Approach to the Patient with Gastrointestinal Disease

Objectives:
1. To describe an introduction to the gastrointestinal (GI) diseases.
2. To apply the student’s anatomical, histological, and physiological knowledge to predict the cause of the GI complain.
3. To give the student a simple introduction to approach a patient of GI disease.
4. To describe the common signs and symptoms of GI diseases.

ANATOMIC CONSIDERATIONS
The gastrointestinal (GI) tract extends from the mouth to the anus and is composed of several organs with distinct functions. Specialized independently controlled thickened sphincters that assist in gut compartmentalization separate the organs. The gut wall is organized into well-defined layers that contribute to functional activities in each region. The mucosa is a barrier to luminal contents or as a site for transfer of fluids or nutrients. Gut smooth muscle mediates propulsion from one region to the next. Many GI organs possess a serosal layer that provides a supportive foundation but that also permits external input. Interactions with other organ systems serve the needs both of the gut and the body. Pancreaticobiliary conduits deliver bile and enzymes into the duodenum. A rich vascular supply is modulated by GI tract activity. Lymphatic channels assist in gut immune activities. Intrinsic gut wall nerves provide the basic controls for propulsion and fluid regulation. Extrinsic neural input provides volitional or involuntary control to degrees that are specific for each gut region.

EXTRINSIC MODULATION OF GUT FUNCTION
GI function is modified by influences outside of the gut. Unlike other organ systems, the gut is in continuity with the outside environment. Thus, protective mechanisms are vigilant against deleterious effects of foods, medications, toxins, and infectious organisms. Mucosal immune mechanisms include
chronic lymphocyte and plasma cell populations in the epithelial layer and lamina propria backed up by lymph node chains to prevent noxious agents from entering the circulation. All substances absorbed into the bloodstream are filtered through the liver via the portal venous circulation. In the liver, many drugs and toxins are detoxified by a variety of mechanisms. Although intrinsic nerves control most basic gut activities, extrinsic neural input modulates many functions. Two activities under voluntary control are swallowing and defecation. Many normal GI reflexes involve extrinsic vagus or splanchnic nerve pathways. The brain gut axis further alters function in regions not under volitional regulation. As an example, stress has potent effects on gut motor, secretory, and sensory functions.

OVERVIEW OF GASTROINTESTINAL DISEASES
GI diseases develop as a result of abnormalities within or outside of the gut and range in severity from those that produce mild symptoms and no long-term morbidity to those with intractable symptoms or adverse outcomes. Diseases may be localized to one organ or exhibit diffuse involvement at many sites.

Classification of GI Diseases
GI diseases are manifestations of alterations in nutrient assimilation or waste evacuation or in the activities supporting these main functions.

IMPAIRED DIGESTION AND ABSORPTION
Diseases of the stomach, intestine, biliary tree, and pancreas can disrupt digestion and absorption. The most common intestinal maldigestion syndrome, lactase deficiency, produces gas and diarrhea after dairy products and has no adverse outcomes. Other intestinal enzyme deficiencies produce similar symptoms after ingestion of other simple sugars. Conversely, celiac disease, bacterial overgrowth, infectious enteritis, and radiation damage, which affect digestion and/or absorption more diffusely, produce anemia, dehydration, electrolyte disorders, or malnutrition. Gastric hypersecretory conditions such as Zollinger-Ellison syndrome damage the intestinal mucosa, impair pancreatic enzyme activation, and accelerate transit due to excess gastric acid. Biliary obstruction from stricture or neoplasm impairs fat digestion. Impaired pancreatic enzyme release in chronic pancreatitis or pancreatic cancer decreases intraluminal digestion and can lead to malnutrition.

ALTERED SECRETION
Selected GI diseases result from dysregulation of gut secretion. Gastric acid hypersecretion occurs in Zollinger-Ellison syndrome, and some individuals with duodenal ulcers. Conversely, patients with atrophic gastritis or pernicious anemia release little or no gastric acid. Inflammatory and infectious small-intestinal and colonic diseases produce fluid loss through impaired absorption or enhanced secretion. Common intestinal and colonic hypersecretory conditions cause diarrhea and include acute bacterial or viral infection, chronic *Giardia* infections, small-intestinal bacterial overgrowth, bile salt diarrhea, microscopic colitis, diabetic diarrhea, and abuse of certain laxatives.

**ALTERED GUT TRANSIT**
Impaired gut transit may be secondary to mechanical obstruction. Esophageal occlusion often results from acid-induced stricture or neoplasm. Gastric outlet obstruction develops from peptic ulcer disease or gastric cancer. Small-intestinal obstruction most commonly results from adhesions but may also occur with Crohn's disease, radiation- or drug-induced strictures, and less likely malignancy. The most common cause of colonic obstruction is colon cancer, although inflammatory strictures develop in patients with inflammatory bowel disease, after certain infections such as diverticulitis, or with some drugs.

**IMMUNE DYSREGULATION**
Many inflammatory GI conditions are consequences of altered gut immune function. The mucosal inflammation of celiac disease results from dietary ingestion of gluten-containing grains. Some patients with food allergy also exhibit altered immune populations.

**IMPAIRED GUT BLOOD FLOW**
Different GI regions are at variable risk for ischemic damage from impaired blood flow. For example, intestinal and colonic ischemias which are consequences of arterial embolus, arterial thrombosis, venous thrombosis, or hypoperfusion from dehydration, sepsis, hemorrhage, or reduced cardiac output. These may produce mucosal injury, hemorrhage, or even perforation.

**NEOPLASTIC DEGENERATION**
All GI regions are susceptible to malignant degeneration to varying degrees. In the United States, colorectal cancer is most common and usually presents after age 50 years. Worldwide, gastric cancer is prevalent especially in certain Asian regions. Esophageal cancer develops with chronic acid reflux or after an extensive alcohol or tobacco use history. Small-intestinal neoplasms are rare and occur with underlying inflammatory disease. Anal cancers arise after prior
anal infection or inflammation. Pancreatic and biliary cancers elicit severe pain, weight loss, and jaundice and have poor prognoses. Hepatocellular carcinoma usually arises in the setting of chronic viral hepatitis or cirrhosis secondary to other causes.

**DISORDERS WITHOUT OBVIOUS ORGANIC ABNORMALITIES**
The most common GI disorders show no abnormalities on biochemical or structural testing and include irritable bowel syndrome, functional dyspepsia, functional chest pain, and functional heartburn. These disorders exhibit altered gut motor function; however, the pathogenic relevance of these abnormalities is uncertain. Exaggerated visceral sensory responses to noxious stimulation may cause discomfort in these disorders. Symptoms in other patients result from altered processing of visceral pain sensations in the central nervous system. Functional bowel patients with severe symptoms may exhibit significant emotional disturbances on psychometric testing.

**GENETIC INFLUENCES**
Although many GI diseases result from environmental factors, others exhibit hereditary components. Family members of inflammatory bowel disease patients show a genetic predisposition to disease development themselves. For example, Colonic and esophageal malignancies arise in certain inherited disorders.

**EVALUATION OF THE PATIENT WITH GASTROINTESTINAL DISEASE**
Evaluation of the patient with GI disease begins with a careful history and examination. Subsequent investigation with a variety of tools designed to test gut structure or function are indicated in selected cases. Some patients exhibit normal findings on diagnostic testing. In these individuals, validated symptom profiles are employed to confidently diagnose a functional bowel disorder.

**History**
The history of the patient with suspected GI disease has several components. Symptom timing suggests specific etiologies. Symptoms of short duration commonly result from acute infection, toxin exposure, or abrupt inflammation or ischemia. Long-standing symptoms point to underlying chronic inflammatory or neoplastic conditions or functional bowel disorders. Symptoms from mechanical obstruction, ischemia, inflammatory bowel disease, and functional bowel disorders are worsened by meals. Conversely, ulcer symptoms may be relieved by eating or antacids. Symptom patterns and
duration may suggest underlying etiologies. Ulcer pain occurs at intermittent intervals lasting weeks to months, while biliary colic has a sudden onset and lasts up to several hours. Pain from acute inflammation as with acute pancreatitis is severe and persists for days to weeks. Meals elicit diarrhea in some cases of inflammatory bowel disease and irritable bowel syndrome. Defecation relieves discomfort in inflammatory bowel disease and irritable bowel syndrome. Functional bowel disorders are exacerbated by stress. Sudden awakening from sound sleep suggests organic rather than functional disease. Diarrhea from malabsorption usually improves with fasting, while secretory diarrhea persists without oral intake. Symptom relation to other factors narrows the list of diagnostic possibilities. Obstructive symptoms with prior abdominal surgery raise concern for adhesions, whereas loose stools after gastrectomy or gallbladder excision suggest dumping syndrome or postcholecystectomy diarrhea. Symptom onset after travel prompts a search for enteric infection. Medications may produce pain, altered bowel habits, or GI bleeding. Lower GI bleeding is likely results from neoplasms, diverticula, or vascular lesions in an older person and from anorectal abnormalities or inflammatory bowel disease in a younger individual. Celiac disease is prevalent in people of northern European descent, while inflammatory bowel disease is more common in certain Jewish populations. A sexual history may raise concern for sexually transmitted diseases or immunodeficiency.

For more than two decades, working groups have been convened to devise symptom criteria to improve the confident diagnosis of functional bowel disorders and to minimize the numbers of unnecessary diagnostic tests performed. The most widely accepted symptom-based criteria are the Rome criteria.

When tested against findings of structural investigations, the Rome criteria exhibit diagnostic specificities exceeding 90% for many of the functional bowel disorders.

**Physical Examination**

The physical exam complements information from the history. Abnormal vital signs provide diagnostic clues and determine the need for acute intervention. Fever suggests inflammation or neoplasm. Orthostasis is found with significant blood loss, dehydration, sepsis, or autonomic neuropathy. Skin, eye, or joint findings may point to specific diagnoses. Neck exam with swallowing assessment evaluates dysphagia. Cardiopulmonary disease may present with abdominal pain or nausea, thus lung and cardiac exams are important. Pelvic examination tests for a
gynecologic source of abdominal pain. Rectal exam may detect blood, indicating gut mucosal injury or neoplasm or a palpable inflammatory mass in appendicitis. Metabolic conditions and gut motor disorders have associated peripheral neuropathy.

Inspection of the abdomen may reveal distention from obstruction, tumor, or ascites or vascular abnormalities with liver disease. Ecchymoses develop with severe pancreatitis. Auscultation can detect bruits or friction rubs from vascular disease or hepatic tumors. Loss of bowel sounds signifies ileus, while high-pitched, hyperactive sounds characterize intestinal obstruction. Percussion assesses liver size and can detect shifting dullness from ascites. Palpation assesses for hepatosplenomegaly as well as neoplastic or inflammatory masses. Abdominal exam is helpful in evaluating unexplained pain. Intestinal ischemia elicits severe pain but little tenderness. Patients with visceral pain may exhibit generalized discomfort, while those with parietal pain or peritonitis have directed pain, often with involuntary guarding, rigidity, or rebound. Patients with musculoskeletal abdominal wall pain may note tenderness exacerbated by Valsalva or straight-leg lift maneuvers.

**Tools for Patient Evaluation**

Laboratory, radiographic, and functional tests can assist in diagnosis of suspected GI disease. The GI tract also is amenable to internal evaluation with upper and lower endoscopy and to examination of luminal contents. Histopathologic exams of GI tissues complement these tests.

**LABORATORY**

Selected laboratory tests facilitate the diagnosis of GI disease. Iron-deficiency anemia suggests mucosal blood loss, while vitamin B12 deficiency results from small-intestinal, gastric, or pancreatic disease. Either also can result from inadequate oral intake. Leukocytosis and increased sedimentation rates and C-reactive proteins are found in inflammatory conditions, while leukopenia is seen in viremic illness. Severe vomiting or diarrhea elicits electrolyte disturbances, acid-base abnormalities, and elevated blood urea nitrogen. Pancreaticobiliary or liver disease is suggested by elevated pancreatic or liver chemistries.

Thyroid chemistries, cortisol, and calcium levels are obtained to exclude endocrinological causes of GI symptoms. Pregnancy testing is considered for women with unexplained nausea. Serologic tests can screen for celiac disease, inflammatory bowel disease, and rheumatologic diseases like lupus or scleroderma.

Urine samples screen for bilirubin, and heavy metal intoxication.
LUMINAL CONTENTS
Luminal contents can be examined for diagnostic clues. Stool samples are cultured for bacterial pathogens, examined for leukocytes and parasites, or tested for *Giardia* antigen occult blood and undigested food or fungi. Duodenal aspirates can be examined for parasites or cultured for bacterial overgrowth. Fecal fat is quantified in possible malabsorption. Stool electrolytes can be measured in diarrheal conditions.

ENDOSCOPY
The gut is accessible with endoscopy, which can provide the diagnosis of the causes of bleeding, pain, nausea and vomiting, weight loss, altered bowel function, and fever. Upper endoscopy evaluates the esophagus, stomach, and duodenum, while colonoscopy assesses the colon and distal ileum. Upper endoscopy is advocated as the initial structural test performed in patients with suspected ulcer disease, esophagitis, neoplasm, malabsorption, and Barrett's metaplasia because of its ability to directly visualize as well as biopsy the abnormality. Colonoscopy is the procedure of choice for colon cancer screening and surveillance as well as diagnosis of colitis secondary to infection, ischemia, radiation, and inflammatory bowel disease.

RADIOGRAPHY/NUCLEAR MEDICINE
Radiographic tests evaluate diseases of the gut and extraluminal structures. Oral or rectal contrast agents like barium provide mucosal definition from the esophagus to the rectum. Contrast radiography also assesses gut transit and pelvic floor dysfunction. Ultrasound and computed tomography (CT) evaluate regions not accessible by endoscopy or contrast studies, including the liver, pancreas, gallbladder, kidneys, and retroperitoneum. These tests are useful for diagnosis of mass lesions, fluid collections, organ enlargement, and in the case of ultrasound gallstones. CT and magnetic resonance (MR) colonography are being evaluated as alternatives to colonoscopy for colon cancer screening.

HISTOPATHOLOGY
Gut mucosal biopsies obtained at endoscopy evaluate for inflammatory, infectious, and neoplastic disease. Biopsies obtained during CT or ultrasound can evaluate for intra-abdominal conditions not accessible by endoscopy.

FUNCTIONAL TESTING
Tests of gut function provide important data when structural testing is non-diagnostic. In addition to gastric acid and pancreatic function testing, functional testing of motor activity is provided by manometric techniques. Esophageal manometry is useful for suspected achalasia, whereas small-intestinal manometry tests for pseudoobstruction. A wireless motility capsule is now available to measure transit and contractile activity in the stomach, small intestine, and colon in a single test.

**Symptoms of Gastrointestinal Disease**
The most common GI symptoms are abdominal pain, heartburn, nausea and vomiting, altered bowel habits, GI bleeding, and jaundice

**ABDOMINAL PAIN**
Abdominal pain results from GI disease and extra-intestinal conditions involving the genitourinary tract, abdominal wall, thorax, or spine. Visceral pain generally is midline in location and vague in character, while parietal pain is localized and precisely described. Common inflammatory diseases with pain include peptic ulcer, appendicitis, diverticulitis, inflammatory bowel disease, and infectious enterocolitis. Other intraabdominal causes of pain include gallstone disease and pancreatitis. Non-inflammatory visceral sources could be the cause such as mesenteric ischemia.

**DYSPHAGIA**
Dysphagia—difficulty with swallowing—refers to problems with the transit of food or liquid from the mouth to the hypopharynx or through the esophagus. Severe dysphagia can compromise nutrition, cause aspiration, and reduce quality of life. Additional terminology pertaining to swallowing dysfunction is as follows. Aphagia denotes complete esophageal obstruction, most commonly encountered in the acute setting of a food bolus or foreign body impaction. Odynophagia refers to painful swallowing, typically resulting from mucosal ulceration within the oropharynx or esophagus. It commonly is accompanied by dysphagia, but the converse is not true. Globus pharyngeus is a foreign body sensation localized in the neck that does not interfere with swallowing and sometimes is relieved by swallowing.

**NAUSEA, VOMITING, AND INDIGESTION**
Nausea is the subjective feeling of a need to vomit. Vomiting (emesis) is the oral expulsion of gastrointestinal contents resulting from contractions of gut and thoracoabdominal wall musculature. Vomiting is contrasted with regurgitation, the effortless passage of gastric contents into the mouth. Rumination is the repeated regurgitation of stomach contents, which may be rechewed and reswallowed. In contrast to vomiting, these phenomena often exhibit volitional control. Indigestion is a nonspecific term that encompasses a variety of upper abdominal complaints including nausea, vomiting, heartburn, regurgitation, and dyspepsia (the presence of symptoms thought to originate in the gastroduodenal region). Some individuals with dyspepsia report predominantly epigastric burning, gnawing discomfort, or pain. Others with dyspepsia experience a constellation of symptoms including postprandial fullness, early satiety (an inability to complete a meal due to premature fullness), bloating, eructation (belching), and anorexia.

Nausea and vomiting are caused by conditions within and outside the gut as well as by drugs and circulating toxins.

Cause of nausea and vomiting could be Intraperitoneal like Obstructing disorders such as Pyloric obstruction or Small bowel obstruction or Colonic obstruction or could be vascular like Superior mesenteric artery syndrome or Enteric infections which could be Viral or Bacterial or Inflammatory diseases like Cholecystitis, Pancreatitis, or Appendicitis or liver disease like Hepatitis and rarely altered sensorimotor function.

Extraperitoneal cause could be cardiopulmonary disease like Myocardial infarction or Labyrinthine disease such as Motion sickness, Labyrinthitis or intracerebral disorders such as malignancy, Hemorrhage, abscess, or hydrocephalus.

Psychiatric illness should also be excluded such as Anorexia and bulimia or Nervosa.

Medications/Metabolic Disorders are also common causes which include drugs, Cancer chemotherapy, antibiotics, cardiac antiarrhythmics, Digoxin, oral hypoglycemics or oral contraceptives and also Toxins that should be excluded. Endocrine/metabolic disease could also cause nausea and vomiting such as pregnancy, Uremia, Ketoacidosis, or Thyroid and parathyroid disease.

**DIARRHEA AND CONSTIPATION:**

Diarrhea and constipation are exceedingly common and, together; exact an enormous toll in terms of mortality, morbidity, social inconvenience, loss of work productivity, and consumption of medical resources. Worldwide, >1 billion individuals suffer one or more episodes of acute diarrhea each year.
Although diarrhea and constipation may present as mere nuisance symptoms at one extreme, they can be severe or life-threatening at the other. Even mild symptoms may signal a serious underlying gastrointestinal lesion, such as colorectal cancer, or systemic disorder, such as thyroid disease. Given the heterogeneous causes and potential severity of these common complaints, it is imperative for clinicians to appreciate the pathophysiology, etiologic classification, diagnostic strategies, and principles of management of diarrhea and constipation, so that rational and cost-effective care can be delivered.

**DIARRHEA**
Diarrhea is loosely defined as passage of abnormally liquid or unformed stools at an increased frequency. For adults on a typical Western diet, stool weight >200 g/d can generally be considered diarrheal. Diarrhea may be further defined as *acute* if <2 weeks, *persistent* if 2–4 weeks, and *chronic* if >4 weeks in duration.

It is important to concentrate on hydration state of the patient as a first line of managements. Diarrhea classified according to the duration into:

**Acute Diarrhea**
More than 90% of cases of acute diarrhea are caused by infectious agents; these cases are often accompanied by vomiting, fever, and abdominal pain. The remaining 10% or so are caused by medications, toxic ingestions, ischemia, and other conditions.

**Chronic Diarrhea**
Diarrhea lasting >4 weeks warrants evaluation to exclude serious underlying pathology. In contrast to acute diarrhea, most of the causes of chronic diarrhea are noninfectious.

**CONSTIPATION**
Constipation is a common complaint in clinical practice and usually refers to persistent, difficult, infrequent, or seemingly incomplete defecation. Because of the wide range of normal bowel habits, constipation is difficult to define precisely. Most persons have at least three bowel movements per week; however, low stool frequency alone is not the sole criterion for the diagnosis of constipation. Many constipated patients have a normal frequency of defecation but complain of excessive straining, hard stools, lower abdominal fullness, or a sense of incomplete evacuation. The individual patient's symptoms must be analyzed in detail to ascertain what is meant by "constipation" or "difficulty" with defecation.
Types of Constipation and Causes:
Recent onset could be:
1. Colonic obstruction because of neoplasm; stricture: ischemic, or diverticular.
2. Anal sphincter spasm because of anal fissure or painful haemorrhoids.
3. Medications

Or Chronic that could be:
1. Irritable bowel syndrome.
2. Because of medications like Ca2+ blockers, antidepressants
3. Colonic pseudoobstruction

GASTROINTESTINAL BLEEDING:
Bleeding from the gastrointestinal (GI) tract may present in five ways. Hematemesis is vomitus of red blood or "coffee-grounds" material. Melena is black, tarry, foul-smelling stool. Hematochezia is the passage of bright red or maroon blood from the rectum.
Occult GI bleeding (GIB) may be identified in the absence of overt bleeding by a fecal occult blood test or the presence of iron deficiency. Finally, patients may present only with symptoms of blood loss or anemia such as lightheadedness, syncope, angina, or dyspnea.

Sources of Gastrointestinal Bleeding
UPPER GASTROINTESTINAL SOURCES OF BLEEDING
The annual incidence of hospital admissions for upper GIB (UGIB) in the United States and Europe is 0.1%, with a mortality rate of 5–10%. Patients rarely die from exsanguination; rather, they die due to decompensation from other underlying illnesses. The mortality rate for patients <60 years in the absence of major concurrent illness is <1%. Independent predictors of rebleeding and death in patients hospitalized with UGIB include increasing age, comorbidities, and hemodynamic compromise (tachycardia or hypotension).

Sources of Bleeding Proportion of Patients, %
Ulcers 31–67%
Varices 6–39%
Mallory-Weiss tears 2–8%
Gastroduodenal erosions 2–18%
Erosive esophagitis 1–13%
Neoplasm 2–8%
Vascular ectasias 0–6%
No source identified 5–14%
Approach to the Patient of Gastrointestinal Bleeding
Measurement of the heart rate and blood pressure is the best way to initially assess a patient with GIB. Clinically significant bleeding might lead to postural changes in heart rate or blood pressure, tachycardia, and, finally, recumbent hypotension. In contrast, the hemoglobin does not fall immediately with acute GIB, due to proportionate reductions in plasma and red cell volumes (i.e., "people bleed whole blood"). Thus, hemoglobin may be normal or only minimally decreased at the initial presentation of a severe bleeding episode. As extravascular fluid enters the vascular space to restore volume, the hemoglobin falls, but this process may take up to 72 h. Patients with slow, chronic GIB may have very low hemoglobin values despite normal blood pressure and heart rate. With the development of iron-deficiency anemia, the mean corpuscular volume will be low and red blood cell distribution width will increase.

DIFFERENTIATION OF UPPER FROM LOWER GIB
Hematemesis indicates an upper GI source of bleeding (above the ligament of Treitz). Melena indicates that blood has been present in the GI tract for at least 14 h.

JAUNDICE:
Jaundice, or icterus, is a yellowish discoloration of tissue resulting from the deposition of bilirubin. Tissue deposition of bilirubin occurs only in the presence of serum hyperbilirubinemia and is a sign of either liver disease or, less often, a hemolytic disorder. The degree of serum bilirubin elevation can be estimated by physical examination. Slight increases in serum bilirubin are best detected by examining the sclerae, which have a particular affinity for bilirubin due to their high elastin content. The presence of scleral icterus indicates a serum bilirubin of at least 51 mol/L (3 mg/dL). The ability to detect scleral icterus is made more difficult if the examining room has fluorescent lighting. If the examiner suspects scleral icterus, a second place to examine is underneath the tongue. As serum bilirubin levels rise, the skin will eventually become yellow in light-skinned patients and even green if the process is long-standing; the green color is produced by oxidation of bilirubin to biliverdin.

Approach to the Patient: Bilirubin
The bilirubin present in serum represents a balance between input from production of bilirubin and hepatic/biliary removal of the pigment. Hyperbilirubinemia may result from (1) overproduction of bilirubin; (2) impaired uptake, conjugation, or excretion of bilirubin; or (3) regurgitation of
unconjugated or conjugated bilirubin from damaged hepatocytes or bile ducts. An increase in unconjugated bilirubin in serum results from either overproduction, impairment of uptake, or conjugation of bilirubin. An increase in conjugated bilirubin is due to decreased excretion into the bile ductules or backward leakage of the pigment. The initial steps in evaluating the patient with jaundice are to determine (1) whether the hyperbilirubinemia is predominantly conjugated or unconjugated in nature, and (2) whether other biochemical liver tests are abnormal.

**ABDOMINAL SWELLING**
Abdominal swelling is a manifestation of numerous diseases. Patients may complain of bloating or abdominal fullness and may note increasing abdominal girth on the basis of increased clothing or belt size. Abdominal discomfort is often reported, but pain is less frequent. When abdominal pain does accompany swelling, it is frequently the result of an intraabdominal infection, peritonitis, or pancreatitis.

Patients with abdominal distention from ascites (fluid in the abdomen) may report the new onset of an inguinal or umbilical hernia. Dyspnea may result from pressure against the diaphragm and the inability to expand the lungs fully.

The causes of abdominal swelling can be remembered conveniently by the six Fs: flatus, fat, fluid, fetus, feces, or a "fatal growth" (often a neoplasm).

**Flatus**
Abdominal swelling may be the result of increased intestinal gas. The normal small intestine contains approximately 200 mL of gas made up of nitrogen, oxygen, carbon dioxide, hydrogen, and methane. Nitrogen and oxygen are consumed (swallowed), whereas carbon dioxide, hydrogen, and methane are produced intraluminally by bacterial fermentation. Increased intestinal gas can occur in a number of conditions. Aerophagia, the swallowing of air, increased intestinal gas is the result of bacterial metabolism of excess fermentable substances such as lactose and other oligosaccharides that can lead to production of hydrogen, carbon dioxide, or methane.

In irritable bowel syndrome and bloating, the subjective sense of abdominal pressure is attributable to impaired intestinal transit of gas rather than increased gas volume.

**Fat**
Weight gain with an increase in abdominal fat can result in an increase in abdominal girth and can be perceived as abdominal swelling. Abdominal fat may be the result of an imbalance between calorie intake and energy expenditure associated with a poor diet and sedentary lifestyle and also can be a manifestation of certain diseases such as Cushing's syndrome. Excess abdominal fat has been associated with an increased risk of insulin resistance and cardiovascular disease.

**Fluid**
Fluid within the abdominal cavity, or ascites, often results in abdominal distention and will be discussed in detail later.