Abdominal pain is an unpleasant experience commonly associated with tissue injury. The sensation of pain represents an interplay of pathophysiologic and psychosocial factors. Physiologic determinants of pain include the nature of the stimuli, the type of receptor involved, the organization of the neuroanatomic pathways from the site of injury to the central nervous system, and a complex interaction of modifying influences on the transmission, interpretation, and reaction to pain messages. Psychosocial factors modifying the sensation of pain include personality, ethnic and cultural background, and circumstances surrounding the injury. Thus, pain represents a complex sensation with different manifestations in different individuals. It is the clinician’s responsibility to interpret the patient’s complaint of pain with complete understanding of factors modifying its sensation and manifestations.

ANATOMIC BASIS OF PAIN

Sensory neuroreceptors in abdominal organs are located within the mucosa and muscularis of hollow viscera, on serosal structures such as the peritoneum, and within the mesentery. In addition to nociception (the perception of noxious stimuli), sensory neuroreceptors are involved in the regulation of secretion, motility, and blood flow via local and central reflex arcs. Although sensory information conveyed in this manner is usually not perceived, disordered regulation of these gastrointestinal functions can cause pain. For example, patients with irritable bowel syndrome perceive pain related to a heightened sensitivity of gut afferent neurons to normal endogenous stimuli, resulting in altered gut motility and secretion (see Chapter 91).

The neuroreceptors involved in nociception are the peripheral ends of two distinct types of afferent nerve fibers: myelinated A-delta fibers and unmyelinated C fibers. A-delta fibers are distributed principally to skin and muscle and mediate the sharp, sudden, well-localized pain that follows an acute injury. These fibers convey somatotopical pain sensations through spinal nerves. C fibers are found in muscle, periosteum, mesentery, peritoneum, and viscera. Most nociception from abdominal viscera is conveyed by this type of fiber and tends to be dull, burning, poorly localized, more gradual in onset, and longer in duration. These C fibers utilize substance P and calcitonin gene-related peptide as neurotransmitters. Stimulation of these fibers activates local regulatory reflexes mediated by the enteric nervous system and long spinal reflexes mediated by the autonomic nervous system, in addition to the transmission of pain sensation to the central nervous system.

Afferent pain impulses are modified by inhibitory mechanisms at the level of the spinal cord. Somatic A-delta fibers mediating touch, vibration, and proprioception from a dermatomal distribution matching the visceral innervation of the injured viscera synapse with inhibitory interneurons of the
substantia gelatinosa in the spinal cord. In addition, inhibitory neurons originating in the mesencephalon, periventricular gray matter, and caudate nucleus descend within the cord to modulate afferent pain pathways. These inhibitory mechanisms allow cerebral influences to modify afferent pain impulses.  

STIMULANTS OF PAIN

Abdominal visceral nociceptors respond to mechanical and chemical stimuli. The principal mechanical signal to which visceral nociceptors are sensitive is stretch. Unlike for somatoparietal nociceptors, cutting, tearing, or crushing of viscera does not result in pain. Visceral stretch receptors are located in the muscular layers of the hollow viscera, between the muscularis mucosa and submucosa, in the serosa of solid organs, and in the mesentery (especially adjacent to large vessels).  

Mechanoreceptor stimulation can result from rapid distention of a hollow viscus (e.g., intestinal obstruction), forceful muscular contractions (e.g., biliary or renal colic), and rapid stretching of solid organ serosa or capsule (e.g., hepatic congestion). Similarly, torsion of the mesentery (e.g., cecal volvulus) or tension from traction on the mesentery or mesenteric vessels (e.g., retroperitoneal or pancreatic tumor) results in stimulation of mesenteric stretch receptors.

Abdominal visceral nociceptors also respond to various chemical stimuli. Chemical nociceptors are contained mainly within the mucosa and submucosa of the hollow viscus. These receptors are directly activated by substances releasing in response to local mechanical injury, inflammation, ischemia and necrosis, and noxious thermal or radiatioinjury. Such substances include H+ and K+ ions, histamine, serotonin, bradykinin and other vasoactive amines, substance P, calcitonin gene-related peptide, prostaglandins, leukotrienes.  

Accumulation of nocireactive substances may change the microenvironment of the injured tissue, resulting in a reduction of the pain threshold. This increases the sensation of pain to a given stimulus and makes otherwise innocuous stimuli painful. For example, the use of chemical irritants or pressure on normal gastric mucosa is not painful, whereas the application of the same stimulant to an inflamed or injured mucosa causes pain.

TYPES OF PAIN

Abdominal pain may be classified into three categories: visceral pain, somatoparietal pain, and referred pain.  

Visceral pain is experienced when noxious stimuli to visceral nociceptors. The pain is usually dull and per localized in the midline epigastrium, periumbilical region, or lower midabdomen because abdominal organs transmit sensory afferents to both sides of the spinal cord (Fig. 4-1). The site where the pain is felt corresponds roughly to dermatomes appropriate to the diseased organ's innerv
The pain is not well localized because the innervation of most viscera is multisegmental and the number of nerve endings in viscera is lower than that in highly sensitive organs such as the skin. The pain is generally described as cramping, burning, or gnawing. Secondary autonomic effects such as sweating, restlessness, nausea, vomiting, perspiration, and pallor often accompany visceral pain. The patient may move about in an effort to relieve the discomfort.

Somatoparietal pain arises from noxious stimulation of the parietal peritoneum and is generally more intense and more precisely localized than visceral pain. An example of this difference is the early vague periumbilical visceral pain in acute appendicitis, which is followed by the localized somatoparietal pain at McBurney's point produced by inflammatory involvement of the parietal peritoneum. Parietal pain is usually aggravated by movement or coughing. The nerve impulses mediating parietal pain travel within somatic sensory spinal nerves. The fibers reach the spinal cord in the peripheral nerves corresponding to the cutaneous dermatomes from the sixth thoracic (T6) to the first lumbar (L1) region. Lateralization of the discomfort of parietal pain is possible because only one side of the nervous system innervates a given part of the parietal peritoneum.

Referred pain is felt in areas remote to the diseased organ. It is a result of convergence of visceral afferent neurons with somatic afferent neurons from different anatomic regions on second-order neurons in the spinal cord at the same spinal segment. Referred pain may be felt in skin or deeper tissues but is usually well localized. Generally, referred pain appears as the noxious visceral stimulus becomes more intense. An example is illustrated in Figure 4–3, in which diaphragmatic irritation from a subphrenic hematoma or abscess results in shoulder pain.

**APPRAOH TO THE PATIENT WITH ACUTE ABDOMINAL PAIN**

The goal of the evaluation of the patient with acute abdominal pain is an early, efficient, and accurate diagnosis. When evaluating a patient with acute abdominal pain, the most important elements in making an accurate early diagnosis are the history and physical examination. A careful description of the chronology, location, intensity, and character of the pain as well as aggravating and alleviating factors, other symptoms, and other medical history usually allows an accurate diagnosis to be made. A thorough physical examination can confirm a diagnosis that was suspected during the history. Finally, selective use of appropriate laboratory and radiographic examinations provides further objective evidence in support of a specific diagnosis.

In some cases, the diagnosis is obscure despite an exhaustive evaluation. In most settings in which the patient’s clinical status is stable, repetitive examination over time eliminates diagnostic uncertainty. In this situation, admission to the hospital for serial abdominal examinations or close telephone or office follow-up is necessary. When the patient’s clinical status is deteriorating and diagnostic uncertainty remains, surgical exploration may be necessary.

**Clinical Evaluation**

**History**

The most important part of the evaluation of a patient with abdominal pain is the history (Table 4–1).
M: many causes of abdominal pain have a catastrophic onset, such as ruptured aortic aneurysm. D: Certain conditions, such as diverticulitis or appendicitis, are progressive, as with appendicitis or diverticulitis. C: Commonly, abdominal pain may vary widely from minutes in intestinal and renal colic to days, weeks, or even months in biliary colic. Some pain is colicky (i.e., the pain progresses and remits over time); examples include intestinal, renal, and biliary colic. The time course may vary widely from minutes in intestinal and renal colic to days, weeks, or even months in biliary colic. C: Commonly, abdominal pain is progressive, as with appendicitis or diverticulitis. D: Certain conditions have a catastrophic onset, such as ruptured aortic aneurysm.

**Table 4-1 | Comparison of Common Causes of Acute Abdominal Pain**

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>ONSET</th>
<th>LOCATION</th>
<th>CHARACTER</th>
<th>DESCRIPTOR</th>
<th>RADIATION</th>
<th>INTENSITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appendicitis</td>
<td>Gradual</td>
<td>Periumbilical early; RLQ late</td>
<td>Diffuse, early, localized</td>
<td>Ache</td>
<td>RLQ</td>
<td>+</td>
</tr>
<tr>
<td>Cholecystitis</td>
<td>Rapid</td>
<td>RUQ</td>
<td>Localized</td>
<td>Constricting</td>
<td>Scapula</td>
<td>++</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Rapid</td>
<td>Epigastric, back</td>
<td>Localized</td>
<td>Boring</td>
<td>Midback</td>
<td>+ to ++</td>
</tr>
<tr>
<td>Diverticulitis</td>
<td>Gradual</td>
<td>LLQ</td>
<td>Localized</td>
<td>Ache</td>
<td>None</td>
<td>+ to ++</td>
</tr>
<tr>
<td>Perforated peptic ulcer</td>
<td>Sudden</td>
<td>Epigastric</td>
<td>Localized, early, diffuse</td>
<td>Burning</td>
<td>None</td>
<td>+++</td>
</tr>
<tr>
<td>Small bowel obstruction</td>
<td>Gradual</td>
<td>Periumbilical</td>
<td>Diffuse</td>
<td>Crampy</td>
<td>None</td>
<td>++</td>
</tr>
<tr>
<td>Mesenteric ischemia/infarction</td>
<td>Sudden</td>
<td>Periumbilical</td>
<td>Diffuse</td>
<td>Agonizing</td>
<td>None</td>
<td>+++</td>
</tr>
<tr>
<td>Ruptured abdominal aortic aneurysm</td>
<td>Sudden</td>
<td>Abdominal, back, flank</td>
<td>Diffuse</td>
<td>Tearing</td>
<td>Back, flank</td>
<td>+++</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>Gradual</td>
<td>Periumbilical</td>
<td>Diffuse</td>
<td>Spasmodic</td>
<td>None</td>
<td>+ to ++</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>Gradual</td>
<td>Either LQ, pelvic</td>
<td>Localized</td>
<td>Ache</td>
<td>Upper thigh</td>
<td>++</td>
</tr>
<tr>
<td>Ruptured ectopic pregnancy</td>
<td>Sudden</td>
<td>Either LQ, pelvic</td>
<td>Localized</td>
<td>Light-headed</td>
<td>None</td>
<td>++</td>
</tr>
</tbody>
</table>

**CHRONOLOGY.** Temporal considerations in the evaluation of a patient with acute abdominal pain include the rapidity of onset and the progression and duration of symptoms (Fig. 4-4). The rapidity of onset of pain is often a measure of its significance. Pain that is sudden in onset, severe, and well localized is likely to be the result of an intra-abdominal catastrophe such as a perforated viscus, mesenteric infarction, or ruptured aneurysm. Such patients usually recall the exact moment of onset of their pain. A second important temporal factor in abdominal pain is its progression. Pain in some disorders, such as gastroenteritis, is self-limited, whereas in others, such as appendicitis, it is progressive. Colicky pain has a crescendo-decrescendo pattern that may be diagnostic, such as in renal colic. The duration of abdominal pain is also important. Patients who seek evaluation of abdominal pain that has been present for an extended period (e.g., weeks) are less likely to have an acutely threatening illness than are those who do so within hours to days of the onset of symptoms.

**LOCATION.** The location of abdominal pain provides clues when interpreting the cause. As previously discussed, a given noxious stimulus may result in a combination of visceral, somatoparietal, and referred pain. This may create confusion in interpretation unless the neuroanatomic pathways are considered. For example, the pain of diaphragmatic irritation from a left-sided subphrenic abscess may be referred to the shoulder and misinterpreted as the pain from ischemic heart disease. Changes in location may represent progression from visceral to parietal irritation, as in appendicitis, or represent the development of diffuse peritoneal irritation, as with a perforated ulcer.

**INTENSITY AND CHARACTER.** The intensity of pain is difficult to measure. Perception of intensity is dependent upon the point of reference from which the patient is describing the pain. This point of reference varies among individuals and depends on the setting in which the pain is occurring, past experience with various types of pain, personality, and cultural differences. For these reasons, estimates of pain severity are not uniformly reliable diagnostic clues. However, the severity of the pain is loosely related to the magnitude of the noxious stimulus. Several classic descriptors have been assigned to certain acute abdominal conditions (see Table 4-1). The clinician should be cautious, however, to prevent assigning too much importance to descriptions of pain. It must be recognized that the exceptions may outnumber the rule and a given description may be applied to a number of conditions.

**AGGRAVATING AND ALLEVIATING FACTORS.** The setting in which pain occurs or is exacerbated may yield important diagnostic information. The relationship to positional changes, meals, bowel movements, and stress may be significant. Patients with peritonitis, for example, lie motionless, whereas those with renal colic may writhe in an attempt to find a comfortable position. Sometimes, certain foods exacerbate pain. A classic example is the relationship between fatty foods and the development of biliary colic. Pain associ-
ated with duodenal ulcer is often alleviated by meals. In contrast, patients with gastric ulcer or chronic mesenteric ischemia may report exacerbation of pain with eating. Patients often self-medicate to alleviate symptoms. A history of chronic antacid use, for example, may suggest the presence of peptic ulcer disease.

ASSOCIATED SYMPTOMS AND REVIEW OF SYSTEMS. A careful history of other symptoms coexisting with the presentation of abdominal pain should be elicited. Information regarding changes in constitutional symptoms (e.g., fever, chills, night sweats, weight loss, myalgias, arthralgias), digestive function (e.g., anorexia, nausea, vomiting, flatus, diarrhea, and constipation), jaundice, dysuria, menstruation, and pregnancy should be solicited. A careful review of these symptoms may reveal important diagnostic information. For example, vomitus that is clear suggests gastric outlet obstruction, whereas feculent vomitus suggests more distal small bowel or colonic obstruction. A constellation of findings may indicate a particular disease entity.

PAST MEDICAL HISTORY. A careful review of the patient’s other medical problems often sheds light on the current presentation of acute abdominal pain. Previous experience with similar symptoms suggests a recurrent problem. Patients with a history of partial small bowel obstructions, renal calculi, or pelvic inflammatory disease are likely to have recurrences. Systemic illnesses such as scleroderma, lupus, nephrotic syndrome, porphyrias, and sickle cell disease often have abdominal pain as a manifestation of their illness. Abdominal pain may also arise as a side effect of medication taken for other illness.

FAMILY AND SOCIAL HISTORY. A careful review of the patient’s family history may yield information relevant to the diagnosis. This is especially true in the pediatric population. Sickle cell disease in black patients and familial Mediterranean fever in patients of Armenian or Sephardic Jewish heritage are examples. Likewise, the patient’s social history, including habits or history of substance abuse, occupational history, travel history, and history of contact with animals or other ill people, may provide useful diagnostic information.

**Physical Examination**

The physical examination must be pursued systematically to test specific hypotheses formed while eliciting the history and to uncover unsuspected abnormalities. When examining a patient, the clinician must interpret his or her findings in the context of the patient’s history. For example, the elderly, immunocompromised, or long-term diabetic patient is less likely to show signs of peritoneal irritation, even in the presence of a perforated viscus. When the source of the pain is intra-abdominal, many important clues are derived from a complete physical examination. Therefore, a careful systemic examination must be performed, in addition to a thorough abdominal examination.

SYSTEMIC EXAMINATION. The physical examination begins with an assessment of the patient’s appearance, ability to converse, breathing pattern, position in bed, posture, degree of discomfort, and facial expression. A patient lying still in bed, in the fetal position, reluctant to move or speak, with a distressed facial expression is likely to have peritonitis. On the other hand, patients who writhe with frequent position changes likely have pure visceral pain, such as in bowel obstruction or gastroenteritis. Vital signs should be obtained to exclude conditions such as hypovolemia, tachypnea related to metabolic acidosis, or atrial fibrillation as a cause of mesenteric arterial embolus. Careful lung examination may yield findings suggestive of pneumonia. Examination of the extremities may provide evidence for inadequate perfusion, as in shock, or the presence of chronic vascular disease.

ABDOMINAL EXAMINATION. An assessment of the degree of tenderness and its location must be made in each patient with abdominal pain. Severe diffuse tenderness with rigidity suggests generalized peritonitis. Mild tenderness without signs of peritoneal irritation is more characteristic of conditions that do not require surgical treatment (e.g., salpingitis and gastroenteritis). The abdomen should be inspected for distention, scars, hernias, muscle rigidity, splinting during respiration, ecchymoses, and visible hyperperistalsis. Hyperperistalsis may be detected by auscultation in patients with intestinal obstruction or enteritis. Generalized peritonitis usually causes diminished peristalsis. Bruits may point to a vascular stenosis. Abdominal percussion may elicit tympany from excess abdominal gas, whether it is intraluminal (as occurs with intestinal obstruction) or extraluminal (as occurs with perforated viscus). Light, gentle palpation is superior to deep palpation in the identification of peritoneal irritation. Peritonitis may also be detected through innocuous measures such as gently shaking the bed or asking the patient to breathe deeply or cough. Palpation should begin at the point of least tenderness and proceed to the point of greatest tenderness. The degree of tenderness, guarding, and rigidity should be determined. Enlargement of a diseased organ, tumor, or inflammation may produce a palpable mass. Potential hernia orifices should be examined.

GENITAL, RECTAL, AND PELVIC EXAMINATION. The pelvic organs and external genitalia should be examined in every patient with abdominal pain. The rectum and vagina provide additional avenues for gentle palpation of pelvic viscera. The presence of gynecologic abnormality should be excluded in all women with abdominal pain.

**Laboratory Data**

Laboratory tests ordered should reflect the clinical suspicion raised during the history and physical examination. Unnecessary laboratory testing is costly and often clouds the diagnostic picture. All patients with acute abdominal pain should have a complete blood count with differential count and urinalysis. The determination of serum electrolyte, blood urea nitrogen, creatinine, and glucose concentrations is useful in ascertaining fluid status, acid-base status, renal function, and metabolic state but is not necessary for every patient. Urine or serum pregnancy testing should be performed in all women of reproductive age with lower abdominal pain. Liver function tests and serum amylase determination should be ordered in patients with upper abdominal pain. Other tests are obtained on the basis of clinical history (e.g., prothrombin time and a blood albumin in patients with suspected liver disease).
Radiographic Evaluation

As is the case with laboratory tests, diagnostic imaging must be tailored to answer specific questions arising from a carefully derived differential diagnosis based on history, physical examination, and laboratory testing. A patient who has a clinical picture suggestive of a bowel obstruction, for example, is best served by obtaining plain radiographs of the abdomen, whereas a patient with suspected acute cholecystitis is best evaluated via ultrasonography.

The most common imaging examination ordered in the evaluation of the patient with acute abdominal pain is the plain abdominal series. This should include two views of the abdomen: one in the supine and one in the upright position. In patients unable to sit upright, a lateral decubitus film with the left side down may identify abnormal gas patterns. In addition, an upright chest radiograph should be performed to exclude intrathoracic causes of abdominal pain (e.g., lower lobe pneumonia) and pneumoperitoneum (Fig. 4–5). Only 10% of abdominal radiographs reveal findings diagnostic of abdominal abnormality. Even so, the examination is readily available and inexpensive and should be obtained in most circumstances.15

Ultrasoundography can provide rapid, accurate, and inexpensive anatomic information about the liver, biliary tree, spleen, pancreas, kidneys, and pelvic organs. In some cases (e.g., biliary colic, cholecystitis, ectopic pregnancy, ovarian cyst, or tubo-ovarian abscess), ultrasonography is the preferred initial imaging test. Endovaginal and endorectal ultrasonography can be useful in identifying pelvic abnormalities not seen by other imaging modalities. Doppler technology permits evaluation of vascular lesions such as aortic or visceral aneurysms, venous thrombi, and anomalies.16

The most versatile imaging tool in the evaluation of acute abdominal pain is computed tomography (CT). CT of the abdomen and pelvis provides information about the presence of pneumoperitoneum, abnormal bowel gas patterns, and calcifications similar to that obtained with plain radiographs. In addition, CT permits detection of inflammatory lesions (e.g., appendicitis, diverticulitis, pancreatitis, and abscesses), neoplastic lesions (e.g., obstructing colon cancer, pancreatic tumors), and trauma (e.g., spleen, liver, and kidney injury). CT also provides information about vascular lesions (e.g., portal vein thrombosis, pylephlebitis, aneurysm disease) and intra-abdominal or retroperitoneal hemorrhage (e.g., trauma, adrenal hemorrhage, ruptured hepatoma).16, 17

Recent improvements in CT technology have enhanced image resolution and expanded its utility. Helical CT examinations generate high-resolution images rapidly and have largely replaced previous techniques. These improvements in CT technology include focused or organ-specific examinations and CT angiography. An example of a focused examination is the focused helical CT of the appendix. In a 1998 study of 100 patients with clinically suspected appendicitis, treatment plans were altered in over half of the patients and unnecessary appendectomy was prevented in 13 patients. In this study, the savings realized exceeded the cost of the scans by $447 per patient, making the focused examination highly cost-effective.18 Other examples of focused helical CT examinations include the use of organ-specific protocols in which timing of contrast ingestion or injection is coordinated with image acquisition to optimize visualization of specific organs. Examples of these techniques include CT of the esophagus or upper abdomen with oral contrast medium to evaluate perforated viscera and CT of the pancreas or liver in which the image acquisition is coordinated with the arterial and/or venous phase to evaluate for perfusion defects from ischemia, trauma, or neoplasia. CT arteriography is useful in evaluating the aorta and visceral vasculature. These improvements in technology make CT the most versatile adjunct to the clinical history and physical examination in the evaluation of the acute abdomen.

Other imaging modalities are occasionally useful in the evaluation of the patient with acute abdominal pain. These include magnetic resonance imaging (MRI) angiography and endoscopy. The former is a useful, noninvasive method to evaluate the visceral vasculature. Endoscopy can be useful in the evaluation of the stomach, duodenum, and colon mucosa for ulceration, neoplasia, ischemia, and inflammation.

Other Diagnostic Tests

Other diagnostic tests occasionally useful in evaluating the patient with acute abdominal pain include peritoneal lavage, laparoscopy, and exploratory laparotomy. Peritoneal lavage is useful in detecting the presence of hemo peritoneum after blunt or penetrating trauma and of purulent or feculent material after hollow viscus injury, ischemia, or perforation.

Diagnostic laparoscopy is useful when diagnostic uncertainty exists and the patient's clinical condition demands intervention. Recent improvements in minimally invasive technology and techniques have increased the utility of laparoscopy in the evaluation and treatment of the acute abdomen. Refinements and miniaturization of instruments, the use of laparoscopic ultrasonography, and increased experience
with advanced laparoscopic techniques have led to a wider application of minimally invasive surgery to the evaluation and treatment of almost all intra-abdominal diseases, including most causes of the acute abdomen. For example, in a female patient of reproductive years with obvious peritonitis localized to the right lower quadrant, diagnostic laparoscopy permits differentiation between adnexal disease and acute appendicitis. In addition to its diagnostic usefulness, laparoscopy can be used in the treatment of these disorders. Since the late 1990s, the addition of laparoscopic ultrasonography technology has improved the evaluation of the solid visceral organs and retroperitoneum. The diagnostic accuracy of laparoscopy in patients with acute nontraumatic abdominal pain is 93% to 98%. In various series, 57% to 77% of patients who underwent diagnostic laparoscopy for acute abdominal pain were successfully treated by laparoscopic or laparoscopically assisted methods.²⁹-²¹

Exploratory laparotomy is reserved for patients with intra-abdominal catastrophe whose diagnosis is obvious from the clinical history and examination (e.g., ruptured spleen from blunt trauma, ruptured abdominal aortic aneurysm) or patients in extremis in whom delay in therapy would be life-threatening.

Intra-abdominal Causes of the Acute Abdomen

Acute abdominal pain is pain of less than 24 hours' duration. It has many causes, and only after a careful history, physical examination, and appropriate laboratory and radiographic examination can the clinician differentiate between those conditions that require surgery and those that can be treated nonoperatively. Acute abdomen, therefore, does not mandate surgery. If, after the initial evaluation, the diagnosis is unclear, periodic physical and laboratory re-examination can often eliminate any uncertainty. The list of intra-abdominal causes of the acute abdomen is exhaustive. In this chapter, the most common causes are discussed; for more detailed information, refer to the corresponding organ system chapters.

ACUTE APPENDICITIS. Acute appendicitis begins with prodromal symptoms of anorexia, nausea, and vague periumbilical pain. Within 6 to 8 hours, the pain migrates to the right lower quadrant and peritoneal signs develop. In uncomplicated appendicitis, a low-grade fever to 38°C and mild leukocytosis are usually present. Higher temperatures and white blood cell counts are associated with perforation and abscess formation. The mnemonic "PANT" can help the novice remember the classic progression of symptoms in appendicitis (pain followed by anorexia followed by nausea followed by temperature elevation). Plain abdominal radiographs are not diagnostic, but suggestive findings include a localized right lower quadrant ileus, an appendicolith, or spasm of the right psoas muscle. Ultrasonography and CT are useful diagnostic adjuncts in selected patients. Treatment of uncomplicated cases is via appendectomy (see Chapter 107).

ACUTE CHOLECYSTITIS. Acute cholecystitis is caused by gallstone obstruction of the cystic duct, except in acalculous cholecystitis, which may be a result of gallbladder ischemia, stasis, or viral infection. Acute cholecystitis causes pain that is almost indistinguishable from the pain of biliary colic. The pain is usually a persistent, dull ache. It is usually localized to the right upper quadrant or epigastrium but may radiate around the back to the right scapula. The pain usually subsides within 6 hours of onset in biliary colic but persists in acute cholecystitis. Nausea, vomiting, and low-grade fever are commonly present. On examination, right upper quadrant tenderness, guarding, and Murphy’s sign are diagnostic of acute cholecystitis. The white blood cell count is usually mildly elevated, although it may be normal. Mild elevations in total bilirubin and alkaline phosphatase concentrations are typical. More marked liver function test result abnormalities are associated with choledocholithiasis, Mirizzi’s syndrome, or hepatitis. Acute cholecystitis may be differentiated from cholangitis by the high fevers (especially with chills), jaundice, and leukocytosis observed in the latter. Treatment of acute cholecystitis includes intravenous fluid replacement, antibiotics, bowel rest, and early laparoscopic cholecystectomy (see Chapters 55 to 58).

ACUTE PANCREATITIS. Pancreatitis typically begins with the acute onset of epigastric and upper abdominal pain, which rapidly increases in severity. The pain may bore through to the back or be referred to the left scapular region. The pain is constant and unremitting. Fever, anorexia, nausea, and vomiting are typical. Physical examination reveals an acutely ill patient in considerable distress. Patients are usually tachycardic and tachypneic. Hypotension is a late finding, related to extravasation of intravascular fluid and/or hemorrhage. Abdominal examination reveals hypoaic bowel sounds and marked tenderness to percussion and palpation in the epigastrium. Abdominal rigidity is a variable finding. In rare cases, flank or periumbilical ecchymoses (Turner’s and Cullen’s signs) develop in the setting of hemorrhagic pancreatitis. Extremities are often cool and cyanotic, reflecting underperfusion. White blood cell counts of 12,000 to 20,000/µL are common. Elevated serum and urine amylase levels are usually present within the first few hours. Other useful laboratory tests include concentrations of serum electrolytes, including calcium, liver function tests; levels of blood glucose; and arterial blood gas evaluation. Plain abdominal films may show a “cutoff sign” or sentinel loop and may exclude other causes of pain, including perforated peptic ulcer. Ultrasonography is useful in identifying gallstones as the cause of pancreatitis. Computed tomography is reserved for complicated pancreatitis (see Chapter 48).

ACUTE DIVERTICULITIS. Acute diverticulitis is a disease common in the older population. Although the entire colon may be involved with diverticula, diverticulitis most often occurs in the sigmoid colon. Symptoms relate to inflammation or obstruction. Early in the course of diverticulitis, patients describe mild anorexia, nausea, vomiting, and a visceral-type pain located in the hypogastrium. Later, with the onset of somatoparietal irritation, the pain shifts to the left lower quadrant. Obstipation or diarrhea may be present. Fever is common. Abdominal examination reveals slight distention with left lower quadrant tenderness and guarding. A mass is sometimes palpable. Leukocytosis is present. Abdominal radiography may rule out perforation or obstruction. CT is useful to define the extent of inflammation and exclude the presence of abscess or underlying perforated cancer. Barium enema and colonoscopy are contraindicated dur-
ing the acute illness. Colonoscopy performed 4 to 6 weeks later is recommended, however, to define the extent of diverticula and exclude other colonic abnormalities, especially neoplasm. Treatment is supportive with bowel rest and antibiotics. Surgery is reserved for patients with obstruction, failure of conservative therapy, or recurrent episodes (see Chapter 108).

PERFORATED DUODENAL ULCER. Perforation due to duodenal ulcer usually occurs in the anterior portion of the first part of the duodenum. The pain is sudden, sharp, and severe. At first, it is located in the epigastrium, but it quickly spreads over the entire abdomen, especially along the right side, as the chemical peritonitis descends down the right periolic gutter, where it can mimic appendicitis (Valentino’s syndrome). Nausea is common. The patient typically lies motionless, but in obvious distress. Tachypnea and tachycardia are present early. Hypotension and fever develop 4 to 6 hours into the illness. Examination reveals diffuse peritonitis, with a characteristic “board-like” abdomen caused by involuntary guarding. Laboratory study findings reveal leukocytosis and volume depletion. Pneumoperitoneum is identified on abdominal radiographs in 75% of patients. In equivocal cases, water-soluble contrast studies or computed tomography reveals localized perforation. Most patients require immediate surgery (see Chapters 40 and 42).

SMALL BOWEL OBSTRUCTION. Intestinal obstruction occurs in patients of all ages. In pediatric patients, intussusception, atresia, and meconium ileus are the most common causes. In adults, about 70% of cases are caused by postoperative adhesions. Incarcerated hernias make up the majority of the remainder. Small bowel obstruction is characterized by sudden, sharp periumbilical abdominal pain. Nausea and vomiting occur soon after the onset of pain and provide temporary relief of discomfort. Frequent bilious emesis with epigastric pain is suggestive of high intestinal obstruction. In contrast, crampy periumbilical pain with infrequent feculent emesis is more typical of distal obstruction. Examination reveals an acutely ill, restless patient. Fever, tachycardia, and orthostatic hypotension are common. Abdominal distension is usually present with hyperactive bowel sounds and audible rushes. Diffuse tenderness to percussion and palpation is present, but peritoneal signs are absent, unless a complication such as ischemia or perforation has occurred. Leukocytosis suggests the presence of ischemia. Plain radiographs are diagnostic when they reveal dilated loops of small bowel with air-fluid levels and decompressed distal small bowel and colon. Plain films can be misleading in patients with proximal jejunal obstruction, as dilated bowel loops and air-fluid levels may be absent. Treatment is surgical (see Chapter 109).

ACUTE MESENTERIC ISCHEMIA. Acute ischemic syndromes include embolic arterial occlusion, thrombotic arterial occlusion, nonocclusive mesenteric ischemia, and venous thrombosis. An antecedent history of “intestinal angina,” weight loss, diarrhea, abdominal bruise, cardiac arrhythmias, coronary or peripheral vascular disease, and valvular heart disease is common. The hallmark of the diagnosis is acute onset of crampy epigastric and periumbilical pain out of proportion to the physical findings. Other symptoms include diarrhea, vomiting, bloating, and melena. On examination, most patients appear acutely ill; however, the presentation may be subtle. Shock is present in about 25% of cases. Peritoneal signs usually denote intestinal infarction. Leukocytosis and hemoconcentration are present. Metabolic acidosis is a late finding. CT is the best initial diagnostic test. Visceral angiography may be useful to differentiate the causes of intestinal ischemia and define the extent of disease. Immediate surgery is mandated except in nonocclusive mesenteric ischemia (see Chapter 119).

ABDOMINAL AORTIC ANEURYSM. Rupture or dissection of an abdominal aortic aneurysm is heralded by acute, sudden-onset, severe abdominal pain localized to the midesophageal, paraaortic, or flank area. The pain is tearing in nature and associated with light-headedness, diaphoresis, and nausea. If the patient survives transit to the hospital, shock is the most common presentation. Physical examination reveals a pulsatile, tender abdominal mass in about 90% of cases. The classic triad of hypotension, a pulsatile mass, and abdominal pain is present in 75% of cases. Once the clinical diagnosis is made, emergency surgery is required.5

OTHER CAUSES. Other intra-abdominal causes of acute abdominal pain include gynecologic conditions such as endometritis, acute salpingitis with or without tubo-ovarian abscess, ovarian cysts or torsion, and ectopic pregnancy; spontaneous bacterial peritonitis (see Chapter 78); peptic ulcer disease and nonulcer dyspepsia (see Chapters 7 and 40); gastroenteritis (see Chapters 96 and 97); viral hepatitis and liver infections (see Chapters 68 and 69); pyelonephritis; cystitis; mesenteric lymphadenitis; inflammatory bowel disease (see Chapters 103 and 104); and functional abnormalities such as irritable bowel syndrome (see Chapter 91) and intestinal pseudo-obstruction (see Chapter 111).

Extra-abdominal Causes of Acute Abdominal Pain

Acute abdominal pain may arise from disorders involving extra-abdominal organs and systemic illnesses. Examples are summarized in Table 4–2. Surgical intervention in patients with acute abdominal pain arising from extra-abdominal or systemic illnesses is seldom required. Instances in which surgery is required include pneumothorax, empyema, and esophageal perforation. The latter may be iatrogenic, result from blunt or penetrating trauma, or occur spontaneously (Boerhaave’s syndrome) (see Chapter 34).

Special Circumstances

EXTREMES OF AGE. Evaluation of acute abdominal pain in patients at the extremes of age is a challenge. Historical information and physical examination findings are often difficult to elicit and/or unreliable. Similarly, laboratory findings may be misleadingly normal in the face of significant intra-abdominal abnormality. For these reasons, patients at the extremes of age are often diagnosed late in their disease course and have higher rates of morbidity. For example, the perforation rate for appendicitis in the general population averages 10%, but it exceeds 50% in infants. A careful history, thorough physical examination, and high level of
suspicion are the most useful tools in aiding diagnosis. The presentation of acute abdominal conditions is highly variable in these populations and alert observation is required.

In the pediatric population, the causes of acute abdominal pain vary with age. In infancy, intussusception, pylonephritis, gastroesophageal reflux, Meckel’s diverticulitis, and bacterial or viral enteritis are common. In children, Meckel’s diverticulitis, cystitis, pneumonitis, enteritis, mesenteric lymphadenitis, and inflammatory bowel disease are prevalent. In adolescents, pelvic inflammatory disease, inflammatory bowel disease, and the common adult causes of acute abdominal pain prevail. In children of all ages, two of the most common causes of pain are acute appendicitis and abdominal trauma secondary to child abuse.26, 27

In the geriatric population, biliary tract disease accounts for nearly 25% of cases of acute abdominal pain, followed by nonspecific pain, malignancy, bowel obstruction, complicated peptic ulcer disease, and incarcerated hernias. Appendicitis, although rare in elderly patients, usually becomes apparent late in its course with high morbidity and mortality rates.13, 28

PREGNANCY. Pregnancy poses unique problems in the evaluation of the patient with acute abdominal pain. In pregnancy, the enlarged uterus displaces lower abdominal organs from their usual position, compromises abdominal examination, alters clinical manifestations, and interferes with natural mechanisms that localize infection. Acute abdominal pain in pregnant patients results from diseases similar to those that affect age-matched nonpregnant counterparts and with equal frequency. The most common causes of acute abdominal pain in pregnancy are appendicitis, cholecystitis, pylonephritis, and adnexal problems, including ovarian torsion and ovarian cyst rupture. The rate of fetal loss in intra-abdominal disease is related more to the severity of the disease than to the treatment, including surgery. Therefore, early diagnosis and therapy are indicated. Appendicitis, for example, occurs in about 7 of every 10,000 pregnant women. Appendectomy for uncomplicated appendicitis results in a 3% fetal loss rate, which increases to 20% in perforated appendicitis. After cholecystectomy, the rate of preterm labor is about 7% and the rate of fetal loss is 8%.29, 30

IMMUNOCOMPROMISED HOST. The immunocompromised patient population includes patients undergoing organ transplantation, chemotherapy for cancer, and immunosuppressive therapy for autoimmune disease and those with congenital or acquired immunodeficiency syndromes (see Chapters 27 and 28). As in the elderly population, immunocompromised hosts often demonstrate few abdominal signs and symptoms, minimal systemic manifestations of peritonitis, and little change in laboratory data in the face of acute abdominal pathologic conditions. Therefore, a thoughtful approach to diagnosis is necessary.

Two categories of disease cause acute abdominal pain in these patients: (1) diseases that occur in the general population independently of immune function (e.g., appendicitis, cholecystitis), and (2) diseases unique to the immunocompromised host (e.g., neutropenic enterocolitis, drug-induced pancreatitis, graft-versus-host disease, pneumatoasis intestinalis, cytomegalovirus [CMV], and fungal infections). Intestinal obstruction or perforation is the most common indication for surgery and may occur in the setting of Kaposi’s sarcoma of the intestine, lymphoma or leukemia after chemotherapy, atypical mycobacterial infections, CMV infections, iatrogenic perforations, and neutropenic enterocolitis.31–34

ACUTE ABDOMEN IN THE INTENSIVE CARE UNIT PATIENT. The gastroenterologist or surgeon is occasionally asked to evaluate patients in the intensive care unit for acute abdominal pain or intra-abdominal causes of sepsis. Critical care patients often have altered sensorium as a result of medication, injury, or metabolic disorders. Often one cannot obtain a thorough history and physical examination in these patients. In this situation, a greater reliance on helical CT and diagnostic laparoscopy is necessary. In the intensive care unit an acute abdominal condition unrelated to the main reason for hospitalization may develop. In addition, these patients are at risk for unusual illness related to their hospitalization or underlying condition. Examples of causes of acute abdominal pain in the intensive care unit patient include overlooked trauma injuries; postoperative complications, such as anastomotic leak and obstruction; and complications of critical illness, including acalculous cholecystitis and stress ulcer.
**Pharmacologic Management of the Acute Abdomen**

Early in the course of the evaluation of the patient with acute abdominal pain, the clinician must consider the important role of analgesics and antibiotics in both the evaluation and the early treatment of the underlying problem. Patients with acute abdominal pain are often in great distress, which often obviates their ability to participate in the history and physical examination. Despite data from well-designed studies showing that the administration of analgesics to patients with acute abdominal pain does not adversely affect the clinician's ability to make a timely and accurate diagnosis, 75% of emergency room physicians withhold analgesics pending evaluation of the patient by a surgeon.\(^{35-37}\) This delay results in unnecessary suffering and is not warranted. Patients with moderate to severe abdominal pain should receive analgesics during their evaluation.

Similarly, patients with acute abdominal pain from primary or secondary bacterial peritonitis should receive antibiotics empirically directed against the likely offending organisms. Primary bacterial peritonitis has an extra-abdominal source, often hematogenous, of transmitted bacterial infection to the peritoneal fluid. Examples include spontaneous bacterial peritonitis, tuberculosis peritonitis, and peritonitis associated with chronic ambulatory peritoneal dialysis. In contrast, secondary bacterial peritonitis arises as a consequence of an intra-abdominal process. Causes include appendicitis, diverticulitis, perforated viscus, intestinal ischemia, biliary tract disease, and pelvic inflammatory disease. Although the treatment for secondary peritonitis is usually surgical, appropriate antibiotics should be started soon after the diagnosis is made (see Chapter 121).

**APPROACH TO THE PATIENT WITH CHRONIC ABDOMINAL PAIN**

Chronic abdominal pain is common. A survey of more than 1 million Americans revealed that 13% experienced "stomach pain" and 15% experienced "pain in the lower abdomen."\(^{38}\) Most of these are minor discomforts, but some reflect genuine disease. Causes of chronic abdominal pain may be divided into those that are diagnosable, either intermittent or constant and unremitting, and those that are undiagnosable. Patients with chronic abdominal pain are plagued not only by their symptoms, but also by the disruption of their lives, including increased dependency, altered self-image, and interference with work, family, and social relationships. Often psychologic disturbances, including affective disorders (see Chapters 5 and 122),\(^{8}\) result.

Chronic intractable abdominal pain or chronic undiagnosed abdominal pain is defined as abdominal pain that is present for at least 6 months without diagnosis despite appropriate evaluation. Women are more likely to be afflicted than men. A history of sexual or physical abuse is common. The pain is described in vague, peculiar terms; is exacerbated by psychologic stresses; and is associated with multiple somatic complaints. It is unresponsive to standard treatment and often provokes multiple unnecessary procedures. The pain is disruptive to the patient's relationships and work and often leads to depression, anxiety, illness behavior, and somatoform disorders or traits.\(^{39,40}\) (See Chapter 5 for a more detailed discussion.)

The remainder of this chapter focuses on the evaluation of diagnosable causes of chronic abdominal pain.

**Clinical Evaluation**

**History**

The pattern of chronic diagnosable abdominal pain may be intermittent or constant. Pain that is intermittent is characterized by episodes that last minutes or hours to several days separated by pain-free periods. In these patients, a careful history of the chronology, location, character, and aggravating and alleviating factors often narrows the differential diagnosis. Description of associated symptoms, a careful past medical history, and review of systems are also necessary. Pain stimulated by eating suggests chronic mesenteric ischemia. Pain associated with abnormal bowel habits or bloating may be due to irritable bowel syndrome or recurrent intestinal obstruction. Ulcer-like pain may represent nonulcer dyspepsia or recurrent pancreatitis. Pelvic pain at monthly intervals suggests endometriosis.\(^{41}\) Pain that is constant and unrelenting suggests other causes. In these patients, history of weight loss, fever, and medical and surgical histories are particularly important. Weight loss suggests malignancy or malabsorption. The latter may result from intestinal or pancreatic disorders (e.g., sprue and chronic pancreatitis). Fever is common in occult intra-abdominal abscesses, autoimmune disorders, and hematologic malignancies such as lymphoma.

**Physical Examination**

The physical examination should include both a careful abdominal examination and a systemic examination for extra-abdominal manifestations of the underlying disease. For example, jaundice may be associated with chronic hepatitis, choledocholithiasis, or hepatic or biliary cancer. Perianal lesions may suggest the presence of inflammatory bowel disease.

**Laboratory Data**

As in the patient with acute abdominal pain, laboratory testing should reflect the differential diagnosis generated by the history and physical examination. Anemia may reflect chronic blood loss from a gastrointestinal source. An elevated sedimentation rate may signify an inflammatory disease or autoimmune disease.

**Diagnostic Studies**

Diagnostic imaging, including abdominal radiography, ultrasonography, and computed tomography, is valuable in establishing a diagnosis. In addition, upper and lower endoscopy and laparoscopy should be considered. For many patients with chronic abdominal pain diagnosis requires extensive evaluation.
Mechanical
Intestinal obstruction (hernia, intussusception, adhesions, volvulus)
Gallstones
Ampullary stenosis
Inflammatory
Inflammatory bowel disease
Endometriosis/endometritis
Acute relapsing pancreatitis
Familial Mediterranean fever
Neurologic and metabolic
Porphyria
Abdominal epilepsy
Diabetic radiculopathy
Nerve root compression or entrapment
Uremia
Miscellaneous
Irritable bowel syndrome
Nonulcer dyspepsia
Chronic mesenteric ischemia
Mittelschmerz

Diagnosable Causes

Table 4–3 lists some commonly overlooked causes of chronic abdominal pain. For further discussion, refer to the appropriate chapter within the text.

Treatment

The goals of treatment of patients with chronic abdominal pain are identification and cure of the responsible underlying disease. Cure, however, is often not possible; in these patients, palliation of symptoms may be worthwhile. Palliative care may involve medications, surgery, or psychologic support. For example, a patient with metastatic colon cancer may benefit from excision of the primary lesion to alleviate pain caused by local invasion or obstruction. Similarly, patients with advanced malignancy and biliary obstruction may experience significant relief of symptoms through surgical, endoscopic, or percutaneous biliary decompression. Palliation may also be achieved by pharmacologic and mechanical means. Analgesics, antidepressants, antiepileptics, and anxiolytics are often useful tools in palliative care. In addition, chemical and surgical nerve ablation or transcutaneous electrical nerve stimulation may be useful in relieving pain.

The treatment strategy pursued must also address the physical and psychologic symptoms associated with chronic pain. This is often the most difficult aspect of treatment facing the clinician. A multidisciplinary approach, including psychiatrists, physiotherapists, psychologists, and social workers, may help the patient and the clinician cope with chronic abdominal pain and its associated physical and psychologic manifestations. Where these resources are not available, however, the presence of a caring physician with an unhurried attitude can provide significant reassurance and comfort to an afflicted patient.

REFERENCES