CHAPTER 43

ACUTE ABDOMEN

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The term *acute abdomen* designates symptoms and signs of intra-abdominal disease usually treated best by surgical operation. Many diseases, some of which do not require surgical treatment, produce abdominal pain, so the evaluation of patients with abdominal pain must be methodical and careful. The proper management of patients with acute abdominal pain requires a timely decision about the need for surgical operation. This decision requires evaluation of the patient's history and physical findings, laboratory data, and imaging tests. The syndrome of acute abdominal pain generates a large number of hospital visits and may affect the very young, the very old, either sex, and all socioeconomic groups.[1-4] All patients with abdominal pain should undergo evaluation to establish a diagnosis so that timely treatment can minimize morbidity and mortality.

Abdominal pain accounts for 5% to 10% of all emergency department visits or 5 to 10 million patient encounters in the United States annually.[5] Another study demonstrated that 25% of patients presenting to the emergency department complained of abdominal pain.[6] Diagnoses vary according to age group: pediatric, geriatric, and everyone else. Chapter 70 deals with abdominal pain in children. Appendicitis is more common in children, whereas biliary disease, colonic diverticulitis, and intestinal infarction occur more commonly in the elderly. Hospitalized patients may develop abdominal pain during the course of their illness, making diagnosis and treatment more difficult.

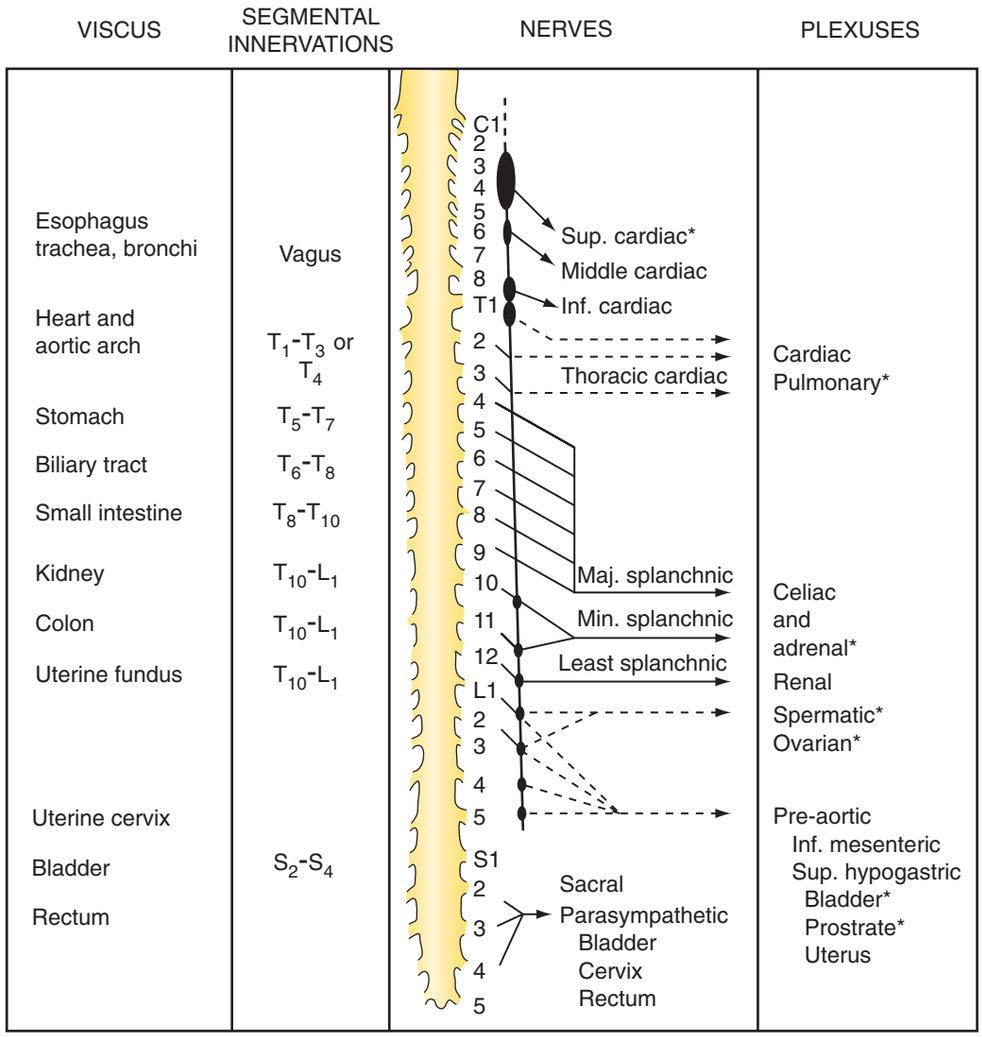
ANATOMY AND PHYSIOLOGY

Developmental Anatomy

The developmental anatomy of the abdominal cavity and of its viscera determines normal structure and influences the pathogenesis and clinical manifestations of most abdominal diseases.[7] Peritoneal attachments and visceral sensory innervation are particularly important to the evaluation of acute abdominal disease. After the 3rd week of fetal development, the primitive gut divides into foregut, midgut, and hindgut. The superior mesenteric artery supplies the midgut (the fourth portion of the duodenum to the midtransverse colon). The foregut includes the pharynx, the esophagus, the stomach, and the proximal duodenum, whereas the hindgut comprises the distal colon and the rectum. The afferent fibers accompanying the vascular supply provide sensory innervation to the bowel and associated visceral peritoneum.

Thus, disease in the proximal duodenum (foregut) stimulates celiac axis afferents to produce epigastric pain. Stimuli in the cecum or appendix (midgut) activate afferent nerves accompanying the superior mesenteric artery to cause periumbilical pain, and distal colon disease induces inferior mesenteric artery afferent fibers to cause suprapubic pain. The phrenic nerve and afferent fibers in C3, C4, and C5 dermatomes accompanying the phrenic arteries innervate the diaphragmatic musculature and the peritoneum on its undersurface. Stimuli to the diaphragm therefore cause referred shoulder pain. The parietal peritoneum, abdominal wall, and retroperitoneal soft tissue receive somatic innervation corresponding to the segmental nerve roots (Fig. 43-1).

The richly innervated parietal peritoneum is particularly sensitive. Parietal peritoneal surfaces sharply localize painful stimuli to the site of the stimulus. When visceral inflammation irritates the parietal peritoneal surface, localization of pain occurs. Maneuvers that exacerbate this irritation then intensify the pain. The many "peritoneal signs" useful in the clinical diagnosis of the acute abdomen originate in this fashion. Dual-sensory innervation of the abdominal cavity by both visceral



* No known sensory fibers in sympathetic rami.

FIGURE 43-1. Sensory innervation of the viscera. (From White JC, Sweet WH: Pain and the Neurosurgeon. Springfield, IL, Charles C Thomas, 1969, p 526.)

afferents and somatic nerves produces clinical pain patterns that aid in diagnosis. For example, the pain of acute appendicitis originates with poorly localized periumbilical pain progressing to sharply localized right lower quadrant pain when the inflammation involves the parietal peritoneal surface.

Peripheral nerves mediate sharp, sudden, well-localized pain. Sensory afferents involved with intraperitoneal abdominal pain transmit dull, sickening, poorly localized pain of more gradual onset and protracted duration. The vagus nerve does not transmit pain from the gut. Small, unnamed sympathetic afferent nerves transmit pain from the esophagus to the spinal cord. Afferent nerves from the liver capsule, the hepatic ligaments, the central portion of the diaphragm, the splenic capsule, and the pericardium enter the central nervous system from C3 to C5. The spinal cord from T6 to T9 receives pain fibers from the periphery of the diaphragm, the gallbladder and the stomach, the pancreas, and the small intestine. Pain fibers from the colon, appendix, and pelvis viscera enter the central nervous system at the 10th and 11th thoracic segments. The sigmoid colon, rectum, renal pelvis and capsule, ureter, and testes pain fibers enter the central nervous system at T11 and L1. The bladder and the rectosigmoid colon send afferent nerves to the spinal cord from S2 to S4.[8,9]

Cutting, tearing, crushing, or burning usually does not produce pain in the abdominal viscera. However, stretching or distention of the peritoneum produces pain. Bacterial or chemical peritoneal inflammation produces visceral pain, as does ischemia. Cancer can cause intra-abdominal pain by invading sensory nerves. Abdominal pain may be visceral, parietal, or referred. *Visceral pain* is dull and poorly localized, usually in the epigastrium,

periumbilical region, or suprapubic region, and it usually does not lateralize well. Patients with visceral pain may also experience sweating, restlessness, and nausea. The *parietal* or *somatic pain* associated with intra-abdominal disorders may be more intense and precisely localized. *Referred pain* is perceived at a site distant from the source of stimulus. For example, irritation of the diaphragm may produce pain in the shoulder. Disease in the bile duct or gallbladder may produce shoulder pain. Distention of the small bowel can produce pain referred to the back.

During the 5th week of fetal development, the bowel outgrows the peritoneal cavity, protrudes through the base of the umbilical cord, and undergoes a 180-degree counterclockwise rotation. During this process, the bowel remains outside the peritoneal cavity until approximately the 10th week, when it returns to the abdomen, and an additional 90-degree counterclockwise rotation occurs. This embryologic rotation places the viscera in their adult positions, and subsequent fusion of the portions of the colonic and duodenal mesenteries with the mesothelium of the posterior abdomen forms the normal ultimate peritoneal attachments. Knowledge of these attachments is clinically important during the evaluation of patients with the acute abdomen because of variation in the exact position of the viscera (e.g., pelvic or retrocecal appendix) and the compartmentalization of the abdomen by mesenteric attachments.[10] The latter, for example, may channel duodenal or gastric contents from the site of a perforated ulcer to the right lower quadrant.

Peritoneal Pathophysiology

Mesothelial cells cover the visceral and parietal peritoneal surfaces. Openings into radially arranged lymphatics penetrate the diaphragmatic peritoneal surface. Introduction of bacteria into the peritoneal cavity can cause an outpouring of fluid from the peritoneal membrane. This loss of fluid from the circulation may lead to dehydration and may produce the clinical signs of resting or orthostatic hypotension and tachycardia. Diaphragmatic lymphatics are the major route for the clearance of bacteria and cellular debris from the abdominal cavity. This process leads to an intraperitoneal circulation of fluid toward the subdiaphragmatic regions bilaterally. Fluid not cleared in this fashion tends to accumulate in the deep end of the pelvis. Thus, subdiaphragmatic, subhepatic, paracolic, or pelvic fluid collections can accompany visceral perforation. The peritoneal surfaces localize bacteria and the products of inflammation. The peritoneum responds to inflammation by increased blood flow, increased permeability, and the formation of a fibrinous exudate on its surface. The bowel also responds to inflammation with localized or generalized paralysis. The fibrinous surface thus created, aided by decreased intestinal movement, causes adherence between bowel and omentum and effectively walls off inflammation. An abscess may produce sharply localized pain with normal bowel sounds and gastrointestinal function, whereas a disseminated process, such as a perforated ulcer, produces generalized abdominal pain with a quiet abdomen. Peritonitis may affect the entire abdominal cavity or a portion of the visceral or parietal peritoneum. Transudation can produce an increase in the peritoneal fluid, which is rich in protein and leukocytes that facilitate the formation of fibrin on peritoneal surfaces.

Peritonitis denotes peritoneal inflammation from any cause. Primary or spontaneous peritonitis can occur as a diffuse bacterial infection without an obvious intra-abdominal source of contamination. Primary peritonitis, most commonly caused by *Pneumococcus* or hemolytic *Streptococcus,* occurs more commonly in children than in adults. However, adults with ascites and cirrhosis are susceptible to spontaneous peritonitis resulting from *Escherichia coli* and *Klebsiella.*[11]

The more common secondary peritonitis results from perforation, infection, or gangrene of an intra-abdominal organ, usually of the gastrointestinal tract. Gastrointestinal secretions, pancreatic secretions, bile, blood, urine, and meconium cause chemical peritonitis when in contact with the peritoneum. A common form of chemical peritonitis follows perforation of a peptic ulcer. Bile peritonitis may result from perforation of the gallbladder or leakage from the bile ducts. Ordinarily, slow bleeding into the abdominal cavity produces relatively few signs of inflammation; the addition of bacteria to blood produces suppuration (Box 43-1). The sickest postoperative patients may have tertiary peritonitis that kills 30% to 64% of affected patients. The syndrome of poorly localized intra-abdominal infection, an altered microbial flora, progressive organ dysfunction, and high mortality define tertiary peritonitis.[12,13]

Peritonitis causes abdominal pain, either generalized or localized, depending on the disease. Appendicitis usually causes localized pain. Perforated peptic ulcer usually produces generalized abdominal pain. Acute cholecystitis causes right upper quadrant pain referred to the right scapula or shoulder. Physical findings of patients with peritonitis are abdominal tenderness, guarding, and rebound tenderness.

Box 43-1. Causes of Hemoperitoneum

Gastrointestinal
- Traumatic laceration of liver, spleen, pancreas, mesentery, bowel

Gynecologic
- Ruptured ectopic pregnancy
- Ruptured graafian follicle
- Ruptured uterus

Vascular
- Ruptured aneurysm: aortoiliac, hepatic, renal, and splenic artery

Urologic
- Ruptured bladder

Hematologic
- Ruptured spleen

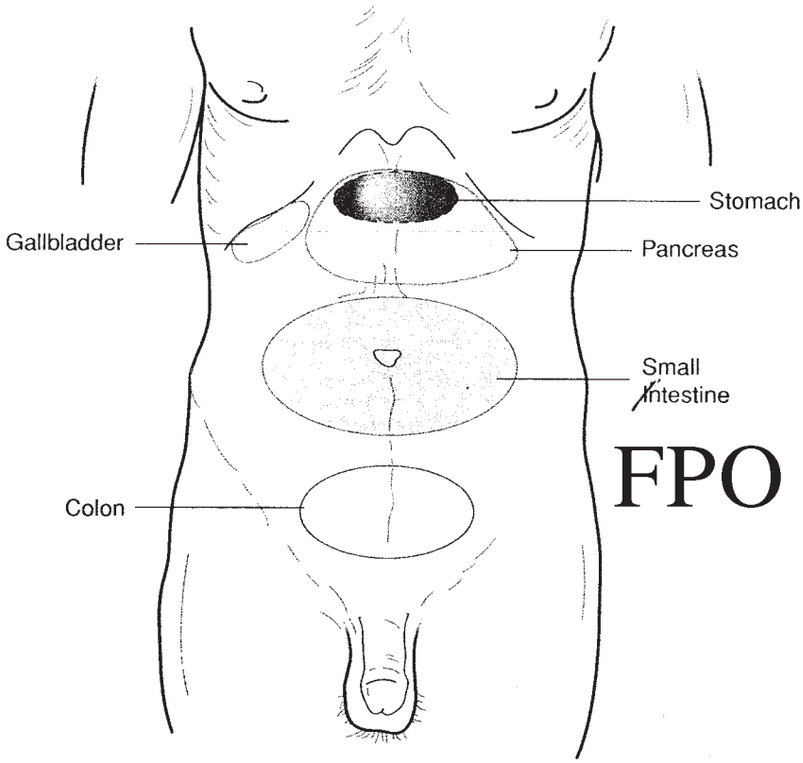


FIGURE 43-2. Pain from intra-abdominal viscera.

CLINICAL DIAGNOSIS

History and Present Illness

Pain is the focal issue in the evaluation of the patient suspected of having an acute abdomen.[8,9,14] The history should therefore characterize and document the pain as precisely as possible. The duration of the pain is important, but the location, mode of onset, and character of the pain help in making a diagnosis. Abdominal pain that persists for 6 hours or more with severe intensity increases the likelihood that surgical operation will be required. If the pain ebbs after a few hours, however, the probability of surgical disease decreases, but not to zero. Visceral pain caused by distention, inflammation, or ischemia usually feels dull and poorly localized in the midabdomen. Depending on the organ involved, the pain may be felt in the epigastrium, the periumbilical area, or the lower abdomen (Fig. 43-2). Diseases of the kidneys or ureters produce pain in the flanks. Parietal pain, however, is sharper and better localized. Localized parietal peritonitis can produce pain confined to one of the four quadrants of the abdomen.

In an evaluation of the location of the pain, the concept of referred pain becomes important. Subdiaphragmatic disorders can produce pain referred to the shoulder. Blood or pus beneath the left diaphragm can cause left shoulder pain. Biliary disease can cause referred pain in the right shoulder or the back. Diseases above the diaphragm such as basal pneumonia can cause pain referred to the neck or shoulder in the C4 distribution. Upper abdominal pain suggests peptic ulcer, acute cholecystitis, or pancreatitis. Conversely, ovarian cysts, diverticulitis, and ruptured tubo-ovarian abscesses produce lower abdominal pain. Small bowel obstruction usually causes midabdominal pain sometimes referred to the back (Fig. 43-3).

Migratory pain shifting from one place to another can give insight into the diagnosis. For example, pain that moves from the epigastrium to the periumbilical area to the right lower quadrant suggests acute appendicitis. Distention and inflammation of the appendix produce visceral pain perceived in the periumbilical area.[15] When the inflammation spreads and produces parietal peritonitis, the pain localizes in the right lower quadrant of the abdomen. Another example of moving or migratory pain occurs with perforated duodenal ulcer. The leakage of duodenal contents from a perforated ulcer causes intense and localized epigastric pain. However, if the leaked duodenal content gravitates down the right paracolic gutter into the right lower quadrant, the patient may also experience right lower quadrant pain. Although the location of abdominal pain may be helpful, particularly early in the course of the disease, it may not be typical in all patients. Late in many cases, the pain may become generalized because of diffuse peritonitis.

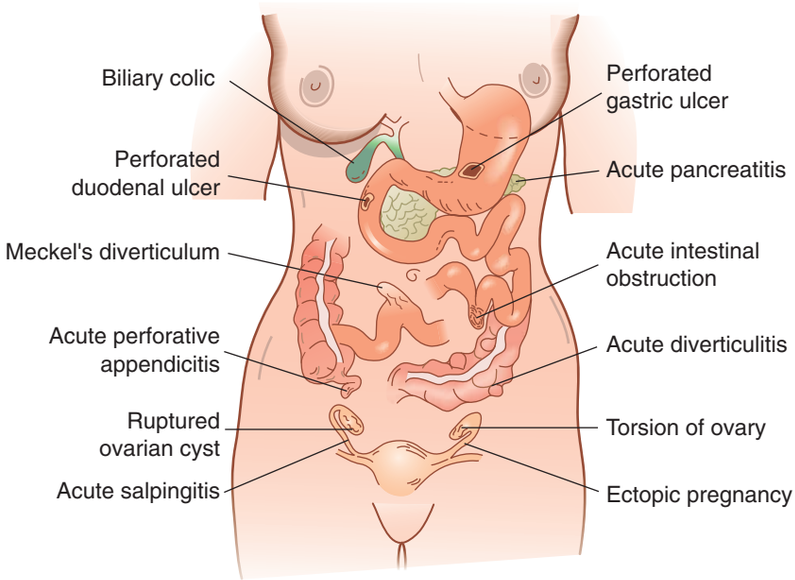


FIGURE 43-3. Common causes of abdominal pain.

Box 43-2. Abdominal Pain Secondary to Inflammatory Lesions of the Gastrointestinal Subsystem

Stomach
- Gastric ulcer
- Duodenal ulcer

Biliary tract
- Acute cholecystitis with or without choledocholithiasis

Pancreas
- Acute, recurrent, or chronic pancreatitis

Small intestine
- Crohn's disease
- Meckel's diverticulum

Large intestine
- Appendicitis
- Diverticulitis

Box 43-3. Abdominal Pain Secondary to Obstructing Lesions of the Gastrointestinal Tract

Jejunum
- Malignancy
- Volvulus
- Adhesions
- Intussusception

Ileum
- Malignancy
- Volvulus
- Adhesions
- Intussusception

Colon
- Malignancy
- Volvulus: cecal or sigmoid
- Diverticulitis

The initial manifestations of the acute abdomen and the evolution of the pain syndrome may give some insight into the cause of the pain. The pain can start suddenly or instantly with no prior symptoms. Sudden or explosive onset of severe abdominal pain suggests free perforation of a viscus such as the duodenum or acute intestinal ischemia from a visceral artery embolus. This type of pain onset can awaken patients from sleep or can incapacitate them during work or play. Sudden, generalized, excruciating pain suggests an intra-abdominal catastrophe that may produce shock requiring resuscitation and prompt operation. In other conditions, the pain comes on with progressively increasing intensity over 1 to 2 hours. This progressive pain represents the usual manifestation of the diseases that commonly produce the acute abdomen such as acute cholecystitis, acute pancreatitis, and proximal small bowel obstruction. Some illness begins with vague general abdominal discomfort that progresses to abdominal pain over a few hours. The pain becomes more intense and subsequently localizes. This group of illnesses generally includes acute appendicitis, incarcerated hernia, distal small bowel obstruction, colon obstruction, diverticulitis, and contained or walled-off visceral perforation (Box 43-2).

The quality, severity, and periodicity of the pain may provide clues to the diagnosis. Steady, sharp pain accompanies perforated duodenal ulcer or perforated appendix. The early pain of small bowel obstruction is vague and deep seated. This pain then assumes a crescendo-decrescendo character described as *colicky pain* (Box 43-3). However, if obstruction produces intestinal infarction, then the pain becomes dull and constant. The pain of ureteral obstruction is extremely severe and intense.

Patients with kidney stones appear restless, agitated, or hyperactive and tend to move about, in contrast to patients with peritoneal inflammation, who prefer to lie quietly and remain undisturbed. Sudden, excruciating pain in the upper abdomen or the lower chest or interscapular region suggests aortic dissection.

Radiation of pain or referral of pain may help in diagnosis. Radiation of pain around the right costal margin to the right shoulder and scapula suggests acute cholecystitis. Pancreatitis usually produces epigastric pain that may radiate along the costal margins to the back or straight through to the back. Kidney stones may cause pain radiating to the groin or the perineal area.

Vomiting may occur from the severity of the pain or because of disease in the gastrointestinal tract. Generally, patients with abdominal pain requiring surgical treatment experience the pain before vomiting occurs. Vomiting frequently precedes the pain in patients with medical conditions. Patients with appendicitis usually have pain and anorexia for a while before vomiting, and patients with gastroenteritis experience vomiting before abdominal pain. Vomiting frequently occurs in patients with acute cholecystitis, acute gastritis, acute pancreatitis, and bowel obstruction. Proximal small bowel obstruction produces more vomiting than distal small bowel obstruction. Vomiting occurs uncommonly in patients with colon obstruction. Small bowel obstruction of longer duration can cause feculent vomiting. Obstruction distal to the ampulla of Vater causes bile-stained vomitus, whereas obstruction proximal to the ampulla causes clear vomitus. Most patients with acute abdominal pain have no desire to eat. Anorexia may precede the pain of acute appendicitis.

Bowel function, including a history of constipation, diarrhea, or a recent change in bowel habits, can be important. Watery diarrhea associated with abdominal pain suggests gastroenteritis. Immunosuppressed patients can contract cytomegalovirus (CMV) infection, salmonellosis, or cryptosporidiosis, which may produce diarrhea. A past history of diarrhea raises the suspicion of inflammatory bowel disease, either Crohn's disease or ulcerative colitis. Failure to pass gas or bowel movements suggests mechanical intestinal obstruction. A history of jaundice, hematemesis, hematochezia, or hematuria is important in the evaluation of acute abdominal pain.

A careful menstrual history is important in women with abdominal pain. Ovulation can produce significant abdominal pain. Furthermore, abdominal pain in a woman with a missed menstrual period or irregular menstrual periods can be related to complications of an undiagnosed pregnancy or an ectopic pregnancy.

The drug history is important in managing patients with acute abdominal pain. Corticosteroids predispose to gastroduodenal ulceration and the possibility of perforation. Corticosteroids also immunosuppress patients and obscure the manifestations of acute intra-abdominal disease. Furthermore, patients who have taken steroids for long periods require perioperative steroid supplementation. Patients who take diuretics need evaluation of their fluid and electrolyte status. Anticoagulants can cause intra-abdominal, intestinal, and mesenteric bleeding. The effects of anticoagulants must be reversed preoperatively. Cocaine can cause abdominal pain. Of course, many patients developing acute abdominal pain are taking cardiovascular drugs, hormones, tranquilizers, diuretics, and numerous other classes of agents that must be managed in the perioperative period.

Past history becomes important, especially regarding prior surgery. For example, if a patient has had an appendectomy, cholecystectomy, and so forth, it has a significant impact on the differential diagnosis of acute abdominal pain. Past history can also give clues to the diagnosis of the present illness. In addition, past history may reveal significant comorbid conditions requiring careful management during the perioperative period. Systemic illnesses or cardiac or pulmonary disease must be excluded as possible causes of the abdominal pain syndrome.

Physical Examination

The physical examination usually provides important information that helps in the diagnosis and management of patients with acute abdominal pain.[8,14] The patient's overall appearance, ability to communicate, habitus, and signs of pain should be noted. Does the patient lie quietly in bed or actively move about? Does the patient lie on his or her side with knees and hips flexed? Does the patient appear dehydrated with dry mucous membranes? An apprehensive patient lying quietly in bed, avoiding motion, and complaining of abdominal pain probably has serious intra-abdominal disease. The physical examination continues with the evaluation of the vital signs. Low fever often accompanies diverticulitis, appendicitis, and acute cholecystitis. High fever more often occurs in pneumonia, urinary tract infection, septic cholangitis, or gynecologic infection. Rapid heart rate and hypotension may mean advanced complicated disease with peritonitis. Peritonitis causes hypovolemia as plasma volume leaves the intravascular space. The general appearance of the patient and the vital signs determine the urgency of the diagnostic workup and implementation of therapy.

Examination of the abdomen always begins with inspection, with particular attention to scars, hernias, masses, or abdominal wall defects. Hernias incarcerated in the groin, umbilicus, or incisions of obese patients can be difficult to detect. The examiner should observe whether the contour of the abdomen appears scaphoid, flat, or distended. Abdominal distention can mean intestinal obstruction, ileus, or fluid including ascites, blood, or bile.

Palpation is a crucial step in evaluating the patient with acute abdominal pain. For this examination, the patient and the examiner should be positioned comfortably to conduct gentle palpation. The examiner should assess the patient's facial expression for signs of pain or discomfort during the examination. Careful palpation for tenderness is important. This must be done gently to avoid hurting the patient and should begin in an area away from the pain site if possible. The finding and the description

of tenderness are the most important steps in palpation of the abdomen of patients with acute abdominal pain. Localized tenderness over the McBurney point suggests appendicitis. Tenderness in the right upper quadrant suggests an inflamed gallbladder. Diverticulitis produces tenderness in the left lower quadrant. Tenderness throughout the abdomen may reflect diffuse peritonitis.

The detection of increased abdominal muscle tone during palpation is called *guarding.* Guarding may be voluntary, involuntary, localized, or generalized. To detect guarding, the examiner should press gently but slowly and firmly on the patient's abdomen. Using two hands works best. The detection of muscle spasm denotes guarding. If, after asking the patient to relax and breathe deeply, the patient's muscles relax, it denotes voluntary guarding. If the muscles remain rigid or tense, it indicates involuntary guarding, which means underlying peritonitis. Guarding may be localized or generalized. Generalized intense guarding produces the boardlike abdomen characteristic of perforated duodenal ulcer. Careful deep palpation can detect abdominal masses. Acute cholecystitis, acute pancreatitis, abdominal aortic aneurysm, and diverticulitis can produce abdominal masses. Severe guarding can interfere with the detection of abdominal masses by palpation.

Rebound tenderness is also a sign of peritonitis. To detect rebound tenderness, the examiner presses deep into the patient's abdomen with flattened fingers. Sudden withdrawal of that hand may cause an increase in the abdominal pain, and this symptom indicates peritonitis. Rebound tenderness can be elicited directly over the site of the abdominal pain. Pressing and releasing the abdomen away from the site of pain can exacerbate the pain at the original site. Careful, deep palpation can detect abdominal masses. Severe guarding can interfere with the detection of abdominal masses by palpation. In acute cholecystitis, palpation in the right subcostal area during deep inspiration by the patient may elicit pain. This finding is called a positive *Murphy's sign.* This sign can be detected either with the patient sitting or supine. The gallbladder may be palpated during this maneuver. Direct compression by the probe may cause pain during ultrasound examination.

Auscultation of the abdomen should give information about the presence or absence of bowel sounds. A quiet abdomen indicates ileus. Hyperactive bowel sounds may occur in gastroenteritis. Periods of quiet interrupted by the onset of high-pitched hyperactive bowel sounds characterize the peristaltic rushes of mechanical small bowel obstruction. Evaluation of bowel sounds requires careful auscultation for several minutes. During auscultation of the abdomen, the examiner can effectively evaluate tenderness and guarding further by palpating gently with the stethoscope. The examiner should also note the presence or absence of bruits in the abdomen.

Percussion is an important part of the abdominal examination. When percussion elicits tenderness, it indicates inflammation and has the same implication as rebound tenderness. Hyper-resonance or tympany to percussion of the abdomen means gaseous distention of the intestine or stomach. Resonance to percussion over the liver suggests free intra-abdominal gas.

Box 43-4. Abdominal Pain Secondary to Lesions of the Gynecologic Subsystem

Ovary
- Ruptured graafian follicle
- Torsion of ovary

Fallopian tube
- Ectopic pregnancy
- Acute salpingitis
- Pyosalpinx

Uterus
- Uterine rupture
- Endometritis

Other tests or maneuvers can aid in the assessment of patients with abdominal pain. Pain during gentle tapping of a fist or deep palpation at the costovertebral angles may suggest pyelonephritis. An inflamed retrocecal appendix or a psoas abscess can produce pain or tenderness on motion of the psoas muscle. If passively extending the hip or actively flexing the hip against resistance causes pain, this is called a positive *iliopsoas sign.* If internal or external rotation of the flexed hip causes pain, it is referred to as a positive *obturator sign.*

During the bimanual pelvic examination, the physician should seek evidence of uterine or adnexal masses or tenderness. Acute salpingitis, tubo-ovarian abscess, or torsion of an ovarian cyst can cause acute abdominal pain (Box 43-4). The speculum examination allows inspection of the cervix for discharge. Rectal examination should include tests for occult blood, and the examiner should note the presence of masses or tenderness. An inflamed pelvic appendix or a pelvic abscess can cause tenderness detected by rectal examination.

Laboratory Testing

Laboratory investigation of most patients with acute abdominal pain usually includes a complete blood count. Intra-abdominal inflammation can produce elevation in the white blood cell count, although this is not always true. One study demonstrated a poor correlation between the white blood cell count and the degree of intra-abdominal inflammation in patients operated on because of acute abdominal pain.[16] If a patient with unequivocal and persistent abdominal pain has a normal or low white blood cell count, a differential count may disclose a marked left shift, which can be more significant than finding an elevation in the white blood cell count. If patients have obvious dehydration, a history of vomiting or diarrhea, or if they have been taking medications such as diuretics that may influence their serum electrolyte values, one should measure the concentrations of serum sodium, potassium,

blood urea nitrogen, creatinine, glucose, chloride, and carbon dioxide. In addition, these laboratory tests enable one to detect diabetes, renal failure, or other systemic diseases. Measurements of serum amylase and lipase may help in the evaluation of upper abdominal pain by giving evidence of pancreatitis. Although elevated serum amylase accompanies pancreatitis, other diseases such as perforated duodenal ulcer and small bowel infarction can also cause increased serum amylase concentrations. Patients with right upper quadrant abdominal pain should have measurements of serum bilirubin, alkaline phosphatase, and serum transaminase because of the possibility of obstructive jaundice or acute hepatitis. Urinalysis can detect evidence of urinary tract infection, hematuria, proteinuria, or hemoconcentration. Women of childbearing age who have acute abdominal pain or hypotension should have measurement of the serum or urine β-human chorionic gonadotropin concentration.

Diagnostic Imaging

History and physical examination are the most important and useful steps in the evaluation of patients with abdominal pain. However, advances in imaging of the abdomen have improved the diagnostic accuracy and the overall management of patients experiencing acute abdominal pain. Before the widespread availability of ultrasonography and computed tomography (CT), surgeons performed a careful history and physical examination, obtained laboratory tests, and reviewed plain films of the abdomen and chest. With that information, a decision to operate or not was made usually on the basis that the patient probably had some disease best treated surgically. The laparotomy was considered diagnostic as well as therapeutic. Historically, before modern imaging tests, as many as 20% of patients operated on for acute appendicitis did not have it.

Plain films still have usefulness in several circumstances. A radiograph centered on the diaphragm detects pneumoperitoneum better than other radiographic techniques. An upright chest radiograph can detect under the diaphragm as little as 1 mL of air injected into the peritoneal cavity.[17] For the occasional patient who cannot stand up, a lateral decubitus radiograph of the abdomen can also detect pneumoperitoneum effectively. A cross-table lateral radiograph with the patient in the left lateral position can detect 5 to 10 mL of gas under the lateral abdominal wall. Free air in the peritoneal cavity indicates a perforation of the gastrointestinal tract. Perforated duodenal ulcers usually allow small amounts of air to escape into the peritoneal cavity. About 75% of patients with perforated duodenal ulcers have radiographically detectable pneumoperitoneum. Perforations of the stomach and the colon can cause extensive pneumoperitoneum. The amount of pneumoperitoneum can also depend on the duration of the leak from the perforation. Plain films of the abdomen can show extensive pneumoperitoneum. If the film defines both the serosal and the related mucosal walls of the bowel, it means free air is at that serosal surface. In addition, free air can delineate the falciform ligament on plain abdominal films. An extensive hydropneumoperitoneum appears as an extremely long air-fluid level on an upright film. A supine film can show a large air collection beneath the abdominal wall that does not conform to any bowel loop (Fig. 43-4).

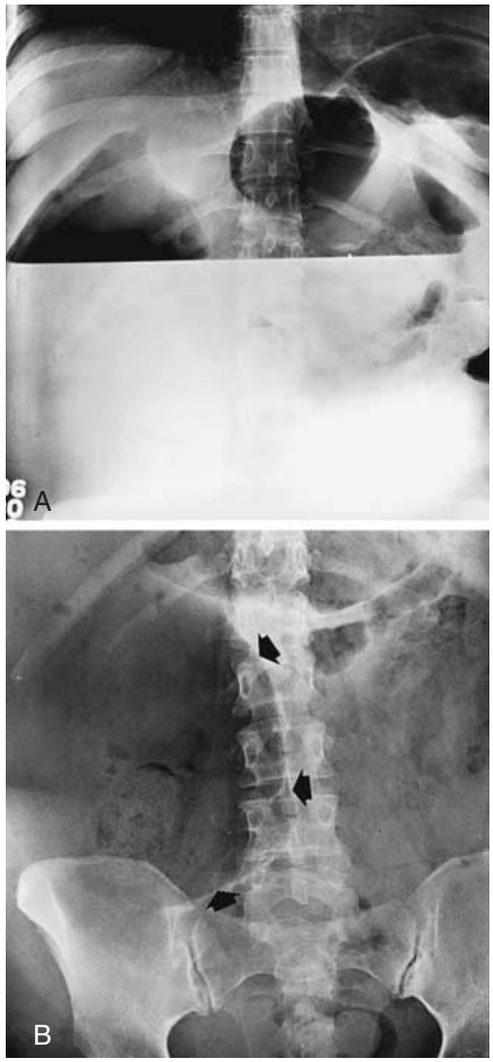


FIGURE 43-4. Plain film findings in hydropneumoperitoneum. **A,** Upright view shows fluid level too long to be within a loop of bowel. **B,** In the supine position, the free air is well defined by the interface with the fluid in the peritoneal cavity *(arrows)*.

Plain films show abnormal calcifications. About 10% of gallstones and 90% of kidney stones contain sufficient calcium to be radiopaque. Appendicoliths can calcify and appear radiographically in 5% of patients with appendicitis. Pancreatic calcifications characteristic of chronic pancreatitis show on plain films, and vascular calcifications can aid in the evaluation of abdominal aortic aneurysms, visceral artery aneurysms, and atherosclerosis of visceral vessels.

Supine and erect plain films of the abdomen show gastric outlet obstruction; proximal, mid, and distal small bowel obstruction; and colon obstruction. The character-

istics of small bowel obstruction include multiple air-fluid levels in dilated, centrally located loops of intestine with visible valvulae conniventes and an absence or paucity of colon gas. Obstructed colon usually appears as peripherally located distended bowel with haustral markings. If the ileocecal valve is incompetent, colon obstruction will cause distention of the distal small bowel.

Some patients with an acute abdomen have plain abdominal films that show a bowel pattern suggesting mechanical obstruction when no obstruction exists. Paralytic ileus can produce distended bowel with multiple air-fluid levels. Plain radiographs show paralytic ileus resulting from intra-abdominal or retroperitoneal inflammation. The radiographic findings of paralytic ileus include excessive distention and fluid with gas distributed from stomach to rectum.

Plain films of the abdomen may also detect gas in the portal or mesenteric venous system, intramural gas in the gastrointestinal tract, gas in the biliary ducts or gallbladder, and gas in the urinary tract or retroperitoneal areas. When plain films show gas in the portal or mesenteric veins, it usually means advanced and serious disease. CT can show small amounts of gas in veins and also may delineate the cause of the abnormality. If the patient's history suggests renal colic, an intravenous pyelogram may confirm the diagnosis of a kidney stone.

CT scanning has provided definite improvements in diagnostic accuracy in evaluating patients with abdominal pain and also reveals anatomic and pathologic detail not possible with plain radiographs (Fig. 43-5).[18] Therefore, CT and ultrasonography now occupy the central imaging role in this situation. Although history and physical examination provide essential information in evaluating patients with the acute abdomen, modern imaging techniques, including ultrasound and CT, can lead to an anatomic diagnosis in most cases. One prospective study of 40 patients with acute abdominal pain revealed that CT significantly improved the diagnostic accuracy of clinical evaluation plus plain radiographs.[19] Clinical examination and plain films were 50% correct, but CT scanning was 95% correct. CT scans accurately detected the specific anatomic lesion in 57.5% of cases compared with 17.5% with clinical examination and plain films. This study included no patients with appendicitis, the most common cause of the acute abdomen, because the surgeons did not refer any cases of suspected appendicitis for inclusion in the study. However, other investigators evaluated the role of CT in the diagnosis of acute appendicitis in 100 consecutive patients studied prospectively.[20] The CT interpretation had 98% sensitivity, 98% specificity, 98% positive predictive value, 98% negative predictive value, and 98% overall accuracy for diagnosing or ruling out appendicitis. According to the authors' calculations, these 100 CT scans produced a net savings of $44,731 in the care of the study patients because of improved diagnostic accuracy. CT scans can add important value to the diagnosis of acute appendicitis. However, focused specialists using excellent equipment in an environment of inquiry conducted this study, and the results may not be reproducible in all hospitals.[20] Other workers questioned the value of CT scanning in the diagnosis of acute appendicitis.[21]

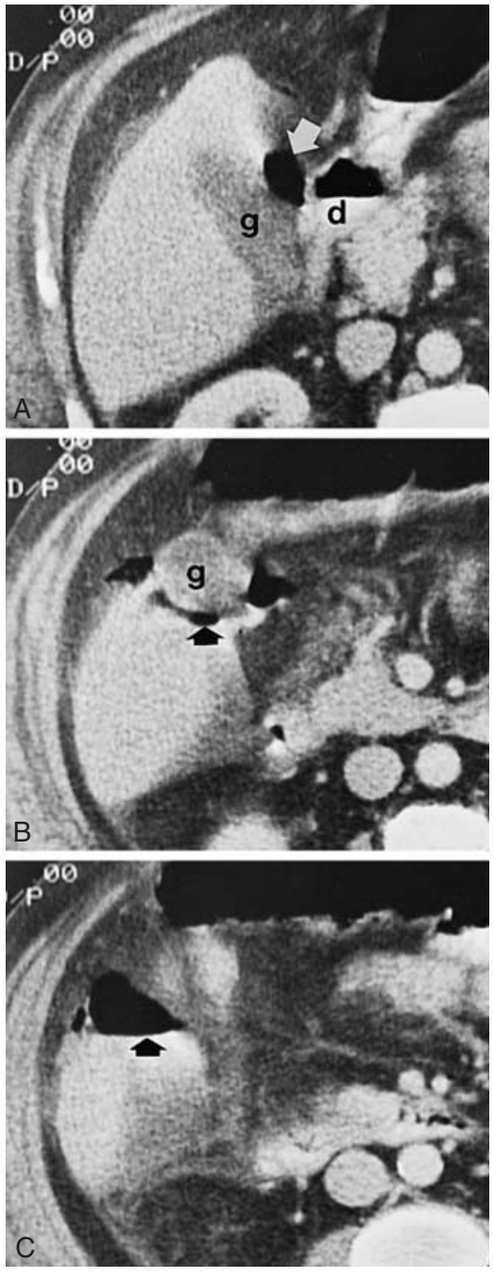


FIGURE 43-5. Unsuspected perforated duodenal ulcer. **A,** Small amount of extraluminal gas *(arrow)* lies lateral to duodenal bulb (d). g, gallbladder. **B,** At 3 cm caudad, gas *(arrow)* tracks behind the gallbladder (g) laterally. **C,** The air-fluid level *(arrow)* identifies the loculated extravasated duodenal contents. Inflammatory changes are present in the surrounding mesenteric fat.

Ultrasonography is useful for patients with acute abdominal pain because it provides rapid, safe, low-cost evaluation of the liver, gallbladder (Fig. 43-6), bile ducts, spleen, pancreas, appendix, kidneys, ovaries, adnexa, and uterus. Transabdominal and intravaginal ultrasonography can aid in the evaluation of the ovaries, adnexa, and uterus. Ultrasonography also detects and characterizes the

FIGURE 43-6. Acute cholecystitis. Ultrasound evaluation shows two small stones *(curved arrow)* present in the neck of the gallbladder. The wall of the gallbladder in the fundus *(straight arrow)* is thickened, and pericholecystic fluid is present.

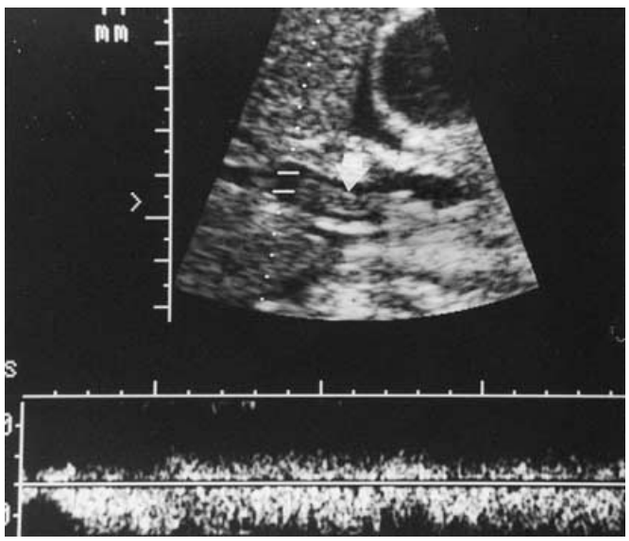


FIGURE 43-7. Thrombus in portal vein evident on pulsed Doppler ultrasonography. An echogenic thrombus *(arrow)* is within the lumen of the portal vein. The Doppler tracing indicates flow within the portal vein.

distribution of intra-abdominal fluid. Color-Doppler ultrasonography allows evaluation of the intra-abdominal and retroperitoneal blood vessels. Aortic and visceral artery aneurysms, venous thrombosis, arteriovenous fistulas, and vascular anomalies can be evaluated with ultrasound (Fig. 43-7). Unfortunately, patients with acute abdominal disease frequently have excessive abdominal gas that interferes with careful and detailed sonographic evaluation of the abdominal organs, but overlying gas, bone, and

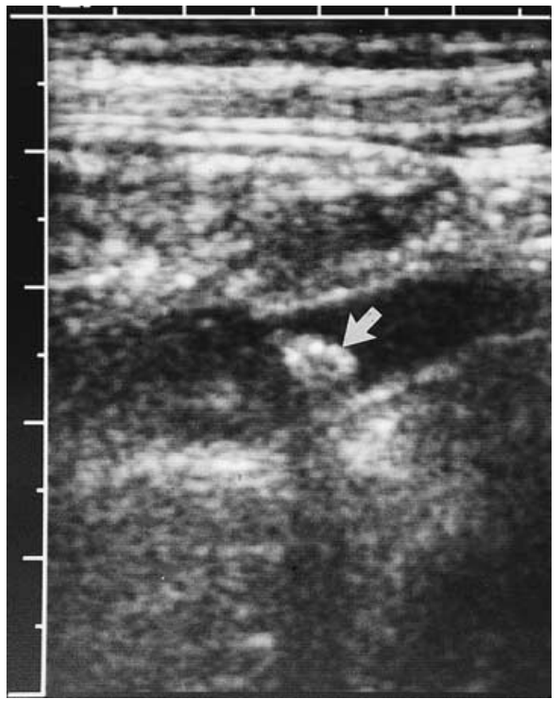


FIGURE 43-8. Acute appendicitis. On ultrasonography, a radiographically nonopaque appendicolith *(arrow)* is evident within a thick-walled, distended appendix (longitudinal view).

fat do not impair imaging with CT. Therefore, CT has become important for evaluating causes of the acute abdomen.

Appendicitis, the most common cause of the acute surgical abdomen in North America, can be difficult to diagnose.[1,2] Plain films and barium enema studies generally add little to the diagnosis. However, in patients with uncomplicated appendicitis, ultrasonography can detect appendicoliths, demonstrate a distended or thick-walled appendix, or detect periappendiceal and pericecal inflammatory changes (Fig. 43-8). Ultrasound is reliable and sensitive for the detection of appendicoliths and the demonstration of an abnormally distended or thick-walled appendix.[22] Conversely, CT detects acute appendicitis and defines the changes of complicated appendicitis (Fig. 43-9). CT scans can enable the examiner to differentiate diffuse periappendiceal inflammation from an abscess. In addition, CT scans detect many of the diseases included in the differential diagnosis of acute appendicitis.

CT detects blood and other fluids in the abdominal cavity. Intramural intestinal hemorrhage is readily detected by CT (Fig. 43-10). CT scans accurately reveal mesenteric venous thrombosis (Fig. 43-11). CT scans can delineate diverticulitis and its complications, such as abscess and even pyelophlebitis (Fig. 43-12). CT is especially helpful in evaluating pancreatitis by revealing minimal edema, extensive edema, fluid collections, hemorrhage, and necrosis; in addition, it effectively evaluates the complications of pancreatitis such as abscess or

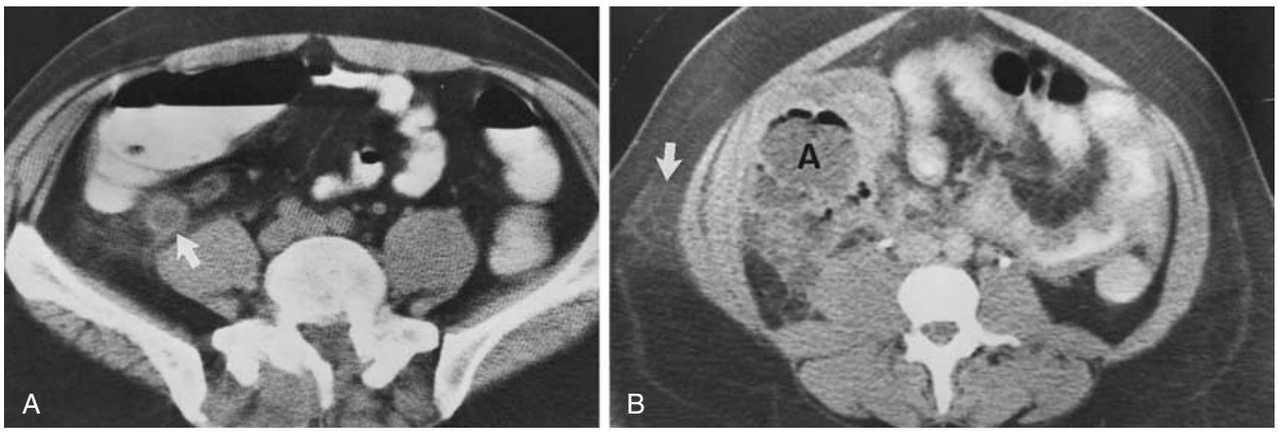


FIGURE 43-9. Appendicitis. **A,** CT scan of uncomplicated appendicitis. A thick-walled, distended, retrocecal appendix *(arrow)* is seen with inflammatory change in the surrounding fat. **B,** CT scan of complicated appendicitis. A retrocecal appendiceal abscess (A) with an associated phlegmon posteriorly was found in a 3-week-postpartum, obese woman. Inflammatory change extends through the flank musculature into the subcutaneous fat *(arrow)*.

FIGURE 43-10. Intramural hematoma of small bowel. Uniform, concentric, high-density thickening of the wall of jejunal loops is characteristic.

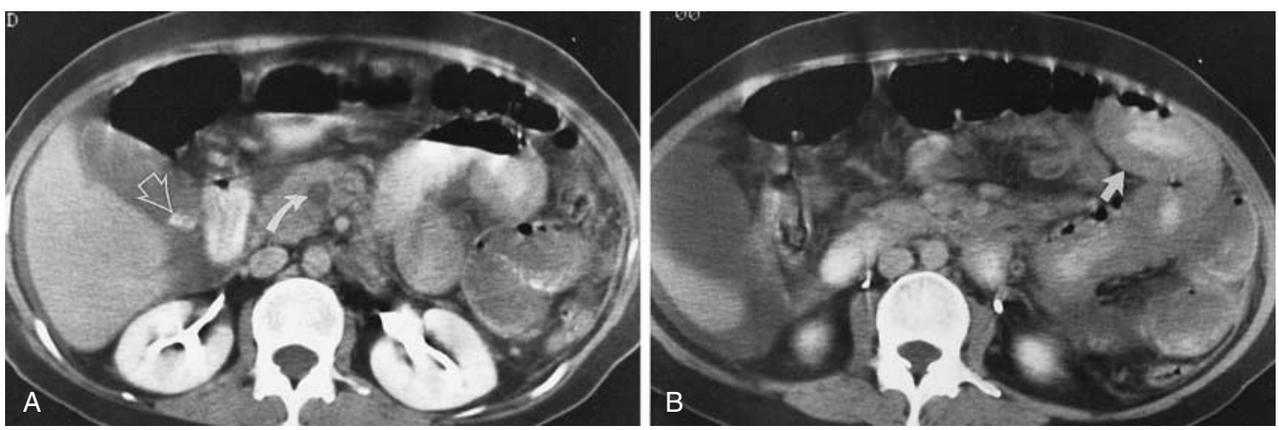


FIGURE 43-11. Small bowel infarction associated with mesenteric venous thrombosis. **A,** Note the low-density thrombosed superior mesenteric vein *(solid arrow)* and incidental gallstones *(open arrow)*. **B,** Thickening of proximal small bowel wall *(arrow)* coincided with several feet of infarcting small bowel at time of surgery.

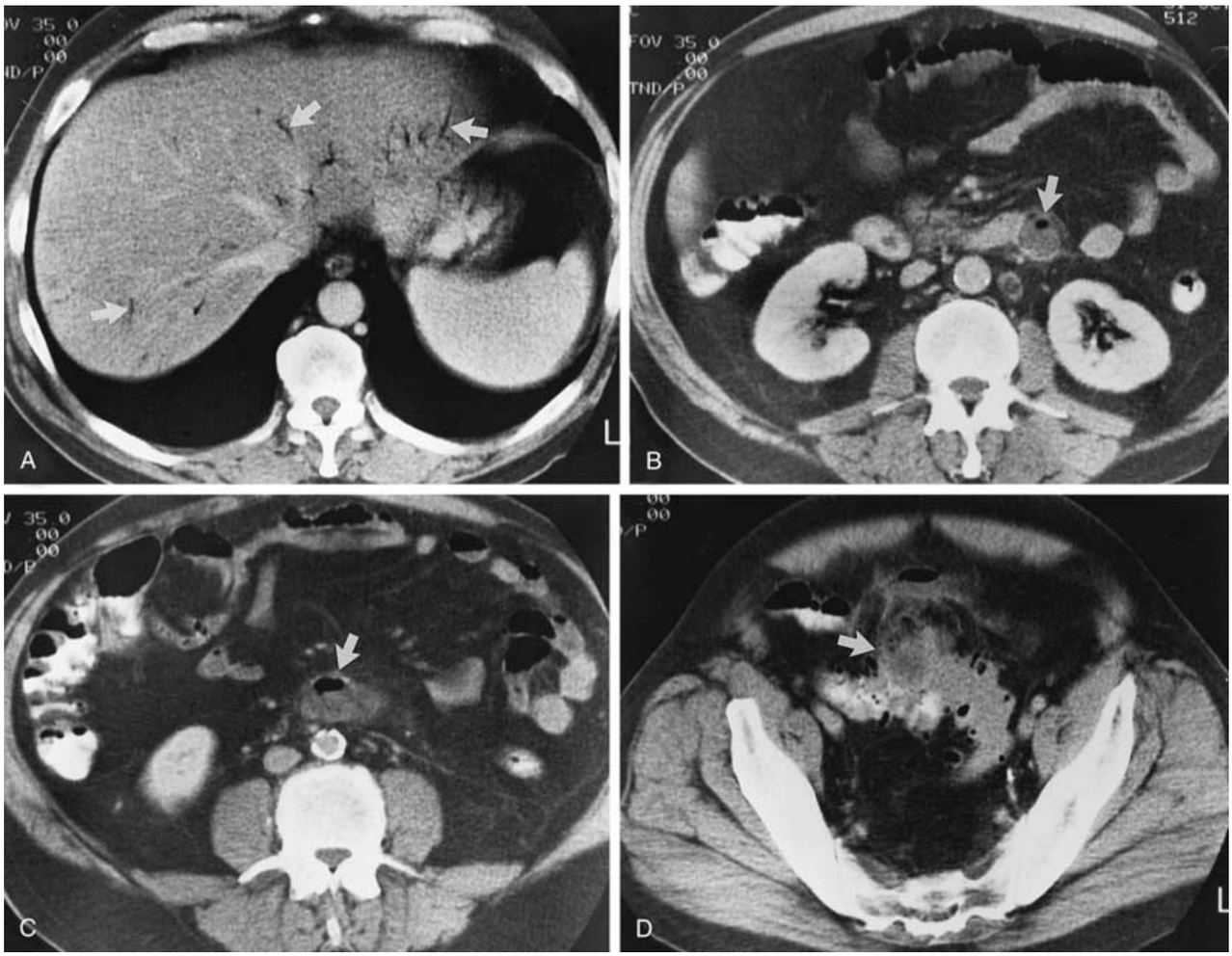


FIGURE 43-12. Acute pyelophlebitis resulting from diverticulitis with abscess. **A,** Minute quantities of gas *(arrows)* within peripheral branches of the portal venous system were not visible on a plain radiograph. **B,** A gas-containing thrombus *(arrow)* is visible in the inferior mesenteric vein at its junction with the splenic vein. **C,** A chain of abscesses *(arrow)* extended along the course of the thrombosed inferior mesenteric vein. **D,** The septic thrombus led directly to a pericolonic abscess *(arrow)* caused by diverticulitis of the sigmoid colon.

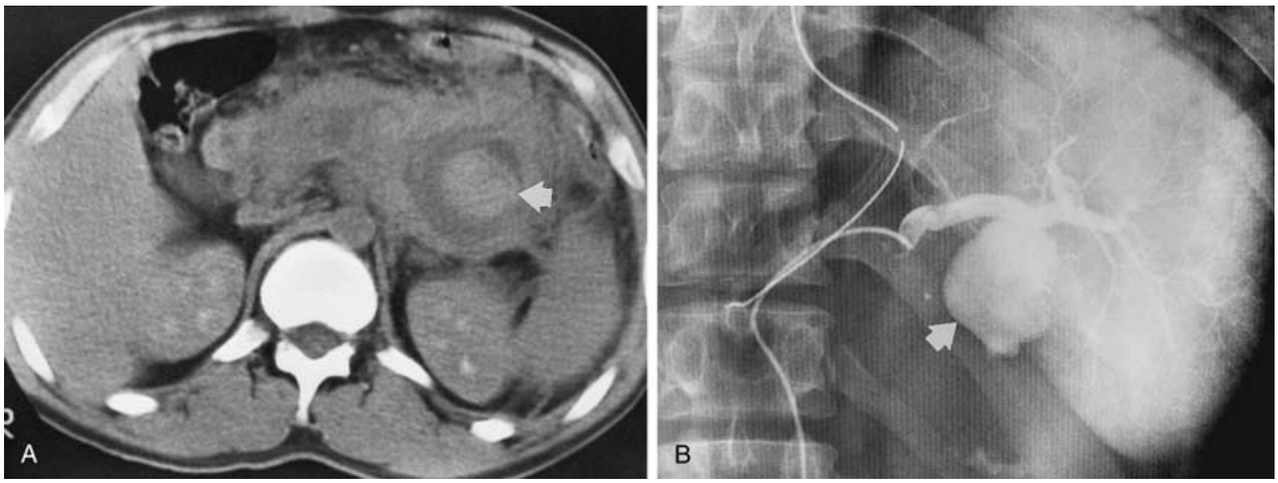


FIGURE 43-13. Hemorrhage and false aneurysm complicating pancreatitis. **A,** Intraparenchymal hemorrhage enlarges the body and tail of the pancreas. The lumen of the false aneurysm *(arrow)* is shown as an area of increased density resulting from the enhancement of the flowing blood. **B,** Selective splenic arteriogram. A false aneurysm *(arrow)* arises from a branch of the splenic artery and was successfully treated with transcatheter embolization.

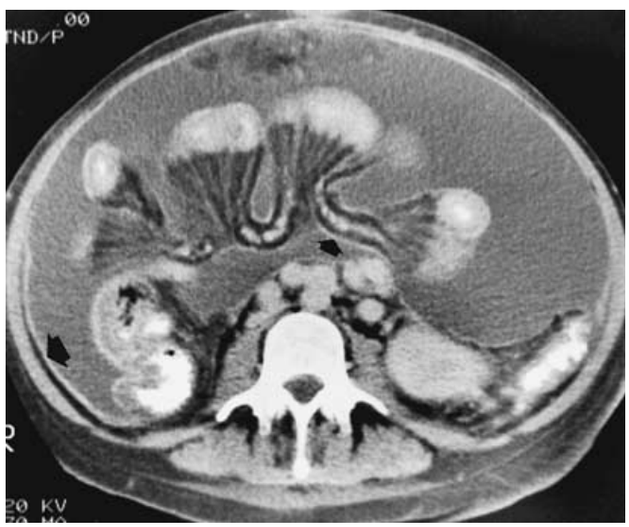


FIGURE 43-14. Peritonitis. CT scan shows inflammatory thickening of the parietal *(large arrow)* and visceral *(small arrow)* peritoneum. The ascitic fluid is of high density, characteristic of peritonitis.

pseudocyst (Fig. 43-13). CT scans show the signs of advanced peritonitis (Fig. 43-14). With this technique, one can also evaluate the complications of colon perforation (Fig. 43-15) and of small bowel disease such as intussusception (Fig. 43-16). Although history and physical examination provide essential information in evaluating patients with the acute abdomen, modern imaging techniques, including ultrasound and CT, can lead to an anatomic diagnosis in the majority of cases.

CLINICAL MANAGEMENT

Differential Diagnosis

Information from the patient's history, physical examination, laboratory tests, and imaging studies usually permits a diagnosis, but uncertainty can still remain (see Fig. 43-3). Because appendicitis is a common disease, it must remain in the differential diagnosis of any patient with persistent abdominal pain, particularly right lower quadrant pain.[3,4] The diagnosis of appendicitis is easy to miss, and perforation substantially increases morbidity and mortality from the disease.[1,2] Delay in diagnosis is the principal reason for unfavorable outcomes in appendicitis. Appendicitis is the most common cause of the acute abdomen in childhood; however, in older patients, acute cholecystitis, bowel obstruction, cancer, and acute vascular conditions assume importance in addition to appendicitis. The differential diagnosis in young women can be difficult because they can have salpingitis, dysmenorrhea, ovarian lesions, and urinary tract infections as well as complications of pregnancy, which can confound the evaluation of abdominal pain. Of course, the medical causes of abdominal pain must be considered, but patients with medical disease generally lack specific localized tenderness and guarding. The other problem is that about one third of patients who present with acute abdominal pain have nonspecific abdominal pain, and no clear diagnosis is ever established.

Decision to Operate

These difficulties notwithstanding, the surgeon must make a decision to operate or not. Certain indications for surgical treatment exist. For example, definite signs of

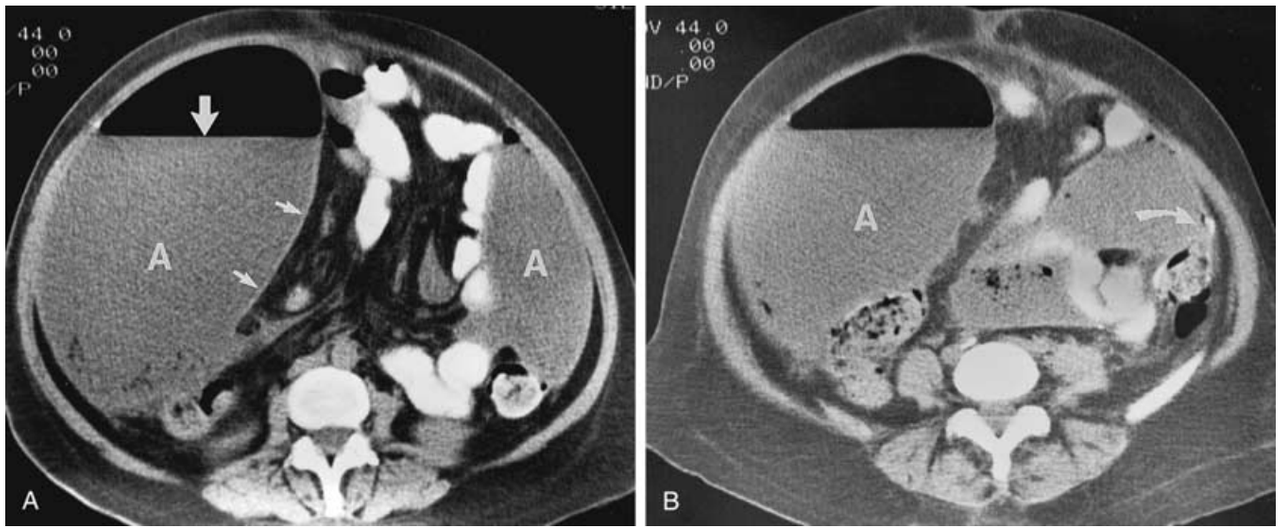


FIGURE 43-15. Pyopneumoperitoneum secondary to a perforated descending colon. **A,** Pyopneumoperitoneum interface *(large arrow)* and inflammatory thickening of visceral peritoneum *(small arrows)* are shown. Seven liters of grossly infected ascitic fluid (A) were drained percutaneously. **B,** A trail of small gas bubbles in the left flank led to a point of discrete perforation of the descending colon *(arrow)*, which was confirmed by contrast material enema and was surgically repaired.

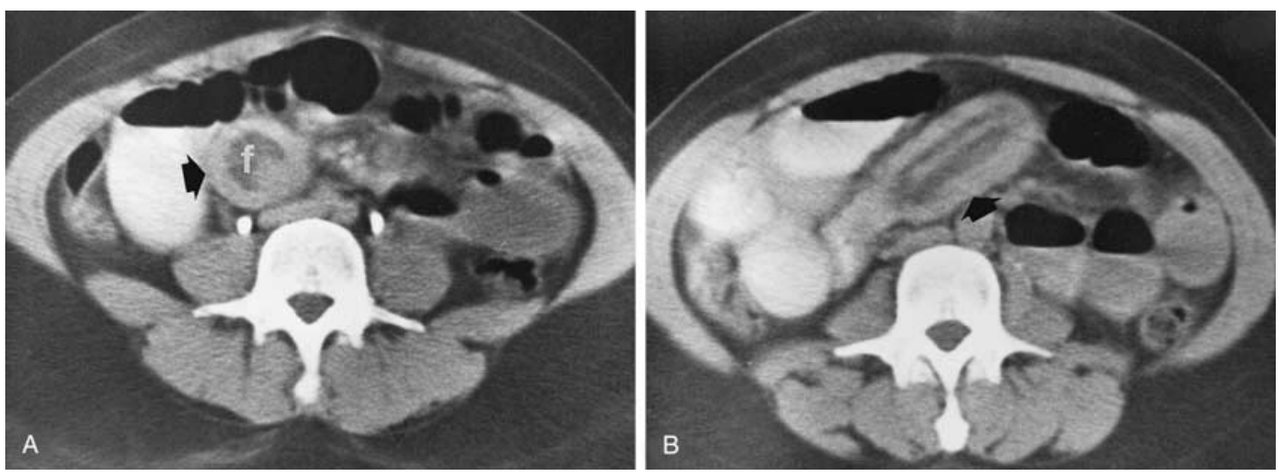


FIGURE 43-16. Acute small bowel intussusception. The patient had a sudden onset of severe mid-abdominal pain with nonspecific plain film findings. Cross-sectional (**A**) and longitudinal (**B**) CT scans showed a small bowel intussusception *(arrows)*. Mesenteric fat (f) accompanies the intussusceptum. A benign spindle cell tumor was the cause.

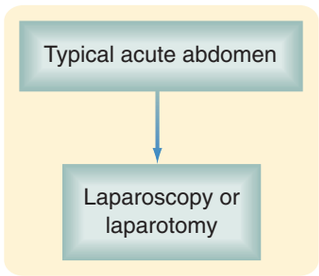


FIGURE 43-17. Patients with unrelenting abdominal pain, tenderness, guarding, and rebound should undergo laparoscopy or laparotomy following suitable resuscitation and preparation.

peritonitis such as tenderness, guarding, and rebound tenderness support the decision to operate (Fig. 43-17). Likewise, severe or increasing localized abdominal tenderness should prompt an operation. Patients with abdominal pain and signs of sepsis that cannot be explained by any other finding should undergo operation. Those patients suspected of having acute intestinal ischemia should be operated on after complete evaluation. Certain radiographic findings confidently predict the need for operation. These findings include pneumoperitoneum and radiologic evidence of gastrointestinal perforation. Patients presenting with abdominal pain and free intra-abdominal gas seen on radiograph warrant operation with limited exceptions (Fig. 43-18). Observation with serial examinations may be

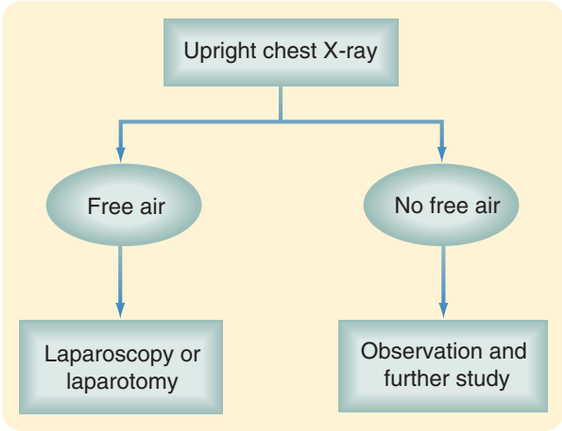


FIGURE 43-18. Most patients with free air in the peritoneal cavity should undergo laparoscopy or laparotomy following suitable resuscitation and preparation.

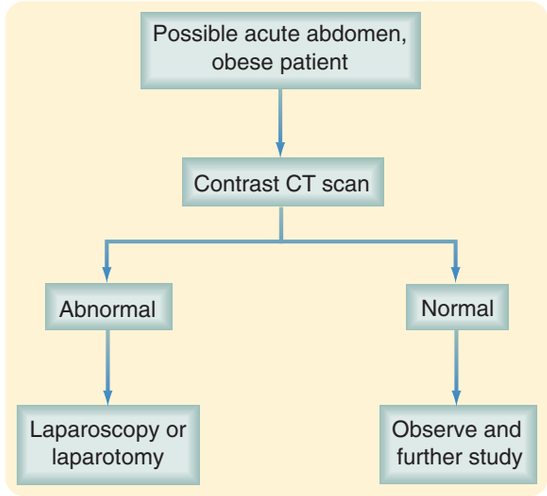


FIGURE 43-20. When obesity impairs physical examination, CT scan of the abdomen can aid in the evaluation of abdominal pain.

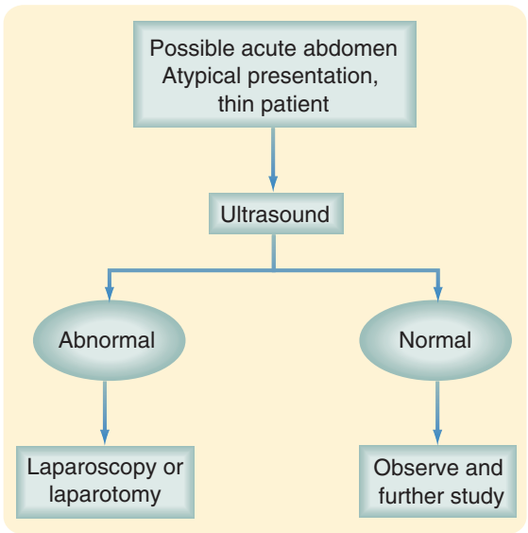


FIGURE 43-19. Patients with abdominal pain and doubtful findings for an acute abdomen should undergo imaging tests beginning with abdominal ultrasound.

appropriate for a patient with free gas after a colonoscopy.[23] Intra-abdominal gas can persist for a day or two following celiotomy. Imaging tests can reveal signs of vascular occlusion requiring operation.

After careful examination and evaluation, diagnostic uncertainty can remain. Some patients may have equivocal physical findings (Figs. 43-19 and 43-20). When this occurs and the diagnosis is unclear and the patient's wellness is unclear, it may be advisable to defer operation and to re-examine the patient carefully after several hours.[24] This is best done in a short-stay unit in the hospital, in a special unit in the emergency department, or if necessary, by regular hospital admission. In a period of hours, vague pain with minimal physical findings may proceed to definite localized pain with tenderness, guarding, and rebound tenderness; if that occurs, operation should follow. After several hours, the patient's symptoms and signs may also resolve. When that happens, the patient can be dismissed, although the patient should have a follow-up appointment scheduled within a day or so to permit re-examination to be certain that an important diagnosis was not missed. Certain patients are difficult to evaluate because of special characteristics. For example, patients who are neurologically impaired as a result of a stroke or a spinal cord injury may be difficult to evaluate.[25] Patients who are under the influence of drugs or alcohol may require special or subsequent examination. Patients who take steroids or are otherwise immunosuppressed deserve special mention because steroids and immunosuppression mask the intensity of abdominal pain and the physical findings of severe, life-threatening intra-abdominal disease. Patients in this category who have persistent, unequivocal abdominal pain and even minimal findings should be considered for surgical operation.

Some patients with clear findings of the acute abdomen may be treated without surgical operation. For example, patients with perforated duodenal ulcer who seek attention late in the course of their disease after they have been sick for several days may be treated best by careful supportive care including nasogastric suction, intravenous fluids, and pain relief. Certain patients with empyema of the gallbladder, especially those with other serious concomitant illnesses, can be treated by percutaneous drainage of the infected gallbladder and careful supportive care rather than with cholecystectomy. Some patients who have acute appendicitis may not seek attention until several days into the course of the illness, at which time they may have walled off the perforation and may have an appendiceal abscess. These patients have right lower quadrant pain, tenderness, and perhaps guarding, but if they have an appendiceal abscess, this is usually best managed by percutaneous drainage of the abscess and

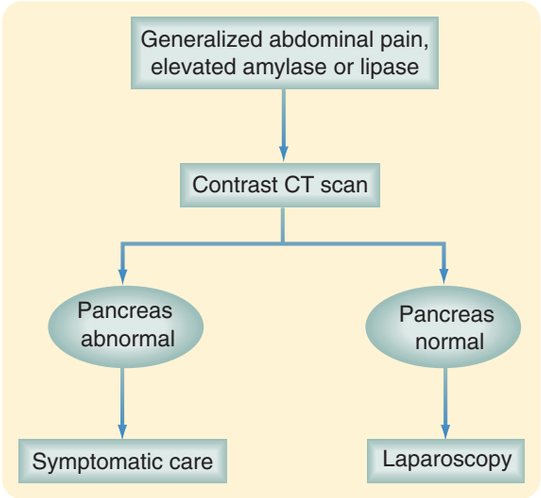


FIGURE 43-21. Pancreatitis can produce the acute abdomen. Acute pancreatitis should be managed with supportive care. Complications of acute pancreatitis may require operation.

avoidance of appendectomy at that time. Acute diverticulitis is usually best managed nonsurgically. If a patient with diverticular disease has a diverticular abscess, percutaneous drainage and supportive care will suffice, and the diverticular disease may be removed electively later. Most patients with acute pancreatitis should be managed without operation unless they have a specific indication for operation (Fig. 43-21). Indications for operation include the development of an abscess.

Preoperative Preparation

In a stable, otherwise healthy patient, preoperative procedures should include insertion of a nasogastric tube, establishment of access for intravenous fluid administration, insertion of a Foley catheter in the urinary bladder to record urinary output, and pain relief. Once a decision has been made to perform an operation, the patient should be given a narcotic or other suitable analgesic unless he or she is being taken immediately to the operating room. Most patients with an acute abdomen requiring an operation have conditions in which infection is either present or likely. For that reason, antibiotics should be administered preoperatively.

Unstable patients must have more careful evaluation and resuscitation before one proceeds to surgical intervention. These patients also require a nasogastric tube, a Foley catheter, intravenous fluids, and antibiotics, but if patients have hypotension, tachycardia, or oliguria and evidence of dehydration, they will need a period of supportive care and intravenous fluids before they undergo general anesthesia and operation. After evaluation of their fluid and electrolyte status and degree of dehydration, these patients should receive sufficient quantities of intravenous fluid to establish urinary output of 0.5 mL/kg per hour. Preoperative blood pressure should be greater than 100 mm Hg systolic, and the pulse should fall to less than 100 beats/min. Patients taking steroids should have supplemental doses administered before and after the operation, including their maintenance dose. Diabetic patients should have attention to control their hyperglycemia and acid-base balance. Cardiovascular function must be monitored in patients with a history of heart disease, and they should have preoperative and postoperative management of their current drugs. Patients with an acute abdomen should be operated on when they become hemodynamically stable and have satisfactory urinary output. Patients who are hypokalemic should have potassium infusion after the establishment of urine flow.

Operation

After concluding that a patient with abdominal pain needs an operation, the surgeon must plan the surgical approach. General inhalation anesthesia administered through an endotracheal tube should be used in most cases. Then the surgeon must choose whether to employ laparotomy or laparoscopy. That choice depends on the surgeon's experience and the probable diagnosis. Some factors such as multiple prior laparotomies, hemodynamic instability, or advanced abdominal distention preclude laparoscopy. For open operation, the surgeon must choose an incision. In cases of probable appendicitis, a right lower quadrant muscle-splitting incision works well. If acute cholecystitis is nearly certain, a right subcostal incision should be used. An incarcerated groin hernia should be approached through a groin incision. When the diagnosis is uncertain, a midline incision works well.

The use of laparoscopy has become more common and more effective in the management of acute abdominal pain. In 1975, Sugarbaker and associates[26] demonstrated the utility of laparoscopy in the management of patients with acute abdominal pain. In this study, 56 patients required hospitalization because of acute abdominal pain. Twenty-seven of these patients had a "definite" clinical diagnosis and underwent laparotomy. Six, or 22%, of these patients had a negative laparotomy, whereas 21 patients had diseases managed best by laparotomy. Twenty-nine patients without an "exact" diagnosis underwent laparoscopy. Eighteen of those patients had, at laparoscopy, a definitive diagnosis of a disease that did not require laparotomy, and 11 patients required laparotomy after laparoscopy. Laparoscopy required 20 minutes on average and incurred no complications. The patients in the laparoscopy group had shorter hospital stays and lower hospital charges. Since 1975, of course, laparoscopic surgical skills and technology have improved dramatically, and the usefulness of laparoscopy in managing patients with acute abdominal pain is generally recognized and accepted.

Laparoscopy has become an important technique in the management of patients with acute abdominal pain. In a study of 255 patients with acute abdomen, laparoscopy

proved helpful.[27] In this set of patients, laparoscopy provided a definitive diagnosis in 93%, and the remaining 7% required laparotomy for diagnosis. The treatment of the acute abdominal pain was exclusively laparoscopic in 73% of the patients, whereas 23% were treated by conventional surgery. Four percent had a combined procedure of conventional surgery assisted by laparoscopy. Eight patients died from the natural course of their disease, five from nonresectable intestinal infarctions, and three from disseminated peritoneal malignant disease. Excluding these patients, the operative mortality was 2%, that is, 5 of 247 cases. One 80-year-old patient had a fatal stroke, an 89-year-old patient who was operated on for a large intestinal obstruction had multiple organ failure, an 82-year-old patient had an intraoperative complication resulting in massive blood loss and died on the 48th postoperative day, and an 89-year-old patient died of a thoracic empyema.

More recently, several authors reported favorable experiences using laparoscopy in the diagnosis and treatment of patients with acute abdominal pain.[28-33] The diagnostic accuracy of laparoscopy varied from 93% to 100%. Laparoscopic techniques accomplished definitive treatment of the underlying disease in 44% to 73% of cases. From 10% to 38% of patients required laparotomy for definitive treatment. In 20% to 38% of patients, laparoscopy revealed either no abnormality or discovered a disease requiring no surgery for proper treatment. The morbidity rates ranged from 0 to 20%, and the mortality rates ranged from 0 to 5%.

Diagnostic and therapeutic laparoscopic techniques have an important place in the management of patients with acute abdominal pain. The diagnostic accuracy spares many patients an unnecessary laparotomy and also allows definitive laparoscopic therapy that prevents additional patients from undergoing unnecessary laparotomy. Evidence suggests that diagnostic laparoscopy reduces the cost of managing patients with acute abdominal pain. Whether diagnostic laparoscopy and therapeutic laparoscopy reduce the cost remains unclear. Most patients with acute abdominal pain should be suitable candidates for laparoscopy. Laparoscopy should be avoided in hemodynamically unstable patients and in patients with extensive gaseous distention of the abdomen. Whether pregnant women with the acute abdomen should undergo laparoscopy is a practical question. One study suggested that laparoscopy in this setting was safe and effective.[34]

Outcomes

It is difficult to know the mortality rate for patients with the acute abdomen. A study from the United Kingdom of patients hospitalized with abdominal pain revealed a mortality rate for all patients of 3.0% and an operative mortality of 7.7%.[35] Another study of 300 consecutive patients undergoing laparotomy within 6 hours of consultation for gastrointestinal perforation, intestinal infarction, or hemorrhage demonstrated a mortality rate of 20%.[36] This study included mostly critically ill patients. Other studies revealed a 16% to 40% mortality rate for emergency in older patients.[35]

ACUTE VISCERAL ISCHEMIA

Although patients experiencing acute visceral ischemia account for a small percentage of the population seeking medical attention for acute abdominal pain, this topic deserves special attention because of extreme difficulty in establishing a correct and timely diagnosis and because the condition has a high mortality rate. Acute arterial disease may be either occlusive or nonocclusive, and venous disease can also produce the syndrome. Arterial occlusion may be either embolic or thrombotic. Generally, acute superior mesenteric artery embolism causes a sudden onset of extremely severe abdominal pain. This ischemic pain persists for a long time before the development of intestinal necrosis. Because the pain results from ischemia and not from peritonitis, these patients have no abdominal tenderness, guarding, or rebound. Therefore, abdominal pain out of proportion to the abdominal physical findings should raise a question about this diagnosis. Because ischemia stops bowel motility promptly, the abdomen may be quiet to auscultation, depending on the amount of ischemic bowel. The heart is the most likely source of a superior mesenteric artery embolus. Therefore, any patient with cardiac arrhythmias, particularly atrial fibrillation, a known mural thrombus, or a recent myocardial infarction who develops acute abdominal pain should have acute superior mesenteric artery embolism high in the differential diagnosis. Patients with atherosclerosis can develop thrombosis at a superior mesenteric artery stenosis. Patients with acute visceral ischemia usually have marked leukocytosis and acidosis. Because cardiovascular disease is important in the development of acute visceral ischemia, most patients with that condition are persons who are middle aged or older.

Conversely, venous thrombosis can cause visceral ischemia, and those patients can be younger. Birth control pills have been implicated in venous thrombosis in young women. Patients suspected of having acute visceral ischemia should undergo arteriography. Although duplex scanning can provide information about the visceral circulation, arteriography provides better images for planning arterial reconstruction or embolectomy. However, arteriography may not help in venous disease. CT scans or magnetic resonance imaging studies can reveal and delineate clots in visceral veins. Most patients with acute visceral ischemia should undergo laparotomy. Some patients develop visceral ischemia because of poor perfusion resulting from decreased cardiac output. Patients usually develop nonocclusive visceral ischemia while they are in the hospital, particularly in an intensive care setting. Improving cardiac output to restore intestinal perfusion is an important step in managing this problem. Arteriography may be required for complete evaluation and allows direct infusion of vasodilators for therapy.

ACUTE ABDOMINAL PAIN

During Pregnancy

The development of acute abdominal pain during pregnancy presents a diagnostic challenge because of the enlarged uterus and the difficulty in evaluating the abdomen.[34] Appendicitis occurs once in 1500 pregnancies, evenly distributed in the trimesters. The diagnosis may be particularly difficult because the pregnant uterus can push the cecum and appendix into the right upper quadrant. Cholecystitis also occurs during pregnancy. Cholecystectomy has been performed in 3% to 8% of 10,000 pregnancies. Other conditions, such as acute pancreatitis and perforated ulcer, occur less frequently. Preeclamptic patients may experience spontaneous rupture of the liver. This is a serious and difficult complication to manage. Other causes of abdominal pain during pregnancy include placental abruption, ruptured uterus, torsion of the ovary, urinary tract infection, and pulmonary embolus.

The pregnant patient with right-sided abdominal pain, tenderness, and guarding should be strongly suspected of having appendicitis. Ultrasound examination may help to detect evidence of appendicitis. In this setting, the patient should undergo operation, probably laparoscopy. The patient and the fetus are likely to face more risk from a ruptured appendix than from the procedure. If possible, surgical treatment of symptomatic cholelithiasis should be avoided during pregnancy. Patients with infrequent, mild, self-limited attacks of right upper quadrant pain should delay the operation until after delivery. If biliary colic becomes disabling but not an emergency, operation should be delayed and performed in the second trimester. Procedures are safer during the second trimester of pregnancy. Procedures during the first trimester pose a risk to the fetus, whereas procedures during the third trimester carry the risk of premature labor. If a pregnant patient with cholelithiasis develops unrelenting right upper quadrant abdominal pain, tenderness, guarding, and fever, she should undergo operation, probably laparoscopic cholecystectomy. Hemodynamic monitoring, perhaps including an arterial line, should be used. When laparoscopy is used, intra-abdominal pressures up to 15 mm Hg should be safe. Carbon dioxide values should be monitored. Fetal heart tones should be monitored, and exsufflation should follow any sign of fetal distress.

The Patient in the Medical Intensive Care Unit

Patients in the medical intensive care unit (MICU) who develop abdominal pain while undergoing treatment for another primary condition pose a common and difficult management challenge. Gajic and associates[37] studied a cohort of 77 abdominal catastrophe patients from 6000 MICU admissions (1.3%). The conditions producing the acute abdomen in that cohort included peptic ulcer, ischemic bowel, cholecystitis, bowel obstruction, and bowel inflammation. The APACHE III score on admission to the MICU predicted a mortality rate of 31% in this group of patients who experienced an actual mortality rate of 63%. The development of an acute abdomen in this setting doubled the mortality risk. All of the 26 patients not undergoing operation died, while 23 of the 51 patients undergoing operation died postoperatively. In the unoperated group some patients were judged too ill for surgery, 2 died during resuscitation, and 3 cases were only diagnosed at autopsy. For the patients undergoing operation significant predictors of mortality included delay in surgical evaluation, delay in surgical intervention, admission APACHE III scores, renal insufficiency, and ischemic bowel. Surgical delay occurred in patients with altered mental state, absence of peritoneal signs, opioid analgesia, antibiotics, and mechanical ventilation. It is noteworthy that in this cohort 84% of patients had abdominal pain, 95% had abdominal tenderness, 73% had abdominal distention, and 33% had free intra-abdominal air on radiograph or CT.

MICU intensivists should maintain a low threshold for obtaining surgical consultation for patients with abdominal pain. The surgeon will approach such patients with high clinical suspicion. Repeated abdominal examinations, radiologic and sonographic investigations, and abdominal paracentesis must be evaluated carefully. Laparoscopy may help in this setting. Gagne and colleagues[38] reported using bedside minilaparoscopy to evaluate abdominal pain in ICU patients. Minilaparoscopy can be performed with a 3.3-mm laparoscope and instruments using local anesthesia and intravenous sedation. In any case, early surgical intervention remains crucial to survival of patients developing the acute abdomen in the MICU.

AIDS, IMMUNOSUPPRESSION, AND THE ACUTE ABDOMEN

The diagnosis and treatment of acute abdominal pain in patients with immunodeficiency pose special problems.[39,40] One must recognize the immunosuppressed patient and determine the degree of immunosuppression.[41] Mild to moderate immunodeficiency occurs in the elderly, the malnourished, the diabetic, the uremic, and patients with malignancy. In addition transplant patients on maintenance immunosuppression therapy and acquired immunodeficiency syndrome (AIDS) patients with $CD4^+$ counts greater than $200/mm^3$ fall in this category. Patients in this mild to moderate immunodeficient category have the same kinds of diagnoses and surgical problems as other patients except they present in later or more advanced stages of the acute abdominal disease. Severe immunodeficiency includes AIDS patients with $CD4^+$ counts less than $200/mm^3$, transplant patients taking high doses or potent immunosuppressants, and cancer patients taking chemotherapy especially if neutropenic. Severe immunodeficient patients with the acute abdomen have unusual diseases and seek medical attention late in the course of their disease. Their symptoms are vague, and they are unlikely to have fever, abdominal tenderness, or guarding. Immunodeficient patients have particular susceptibility to unusual infections caused by fungi, mycobacteria, viruses, and infesta-

tions by parasites. They are also prone to develop unusual malignant tumors.

Parente and coworkers[42] studied 458 AIDS patients hospitalized 752 times over 4 years. Seventy-one of the patients had an episode of abdominal pain severe enough to require surgical consultation. Forty-two of those patients had a premortem diagnosis of the condition causing the pain. Twenty-three patients had the cause of the abdominal pain explained by postmortem examination. The most common causative disorders in decreasing order included gastrointestinal non-Hodgkin's lymphoma, acute pancreatitis, CMV colitis/enteritis, *Mycobacterium avium-intracellulare* colitis/enteritis, sclerosing cholangitis, CMV gastritis, cryptosporidial infection, acute cholecystitis, and gastrointestinal Kaposi's sarcoma. Ten patients underwent emergency laparotomy: six for perforated viscus or peritonitis, two for intestinal obstruction, one for toxic megacolon, and one for hemoperitoneum. The postoperative survival was 40% at 1 month, 30% at 3 months, and 10% at 6 months. The median survival of the abdominal pain patients was 180 days from the diagnosis of AIDS, significantly lower than the median survival rate of the patients without abdominal pain, which was 540 days.

Patients with advanced AIDS are debilitated, malnourished, and catabolic. In addition to being particularly susceptible to unusual bacterial, viral, and fungal agents, these patients are also at risk for the common causes of the acute abdomen. Patients with AIDS are particularly prone to CMV infections, and these infections commonly invade the gastrointestinal tract and produce mucosal ulceration, bleeding, and even perforation. One should avoid surgery in patients with CMV infection unless perforation occurs. If the diagnostic work-up indicates that a patient with AIDS has an acute abdomen from a common AIDS-unrelated disease, he or she should have conventional treatment without delay.

Patients who have received organ transplants, particularly patients taking high doses of steroids, are at risk of developing the same diseases as those that occur in AIDS patients.[8,43] Because immunosuppression obscures the signs and symptoms of intra-abdominal infections and perforation, physicians caring for organ transplant recipients must have a heightened awareness of the serious significance of acute abdominal pain in their patients. Transplant recipients with intestinal perforation, appendicitis, and so forth should be operated on as soon as possible.

Any new complaint of abdominal pain expressed by an immunocompromised patient requires professional attention. Internists and family physicians should consult surgeons promptly in this situation. The surgeon should obtain a careful description of the nature of the pain and its onset. Immunocompromised patients may harbor advanced intra-abdominal disease yet exhibit minimal physical findings including fever, abdominal tenderness, guarding, and rebound. Imaging tests may help with the decision to operate. Although establishing strict criteria or guidelines for operating remains difficult, severely immunocompromised patients with unrelenting abdominal pain should undergo laparoscopy or laparotomy. In such cases the risks of intervention remain far less than the risks of untreated potentially catastrophic disease.

Box 43-5. Nonsurgical Causes of Abdominal Pain

Cardiac
- Myocardial infarction
- Acute pericarditis

Pulmonary
- Pneumonia
- Pulmonary infarction

Gastrointestinal
- Acute pancreatitis
- Gastroenteritis
- Acute hepatitis

Endocrine
- Diabetic ketoacidosis
- Acute adrenal insufficiency

Metabolic
- Acute porphyria
- Familial Mediterranean fever
- Hyperlipidemia

Musculoskeletal
- Rectus muscle hematoma

Central and peripheral nervous system
- Tabes dorsalis
- Nerve root compression

Genitourinary
- Pyelonephritis
- Acute salpingitis

Hematologic
- Sickle cell crisis

NONSURGICAL CAUSES OF ACUTE ABDOMINAL PAIN

Many diseases produce acute abdominal pain and may be treated best by means other than surgery.[44] Certain nonsurgical conditions can cause acute abdominal pain, such as spontaneous bacterial peritonitis, as mentioned earlier (Box 43-5). Sickle cell anemia may produce an attack of severe abdominal pain, referred to as *sickle cell crisis,* and this condition may result from a splenic infarction. These patients also may have attacks of bone and joint pain. Gastroenteritis may produce severe abdominal pain. Patients who develop abdominal pain and who have had a recent exposure to antibiotic therapy may have *Clostridium difficile* colitis or pseudomembranous colitis, which can mimic the acute abdomen. This diagnosis can usually be clarified by a careful history, and sigmoidoscopy reveals the pseudomembrane, which is virtually pathognomonic for the condition. Other diseases, such as lead poisoning, acute porphyria, and familial Mediterranean fever, may also cause abdominal pain. Pneumonia can produce abdominal pain, and of course, acute myocardial infarction may produce epigastric pain and can mimic acute pancreatitis or perforated ulcer. Hepatitis may produce abdominal pain. Acute adrenal insufficiency may cause abdominal pain, and patients with hyperlipidemia may have acute abdominal pain with or without acute pancreatitis.

To manage patients with abdominal pain effectively, the surgeon must always remember that many nonsurgical diseases cause abdominal pain and may mimic the acute abdomen. Surgical and nonsurgical causes of abdominal pain are not mutually exclusive. Patients with sickle cell disease can develop acute cholecystitis or appendicitis. After performing a careful history, physical examination, and imaging tests, the surgeon must evaluate the strength of the evidence that the patient actually has a nonsurgical disease versus the strength of the evidence for an acute surgical abdomen. Diagnostic laparoscopy should find liberal application in this situation.

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