CHAPTER 14

ABDOMINAL PAIN

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ACUTE ABDOMINAL PAIN

Acute abdominal pain is a popular descriptor that has evolved from the seminal definition by Sir Zachary Cope of abdominal pain of recent onset that triggers an urgent need for prompt diagnosis and active treatment.[1] The nature of the pain is usually such that the parent seeks immediate medical evaluation by a general pediatrician or emergency room physician. An acute complaint of abdominal pain precipitates at least 5% of unscheduled pediatric visits.[2] Although most children with acute abdominal pain have self-limiting conditions, the pain may herald a serious medical or surgical emergency. The diverse etiologies include acute surgical emergencies, disorders that will or may require surgical intervention, and specific intra-abdominal, extraintestinal, or systemic medical disorders. Severe acute pain may also present against a background of chronic illness. Most often, the child appears well at presentation, and the abdominal pain is accompanied by multiple complaints and is usually associated with a self-limited disease. The major challenge is to make a timely diagnosis of the acute surgical abdomen. In patients who present with acute abdominal pain to a primary care practice or community emergency room, the frequency of diseases requiring surgical intervention may be as low as 1%.[2] A physician can easily become complacent when dealing with a child with acute pain. It is important to have a consistent approach that addresses key diagnostic categories and gives parents guidelines to recognize warning signs that require re-evaluation. The primary care physician or emergency physician will most often call on the pediatric or general surgeon for consultation when concerned about a particular presentation of acute abdominal pain. There are, however, occasions when, with indefinite symptoms, there may be a tendency to wait for evolution of an acute process, in which case, the pediatric gastroenterologist may become involved in diagnosis and management.

Pathophysiology of Abdominal Pain

Abdominal pain can be perceived by autonomic sensory pathways from the abdominal viscera; somatic sensory pathways from the parietal peritoneum, abdominal wall, or retroperitoneal skeletal muscles; or somatic sensory pathways from extra-abdominal sites that share central projections with sensory pathways from the abdominal wall (referred pain).[3,4] Visceral pain is a dull or aching sensation generally perceived in one of three regions: the periumbilical, epigastric, or suprapubic midline area. Unfortunately, pain and tenderness are not always felt immediately over the site of disease. For example, the initial pain of appendicitis is usually felt in the periumbilical or epigastric areas, whereas pain caused by obstruction of the transverse colon is usually felt in the suprapubic midline area. Somatic pain, in contrast, is usually well localized and intense (often sharp) in character. An intra-abdominal process will manifest somatic pain if an inflammatory process affecting a viscus touches a somatic organ (ie, the anterior parietal peritoneum or abdominal wall). The classic example of referred abdominal pain is the shared central projections of the parietal pleura of the lung and the abdominal wall, such that abdominal pain may be the initial presentation of pneumonia. All three types of pain may be modified by a child's level of tolerance. Psychogenic and environmental factors augment or inhibit the perception of pain to varying degrees in different individuals.

Pain arising from the small intestine, regardless of the etiology, is always felt first and chiefly in the periumbilical or midepigastric area of the abdomen. Because the appendicular nerves are derived from the same thoracic nerves that supply the small intestine, it is not surprising that the pain at the onset of appendicitis is usually felt in the epigastric or umbilical area of the abdomen. The pain of disorders affecting the cecum, ascending colon, and descending colon are characteristically first felt at the actual site of the lesion because of the corresponding short mesocecum or mesocolon. An evolving change in localization of abdominal pain is often significant. Localization of pain in the right iliac fossa some hours after acute epigastric or periumbilical pain is usually due to appendicitis, although, rarely, the same sequence is seen with perforated pyloric or duodenal ulcer or in cases of acute pancreatitis. Radiation of the pain is also frequently helpful in diagnosis. In biliary colic, the pain is frequently referred to the region just under the inferior angle of the right scapula, whereas renal colic may be felt in the testicle on the same side. Testicular pain may also occur with appendicitis. A pelvic abscess, which lies close to the bladder, or an inflamed appendix that irritates the right ureter frequently causes pain on micturition. In many cases of peritonitis, intraperitoneal abscess, or abdominal distention owing to intestinal obstruction, abdominal pain will be caused or increased on inspiration.

DIFFERENTIAL DIAGNOSIS

Figure 14-1 divides the differential diagnosis of acute abdominal pain into three categories: (1) conditions requiring acute surgical intervention, (2) conditions that may initially be managed conservatively but will or may eventually require surgical intervention, and (3) specific intra-abdominal, extra-abdominal, and systemic conditions that require medical management. Detailed descriptions of the clinical presentation, diagnosis, and management of the specific disorders associated with acute abdomen are discussed in other chapters. The age of the patient is particularly helpful because the incidence of certain conditions is limited within a particular range of years (Table 14-1). Figure 14-2 presents an algorithm for sequential evaluation of a patient with acute abdominal pain. By thinking and working up selected key diagnoses, rare conditions will not be missed.

IS THERE EVIDENCE OF A CATASTROPHIC EVENT REQUIRING EMERGENCY SURGERY?

Catastrophic events include acute generalized peritonitis, intestinal infarction, or infarction of the ovaries or testes. Such complications may follow blunt or penetrating abdominal injury, high-grade acute intraluminal intestinal
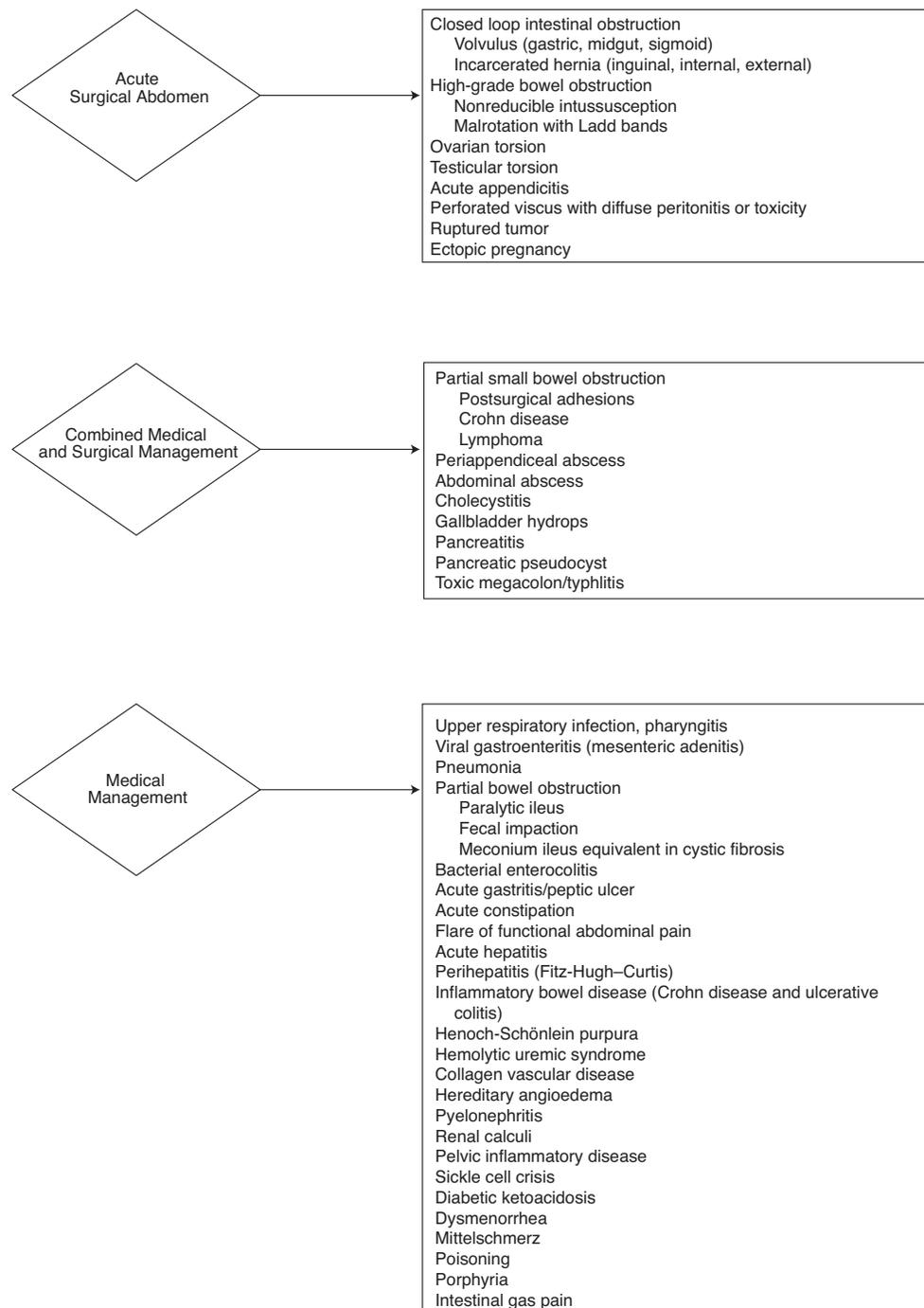

Acute Surgical Abdomen
Closed loop intestinal obstruction
    Volvulus (gastric, midgut, sigmoid)
    Incarcerated hernia (inguinal, internal, external)
High-grade bowel obstruction
    Nonreducible intussusception
    Malrotation with Ladd bands
Ovarian torsion
Testicular torsion
Acute appendicitis
Perforated viscus with diffuse peritonitis or toxicity
Ruptured tumor
Ectopic pregnancy

Combined Medical and Surgical Management
Partial small bowel obstruction
    Postsurgical adhesions
    Crohn disease
    Lymphoma
Periappendiceal abscess
Abdominal abscess
Cholecystitis
Gallbladder hydrops
Pancreatitis
Pancreatic pseudocyst
Toxic megacolon/typhlitis

Medical Management
Upper respiratory infection, pharyngitis
Viral gastroenteritis (mesenteric adenitis)
Pneumonia
Partial bowel obstruction
    Paralytic ileus
    Fecal impaction
    Meconium ileus equivalent in cystic fibrosis
Bacterial enterocolitis
Acute gastritis/peptic ulcer
Acute constipation
Flare of functional abdominal pain
Acute hepatitis
Perihepatitis (Fitz-Hugh–Curtis)
Inflammatory bowel disease (Crohn disease and ulcerative colitis)
Henoch-Schönlein purpura
Hemolytic uremic syndrome
Collagen vascular disease
Hereditary angioedema
Pyelonephritis
Renal calculi
Pelvic inflammatory disease
Sickle cell crisis
Diabetic ketoacidosis
Dysmenorrhea
Mittelschmerz
Poisoning
Porphyria
Intestinal gas pain

FIGURE 14-1   Differential diagnosis of acute abdominal pain.

TABLE 14-1   PRINCIPAL CAUSES OF ACUTE
ABDOMINAL PAIN BASED ON AGE

NEONATE
Necrotizing enterocolitis
Spontaneous gastric perforation
Hirschsprung disease
Meconium ileus
Intestinal atresia or stenosis
Peritonitis owing to gastroschisis or ruptured omphalocele
Traumatic perforation of viscus (difficult birth)

INFANT (< 2 YR)
Colic (< 3 mo)
Acute gastroenteritis or "viral syndrome"
Traumatic perforation of viscus (child abuse)
Intussusception
Incarcerated hernia
Volvulus (malrotation)
Sickling syndromes

SCHOOL AGE (2–13 YR)
Acute gastroenteritis or "viral syndrome"
Urinary tract infection
Appendicitis
Trauma
Constipation
Pneumonia
Sickling syndromes

ADOLESCENT
Acute gastroenteritis or "viral syndrome"
Urinary tract infection
Appendicitis
Trauma
Constipation
Pelvic inflammatory disease
Pneumonia
Mittelschmerz

obstruction (intussusception), closed-loop intestinal obstruction (volvulus or incarcerated hernia), torsion of the ovaries or testes, and perforation secondary to peptic ulcer, intestinal foreign body, gallbladder hydrops, or acute cholecystitis. An obvious "sick" general appearance of a child who presents with acute abdominal pain suggests a late stage of all varieties of acute abdominal disease. A pale, ashen, diaphoretic facial appearance leaves little doubt about a serious abdominal disorder. The signs and symptoms of a catastrophic event can vary according to the time that has elapsed since the acute event has occurred. High fever is unusual in the early stages. An initial stage of generalized, continuous abdominal pain accompanied by prostration, hypothermia, retching, and vomiting is followed by a period in which abdominal pain lessens, vomiting ceases, and temperature and pulse return to normal. Sending patients home or admitting them to a general hospital ward during this "honeymoon phase" can have disastrous consequences because this stage is soon followed by a shock-like picture accompanied by high fever, abdominal distention, gastrointestinal bleeding, and generalized peritoneal signs. Well-known features of peritonitis include abdominal wall rigidity, involuntary guarding, cutaneous hyperesthesia, rebound tenderness, absent bowel sounds, positive psoas or obturator signs, and tenderness on palpation of the anterior or right lateral rectal wall during rectal

examination. Rebound tenderness may or may not be a sign of a surgical abdomen. Rebound has also been reported in association with severe gastroenteritis, pneumonia, lead poisoning, and Henoch-Schönlein purpura.[1]

There is no reliable clinical, laboratory, or radiologic test that can distinguish between simple and strangulation obstruction of the small intestine.[5] Abdominal upright or decubitus plain films may show the presence of free air in the peritoneal cavity. A gasless abdomen is not uncommon in closed-loop or strangulating obstruction in which the obstructed loops are fluid filled.[6] Computed tomographic (CT) signs of strangulation include bowel wall thickening with or without target sign, pneumatosis, portal venous gas, mesenteric haziness, fluid, or hemorrhage often associated with ascites and abnormal bowel wall enhancement patterns following intravenous contrast.[7] CT may be oversensitive in diagnosing strangulation because bowel wall thickening, mesenteric haziness, fluid, and ascites are nonspecific findings that may also accompany other inflammatory processes, including appendicitis or Crohn disease.[7]

DOES A DIAGNOSIS OF ACUTE VIRAL SYNDROME (GASTROENTERITIS, UPPER RESPIRATORY INFECTION, PHARYNGITIS) MAKE SENSE?

An acute viral syndrome is the most common cause of acute abdominal pain in any age group beyond the neonatal period.[2] Think viral syndrome when multiple symptoms, including fever, vomiting, diarrhea, decreased appetite, headache, cough, sore throat, and rhinorrhea, occur simultaneously with onset of crampy, diffuse abdominal pain. The abdomen is soft and nondistended in most cases. Although the child may perceive palpation as uncomfortable, this maneuver does not elicit localized or rebound tenderness. Bowel sounds are normal or hyperactive. Gastroenteritis may present with predominant upper gastrointestinal tract symptoms, including epigastric pain, nausea, and variable vomiting. The pain most often occurs during eating or in the immediate postprandial period. Alternatively, gastroenteritis may present with predominant lower tract symptoms with generalized, periumbilical, or lower abdominal pain associated with diarrhea. The key to diagnosis is that the pain is self-limiting and does not progress or localize.

The differential diagnosis of acute viral syndrome includes bacterial enterocolitis, food poisoning, acute infection with *Helicobacter pylori*, acute pneumonia, pyelonephritis, diabetic ketoacidosis, Henoch-Schönlein purpura, hemolytic uremic syndrome, and angioedema. Bacterial enterocolitis should be suspected by the abrupt onset of fever and diffuse abdominal pain, followed shortly by diarrhea. Small-volume, frequent stools, blood and mucus in the stool, fever greater than 102.5°F, and polymorphonuclear leukocytes in the stool suggest a bacterial rather than a viral etiology. Although the severity of the abdominal pain may simulate appendicitis, palpation of the abdomen elicits diffuse tenderness and no evidence of peritoneal irritation. Common bacterial food poisoning may present with generalized abdominal pain associated with profuse watery diarrhea (*Clostridium perfringens*), or profuse vomiting and generalized abdominal pain, followed by
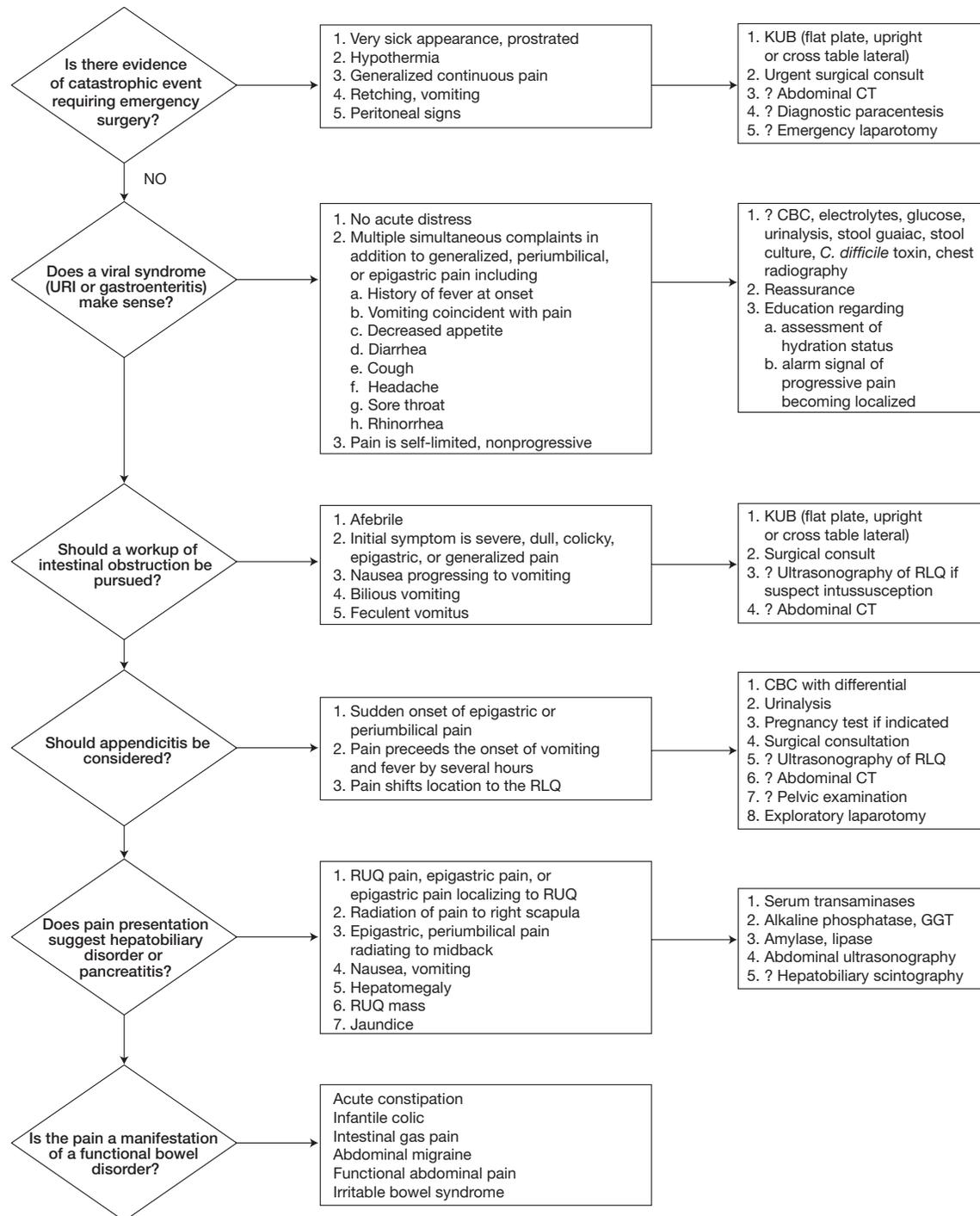
| Is there evidence of catastrophic event requiring emergency surgery? | 1. Very sick appearance, prostrated
2. Hypothermia
3. Generalized continuous pain
4. Retching, vomiting
5. Peritoneal signs | 1. KUB (flat plate, upright or cross table lateral)
2. Urgent surgical consult
3. ? Abdominal CT
4. ? Diagnostic paracentesis
5. ? Emergency laparotomy |

NO

| Does a viral syndrome (URI or gastroenteritis) make sense? | 1. No acute distress
2. Multiple simultaneous complaints in addition to generalized, periumbilical, or epigastric pain including
  a. History of fever at onset
  b. Vomiting coincident with pain
  c. Decreased appetite
  d. Diarrhea
  e. Cough
  f. Headache
  g. Sore throat
  h. Rhinorrhea
3. Pain is self-limited, nonprogressive | 1. ? CBC, electrolytes, glucose, urinalysis, stool guaiac, stool culture, *C. difficile* toxin, chest radiography
2. Reassurance
3. Education regarding
  a. assessment of hydration status
  b. alarm signal of progressive pain becoming localized |

| Should a workup of intestinal obstruction be pursued? | 1. Afebrile
2. Initial symptom is severe, dull, colicky, epigastric, or generalized pain
3. Nausea progressing to vomiting
4. Bilious vomiting
5. Feculent vomitus | 1. KUB (flat plate, upright or cross table lateral)
2. Surgical consult
3. ? Ultrasonography of RLQ if suspect intussusception
4. ? Abdominal CT |

| Should appendicitis be considered? | 1. Sudden onset of epigastric or periumbilical pain
2. Pain preceeds the onset of vomiting and fever by several hours
3. Pain shifts location to the RLQ | 1. CBC with differential
2. Urinalysis
3. Pregnancy test if indicated
4. Surgical consultation
5. ? Ultrasonography of RLQ
6. ? Abdominal CT
7. ? Pelvic examination
8. Exploratory laparotomy |

| Does pain presentation suggest hepatobiliary disorder or pancreatitis? | 1. RUQ pain, epigastric pain, or epigastric pain localizing to RUQ
2. Radiation of pain to right scapula
3. Epigastric, periumbilical pain radiating to midback
4. Nausea, vomiting
5. Hepatomegaly
6. RUQ mass
7. Jaundice | 1. Serum transaminases
2. Alkaline phosphatase, GGT
3. Amylase, lipase
4. Abdominal ultrasonography
5. ? Hepatobiliary scintography |

| Is the pain a manifestation of a functional bowel disorder? | Acute constipation
Infantile colic
Intestinal gas pain
Abdominal migraine
Functional abdominal pain
Irritable bowel syndrome | |

FIGURE 14-2 Approach to the patient with acute abdominal pain. CBC = complete blood count; *C. difficile* = *Clostridium difficile*; CT = computed tomography; GGT = γ-glutamyltransferase; KUB = flat plate of abdomen; RLQ = right lower quadrant; RUQ = right upper quadrant; URI = upper respiratory infection.

diarrhea (*Staphylococcus aureus*). Acute infection with *H. pylori* results in a neutrophilic gastritis with transient hypochlorhydria associated with epigastric abdominal pain and nausea. Pain is rarely severe enough to seek acute evaluation. As a general rule, serologic or stool antigen testing for evidence of *H. pylori* is not indicated in a child presenting with symptoms of viral gastroenteritis. Symptoms and signs of pneumonia are invariably present, including tachypnea out of proportion to fever, grunting respiration, cough, decreased breath sounds, and inspiratory rales. Fever, often accompanied by gastrointestinal symptoms suggestive of viral gastroenteritis, is frequent in the infant with urinary tract infection or pyelonephritis. In older children, fever accompanied by diffuse abdominal or flank pain may be the presenting symptom of pyelonephritis. Frequency, urgency, and dysuria, symptoms of cystitis, may be absent. Abdominal pain accompanied by vomiting may herald the onset of ketoacidosis in diabetes mellitus. There is usually an antecedent history of polydypsia, polyuria, and weight loss. There may be exquisite abdominal tenderness with guarding

and rigidity that may mimic peritonitis. The smell of ketones on the breath and the presence of deep sighing (Kussmaul breathing) reflect the ketoacidosis. In Henoch-Schönlein purpura, diffuse abdominal pain and vomiting with or without hematochezia may precede skin involvement by 1 week or occur 1 week after skin involvement. Diagnosis is suspected by evidence of other organ involvement, including joint pain, hematuria, and proteinuria. Intussusception occurs in 4 to 5% of children with abdominal pain. In hemolytic uremic syndrome, diffuse abdominal pain, vomiting, and hematochezia precede the onset of thrombocytopenia, coagulopathy, and oliguric renal failure by up to several weeks. At times, peritoneal signs may be prominent. Hereditary angioedema occurs in persons born without the ability to synthesize a normally functioning C1 inhibitor. Patients usually present with episodic, localized, nonpitting subcutaneous edema without urticaria, pruritus, or redness. Swelling of the intestinal wall without concurrent subcutaneous edema can lead to intense abdominal pain, sometimes with vomiting or diarrhea.

Acute abdominal pain secondary to acute infection is usually a clinical diagnosis that requires no confirmatory testing. Decision to do tests such as the complete blood count (CBC) with differential, electrolytes, blood urea nitrogen, creatinine, blood glucose, stool guaiac, stool culture, stool for *Clostridium difficile* toxin, urinalysis, or chest radiography should be based on clinical suspicion. The key to management is reassurance and education of the parents about the signs and symptoms of dehydration and the need for re-evaluation if pain progresses or localizes in the following 24 to 36 hours.

Should a Diagnosis of
Intestinal Obstruction Be Entertained?

The differential diagnosis of intestinal obstruction includes closed-loop obstruction (volvulus, incarcerated hernia), high-grade or complete intraluminal obstruction (intussusception), partial obstruction (incomplete intussusception, postoperative adhesions, Crohn disease, fecal impaction), and paralytic ileus. Early diagnosis of closed-loop and high-grade intraluminal obstruction is essential to avoid intestinal ischemia. Intestinal obstruction is suggested by a history of episodic, crampy visceral pain and vomiting and is supported by physical signs of abdominal distention, diffuse pressure tenderness, visible peristalsis, and absent or high-pitched bowel sounds. In acute intestinal obstruction, the temperature, as a rule, is normal. Visceral pain is usually present from the onset and frequently comes in bouts and spasms. Frequent bilious emesis, beginning soon after the onset of epigastric pain, suggests high intestinal obstruction (malrotation with Ladd bands). If the obstruction is in the distal small bowel or colon, nausea is constant from the onset, but vomiting is usually a late symptom. In most cases, the character of the vomiting changes with time. First, the stomach contents are expelled, and then yellow-green bilious material appears. The color of the emesis gradually changes to greenish-brown and becomes "feculent" (foul smelling). Feculent vomiting is diagnostic of distal intestinal obstruction.

Causes of intestinal obstruction requiring surgery include intussusception, postoperative adhesions, and incarcerated hernia (inguinal, internal, or external). Intussusception occurs most often in infants aged 6 to 18 months and is usually ileocolic and idiopathic. Intussusception is heralded by episodic crampy abdominal pain often following signs of viral gastroenteritis or upper respiratory illness. Prior abdominal surgery or peritonitis places a child at risk for intestinal obstruction from adhesions. Adhesions can occur relatively early in the postoperative course or months or even years later. Small incarcerated indirect inguinal hernia can easily escape detection if the whole abdomen is not observed and palpated, especially in obese patients. A firm, discrete mass can be palpated at the internal inguinal ring and may or may not extend into the scrotum. The testes may appear dark because of pressure on the spermatic cord causing congestion.

Small bowel obstruction can be diagnosed on an abdominal plain film with the demonstration of dilated loops of small intestine with air-fluid levels and no or little colonic gas, whereas colonic obstruction appears as colonic distention. Typically, in intussusception, no stool or air-fluid levels are seen in the cecum. In suspected obstruction, serial abdominal radiographs reveal progressive bowel distention and disappearance of gas from the distal bowel. Unfortunately, the plain film is diagnostic in only 46 to 80% of cases of small bowel obstruction.[5] The lower percentage probably reflects the radiographic findings at the patient's initial presentation, whereas the higher percentage includes patients who received follow-up studies. Ultrasonography can readily detect distended fluid-filled bowel loops, which certainly suggests the possibility of obstruction, but defining the location, type, and cause of the obstruction is extremely operator dependent. Also, where gaseous distention predominates or if the child resists abdominal compression because of pain, ultrasonography may be technically limited. If the question is to rule out ileocolic intussusception, the sensitivity of abdominal ultrasonography has been reported to be close to 100%, even in relatively inexperienced hands.[8,9] The position of the leading edge can be determined, the presence or absence of a lead point can be ascertained, and the presence or absence of blood flow within the intussusception can be identified with Doppler examination. The presence of flow on Doppler interrogation has been shown to predict radiographic reducibility by barium or air and diminish the danger of perforation during reduction. Abdominal CT has significantly advanced the evaluation of small and large bowel obstruction, especially in the acute situation in which high-grade or possibly strangulating obstruction is being encountered.[7] The abdominal CT diagnosis of small bowel obstruction requires a dilated proximal small bowel and collapsed distal bowel. Although CT may miss low-grade partial small bowel obstruction (eg, secondary to Crohn disease), incomplete obstruction rarely results in strangulation and, therefore, can be managed conservatively, at least initially. The diagnosis of closed-loop obstruction (volvulus or incarcerated hernia) by CT may be difficult to ascertain.

Causes of intestinal obstruction requiring surgery must be distinguished from paralytic ileus, which generally presents with increasing abdominal distention, minimal abdominal pain and tenderness, nausea, and increased frequency of flatus and loose stools. Vomiting is uncommon. Bowel sounds are characteristically diminished or absent. Paralytic ileus may be seen in a number of clinical settings, including hypokalemia, uremia, lead poisoning, drug therapy that interferes with gastrointestinal motor function, postsurgical period, posttraumatic shock, and viral gastroenteritis. Radiographs in a child with paralytic ileus demonstrate multiple, small air-fluid levels throughout the abdomen, but serial films show either no worsening or gradual improvement of the bowel gas pattern. The abdominal CT finding that suggests paralytic ileus is small bowel dilatation associated with a colon that is distended by gas and fluid.[7] Although fecal impaction is a frequent complication of chronic fecal retention, complete obstruction is rare. Partial obstruction from fecal retention responds to a combination of serial enemas and a large volume of polyethylene glycol (PEG) electrolyte solution given by nasogastric tube with or without manual evacuation of the distal rectum under general anesthesia. Distal ileal obstruction syndrome is a complication of cystic fibrosis that may result in partial small bowel obstruction from inspissation of intestinal contents in the distal ileum. The obstruction usually responds to a large volume of PEG electrolyte solution given by nasogastric tube or Gastrografin enemas.

Should Appendicitis Be Considered?

The first symptom of appendicitis is characteristically epigastric or periumbilical pain. The awakening out of sleep by acute abdominal pain in a previously well child is a common presentation of acute appendicitis. The temperature at the onset of acute appendicitis is usually normal but may rise to 100 or 100.5°F within a few hours. Similarly, vomiting usually begins a few hours after the onset of abdominal pain. Frequent vomiting may occur at the onset of acute appendicitis if the distal tip of the appendix distends acutely behind a proximal appendiceal concretion. Diarrhea is not a common symptom associated with uncomplicated appendicitis. Characteristically, over time, the pain shifts to the right lower quadrant. The most reliable sign of acute appendicitis is localized tenderness in the right lower quadrant. In fact, the localization of pain and tenderness on physical examination depends on the anatomic position of the appendix. In the case of the retrocecal appendix, pain may be localized to the lateral abdomen or flank. Alternatively, pain associated with retrocecal appendicitis may never localize. An appendix pointing to the left lower quadrant may present with suprapubic tenderness.

An elevated total white blood cell count (WBC) in the range of 11,000 to 17,000/mm³ is seen in approximately 80% of patients, but the specificity of leukocytosis for acute appendicitis is poor.[10] It is important to note that a normal WBC and differential should not delay surgical exploration in a child with localized right lower quadrant tenderness. A WBC that is higher than 20,000 mm³ suggests an acute bacterial infection or intra-abdominal abscess.[11] The plain abdominal radiograph is most often normal in children with appendicitis. Conversely, patients with a right lower quadrant process of any etiology, including appendicitis or gastroenteritis, may present with air-fluid levels in the right lower quadrant, indicative of a localized ileus. Unless the conventional abdominal radiograph reveals a calcified appendicolith (seen in 10% of patients with appendicitis), it is too nonspecific to help in the diagnosis of appendicitis.[12]

Many surgeons now advocate imaging studies to improve diagnostic accuracy and decrease the need for hospital admission to observe patients with suspicion but a lower probability of appendicitis. High-resolution graded compression ultrasonography is an excellent test for detection of acute nonperforated appendicitis.[13] Appendicitis is suspected by visualization of a rigid, noncompressible, aperistaltic, tubular structure in the appropriate location. In children, the sensitivity and specificity of sonography as applied to the diagnosis of appendicitis are very high, reported at 94% and 89%, respectively, with an overall accuracy of 91%.[14] Ultrasound visualization of the appendix must be interpreted in light of the clinical findings. False-negative results may occur for a number of reasons, including a lack of patient cooperation, inadequate compression to displace bowel gas, and operator inexperience. Abdominal CT can also be performed quickly, does not require graded compression, requires less initial experience in interpretation, and is highly accurate in both the diagnosis and exclusion of appendicitis.[15] On CT, an inflamed appendix is fluid filled, often contains a fecolith, and shows "stranding" or inflammatory changes in the periappendiceal fat.[12]

Many cases of appendicitis progress to perforation without the occurrence of vomiting. A large percentage of very young children will have perforated by the time of presentation. Immediately following perforation, abdominal pain may improve, and the temperature may become normal or even subnormal. Within 1 to 2 hours, however, there are usually signs of generalized rather than localized peritonitis accompanied by frequent vomiting, pallor, tachycardia, and fever of 101°F or greater. Secretory diarrhea may be a predominant symptom following a perforated appendix if the inflammatory mass lays against the sigmoid colon. Following appendiceal perforation, the child characteristically prefers to lie still. Any movement usually evokes pain and irritability. Bowel sounds are absent. Following perforation, the diagnostic accuracy of ultrasonography decreases because of guarding and focal ileus. Perforation may be suspected by visualization of asymmetric mural thickening, areas of increased intramural echogenicity, and fluid in the right paracolic gutter with adjacent atonic bowel loops. Abdominal CT is more sensitive, more specific, and less operator dependent for assessing a perforated appendix. CT signs of perforation include periappendiceal phlegmon or abscess.[15]

The differential of right lower quadrant abdominal pain includes Crohn disease, small bowel obstruction, pyelonephritis, renal colic, acute salpingitis (pelvic inflammatory disease), ovarian torsion, dysmenorrhea, ruptured

ovarian cyst, mittelschmerz, typhlitis, ectopic pregnancy, and mesenteric adenitis. Acute onset of Crohn disease should be suspected if there is right lower quadrant mass and diarrhea. Children with urolithiasis rarely present with the excruciating pain of stone passage seen in adults. Colicky pain in the abdomen or flank is more common. Hematuria, either microscopic or macroscopic, occurs in the vast majority of children. The presence of fever greater than 101°F suggests pyelonephritis and salpingitis in addition to a perforated appendix. Urinalysis should be performed in all patients with right lower quadrant abdominal pain, flank pain, or pain radiating into the groin. Pelvic examination with appropriate examinations for sexually transmitted diseases is indicated in an adolescent female who has just completed a menstrual period and presents with lower abdominal pain and fever. The patient may report an increased vaginal discharge or irregular bleeding. A complication of salpingitis that evokes clinical signs of peritonitis and shock is a ruptured tubo-ovarian abscess. Typical primary dysmenorrhea consists of crampy, dull, midline, or generalized lower abdominal pain at the onset of the menstrual period. The pain may coincide with the start of bleeding or precede the bleeding by several hours. Associated symptoms include backache, thigh pain, diarrhea, nausea, vomiting, and headache. Endometriosis must be considered when there is chronic, cyclic, undiagnosed pelvic pain in teenagers. Unilateral abdominal pain at the midpoint of the menstrual cycle (time of ovulation), with or without spotty bleeding for 24 hours, is characteristic of mittelschmerz. Typhlitis should be considered in a neutropenic patient receiving antineoplastic drugs who presents with right lower quadrant abdominal pain, fever, diarrhea, nausea, and vomiting. Localized tenderness may progress rapidly to diffuse signs of peritonitis as a result of intestinal perforation. Urine or serum pregnancy testing should be performed in adolescent females of reproductive age with lower abdominal pain. Mesenteric adenitis is a commonly used term to describe clustering of inflamed lymph nodes in the region of the terminal ileum in patients undergoing appendectomy. Mesenteric adenitis should not be considered a separate diagnosis but rather a sequela of viral or bacterial gastroenteritis.

DOES THE PAIN PRESENTATION SUGGEST A HEPATOBILIARY DISORDER OR PANCREATITIS?

Epigastric, right upper quadrant, or initial epigastric pain localizing to the right upper quadrant should suggest hepatobiliary disease, pancreatitis, or, rarely, acute peptic ulcer disease. Screening tests include serum transaminases, alkaline phosphatase, γ-glutamyltransferase, amylase, and lipase, as well as possible abdominal ultrasonography and hepatobiliary scintography.

Hepatobiliary disorders that may present with acute abdominal pain include viral hepatitis, biliary colic, acute calculous cholecystitis, cholangitis, acalculous cholecystitis, gallbladder hydrops, and perihepatitis (Fitz-Hugh–Curtis syndrome). Acute hepatitis is suspected if epigastric or right upper quadrant abdominal pain is accompanied by flu-like symptoms, including low-grade fever, anorexia, nausea, vomiting, malaise, and fatigability associated with palpation of tender hepatomegaly. Clinical manifestations of gallstone disease include biliary colic, acute cholecystitis, and cholangitis. Biliary colic is triggered by a gallstone(s) obstructing the cystic duct. The pain of biliary colic frequently follows a meal and can be localized to the right upper quadrant or epigastric areas. Sustained pain rises to a plateau of intensity over 5 to 20 minutes and gradually resolves over a 1- to 6-hour period. The patient tends to be restless, and the position does not help the pain. Pain lasting longer than 6 hours suggests acute cholecystitis. Cholecystitis implies an chemical inflammatory process within the gallbladder triggered by prolonged obstruction of the cystic duct. Referred pain to the dorsal lumbar back near the right scapula, nausea with some vomiting, and low-grade fever (< 101°F) are common. As inflammation worsens, the pain becomes more generalized in the upper abdomen and is increased by deep inspiration (Murphy sign: production of pain by deep inspiration or cough while the physician's fingers are compressing the abdomen below the right costal margin in the midclavicular line) and jarring movements. A common bile duct stone should be considered if the patient is jaundiced. Cholangitis should be suspected in a patient who has right upper quadrant pain, shaking chills, and a spiking fever greater than 102.5°F. A rigid abdomen or rebound tenderness suggests local perforation or gangrene of the gallbladder. Acute acalculous cholecystitis is acute inflammation of the gallbladder in the absence of stones. It is rare in children but has been associated with systemic illness or enteric infections. Acalculous cholecystitis should be included in the differential of a patient with the simultaneous onset of high fever and pain symptoms suggesting biliary colic. Gallbladder hydrops, or acute noncalculous, noninflammatory distention of the gallbladder, has been associated with Kawasaki disease, Henoch-Schönlein purpura, and scarlet fever. In addition to right upper quadrant pain, the distended gallbladder may be palpated. Perihepatitis is a complication of pelvic inflammatory disease in adolescent females that presents with severe right upper quadrant pain and tenderness produced by inflammation of the liver capsule. Fever may or may not be present. The syndrome has been associated with both *Neisseria gonorrhoeae* and *Chlamydia trachomatis*.

In pancreatitis, onset of pain is usually insidious over several hours. Constant epigastric or upper quadrant pain with or without radiation to the back, which is aggravated when the patient lies down, is an indication to check pancreatic enzymes. The pain may be referred to the left scapula or be generalized in both upper abdominal quadrants. Vomiting may be severe and protracted. A low-grade fever (< 101°F) may be present. The abdomen may be distended but is rarely rigid. Rebound tenderness is rare. Bowel sounds may be decreased.

IS THE ACUTE ABDOMINAL PAIN A MANIFESTATION OF A FUNCTIONAL BOWEL DISORDER?

The three main considerations are acute constipation, aerophagia, and flare of functional abdominal pain. Acute

constipation may complicate a viral illness that causes decreased bowel motility and results in dietary changes. Rectal pain produced by anal fissure may be a cause of constipation. Abdominal pain is usually left-sided or suprapubic, antedated by decreased frequency or volume of usual bowel movements for several days. Acute constipation may be accompanied by sensations of urgency, tenesmus, and rectal pain. Abdominal examination may reveal distention or hard feces in a palpable colon.

In emergency rooms, acute constipation is frequently given as a cause of acute abdominal pain after abdominal plain film performed to screen for obstruction is interpreted by the radiologist as showing a moderate or large amount of stool. In fact, abdominal plain film has a low specificity for diagnosing constipation. In the absence of a confirming history and digital rectal examination showing a rectum full of stool, a child with acute abdominal pain should not be treated for acute constipation based on an abdominal radiograph.

Intestinal gas is also overplayed as a cause of acute abdominal pain. In the absence of a history of excessive air swallowing and distention, parents should not be told that acute abdominal pain is the result of intestinal gas. As with constipation, the abdominal plain film has a low specificity for diagnosing excessive intraluminal gas.

A significant percentage of children who present to emergency rooms with acute abdominal pain have a flare of chronic functional abdominal pain, described below. A history of chronic pain, a normal abdominal examination, and the absence of alarm signals suggest a flare of functional abdominal pain. Emergency physicians should reassure the patient and parents regarding normal examination and resist initiating further workup that might confuse management initiated by the patient's primary caregiver or pediatric gastroenterologist.

CHRONIC ABDOMINAL PAIN

Chronic abdominal pain is one of the most commonly encountered symptoms in childhood and adolescence. The definition of "chronic" has evolved from the seminal definition by Apley of recurrent paroxysmal abdominal pain in children between the ages of 4 and 16 years that persists for greater than 3 months duration and affects normal activity.[16] Chronic abdominal pain has been reported to occur in 10 to 15% of children.[17–19] At least as many children experience chronic pain but maintain normal activity and rarely come to the attention of the physician.[17–19]

The Pediatric Rome group has proposed that chronic abdominal pain can be subcategorized based on clinical presentation: (1) isolated paroxysmal abdominal pain, (2) abdominal pain associated with symptoms of dyspepsia, (3) abdominal pain associated with altered bowel pattern, and (4) abdominal migraine.[20] Symptoms of dyspepsia include pain associated with eating, epigastric location of pain, nausea, episodic vomiting, early satiety, occasional heartburn and acid regurgitation, and excessive belching. Symptoms of altered bowel pattern include diarrhea, constipation, or a sense of incomplete evacuation with bowel movements. The differential diagnosis of each subcategory of chronic pain includes a heterogeneous group of anatomic, infectious, noninfectious inflammatory, and biochemical organic disorders. Yet, although the exact prevalence figures are unknown, the most common cause in each subcategory is functional abdominal pain. The modifier "functional" is used in gastroenterology if no specific structural, infectious, inflammatory, or biochemical cause for the abdominal pain can be determined. The vast majority of children classified by Apley as having "recurrent abdominal pain" had functional abdominal pain.

PATHOPHYSIOLOGY OF FUNCTIONAL ABDOMINAL PAIN

There is general agreement that functional pain is genuine and not simply social modeling or imitation of parental pain or a means to avoid an unwanted experience (eg, school phobia or malingering). The etiology and pathogenesis of the pain are unknown. Many physicians conceptualize functional pain as "nonorganic." Yet there is a growing body of evidence that the pain is the result of disordered brain-gut communication involving both the efferent and afferent pathways by which the enteric and central nervous systems communicate. It is not clear whether the different subcategories of functional abdominal pain result from a heterogeneous group of disorders or represent variable expressions of the same disorder. The frequent occurrence of upper and lower bowel symptoms in the same patient (particularly nonulcer dyspepsia and irritable bowel in an adolescent) suggests that the latter scenario may indeed be the case. Although there is no evidence that the etiology of functional pain in children differs from functional pain in adults, the tendency of children to outgrow functional pain suggests that self-limiting developmental factors may be involved in the pathophysiology of pain in children.

The prevailing viewpoint is that the pathogenesis of the functional pain involves the interrelationship between altered gastrointestinal motility and visceral hypersensitivity.[21] Motility disturbances have been described in children, including altered intestinal transit, enhanced rectal contractility to cholinergic agonists, clusters of jejunal pressure activity that coincide with pain, lower rectal compliance, and altered rectal contractile response to a meal.[22–24] Visceral hypersensitivity in children with functional abdominal pain is supported by reports of enhanced awareness of balloon distention in the rectum and pain associated with physiologic stimuli such as the intestinal migrating motor complex.[25,26] A common link among these various motor and sensory phenomena is the autonomic nervous system. In some patients, associated symptoms including headache, dizziness, motion sickness, pallor, temperature intolerance, and nausea suggest a generalized dysfunction of the autonomic nervous system. In fact, abnormalities of sympathetic cardiac, vasomotor, and sudomotor function by autonomic testing have been described in patients with functional pain.[27] In adult studies, there is growing evidence that the initiating factors for autonomic dysfunction are found in the central nervous system, namely, the limbic system and the thalamus. Furthermore, there is increasing

evidence that serotonin (5-hydroxytryptamine [HT]) and its receptors (particularly the 5-HT$_3$ and 5-HT$_4$ receptors) play a major role in the pathogenesis of functional pain.[28]

In a child with visceral hypersensitivity, painful sensations may be provoked by physiologic phenomena or concurrent physical and psychological stressful life events. Examples of physiologic phenomena that may trigger pain include postprandial gastric or intestinal distention, gastric emptying, intestinal contractions or the migrating motor complex, intestinal gas, or gastroesophageal reflux. Intraluminal physical stress factors that may trigger pain include aerophagia, simple constipation, lactose intolerance, minor noxious irritants such as spicy foods, *H. pylori* gastritis, celiac disease, or drug therapy. Systemic physical or psychological stress factors may also provoke or reinforce the pain behavior by altering the conscious threshold of gastrointestinal sensory input in the central nervous system. Acute or chronic physical illness may unmask functional pain. Psychological stress factors may include death or separation of a significant family member, physical illness or chronic handicap in parents or a sibling, school problems, altered peer relationships, family financial problems, or a recent geographic move.

The concept of visceral hypersensitivity can be better understood by examining the role of lactose intolerance as a trigger of functional abdominal pain. There does not appear to be a difference in the incidence of lactose intolerance between patients with functional abdominal pain and age-matched patients without pain. Yet Barr and colleagues have reported a qualitative improvement in pain symptoms in 70% of intolerant children treated with a lactose-free diet.[29] They observed that such children lack an awareness of intolerance to lactose because there is no temporal relationship between lactose ingestion and abdominal discomfort. These results suggest that lactose intolerance is not directly the cause of the pain but the trigger that unmasks visceral hypersensitivity (perhaps by luminal distention) in susceptible patients. That lactose is but one provocative stimulus in such patients is supported by observations that a lactose-free diet does not induce complete resolution of the pain or alter the natural history of the condition.[29]

Recently, there has been progress in defining a subgroup of adults with symptoms of irritable bowel syndrome (IBS) developing after an episode of infective gastroenteritis.[30] The role of inflammation in the pathogenesis of functional abdominal pain must also be considered in view of the frequent finding of mild nonspecific histologic inflammatory changes at all levels of the gastrointestinal tract in patients with functional abdominal pain.[31,32] It may be speculated that such mild inflammatory changes that persist after gastroenteritis may be the cause of visceral hypersensitivity or altered intestinal motility. Immune responses alter neural and endocrine function, and, in turn, neural and endocrine activity modifies immunologic function.[33] Activated immunocompetent cells such as monocytes, lymphocytes, macrophages, serotonin-containing enterochromaffin cells, and mast cells that take up residence in the intestinal tract may secrete a repertoire of cytokines and inflammatory mediators that can lead to profound changes in enteric neural function. The main symptoms of postinfective irritable bowel (ie, diarrhea and loose stools) may reflect the prokinetic and secretory effect of 5-HT and inflammatory mediators derived from enterochromaffin cells and lymphocytes.[30] The possibility that some aspect of personality, behavior, coping style, or emotional state influences immune responses may also have implications in functional abdominal pain. The enteric or central nervous system may also modulate intestinal immune responses. Activation of the sympathetic nervous system causes leukocytosis, sequestration of lymphocytes, and inhibition of natural killer cell activity.[33] Sensory neurons also contain a variety of neurotransmitters and neuropeptides that can affect lymphocyte function, including substance P, vasoactive intestinal polypeptide, angiotensin II, calcitonin gene–related peptide, and somatostatin.

There also appears to be a genetic vulnerability because of the high frequency of pain complaints in family members.[16] Recent studies suggest that patients with functional abdominal pain who make their way to a subspecialty setting commonly exhibit "internalizing" behavior characterized by anxiety, mild depression, withdrawal, and low self-esteem.[34,35] Such a behavior profile may be primary and part of the genetic vulnerability of such patients. Alternatively, it has been postulated that such internalizing behavior is fostered within a family structure characterized by maternal depression, enmeshment, overprotectiveness, rigidity, and a lack of conflict resolution.[36] A third possibility is that the internalizing behavior is a common psychological adaptation to both organic and nonorganic chronic conditions.[35] Whether primary or secondary, the behavior pattern of the child and the family structure may both influence how the disorder is experienced and acted on.

The morbidity associated with recurrent abdominal pain is not physical but results from interference in normal school attendance and performance, peer relationships, participation in organizations and sports, and personal and family activities. Liebman found that only 1 of 10 children with functional abdominal pain attended school regularly and that absenteeism was greater than 1 day in 10 in 28% of patients.[37] A common misconception is that pain is the direct cause of the morbidity. In fact, the environmental consequences of the pain probably contribute significantly to the morbidity. Fordyce and colleagues observed that although pain does not originate from its consequences, much pain behavior is accounted for and modified by its consequences.[38] As described below, the usual parental, school, and medical management of recurrent abdominal pain is focused on symptom relief, which reinforces the pain behavior with attention, rest, and medication. This approach fails to reinforce nonpain responses such as normal activity.

DIAGNOSIS OF CHRONIC ABDOMINAL PAIN

Because the exact etiology and pathogenesis of the pain are unknown and no specific diagnostic markers exist for any group, functional abdominal pain is too often perceived as a diagnosis of exclusion. Yet it is the clinical presentation, together with a well-structured medical history and physical examination, that usually indicates that functional

abdominal pain is the likely diagnosis in an individual child presenting with chronic abdominal pain. Rather than a shotgun approach to rule out all potential infectious, inflammatory, structural, and biochemical causes of a particular pain presentation, diagnostic evaluation should be driven by an index of suspicion based on pertinent alarm signals in the history and physical examination. In clinical practice, functional abdominal pain should not be a diagnosis of exclusion. Primary care physicians should be able to make a primary diagnosis of functional abdominal pain without resorting to a large battery of biochemical or radiography tests. Management of functional pain is facilitated by early diagnosis, parental education and reassurance, and clear delineation of goals of therapy. The major outcome variable in the management of functional abdominal pain in children is lifestyle, not cure of the pain.

One reason why primary care physicians have difficulty making a positive diagnosis of a functional abdominal pain is that there is rarely a clear distinction between acute and chronic abdominal pain. A parent's decision to consult a physician is usually based on the age of the child, the severity of the pain, and the effects of pain on the child's lifestyle. Primary caregivers must often deal with the evolution of pain from the initial acute presentation to a chronic or recurring problem. A stepwise series of diagnostic studies is often initiated during early stages of the pain when an organic etiology is considered to be more likely. Empiric therapy with nonopioid analgesic medications, antispasmodic and anticholinergic agents, and gastric acid–reducing agents may be tried before time criteria for functional abdominal pain are met. Parents tend to become more frustrated and anxious, particularly if they perceive that a serious disorder is being missed or if the physician implies that the primary factors that influence the perception of pain are cognitive and emotional. Parental uncertainty only increases the stressful environment that provokes or reinforces the pain behavior.

Thus, the concept of functional abdominal pain must be introduced into the differential diagnosis of abdominal pain in children before the 3-month time criteria for duration of pain are met. Functional abdominal pain lacks a symptom-based diagnostic marker. None of the following have been shown to help the physician discriminate between organic, psychosomatic, and functional abdominal pain: frequency of pain; character of pain; location of pain; pain awakening patient at night; associated gastrointestinal symptoms, including anorexia, nausea, episodic vomiting, increased gas, or altered bowel pattern; or associated extraintestinal symptoms, including fatigue, pallor, headache, and arthralgia. Similarly, there is no evidence that anxiety, depression, behavior problems, or recent negative life events discriminate between organic, psychosomatic, and functional abdominal pain. Because there are no prospective studies on natural history or incidence, it cannot be stated that the duration of pain itself, beyond 3 months without an organic diagnosis, supports a diagnosis of functional pain.

Although there are no evidence-based data, clinical experience suggests that subclassifying pain presentations may facilitate the choice of testing by narrowing the differential diagnosis (Figure 14-3). Children with abdominal pain may be subclassified by one of four clinical presentations: (1) abdominal pain associated with symptoms of upper abdominal distress, (2) abdominal pain associated with altered bowel pattern, (3) isolated paroxysmal abdominal pain alone, and (4) cyclical pain syndrome. Cyclical pain refers to episodes of intense acute midline pain lasting several hours to a few days with intervening symptom-free intervals lasting weeks to months. Functional abdominal pain should be presented as the most common cause of all four clinical presentations. The frequent occurrence of upper and lower bowel symptoms in the same patient is not uncommon.

ESTABLISHING A WORKING DIAGNOSIS OF FUNCTIONAL ABDOMINAL PAIN

The key variables that point toward a functional diagnosis are a normal physical examination, other than abdominal pressure tenderness, and absence of alarm signals for an organic disorder. Even with a normal physical examination, further diagnostic testing is definitely indicated in the presence of the following alarm signals: involuntary weight loss, growth retardation, significant vomiting, significant diarrhea, gastrointestinal blood loss, associated fever, arthritis, rash, symptoms of a psychiatric disorder, or a family history of inflammatory bowel disease. Alarm signals in the physical examination include evidence of linear growth deceleration, localized tenderness in the right upper or lower quadrants, localized fullness or mass effect, hepatomegaly, splenomegaly, spine or costovertebral angle tenderness, perianal fissure, perianal fistula, visible soiling, and guaiac-positive stools. Although a family history of patients with functional pain who consult physicians is more likely to be positive for parental health complaints, including marital discord, psychiatric illness, and past surgery, this cannot be used to discriminate between functional and organic pain.

Diagnostic testing is indicated when alarm signals or abnormal physical findings suggest a possibility of an organic disorder. No studies have evaluated the value of common laboratory tests (CBC, erythrocyte sedimentation rate [ESR], comprehensive metabolic panel, urinalysis, stool parasite analysis) to distinguish between organic and functional pain. Diagnostic testing may also be considered to reassure the parent, patient, or physician that the most likely diagnosis is functional pain. The physician may also need to do testing to rule out organic disease in the patient in whom pain continues to severely affect lifestyle despite a functional diagnosis. Clinical experience suggests that subclassifying pain presentations may facilitate the choice of testing by narrowing the differential diagnosis (see Figure 14-3).

Establishing a working diagnosis of functional pain and initiating conservative therapy before time criteria are achieved does not preclude an ongoing, focused, diagnostic workup. Synonyms of functional pain that may be useful for individualizing diagnosis in a given patient are functional dyspepsia for pain with upper abdominal symptoms, IBS for pain associated with altered bowel pattern, func-
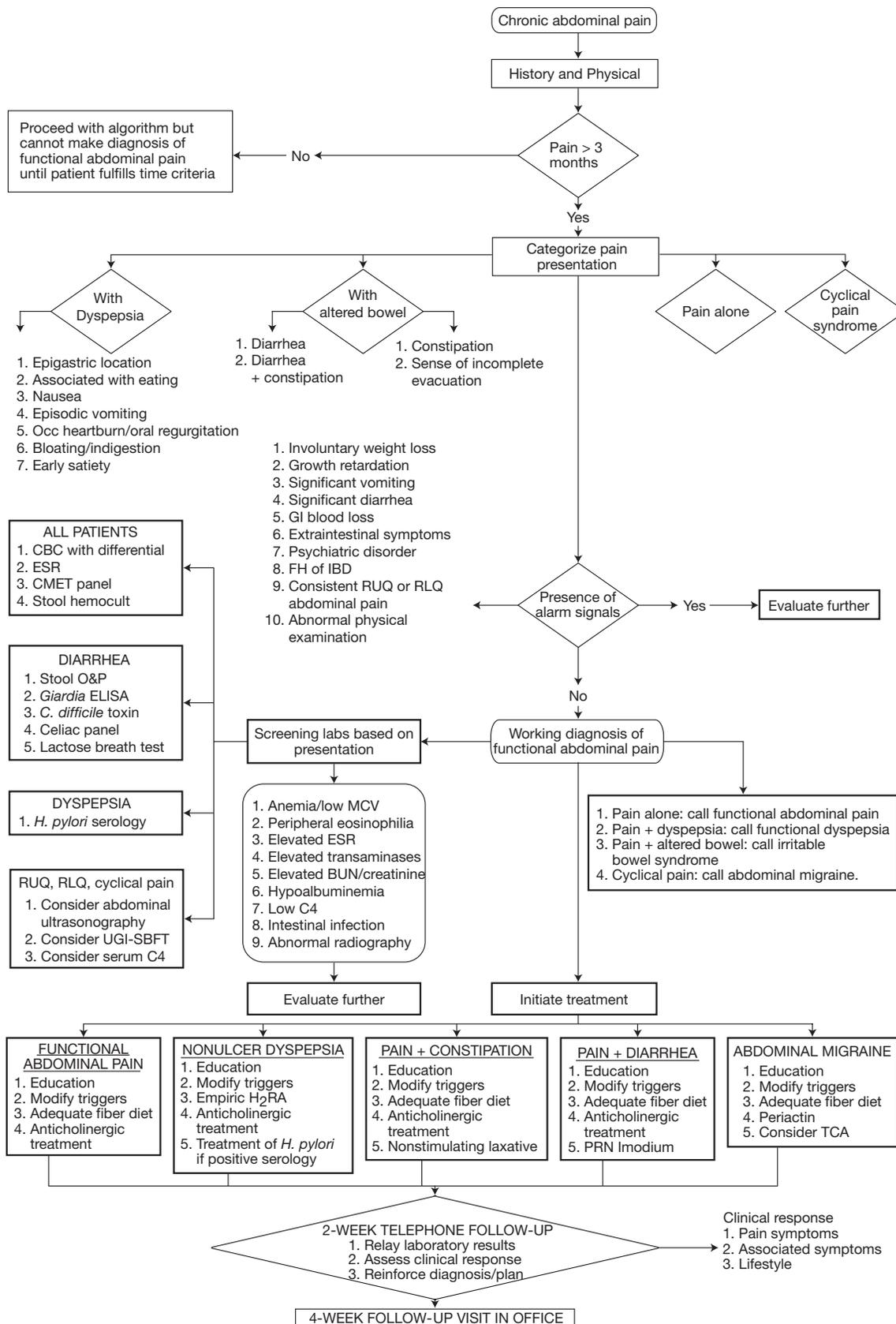
Chronic abdominal pain

History and Physical

Pain > 3 months

No → Proceed with algorithm but cannot make diagnosis of functional abdominal pain until patient fulfills time criteria

Yes

Categorize pain presentation

With Dyspepsia
1. Epigastric location
2. Associated with eating
3. Nausea
4. Episodic vomiting
5. Occ heartburn/oral regurgitation
6. Bloating/indigestion
7. Early satiety

With altered bowel
1. Diarrhea
2. Diarrhea + constipation
1. Constipation
2. Sense of incomplete evacuation

Pain alone

Cyclical pain syndrome

1. Involuntary weight loss
2. Growth retardation
3. Significant vomiting
4. Significant diarrhea
5. GI blood loss
6. Extraintestinal symptoms
7. Psychiatric disorder
8. FH of IBD
9. Consistent RUQ or RLQ abdominal pain
10. Abnormal physical examination

Presence of alarm signals → Yes → Evaluate further

No

ALL PATIENTS
1. CBC with differential
2. ESR
3. CMET panel
4. Stool hemocult

DIARRHEA
1. Stool O&P
2. Giardia ELISA
3. C. difficile toxin
4. Celiac panel
5. Lactose breath test

DYSPEPSIA
1. H. pylori serology

RUQ, RLQ, cyclical pain
1. Consider abdominal ultrasonography
2. Consider UGI-SBFT
3. Consider serum C4

Screening labs based on presentation

Working diagnosis of functional abdominal pain

1. Pain alone: call functional abdominal pain
2. Pain + dyspepsia: call functional dyspepsia
3. Pain + altered bowel: call irritable bowel syndrome
4. Cyclical pain: call abdominal migraine.

1. Anemia/low MCV
2. Peripheral eosinophilia
3. Elevated ESR
4. Elevated transaminases
5. Elevated BUN/creatinine
6. Hypoalbuminemia
7. Low C4
8. Intestinal infection
9. Abnormal radiography

Evaluate further

Initiate treatment

FUNCTIONAL ABDOMINAL PAIN
1. Education
2. Modify triggers
3. Adequate fiber diet
4. Anticholinergic treatment

NONULCER DYSPEPSIA
1. Education
2. Modify triggers
3. Empiric H2RA
4. Anticholinergic treatment
5. Treatment of H. pylori if positive serology

PAIN + CONSTIPATION
1. Education
2. Modify triggers
3. Adequate fiber diet
4. Anticholinergic treatment
5. Nonstimulating laxative

PAIN + DIARRHEA
1. Education
2. Modify triggers
3. Adequate fiber diet
4. Anticholinergic treatment
5. PRN Imodium

ABDOMINAL MIGRAINE
1. Education
2. Modify triggers
3. Adequate fiber diet
4. Periactin
5. Consider TCA

2-WEEK TELEPHONE FOLLOW-UP
1. Relay laboratory results
2. Assess clinical response
3. Reinforce diagnosis/plan

Clinical response
1. Pain symptoms
2. Associated symptoms
3. Lifestyle

4-WEEK FOLLOW-UP VISIT IN OFFICE

FIGURE 14-3 The author's algorithm for the evaluation and management of chronic abdominal pain. BUN = blood urea nitrogen; CBC = complete blood count; *C. difficile* = *Clostridium difficile*; CMET = comprehensive metabolic panel (Chem 12); ELISA = enzyme-linked immunosorbent assay; ESR = erythrocyte sedimentation rate; FH = family history; GI = gastrointestinal; *H. pylori* = *Helicobacter pylori*; H2RA= histamine2 receptor antagonist; IBD = inflammatory bowel disease; MCV = mean corpuscular volume; O&P = ovum parasite examination; PRN = as needed; RLQ = right lower quadrant; RUQ = right upper quadrant; TCA = tricyclic antidepressant; UGI-SBFT = upper gastrointestinal small bowel follow-through .

tional abdominal pain for patients with isolated paroxysmal abdominal pain alone, and abdominal migraine for cyclical acute pain episodes. Two of the following features are required for diagnosis of abdominal migraine: (1) a headache during episodes, (2) photophobia during episodes, (3) associated classic unilateral migraine headaches that may or may not be associated with abdominal pain, (4) a family history of migraine, and (5) visual, sensory, or motor aura antedating acute pain.[39,40]

DIAGNOSTIC EVALUATION BASED ON SUBCATEGORIES OF CHRONIC ABDOMINAL PAIN

Chronic Abdominal Pain Associated with Symptoms of Dyspepsia. Table 14-2 lists the differential diagnosis in patients with chronic abdominal pain and upper gastrointestinal symptoms. The key to deciding on the extent of initial workup is the presence or absence of significant vomiting. A reasonable focused laboratory evaluation in all patients includes a CBC with differential, ESR, *H. pylori* serology and/or stool antigen, hepatic panel, and pancreatic enzyme measurement. In cases in which recurrent vomiting is a significant part of the history, an upper gastrointestinal series with small bowel follow-through and abdominal ultrasonography should be considered to rule out gastric outlet disorder, malrotation, partial small bowel obstruction, small bowel Crohn disease, gallstones, pancreatic pseudocyst, hydronephrosis secondary to ureteropelvic junction obstruction, and retroperitoneal mass.

Gastroesophageal reflux disease should be suspected when heartburn and acid regurgitation are prominent parts

TABLE 14-2   DIFFERENTIAL DIAGNOSIS OF RECURRENT ABDOMINAL PAIN ASSOCIATED WITH SYMPTOMS OF DYSPEPSIA

ASSOCIATED WITH UPPER GASTROINTESTINAL INFLAMMATION
Gastroesophageal reflux disease
Peptic ulcer
Helicobacter pylori gastritis
Nonsteroidal anti-inflammatory drug ulcer
Crohn disease
Eosinophilic gastroenteritis
Ménétrier disease
Cytomegalovirus gastritis
Parasitic infection (*Giardia, Blastocystis hominis*)
Varioliform gastritis
Lymphocytic gastritis/celiac disease
Henoch-Schönlein purpura

MOTILITY DISORDERS
Idiopathic gastroparesis
Biliary dyskinesia
Intestinal pseudo-obstruction

OTHER DISORDERS
Obstructive disorders from Table 14-1
Chronic pancreatitis
Chronic hepatitis
Chronic cholecystitis
Ureteropelvic junction obstruction
Abdominal migraine
Psychiatric disorders

of the history. Gastroparesis following a viral infection may begin within 7 days following resolution of acute viral illness (especially post rotavirus) and lead to chronic epigastric pain associated with persistent nausea and episodic vomiting.[41,42] Diagnosis is confirmed by demonstrating delayed gastric emptying by scintigraphy. Recurrent epigastric or right upper quadrant pain associated with tender hepatomegaly suggests chronic hepatitis. Biliary colic is episodic, severe, constant pain in the right upper quadrant or epigastrium that persists for 20 minutes to 2 hours and that is usually triggered by eating. Choledocholithiasis is confirmed by abdominal ultrasonography. Gallbladder dyskinesia remains a controversial primary diagnosis to explain chronic dyspepsia. Diagnosis should be suspected in patients with protracted symptoms suggesting biliary colic, a positive family history of gallstones, normal abdominal ultrasonography, and hepatobiliary scintigraphy with delayed ejection fraction after cholecystokinin infusion.[43] Dramatic improvement has been reported in children after elective cholecystectomy.[44,45] Experience in adults has been less dramatic, with only 47% of patients becoming completely asymptomatic.[46] Adult norms for ejection fraction have been used to assess pediatric patients. In relapsing pancreatitis, recurrent severe epigastric pain persists for days and may radiate to the back. Endoscopic retrograde cholangiopancreatography is indicated only if there is biochemical or radiologic evidence of recurrent pancreatitis or biliary-type abdominal pain following cholecystectomy. Continuous pain, especially in the context of multisystem complaints, is an alarm signal for possible psychiatric disease. Eating disorder should also be considered in any young patient with significant weight loss.

H. pylori gastritis and nonsteroidal anti-inflammatory drugs (NSAIDs) are the most important exogenous factors associated with peptic ulcer in adults. However, in children, clinically significant ulceration occurs infrequently with NSAID use or *H. pylori* gastritis.[31,47] The pathogenic mechanisms distinguishing those individuals at risk have not been identified. A careful history is required to ensure that NSAID consumption is detected in any patient being evaluated for recurrent abdominal pain with dyspepsia. The incidence of *H. pylori* infection in children increases with age, is inversely related to socioeconomic class, and increases in families in which an adult has had either an ulcer or documented *H. pylori* infection.[48] In the absence of peptic ulcer disease, the relationship between *H. pylori* infection and abdominal pain remains unclear. Although there are no evidence-based data to establish a clear link between *H. pylori* gastritis without ulcer and abdominal pain associated with symptoms of upper abdominal distress,[47] most gastroenterologists will treat a symptomatic child who has been identified as *H. pylori* positive. The rationale is that *H. pylori* may act as a physical trigger of functional dyspepsia in selected patients. Some authors have concluded that the most cost-effective approach is to test serologically for *H. pylori* and to treat all infected cases. However, many investigators have pointed out that commercially available serologic assays do not appear to have the necessary sensitivity or specificity to screen pediatric

patient populations.[47] Empiric treatment of *H. pylori* should be considered only in patients with an elevated immunoglobulin (Ig)G antibody and is not recommended for patients with a positive IgM or IgA antibody. It is not unreasonable to avoid antibody testing altogether and consider treatment only in patients with endoscopically proven infection who have not responded to treatment of functional dyspepsia.

Upper endoscopy should be considered in untreated patients with alarm signals, patients who fail to respond to time-limited gastric acid reduction therapy for functional dyspepsia, and patients in whom symptoms recur after attempting to step off seemingly effective therapy. Upper endoscopy is the gold standard to rule out infectious and inflammatory disorders in the upper gastrointestinal tract. Recognizable objective findings by gross endoscopic examination include superficial erosions, ulcer, stricture, antral nodularity associated with *H. pylori* gastritis, gastric rugal hypertrophy associated with Ménétrier disease and cytomegalovirus gastritis, and the small, heaped up, volcanic-like mounds, pocked with a central crater, associated with chronic varioliform gastritis. Objective histologic findings may help to diagnose reflux esophagitis, eosinophilic gastroenteritis, cytomegalovirus gastritis, *H. pylori* gastritis, Crohn disease, and celiac disease. In the absence of gross ulcer or histologic evidence of *H. pylori*, superficial antral gastritis or duodenitis is of questionable clinical significance and should not dissuade a diagnosis of functional dyspepsia. There is no evidence in children that nonspecific superficial antral gastritis or duodenitis progresses to peptic ulcer. A diagnosis of postviral gastroparesis or gallbladder dyskinesia should not be entertained without first ruling out upper gastrointestinal tract inflammation and infection by upper endoscopy.

Chronic Abdominal Pain Associated with Symptoms of Altered Bowel Pattern. Altered bowel pattern may include a change in the frequency and/or consistency of stools (diarrhea or constipation), pain relieved with defecation, straining or urgency, a feeling of incomplete evacuation, passage of mucus, or a feeling of bloating or abdominal distention. Table 14-3 lists the major differential of chronic abdominal pain associated with an altered bowel pattern. The key to deciding on the extent of initial workup is the volume of diarrhea, evidence of gross or occult blood in the stool, and the presence of encopresis. In patients with diarrhea, a focused laboratory evaluation should include a CBC with differential, ESR, stool for *Giardia* antigen, stool for ovum parasites, and stool for *C. difficile* toxin. Alarm signals, including evidence of gastrointestinal bleeding, tenesmus, pain or diarrhea repeatedly wakening the patient from a sound sleep, involuntary weight loss, linear growth deceleration, extraintestinal symptoms (fever, rash, joint pain, recurrent aphthous ulcers), a positive family history of inflammatory bowel disease, iron deficiency anemia, and an elevated ESR are indications to pursue a diagnosis of inflammatory bowel disease by colonoscopy and barium contrast upper gastrointestinal series with small bowel follow-through. Lactose intolerance

TABLE 14-3	DIFFERENTIAL DIAGNOSIS OF RECURRENT ABDOMINAL PAIN ASSOCIATED WITH ALTERED BOWEL PATTERN

IDIOPATHIC INFLAMMATORY BOWEL DISORDERS
Ulcerative colitis
Crohn disease
Microscopic colitis with crypt distortion
Lymphocytic colitis
Collagenous colitis

INFECTIOUS DISORDERS
Parasitic (*Giardia*, *Blastocystis hominis*, *Dientamoeba fragilis*)
Bacterial (*Clostridium difficile*, *Yersinia*, *Campylobacter*, tuberculosis)

LACTOSE INTOLERANCE
COMPLICATION OF CONSTIPATION (MEGACOLON, ENCOPRESIS, INTERMITTENT SIGMOID VOLVULUS)
DRUG-INDUCED DIARRHEA, CONSTIPATION
GYNECOLOGIC DISORDERS
NEOPLASIA (LYMPHOMA, CARCINOMA)
PSYCHIATRIC DISORDERS

should be considered as a potential primary etiology of chronic abdominal pain in the presence of diarrhea. A trial of a lactose-free diet or performance of a lactose breath hydrogen test is prudent in children with pain associated with loose bowels, bloating, and increased flatulence. Diarrhea associated with encopresis suggests chronic fecal retention and megacolon. Serologic testing for celiac disease should be considered in patients with pain and an altered bowel pattern, especially in patients with iron deficiency anemia or secondary amenorrhea. Large-volume diarrhea is also an indication to pursue colonoscopy to rule out microscopic inflammation, which may alter colonic motility and absorptive function, including lymphocytic, collagenous, or eosinophilic colitis.[49–51]

Table 14-4 lists the indications for colonoscopy in children with chronic abdominal pain and an altered bowel pattern. The accuracy of colonoscopy in diagnosing inflammatory conditions of the colon is superior to barium enema because of the direct visualization of the mucosal surface and the ability to obtain biopsy and culture specimens. Intubation of the terminal ileum can also aid in the diagnosis of Crohn disease. Recognizable objective findings by gross examination with a flexible endoscope include edema, erosions, ulceration, pseudomembranes (discrete yellow plaques on the colonic mucosa), and polyps. Subjective gross endoscopic findings, including erythema, increased vascularity, and spontaneous friability,

TABLE 14-4	INDICATIONS FOR COLONOSCOPY IN PATIENTS WITH RECURRENT ABDOMINAL PAIN AND ALTERED BOWEL PATTERN

Evidence of gastrointestinal bleeding
Profuse diarrhea
Involuntary weight loss or growth deceleration
Iron deficiency anemia
Elevated acute-phase reactants (sedimentation rate, *C*-reactive protein)
Extraintestinal symptoms suggestive of inflammatory bowel disease (fever, rash, joint pains, recurrent aphthous ulceration)

become meaningful only in the context of histology because they are subject to more interobserver variation in interpretation. Objective histologic findings include (1) cryptitis, crypt abscesses, and crypt distortion with branching and dropout, suggesting ulcerative colitis or Crohn disease; (2) noncaseating granuloma specific for Crohn disease; (3) fibrosis and histiocyte proliferation in the submucosa suggesting Crohn disease; and (4) epithelial and intraepithelial lymphocytes or eosinophils with or without subepithelial collagen thickening in lymphocytic colitis, eosinophilic colitis, and collagenous colitis, respectively. Mild superficial increases in interstitial lymphocytes or eosinophils in the absence of crypt distortion or significant diarrhea are nonspecific and should not dissuade the physician from making a positive diagnosis of irritable bowel syndrome.

Chronic Isolated Paroxysmal Abdominal Pain. Table 14-5 lists the major differential of recurrent paroxysmal periumbilical abdominal pain in children. It is often important to try to see the patient during an attack of pain. The Carnett test may help to determine whether pain is arising from the abdominal wall or has an intra-abdominal origin.[52] The site of maximum tenderness is found through palpation. The patient is then asked to cross arms and assume a partial sitting position (crunch), which results in tension of the abdominal wall. If there is greater tenderness on repeat palpation in this position, abdominal wall disorders such as cutaneous nerve entrapment syndromes, abdominal wall hernia, myofascial pain syndromes, rectus sheath hematoma, or costochondritis should be suspected. Discitis, which is an osteomyelitis of the vertebral end plate, may present as a combination of back and abdominal pain.[53] The condition is usually associated with intermittent fever, an elevated peripheral WBC, and an elevated ESR. Unrecognized constipation should be suspected if a left lower quadrant or suprapubic fullness or mass effect is appreciated on abdominal examination and rectal examination reveals evidence of firm stool in the rectal vault or soft stool in a dilated rectal vault with evidence of perianal soiling. Often a history of constipation or encopresis is unknown to the parent. Parasitic infections, particularly *Giardia lamblia*, *Blastocystis hominis*, and *Dientamoeba fragilis*, may present with chronic pain in children in the absence of altered bowel pattern. Alarm signals are also indications to evaluate for Crohn disease or rare disorders such as polyarteritis nodosa, intestinal ischemia, and eosinophilic gastroenteritis, and angioneurotic edema can be indistinguishable from Crohn disease on clinical grounds. Suspicion of polyarteritis nodosa rests on evidence of extraintestinal disease, particularly renal involvement. Mesenteric vein obstruction should be considered in adolescents using oral contraceptives. Clinically, it can present gradually with progressive abdominal pain over a period of weeks. Pneumatosis is usually a late finding. The clinical presentation of eosinophilic gastroenteritis depends on the depth of the infiltration by the eosinophilic process. Submucosal disease can become manifest with abdominal pain and signs of obstruction. Any region of the gastrointestinal tract can be involved. Angioneurotic edema can be heralded by recurrent episodes of pain in the absence of cutaneous or oropharyngeal edema.[54] The family history is usually positive for allergy. Recurrent fever associated with generalized abdominal pain and peritoneal signs suggests the possibility of familial Mediterranean fever. Appendiceal colic is a controversial cause of chronic abdominal pain.[55,56] Appendiceal spasm has been postulated to be caused by inspissated casts of fecal material within the appendix. A number of anecdotal surgical reports have described complete resolution of pain symptoms following elective appendectomy. Appendiceal colic should be suspected in patients with recurrent acute episodes of well-localized abdominal pain and tenderness, most commonly in the right lower quadrant, demonstrated on several examinations. Ureteropelvic junction obstruction is well known to present with recurrent episodes of crampy periumbilical pain, but in all cases reported in the literature to date, the pain has been associated with vomiting.[57] Dull, midline, or generalized lower abdominal pain at the onset of a menstrual period suggests dysmenorrhea. The pain may coincide with the start of bleeding or precede the bleeding by several hours. Gynecologic disorders associated with secondary dysmenorrhea include endometriosis, partially obstructed genital duplications, ectopic pregnancy, and adhesions following pelvic inflammatory disease. Cystic teratoma has been described in prepubertal patients presenting with right or left lower quadrant pain. The vast majority of such patients have a palpable abdominal mass. Benign ovarian cysts in adolescent females do not cause recurrent abdominal pain. Acute intermittent porphyria is a rare disorder characterized by the temporal association of paroxysmal abdominal pain and a wide variety of central nervous system symptoms, including headache, dizziness, weakness, syncope, confusion, memory loss, hallucinations, seizures, and transient blindness.[58] Acute

TABLE 14-5   DIFFERENTIAL OF RECURRENT ABDOMINAL PAIN PRESENTING AS ISOLATED PAROXYSMAL ABDOMINAL PAIN

OBSTRUCTIVE DISORDERS
Crohn disease
Malrotation with or without volvulus
Intussusception with lead point
Postsurgical adhesions
Small bowel lymphoma
Endometriosis
Infection (tuberculosis, *Yersinia*)
Vascular disorders
Eosinophilic gastroenteritis
Angioneurotic edema

APPENDICEAL COLIC
DYSMENORRHEA
MUSCULOSKELETAL DISORDERS
URETEROPELVIC JUNCTION OBSTRUCTION
ABDOMINAL MIGRAINE
ACUTE INTERMITTENT PORPHYRIA
MENTAL DISORDERS (FACTITIOUS DISORDER, CONVERSION REACTION, SOMATIZATION DISORDER, SCHOOL PHOBIA)
FUNCTIONAL ABDOMINAL PAIN

intermittent porphyria is often precipitated by a low intake of carbohydrate or by specific drugs such as barbiturates or sulfonamides.

Focused laboratory evaluation might include CBC with differential and ESR to screen for occult systemic inflammatory condition. Decision to do stool ovum parasite examination is dependent on the incidence of *G. lamblia*, *B. hominis*, and *D. fragilis* within the community. The most valuable diagnostic test in a patient with symptoms suggesting obstruction is an upper gastrointestinal series and small bowel follow-through. Rare conditions such as lymphoma, angioneurotic edema, mesenteric vein thrombosis with ischemia, eosinophilic gastroenteritis, and pseudo-obstruction will also be suggested by barium contrast radiography. Abdominal ultrasonography and abdominal CT have low diagnostic yield for picking up appendiceal abnormalities with recurrent right lower abdominal pain. Colonoscopy and ileoscopy should be performed to rule out Crohn disease in such patients if bloodwork or upper gastrointestinal small bowel follow-through suggests the possibility of inflammatory disease. Elective laparoscopy with planned appendectomy should be considered in patients with chronic right lower quadrant pain and negative infectious, inflammatory, and anatomic evaluation. Head CT to rule out intracranial space-occupying lesions should be considered in patients with recurrent abdominal pain and headache.

TREATMENT OF FUNCTIONAL ABDOMINAL PAIN

Management of all four presentations of functional abdominal pain begins with a positive diagnosis and explanation of suspected pathophysiology and goals of therapy. Specific treatments include identification and modification of physical and psychological stress factors, dietary modification, drug therapy, and active psychological support. Hospitalization is rarely indicated for patients with functional abdominal pain.

Positive Diagnosis, Explanation of Suspected Pathophysiology, and Goals of Therapy. A positive diagnosis is based on normal physical examination and absence of alarm signals in the history, as described above. Focused laboratory and/or radiograph evaluations are based on subcategorizing pain presentation. It is important to emphasize that functional pain is the most common etiology of chronic abdominal pain in children and that the pain is real. Although the exact etiology and pathogenesis of functional abdominal pain in children are unknown, a substantial body of evidence suggests that it is caused by a disturbance of the autonomic nervous system, which results in altered communication between the gut and the brain. The prevailing viewpoint is that the pathogenesis of the pain involves visceral hypersensitivity and altered conscious awareness of gastrointestinal sensory input, with or without disordered gastrointestinal motility. Many parents and children can conceptualize the pain as a "headache" within the abdomen. Parents and child must be told that the primary goal of treatment is resumption of a normal lifestyle, not eradication of abdominal pain. Goals of treatment

include regular school attendance, school performance to the child's ability, participation in desired extracurricular activities, normal weight gain and growth, and a normal sleep pattern. Reassurance that functional pain disorders will not affect future health can have positive therapeutic effects. Many patients lose their symptoms spontaneously after a positive diagnosis, suggesting that allaying the patient's and/or parents' fears may remove a significant stress factor triggering symptoms.

Modify Triggers of Pain. The first goal is to identify, clarify, and possibly reverse physical and psychological stress factors (see above) that may have an important role in the onset, severity, exacerbations, or maintenance of pain. In some cases, painful sensations may be provoked by physiologic phenomena, including postprandial gastric or intestinal distention, gastric emptying, intestinal contractions or the migrating motor complex, intestinal gas, or gastroesophageal reflux. Concurrent physical and psychological stressful life events may also trigger flares of pain. Intraluminal physical stress factors that may trigger pain include aerophagia, simple constipation, lactose intolerance, minor noxious irritants such as spicy foods, *H. pylori* gastritis, celiac disease, or drug therapy. Systemic physical or psychological stress factors may also provoke or reinforce the pain behavior by altering the conscious threshold of gastrointestinal sensory input in the central nervous system. Acute or chronic physical illness may unmask functional pain. Psychological stress factors may include death or separation of a significant family member, physical illness or chronic handicap in parents or a sibling, school problems, altered peer relationships, family financial problems, or a recent geographic move.

Equally important is to reverse environmental reinforcement of the pain behavior. Parents and teachers must be engaged to support the child rather than the pain. Regular school attendance is essential regardless of the continued presence of pain. In many cases, it is helpful for the physician to communicate directly to school officials to explain the nature of the problem. School officials must be encouraged to be responsive to the pain behavior but not to let it disrupt attendance, class activity, or performance expectations. Within the family, less social attention should be directed toward the symptoms. Consultation with a child psychiatrist or psychologist may be indicated when there is concern about maladaptive family coping mechanisms or if attempts at environmental modification do not result in return to a normalized lifestyle.

It is important to address symptoms of mental disorders that may contribute to the pathogenesis of pain symptoms. Failure to treat attention-deficit/hyperactivity, anxiety, or depression will adversely affect pain management. Anxiety may be primary, part of adjustment to an identifiable stress, or associated with panic disorder. Symptoms of anxiety include irritability, exaggerated startle response, poor concentration, worry, hypervigilance, motor restlessness, nervousness, difficulty sleeping, school phobia, fear of separation, and being easily fatigued. Depressive mood is suggested by insomnia, hypersomnia, anorexia, overeat-

ing, low energy, poor concentration, tearfulness, low self-esteem, poor concentration, feelings of hopelessness, and recurrent thoughts of death.

Dietary Modification.   The role of dietary modifications in the management of functional pain disorders is not established. Postprandial symptoms in functional dyspepsia may be improved by eating low-fat meals or by ingesting more frequent but smaller meals throughout the day. A high-fiber diet is recommended for both diarrhea-predominant and constipation-predominant irritable bowel and isolated functional pain. The goal for fiber intake in grams is calculated by adding the patient's age + 5. Excessive fiber in the diet may result in increased gas and distention and actually provoke pain. Malabsorption of dietary carbohydrates may act as provocative stimuli in functional abdominal pain. Most often, the patient does not perceive a temporal association between ingestion of a particular sugar and the abdominal pain. Avoidance of excessive intake of milk products (lactose), carbonated beverages (fructose), dietary starches (corn, potatoes, wheat, oats), or sorbitol-containing products (vehicle for oral medication, sugar substitute in gum and candy, ingredient in toothpaste, and a plasticizer in gelatin capsules) is not unreasonable. Confirmation of lactose intolerance by a lactose breath hydrogen test should be considered before recommending prolonged lactase enzyme replacement therapy or commercial milk products that have been pretreated with lactase enzyme. Excessive gas in patients with irritable bowel syndrome can be managed by advising the patient to eat slowly, to avoid chewing gum, and to avoid excessive intake of carbonated beverages, legumes, foods of the cabbage family, and foods or beverages sweetened with aspartame.

Medications.   There are no evidence-based data to support antisecretory therapy in pediatric patients with functional dyspepsia. Response rates in controlled clinical trials using antisecretory agents, both H_2 receptor antagonists and proton pump inhibitors, in adults with functional dyspepsia range from 35 to 80% compared with placebo response rates of 30 to 60%.[49] Meta-analyses of these trials suggest that acid reduction therapy is 10 to 30% more effective than placebo in relieving symptoms of ulcer-like (predominant abdominal pain) dyspepsia.[59] Conversely, there is no evidence that symptoms of nausea or bloating are relieved by antisecretory therapy. Given that acid reduction therapy may be beneficial in a subset of patients, it is not unreasonable to treat pediatric patients with ulcer-like dyspepsia with 4 to 6 weeks of an H_2 receptor antagonist. Patients who fail to respond or who relapse with step-down therapy should have upper endoscopy to establish a firm diagnosis of functional dyspepsia. If a firm diagnosis of functional dyspepsia is established by upper endoscopy, it is not unreasonable to continue acid inhibition therapy in patients who initially responded to shot-term empiric treatment but had recurrence of pain symptoms with attempts at step-down therapy. Short-term step-up to a proton pump inhibitor may be tried in patients who previously did not respond to an H_2 blocker. Metoclopramide, the only pro-

motility agent available in the United States, has not been studied in pediatric patients and has only limited testing in adults with functional dyspepsia. It is not unreasonable to treat dysmotility-like dyspepsia (strong component of nausea, early satiety, and bloating) with a time-limited course of metoclopramide, but the high incidence of adverse central nervous system side effects and extrapyramidal symptoms associated with metoclopramide makes it risky for long-term use. As stated above, although *H. pylori*–eradication therapy is not established to be effective in adults with functional dyspepsia, the available data clearly do not rule out the possibility. Thus, most pediatric gastroenterologists still will treat documented *H. pylori* in functional dyspepsia. There are no evidence-based data to support the use of antispasmodic or antinauseant drugs to treat dyspepsia.

There are also no evidence-based data on the effects of pharmacologic therapy in pediatric patients with IBS. Synthetic opioids such as loperamide and diphenoxylate or the bile salt binding agent cholestyramine may be helpful in treating diarrhea associated with IBS. Loperamide is preferred over diphenoxylate because it does not traverse the blood-brain barrier. Fiber supplements such as psyllium, methycellulose, or polycarbophil are effective in treating both constipation and diarrhea, but their value in relief of abdominal pain associated with IBS is controversial. Nonstimulating laxatives such as PEG powder, mineral oil, milk of magnesia, and lactulose are effective adjuncts in treating constipation-predominant IBS. Antispasmodic or anticholinergic agents are commonly used in clinical practice to treat visceral abdominal pain, although efficacy is controversial. Only enteric-coated peppermint oil capsules (with possible smooth muscle–relaxing properties) have been shown to be superior to placebo for reducing functional pain by a randomized, double-blinded control study.[60] The duration of therapy at which time pain response was assessed, however, was only 2 weeks. Excessive gas can be managed by advising the patient to eat slowly, to avoid chewing gum, and to avoid excessive intake of carbonated beverages, legumes, foods of the cabbage family, and foods or beverages sweetened with fructose or sorbitol. Simethicone or activated charcoal may help individual patients.

In uncontrolled, retrospective case series, prophylactic cyproheptadine and propranolol have been reported to reduce the frequency of attacks of abdominal migraine.[61,62]

Although there is a lack of formal randomized, placebo-controlled trials, there has been a recent surge in using antidepressant and psychotropic agents to treat both diarrhea-predominant IBS and functional dyspepsia in adults.[63] Anecdotally, this class of drugs appears to be effective in adults with or without psychiatric abnormalities, especially low-dose tricyclic antidepressants. These drugs may act as "central analgesics" to raise the perception threshold for abdominal pain or down-regulate pain receptors in the intestine. There are as yet no data on treatment of pediatric patients.

There has been a recent surge in the development of novel drugs for IBS in adults, such as 5-HT$_3$ receptor antagonists and 5-HT$_4$ agonists aimed at modifying gas-

trointestinal motor activity and restoring normal visceral sensation. A significant beneficial effect of the 5-HT$_3$ antagonist alosetron has been reported in diarrhea-predominant adult women with IBS.[64] A significant beneficial effect of the 5-HT$_4$ agonist tegaserod has been reported in constipation-predominant adult women with IBS.[65]

Direct Psychological Support. Consultation with a child psychiatrist or psychologist may be indicated when there is concern about maladaptive family coping mechanisms or if attempts at environmental modification do not result in return to a normalized lifestyle. Referral for psychological treatment can be proposed as part of a multi-specialty treatment package to help the patient manage the pain symptoms better. It is critical that the psychologist or psychiatrist initially focus on illness behavior and expand psychotherapeutic treatments as indicated only as the patient or parents begin to see the benefits of referral.

Cognitive behavioral therapies add strategies such as cognitive restructuring to behavioral interventions such as teaching relaxation and behavior management techniques. For example, a therapist would evaluate a patient's cognitive interpretation of bodily sensations and teach how cognition impacts affective experience and behavior. The perception that abdominal pain is a sign of impending physical disease must be countered both to address functional disability and to reassure the family that a functional diagnosis is credible. Attribution styles can also be examined for distortions. Patients are taught to treat their beliefs as hypotheses to be tested rather than accept their beliefs as inherently valid. Cognitive behavioral interventions targeting children's competence in social roles may be a useful adjunct to other medical treatment in reducing illness behavior. In addition, parents are trained to behaviorally reinforce appropriate coping behavior. There are evidence-based data that cognitive behavioral treatment helps to reduce pain and improve functioning. Cognitive behavioral therapy has been compared to standard supportive care of children with functional abdominal pain.[66–68] Both groups demonstrated reductions in pain at 3 months; however, those receiving cognitive behavioral treatment were more likely to be pain free at 6-month (55.6% vs 23.8%) and 12-month follow-up (58.8% vs 36.8%). These findings are very encouraging, although replication by different investigators is still needed.

Hospitalization. Hospitalization is rarely indicated for patients with functional abdominal pain. Fifty percent of patients experience relief of symptoms during hospitalization. However, no data have been presented that the natural history of the pain is affected. Hospitalization does not enhance the fundamental goals of environmental modification. More commonly, it will reinforce pain behavior.

PROGNOSIS OF FUNCTIONAL ABDOMINAL PAIN IN CHILDREN

There are no prospective studies of the outcome of any of the various presentations of functional abdominal pain. Once functional abdominal pain is diagnosed, subsequent follow-up rarely identifies an occult organic disorder. Interestingly, pain resolves completely in 30 to 50% of patients by 2 to 6 weeks after diagnosis. This high incidence of early resolution suggests that the child and parent accept reassurance that the pain is not organic and that environmental modification is effective treatment. Nevertheless, more long-term studies suggest that 30 to 50% of children with functional abdominal pain in childhood experience pain as adults, although in 70% of such individuals, the pain does not limit normal activity.[69–71] Thirty percent of patients with functional abdominal pain develop other chronic complaints as adults, including headaches, backaches, and menstrual irregularities. Based on a small number of patients, Apley and Hale have described several factors that adversely influence prognosis for a lasting resolution of pain symptoms during childhood, including male sex, age at onset less than 6 years, a strong history of a "painful family," and greater than 6 months elapsed time from the onset of pain symptoms to an established functional diagnosis.[72]

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