Chapter 1: Training in Acid-Peptic Disease

Author: Arthur J. DeCross, MD, AGAF

A 30-year-old patient is placed on a Proton Pump Inhibitor (PPI) for post-prandial heartburn. There is no complaint of dysphagia, emesis, weight loss, or GI bleeding. Several weeks later, the patient is seen in follow up and complains of abdominal ache with several loose stools daily, although the heartburn is relieved. A serum gastrin level is elevated at twice the upper limit of normal. You are consulted for further evaluation. The most appropriate approach to this scenario is:

A. Order a secretin stimulation test and gastric acid analysis
B. Order an octreotide scan and perform an endoscopic ultrasound
C. Perform an upper endoscopy
D. **Discontinue the PPI, and recheck the serum gastrin in a few weeks**
E. Change PPI brands

The recommended response is D.

Abdominal ache, loose stools, and headache are among the most common side effects of the PPI class. Significant elevations in serum gastrin secondary to the acid suppression are not uncommon. The most rational, cost effective, and sensible approach to this patient is to discontinue the medication, and repeat the serum gastrin which will return to normal in just 2-3 weeks. Endoscopic imaging is not indicated for uncomplicated dyspepsia. Gastric physiology testing would be inappropriate prior to excluding a diagnosis of drug induced hypergastrinemia, and imaging studies for gastrinoma would be inappropriate before confirmed abnormalities of gastric physiology testing. Changing PPI brands would be unlikely to resolve the subjective symptoms, and would not change the hypergastrinemia.
Chapter 1: Training in Acid-Peptic Disease

Author: Arthur J. DeCross, MD, AGAF

The following groups of patients should be considered empirically for peptic ulcer prophylaxis when initiated on a new prescription of NSAIDs, except:

A. Elderly patients
B. Patients concurrently on other NSAID prescriptions
C. Patients concurrently on steroids
D. Patients on anti-coagulation
E. Young women of child-bearing potential

The recommended response is E.

The elderly, and patients on concurrent anti-coagulants, concurrent NSAID prescriptions, and concurrent steroids are all at significantly higher risk of peptic ulcer incidence and/or complications when initiating a new NSAID medication, and should be considered for empiric prophylactic therapy with either PPI or misoprostol. Not only are young women not particularly at increased risk, but the use of misoprostol is relatively contraindicated in women of child-bearing potential because it is an abortifacient.
Chapter 1: Training in Acid-Peptic Disease

Author: Arthur J. DeCross, MD, AGAF

A 25-year-old with uncomplicated dyspepsia has a positive serology for H. pylori infection, and is treated appropriately with a standard triple antibiotic therapy protocol. At follow up 3 months later, the patient remains asymptomatic. The most clinically appropriate management at this time is:

A. Repeat serology for evidence of cure
B. No further assessment is indicated
C. Perform endoscopy for mucosal biopsy and special silver stains
D. Perform a C-14 urea breath test no earlier than one month after completing antibiotic therapy
E. Perform a stool antigen assay for H. pylori no earlier than one month after completing antibiotic therapy

The recommended response is B.

In this case, uncomplicated dyspepsia in young people does not require imaging, and there is no obligation to demonstrate eradication of the H. pylori infection in successfully resolved dyspepsia cases. H. pylori eradication should be demonstrated when associated with peptic ulcer disease in particular, as the infection affects the natural history and complications of the disease. When indicated, endoscopy, stool antigen tests, and C-14 urea breath tests are all methods of assessing the presence of viable H. pylori infection after attempted eradication therapy. In contrast, serology may take several years to normalize, and therefore is not an effective tool for short term follow-up of eradication therapy.
Chapter 2: Training in Biliary Tract Diseases and Pancreatic Disorders

DDSEP Chapter 7: Question 2

Hereditary Pancreatitis is caused by mutations in:

A. The pancreatic secretory trypsin inhibitor
B. The Cystic Fibrosis Transmembrane Conductance Regulator (CFTR)
C. Chymotrysinogen
D. **Cationic trypsinogen**
E. Procarboxypeptidase A1

The recommended response is D.

Hereditary pancreatitis is distinct clinical entity inherited as an autosomal dominant trait with incomplete penetrance and caused by mutations in cationic trypsinogen (PRSS1). While specific trypsinogen mutations cause disease, the disease mechanism remains unclear. Classic Cystic Fibrosis (CF) is associated with pancreatic insufficiency and chronic pancreatitis. Minor mutations in CFTR that do not cause classic CF can occasionally cause chronic pancreatitis. Mutations in the pancreatic trypsin inhibitor (SPINK1) may increase the risk of chronic pancreatitis in susceptible individuals (e.g. those with minor CFTR mutations), but do not appear themselves to cause chronic pancreatitis.


Chapter 2: Training in Biliary Tract Diseases and Pancreatic Disorders

DDSEP Chapter 8: Question 2

Which of the following is true regarding gallbladder motility?

A. A gallbladder ejection fraction of less than 50% indicates dyskinesia
B. Patients with gallbladder dysmotility commonly have histologic changes of chronic cholecystitis
C. Contractile function is mediated by secretin
D. Short-chain fatty acids are the most potent stimulators of intestinal CCK release
E. Somatostatin release is associated with accelerated gallbladder emptying time

The recommended response is B.

Biliary dysmotility is usually associated with specific conditions such as gallstones or choledocholithiasis (Hyperlink to Gallbladder Dysmotility section). Biliary dyskinesia and sphincter of Oddi dysfunction are the two main categories of biliary motility disorders. Most patients present with unexplained chronic abdominal pain which has some features of biliary-type pain. The duration of pain, however, is often long-standing and may resemble luminal gastrointestinal disorders such as irritable bowel syndrome or functional dyspepsia. Radionuclide scintigraphy (e.g. HIDA scan) enables the calculation of a gallbladder ejection fraction to objectively assess the severity of dysmotility. Biliary dyskinesia is defined as a gallbladder ejection fraction of less than 35% in the absence of cholelithiasis. Conflicting evidence exists regarding the effectiveness of cholecystectomy for the treatment of biliary dyskinesia. Relief of symptoms has been reported in 50-85% of cases. These patients have histologic changes of chronic cholecystitis in most cases.

Gallbladder contractility is regulated by a neurohumoral axis stimulated by the fat content of ingested food, vagal nerve innervation, and release of CCK. Long-chain fatty acids are the most potent stimulus for the intestinal release of CCK which mediates postprandial gallbladder contraction and sphincter of Oddi relaxation. Somatostatin acts as a physiological inhibitor of gallbladder contraction.

DDSEP Chapter 12: Question 5

Which one of the following is not a poor prognostic sign for pancreatic cancer at presentation:

A. Weight loss
B. Back pain
C. Jaundice
D. Depression
E. Fatigue

The recommended response is C.

The fact that most symptoms are so non-specific can lead to significant delays in diagnosis of pancreatic cancer. Obstructive jaundice from a pancreatic head mass usually prompts a work-up and may pick up early, resectable lesions, while weight loss, depression, and fatigue are usually indicative of high tumor burden and or disseminated disease and back pain may indicate local invasion.

Chapter 3: Training in Cellular and Molecular Physiology

DDSEP Chapter 12: Question 16

Which of the following statements is true regarding the pathogenesis of colorectal cancer?

A. Colorectal cancers that demonstrate chromosomal instability tend to be diploid
B. Colorectal cancers that demonstrate microsatellite instability often have p53 mutations
C. Colorectal cancers from familial adenomatous polyposis patients tend to follow the chromosomal instability pathway
D. Colorectal cancers that demonstrate microsatellite instability often lack mucin within their tumors
E. Colorectal cancers that arise from inflammatory bowel disease patients follow the chromosomal instability pathway

The recommended response is C.

The chromosomal instability pathway is observed in 80-85% of sporadic colorectal cancers, and also FAP cancers. It is mainly characterized by aneuploidy, with loss of heterozygosity at key tumor suppressor gene loci, including APC, chromosome 18q (DCC, SMAD2, SMAD4), and p53. Tumors that demonstrate microsatellite instability have a different genetic pattern that does not involve p53, tend to be right-sided in colon location, poorly differentiated, and more likely to be mucinous. Inflammatory bowel disease-associated colorectal cancers demonstrate a different timing and pattern of molecular alterations than the CIN and MSI pathways, with p53 mutations occurring early but with rare APC and K-RAS mutations.

An 18-year-old man presented for evaluation of constipation. His mother told him that he had problems with stooling from the second half of his first year of life and he required enemas on a regular basis. Toilet training was relatively easy; he never soiled himself. During childhood he had clogged the commode regularly when defecating. During high school his use of enemas declined. Spontaneous bowel movements now occurred only every two weeks.

Physical examination revealed a large fecal mass filling the sigmoid colon and extending up to the umbilicus. Rectal examination showed a large soft fecal impaction in the rectum. Anal sphincter resting tone was normal. Sphincter squeeze was normal.

Following disimpaction with the use of a colon lavage solution, his colon was kept empty for one month by ingestion of one glassful of lavage solution nightly. He then presented for anorectal manometry. This showed an elevated distention threshold for rectal sensation (60 mL) and absence of the rectoanal inhibitory reflex. Barium contrast study showed megarectum and enlargement and lengthening of the sigmoid colon.

The most likely diagnosis in this case is:

A. Hirschsprung’s disease
B. Ultrashort segment Hirschsprung’s disease
C. Idiopathic megarectum
D. Encopresis
E. Pelvic floor dyssynergia

The recommended response is B.

Young adults who present with constipation dating back to infancy need to be evaluated for a congenital problem such as Hirschsprung’s disease (Answer A). Classical Hirschsprung’s disease starts during the first weeks of life and is associated with failure of formation of the more distal ganglia in the enteric nervous system. The aganglionic segment is contracted and the colon proximal to the aganglionic segment (which is innervated normally) is dilated. In addition to the lack of enteric ganglia in the most distal parts of the colon, nerve trunks with increased acetylcholinesterase activity are noted in the lamina propria. Most cases are associated with a variety of mutations of the RET receptor tyrosine kinase gene, the same gene associated with multiple endocrine neoplasia, type 2.
A 24-year-old male presents with severe malnutrition, known small bowel diverticulosis, prior surgeries for intra-abdominal abscesses around perforated diverticula. Pupillary reaction to light is absent, and he has lateral rectus palsy in both eyes. Any physical exercise results in severe skeletal muscle cramps and pain. He has a past history of lactic acidosis and seizures in childhood. The most likely diagnosis is:

A. **Mitochondrial cytopathy**
B. Systemic sclerosis
C. Jejunal diverticulosis (idiopathic) with malnutrition
D. Polymyositis with gastrointestinal involvement
E. Duchenne dystrophy with gastrointestinal involvement

The recommended response is A.

The patient has mitochondrial cytopathy with multiple organs affected including GI, skeletal muscle, external ophthalmoplegia, encephalopathy. This is a genetic disorder; screening tests include searching for acidosis (lactate, pyruvate), skeletal muscle injury, elevated CPK (ragged red fibers on biopsy), and mitochondrial DNA test. Genetic counseling is required.

Chapter 4: Training in Endoscopy

DDSEP Chapter 5: Question 9

For the patient with an UGI bleed and the endoscopic finding of a clean ulcer, the most appropriate management includes:

A. Endoscopic hemostasis with multipolar or heater probe or injection treatment
B. Endoscopic hemostasis with combination therapy
C. Emergent surgery
**D. Medical therapy, early refeeding, same day discharge if stable (medically) and reliable**
E. Medical therapy alone in a monitored setting for three days

The recommended response is D.

For a patient with an UGI bleed and a clean ulcer base at endoscopy, rebleeding is rare -- 3% or less. Endoscopic therapy is not necessary, and the patient may be fed early and considered for same day discharge. A three-day admission is not necessary in stable patients without severe co-morbid conditions. Endoscopic hemostasis is not indicated nor is emergency surgery.


Chapter 4: Training in Endoscopy

DDSEP Chapter 5: Question 12

The two most common causes of severe hematochezia requiring hospitalization are:

A. Angioma and internal hemorrhoids
B. Diverticulosis and internal hemorrhoids
C. Colon cancer and colitis
D. Angioma and diverticulosis
E. Colon polyps and internal hemorrhoids

The recommended response is B.

In a large recent prospective CURE study, diverticulosis and internal hemorrhoids were the two most common colonic causes of severe hematochezia for patients hospitalized with BRB – bright red blood per rectum.


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.

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. 10,000  
E. 50,000

The recommended response is C.

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

Chapter 6: Training in Geriatric Gastroenterology

DDSEP Chapter 4: Question 6

A 40-year-old woman with intermittent heartburn and dysphagia to liquids and solids is seen in consultation. Review of systems reveals she has occasional arthralgias and Raynaud’s phenomenon. The esophageal manometry in this patient would be characterized by:

A. Hypertensive LES with normal esophageal peristalsis
B. Hypertensive EUS with incomplete relaxation
C. **Hypotensive LES and aperistalsis in esophageal body**
D. Increased frequency of transient LES relaxation
E. Normal peristalsis in the distal smooth muscle portion of the esophageal body

The recommended response is C.

In scleroderma of the esophagus, collagenous deposits in the smooth muscle of the LES eventually prevent the smooth muscle from contracting and result in hypotensive LES and aperistalsis in the esophageal body. The aperistalsis in the esophageal body results from both loss of neural elements in the esophageal wall as well as loss of smooth muscle and contractility of the esophageal wall. In scleroderma, the striated muscle function in the proximal esophageal body maintains normal contractility. Thus, esophageal dysphagia resulting from scleroderma can involve both decreased peristalsis in the esophageal body and various degrees of peptic stricture in the LES area due to gastroesophageal acid reflux. The best option is C.

In a patient hospitalized for severe alcoholic hepatitis, which of the following would be an indication to treat with methylprednisolone?

A. Maddrey discriminant function greater than 32
B. Massive variceal hemorrhage
C. Ascites with spontaneous bacterial peritonitis
D. Albumin less than 3.0 g/dl
E. AST/ALT ratio greater than 2

The recommended response is A.

Severe jaundice and coagulopathy with a Maddrey discriminant function greater than 32 (calculated according to the formula $\text{Bilirubin (mg/dl)} + 4.6 \times (\text{Prothrombin time – control})$) identifies patients with high mortality. In these groups steroids appear to improve short term survival. Patients lacking these findings have not been shown to benefit from steroids. Steroid use is relatively contraindicated in the setting of infection or gastrointestinal bleeding.

Chapter 7: Training in Hepatology

DDSEP Chapter 9: Question 8

A husband and wife seek your advice. Last year their daughter died of cirrhosis at the age of thirty. A nephew on the husband’s side also developed cirrhosis as a young adult. The couple’s older son, age 25, has recently been found to have a mild abnormality of serum transaminases with normal bilirubin. A younger son, age 20, has normal liver tests. None of the affected individuals uses alcohol, none is obese, and none is chronically infected with hepatitis B or C. The couple are in their mid-50’s and apparently healthy. You suspect a hereditary liver disease. Possibilities include mutations involving any of the following EXCEPT:

A. HFE  
B. TfR2 (coding for the type 2 transferrin receptor)  
C. ATP7B (coding for hepatic copper transporting p-type ATPase)  
D. SERPINA1 (coding for alpha-1-antitrypsin)  
E. UGT1A1 (coding for bilirubin UDP-glucuronosyl transferase)

The recommended response is E.

UGT1A1 defects, including Gilbert’s syndrome and Criggler-Najjar syndrome, lead to impaired bilirubin conjugation with indirect hyperbilirubinemia. Liver function is otherwise unimpaired and cirrhosis does not occur. HFE and TfR2 defects are associated with classical adult hemochromatosis. ATP7B defects cause Wilson’s disease. SERPINA1 mutations are responsible for alpha-1-antitrypsin deficiency. All can cause cirrhosis in young adults.

Chapter 7: Training in Hepatology

DDSEP Chapter 10: Question 2

Which of the following patients are at increased risk to develop fulminant hepatic failure following acute hepatitis A infection:

A. Women during pregnancy
B. Patients with chronic hepatitis C virus infection
C. Exposure to hepatitis A during infancy
D. Patients with chronic renal failure
E. Patients with previous exposure to HBV who developed anti-HB surface antibody

The recommended response is B.

Patients with chronic HCV and likely also patients with chronic liver disease of other etiologies are at increased risk to develop acute liver failure if they develop acute HAV infection. It is therefore recommended that all patients with chronic HCV be vaccinated against HAV.

Chapter 8: Training in Inflammation and Enteric Infectious Disease

DDSEP Chapter 8: Question 7

After secretion into bile, the majority of primary bile acids are:

A. All excreted in the feces  
B. Absorbed in the small intestine and undergo enterohepatic circulation  
C. Normally deconjugated by bacteria in the small intestine  
D. Absorbed in the conjugated form in the colon to undergo enterohepatic circulation  
E. Used metabolically by bacteria in the colon

The recommended response is B.

Among healthy individuals, the enterohepatic circulation carries nutrient lipids and fat-soluble vitamins to the intestinal mucosa and transports cholesterol into bile, permitting its unchanged elimination from the body in feces. The enterohepatic circulation also transports bile acids from the liver to the small intestine and back to the liver after reabsorption on the terminal ileum (Hyperlink to Enterohepatic Circulation section). The circulating bile acid pool is primarily maintained by efficient ileal reabsorption (>95% per pool cycle). Under stable conditions, the rate of loss of bile acids is matched by an equivalent rate of hepatic synthesis. Unabsorbed bile acids pass into the colon where they are metabolized by bacteria to secondary bile acids including deoxycholic acid, ursodeoxycholic acid, and lithocholic acid. These deconjugated bile acids are absorbed inefficiently in the colon and undergo enterohepatic circulation for hepatic reprocessing and ultimate secretion into bile. Over half of the secondary bile acids in the colon are excreted in feces. The exposure of bile acids to anaerobic bacteria (primarily in the cecum) allows for hydroxyl substituent oxidation to facilitate deconjugation. There are reports that intestinal bile acid dehydroxylating bacteria employ bile acids as metabolic substrates, but this is not the main fate of primary bile acids.

**Chapter 8: Training in Inflammation and Enteric Infectious Disease**

**DDSEP Chapter 8: Question 11**

Which one of the following is true about gallbladder polyps?

A. They are primarily found among patients with symptomatic cholelithiasis  
B. The most common histologic type is inflammatory  
C. The risk of malignant transformation approaches 50% at 15 years  
D. **Elective cholecystectomy is indicated when polyps >18 mm are detected**  
E. Polyps <10 mm in size require cholecystectomy in the presence of asymptomatic cholelithiasis

The recommended response is D.

Gallbladder polyps are often incidentally detected by ultrasonography when performed among asymptomatic patients for other reasons (Hyperlink to Gallbladder polyps section). Prevalence rates are estimated at 1-4% in the general population. The most common histologic type of polyps is cholesterol-based, followed by inflammatory and adenomatous types. Cholesterol polyps are usually less than 10 mm in diameter and are echogenic without acoustic shadowing. Endoscopic ultrasound (EUS) has high accuracy in distinguishing cholesterol polyps from other lesions of the gallbladder wall, including adenomyomatosis. Natural history studies suggest that a less than 10% risk for malignant transformation over 15 years is associated with gallbladder polyps. For most polyps <10 mm in diameter in the absence of symptomatic cholelithiasis, operative treatment is generally not indicated. Surveillance by abdominal ultrasonography at 3-to-6 month intervals to ensure polyp stability has been recommended. Otherwise, the treatment of choice for symptomatic cholelithiasis and polyps <10 mm in diameter is cholecystectomy. Polyps between 10-18 mm in diameter independent of cholelithiasis have a small but appreciable risk for developing into carcinoma. Thus, cholecystectomy is recommended in patients who are acceptable operative candidates. A significant risk for carcinoma does appear to be associated with polyps >18 mm and requires cholecystectomy if possible.


Chapter 8: Training in Inflammation and Enteric Infectious Disease

DDSEP Chapter 6: Question 4

Which one of the following accurately (i.e., is true) reflects the epidemiologic features of IBD?

A. The prevalence and incidence of UC is more common in Asian Americans than in Caucasians
B. Ulcerative colitis has increased in incidence and prevalence at twice the rate of CD
C. In general, there is a higher prevalence of IBD in the southern parts of North America and Europe than in the northern parts
D. North American males are more likely to develop UC than North American Females
E. Jews who were born in North America or who migrated to Israel are at a higher risk of UC than those born in Israel, who are themselves at a higher risk than those born in Africa or Asia

The recommended response is E.

The prevalence of both UC and CD is at least 100 patients per 100,000 general population. Thus, in the United States, the total number of patients with IBD is at least 500,000 people, perhaps more. Men and women in North America are equally as likely to develop inflammatory bowel disease (either CD or UC). Inflammatory bowel disease spares no socioeconomic class. Jews of Ashkenazi descent are 2- to 3-fold more likely to develop IBD, particularly CD, than non-Jews. The country of birth of a person plays a role in the risk of developing inflammatory bowel disease. Jews who were born in North America or who migrated to Israel are at a higher risk of UC than those born in Israel, who are themselves at a higher risk than those born in Africa or Asia. It has been suggested that there is a higher prevalence of IBD in the northern parts of North America and Europe than in the southern parts. Finally, people who smoke are more likely to get CD and less likely to get UC.


Chapter 9: Training in Malignancy

DDSEP Chapter 12: Question 14

A 48 y.o. male presents with *S. bovis* endocarditis. Which of the following is appropriate follow-up?

A. Flexible sigmoidoscopy, age 48  
B. Flexible sigmoidoscopy, age 50  
**C. Colonoscopy, age 48**  
D. Colonoscopy, age 50  
E. No need for follow-up

The recommended response is C.

*Streptococcus bovis* bacteremia has been associated with the presence of an adenoma or cancer in the colon, and a full examination of the colon via colonoscopy should be pursued once the patient is stable from the bacteremia.

Chapter 9: Training in Malignancy

DDSEP Chapter 12: Question 4

Risk factors for pancreatic cancer include all but the following:

A. Hereditary pancreatitis
B. History of pancreatic cancer in 2 first degree relatives over age 60
C. Smoking
D. Alcohol
E. BRCA2 mutation

The recommended response is D.

Alcohol alone is not a significant risk factor for pancreatic cancer. While the risk is increased in sporadic and hereditary pancreatitis with glandular destruction, smoking is the environmental factor with the most impact. Individuals with germline mutations in the breast cancer gene 2 (BRCA2) carry an increased risk of developing pancreatic cancer. Two first degree relatives affected with pancreatic cancer is indicative of Familial Pancreatic Cancer, the risk in this individual is increased regardless of the age of the affected the kindred, although it would be significantly higher, if any relative was less than 50 years upon diagnosis.

Chapter 10: Training in Motility and Functional Illnesses

DDSEP Chapter 4: Question 4

The mechanism by which a peristaltic contraction is propagated in the smooth muscle part of the tubular esophagus is:

A. A myogenic mechanism related to progressively more negative membrane potentials toward the distal esophagus
B. A result of sequential activation of the myenteric plexus by the vagus nerves
C. A result of sequential activation of the musculature by the vagus nerves
D. A function of the balance between inhibitory and excitatory myenteric plexus neurons at each esophageal locus
E. Under complete control of the CNS at the level of the medulla

The recommended response is D.

The sequencing of peristalsis is a result of increasing dominance of inhibitory neurons toward the distal esophagus with the effect of delaying the excitation. By experimentally eliminating the influence of the inhibitory neurons, the result is a simultaneous contraction. Removing the vagal influences (and hence CNS influences) altogether does not change the propagation of peristalsis, though it does prevent deglutition-induced primary esophageal peristalsis.
DDSEP Chapter 4: Question 8

Gastric peristaltic contractions occur normally at a rate of 3 per minute, due to:

A. Vagal excitatory innervation
B. Gastric slow waves
C. Antroduodenal contraction and coordination
D. Duodenal pacemaker activity
E. Rhythmic pyloric contractions

The recommended response is B.

Gastric peristaltic contractions occur at a rate of 3 per minute because the gastric slow waves occur at 3 cpm. Gastric slow waves are also called pacesetter potentials or electrical control activity. These electrical waves set the timing and the propagation velocity of the peristaltic contractions of the stomach. Action potentials and plateau potentials are the electrical correlate of circular muscle contractions. Antroduodenal coordination occurs with the normal emptying of the stomach but does not control the rate of contraction. The pylorus contracts in synchrony with or independently from the antrum and is not responsible for the frequency of gastric contractions. Finally, the fundus is electrically silent and does not have rhythmic electrical pacemaker activity, and the duodenal pacing does not determine the slow wave frequency of the stomach.


All of the following physiological defects have been described in patients with IBS without fibromyalgia except:

A. Increased colonic motility in response to a meal
B. Prolonged propagated contractions in the small and large intestine
C. Increased visceral sensitivity to rectosigmoid balloon distension
D. **Increased somatic sensitivity to tactile and thermal stimuli**
E. Altered CNS activation in response to bowel stimuli as measured by PET and functional MRI scanning

The recommended response is D.

A number of both psychologic and physiologic abnormalities have been described in patients with IBS. Present understanding of the basis of IBS has shifted away from motility being the cause to motility disturbances being secondary phenomena. Visceral hypersensitivity is demonstrated in two-thirds of patients with IBS. Interestingly, somatic hypersensitivity has not been found, at least in some studies.

*Cook IJ, van Eeden A, Collins SM. Patients with irritable bowel syndrome have greater pain tolerance than normal subjects. Gastroenterology 1987;93:727-733.*


DDSEP Chapter 13: Question 7

Which of the following would be most responsive to a recent change in food intake and nutritional status?

A. Triceps skin fold  
B. Mid-arm muscle circumference  
C. Serum albumin  
D. Serum pre-albumin  
E. Serum transferrin

The recommended response is D.

Anthropometric measurements of fat (triceps skin fold) and muscle (mid-arm muscle circumference) mass will change slowly due to alterations in nutritional status. Pre-albumin has a short half-life of 2-3 days and is a good measure of recent changes in food intake and nutritional status. Albumin and transferrin are also affected by nutritional status, but have longer half-lives, 20 days and 7 days respectively.

A 26 year-old woman in her 9th week of pregnancy is admitted because of intractable nausea and vomiting. She is begun on intravenous fluids with 5% dextrose, half normal saline, and supplemental potassium. Three days after admission she is noted to be confused and disoriented with abnormal eye movements. Deficiency of which of the following is most likely the cause of her symptoms?

A. **Thiamine**
B. **Riboflavin**
C. **Vitamin E**
D. **Vitamin B_{12}**
E. **Copper**

The recommended response is A.

Women with hyperemesis gravidarum may develop thiamine deficiency. Administration of glucose containing intravenous solutions without supplemental thiamine can precipitate thiamine deficiency and neurologic symptoms. Vitamins E and B_{12} can cause neurologic abnormalities, but deficiency would not develop acutely. Riboflavin deficiency causes skin rash and oral mucosal abnormalities. Copper deficiency causes anemia and neutropenia.

Chapter 11: Training in Nutrition

DDSEP Chapter 13: Question 20

A 64-year-old male with a 15-year history of type II diabetes mellitus presents with nausea, vomiting, and early satiety associated with meals. You suspect gastroparesis, which you confirm with a delayed gastric emptying scan. All of the following are recommended except:

A. Eat smaller meals  
B. High fiber diet  
C. Increase liquid nutrition  
D. Eat more frequent meals  
E. Reduced fat diet

The recommended response is B.

You would advise the patient to decrease, not increase the fiber in his diet because high fiber foods lead to delayed gastric emptying. High fat foods lead to delayed gastric emptying as well, so you would recommend a low fat diet. Patients should be advised to eat smaller meals more often throughout the day. Liquid emptying is often normal in patients with gastroparesis, so increased liquid nutrition is recommended.

Chapter 12: Training in Pathology

DDSEP Chapter 13: Question 19

A 70 year-old male with a history of celiac disease diagnosed in his forties reports abdominal pain, dark stools, and 20-pound weight loss. He reports complete compliance with a gluten free diet. Tissue transglutaminase is normal. What test would you order next to evaluate his complaints?

A. Repeat tissue transglutaminase  
B. Antigliaden antibodies  
C. Antiendomysial antibodies  
D. EGD with small bowel biopsy  
E. Magnetic Resonance Enterography

The recommended response is E.

The patient’s symptoms (especially the 20-pound weight loss) are worrisome for small bowel lymphoma. This is a rare complication that usually occurs after 20-40 years of disease. An evaluation of his small bowel should be ordered to rule out this complication. The other choices would not be helpful in ruling out small intestinal lymphoma.

Ciclitira PJ. AGA technical review on celiac sprue. Gastroenterology 2001; 120: 1526-1540.  
Chapter 12: Training in Pathology

DDSEP Chapter 1: Question 28

A 55-year-old man presents with a long-standing history of heartburn for 10 years which has been partially treated with over-the-counter antacids and H-2 antagonists. He is otherwise healthy without any past medical or surgical histories. An endoscopic view of the distal esophagus is shown in Figure 19A. The pathology findings of multiple biopsies are shown in Figure 19B. Which of the following is the most appropriate next step in management in addition to treatment with proton pump inhibitors?

A. Inform the patient that he has Barrett’s esophagus without dysplasia and recommend returning in 1 year for routine EGD with surveillance biopsies
B. Inform the patient that he has Barrett’s esophagus with low-grade dysplasia and recommend returning for repeat EGD in 3 months for more aggressive biopsies
C. Inform the patients that he has Barrett’s esophagus with low-grade dysplasia and recommend returning in 1 year for routine EGD with surveillance biopsies
D. Inform the patient that he has Barrett’s esophagus with high-grade dysplasia and recommend immediate evaluation for surgery, radiofrequency ablation, or intensive surveillance
E. Inform the patient that he has Barrett’s esophagus with high-grade dysplasia and recommend immediate surgical referral for Nissen’s fundoplication

The recommended response is D.

The endoscopic figure shows long-segment Barrett’s esophagus (BE) with the pathology confirming the presence of intestinal metaplasia with high-grade dysplasia (HGD). The best management of HGD remains highly controversial. Given the uniformly poor survival reported in patients with esophageal adenocarcinoma, the guideline for endoscopic surveillance programs has been developed. The interval is contemplated on the basis of the degree of dysplasia noted on biopsies. Patients without dysplasia are generally followed up with EGD at intervals of 2-3 years after an initial confirmation at 1 year, whereas those with HGD are considered for intervention. Interobserver variability in the interpretation of HGD mandates expert confirmation. If confirmed, the presence of HGD is considered an appropriate criterion for surgical resection because of the high risk of progression and the likelihood of metachronous adenocarcinoma. On the other hand, two other management strategies have been recently proposed and can be considered in the selected group of patients, especially those with the high risk for surgery. These strategies include intensive endoscopic surveillance (every 3 months for the first year, every 6 months for the second year, and yearly thereafter) and endoscopic ablative therapies.


Figure 19A
Colorectal biopsy can help distinguish acute self limited colitis (ASLC) from IBD. Which feature is unlikely in ASLC?

A. Crypt abscess  
**B. Basal plasmacytosis**  
C. Mucin depletion  
D. Lymphocytes  
E. Plasma cells

The recommended response is B.

Chronic inflammatory changes are seen biopsies with inflammatory bowel disease even early in the course of disease. Specifically, these are distorted crypts, basal or lymphoid aggregates, basal plasmacytosis and mixed acute and chronic inflammatory cells in the lamina propria. With infectious colitis normal crypt architecture is preserved and the inflammation when present is predominantly acute. Crypt abscesses are seen in both as is mucin depletion.

A 6-week-old baby boy is seen because of persistent jaundice, dark urine and acholic stools. Jaundice had been present since shortly after birth, but was not adequately assessed. He had a normal birth weight. No important elements are disclosed by family history. On physical examination, the baby is jaundiced, has both liver and spleen palpable 4 cm below the costal margins. The following laboratory studies are obtained:

- CBC: WBC 13.8 K/mm³; Hb 9.8 g/dl
- PT 13.8 sec; INR 1.2
- AST 280; ALT 350 U/L; GGT 400, Alkaline Phosphatase 550
- Bilirubin: total 9.3/conjugated 6.1 mg/dl

You perform a liver biopsy that shows: canalicular and cellular bile stasis and bile duct proliferation and portal fibrosis.” What diagnosis is consistent with this clinical picture?

A. Alagille’s syndrome
B. Biliary atresia
C. Idiopathic neonatal hepatitis
D. Neonatal hemochromatosis
E. Wilson’s disease

The recommended response is B.

Alagille’s syndrome occurs in approximately 1/100,000 live births, There is often a family history for related clinical features in at least 20% of cases. Classic pathology findings include bile duct paucity. Idiopathic neonatal hepatitis represents the most common diagnosis in infants with neonatal cholestasis. Often children have low birth weight and jaundice present within the first week. Acholic stools are uncommon. Often see giant cell transformation and extramedullary hematopoiesis on liver biopsy.

Neonatal iron storage disease (or “hemochromatosis”) presents shortly after birth with fulminant hepatic failure and is, therefore, not in this differential diagnosis. Wilson’s disease, a rare autosomal recessive disease resulting in accumulation of copper in several organs, has not been reported in children younger than 4 years of age and is, thus, not consistent with this presentation. Biliary atresia accounts for 30% of cases of neonatal cholestasis. It is characterized by prolonged cholestatic jaundice in the newborn period, then hepatosplenomegaly. Patients late in the process present with abdominal distension, ascites and failure to thrive. The classic histopathology changes include variable canalicular and cellular bile stasis and bile duct proliferation.
Chapter 13: Training in Pediatric Gastroenterology

DDSEP Chapter 14: Question 19

Which of the following statements is not true when dealing with IBD in children?

A. Its incidence appears to be increasing worldwide  
B. Children with UC tend to have a higher likelihood of pancolitis at presentation  
C. Steroid sparing strategies are important for the treatment of pediatric IBD  
D. Isolated small bowel disease is more common in children than adults  
E. Growth failure is more common in UC then CD

The recommended response is E.

The only false statement is reported in option E. Growth failure is more common in CD then UC. In patients with significant growth failure carrying a diagnosis of UC must be investigated for evidence of Crohn’s disease most noticeable in the more proximal small bowel. The video capsule endoscopy may be a very useful diagnostic tool in this case.
Chapter 13: Training in Pediatric Gastroenterology

DDSEP Chapter 14: Question 20

You examine a 1-month-old-boy who is brought to your attention for a distended abdomen. The parents tell you that he was born at full term by a normal delivery, he is their first-born, and was breast-fed from birth. He is reported to have had passage of meconium on day 2 of life. He did well and was sent home on day 3, where he continued to thrive while breast-fed, but the frequency of his bowel movements decreased, and currently he has 1 bowel movement of normal stools, passed with evident straining, every 2 to 3 days.

At physical examination, he is well nourished and alert. Abdomen is very distended and full: palpation does not elicit tenderness. No masses are evident. Perianal inspection is normal. Rectal exam: your finger enters with difficulty through a narrow anus, and you do not touch stools; at retraction, loose, yellowish stools are passed explosively. A plain x-ray of the abdomen confirms the distention and shows dilated loops of colon, but no stool accumulation.

What is the first priority in caring for this patient?

A. Rule out the diagnosis of Hirschsprung’s disease
B. Provide him with a stool softener
C. Administer an enema
D. Change the diet to soy milk to eliminate cow’s milk protein introduced via breast milk
E. No action is needed, as this baby obviously is healthy

The recommended response is A.

Hirschsprung’s disease is a diagnostic possibility and needs to be ruled out as soon as possible. In fact, even though this case lacks the most obvious landmarks of the condition such as the delayed passage of meconium, this presentation is not unusual for patients who have a short segment. The physical examination findings are highly supportive of this diagnosis. Using stool softeners or enemas is not indicated, as this patient does not have hard stools (as seen both from history and at the physical examination), and they would be contraindicated in suspected HD. No signs consistent with Cow’s Milk Protein Allergy (CMPA) are reported, and discontinuing breast-feeding would be a poor option. Finally, although this baby is well nourished, he is not healthy, as the abdomen is very distended and diagnostic steps need to be undertaken. Testing may involve first a barium enema and/or an anorectal manometry, followed—in cases still consistent with HD—by the definitive diagnostic test: the rectal biopsy.
Chapter 14: Training in Radiology

DDSEP Chapter 1: Question 12

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 H$_2$-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 moxifloxacin 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 hemicolectomy
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 hemicolectomy 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. Schatzki 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
**Chapter 16: Training in Surgery**

**DDSEP Chapter 1: Question 24**

Which of the following statements is true regarding the role of antireflux surgery in Barrett’s esophagus?

A. **Antireflux surgery will control symptoms of GERD**
B. **Antireflux surgery is more effective than medical therapy for the treatment of Barrett’s esophagus**
C. **Antireflux surgery causes regression of Barrett’s epithelium**
D. **Antireflux surgery decreases the risk of esophageal cancer**
E. **Antireflux surgery eliminates the need for surveillance in Barrett’s patients**

The recommended response is A.

Antireflux surgery effectively alleviates GERD symptoms in Barrett’s esophagus patients. While studies consistently show the development of squamous islands after antireflux surgery, complete regression of Barrett’s epithelium is uncommon and may well represent “pseudoregression” due to surgical repositioning of the esophagus. Some surgical enthusiasts suggest that antireflux surgery decreases the subsequent risk of developing esophageal cancer. However, a large Swedish cohort study found that the risk of developing adenocarcinoma of the esophagus remains elevated after antireflux surgery in GERD patients (Barrett’s status unknown) and was no different for GERD patients not undergoing antireflux surgery. A recent meta-analysis found a decrease in the risk of adenocarcinoma after antireflux surgery for Barrett’s esophagus. There are no data to support an advantage of antireflux surgery compared to medical therapy for symptoms control in Barrett’s esophagus patients. Thus, while surgery provides an excellent means of symptom control in Barrett’s patients, it does not appear to influence the natural history of Barrett’s esophagus and as such patients remain in need of surveillance endoscopy and biopsy.


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

DDSEP Chapter 4: Question 26

A 65-year-old woman presents with long standing history of diarrhea and urgency. She reluctantly admits to being incontinent for semi-formed stool on several occasions in the past 6 months. She has 3 vaginal deliveries with episiotomies but no muscular tear. Anal endosonography is performed and shows in Figure 13. Which of the following is the finding from anorectal function testing of this patient?

A. Decreased resting pressure
B. Decreased squeezing pressure
C. Decreased rectal compliance
D. Failed expulsion of balloon
E. Absence of rectoanal inhibitory reflex

The recommended response is B.

An endosonographic figure shows a large, wedge-shaped anterior defect of external anal sphincter, characteristic for childbirth injury from episiotomies. Fecal incontinence is defined as either the involuntary passage or the inability to control the discharge of fecal matter through the anus. Three clinical subtypes have been described, including passive incontinence, urge incontinence, and fecal seepage. Detailed history and physical examination play an important role in evaluation of patients with fecal incontinence. Several modalities have been developed in order to identify the mechanism and etiology of fecal incontinence. The American Gastroenterological Association (AGA) has published the technical review on anorectal testing techniques providing the detailed descriptions of the process and clinical significance of these tests, including anorectal manometry, anal endosonography, MRI, and pudendal nerve terminal motor latency.

Anal endosonography is a very useful technique for demonstrating anal sphincter anatomic features, both the thickness and structural integrity. Normal findings have been described, and sphincter defects due to scarring, loss of muscle tissue, and other local pathology have been demonstrated to correlate with surgical findings. This test is usually performed with a 7.5-MHz radial endosonographic probe, providing 360-degree view. Higher-frequency probes (10-15 MHz) are now being used for better delineation of the sphincter complex. A high concordance rate was established for identifying sphincter defects in the studies comparing anal endosonography with the other modalities, including EMG mapping, MRI, and anorectal function test. The advantage is that the test is simple, inexpensive, widely available, and less painful than needle insertion for EMG. However, the identification of abnormalities is dependent on the training and experience of the operator, and the test is subject to interobserver variability. In addition, anatomic variations with aging and gender differences may influence the interpretations.

In patients with external anal sphincter defect, the squeezing pressure during voluntary contraction is often reduced, whereas decreased resting pressure can be detected in patients with internal anal sphincter defect. Decrease rectal compliance is often seen in conditions associated with fibrosis (postradiation) or inflammatory (proctitis). Failure to expel the balloon may indicate underlying pelvic floor dysfunction or dyssynergic defecation, while the absence of rectoanal inhibitory reflex is pathognomonic for anorectal aganglionosis (Hirschsprung’s disease). Both conditions usually result in constipation rather than fecal incontinence.


Figure 13