent GERD as a cause of their cough are characterized by the following findings: 1) normal or clear chest X-ray; 2) no smoking or exposure to environmental irritants; 3) no use of ACE inhibitors; 4) negative methacholine challenge or failure of cough to improve with treatment of asthma; and 5) failure of cough to improve with treatment of postnasal drip syndrome. The most common causes of unexplained cough are asthma, postnasal drip and GERD. Often, these patients may have more than one etiology for their symptoms. A definite diagnosis of cough due to GERD requires that cough nearly or completely be resolved with anti-reflux treatment. This patient failed to respond to an empiric trial of twice daily PPI therapy with documentation of adequate acid suppression. As such, the best management approach in this patient is to exclude asthma with a methacholine
challenge test and sinus disease with either a CT scan of the sinuses or administration of empiric therapy for postnasal drip.

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An 18-year-old woman freshman student in college presents with “reflux” of food unresponsive to a proton pump inhibitor taken at full dose t.i.d. The problem started during her first semester in college; she is 5 ft. 4 in. tall and weighs 170 lb. Her roommate is a junior who is a cheerleader for the college. The patient denies heartburn at night and complains that food comes up, almost without any antecedent nausea or retching. Your surgical colleague wants to offer her a once-and-for-all operation for reflux. She has lost 14 lb. Optimal management is:

A. Full fundoplication
B. Loose fundoplication (e.g., Toupet procedure) to avoid postoperative dysphagia
C. Referral to a psychologist for behavioral therapy
D. A prokinetic (e.g., tegaserod 6 mg) before each meal
E. Partial gastrectomy with Roux-Y reconstruction to avoid bile reflux gastritis.

The recommended response is C.

The history is highly suggestive of rumination syndrome due to social pressures. Behavioral therapy such as teaching postcibal diaphragmatic breathing is indicated. Fundoplication should be avoided at all costs. It stops the food regurgitation, but replaces that symptom with severe nausea, upper abdominal pain, gas, and bloating with inability to belch.


Chapter 17: Training in Women's Health in Digestive Diseases

DDSEP Chapter 4: Question 20

A 38-year-old woman with 4 prior vaginal deliveries and episiotomies and with a past history of cold hands that turn intensely blue and painful in cold water presents with diarrhea, foul smelling floating stools, and occasional nocturnal stool incontinence. She denies difficulty swallowing. X-rays of her hands show calcifications. Rectal examination: normal anal squeeze pressure. The most likely diagnosis is:

A. Hyperparathyroidism and malabsorption
B. Celiac disease
C. Postpartum external anal sphincter damage
D. Progressive systemic sclerosis
E. Crohn’s disease with small joint arthropathy

The recommended response is D.

The clinical features suggest bacterial overgrowth and internal anal sphincter weakness due to scleroderma. The normal anal squeeze pressure suggests normal external sphincter, unlikely to be associated with partum injury. About 5% of patients with gut scleroderma do not have esophageal involvement. In this patient, the peripheral manifestations and any positive serology will obviate the need for gastrointestinal manometry. If there is dilation of the gut (esophagus, small bowel) on x-ray, no further test is usually needed.