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GOLDMAN'S
CECIL
MEDICINE

西氏内科学

第24版

消化系统疾病分册

LEE GOLDMAN
ANDREW I. SCHAFER

北京大学医学出版社

GOLDMAN'S CECIL MEDICINE

24TH EDITION

西氏内科学

（第24版）

消化系统疾病分册

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PREFACE

The 24TH Edition of *Goldman's Cecil Medicine* symbolizes a time of extraordinary advances in medicine and in technological innovations for the dissemination of information. This textbook and its associated electronic products incorporate the latest medical knowledge in formats that are designed to appeal to learners who prefer to access information in a variety of ways.

The contents of *Cecil* have remained true to the tradition of a comprehensive textbook of medicine that carefully explains the *why* (the underlying normal physiology and pathophysiology of disease, now at the cellular and molecular as well as the organ level) and the *how* (now frequently based on Grade A evidence from randomized controlled trials). Descriptions of physiology and pathophysiology include the latest genetic advances in a practical format that strives to be useful to the nonexpert. Medicine has entered an era when the acuity of illness and the limited time available to evaluate a patient have diminished the ability of physicians to satisfy their intellectual curiosity. As a result, the acquisition of information, quite easily achieved in this era, is often confused with knowledge. We have attempted to counteract this tendency with a textbook that not only informs but also stimulates new questions and gives a glimpse of the future path to new knowledge. Grade A evidence is specifically highlighted in the text and referenced at the end of each chapter. In addition to the information provided in the textbook, the Cecil website supplies expanded content and functionality. In many cases, the full articles referenced in each chapter can be accessed from the Cecil website. The website is also continuously updated to incorporate subsequent Grade A information, other evidence, and new discoveries.

The sections for each organ system begin with a chapter that summarizes an approach to patients with key symptoms, signs, or laboratory abnormalities associated with dysfunction of that organ system. As summarized in Table 1-1, the text specifically provides clear, concise information regarding how a physician should approach more than 100 common symptoms, signs, and laboratory abnormalities, usually with a flow diagram, a table, or both for easy reference. In this way, *Cecil* remains a comprehensive text to guide diagnosis and therapy, not only for patients with suspected or known diseases but also for patients who may have undiagnosed abnormalities that require an initial evaluation.

Just as each edition brings new authors, it also reminds us of our gratitude to past editors and authors. Previous editors of *Cecil Medicine* include a short but remarkably distinguished group of leaders of American medicine: Russell Cecil, Paul Beeson, Walsh McDermott, James Wyngaarden, Lloyd H. Smith, Jr., Fred Plum, J. Claude Bennett, and Dennis Ausiello. As we welcome new associate editors—Wendy Levinson, Donald W. Landry, Anil Rustgi, and W. Michael Scheld—we also express our appreciation to Nicholas LaRusso and other associate editors from the previous editions on whose foundation we have built. Our returning associate editors—William P. Arend, James O. Armitage, David Clemmons, Jeffrey M. Drazen, and Robert C. Griggs—continue to make critical contributions to the selection of authors and the review and approval of all manuscripts. The editors, however, are fully responsible for the book as well as the integration among chapters.

The tradition of *Cecil Medicine* is that all chapters are written by distinguished experts in each field. We are also most grateful for the editorial assistance in New York of Theresa Considine and Silva Sergenian. These individuals and others in our offices have shown extraordinary dedication and equanimity in working with authors and editors to manage the unending flow of manuscripts, figures, and permissions. We also thank Faten Aberra, Reza Akari, Robert C. Brunham, Ivan Ciric, Seema Daulat, Gregory F. Erikson, Kevin Ghassemi, Jason H. Huang, Caron Jacobson, Lisa Kachnic, Bryan T. Kelly, Karen Krok, Heather Lehman, Keiron Leslie, Luis Marcos, Michael Overman, Eric Padron, Bianca Maria Piraccini, Don W. Powell, Katy Ralston, James M. Swain, Tania Thomas, Kirsten Tillisch, Ali Turabi, Mark Whiteford, and Y. Joseph Woo, who contributed to various chapters. At Elsevier, we are most indebted to Dolores Meloni and Linda McKinley, and also thank Cathy Carroll, Taylor Ball, Virginia Wilson, Linda Van Pelt, Suzanne Fannin, and Steve Stave, who have been critical to the planning and production process under the direction of Mary Gatsch. Many of the clinical photographs were supplied by Charles D. Forbes and William F. Jackson, authors of *Color Atlas and Text of Clinical Medicine*, Third Edition, published in 2003 by Elsevier Science Ltd. We thank them for graciously permitting us to include their pictures in our book. We have been exposed to remarkable physicians in our lifetimes and would like to acknowledge the mentorship and support of several of those who exemplify this paradigm—Robert H. Gifford, Lloyd H. Smith, Jr., Frank Gardner, and William Castle. Finally, we would like to thank the Goldman family—Jill, Jeff, Abigail, Mira, Daniel, and Robyn Goldman—and the Schafer family—Pauline, Eric, Pam, John, Evan, and Kate—for their understanding of the time and focus required to edit a book that attempts to sustain the tradition of our predecessors and to meet the needs of today's physician.

LEE GOLDMAN, MD
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CONTENTS

XII

GASTROINTESTINAL DISEASES

134 APPROACH TO THE PATIENT WITH GASTROINTESTINAL DISEASE

KENNETH MCQUAID

The luminal gastrointestinal (GI) tract (esophagus, stomach, duodenum, small and large intestine, and anus) and pancreas are responsible for digestion, for the absorption of nutrients and fluids, and for the temporary storage and excretion of undigested waste. The GI tract has an epithelial lining with an enormous surface area that provides nutrient absorption and serves as a barrier to microorganisms. In addition, the GI tract has a large innate and adaptive immune system that interfaces with luminal food antigens, host proteins, commensal and pathogenic bacteria, and parasites and must decide which antigens to tolerate and which require immune activation. The GI tract also contains an extensive enteric endocrine system that regulates food intake, weight control, and glucose homeostasis, as well as secretions from the stomach, intestine, and pancreas. Finally, it has an enteric nervous system that is integrated with the autonomic and central nervous systems to control gastric emptying, intestinal motility, and defecation.

Numerous diseases within and outside the GI tract may alter normal function by causing structural damage (erosion, ulceration, perforation, stenosis, or obstruction), bleeding, inflammation, abnormal absorption or secretion of nutrients and electrolytes, or abnormal motility. Despite its anatomic and physiologic complexity, the GI system has only a limited repertoire of symptoms and signs to express conditions that may be either serious or clinically insignificant: abdominal pain, heartburn, regurgitation, dysphagia, odynophagia, dyspepsia, nausea and vomiting, gas and bloating, weight loss, diarrhea, constipation, overt or occult gastrointestinal bleeding, and incontinence.

GENERAL APPROACH TO PATIENTS WITH GASTROINTESTINAL SIGNS AND SYMPTOMS

An appropriate history and physical evaluation usually can narrow the differential diagnosis of GI complaints. A specific diagnosis can almost always be established thereafter by the judicious use of laboratory, endoscopic, or imaging studies (Table 134-1).

Clinical History

The clinician should elicit the nature of the complaint, including its acuity, severity, location, radiation, duration, pattern (steady vs. colicky; abrupt vs. gradual onset), and relationship to food, meals, and bowel movements. Symptoms that arise from the GI tract are almost always improved or worsened by eating or by bowel movements. For symptoms of recent onset, it is important to elicit recent dietary intake, a medication history, potential exposure to enteric infections or sexually transmitted diseases (Chapter 293), and recent travel. It is also useful to establish whether there are signs or symptoms that suggest a systemic illness, including fever, weight loss, arthralgias, fatigue, weakness, or skin rash.

Most nonsurgical GI diseases manifest with mild to moderate symptoms that develop gradually and do not require immediate attention. Acute symptoms that require urgent assessment are severe abdominal pain and overt GI bleeding (Chapter 137) that manifests by hematemesis, melena, or large-volume hematochezia. Severe or dramatic abdominal pain that develops acutely over minutes to hours requires urgent evaluation to determine whether surgical intervention is required. Severe vomiting or diarrhea with signs of dehydration also warrants urgent attention.

Mild to moderate chronic or intermittent symptoms that have been present for a long period can be evaluated in a deliberate fashion. A substantial proportion of chronic GI complaints have no obvious organic or biochemical basis and ultimately are classified as *functional* GI disorders (Chapter 139). Complaints that have been ongoing for years rarely are attributable to readily remedied structural disorders. In patients with chronic GI symptoms, it is important to elicit and address the current reason for seeking evaluation, which may include concern for underlying serious illness (especially cancer), a change in the character or severity of symptoms, life stressors, or depression. Asking the patient what he or she thinks or fears may provide insights into the proportion of the complaint attributable to these amplifying issues, regardless of whether the problem is functional or structural in origin.

A dietary history (Chapter 221) should be obtained. For acute symptoms of nausea, vomiting, diarrhea, or abdominal pain, intake over the previous 24 to 48 hours should be reviewed for clues to a food-borne illness, including possible exposure to a contaminated food or water source and similar symptoms in other people (Chapter 291). For chronic or intermittent complaints, a recall of meals and types of foods eaten over the previous 1 to 2 days provides insight into eating habits and the amounts and types of fruits and vegetables, whole grains, fiber, protein, fat, and dairy products ingested. A relationship between specific foods and symptoms may be found. For example, pain, flatulence, or diarrhea may be caused by dairy products (lactose intolerance), whole grains, legumes or cruciferous vegetables, or fatty meals (malabsorption), and chronic constipation may be due to a low-fiber diet. Recent and long-term changes in body weight should be elicited. Involuntary loss of greater than 5% of body weight over the prior 12 months is worrisome for serious disease and significant malnutrition (Chapter 222).

The number and consistency of bowel movements should be elicited, and any change in bowel habits must be explored. Signs of acute GI bleeding (melena or hematochezia) or inflammatory colitis (blood, mucus, or pus) should be elicited. Improvement in symptoms after passage of flatus or a bowel movement suggests a disorder of the colon or anorectum.

Past Medical History

The past medical history should be reviewed for conditions that may cause acute or chronic GI symptoms, including endocrine disorders such as diabetes (Chapter 237) or thyroid dysfunction (Chapter 233), cardiovascular diseases such as heart failure (Chapter 58) or peripheral vascular disease (Chapter 81), chronic liver disease and portal hypertension (Chapter 156), neurologic conditions such as Parkinson's disease (Chapter 416) or neuromuscular disorders (Chapter 403), and rheumatologic and collagen vascular disorders (Chapter 264). In addition to their impact on GI tract function, the severity of these conditions must be considered when weighing the risks of diagnostic studies, especially endoscopy. Patients with symptomatic or advanced respiratory insufficiency (Chapter 83), sleep apnea (Chapter 100), valvular heart disease (Chapter 75), coronary artery disease (Chapter 50), heart failure (Chapter 58), cirrhosis (Chapter 156), cerebrovascular disease (Chapter 413), neuromuscular disease (Chapter 430), or dementia (Chapter 409) have an increased risk of sedation-related complications during endoscopy.

A list of prescription and nonprescription medications, vitamins, minerals, and other nutritional supplements should be obtained, paying particular attention to any that were recently initiated or changed. Herbal supplements (Chapter 38) are commonly used but are seldom reported without direct questioning. Medications are potential causes of odynophagia, dyspepsia, nausea or vomiting, abdominal pain, diarrhea, and constipation. The use of antiplatelet agents, including aspirin and anticoagulants, should be determined. The risks of stopping versus continuing these medications must be weighed in patients who have acute or chronic GI bleeding or in whom a therapeutic procedure is to be performed.

Social History

The patient's personal relationships, employment history, quality of life, alcohol intake (Chapter 32), and smoking (Chapter 31) history should be determined. It can be very informative to observe both verbal and nonverbal interactions between the patient and a partner or caregiver during an interview. Alcohol may cause heartburn, dyspepsia, nausea, diarrhea, or chronic liver disease. Many patients are reluctant to disclose the full extent of their alcohol intake on direct questioning; therefore, in addition to asking how often they imbibe (days/week and drinks/day), it may be revealing to inquire about their preferred beverage and how it is purchased (location, volume, and frequency). Cigarette smoking is associated with an increased risk of heartburn, peptic ulcer disease, Crohn's disease, and GI malignancies.

Clinicians should inquire about the degree to which GI symptoms are disrupting a patient's life. GI illness may affect dietary and bowel habits, sleep, and sense of vitality. Concerns about dietary intolerances, inability to eat, inability to have comfortable bowel movements, uncontrolled diarrhea or gas, fecal urgency, or fecal incontinence may affect a patient's social life, personal and sexual relationships, employment, and sense of optimism.

The social history should also be reviewed for recent stressors that may precipitate or exacerbate GI symptoms, including marital or interpersonal

discord, personal or family illness, bereavement, financial pressures, job loss, or change in employment. To elicit such information, it may be helpful to tell the patient that stress worsens many conditions and to inquire whether they believe stress may be contributing to their problem.

For elderly, disabled, or marginally housed patients, it is important to elicit how they obtain and prepare their meals and how they access toilet facilities. For patients undergoing GI procedures, it is important to determine whether they have mental, physical, or social barriers that would make it difficult to comply with pre-procedure instructions (including bowel preparation) and whether they have an able-bodied adult who can accompany them to the procedure and observe them at home, if necessary, afterward.

Family History

The family history should be reviewed for GI disorders with a heritable component, especially celiac disease (Chapter 142), inflammatory bowel diseases (Chapter 143), and GI, gynecologic, and genitourinary neoplasms.

Physical Examination

Nonabdominal Examination

The nonabdominal examination should assess nutritional status (Chapter 221) and any signs of systemic conditions that may cause GI symptoms or that must be considered when weighing the risks and benefits of further testing, especially endoscopy. Vital signs should be obtained in all patients. Low-grade fever (<100.5° F) is common in inflammatory conditions, including gastroenteritis, inflammatory bowel disease, appendicitis, cholecystitis, and diverticulitis. High fever (>102° F) suggests sepsis, pelvic, or intra-abdominal infections (e.g., cholangitis, pelvic inflammatory disease, pyelonephritis) or peritonitis. Hemodynamic instability (hypotension or tachycardia) suggests intravascular depletion due to poor oral intake, acute GI or intra-abdominal bleeding, severe diarrhea, or peritonitis. A body mass index less than 18 suggests malnourishment.

A general survey should be performed to assess for signs of weight loss (fat and muscle wasting), malnutrition (dry or thin skin, hair loss, edema, anasarca), and vitamin deficiencies (pellagra, scurvy). Skin lesions may provide clues to systemic conditions such as liver disease (jaundice, spider telangiectasias, palmar erythema), inflammatory bowel disease (erythema nodosum, pyoderma gangrenosum), celiac disease (dermatitis herpetiformis), vasculitis, and rare gastrointestinal malignancies, polyposis syndromes, and pancreatic endocrine tumors (Chapters 198, 199, and 201). An oral examination looks for mucocutaneous candidiasis (which may reflect immunosuppression), ulcerations (which may reflect inflammatory bowel disease, vasculitis, viral infection, or vitamin deficiencies), and glossitis or angular cheilitis (which may reflect vitamin deficiencies). With the exception of supraclavicular lymph nodes, peripheral lymph nodes are uninvolved with GI diseases but should be examined when systemic infection or advanced malignancy is suspected (Chapter 171). Examination of the lungs and cardiovascular system should focus on evidence of conditions that might increase the risk of moderate sedation in the event endoscopy is required (respiratory insufficiency, heart failure) and for conditions that increase the risk of intestinal ischemia (atrial fibrillation, valvular heart disease, peripheral vascular disease) (Chapter 145). The extremities should be evaluated for edema and peripheral pulses. Finally, a brief neurologic assessment should be performed to screen for intracranial mass lesions or other neurologic disorders that may present with GI symptoms.

Abdominal Examination

The abdominal examination begins with a visual inspection of the abdomen and inguinal region for scars (due to prior surgeries or trauma), asymmetry (suggesting a mass or organomegaly), distention (due to obesity, ascites, or intestinal ileus or obstruction), prominent periumbilical veins (suggesting portal hypertension), or hernias (umbilical, ventral, inguinal). The examination proceeds with auscultation followed by percussion, and it ends with light and deep palpation.

In patients *without* abdominal pain, auscultation of bowel sounds to assess intestinal motility has limited usefulness and may be omitted. Percussion may be performed before or in conjunction with light and deep palpation. Initial cursory light percussion across the upper, mid-, and lower abdomen is useful to denote areas of dullness and tympany, as well as to elicit unanticipated areas of pain or tenderness before palpation. More extensive percussion provides limited but useful information about the size of the liver and spleen, gastric or intestinal distention, bladder distention, and ascites (Chapters 148 and 156). Gentle, light palpation promotes abdominal relaxation and allows the detection of muscular resistance (guarding), abdominal tenderness, and superficial masses of the abdominal wall or abdomen. Deeper palpation of the abdominal organs (liver, spleen, kidneys, aorta) and abdominal cavity may detect enlargement or abnormal masses. Superficial or deep masses should be assessed for size, location, mobility, content (solid, liquid, or air), and the presence or absence of tenderness. The consistency of a patient's response to palpation with and without distraction is particularly useful in those with suspected chronic functional abdominal discomfort. Superficial masses include hernias, lymph nodes, subcutaneous abscesses, lipomas, and hematomas. Deep abdominal masses may be caused by neoplasms (liver, gallbladder, pancreas, stomach, intestine, kidney), abscesses (appendicitis, diverticulitis, Crohn's disease), or aortic aneurysms.

Examination of the right upper quadrant should assess the liver size, contour, texture, and tenderness. Liver size is crudely estimated by percussion of the upper and lower borders of liver dullness in the midclavicular line. Liver contour and tenderness are best assessed during held inspiration by deep palpation along the costal margin. Examination of the left upper quadrant is useful to detect splenomegaly (Chapter 171), although a normal-sized or even an enlarged spleen often cannot be detected. Percussion in the left upper quadrant near the tenth rib (posterior to the midaxillary line) may detect splenic dullness that is distinct from gastric or colonic tympany. The tip of an enlarged spleen may be palpated during inspiration if the examiner supports the left costal margin with the left hand while palpating below the costal margin with the right hand. Ascites should be suspected in a patient with a protuberant abdomen and bulging flanks. To screen for ascites, percussion of the flanks should be performed to assess the level of dullness. If the level of flank dullness appears to be increased, the most sensitive test for ascites is to check for "shifting" dullness when the patient rolls from the supine to the lateral position.

Digital Rectal and Pelvic Examinations

The digital rectal examination is intrusive and uncomfortable and should be performed only when necessary, such as in patients with perianal or rectal symptoms, incontinence, difficult defecation, suspected inflammatory bowel disease, and acute abdominal pain. The digital examination, with or without fecal occult blood testing, is not a useful screening test for colorectal cancer (Chapter 199). However, in patients with acute or chronic GI bleeding (Chapter 137), it is a rapid means of assessing the stool for color and occult blood. The perianal area should be visually inspected for rashes, soilage (suggesting incontinence or fistula), fistulas, fissures, skin tags, external hemorrhoids, and prolapsed internal hemorrhoids (Chapter 147). After gentle digital insertion, the anal canal should be assessed for resting tone and voluntary squeeze. The distal rectal vault should be swept circumferentially to palpate for mass lesions, tenderness, or fluctuance.

Laboratory Studies

Blood Tests

Blood tests routinely obtained in the evaluation of patients with GI symptoms include a complete blood count, liver tests (Chapter 149), serum chemistries, and, in selected cases, pancreatic enzymes and markers of inflammation. GI causes of anemia include acute or chronic GI blood loss, inflammatory bowel disease, nutrient malabsorption (folate, iron, or vitamin B_{12}), and chronic liver disease. Microcytosis suggests iron deficiency due to chronic GI blood loss or malabsorption. Macrocytosis may be attributable to folate or B_{12} malabsorption, medications (e.g., immunomodulators used for inflammatory bowel disease), or chronic liver disease. An elevated platelet count suggests chronic inflammation (e.g., inflammatory bowel disease) or GI blood loss with compensatory marrow production. A low platelet count may be attributable to portal hypertension with splenic sequestration. Low serum albumin may be caused by chronic GI disorders that result in weight loss, nutrient malabsorption, chronic inflammation, loss of protein across abnormal GI mucosa (i.e., protein-losing enteropathy), or decreased hepatic synthesis (e.g., chronic liver disease). Abnormal liver tests may be due to acute or chronic liver diseases, disorders of the pancreas or biliary tract, and medications (Chapter 149). Serum amylase and lipase are obtained to screen for pancreatitis (Chapter 146) in patients with acute abdominal pain. Increased levels of inflammatory markers, such as an elevated erythrocyte sedimentation rate and C-reactive protein, are nonspecific but useful in the management of patients with inflammatory bowel disease (Chapter 143).

Serum ferritin reflects total body iron and may be decreased in patients with chronic GI blood loss or intestinal malabsorption (e.g., celiac disease).

TABLE 134-1

	ABDOMINAL PAIN	GI BLEEDING	DIARRHEA	STEATORRHEA	CONSTIPATION
History (ascertain the following)	Duration: acute vs. chronic Onset: sudden vs. 1-2 hr vs. gradual Character: visceral (vague or dull, steady or cramping, diffuse) or parietal (severe, well localized, worse with movement) Location: upper, middle, or lower; radiation Associated symptoms: vomiting, hematemesis, diarrhea, hematochezia, melena, constipation, obstipation, jaundice Previous episodes Other diseases	Acute vs. chronic (duration); intermittent vs. continuous; quantity; hematemesis, melena, or hematochezia; associated pain and location; symptoms of anemia (e.g., dyspnea, chest pain, lightheadedness); medication use (especially aspirin, NSAIDs, anticoagulants); previous episodes; risk factors for chronic liver disease (alcohol, hepatitis)	Acute (<2 wk) vs. chronic (duration); fever, weight loss, or abdominal pain; stool character: number per 24 hr, watery or bloody, large vs. small volume, change in volume with eating, greasy; dietary history (especially lactose); history of IBD, pancreatic disease, intestinal surgery, DM; recent change in medications or antibiotic use; community outbreak or similar symptoms in family members; potential exposure to contaminated food; elderly immunosuppressed host; risk of HIV or sexually transmitted disease	Duration; weight loss; stool number, consistency (greasy), presence of blood; abdominal pain; flatulence; history of excessive alcohol, chronic liver disease, pancreatitis, intestinal dysmotility, surgery, DM; history of easy bruising, night blindness, bone pain, osteoporosis, dermatitis herpetiformis	Acute vs. chronic (duration); age; number of stools per wk; difficulty with defecation (straining, incomplete evacuation, digital manipulation); bloating or discomfort; blood on stools, weight loss; dietary fiber and fluid intake; chronic illness (DM, neuromuscular, endocrine); abdominal surgeries; medications, impaired mobility
Physical findings (evaluate for the following)	Fever, HR, BP Appearance: calm, restless, motionless Inspect: skin, distention, hernias Bowel sounds: present, absent, roaring Percussion and palpation: organomegaly, mass (abscess), focal tenderness, guarding Peritoneal signs: sharp pain with cough, shaking, percussion, light palpation	HR, BP, orthostatic findings; abdominal pain present or absent; signs of chronic liver disease and portal hypertension (which may indicate varices); jaundice, spider angiomas, hepatosplenomegaly; ascites; examine nasogastric aspirate for blood ("coffee grounds" vs. bright red); examine stool for blood (Hemoccult) and color (melena, maroon, or bright red)	HR, BP, orthostatic findings; fever; wasting; presence of abdominal tenderness or mass; perianal disease; extraintestinal symptoms of IBD (e.g., oral ulcers, arthritis, erythema nodosum)	Wasting, presence or absence of abdominal mass or tenderness; rash (vitamin deficiencies) or excessive bruising (vitamin K deficiency); jaundice or signs of chronic liver disease	Assess mobility and chronic medical conditions; abdominal distention; palpable stool within bowel in left lower quadrant; rectal examination—impacted stool, anal fissure, rectal prolapse, pelvic floor descent with straining, rectocele
Laboratory tests	CBC, BUN, Cr, glucose, amylase, lipase, liver tests (ALT, AST, bilirubin, alkaline phosphatase), albumin, INR, U/A, urine β-HCG	CBC BUN, Cr, liver tests, INR, type and cross	CBC, BUN, Cr, glucose, electrolytes, liver tests, albumin, C-reactive protein Selected cases: consider serum chromogranin A, VIP, calcitonin, gastrin, glucagon, urinary 5-HIAA; stool for culture, ova, parasites; consider fecal weight, fat, electrolytes, laxative screen	CBC, glucose, liver tests, albumin, electrolytes, celiac disease antibodies (anti-tTG or antiendomysial); assessment of vitamin and mineral absorption (A, D) and INR (K is fat soluble), folate, iron, calcium, phosphate, B_{12}; qualitative or quantitative stool for fecal fat; H_2 breath test for bacterial overgrowth	CBC, Chem-7, calcium, magnesium, phosphate, TFTs; selected patients with severe constipation may undergo colonic transit studies and/or anal manometry with balloon expulsion

NAUSEA AND VOMITING	DYSPHAGIA	ODYNOPHAGIA	HEARTBURN AND REGURGITATION	ANOREXIA	WEIGHT LOSS
Nausea with or without emesis; acute vs. chronic (duration); intermittent vs. constant; presence or absence of severe abdominal pain, comorbid illnesses, especially peptic ulcer, endocrine (DM), cardiac, psychiatric; medications; history of excessive alcohol	Oropharyngeal vs. esophageal dysphagia; solids vs. liquids; acute vs. chronic (duration); intermittent vs. progressive; GERD symptoms present or absent; weight loss; history of food impactions, allergies, atopic conditions, skin changes, cold hands (Raynaud's phenomenon)	Duration of pain with swallowing; underlying immunosuppression (e.g., HIV infection, DM); caustic ingestion; use of medications that cause topical injury (especially NSAIDs, KCl, bisphosphonates, iron, antibiotics, zidovudine)	Duration of symptoms; location; relation to meals or specific foods; nocturnal symptoms; dysphagia or chest pain; extraesophageal manifestations: cough, hoarseness, asthma	Acute vs. chronic (duration); association with different foods; psychiatric disease (e.g., depression, dementia); chronic or undiagnosed medical conditions (e.g., DM, thyroid or adrenal disease, COPD, advanced heart failure, renal insufficiency, malignancy, HIV infection); medication use	Acute vs. chronic (duration); age; total amount (>5% is significant); intentional vs. unintentional; appetite increased or decreased; rapid vs. gradual; change in physical activity; documented vs. undocumented; fever or sweats; anorexia, nausea, vomiting; diarrhea, steatorrhea, blood in stool; abdominal pain; history or symptoms of chronic medical, neurologic, or psychiatric illness; medications; alcohol and substance abuse
Acute with severe abdominal pain: evaluate for GI obstruction, pancreatitis, mesenteric ischemia, biliary colic, appendicitis, or other conditions causing peritonitis Acute without abdominal pain: evaluate for pregnancy, medications, food poisoning, infectious gastroenteritis, hepatitis, CNS disease, postoperative ileus Chronic: evaluate for medications, chronic gastric outlet obstruction (due to ulcer disease or malignancy), impaired GI motility (gastroparesis), other chronic medical conditions, intracranial disorders, psychiatric disease (bulimia)	Usually normal; examine oropharynx and neck for lymphadenopathy and masses; evaluate the skin for sclerodermatous changes	Usually normal; evaluate oropharynx for thrush, herpetic lesions, caustic injury; general exam for signs of underlying immunosuppression	Usually normal, unless extraesophageal manifestations are present	Wasting; fever; signs of bulimia (e.g., loss of tooth enamel, knuckle ulcerations and calluses); abdominal masses; enlarged lymph nodes	Wasting; malnutrition; poor dentition or poorly fitting dentures; thyromegaly; COPD or heart failure; abdominal masses; enlarged lymph nodes; pelvic masses in women; diabetic neuropathy; signs of depression, dementia, or bulimia
β-HCG, CBC, serum electrolytes, BUN, Cr, glucose, HbA_{1c}, liver tests, albumin, TFTs, cortisol	CBC; eosinophilia or elevated IgE in some patients with eosinophilic esophagitis	CBC, HIV test, fasting glucose	Usually normal	CBC, Chem-7, liver tests, albumin, HIV test, TFTs	CBC, Chem-7, HbA_{1c}, TFTs, liver tests, C-reactive protein or ESR, calcium, phosphate, albumin, HIV test, morning cortisol

TABLE 134-1

	ABDOMINAL PAIN	GI BLEEDING	DIARRHEA	STEATORRHEA	CONSTIPATION
Endoscopy	EGD, colonoscopy	EGD, colonoscopy, enteroscopy, wireless capsule study	Colonoscopy (including ileal inspection) with biopsies; EGD with duodenal biopsies; wireless capsule study	EGD with duodenal biopsies	Colonoscopy if recent change in bowel habits
Imaging	CT scan or ultrasound; angiography; small bowel enterography	Tagged RBC scan, angiography	Small bowel enterography: CT, MRI, or barium (Crohn's disease); somatostatin scintigraphy	CT of the abdomen (pancreatic calcifications; biliary dilation)	Usually not necessary; MRI or defecography

ALT = alanine transaminase; AST = aspartate transaminase; BP = blood pressure; BUN = blood urea nitrogen; CBC = complete blood count; CNS = central nervous system; COPD = chronic obstructive pulmonary disease; Cr = creatinine; CT = computed tomography; DM = diabetes mellitus; EGD = esophagogastroduodenoscopy; ESR = erythrocyte sedimentation rate; GERD = gastroesophageal reflux disease; GI = gastrointestinal; HCG = human chorionic gonadotropin; 5-HIAA = 5-hydroxyindoleacetic acid; HIV = human immunodeficiency virus; HR = heart rate; IBD = inflammatory bowel disease; INR = international normalized ratio; MRI = magnetic resonance imaging; NSAIDs = nonsteroidal anti-inflammatory drugs; RBC = red blood cell; TFTs = thyroid function tests; tTG = tissue transglutaminase; U/A = urinalysis; VIP = vasoactive intestinal polypeptide.
Adapted from Proctor DD. Approach to the patient with gastrointestinal disease. In: Goldman L, Ausiello D, eds. *Cecil Textbook of Medicine*, 23rd ed. Philadelphia: Saunders-Elsevier; 2008.

Disorders of malabsorption that result in steatorrhea may lead to deficiencies in the fat-soluble vitamins (A, D, E, K) (Chapter 142). The serum international normalized ratio may be elevated in patients with cholestasis owing to malabsorption of vitamin K or in patients with chronic liver disease due to decreased hepatic synthetic function. Serum B_{12} may be decreased in patients with autoimmune gastritis (pernicious anemia), gastric bypass surgery, or malabsorption due to small bowel bacterial overgrowth or disease of the terminal ileum (e.g., Crohn's).

Specialized laboratory tests that are useful for the diagnosis of specific diseases include antibodies to *Helicobacter pylori* in patients with peptic ulcer disease or dyspepsia, antibodies to tissue transglutaminase in celiac disease, antibodies to microbial antigens or autoimmune markers in inflammatory bowel disease (anti–*Saccharomyces cerevisiae* [ASCA], perinuclear antineutrophil cytoplasmic antibody [pANCA]), and CA-19-9 in pancreaticobiliary malignancy. Owing to their limited sensitivity and specificity, these tests are not useful for screening but may be helpful in circumscribed situations in which the results may shift the diagnostic probability.

Stool Examination

Fecal occult blood testing is useful to evaluate iron deficiency anemia and acute or chronic GI blood loss. In patients with acute diarrhea, assessment of fecal leukocytes or culture of common pathogens is routine, and in selected patients, testing for parasites (*Giardia, Entamoeba histolytica*), *Clostridium difficile, Escherichia coli* O157:H7, or other specific organisms may be warranted. To distinguish among the causes of chronic diarrhea (Chapter 142), stool samples may be sent for assessment of electrolytes, leukocytes, and fecal fat.

Endoscopy and Radiology

Endoscopy (Chapter 136) and radiographic studies (Chapter 135) play a major role in the evaluation and management of many GI disorders. Esophageal manometry and esophageal pH and impedance monitoring can be useful for the evaluation of heartburn, reflux, and other esophageal symptoms (Chapter 140). Anorectal manometry may be useful in some patients with fecal incontinence and defecatory dysfunction (Chapter 147). Breath tests are commonly used to diagnose *H. pylori* infection (Chapter 141), lactose intolerance, and small bowel bacterial overgrowth (Chapter 142).

The diagnosis of a functional GI disorder is made after organic disorders have been excluded by clinical evaluation and limited, directed diagnostic testing. "Overtesting" should be avoided. Thereafter, the emphasis should switch from finding a "cause" of the symptoms to implementing successful coping and adaptive behaviors.

ABDOMINAL PAIN

Abdominal pain, which is a frequent complaint among outpatients in the office setting and emergency department, may be benign and self-limited or the presenting symptom of severe, life-threatening disease. Chronic abdominal pain that has been present for months or years in the absence of other organic illness is almost always functional in origin and does not require urgent evaluation. By contrast, most patients with severe acute abdominal pain require a thorough but emergent evaluation, which may quickly reveal an acute surgical illness.

PATHOBIOLOGY

Stimulation of hollow abdominal viscera is mediated by splanchnic afferent fibers within the muscle wall, visceral peritoneum, and mesentery that are sensitive to distention and contraction. Visceral afferent nerves are loosely organized, innervate several organs, and enter the spinal cord at several levels. Thus, visceral pain is vague or dull in character and diffuse; patients attempting to localize the pain often move their entire hand over the upper, middle, or lower abdomen. Most visceral pain is steady, but cramping, intermittent pain or "colic" results from peristaltic contractions caused by partial or complete obstruction of the small intestine, ureter, or uterine tubes. In contrast to visceral innervation, the parietal peritoneum is innervated unilaterally by a dense network of nerve fibers that follow a spinal T6 to L1 somatic distribution. Pain fibers of the parietal peritoneum are stimulated by stretch or distention of the abdominal cavity or retroperitoneum; direct irritation from infection, pus, or secretions (e.g., caused by a ruptured viscus); or inflammation caused by contact between the parietal peritoneum and an adjacent inflamed organ (e.g., appendicitis). Parietal pain is sharp, well characterized, and localized by the patient to a precise location on the abdomen, often by pointing with one finger.

The gastrointestinal viscera (liver, biliary system, pancreas, and GI tract) arise during embryology from midline structures that have bilateral innervation. Thus, GI visceral pain is typically localized to the abdominal midline.

Acute Abdominal Pain

CLINICAL MANIFESTATIONS

History

The history should determine the time course, character, and location and radiation pattern of the pain (Table 134-2). Severe abdominal pain that begins suddenly over seconds to minutes indicates a catastrophic event such as esophageal rupture, perforated peptic ulcer or viscus, ruptured ectopic pregnancy, ruptured aortic aneurysm, acute mesenteric ischemia, or myocardial infarction. Pain that progresses within 1 to 2 hours is consistent with a rapidly progressive inflammatory disorder (e.g., cholecystitis, appendicitis, pancreatitis), acute obstruction of a viscus (small intestinal obstruction, ureteral colic), or organ ischemia caused by a strangulated blood supply (volvulus, strangulated hernia, ovarian torsion). Pain that is less severe and develops over several hours is more commonly caused by a medical rather than a surgical condition, including upper GI disorders (dyspepsia), intestinal disorders (gastroenteritis, inflammatory bowel disease), liver disorders (hepatitis, abscess), urinary disorders (cystitis, pyelonephritis), or gynecologic infections; however, the slow evolution of surgical disorders such as cholecystitis (Chapter 158), appendicitis or diverticulitis (Chapter 144), and intra-abdominal abscesses must not be overlooked.

The character of the pain provides important information about whether the symptoms are due to visceral stimulation or parietal stimulation

NAUSEA AND VOMITING	DYSPHAGIA	ODYNOPHAGIA	HEARTBURN AND REGURGITATION	ANOREXIA	WEIGHT LOSS
EGD to exclude gastric outlet obstruction	EGD with biopsies and/or dilation, esophageal motility study, 24-hr pH probe	EGD with biopsies	EGD (to detect erosive esophagitis or Barrett's esophagus); 24-hr pH probe	Directed at detecting underlying disease, e.g., if a GI cause is suspected, EGD and/or colonoscopy with biopsies may be helpful	Directed at detecting underlying disease, e.g., if a GI cause is suspected, EGD and/or colonoscopy with biopsies may be helpful
CT of the abdomen; if chronic, also consider head CT, gastric emptying study, small bowel enterography	Esophagogram (barium swallow) will show stricture, Schatzki's ring, mass, etc.	Usually not necessary	Usually not necessary	Directed at detecting underlying disease, e.g., if a GI cause is suspected, abdominal CT may be helpful	Directed at detecting underlying disease, e.g., chest or abdominal CT may be helpful

(peritonitis). Patients with peritonitis may report severe localized pain or irritation with activities or maneuvers that stretch or move the parietal peritoneum, such as walking, moving in bed, and coughing; as a result, they tend to lie quietly to avoid painful stimulation. By contrast, patients with visceral pain may move or walk restlessly or attempt a bowel movement in an effort to relieve their symptoms.

The location of pain in the upper, middle, or lower abdomen is a crude but important indicator of the diagnosis (Fig. 134-1). Visceral pain arising from the foregut (esophagus, stomach, proximal duodenum, bile duct, gallbladder, pancreas) most often manifests in the epigastrium. Pain derived from the midgut (small intestine, appendix, ascending colon, proximal transverse colon) presents in a periumbilical location. Pain derived from the hindgut (distal transverse colon, left colon, rectum) localizes to the lower midline between the umbilicus and symphysis pubis. Paired intra-abdominal organs such as the kidneys, ureters, ovaries, and fallopian tubes have unilateral innervation that localizes pain to the side of the involved organ. As some surgical conditions progress, the character and location of the pain shift from a visceral to a parietal pain pattern. Thus, early cholecystitis (Chapter 158) may present with vague midline epigastric pain that progresses to sharp right upper quadrant pain as localized peritoneal irritation develops. Likewise, appendicitis (Chapter 144) commonly begins with vague, diffuse periumbilical pain that evolves to sharp, well-localized right lower quadrant pain as peritonitis ensues.

Anorexia, vomiting, diarrhea, distention, and constipation are commonly seen with abdominal pain caused by both medical and surgical disorders. Although nonspecific, the *absence* of any of these symptoms is evidence against an emergent surgical or medical disorder because severe illness usually leads to reflex stimulation or inhibition of gastric and intestinal peristalsis. Vomiting is common in medical and surgical disorders involving the upper GI tract, including acute gastroenteritis, pancreatitis, gastric and small intestinal obstruction, and biliary tract disease. Pain that precedes the onset of vomiting is typical of surgical conditions, whereas the reverse is true of medical conditions (e.g., food poisoning, gastroenteritis). Abdominal pain with prominent diarrhea is most commonly caused by a medical condition (e.g., gastroenteritis, inflammatory bowel disease). Although constipation alone is a nonspecific complaint, the absence of stool passage and flatus is consistent with complete bowel obstruction or paralytic ileus.

Jaundice accompanying acute abdominal pain virtually always indicates a hepatobiliary disorder (Chapter 149), including obstruction of the biliary duct (choledocholithiasis, pancreatic carcinoma, cholangiocarcinoma), complications of acute cholecystitis, acute hepatitis (viral, ischemic), or hepatic malignancies. The possibility of cholangitis should be considered and excluded in all patients with acute abdominal pain and jaundice, especially if the patient has fever, chills, hypotension, altered mental status, or leukocytosis. Hematemesis with upper abdominal pain suggests a Mallory-Weiss tear, alcoholic gastritis, or peptic ulcer disease. Hematochezia with abdominal pain is most commonly caused by medical conditions such as infectious gastroenteritis or inflammatory bowel disease, but it also may be caused by ischemic colitis or mesenteric ischemia. Gross hematuria may be due to cystitis (Chapter 292) or a ureteral stone (Chapter 125). Abdominal pain with weight loss may be due to inflammatory bowel disease, chronic mesenteric ischemia, or advanced GI malignancies. In women, a missed menstrual period, adnexal pain, spotting, or cramping may suggest pregnancy, ectopic pregnancy, or spontaneous abortion. Acute pain between cycles may be caused by ovarian follicles or ruptured corpus luteum cysts. Pelvic pain with fever, chills, or cervical discharge suggests pelvic inflammatory disease.

The past medical history and review of systems can provide clues about systemic and extra-abdominal conditions that may present with abdominal pain. Acute coronary syndromes (Chapter 72), heart failure (Chapter 58), pneumonia (Chapter 97), or empyema may cause dyspepsia, epigastric or right or left upper quadrant pain, nausea, and vomiting. Metabolic conditions such as uremia (Chapter 132), diabetes with hyperglycemia or ketoacidosis (Chapter 236), hypercalcemia (Chapter 253), or acute adrenocortical insufficiency (Chapter 234) may cause pain, nausea, vomiting, and diarrhea. Acute intermittent porphyria (Chapter 217) and familial Mediterranean fever (Chapter 283) may cause recurrent episodes of severe pain and peritonitis that may be misdiagnosed, leading to unnecessary surgeries. Other causes of acute abdominal pain include narcotic withdrawal (Chapter 33), insect or reptile bites (Chapter 113), and lead or arsenic poisoning (Chapter 21).

Physical Examination

The physical examination must identify life-threatening illnesses that require urgent surgical evaluation. Nevertheless, the examination must be orderly, careful, and complete. If the examiner immediately palpates the site of maximal pain, the patient is unlikely to relax and cooperate for the remainder of the examination.

First, the patient should be observed and the abdomen inspected. Most patients remain calm, cooperative, and freely capable of moving during the examination. Patients who are writhing or restless may have pain due to visceral distention (e.g., renal colic, intestinal obstruction), whereas patients who lie motionless may have peritonitis. Gentle shaking of the bed or having the patient cough may elicit sharp, well-localized pain in patients with parietal but not visceral pain. Auscultation should be performed before percussion or palpation so that intestinal activity is undisturbed. An abdomen that is quiet except for infrequent squeaks or tinkles suggests peritonitis or ileus. Loud peristaltic rushes that occur in synchrony with abdominal pain suggest small bowel obstruction. Light percussion across the upper, middle, and lower abdomen can determine any site of focal tenderness suggestive of peritonitis. Light palpation should be performed with one or two fingers (not the whole hand), beginning away from where the patient localizes the pain and gradually moving to the site of pain. Thereafter, gentle, deeper palpation of the entire abdomen is performed gradually, including the region of tenderness. An attempt should be made to palpate for an abdominal aortic aneurysm (Chapter 78). Examination also should include the inguinal and femoral canals, umbilicus, and surgical scars for evidence of incarcerating hernias. The presence of focal tenderness indicates parietal peritoneal irritation. Voluntary or involuntary tightening of the muscle wall ("guarding") may occur during

TABLE 134-2

CONDITION	LOCATION	QUALITY	ONSET	AGGRAVATING OR RELIEVING FACTORS	ASSOCIATED SYMPTOMS OR SIGNS	DIAGNOSTIC STUDIES
Peptic ulcer disease (Chapter 141)	Epigastric, occasionally RUQ, rarely LUQ	Dyspepsia: mild to moderate aching discomfort, pain, burning, gnawing, postprandial fullness	Days	Variable relief with antacids; may be relieved by, worsened by, or unrelated to meals	Recurrent; associated factors (e.g., *Helicobacter pylori*, aspirin, NSAIDs)	Anemia, upper endoscopy, *H. pylori* testing
Acute pancreatitis (Chapter 146)	Epigastric, radiates to midback (occasionally RUQ or LUQ)	Diffuse, steady, stabbing, penetrating	1-2 hr	Aggravated by food; better when lying still and with narcotics	Severe nausea and vomiting; reduced or absent bowel sounds; associated factors (e.g., alcohol, gallstones)	Elevated amylase and lipase, CT
Acute cholecystitis (Chapter 158)	Epigastric, then moves to RUQ; may radiate to right scapula	Gradual, steady increase, moderate to severe	Hours	May follow a fatty meal; better with narcotics and surgery	Nausea, some vomiting, fever	Elevated WBC count, US or CT
Acute appendicitis (Chapter 144)	Periumbilical, then moves to RLQ	Vague initially; gradual, steady increase to intense, localized, pain	Hours	Unprovoked; better with narcotics and surgery	Anorexia, nausea, obstipation; occasional vomiting, fever late	Elevated WBC count, US or CT
Diverticulitis (Chapter 144)	LLQ or suprapubic	Moderate to severe, steady or cramping, sharp or aching, localized	Hours to days	Unprovoked; better with narcotics and antibiotics or surgery	Anorexia, nausea, distention, constipation or loose stools; partial relief with passage of flatus or BM; fever late	Elevated WBC count, CT
Ruptured viscus and peritonitis (Chapter 144)	Diffuse	Intense	Minutes to hours	Worse with cough or movement; better when lying still or with narcotics or surgery	Fever, anorexia, nausea, vomiting; lack of bowel sounds; tenderness with percussion, light touch, rebound; guarding and rigidity (late); loath to move	Elevated WBC count, CT
Intestinal ischemia (Chapter 145)	Small intestine—periumbilical; proximal (right) colon—periumbilical or RLQ; distal colon—LLQ	Severe, stabbing pain out of proportion to physical findings	Minutes	Chronic ischemia—occurs after eating; acute ischemia—usually unprovoked; better with narcotics, thrombus dissolution, stenting, surgical resection	Nausea, bloody diarrhea; associated factors (e.g., hypotension, cardiac arrhythmias)	Elevated WBC count, angiography or colonoscopy (colonic ischemia)
Strangulated hernia (Chapter 144)	Localized	Sharp, localized, intense; crampy or steady	Minutes to hours	Previous hernia history; unprovoked; better with narcotics and decompression, including surgery	Anorexia, nausea, vomiting, no stool or flatus passage if obstruction; bowel sounds variable—hyperactive early if obstruction present, but absent bowel sounds late, especially with peritonitis	Elevated WBC count, CT, US
Small or large bowel obstruction (Chapter 144)	Small intestine—periumbilical; proximal (right) colon—periumbilical or right abdomen; distal (left) colon—LLQ	Early—diffuse, colicky, crampy; late—steady and better localized	Hours to days	Aggravated by food; better with narcotics, NGT decompression, and/or surgery	Distention, anorexia, nausea, vomiting; no stool or flatus passage; small intestine—increased hyperperistaltic (rushes) bowel sounds (early) or quiet abdomen (late); large intestine—bowel sounds variable; associated factors (e.g., hernia, previous surgery)	CT

TABLE 134-2

CONDITION	LOCATION	QUALITY	ONSET	AGGRAVATING OR RELIEVING FACTORS	ASSOCIATED SYMPTOMS OR SIGNS	DIAGNOSTIC STUDIES
Abdominal abscess (Chapter 144)	Located over the abscess, usually LLQ or RLQ	Insidious, intense, constant	Days	May be aggravated by movement; better with abscess drainage	Fever, anorexia, nausea, abdominal mass	Elevated WBC count, CT
Acute hepatitis (Chapter 150)	RUQ	Dull or intense; localized	Days	Worse with deep inspiration	Jaundice, anorexia, nausea; liver enlarged and tender to palpation; associated factors (e.g., alcohol, infection)	Abnormal liver tests
GERD (Chapter 140)	Substernal or epigastric	Burning, gnawing	Days to years	Provoked by large or fatty meals or recumbency; relief with antacids	Recurrent; may have regurgitation, dysphagia, or extraesophageal manifestations (e.g., asthma, chronic cough, laryngitis)	Upper endoscopy (usually normal), 24-hr pH probe
Nonulcer (functional) dyspepsia (Chapter 139)	Epigastric	Mild to moderate discomfort, pain, burning, gnawing, postprandial fullness	Years	May be worsened by meals; cannot be reliably distinguished from ulcer disease by history alone	Other symptoms of functional disorders (IBS, fibromyalgia, pelvic pain)	Normal EGD
IBS (Chapter 139)	Variable; usually lower abdomen	Vague, crampy, sense of urgency	Years	Pain may be precipitated by dietary factors or stress; associated with change in bowel characteristics (e.g., frequency, form, difficulty with passage); relieved with stool passage	Bloating and abdominal distention	Normal sigmoidoscopy, colonoscopy, and CT, but these are usually not necessary for diagnosis
Chronic pancreatitis (Chapter 146)	Epigastric or periumbilical, radiates to midback	Intense, localized	Days to years	Aggravated by food; better with narcotics	Anorexia, nausea, vomiting; associated factors (e.g., alcohol)	Amylase and lipase may be normal; CT may show calcifications, dilated pancreatic duct, pseudocyst
Inflammatory or infectious enterocolitis (Chapters 142 and 291)	Small intestine—periumbilical; large intestine—right or left side of the abdomen over the colon; rectum—tenesmus	Crampy	Hours to days	Better with stool passage and treatment of underlying cause	Nausea, vomiting, bloody diarrhea; associated factors (e.g., infectious—food transmission, IBD—prolonged duration, family history)	Stool studies for culture, colonoscopy with biopsies
Malignancy (Chapter 199)	Variable, depending on cancer location	Variable; intense and crampy if bowel obstruction; steady and vague if local invasion	Days	Better with narcotics and cancer therapy	Primary vs. metastatic disease	CT and biopsies, PET
Pneumonia/pleurisy (Chapters 97 and 99)	Upper abdomen: epigastric, RUQ, or LUQ	Localized; worse with deep breathing	Hours to days	Painful breathing; better with antibiotics	Cough, fever, dyspnea	CXR
Angina and myocardial infarction (Chapters 71-73)	Retrosternal or epigastric	Pressure, squeezing, heaviness, or intense	Minutes	Worse with exertion; relief with nitroglycerin	Dyspnea, diaphoresis	ECG, cardiac enzymes, stress testing
Genitourinary disorders (Chapters 128 and 293)	Bladder—suprapubic; renal colic—abrupt, excruciating LLQ or RLQ pain radiating to the groin; prostate—dull, suprapubic; kidney—CVA	Constant or colicky; stone passage—restless, cannot find a comfortable position	Minutes to days	Better with antibiotics and pain medications (pyelonephritis or nephrolithiasis)	Hematuria, dysuria, prostate tenderness, fever	Urinalysis, urine culture, CT for stone disease

TABLE 134-2 SPECIFIC MANIFESTATIONS OF KEY CAUSES OF ACUTE AND CHRONIC ABDOMINAL PAIN—cont'd

CONDITION	LOCATION	QUALITY	ONSET	AGGRAVATING OR RELIEVING FACTORS	ASSOCIATED SYMPTOMS OR SIGNS	DIAGNOSTIC STUDIES
Ovarian cysts or torsion (Chapters 205 and 243)	LLQ or RLQ	Constant, intense	Minutes	Better with NSAIDs or surgery (torsion)	Nausea, vomiting; may be recurrent	US
Ruptured ectopic pregnancy (Chapter 247)	LLQ or RLQ	Constant, intense, stabbing	Minutes	Better with surgery	Rebound and guarding present, abnormal menses or amenorrhea	Acute anemia, elevated β-HCG, US
Musculoskeletal disorders	Specific muscle groups	Aching	Days	Better with heat or NSAIDs; aggravated by movement	History of muscle injury or exertion	Normal laboratory results
Herpes zoster (Chapter 383)	Dermatomal distribution	Burning, itching, neuropathic, constant	Days	Aggravated by touching the dermatome; better with pain or antiviral medications	Recurrent; rash may or may not be present	Skin culture or biopsy
Metabolic disorders (e.g., DM; Chapter 236)	Epigastric or generalized	Intense, constant	Hours to days	Worse with poor metabolic control (e.g., poor glucose control)	Recurrent; nausea, vomiting, diabetic neuropathy	Specific metabolic parameters abnormal (e.g., elevated glucose in DM)
Abdominal epilepsy (Chapter 410)	Epigastric or umbilical	Constant	Hours to days	Unprovoked; better with antiseizure therapy	Recurrent; may have associated seizure disorder	EEG
Dissecting or leaking abdominal aortic aneurysm (Chapter 78)	Over the aneurysm, radiates to the back or groin	Severe, searing, constant	Minutes to hours to days	History of HTN or CAD	Shock, pulsatile mass; bruit *not* usually present	Acute anemia, CT, angiography

BM = bowel movement; CAD = coronary artery disease; CT = computed tomography; CVA = costovertebral angle; CXR = chest x-ray; DM = diabetes mellitus; ECG = electrocardiogram; EEG = electroencephalogram; EGD = esophagogastroduodenoscopy; GERD = gastroesophageal reflux disease; HCG = human chorionic gonadotropin; HTN = hypertension; IBD = irritable bowel disease; IBS = irritable bowel syndrome; LLQ = left lower quadrant; LUQ = left upper quadrant; NGT = nasogastric tube; NSAIDs = nonsteroidal anti-inflammatory drugs; PET = positron emission tomography; RLQ = right lower quadrant; RUQ = right upper quadrant; US = ultrasonography; WBC = white blood cell.
Adapted from Proctor DD. Approach to the patient with gastrointestinal disease. In: Goldman L, Ausiello D, eds. *Cecil Textbook of Medicine*, 23rd ed. Philadelphia: Saunders-Elsevier; 2008.

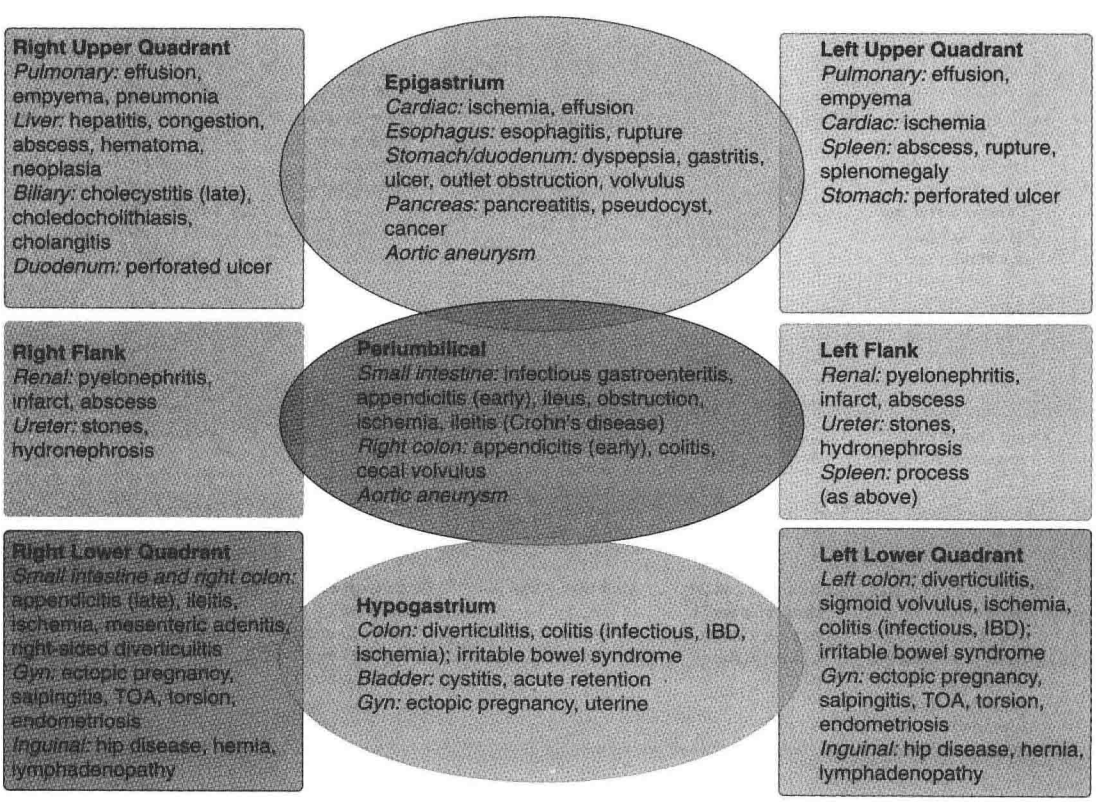

FIGURE 134-1. Differential diagnosis of abdominal pain by its initial location. IBD = inflammatory bowel disease; TOA = tubo-ovarian abscess.

palpation. With gentle, steady compression of the abdomen with one hand, voluntary guarding usually subsides, allowing the examination to proceed. Persistent involuntary guarding indicates peritonitis with reflex muscle wall contraction. Testing for "rebound tenderness" in patients with suspected peritonitis is not recommended because it causes significant pain and is usually not necessary to establish the diagnosis. When the presentation strongly suggests a nonserious GI disorder but the patient has significant tenderness with palpation, it is useful to use the stethoscope ostensibly to listen for bowel sounds but actually to reproduce the pressure of palpation. A significant discrepancy in the tenderness elicited by the stethoscope and by digital palpation may be seen in patients who are anxious, have functional complaints, or are seeking secondary gain. A digital rectal examination should be performed in most patients with acute abdominal pain to evaluate for tenderness or fluctuance that suggests a perirectal abscess and to assess the stool for signs of overt or occult blood. Women with lower abdominal pain should have a pelvic examination by a skilled examiner to evaluate for gynecologic pathology. Some specific and dramatic findings point to particular diagnoses (Table 134-3).

Special Populations

Increased diligence is required in the evaluation of patients in whom abdominal signs and symptoms may be minimal until the disease process is far advanced. Such patients include the elderly (Chapter 24) and patients who have dementia (Chapter 409), psychiatric disturbances (Chapter 404), or spinal cord injuries. An admitting diagnosis of "altered mental status," "failure to thrive," "obstipation," or "fever of unknown origin" may stem from serious intra-abdominal conditions. Disorders that may be overlooked in the elderly include bowel perforation, bowel obstruction, cholecystitis, diverticulitis, volvulus, mesenteric ischemia, and abdominal aortic aneurysm. In patients with chronic liver disease, the presence of ascites may mask the signs and symptoms of serious surgical conditions such as cholecystitis, appendicitis, and diverticulitis. Even in the presence of perforation, signs of peritonitis may be lacking because the ascites fluid separates the visceral peritoneum and parietal peritoneum. Likewise, immunocompromised populations, who are at risk for infectious, drug-related, and iatrogenic complications, may manifest few physical findings or laboratory abnormalities. Owing to the limitations of the clinical evaluation in these vulnerable populations, there should be a low threshold for the use of abdominal imaging.

Abdominal Pain Developing in the Hospital

When pain develops as a new problem in a hospitalized patient, it is usually caused by a limited number of conditions. Postprocedural complications may cause perforation, infection, or bleeding (intraperitoneal, retroperitoneal, or within solid organs). Shunting of splanchnic blood flow in severely ill medical or surgical patients may cause stress gastritis, nonocclusive mesenteric ischemia, or acalculous cholecystitis. Adynamic ileus or acute colonic pseudo-obstruction is common in critically ill or postoperative patients and manifests as diffuse abdominal pain and distention. *Clostridium difficile* (Chapter 304) colitis is a common cause of pain, diarrhea, and distention, especially in patients on antibiotics. Constipation (Chapter 138), which is a common problem in hospitalized patients, may go unnoticed until pain and distention develop. Finally, many medications can cause dyspepsia and abdominal pain.

TABLE 134-3 PHYSICAL SIGNS IN PATIENTS WITH ACUTE ABDOMINAL PAIN

SIGN	DESCRIPTION	DIAGNOSIS
Murphy's sign	Cessation of inspiration during right upper quadrant examination	Acute cholecystitis
McBurney's sign	Tenderness located midway between anterior superior iliac spine and umbilicus	Acute appendicitis
Cullen's sign	Periumbilical bluish discoloration	Retroperitoneal hemorrhage Pancreatic hemorrhage Ruptured abdominal aortic aneurysm
Grey Turner's sign	Bluish discoloration of flanks	Retroperitoneal hemorrhage Pancreatic hemorrhage Ruptured abdominal aortic aneurysm
Kehr's sign	Severe left shoulder pain	Splenic rupture Ectopic pregnancy rupture
Obturator sign	Pain with flexed right hip rotation	Appendicitis
Psoas sign	Pain with straight leg raising against resistance (right side)	Appendicitis

DIAGNOSIS

Patients with acute abdominal pain should have a complete blood count with differential; leukocytosis is present in most acute surgical conditions (Fig. 134-2). A pregnancy test is required in women of childbearing age. Serum electrolytes, glucose, blood urea nitrogen, and creatinine levels assess hydration, acid-base status, and renal function. Liver chemistries and pancreatic enzymes should be obtained in most patients, but especially in those with upper abdominal pain, jaundice, or vomiting. An elevation in aspartate or alanine aminotransferase levels may reflect choledocholithiasis with acute biliary obstruction (Chapter 158), acute gallstone pancreatitis (Chapter 146), or a hepatocellular process (Chapter 150). Painful jaundice with a significant rise in the alkaline phosphatase level usually reflects cholestasis caused by extrahepatic biliary obstruction (Chapter 158). Amylase and lipase levels are elevated in most patients with acute pancreatitis, but minor amylase elevations also occur with a perforated viscus or mesenteric ischemia (Chapter 145). Urinalysis may demonstrate pyuria, hematuria, or bacteriuria owing to ureteral calculi (Chapter 128) or urinary tract infection (Chapter 292).

Imaging

Ultrasound is preferred in suspected pregnancy and to evaluate for other acute gynecologic disorders such as tubo-ovarian abscess, ruptured corpus luteum cyst, or ovarian torsion; it is also preferred for the initial evaluation of suspected acute cholecystitis (Chapter 158) and ureteral stones with hydronephrosis (Chapter 125) and for the bedside evaluation of unstable patients. In most other settings, abdominal computed tomography (CT) with oral and intravenous contrast (when possible) is preferred and can provide a definitive diagnosis in up to 90% of patients with acute severe abdominal pain (Chapter 135). Abdominal CT may be falsely negative early in the course of acute pancreatitis, mesenteric ischemia, cholecystitis, appendicitis, and diverticulitis, especially if performed without contrast.

TREATMENT Rx

Once the diagnosis is clear, treatment of the underlying condition is initiated. In patients with nonspecific acute abdominal pain and no clear diagnosis, early laparoscopy is useful for diagnosis, but outcomes such as complication rates, readmission rates, and length of hospitalization are no better than with a strategy of active observation.[1]

Chronic Abdominal Pain

Chronic or recurrent abdominal pain that has been present for months to years may be caused by structural (organic) disease, but the majority of patients have a functional disorder such as irritable bowel syndrome (Chapter 139). Common organic causes of chronic abdominal pain include medications with GI side effects, peptic ulcer disease (Chapter 141), inflammatory bowel disease (Chapter 143), chronic pancreatitis (Chapter 146), biliary tract disease (Chapter 158), GI cancers (Chapters 198 and 199), and endometriosis (Chapter 244). The clinician should attempt to distinguish patients with symptoms or signs of organic disease, in whom further diagnostic investigation is warranted, from those with probable functional disease (Fig. 134-3). Although functional disorders occur in all age groups, the symptoms usually begin before age 40. "Alarm" features that suggest a structural disorder and are inconsistent with a functional disorder are fever, severe pain, significant weight loss, jaundice, progressive dysphagia, recurrent vomiting, nocturnal pain or diarrhea, and stools that are bloody or positive for fecal occult blood. Laboratory studies should be normal with functional disorders; therefore, an unrevealing evaluation for anemia, leukocytosis, and levels of iron, albumin, C-reactive protein, and vitamins A, D, or B_{12} argues against structural or organic disease.