ELSEVIER
SAUNDERS

Prim Care Clin Office Pract
33 (2006) 659–684

PRIMARY CARE:
CLINICS IN
OFFICE PRACTICE

Acute Abdominal Pain

Mark H. Flasar, MD[a,b,*], Raymond Cross, MD[a,b], Eric Goldberg, MD[a,b]

[a]*Division of Gastroenterology and Hepatology, Department of Medicine, University of Maryland Medical Center, 22 South Greene Street, Baltimore, MD 21201, USA*
[b]*Veterans Affairs, Maryland Health Care System, 10 North Greene Street, Baltimore, MD 21201, USA*

Abdominal pain is a complaint seen commonly in the outpatient setting. Many etiologies, both acute and chronic, can be evaluated on an outpatient basis. However, several causes of abdominal pain necessitate prompt, focused, and structured evaluation, given associated morbidity and mortality. The differential diagnosis of a patient presenting with acute abdominal pain is exhaustive, necessitating that the physician understand not only the underlying pathophysiology of the pain, but also the clinical presentation, course, and initial management of more harmful causes. A focused history, physical examination, and adjunctive testing strategy will allow for those patients with concerning presentations to be identified, initially managed, and appropriately referred for continued care.

Abdominal pain is an extremely common complaint in all settings of medical practice. In primary care practices in 2002, abdominal pain was a complaint in more than 13.5 million patient visits [1]. Oftentimes, patients with severe abdominal pain will self-triage to an emergency department, hospital, or contact emergency medical services. However, some patients with potentially life-threatening abdominal catastrophes will initially present to the primary care physician. Ease of access to the primary care physician and preexisting appointments occurring shortly after the pain onset offer some explanation.

Abdominal pain may often be a symptom of a disease process with a benign course, but it may also herald a severe, life-threatening condition that demands prompt recognition and management. The purpose of this review is to provide the practitioner with a framework for understanding

* Corresponding author.
E-mail address: mflasar@medicine.umaryland.edu (M.H. Flasar).

doi:10.1016/j.pop.2006.06.004

abdominal pain, so that they may determine those patients that need a more expedited evaluation. Furthermore, the pathophysiologic mechanisms underlying abdominal pain will be reviewed. A general approach to the patient with acute abdominal pain will be outlined, and several gastrointestinal etiologies of abdominal pain will be considered in detail, focusing on the most severe and commonly encountered.

A general understanding of abdominal anatomy, physiology, and pathophysiology is vital when formulating a differential diagnosis for abdominal pain. In addition, it is important to understand how abdominal pain is generated and perceived by the patient. The abdominal viscera are innervated with nocioceptive afferents within the mesentery, on peritoneal surfaces, and within the mucosa and muscularis of hollow organs. These afferents respond to both mechanical and chemical stimuli producing dull, crampy, poorly localized pain sensations. The principal mechanical stimulus is stretch, while a variety of chemical stimuli including substance P, serotonin, prostaglandins, and hydrogen ions are perceived as noxious by visceral chemoreceptors [2]. Abdominal pain occurs in three broad patterns: visceral, parietal, and referred. Visceral nocioception typically involves stretch and distension of the abdominal organs, although torsion, and contraction also contribute. The pain stimulus is carried on slow-conducting C-fibers. Patients often describe pain of visceral origin as a dull ache. The location of visceral pain is often midline, because visceral innervation of abdominal organs is typically bilateral. Pain location corresponds to those dermatomes that match the innervation of the injured organ [2]. Generally, visceral pain from organs proximal to the Ligament of Treitz, including the hepatobiliary organs and spleen, is felt in the epigastrum. Visceral pain from abdominal organs between the Ligament of Treitz and the hepatic flexure of the colon is felt in the periumbilical region. Visceral pain generated from organs distal to the hepatic flexure is perceived in the midline lower abdomen.

Parietal pain is typically sharp and well localized, resulting from the direct irritation of the peritoneal lining. Parietal peritoneal afferents are A-δ fibers, with a rapid conduction velocity and result in sharp pain sensation similar to skin and muscle pain. Because parietal innervation is unilateral, lateralization of pain occurs [2]. Referred pain occurs when visceral afferents carrying stimuli from a diseased organ enter the spinal cord at the same level as somatic afferents from a remote anatomic location. It is typically well localized. A single diseased organ may produce all three types of pain. For example, when a patient develops cholecystitis, gallbladder inflammation is initially experienced as a visceral pain in the epigastric region. Eventually, the inflammation extends to the parietal peritoneum, and the patient will experience parietal pain that lateralizes to the right upper quadrant. Gallbladder pain may also refer to the right shoulder.

Awareness of the anatomy and innervation of the abdominal viscera allows one to formulate a differential diagnosis of abdominal pain based on the location and distribution of the pain (Table 1). However, there is

significant overlap between abdominal pain presentations. Furthermore, disease processes from organs outside of the abdominal cavity can present with abdominal pain. To considerably narrow the differential diagnosis, it is crucial to approach each patient in a systematic, logical, and deliberate manner. Having already elicited a chief complaint of abdominal pain, this begins with the patient's history and physical examination.

The history should not only include a thorough assessment of the present condition, but also a detailed assessment of underlying medical problems, medications, family history, substance abuse history, recent travel history and occupational history. Important clues to the etiology of the pain should be determined in the patient's history by inquiring about the nature of the pain, which includes its quality, location, rapidity of onset, chronicity, radiation, intensity, exacerbating factors, alleviating factors, and associated symptoms. Chronicity of symptoms is an important factor in evaluating abdominal pain. Generally, patients with chronic symptoms can be evaluated on an outpatient basis, as the process underlying the pain rarely requires acute intervention. On the other hand, patients with new-onset of symptoms are more likely to have a significant disease process that can bring them harm in the hours to days ahead. An exception to this is an acute worsening of chronic or intermittent abdominal pain symptoms. Examples include acute mesenteric ischemia superimposed on a history of chronic intestinal angina or development of acute cholecystitis in a patient with a history of biliary colic. Additionally, identifying high-risk patients such as the elderly, pregnant, and those with immunodeficiency syndromes proves invaluable in triaging patients.

Following a thorough history, a focused physical examination should be performed. The generation of a differential diagnosis will help the practitioner tailor the examination, whose purpose is to provide confirmatory or contradictory data for each disease process on their differential. Although centered on the abdomen, the examination should also focus on extraabdominal organ systems when indicated. For example, a patient with suspected mesenteric ischemia should have a cardiovascular examination assessing for arrhythmias and evidence of atherosclerotic disease.

Overall, the abdominal examination should begin with general observation of the patient, followed by abdominal inspection. A patient with peritonitis often lies completely still as movement further irritates the peritoneum. Their abdomen will be visually rigid. On the other hand, a patient with renal colic may writhe in pain, may not be able to be consoled to comfort, and have a nonrigid abdomen. Once initial observation is complete, a review of the vital signs is imperative. Any significant abnormality of vital signs should prompt the physician to consider a more life threatening process. Auscultation of the abdomen determines whether intestinal peristalsis is appropriate and whether any abdominal bruits are present. Next, palpation of the abdomen should be performed to distinguish pain, a subjective sensation, from tenderness, which is an objective finding.

Table 1
Diagnosis of abdominal pain based on location and distribution

Right upper quadrant	Middle upper abdomen	Left upper quadrant
Peptic ulcer disease	Peptic ulcer disease	Peptic ulcer disease
Biliary disease	Pancreatitic disease	Splenic disease
Biliary colic	Pancreatitis	Splenic rupture
Choledocholithiasis	Pancreatic neoplasm	Splenic infarct
Cholecystitis	Biliary disease	Pancreatic disease
Cholangitis	Biliary colic	Pancreatitis
Liver disease	Choledocholithiasis	Pancreatic neoplasm
Hepatitis	Cholecystitis	Lung disease
Neoplasm	Cholangitis	Pneumonia
Abscess	Esophageal disease	Subphrenic abscess
Congestive hepatopathy	Reflux esophagitis	Pulmonary embolism
Lung disease	Infectious esophagitis	Pneumothorax
Pneumonia	Pill esophagitis	Kidney disease
Subphrenic abscess	Cardiac disease	Pyelonephritis
Pulmonary embolism	Myocardial ischemia	Perinephric abscess
Pneumothorax	or infarction	Nephrolithiasis
Abdominal wall	Pericarditis	
Herpes zoster	AAA rupture/aortic	
Muscular Stain	dissection	
Kidney disease	Mesenteric ischemia	
Pyelonephritis		
Perinephric abscess		
Nephrolithiasis		
Colonic causes		
Colitis		
Right sided diverticulitis		
	Periumbilical	
	Appendicitis (early)	
	Small bowel obstruction	
	Gastroenteritis	
	Mesenteric ischemia	
	AAA rupture	
	Aortic dissection	
Right lower quadrant	Suprapubic	Left lower quadrant
Appendicitis	Inflammatory bowel disease	Inflammatory bowel disease
Inflammatory bowel disease	OB-GYN causes	OB-GYN causes
OB-GYN causes	Ovarian tumor	Ovarian tumor
Ovarian tumor	Ovarian torsion	Ovarian torsion
Ovarian torsion	Ectopic pregnancy	Ectopic pregnancy
Ectopic pregnancy	PID	PID
Pelvic inflammatory disease (PID)	Dysmenorrhea	Kidney disease
Kidney disease	Colonic disease	Pyelonephritis
Pyelonephritis	Proctocolitis	Perinephric abscess
Perinephric abscess	Diverticulitis	Nephrolithiasis
Nephrolithiasis	Urinary tract disease	Intestinal disease
Intestinal disease	Cystitis	Sigmoid diverticulitis
Right sided diverticulitis	Nephrolithiasis	Ileocolitis
Ileocolitis	Prostatitis	Gastroenteritis
Gastroenteritis		Hernia
Hernia		

Table 1 (*continued*)

Diffuse
Gastroenteritis
Bowel obstruction
Peritonitis
Mesenteric ischemia
Inflammatory bowel disease
Diabetic ketoacidosis
Porphyria
Uremia
Hypercalcemia
Sickle cell crisis
Vasculitis
Heavy metal intoxication
Opiate wihdrawal
Familial mediterranean fever
Hereditary angioedema

When performing palpation, the location of tenderness should be used to narrow the differential diagnosis. Additionally, the presence of guarding or rebound tenderness should be noted, as these findings imply peritoneal irritation. Furthermore, palpation can determine the presence of visceral enlargement, masses, or fluid. It is often useful to begin the abdominal examination by palpating distant from, then working toward the site of pain, palpating with the fingertips. It is helpful to keep the patient's hips and knees in a flexed position during a supine examination to help relax the abdominal musculature. Techniques such as patient distraction during examination or palpation during auscultation with the stethoscope head may help discriminate functional from organic pain.

The importance of a properly executed history and physical examination cannot be underestimated. Although the sensitivity and specificity of a history and physical may not match that of an abdominal CT scan, there is no risk, minimal time required, and essentially no cost. In fact, one observational study revealed that, based on history and physical alone, physicians were able to correctly differentiate between organic and nonorganic causes of abdominal pain nearly 80% of the time [3]. Furthermore, historic features such as pain location have been shown in prospective investigation to be specific for certain disease states [4]. Once the history and physical is completed, the practitioner will usually be armed with sufficient information to sharply narrow the differential diagnosis in the majority of patients presenting with abdominal pain.

The detection of the warning signs of a life-threatening process in a patient with abdominal pain is often up to the primary physician long before the emergency room physician, surgeon, gastroenterologist, or other specialist encounters the patient. Certain historic and examination findings should raise red flags that a life-threatening abdominal process is present, and

prompt early triage to an emergency department or inpatient hospital bed (Table 2). Red flags from the history include fever, vomiting, inability to move the bowels (obstipation), syncope, concomitant chest or back pain, respiratory distress, excessive acute vaginal bleeding, and overt gastrointestinal bleeding. Red flags from the physical examination include any significant abnormality of the vital signs, mental status changes, involuntary guarding, rebound abdominal tenderness, complete absence of bowel sounds, and pain out of proportion to abdominal tenderness on physical examination.

In patients without signs or symptoms of an acute abdominal catastrophe, an acute expedited outpatient evaluation should be performed. Selection of initial laboratory and imaging studies to evaluate abdominal pain should be guided by the differential diagnoses generated from the primary evaluation. Historically, plain abdominal radiographs have been the initial imaging test of choice. They can be obtained rapidly at a relatively low cost, and are often available to primary practitioners in the office. However, with the evolution of more sensitive and specific modalities such as CT and ultrasound, the utility of the plain abdominal series has been debated. Nonetheless, the authors feel that plain films should still be the initial imaging modality in patients with suspected visceral perforation or obstruction.

The abdominal plain film series should include supine and upright abdominal films in conjunction with an upright chest (or lateral decubitus abdominal) film. Plain abdominal imaging has been estimated to be diagnostic in up to 60% of cases of suspected small bowel obstruction [5], although sensitivity is more limited in cases of low-grade obstruction [6]. The location, volume, and distribution of intraluminal air, presence and distribution of air–fluid levels, and luminal diameter can often be helpful in differentiating between an obstructive and nonobstructive process, such as partial or complete bowel obstruction, ileus, or intestinal pseudoobstruction. Unfortunately, overlap in the radiographic appearance of obstructive and nonobstructive processes limits the sensitivity and specificity of plain films in this setting.

The ability of plain films to detect free air depends on the volume of free air within the peritoneal cavity. For the detection of large volumes, as would be expected with a perforated viscus, sensitivity of plain films is reported to be as high as 100%. Sensitivity is maximized if the patient is placed in the upright or decubitus position for 5 to 10 minutes before obtaining an upright chest or lateral decubitus film. This allows small volumes of air to redistribute to and collect within nondependent areas. Detection of volumes as small as 1 to 2 cc of air has been reported using this method [6,7].

CT is an imaging tool that is sensitive for the diagnosis of many etiologies of abdominal pain. Because of its widespread availability, CT is often accessible to primary care providers in the outpatient setting for same-day interpretation. With newer rapid helical scanning methods, advances in intravenous and oral contrast agents, three-dimensional reformatting, and

Table 2
Red flags in abdominal pain

History	Physical exam	Labs	Radiography
* Inability to maintain po intake	* Pathologic changes in vital signs	* Renal failure	* Abdominal free air
* Projectile vomiting	* Bloody, maroon, or melanotic stool	* Metabolic acidosis	* Gallbladder wall thickening
* Overt gastrointestinal blood loss	* Hernia (incarcerated and tender)	* Leukocytosis	* Pericholecystic fluid
* Syncope	* Hypoxia	* Elevated transaminases	* Dilated biliary tree
* Pregnancy	* Cyanosis	* Elevated alkaline phosphatase and bilirubin	* Bowel obstruction
* Recent surgery or endoscopic procedure	* Altered mentation	* Anemia or polycythemia	* Dilated small bowel loops ± air fluid levels
* Fever	* Jaundice	* Hyperlipasemia/hyperamylasemia	* Intraabdominal abscess
* Caustic or foreign body ingestion	* Peritoneal signs	* Hyperglycemia/hypoglycemia	* Bowel wall thickening
	* Abdominal pain out of proportion to examination		* Air in the portal venous system
			* Pneumatosis intestinalis

other advanced software capabilities, CT has become the initial imaging modality of choice for the evaluation of most presentations of acute abdominal pain. For example, CT diagnoses acute appendicitis with a reported sensitivity and specificity as high as 98% and 97%, respectively [8]. In fact, the superior diagnostic capability of CT is rendering plain films obsolete. Even in situations where plain films have proven diagnostic accuracy, such as perforated viscus or small bowel obstruction, many physicians now opt for CT as the initial imaging study. Computed tomography has proven to be more sensitive and specific for nearly all etiologies of acute abdominal pain [9–11].

In patients with abdominal pain suspected to be from a hepatobiliary source, abdominal ultrasonography should be considered as an initial imaging option. It is accurate for the detection of gallstones and dilation of the biliary tree. However, ultrasound is less sensitive for stones in the common bile duct. Although MRI can be highly accurate in the diagnosis of acute abdominal pain, high cost and lack of immediate availability limit its use in the primary care setting.

Following clinical evaluation of patients with abdominal pain, the primary physician must appropriately triage the patient. In addition to red flags revealed by the history and physical examination, there are laboratory and radiographic ''red flags'' that should alert the physician of a potentially more serious cause of the abdominal pain (Table 2).

Although cardiac, pulmonary, urologic, musculoskeletal, and gynecologic causes of abdominal pain will not be specifically addressed in this review, it is important to keep these extraabdominal disease processes in the differential diagnosis of abdominal pain. Red flags that a life-threatening extraabdominal cause of abdominal pain is present include chest pain, back pain, shortness of breath, vaginal bleeding, and hemodynamic instability. Finally, there is a spectrum of systemic medical disorders, such as adrenal insufficiency, diabetic ketoacidosis, porphyria, and sickle cell pain crisis, that can present with abdominal pain. Evidence of these disorders in the past medical history, medications, or physical examination should prompt their consideration as the cause of the patient's pain. Although a detailed discussion of all of the potential etiologies of acute abdominal pain is beyond the scope of this review, there are some etiologies that merit a more detailed discussion. What follows is an overview of those gastrointestinal etiologies of abdominal pain that can often be seen in adults in the outpatient setting, with a focus on those etiologies prone to more serious or life-threatening complications.

Hepatopancreatobiliary

Biliary colic

When patients with cholelithiasis present with abdominal pain, they will most commonly have biliary colic as the cause [12]. Biliary colic occurs when

then one or more gallstones transiently occlude the cystic duct. Tonic cystic duct spasm ensues, causing pain. Patients typically experience visceral epigastric or right upper quadrant abdominal pain, often with radiation to the right shoulder or scapula. The pain is sometimes postprandial, but often there is no trigger; it is not uncommon for patients to have nocturnal pain. The term biliary colic is a misnomer, as the pain is not colicky in nature. The classic time course of pain from biliary colic is one that builds over 15 to 60 minutes, lasting up to several hours before slowly dissipating. Physical examination is typically benign, with tenderness sometimes elicited in the epigastrum or right upper quadrant of the abdomen. Patients suspected of having biliary colic should have further evaluation with liver function tests and abdominal ultrasonography. Transaminases, bilirubin, and alkaline phosphatase are usually normal, and an ultrasound can verify the presence of gallstones. Although biliary colic usually resolves without sequelae, it identifies those patients whom are at higher risk for complications of gallstone disease, such as pancreatitis or cholangitis. Therefore, referral to a general surgeon for elective cholecystectomy is recommended.

Cholecystitis

More than 90% of cases of acute cholecystitis are caused by gallstones. The remainder of cases are termed acalculous cholecystitis, typically occur in critically ill patients, and are rarely seen in the outpatient setting [13]. Acute cholecystitis is most commonly caused by the obstruction of the cystic duct by the offending gallstone. Prolonged obstruction of the cystic duct (>6 hours) impairs gallbladder emptying, leading to inflammation of the gallbladder mucosa. Secondary bacterial infection of the gallbladder may ensue, leading to possible empyema, gallbladder necrosis, and perforation. Acute cholecystitis results in gallbladder perforation in up to 12% of cases, with a subsequent mortality rate of 20% [7]. Emphysematous cholecystitis, characterized by air in the wall of the gallbladder, is most often seen in patients with diabetes mellitus. Approximately 75% of patients who develop acute cholecystitis have a prior history of biliary colic [14]. The pain is similar to that of biliary colic, but with a longer duration. Pain lasting longer than 6 hours signifies cholecystitis rather than biliary colic. As acute gallbladder inflammation irritates the parietal peritoneum, the pain may shift from the epigastrum to the right upper quadrant. The physical examination of patients with acute cholecystitis reveals right upper quadrant tenderness. An inspiratory arrest during deep right upper quadrant palpation is referred to as Murphy's sign.

Laboratory abnormalities include leukocytosis with a predominance of neutrophils, and elevation of alkaline phosphatase, and transaminases. Hyperbilirubinemia generally does not occur with acute cholecystitis owing to the unimpeded flow of bile through the common bile duct. An exception is Mirizzi's syndrome, where a large stone in the cystic duct compresses or

erodes into the common hepatic duct, resulting in variable degrees of biliary obstruction. Right upper quadrant ultrasonography is the test of choice to diagnose acute cholecystitis, with reported sensitivity, specificity, and accuracy approaching 95% [7]. Common findings include cholelithiasis, gallbladder wall thickening, pericholecystic fluid, and a sonographic Murphy's sign. The latter finding occurs when ultrasound transducer pressure on the gallbladder results in tenderness. The finding of cholelithiasis and a positive sonographic Murphy's sign has a positive predictive value of 92% for acute cholecystitis. Conversely, when both of these findings are absent, the negative predictive value is 95% [7]. Radionuclude cholescintigraphy scans, such as the HIDA scan, can be used to confirm the diagnosis of acute cholecystitis when ultrasound findings are equivocal. The sensitivity, specificity, and positive predictive value for acute cholecystitis are 95%, 99%, and 97%, respectively [15].

Patients with suspected acute cholecystitis should have an expedited evaluation with early surgical consultation. Cholecystectomy within 24 to 48 hours of presentation has been shown to reduce mortality and shorten length of hospital stay, compared with surgery performed after weeks of conservative management aimed at "cooling off" the gallbladder [16–18]. The benefits of early cholecystectomy have been prospectively validated for the laparoscopic approach as well [19–23].

Cholangitis

Ascending cholangitis is a potentially lethal entity that occurs when the bile duct become obstructed. Once bile flow is impeded, superinfection of the stagnant bile occurs. As pus builds up under pressure, the infection can rapidly ascend into the liver and spread into the blood stream. Common pathogens include *Escherichia coli*, *Klebsiella* species, *Bacteroides*, *Enterococcus*, and other enteric pathogens [24]. The most common cause of obstruction in the United States is choledocholithiasis, accounting for approximately 85% of cases. Benign biliary strictures, choledochal cysts, biliary parasites, and neoplasms are less common causes of cholangitis [25]. The classic presentation of cholangitis is fever, jaundice, and right upper quadrant pain. These findings are collectively referred to as Charcot's triad, which has a reported sensitivity for cholangitis as high as 75% [26]. If the obstruction is not relieved, mental obtundation and shock can occur. The combination of Charcot's triad with these findings is known as Reynold's pentad, which is associated with a higher morbidity and mortality rate [13].

Laboratory findings include leukocytosis with a predominance of neutrophils, elevated alkaline phosphatase, and elevation of the transaminases. An elevation of pancreatic enzymes can be seen in about one third of patients, especially with concomitant gallstone pancreatitis [27]. As the pathophysiology of this disorder involves common bile duct obstruction, conjugated hyperbilirubinemia is invariably present.

The diagnosis of cholangitis is often made clinically, and should be confirmed with cholangiography. Although ultrasonography may suggest the presence of biliary obstruction, its sensitivity for choledocholithiasis is poor [7]. Therefore, when the clinical diagnosis of ascending cholangitis is suspected, patients should undergo cholangiography even in the setting of an unremarkable right upper quadrant ultrasound.

Patients suspected of having acute cholangitis should be referred quickly to an emergency department or hospitalized, as the clinical course can be rapidly progressive and fatal if left untreated. Patients with suspected cholangitis should have blood cultures drawn at presentation, so that therapy can be directed toward the offending pathogen. The definitive therapy for cholangitis is decompression of the obstructed biliary system. Endoscopic retrograde cholangiopancreatography (ERCP) is the diagnostic and therapeutic procedure of choice, and is successful in relieving the obstruction in more than 95% of cases [15]. This is typically accomplished by stone extraction or placement of a stent into the common bile duct. In cases where therapeutic ERCP is not available or is unsuccessful, options include percutaneous transhepatic cholangiography or surgical decompression. If choledocholithiasis is the cause of ascending cholangitis, patients should undergo elective cholecystectomy once the infection resolves.

Acute pancreatitis

Acute pancreatitis is an inflammatory disease of the pancreas that not only may cause significant morbidity but also carries a case fatality rate of 5% to 9% [28]. Gallstones and alcohol account for more than 80% of cases in the United States [15]. Less common causes of pancreatitis include medications, trauma, hypercalcemia, severe hypertriglyceridemia (>1000 mg/dL), malignancy, sphincter of Oddi dysfunction, infections, iatrogenic (ERCP), and congenital abnormalities of the pancreas such as pancreas divisum. The remainder are termed idiopathic, although as many as 75% of cases of idiopathic pancreatitis may actually be due to biliary sludge or microlithiasis [29]. Regardless of the etiology, diffuse pancreatic inflammation and edema develop. In severe cases, necrosis of pancreatic and peripancreatic tissue occurs, and multiorgan failure may ensue. Necrotizing pancreatitis occurs in up to 25% of patients with pancreatitis and has a mortality rate of 15% to 20% [28,30]. Patients typically present with the acute onset of abdominal pain, nausea, and vomiting. The pain is steady and usually in the epigastrium, although patients may also note discomfort in the right or left upper quadrants of the abdomen. Pain is classically described as a boring sensation that radiates into the back. Patients are often unable to get comfortable when lying supine, and they will lean forward in an attempt to find relief. Because of marked fluid shifts, patients may become severely volume depleted. Resultant tachycardia and hypotension with orthostatic changes may develop. Other vital sign abnormalities include low-grade fever and

tachypnea. The latter is a poor prognostic sign, and may herald the development of sepsis or acute respiratory distress syndrome.

The abdominal examination may reveal distension and diminished or absent bowel sounds secondary to the development of a paralytic ileus. With palpation, focal tenderness in the epigastrum is seen, although the abdomen may also be diffusely tender. With more severe cases, voluntary guarding and rebound tenderness may also be appreciated. Signs of hemorrhagic pancreatitis such as Gray Turner's sign (flank ecchymosis), Cullen's sign (periumbilical ecchymosis), or Fox's sign (inguinal ecchymosis) are seen in less than 1% of cases. When acute pancreatitis is suspected clinically, levels of serum amylase or lipase should be determined. In the setting of suspected acute pancreatitis, levels greater than three times the normal values have a high specificity for acute pancreatitis. Serum lipase remains elevated for longer durations than serum amylase and is more specific for acute pancreatitis [31]. It is important to note that the magnitude of serum amylase and lipase elevation does not correlate well with disease severity. Because of the marked intravascular volume depletion secondary to third spacing, hemoconcentration often occurs in acute pancreatitis. Hematocrit levels higher than 44% are associated with a worse prognosis, indicating potentially dangerous fluid shifts [32]. Hyperbilirubinemia, elevations of the alkaline phosphatase, and alanine aminotransferase levels >150 mg/dL suggest gallstones as the etiologic cause of the pancreatitis [33]. Because of marked fluid shifts that occur with acute pancreatitis, blood urea nitrogren, creatinine, and serum electrolytes including calcium should be assessed.

Imaging of the pancreas with CT can confirm the diagnosis of acute pancreatitis, but is not necessary in all cases. The authors feel that CT scanning should be reserved for patients in whom the diagnosis is in question, in cases of suspected pancreatic necrosis, or in cases of clinical deterioration despite adequate medical therapy. Use of intravenous contrast is highly recommended, and CT should therefore be deferred until the patient has received adequate volume resuscitation to prevent nephrotoxicity.

Because the care of patients with acute pancreatitis is complicated by the difficulty in differentiating whether a patient's course will be mild or severe, several scoring systems have been developed to assess the severity in acute pancreatitis and determine prognosis. The most well known of these criteria is Ranson's criteria, which was originally developed for alcoholic pancreatitis and later modified and validated for gallstone pancreatitis. Ranson's criteria has limited clinical value because it takes 48 hours to determine. The Imrie/Glasgow criteria and APACHE II score are two other prospective systems, but both are cumbersome to perform. A prognostic CT scoring system, known as the Balthazar criteria, has been validated for predicting severity in acute pancreatitis. The score is heavily weighted on the presence of pancreatic necrosis [34]. The cornerstone of therapy in acute pancreatitis is intravenous volume resuscitation coupled with the prevention of pancreatic stimulation. Patients should be kept strictly nothing by mouth, and

therefore require a hospital setting for treatment. Very aggressive intravenous fluid repletion is necessary to maintain intravascular volume and allow adequate perfusion of the pancreas and extrapancreatic organs such as the kidneys.

Luminal and vascular disorders

Acute appendicitis

Acute appendicitis is the most common abdominal surgical emergency in the United States, with over 250,000 appendectomies performed annually [35]. Most cases of appendicitis are believed to result from obstruction of the appendiceal lumen by fecaliths. Following obstruction, increased intraluminal pressure causes local ischemia, leading to transmural inflammation. Secondary bacterial infection occurs, and gangrene and perforation of the appendix may result. A thorough history and physical examination are all that is required to make a clinical diagnosis of appendicitis, thereby heightening the importance of the initial care provider's assessment. As a result of appendiceal hypertension and distension, a crampy visceral type pain is initially felt in the peri-umbilical region. There is often associated nausea, vomiting, and fever. As the inflammatory process progresses, and directly irritates the parietal peritoneum, the quality of the pain becomes sharp and shifts to the right lower quadrant (RLQ). Almost all patients with appendicitis will lose their appetite, and if a patient exhibits hunger, the clinical diagnosis of appendicitis should be questioned.

Auscultation of the abdomen reveals diminished or absent bowel sounds. Classically, the examination of patients with appendicitis reveals tenderness to palpation at McBurney's point, anatomically located two thirds of the way from the umbilicus to the anterior superior iliac spine. Guarding, rigidity, and rebound tenderness may be present from peritoneal irritation. Rovsing's sign may be present, which is RLQ pain upon left lower quadrant palpation. The obturator and iliopsoas signs can be elicited by internal rotation of the right hip and extension of the right hip, respectively. The finding of abdominal pain during either maneuver occurs as a result of the inflammatory process, irritating the respective muscles during passive movement.

Patients who present with acute abdominal pain that migrates from the umbilicus to the right lower quadrant, whom also exhibits RLQ tenderness on palpation, should be referred for emergent appendectomy. The accuracy of the clinical diagnosis in this situation has been estimated to be nearly 95% [36]. However, this classic presentation of appendicitis occurs only in two thirds of patients [37]. Atypical presentations account for the remainder. They result from either anomalous appendiceal anatomy, or certain populations of patients that are more prone to atypical presentations of common diseases, such as the elderly, immunocompromised, or pregnant

patients. For example, a retrocecal appendix that becomes inflamed may produce right flank rather than abdominal pain. Patients older than 55 years of age may present with vague symptoms and exhibit more subtle examination findings, thereby causing a delay in diagnosis. This delay can result in a higher rate of perforation compared with their younger counterparts [37]. Finally, misdiagnosis is more common in premenopausal women owing to a broadened gynecologic differential diagnosis and confusing presentations [38]. Therefore, the importance of considering this diagnosis at any age remains important.

Patients with suspected appendicitis should be made nothing by mouth (NPO) and started on intravenous fluids. The prophylactic use of antibiotics is not supported by the literature, and should only be used in cases of suspected perforation. Because of the potential perforation risk, patients with a clinical diagnosis of appendicitis should undergo emergent surgical intervention. Historically, a 20% presurgical false positive rate has been considered acceptable. In patients where the clinical diagnosis is uncertain, imaging studies and observation admissions for serial abdominal examinations may decrease this false positive rate. In any woman of childbearing age, pregnancy should be ruled out with a serum or urinary β-human chorionic gonadotropin (β-HCG) test before imaging or appendectomy.

Diverticulitis

Nearly a third of patients over the age of 50, and two thirds by the age of 80 have diverticular disease [39]. Diverticulitis, a complication caused by the perforation of a diverticulum, affects up to 25% of patients with diverticular disease [40]. Inspissated food, stool, and increased intraluminal pressure are believed to be involved in the pathogenesis of diverticular perforation. The clinical presentation of patients with diverticulitis is dependent on the extent of the perforation. Small perforations are walled off by surrounding mesentery and pericolonic fat, while larger perforations can result in extensive intraperitoneal abscess formation and frank peritonitis. The location of abdominal pain in patients symptomatic with diverticulitis is dependent on the location of the perforated diverticulum. Diverticular disease most commonly affects the sigmoid colon, so patients most often present with crampy, left lower quadrant abdominal pain. However, patients with a redundant sigmoid colon or diverticular disease involving the right colon may complain of RLQ abdominal pain [41]. Patients may additionally complain of nausea, vomiting, fever, and anorexia. Physical examination often reveals tenderness over the inflamed area, and an inflammatory mass may be palpable in some patients. In patients with free perforation, peritoneal signs such as rebound, guarding, and rigidity may be present.

Although the diagnosis can often be made on clinical grounds alone, an imaging study should be performed during a patient's initial presentation to confirm the presence of diverticula. This can be done as an outpatient,

provided none of the aforementioned red flags are present. CT of the abdomen and pelvis with intravenous and oral contrast is the diagnostic modality of choice, with a reported sensitivity as high as 98% [42]. Colonoscopy should not be performed in patients with suspected diverticulitis, as perforation is a contraindication for endoscopy.

Management of mild, uncomplicated diverticulitis can occur on an outpatient basis, and consists of a clear liquid diet and the administration of oral antibiotics that cover typical gastrointestinal pathogens. Complicated diverticulitis occurs when patients develop intraabdominal abscesses, fistula, free perforation, or intestinal obstruction. Patients with complicated diverticulitis or those with mild disease who fail to respond to above therapies require hospitalization. Patients should be started on intravenous antibiotics, made NPO, and be evaluated by a surgeon. Intraabdominal abscesses can often be managed with percutaneous drainage catheters, but surgery is sometimes required [43]. Free perforation or intestinal obstruction usually mandates emergent surgery.

Obstruction

Bowel obstruction occurs when the normal flow of intestinal contents is interrupted by a mechanical blockage. In patients with a history of abdominal surgery, nearly 75% of cases of small bowel obstruction (SBO) are the result of adhesive peritoneal bands [44–46]. In fact, up to 15% of patients who undergo laparotomy will be readmitted within 2 years with SBO from adhesions, while up to 3% will require operative intervention as a result [46]. Furthermore, it is estimated that the 10-year risk of developing recurrent SBO from adhesions is around 40% [47]. Hernias are the second most common cause of SBO, and account for up to 25% of cases [48]. The remainder of cases of SBO result from a number of etiologies, including Crohn's disease, volvulus, neoplasm, intussusception, gallstones, and ischemia. The clinical presentation of large bowel obstruction (LBO) is very similar to that of SBO. Nearly 60% of cases of LBO are the result of malignancy, with colon cancer being the most common. Other causes include diverticular strictures and colonic volvulus [49]. The cecum and the sigmoid colon are the most common locations of colonic volvulus [50]. Once the bowel is obstructed, the segment of bowel proximal to the obstruction becomes increasingly distended by swallowed air, gas from bacterial fermentation, and luminal secretions. Bacterial overgrowth, bowel edema, and loss of absorptive function follow. If the obstruction is not promptly treated, then ischemia, necrosis and perforation may occur.

The pain caused by SBO is a colicky, diffuse pain that waxes and wanes over 5- minute intervals. Nausea, vomiting, distention, and obstipation are often associated with the abdominal pain. Emesis is often feculent due to bacterial overgrowth. The passage of stool and flatus do not eliminate SBO from the differential diagnosis, as luminal contents distal to the

blockage can still pass. Patients will often exhibit physical signs of volume depletion. Abdominal examination reveals a distended abdomen with either hyperactive high-pitched or hypoactive bowel sounds. Rushes of luminal fluid can often be heard. The abdomen is usually diffusely tender, with findings of rigidity, rebound tenderness, or guarding suggesting peritonitis. A ventral, inguinal, or periumbilical hernia should be sought as a potential etiology for the obstruction. Laboratory analysis is usually nonspecific, but common abnormalities include hemoconcentration, leukocytosis, and electrolyte imbalances.

An abdominal plain film series should be the initial diagnostic imaging test in patients with suspected obstruction. Typical findings include air–fluid levels, small bowel distention, and a paucity of air in the rectal vault. In addition, evidence of complications such as intraperitoneal free air can be seen. Although most diagnoses can be made clinically with the confirmatory assistance of plain films, there are instances where plain films are not sufficient. In these instances, CT may be helpful for both diagnosing SBO and determining the etiology, with a reported sensitivity of 100% and accuracy of 90% [51,52].

Patients with evidence of bowel obstruction should be admitted to the hospital, both for decompression and observation. Patients are initially managed with strict restriction of oral intake, nasogastric tube decompression, intravenous fluids, and electrolyte repletion. Early surgical evaluation is mandatory given the perforation risk if left unattended. The philosophy that "the sun should neither rise nor set on a bowel obstruction," still remains true today.

Peptic ulcer disease

Peptic ulcer disease (PUD) is a common affliction that significantly impacts quality of life. In 1989, more than 5 billion dollars were spent on the care of patients with PUD [53]. *Helicobacter pylori* infection, the most common cause of PUD, has been associated with 75% to 95% of duodenal ulcers (DU) and 65% to 95% of gastric ulcers (GU) [54–56]. Nonsteroidal anti-inflammatory medications (NSAIDs) are the second most common cause of PUD, with an estimated yearly incidence of clinically significant gastric or duodenal ulceration of approximately 1.5% [57]. Use of NSAIDs presents a particular challenge, as up to 40% of patients will not report the use of NSAIDs [58]. Acid hypersecretory syndromes such as Zollinger Ellison syndrome accounts for the majority of the remaining cases. The clinical presentation of PUD depends on the location of the ulcer, and whether complications from the ulcer develop. Complications of PUD include bleeding, obstruction, perforation, and penetration into adjacent structures. Patients with uncomplicated peptic ulcers may be asymptomatic, or they may present with pain in the upper abdomen [59,60]. The pain is typically described as a burning or gnawing pain, but patients may occasionally describe it as

crampy. Nausea and vomiting may also be seen. Relation of pain to meals is unreliable to differentiate DU from GU.

Bleeding from PUD may present with melena, hematochezia, or hematemesis, with or without hemodynamic compromise. Bleeding can generally be managed medically with intravenous fluid, blood transfusions, antisecretory therapy, and endoscopic therapy. Endoscopy is also useful to determine the risk for recurrent bleeding [61]. Surgical or angiographic intervention is reserved for bleeding refractory to endoscopic therapies. Pyloric channel and duodenal bulb ulcers may cause gastric outlet obstruction. In addition to epigastric pain, patients with outlet obstruction may present with nausea, projectile vomiting, early satiety, anorexia, and weight loss. Conservative measures are often successful, although many patients will require surgery or endoscopic dilatation therapy [62,63]. Most ulcers that perforate are located in the duodenal bulb, and are often associated with NSAID use [64,65]. Patients present with the sudden onset of epigastric pain that quickly becomes diffuse as generalized peritonitis ensues. Patients can sometimes develop paradoxic improvement in their pain following perforation despite a markedly rigid and diffusely tender abdomen. Plain films are usually adequate to confirm the diagnosis of ulcer perforation. Perforations require immediate surgical evaluation. Ulcer penetration into adjacent structures occurs in up to 20% of cases of PUD, but only a small proportion become clinically apparent [66]. The most common sites of ulcer penetration include the pancreas, omentum, hepatobiliary system, colon, and adjacent vasculature. Patient presentation reflects the structure that is involved, and the therapy is site-specific.

Ischemic bowel disease

Depending on the location, degree, and acuity of the vascular compromise, ischemic bowel disease is classified into three distinct syndromes: acute mesenteric ischemia, chronic mesenteric ischemia, and colonic ischemia. Acute mesenteric ischemia results from the rapid loss of blood supply to the portion of the intestines supplied by the celiac, superior mesenteric, or inferior mesenteric arteries. The cause is most commonly thromboembolic disease. The consequences of acute mesenteric ischemia are severe, and include bowel necrosis, infarction, and death. Chronic mesenteric ischemia results from the gradual loss of blood supply to the portion of the intestines supplied by the celiac, superior mesenteric, or inferior mesenteric arteries. The cause is usually atherosclerosis. Patients with chronic mesenteric ischemia present with chronic postprandial abdominal pain, which is termed intestinal angina. Because eating worsens the pain, patients develop a fear of eating (sitophobia), and significant weight loss may occur. Colonic ischemia, also known as ischemic colitis, is the most commonly encountered intestinal vascular disorder [67]. Colonic ischemia occurs when there is a decrease in colonic mucosal oxygenation. In the vast majority of patients, colonic

ischemia does not result from an occlusive vascular process, but rather occurs when the oxygen requirements to a specific portion of the colon are not met by the vascular supply. Colonic ischemia occurs in the portions of the colon where blood flow is least redundant, such as the splenic flexure, and rectosigmoid junction. Lower gastrointestinal bleeding, rather than abdominal pain, is the most common presenting symptom. The disorder is self-limited in the majority of cases, and the prognosis is good. Of the three ischemic bowel syndromes, acute mesenteric ischemia is the disease that presents with acute abdominal pain, and will be further discussed below.

Acute interruption of blood supply in the mesenteric vasculature results from either thromboembolic disease or vasospasm. The major risk factors include advanced age, hypercoaguability, vascular disease, and cardiac disorders such as atrial fibrillation or valvular disease. Once the blood supply to the mesenteric vascular is interrupted, acute ischemia ensues. If the vascular compromise persists, bowel infarction, necrosis, and perforation may occur. Patients with acute mesenteric ischemia present with acute onset, severe periumbilical abdominal pain. Early in the disease course, the pain is often out of proportion to tenderness produced during the physical examination. If the patient presents after bowel infarction has already occurred, peritoneal signs may develop. The stool may be positive for occult blood, but hematochezia is uncommon with acute mesenteric ischemia. Common laboratory abnormalities seen with acute mesenteric ischemia include leukocytosis and an elevated hematocrit from hemoconcentration. A low serum bicarbonate, metabolic acidosis, and elevated lactate level are seen once bowel infarction has occurred. Retrospective studies evaluating the role of elevated plasma D-dimer levels in the diagnosis of early mesenteric ischemia showed initial promise, although subsequent prospective evaluations have shown D-dimer to be less helpful [68,69].

Several imaging modalities, including plain films, Doppler ultrasound, conventional CT, and MRI have been studied for the diagnosis of acute mesenteric ischemia. Unfortunately, these imaging techniques lack sensitivity and specificity to accurately make the diagnosis [70]. Mesenteric angiography is the ''gold standard'' test for diagnosing occlusive arterial mesenteric ischemia. Its sensitivity and specificity are 75% to 100% and 100%, respectively [67]. In addition to its diagnostic capabilities, angiography offers the potential for treatment. Several studies demonstrate a decreased mortality in patients who undergo angiography for suspected occlusive mesenteric arterial ischemia [71,72]. The mortality rate for patients with acute mesenteric ischemia in whom the diagnosis is not made before the onset of bowel infarction is reported to be as high as 90% [67]. Therefore, early diagnosis is crucial. Because laboratory findings may be nonspecific early in the disease course, a high index of suspicion, based upon predisposing risk factors, and clinical presentation is required. Patients with suspected acute mesenteric ischemia should have prompt angiography and surgical evaluation [73].

Abdominal aortic aneurysm

It is estimated that as many as 10% of patients over the age of 65 have an abdominal aortic aneurysm (AAA). Rupture of the AAA carries an overall mortality rate of nearly 90%, which only falls to 70% if patients survive to reach the operating room [74,75]. Because of such high mortality and rapid course, a ruptured AAA is unlikely to present in an outpatient clinic. However, any severe abdominal pain complaint in a patient with a known AAA mandates immediate referral to an emergency department for evaluation.

Inflammatory bowel disease

Inflammatory bowel disease (IBD) encompasses ulcerative colitis (UC), Crohn's disease (CD), and indeterminate colitis. All three disorders are chronic, characterized by disease-free intervals, followed by flares of disease. These disorders are generally managed in the outpatient setting, and abdominal pain is often a component of active disease. Nonetheless, there are several acute, potentially life-threatening complications from IBD that may present as abdominal pain in the outpatient setting. These include fulminate colitis, toxic megacolon, bowel obstruction, bowel perforation, and abscess formation.

Fulminate colitis is typically associated with UC, and is the initial presenting scenario in up to 10% of patients with UC [76–78]. It is defined as abdominal pain, >10 bloody bowel movements per day, volume depletion, anemia, and any two of the following: white blood count $>10{,}500$ cells/mL, fever $>38.6^{\circ}$C, tachycardia, and hypoalbuminemia [79]. Patients with known UC will complain of increasingly severe, crampy, generalized abdominal pain in addition to the typical complaints of bloody diarrhea, urgency, and tenesmus.

Toxic megacolon occurs in colitis patients when there is pathologically dilated large bowel in conjunction with evidence of systemic toxicity. Early series reported mortality rates of 19%, although more recent series estimate overall mortality approaching 0%, owing to earlier recognition and improved management strategies [80–82]. Toxic megacolon was originally described, and is most commonly seen in the setting of UC, but can also occur with Crohn's colitis, infectious colitis (especially *Clostridium difficile*), ischemic colitis, diverticulitis, and colon cancer. Toxic megacolon generally occurs early in the course of UC, with 30% of cases occurring within the first 3 months of diagnosis and 60% of cases within 3 years of diagnosis [83]. Physical examination classically reveals abdominal distension with tympany to percussion, as well as tenderness above the underlying colon. However, examination findings are less reliable in the setting of active corticosteroid therapy. In patients with peritoneal signs, perforation should be strongly suspected. The diagnosis is made based on the presence of colonic distension (>6 cm) on imaging plus any three of the following: fever

(>38.6°C), leukocytosis (>10,500 cells/mL), anemia, or tachycardia (>120 beats/min), plus any one of the following: altered mental status, volume depletion, hypotension, or electrolyte abnormalities [84]. The patient with either fulminate colitis or toxic megacolon should be hospitalized immediately for aggressive medical care and surgical evaluation. They should be made NPO, started on intravenous hydration with appropriate electrolyte repletion, and in cases of toxic megacolon, should have a nasogastric tube placed to facilitate decompression.

Bowel obstruction is common in CD, and is most frequently seen in the terminal ileum [85]. Obstruction most commonly results from active inflammatory intestinal strictures, postinflammatory fibrotic intestinal strictures, or peritoneal adhesive disease resulting from previous abdominal surgeries. Although far more common in CD, strictures occur in about 5% of patients with UC, with up to 30% representing malignant disease [86].

Perforation can occur both with CD and UC. In patients with UC, it is most commonly the result of toxic megacolon, and carries a mortality rate as high as 50% [87]. In patients with CD, perforation results from unrelieved small bowel obstructions. Because of the powerful immunosuppressive medications that IBD patients are frequently taking, the clinical severity of a perforation may be muted. Therefore, a high index of suspicion is needed.

Intraabdominal abscess formation is common in CD, occurring in approximately 25% of patients [88–90]. They result from microperforations in patients with penetrating or stricturing disease. Patients typically present with fever, leukocytosis, and abdominal pain. Additionally, they may experience back or groin pain if the abscess involves the ileopsoas or pelvic structures, respectively. A special subset of abscess patients includes those with perianal disease, where the abscess occurs in the perirectal fascia, musculature, or adipose tissue. Approximately 30% of patients with perianal fistulizing CD will develop a perirectal abscess [91]. These patients may note low pelvic/perineal pain, defecatory urgency, or tenesmus in addition to constitutional symptoms. Severity may be blunted secondary to concomitant immunosuppressive medications. Regardless, patients with suspected perirectal or intraabdominal abscess with a history of CD warrant hospital admission for antibiotics, surgical evaluation, and advancement of medical therapy when appropriate.

Irritable bowel syndrome

Irritable bowel syndrome (IBS) has an estimated prevalence in the United States as high as 22%, though only one third will ever present for medical evaluation [92]. It is estimated to account for 12% of primary care visits [93]. The hallmark of the disorder is chronic abdominal pain or discomfort that is associated with altered bowel movements. The pathogenesis is not fully understood, but altered motility, visceral hypersensitivity, luminal factors, and psychologic etiologies are felt to all play a role. Affected patients

exhibit variation in the description of the abdominal pain, including cramping, bloating, aching, and even sharp localized pain. Although IBS is troubling to the patient and detrimental to everyday functioning and quality of life, IBS is not a life-threatening process. Therefore, it should be managed on an outpatient basis. Associated abnormalities such as anemia, poor nutritional status, weight loss, evidence of infection, fever, and electrolyte or metabolic abnormalities suggest an alternative diagnosis.

Special populations

There are several populations of patients who warrant special consideration when presenting with abdominal pain because of either underlying processes that are unique to these groups, or because presentations tend to be atypical in these groups. These include the elderly, immunosuppressed (including patients with AIDS), patients on analgesics, women of childbearing age, and pregnant women. Additionally, patients presenting under the influence of alcohol or illicit substances often exhibit atypical presentations of common disorders.

Patients >65 years of age represent the fastest growing population demographic in the United States [94–97]. The elderly often delay seeking medical care, causing them to present at a potentially more dangerous point in their disease course. Compounding this, the history and physical examination have less reliability in the elderly. Many factors contribute to this, including underlying central nervous system disorders, fear of losing independence, hearing loss, depression, complex medical histories, vague description of the discomfort, polypharmacy, and change in normal physiology (eg, inability to mount leukocytosis or pyrexic response to infection). As a result, diagnostic accuracy has been reported as low as 40% in elderly patients with acute abdominal pain [98]. An important point to consider when evaluating elderly patients is that common disorders may manifest with uncommon presentations. For example, both coronary ischemia and urinary tract infections have been well-described causes of abdominal pain in the elderly. Also, it is not uncommon for an elderly patient to present with altered mental status as the lone sign of an acute abdominal process.

Immunosuppressed patients and patients with immunodeficiency syndromes who present with abdominal pain generate a more expansive differential diagnosis (especially infectious causes) for their pain owing to their inability to mount a normal immune response. As in the elderly, the physical examination may be less accurate owing to an abnormal inflammatory response to pathologic processes.

The differential diagnosis and evaluation of gynecologic and obstetric processes manifesting as acute abdominal pain are beyond the scope of this review. However, a few points need to be stressed. All women of childbearing age require a pelvic examination and evaluation for elevated β-HCG in the workup of acute abdominal pain. Additionally, as the gravid uterus

enlarges in a pregnant patient, the normal topography of the small and large intestines may be altered due to mass effect, thereby making the location of abdominal tenderness atypical. This is classically true with appendicitis [99].

Abdominal pain is a common complaint in the outpatient setting. Although many etiologies have a benign course, some have potentially morbid and lethal complications. Care providers must understand the basis of the perception of abdominal pain, and develop a focused approach to the initial evaluation of these patients. Performing a thorough history and physical evaluation will allow the practitioner to generate a differential diagnosis that will guide further laboratory, imaging, and management decisions.

References

[1] Woodwell DA, Cherry DK. National Ambulatory Medical Care Survey: 2002 summary. Adv Data 2004;(346):1–44.
[2] Feldman M, Friedman LS, Sleisenger MH. Sleisenger and Fordtran's gastrointestinal and liver disease: pathophysiology/diagnosis/management. 7th edition. Philadelphia: Saunders; 2002.
[3] Benedict M, Bucheli B, Battegay E, et al. First clinical judgment by primary care physicians distinguishes well between organic and nonorganic causes of abdominal or chest pain. J Gen Intern Med 1997;12(8):459–65.
[4] Yamamoto W, Kono H, Maekawa M, et al. The relationship between abdominal pain regions and specific diseases: an epidemiologic approach to clinical practice. J Epidemiol 1997;7(1):27–32.
[5] Maglinte DDT, Balthazar EJ, Kelvin FM, et al. The role of radiography in the diagnosis of small bowel obstruction. AJR Am J Roentgenol 1997;168(5):1171–80.
[6] Miller RE, Nelson SW. The roentographic demonstration of tiny amounts of free intraperitoneal gas: experimental and clinical studies. AJR Am J Roentgenol 1971;112(3):574–85.
[7] Billittier AJ, Abrams BJ, Brunetto A. Radiographic imaging modalities for the patient in the emergency department with abdominal complaints. Emerg Med Clin North Am 1996;14(4): 789–850.
[8] Gupta H, Dupuy D. Advances in imaging of the acute abdomen. Surg Clin North Am 1997; 77(6):1245–63.
[9] Ahn SH, Mayo-Smith WW, Murphy BL, et al. Acute nontraumatic abdominal pain in adult patients: abdominal radiography compared with CT evaluation. Radiology 2002;225(1): 159–64.
[10] Tsushima Y, Yamada S, Aoki J, et al. Effect of contrast-enhanced computed tomography on diagnosis and management of acute abdomen in adults. Clin Radiol 2002;57(6):507–13.
[11] Stapakis JP, Thickman D. Diagnosis of pneumoperitoneum: abdominal CT vs. upright chest film. J Comput Assist Tomogr 1992;16(5):713–6.
[12] Berger MY, van der Velden JJ, Lijmer JG, et al. Abdominal symptoms: do they predict gallstones? A systematic review. Scand J Gastroenterol 2000;35(1):70–6.
[13] Yusoff IF, Barkun JS, Barkun AN. Diagnosis and management of cholecystitis and cholangitis. Gastroenterol Clin North Am 2003;32(4):1145–68.
[14] Raine PA, Gunn AA. Acute cholecystitis. Br J Surg 1975;62(9):697–700.
[15] Kadakia SC. Biliary tract emergencies. Med Clin North Am 1993;77(5):1015–36.
[16] van der Linden W, Sunzel H. Early versus delayed operation for acute cholecystitis: a controlled trial. Am J Surg 1970;120(1):7–13.
[17] McArthur P, Cuschieri A, Sells R, et al. Controlled clinical trial comparing early with interval cholecystectomy for acute cholecystitis. Br J Surg 1975;62(10):850–2.

[18] Jarvinen H, Hastbacka J. Early cholecystectomy for acute cholecystitis: a prospective randomized trial. Ann Surg 1980;191(4):501–5.
[19] Lai PB, Kwong KH, Leung KL, et al. Randomized trial of early versus late laparoscopic cholecystectomy for acute cholecystitis. Br J Surg 1998;85(6):764–7.
[20] Lo CM, Liu CL, Fan ST, et al. Prospective randomized study of early versus late laparoscopic cholecystectomy for acute cholecystitis. Ann Surg 1998;227(4):461–7.
[21] Chandler CF, Lane JS, Ferguson P, et al. Prospective evaluation of early versus delayed laparoscopic cholecystectomy for treatment of acute cholecystitis. Am Surg 2000;66(9): 896–900.
[22] Brodsky A, Matter I, Sabo E, et al. Laparoscopic cholecystectomy for acute cholecystitis: can the need for conversion and the probability for complications be predicted? Surg Endosc 2000;14(8):755–60.
[23] Pessaux P, Teuch JJ, Rouge C, et al. Laparoscopic cholecystectomy in acute cholecystitis: a prospective comparative study in patients with acute vs. chronic cholecystitis. Surg Endosc 2000;14(4):358–61.
[24] Maluenda F, Csendes A, Burdiles P, et al. Bacteriologic study of choledocal bile in patients with common bile duct stones, with or without acute suppurative cholangitis. Hepatogastroenterology 1989;36(3):132–5.
[25] Saharia PC, Zuidema GD, Cameron JL. Primary common duct stones. Ann Surg 1977; 185(5):598–604.
[26] Saik RP, Greenburg AG, Farris JM, et al. Spectrum of cholangitis. Am J Surg 1975;130(2): 143–50.
[27] Lipsett PA, Pitt HA. Acute cholangitis. Surg Clin North Am 1990;70(6):1297–312.
[28] Banks PA. Practice guidelines in acute pancreatitis. Am J Gastroenterol 1997;92(3):377–86.
[29] Ros E, Navarro S, Bru C, et al. Occult microlithiasis in "idiopathic" acute pancreatitis: prevention of relapses by cholecystectomy or ursodeoxycholic acid therapy. Gastroenterology 1991;101(6):1701–9.
[30] Gullo L, Migliori M, Olah A, et al. Acute pancreatitis in five European countries: etiology and mortality. Pancreas 2002;24(3):223–7.
[31] Steinberg WM, Goldstein SS, Davis ND, et al. Diagnostic assays in acute pancreatitis. A study of sensitivity and specificity. Ann Intern Med 1985;102(5):576–80.
[32] Brown A, Baillargeon J-D, Hughes MD, et al. Can fluid resuscitation prevent pancreatic necrosis in severe acute pancreatitis? Pancreatol 2002;2(2):104–7.
[33] Tenner S, Dubner H, Steinberg W. Predicting gallstone pancreatitis with laboratory parameters: a meta-analysis. Am J Gastroenterol 1994;89(10):1863–6.
[34] Simchuk EJ, Traverso LW, Nukui Y, et al. Computed tomography severity index is a predictor of outcomes for severe pancreatitis. Am J Surg 2000;179(5):352–5.
[35] Owings MF, Kozak LJ. Ambulatory and inpatient procedures in the United States, 1996. Vital and health statistics. Series 13. No. 139. Hyattsville (MD): National Center for Health Statistics; 1998. p. 26.
[36] Paulson EK, Kalady MF, Pappas TN. Suspected appendicitis. N Engl J Med 2003;348(3): 236–42.
[37] Graffeo CS, Counselman FL. Appendicitis. Emerg Med Clin North Am 1996;14(4):653–71.
[38] Rothrock SG, Green SM, Dobson M, et al. Misdiagnosis of appendicitis in nonpregnant women of childbearing age. J Emerg Med 1995;13(1):1–8.
[39] Freeman SR, McNally PR. Diverticulitis. Med Clin North Am 1993;77(5):1149–67.
[40] Parks TG. Natural history of diverticular disease of the colon. Clin Gastroenterol 1975;4: 53–69.
[41] Markham NI, Li AK. Diverticulitis of the right colon: experience from Hong Kong. Gut 1992;33(4):547–9.
[42] Ambrosetti P, Jenny A, Becker C, et al. Acute left colonic diverticulitis: compared performance of computed tomography and water-soluble contrast enema: prospective evaluation of 420 patients. Dis Colon Rectum 2000;43(10):1363–7.

[43] Schecter S, Eisenstat TE, Oliver GC, et al. Computerized tomographic scan-guided drainage of intra-abdominal abscesses: preoperative and postoperative modalities in colon and rectal surgery. Dis Colon Rectum 1994;37(10):984–8.
[44] Bizer LS, Liebling RW, Delany HM, et al. Small bowel obstruction: the role of nonoperative treatment in simple intestinal obstruction and predictive criteria for strangulation obstruction. Surgery 1981;89(4):407–13.
[45] Greene WW. Bowel obstruction in the aged patient: a review of 300 cases. Am J Surg 1969; 118(4):541–5.
[46] Beck DE, Opelka FG, Bailey HR, et al. Incidence of small-bowel obstruction and adhesiolysis after open colorectal and general surgery. Dis Colon Rectum 1999;42(2): 241–8.
[47] Landercasper J, Cogbill TH, Merry WH, et al. Long-term outcome after hospitalization for small-bowel obstruction. Arch Surg 1993;128(7):765–70.
[48] Mucha P Jr. Small intestinal obstruction. Surg Clin North Am 1987;67(3):597–620.
[49] Kahi CJ, Rex DR. Bowel obstruction and pseudo-obstruction. Gastroenterol Clin North Am 2003;32(4):1229–47.
[50] Ballantyne GH, Brandner MD, Beart RW Jr, et al. Volvulus of the colon: incidence and mortality. Ann Surg 1985;202(1):83–92.
[51] Frager D. Intestinal obstruction: role of CT. Gastroenterol Clin North Am 2002;31(3): 777–99.
[52] Frager D, Baer JW, Medwid SW, et al. Detection of intestinal ischemia in patients with acute small-bowel obstruction due to adhesions or hernia: efficacy of CT. AJR Am J Roentgenol 1996;166(1):67–71.
[53] Sonnenberg A, Everhart JE. Health impact of peptic ulcer in the US. Am J Gastroenterol 1997;92(4):614–20.
[54] Borody TJ, George LL, Brandl S, et al. *Helicobacter pylori*-negative duodenal ulcer. Am J Gastroenterol 1991;86(9):1154–7.
[55] Ciociola AA, McSorley DJ, Turner K, et al. *Helicobacter pylori* infection rates in duodenal ulcer patients in the United States may be lower than previously estimated. Am J Gastroenterol 1999;94(7):1834–40.
[56] Tytgat G, Langenberg W, Rauws E, et al. Campylobacter-like organism (CLO) in the human stomach. Gastroenterology 1985;88(5):1620 [abstract].
[57] Silverstein FE, Graham DY, Senior JR, et al. Misoprostol reduces serious gastrointestinal complications in patients with rheumatoid arthritis receiving nonsteroidal anti-inflammatory drugs. A randomized, double-blind, placebo-controlled trial. Ann Intern Med 1995; 123(4):241–9.
[58] Lanas AI, Remacha B, Esteva F, et al. Risk factors associated with refractory peptic ulcers. Gastroenterology 1995;109(4):1124–33.
[59] Kuipers EJ, Thijs JC, Festen HP. The prevalence of *Helicobacter pylori* in peptic ulcer disease. Aliment Pharmacol Ther 1995;9(Suppl 2):59–69.
[60] Hilton D, Iman N, Burke GJ, et al. Absence of abdominal pain in older persons with endoscopic ulcers: a prospective study. Am J Gastroenterol 2001;96:380.
[61] Laine L, Cohen H, Brodhead J, et al. Prospective evaluation of immediate versus delayed refeeding and prognostic value of endoscopy in patients with upper gastrointestinal hemorrhage. Gastroenterology 1992;102(2):314–6.
[62] Weiland D, Dunn DH, Humphrey EW, et al. Gastric outlet obstruction in peptic ulcer disease: an indication for surgery. Am J Surg 1982;143(1):90–3.
[63] Boylan JJ, Gradzka MI. Long-term results of endoscopic balloon dilatation for gastric outlet obstruction. Dig Dis Sci 1999;44(9):1833–6.
[64] Gunshefski L, Flancbaum L, Brolin RE, et al. Changing patterns in perforated peptic ulcer disease. Am Surg 1990;56(4):270–4.
[65] Lanas A, Serrano P, Bajador E, et al. Evidence of aspirin use in both upper and lower gastrointestinal perforation. Gastroenterology 1997;112(3):683–9.

[66] Norris JR, Haubrich WS. The incidence and clinical features of penetration in peptic ulceration. JAMA 1961;178:386–9.
[67] Walker JS, Dire DJ. Vascular abdominal emergencies. Emerg Med Clin North Am 1996; 14(3):571–92.
[68] Acosta S, Nilsson TK, Bjorck M. Preliminary study of D-dimer as a possible marker of acute bowel ishaemia. Br J Surg 2001;88(3):385–8.
[69] Acosta S, Nilsson TK, Bjorck M. D-dimer testing in patients with suspected acute thromboembolic occlusion of the superior mesenteric artery. Br J Surg 2004;91(8):991–4.
[70] Lefkovitz Z, Cappell MS, Lookstein R, et al. Radiologic diagnosis and treatment of gastrointestinal hemorrhage and ischemia. Med Clin North Am 2002;86(6):1357–99.
[71] Boos S. Angiography of the mesenteric artery 1976 to 1991: a change in the indications during mesenteric circulatory disorders. Radiologe 1992;32(4):154–7.
[72] Clark RA, Gallant TE. Acute mesenteric ischemia: angiographic spectrum. Am J Radiol 1984;142(3):555–62.
[73] Kaleya R, Sammartano R, Boley SJ. Aggressive approach to acute mesenteric ischemia. Surg Clin North Am 1992;35(6):613–23.
[74] Multicentre Aneurysm Screening Study Group. The Multicentre Aneurysm Screening Study (MAS) into the effect of abdominal aneurysm screening on mortality in men: a randomised controlled trial. Lancet 2002;360:1531–9.
[75] Ruttedge RA, Oller DW, Meyer AA, et al. A statewide, population-based, time-series analysis of the outcome of ruptured abdominal aortic aneurysm. Ann Surg 1996;223: 492–505.
[76] Becker JM. Surgical therapy for ulcerative colitis and Crohn's disease. Gastroenterol Clin N Am 1999;28:371–90.
[77] Greenstein AJ, Kark AE, Dreiling DA. Crohn's disease of the colon. III. Toxic dilatation of the colon in Crohn's disease. Am J Gastroenterol 1975;63:117–28.
[78] Present D. Toxic megacolon. Med Clin North Am 1993;77(5):1129–48.
[79] Truelove SC, Witts LF. Cortisone in ulcerative colitis: final report on a therapeutic trial. BMJ 1955;2:1041–8.
[80] Katzka I, Katz S, Morris E. Management of toxic megacolon: the significance of early recognition and medical management. J Clin Gastroenterol 1983;78:557–9.
[81] Mungas JE, Mooja AR, Block GE. Treatment of toxic megacolon. Surg Clin N Amer 1976; 56:95–102.
[82] Strauss RJ, Flint GW, Platt N, et al. The surgical management of toxic dilatation of the colon: a report of 28 cases and review of the literature. Ann Surg 1976;184(6):682–8.
[83] Kleer CG, Appleman HD. Ulcerative colitis: patterns of involvement in colorectal biopsies and changes with time. Am J Surg Pathol 1998;22:983–9.
[84] Jalan KN, Sircus W, Card WI, et al. An experience with ulcerative colitis: toxic dilation in 55 cases. Gastroenterology 1969;57:68–82.
[85] Cheung O, Regueiro MD. Inflammatory bowel disease emergencies. Gastroenterol Clin North Am 2003;32:1269–88.
[86] Chutkan R. Inflammatory bowel disease. Prim Care 2001;28(3):539–56.
[87] Greenstein AJ, Aufses AH Jr. Differences in pathogenesis, incidence, and outcome of perforation in inflammatory bowel disease. Surg Gynecol Obstet 1985;160:63–9.
[88] Cybulsky IJ, Tam P. Intra-abdominal abscesses in Crohn's disease. Am Surg 1990;56: 672–82.
[89] Lambiase RE, Cronan JJ, Dorfman GS, et al. Percutaneous drainage of abscesses in patients with Crohn's disease. AJR. AJR Am J Roentgenol 1988;150:1043–5.
[90] Ribeiro MB, Greenstein AJ, Yamazaki Y, et al. Intra-abdominal abscesses in regional enteritis. Ann Surg 1991;179:495–8.
[91] Makowiec F, Jehle EC, Becker HD, et al. Perianal abscess in Crohn's disease. Dis Colon Rectum 1997;40:443–50.
[92] Borum ML. Irritable bowel syndrome. Prim Care 2001;28(3):523–38.

[93] Mitchell CM, Drossman DA. Survey of the AGA membership relating to patients with functional gastrointestinal disorders. Gastroenterol 1987;92:1282.
[94] Ciccone A, Allegra JR, Cochrane DG, et al. Age-related differences in diagnoses within the elderly population. Am J Emerg Med 1998;16:43–9.
[95] Eliastam M. Elderly patients in the emergency department. Ann Emerg Med 1989;18: 1222–9.
[96] Kamin RA, Nowicki TA, Courtney DS, et al. Pearls and pitfalls in the emergency department evaluation of abdominal pain. Emerg Med Clin North Am 2003;21:61–72.
[97] deDombal FT. Acute abdominal pain in the elderly. J Clin Gastroenterol 1994;19:331–5.
[98] Marco CA, Schoenfeld CN, Keyl PM, et al. Abdominal pain in geriatric emergency patients: variables associated with adverse outcomes. Acad Emerg Med 1998;5:1163–8.
[99] Weingold AB. Appendicitis in pregnancy. Clin Obstet Gynecol 1983;26:801.