Despite an array of sophisticated imaging techniques, the barium examination remains a valuable diagnostic test for evaluating structural abnormalities of the small bowel. Most patients can be examined with conventional small-bowel follow-through studies, in which periodic imaging of the entire small bowel is performed by using fluoroscopic guidance; however, some patients may benefit from enteroclysis, in which contrast agents are instilled into the small bowel via a catheter placed in the proximal jejunum for optimal distention and better depiction of individual small-bowel loops. This review for residents discusses the major diseases involving the mesenteric small bowel and presents a pattern approach for the wide spectrum of abnormalities found on barium studies, including polypoid lesions, cavitated lesions, annular lesions, outpouchings, separation of loops, abnormal folds, nodules without abnormal folds, and dilated small bowel.
The mesenteric small bowel is a long (variable in length) convoluted tube affected by a host of pathologic conditions that can be difficult and challenging to diagnose. Despite the increasing availability of sophisticated small-bowel imaging techniques (including computed tomography [CT] and magnetic resonance [MR] enterography, CT and MR enteroclysis, and capsule endoscopy), the conventional small-bowel follow-through (SBFT) study remains a common diagnostic test for patients suspected of having small-bowel disease. In a survey from 2004 (1), more than 60% of academic and community radiology groups from around the United States indicated that the volume of SBFT examinations in their practices was as large or larger than the volume of SBFT examinations 5 years earlier. The purpose of this review for residents is to discuss the major diseases involving the mesenteric small bowel (excluding the duodenum) and to facilitate diagnosis of these diseases by presenting a pattern approach for the wide spectrum of abnormalities found on barium studies of the small bowel.

### Essentials

- A properly performed small-bowel follow-through examination requires periodic imaging of the entire small bowel by using fluoroscopic guidance with manual palpation to separate individual small-bowel loops and identify abnormalities, rather than rely on serial overhead radiographs.
- The differential diagnosis for multiple submucosal masses or centrally ulcerated bull’s-eye lesions includes hematogenous metastases, lymphoma, carcinoid tumors, neurofibromas (although neurofibromas are rarely ulcerated), and Kaposi sarcoma, whereas the differential diagnosis for cavitated lesions includes hematogenous metastases, lymphoma, Kaposi sarcoma, and malignant gastrointestinal stromal tumors.
- Thickened folds that are straight segmental are usually caused by ischemia, vasculitis, radiation, or intramural hemorrhage, whereas those that are straight diffuse are usually caused by bowel wall edema from low serum albumin level, and those that are irregular segmental or irregular diffuse are caused by a host of infiltrative conditions involving the small bowel.
- Scleroderma is characterized by an increased number of jejunal folds (the hidebound sign) due to small-bowel fibrosis and contraction, whereas celiac disease is characterized by a decreased number of jejunal folds (fewer than four per inch) due to loss of mucosal surface area.
- Tubular narrowing of small bowel (with effaced or obliterated folds) is caused by strongyloidiasis, graft-versus-host disease, cytomegalovirus enteritis, or end-stage fibrosis due to Crohn disease, radiation enteropathy, or ischemic bowel disease.

### Normal Findings

The mesenteric small bowel gradually tapers in diameter from the duodenojejunal junction to the terminal ileum, so that it normally has a larger caliber in the jejunum (up to 3 cm in luminal diameter) than in the ileum (up to 2 cm) on SBFT studies. Closely spaced circumferential folds (also known as the valvulae conniventes or plicae circulares) are also thicker in the jejunum (normal thickness of 2–3 mm) than in the ileum (normal thickness of 1–2 mm) and more numerous in the jejunum than in the ileum on SBFT studies (5). Because of greater luminal distention with enteroclysis (normal jejunal diameter up to 4 cm and normal ileal diameter up to 3 cm), the folds tend to be thinner in both the jejunum (normal fold thickness of 1–2 mm) and the ileum (normal fold thickness of 1–1.5 mm) (Fig 1). It also is easier to assess the number of folds by using enteroclysis, which again are more numerous in the jejunum (four to seven folds per inch) than in the ileum (two to four folds per inch) (Fig 1). The folds are composed of mucosa and submucosa, whereas individual villi lining...
the folds are composed only of mucosa and lamina propria.

### Polypoid Lesions

Polypoid lesions in the small bowel can be mucosal or submucosal in origin. Mucosal and submucosal lesions can more readily be differentiated when they are larger than 2 cm in size, as it is easier to assess the angles these lesions form with the adjacent small-bowel wall for characterizing their site of origin (see following sections, Mucosal Lesions and Submucosal Lesions).

### Mucosal Lesions

Small-bowel polyps can be sessile or pedunculated. Polyps arising from the mucosa manifest on barium studies as protruded lesions that are smooth or lobulated, and, when viewed in profile, they form acute angles with the adjacent bowel wall. Some polyps have a nodular or granular surface due to trapping of barium in the interstices of the lesion (5). The most common benign mucosal polyps in the small bowel are adenomas and hamartomas.

The presence of multiple polyps in the mesenteric small bowel should suggest a polyposis syndrome such as familial adenomatous polyposis syndrome or Peutz-Jeghers syndrome (6). Multiple associated polyps in the duodenum should favor a diagnosis of familial adenomatous polyposis syndrome (7), whereas a small number (2–6) of large polyps in the distal duodenum and jejunum should favor a diagnosis of Peutz-Jeghers syndrome (8). The small-bowel polyps in Peutz-Jeghers syndrome are hamartomas (6). These polyps can be sessile or pedunculated and can occasionally cause bleeding or intussusception. Affected patients may also have pigmented lesions on the skin, lips, and buccal mucosa (6).

In contrast, the presence of one or more small sessile polyps in the distal ileum should suggest a carcinoid tumor (Fig 2), because a carcinoid is by far the most common neoplasm in the distal small bowel (see next section). Carcinoid tumors are thought to arise either from the basal layer of the epithelium or from the submucosa, so that they can appear as mucosal or submucosal lesions.

Primary small-bowel carcinomas may manifest on barium studies as polypoid masses, often containing ulceration due to necrosis of tumor. However, affected individuals usually develop obstructive symptoms only after these tumors have grown circumferentially to become annular lesions. Adenocarcinomas usually occur in the duodenum (at or distal to the papilla of Vater) or in the proximal jejunum within 30 cm of the ligament of Treitz (10).

### Submucosal Lesions

Submucosal masses in the small bowel usually have a smooth contour, and, when viewed in profile, they form right angles or slightly obtuse angles to the adjacent bowel wall. About half of these lesions undergo central ischemia and necrosis as they outgrow their blood supply, resulting in the development of centrally ulcerated submucosal masses, also known as “bull’s-eye” or “target” lesions (Fig 3) (11). When multiple submucosal masses are detected in the small bowel (with or without central ulcers), the differential diagnosis includes hematogenous metastases (especially from malignant melanoma and, less frequently, from carcinoma of the breast or lung), lymphoma (usually generalized lymphoma with associated small-bowel involvement), multiple carcinoid tumors (predominantly in the ileum), multiple neurofibromas (although neurofibromas are rarely ulcerated), and Kaposi sarcoma (in patients with acquired immunodeficiency syndrome [AIDS]) (5). A solitary submucosal mass in the small bowel is more likely to be a benign mesenchymal lesion such as a lipoma (Fig 4) or, less frequently, a gastrointestinal stromal tumor, hemangioma, or neurofibroma.

When large pedunculated lesions are detected in the ileum, the most common causes include a lipoma, an inflammatory fibroid polyp, and an inverted Meckel diverticulum (12,13). All of these lesions can act as the lead point for a small-bowel intussusception in adults.

### Intussusception

Idiopathic small-bowel intussusceptions (particularly jejunal intussusceptions) are often seen as a transient finding on abdominal CT scans in asymptomatic patients (14). In contrast, small-bowel intussusceptions diagnosed on barium
studies usually cause symptoms and in adults are precipitated by an underlying lesion such as a lipoma, carcinoma, metastatic melanoma (Fig 5), or inverted Meckel diverticulum acting as the lead point for the intussusception (Fig 6) (15). Barium studies may reveal a coil spring appearance caused by a small-bowel segment (the intussusceptum) telescoping into an adjacent loop (the intussuscipiens), with the coil spring representing the folds of the intussuscipiens (15). CT may be helpful for confirming the presence of an underlying mass lesion and for determining its cause (eg, fat in a lipoma).

Gallstone Ileus

Gallstone ileus is a complication of acute calculous cholecystitis, in which a gallstone has eroded through the gallbladder wall into the duodenum, producing a cholecystoduodenal fistula. Depending on the size of the stone, it may become impacted in the small bowel (usually near the ileocecal valve), producing a mechanical small-bowel obstruction (so the term gallstone ileus is a misnomer).

Gallstone ileus is characterized on abdominal radiographs by the following three findings (Rigler's triad): dilated small bowel, air in the biliary tree (pneumobilia), and a calcified ectopic gallstone in the abdomen (16). Barium studies may show barium outlining the stone as a smooth polypoid defect causing partial or complete small-bowel obstruction (Fig 7) (17). Barium may occasionally be seen to enter the gallbladder via a cholecystoduodenal fistula (17). CT is a more sensitive technique, however, for detecting pneumobilia or ectopic gallstones that are faintly calcified (18).

Cavitated Lesions

Cavitated lesions are almost always malignant tumors that have undergone extensive necrosis. The differential diagnosis depends on whether the lesions are solitary or multiple.

Hematogenous Metastases

Autopsy investigations have shown that malignant melanoma has the highest percentage of hematogenous metastases to the small bowel of any malignant tumor. Metastases from melanoma have a marked tendency to undergo necrosis, usually resulting in the development of multiple large (>2-cm) cavitated lesions in the stomach, small bowel, or both (19). Hematogenous metastases to the small bowel from carcinoma of the lung, colon, kidney, or other sites may produce identical findings.
Lymphoma
B-cell non-Hodgkin lymphoma involving the small bowel may manifest as cavitated lesions indistinguishable from necrotic metastases (Fig 8); however, lymphoma tends to involve the distal ileum, whereas metastases are located throughout the small bowel and tend to be more numerous. Cavitated lesions in lymphoma should also be distinguished from aneurysmal dilatation, in which involvement of the muscularis propria by tumor causes focal ballooning of the small-bowel lumen (20). Most patients with two or more discrete lymphomatous lesions (whether cavitated or not) have generalized lymphoma with associated bowel involvement, whereas most patients with a solitary lesion have primary small-bowel lymphoma (21).

Kaposi Sarcoma
Kaposi sarcoma most commonly develops in homosexual men with AIDS. Gastrointestinal involvement occurs in about 50% of patients with Kaposi sarcoma, and affected individuals almost always have concomitant skin lesions (22). Barium studies may reveal multiple submucosal masses, bull’s-eye lesions, or cavitated lesions in the stomach, small bowel, or both (23).

Malignant Gastrointestinal Stromal Tumors
Malignant gastrointestinal stromal tumors are slow-growing malignant tumors involving the stomach or small bowel, particularly the jejunum. Affected patients often develop intraperitoneal metastases or hematogenous metastases to the liver, but lymphatic metastases are extremely uncommon (20). These tumors frequently undergo liquefactive necrosis, resulting in the development of large cavitated lesions seen on barium studies (Fig 9) or CT scans (20). Unlike metastases, however, malignant gastrointestinal stromal tumors are almost always solitary lesions.

Annular Lesions
Circumferential luminal narrowing may be caused by a host of conditions involving the small bowel. The radiographic features help differentiate benign from malignant lesions.

Small-Bowel Adenocarcinoma
Primary small-bowel adenocarcinomas are usually located in the distal duodenum or proximal jejunum (10). As in the colon, these tumors typically appear as polypoid or annular lesions with mucosal destruction, ulceration, and shell-like overhanging margins (Fig 10).

Annular Metastases
Hematogenous metastases are the single most common type of annular tumor in the small bowel (24). Barium studies may reveal marked narrowing and angulation of the lumen and high-grade obstruction due to a severe desmoplastic response incited by these tumors (24).

Intraperitoneal Adhesions
Postoperative intraperitoneal adhesions are by far the most common cause of small-bowel obstruction, accounting for more than 60% of cases (15). Obstructing adhesions are characterized on barium studies by one or more focal areas of tapered narrowing, angulation, and fixation of the bowel, with preserved but tethered mucosal folds (Fig 11) (15). An adhesive band can sometimes
be seen as an extrinsic bandlike defect traversing the small bowel at the site of obstruction (Fig 12).

Other Causes
Other advanced malignant tumors that grow circumferentially (including carcinoids, lymphoma, and malignant gastrointestinal stromal tumors) may manifest on barium studies as annular lesions with effaced folds and relatively abrupt proximal and distal margins (24). In contrast, short weblike strictures may develop as a sequela of chronic ulcerative jejunoileitis in patients with celiac disease (25) or of scarring from nonsteroidal antiinflammatory drugs (26). Longer strictures with tapered margins and asymmetric sacculcation can also be seen in patients with Crohn disease, chronic ischemia, and radiation enteropathy.

Outpouchings
Focal outpouchings from the small bowel may be classified as ulcers (discussed in sections on various lesions that cause ulceration), diverticula, or sacculations. These outpouchings have different pathophysiologies and are associated with different clinical and radiographic findings.

Jejunal Diverticulosis
Jejunal diverticulosis (also known as jejunoileal diverticulosis) results from herniation of the mucosa at sites of weakening, predominantly on the mesenteric border of the jejunum or, less frequently, the ileum (27). The diverticula are characterized on barium studies by multiple rounded outpouchings that have discrete necks (Fig 13), in contrast to the wide-mouthed sacculations in scleroderma. Paradoxically, the diverticula can be more difficult to detect in patients with massive diverticulosis, because the diverticula are easily mistaken for overlapping loops of small bowel (Fig 14). Severe jejunal diverticulosis can lead to stasis and intestinal pseudo-obstruction with bacterial overgrowth, diarrhea, and malabsorption (28). Other rare complications include gastrointestinal bleeding and jejunal diverticulitis (29).

Meckel Diverticulum
Meckel diverticulum is an embryologic anomaly resulting from failure of obliteration of the omphalomesenteric duct, which occurs in 3% of the population. Meckel diverticulum is a true diverticulum, seen on barium studies to arise from the antimesenteric border of the distal ileum within 100 cm of the ileocecal valve (Fig 15). The diverticula have an average length of 2–3 cm and are often found to contain heterotopic gastric mucosa. Complications include gastrointestinal bleeding, obstruction, intussusception, perforation, and diverticulitis. Meckel diverticulum is easier to detect on enteroclysis studies than on SBFT images because this technique permits
greater luminal distention with less overlap of distal small-bowel loops in the lower abdomen and pelvis (30).

Sacculations
Small-bowel sacculations are wide-mouthed outpouchings containing all layers of the bowel. Sacculations are frequently encountered in patients with scleroderma due to asymmetric bowel wall fibrosis, especially in the jejunum, and are associated with an increased number of closely spaced small-bowel folds (Fig 16). Ischemia and Crohn disease are also characterized by asymmetric sacculations, but the sacculations are associated with strictures and, in Crohn disease, with mesenteric border ulcers opposite the sacculations.

Separation of Loops
Small-bowel loops may be separated by excessive mesenteric fat in overweight patients or by fibrofatty proliferation of the mesentery in patients with Crohn disease (31). Small-bowel loops may also be separated by mesenteric adenopathy in patients with lymphoma or nodal metastases to the mesentery. Other causes of displaced small bowel include ascites (in which the small-bowel loops tend to be centrally located), interloop abscesses in Crohn disease, carcinoids, metastases, and, rarely, mesenteric tumors such as lymphangiomas, hemangiomas, cysts, and mesenteric desmoids in patients with familial adenomatous polyposis syndrome.

Abnormal Folds

Thickened Folds
Because small-bowel folds contain a submucosal core, pathologic processes that involve the submucosa may cause the folds to become abnormally thickened (defined as folds larger than 3 mm in thickness in the jejunum and 2 mm in the ileum on SBFT studies). When thickened folds are seen on barium studies, two major variables should be considered: (a) whether the thickened folds have a segmental (ie, one or several small-bowel loops) or a diffuse (ie, the majority or all of the small bowel) distribution and (b) whether the thickened folds are predominantly smooth and straight or predominantly nodular, lobulated, and irregular. Radiographic analysis of these two variables allows all cases of thickened small-bowel folds to be classified into one of four categories (straight-segmental, straight-diffuse, irregular-segmental, and irregular-diffuse thickening), which are considered separately in the following sections.

Straight-segmental Thickening
Thickened folds that are straight segmental tend to be uniformly thickened and have a relatively parallel configuration, producing a “stack of coins” appearance. This finding almost always results from some combination of localized submucosal edema and bleeding due to ischemia, vasculitis, radiation, or hemorrhage.

Ischemia.—Small-bowel ischemia may be caused by atherosclerotic or thromboembolic disease, mesenteric venous stasis or thrombosis, and low-flow states (eg, hypotension, congestive heart failure) that decrease small-bowel perfusion. The ischemic loops may man-
ifest on barium studies as thickened folds that are straight segmental due to localized submucosal edema and hemorrhage (Fig 17), sometimes associated with mesenteric border thumbprinting (5). With progression of disease, there may be pneumatosis or portomesenteric venous gas, ominous signs of developing bowel necrosis and impending perforation.

Vasculitis.—Small-bowel vasculitis may be caused by systemic lupus erythematosus or, less frequently, polyarteritis nodosa and Henoch-Schönlein purpura. Systemic lupus erythematosus is a connective-tissue disease characterized by recurrent small-vessel arteritis involving multiple organs, including the small bowel (32). Affected individuals may present with diarrhea and abdominal pain that resolves after treatment with steroids. Barium studies usually reveal thickened folds that are straight segmental due to localized submucosal edema and bleeding.

Radiation.—Radiation enteropathy occurs in patients who receive more than 5000 Gy to the pelvis for cervical or prostatic carcinoma. During the subacute phase (2–12 months after radiation therapy), these patients may develop severe endarteritis obliterans in pelvic loops of ileum with thickened folds that are straight segmental due to localized submucosal edema and hemorrhage (Fig 18) (33). During the chronic phase (more than 6 months after therapy), these loops may become fixed and angulated due to radiation serositis, with fistulas, strictures, and obstruction.

Intramural hemorrhage.—Intramural hemorrhage may be caused by a bleeding diathesis, anticoagulation, or trauma. The findings are indistinguishable from those of ischemia or vasculitis, with thickened folds that are straight segmental due to localized submucosal bleeding (Fig 19). Intramural hemorrhage should be differentiated from a traumatic small-bowel hematoma, which is characterized on barium studies or CT scans by a discrete intramural mass.

Straight-diffuse Thickening

Bowel wall edema is by far the most common cause of thickened small-bowel folds that are straight diffuse (Fig 20). When serum albumin levels fall below 2 g/dL, decreased oncotic pressure may cause fluid to accumulate diffusely in the submucosa. Major causes of low serum albumin levels include cirrhosis, nephrotic syndrome, and congestive heart failure. Other less
common causes of straight-diffuse small-bowel fold thickening include eosinophilic enteritis, abetalipoproteinemia, and amyloidosis.

Irregular-segmental Thickening

Crohn disease.—Crohn disease has a marked predilection for the terminal ileum because of the high concentration of lymphoid tissue in the distal ileum; however, additional “skip” lesions can be seen more proximally in the small bowel, and sparing of the terminal ileum occurs in about 1% of patients. Characteristic findings on barium studies include aphthoid ulcers (Fig 21), larger ulcers, linear mesenteric border ulcers, antimesenteric border sacculations, focally thickened irregular folds, an ulceronodular pattern (“cobblestoning”) (Fig 22), marked ileal narrowing with a “string” sign due to a combination of severe edema and spasm or fibrosis (Fig 23), and separation of ileal loops by fibrofatty proliferation in the mesentery (31–37). Complications of Crohn disease include fistulas (especially ileoileal, ileocecal, and ileosigmoid fistulas) (Fig 24), abscesses, perforation, obstruction, and, in patients with longstanding disease, an increased risk of developing small-bowel carcinoma or lymphoma.

Lymphoma.—The small bowel is the second most common site of gastrointestinal involvement by lymphoma after the stomach. Most patients have B-cell non-Hodgkin lymphoma that is predominantly found in the distal ileum because of the high concentration of lymphoid tissue (ie, Peyer patches) in this location (38). Barium studies may reveal thickened small-bowel folds that are irregular segmental because of lymphomatous infiltration of the submucosa. This type of fold thickening is so characteristic of lymphoma that it should be a leading consideration when focally thickened, relatively smooth, lobulated folds are detected in one or more small-bowel loops (Fig 25). Lymphoma may also manifest on SBFT studies as polypoid masses, submucosal masses, bull’s-eye lesions, cavitated lesions (Fig 8), infiltrative lesions, aneurysmal dilatation, and, rarely, innumerable small-bowel nodules (Fig 26) (38).

Tuberculosis.—Gastrointestinal tuberculosis tends to involve the ileocecal region, and the cecum and ascending colon are usually involved to a far greater degree than is the terminal ileum (5). This disease is most commonly encountered in patients from areas where tuberculosis is endemic, such as India, China, and Korea. Like Crohn disease, tuberculosis is characterized on barium studies by thickened nodular folds in the terminal ileum, often associated with a markedly
deformed contracted cecum. Unlike in Crohn disease, however, the ileocecal valve is widely patent, and longitudinal ulcers and cobblestoning are extremely uncommon.

**Yersiniosis.**—Yersiniosis is caused by *Yersinia enterocolitica*, a Gram-negative bacillus associated with self-limited diarrhea. This infection is usually confined to the terminal ileum. As in patients with Crohn disease, barium studies may reveal thickened irregular folds, sometimes associated with aphthoid ulcers or larger areas of ulceration (39). Unlike in Crohn disease, however, mesenteric border ulcers and fistulas are extremely uncommon.

**Giardiasis.**—Giardiasis is caused by *Giardia lamblia*, a protozoan infection associated with diarrhea and malabsorption. Affected individuals may be immunocompromised, or they may have a history of travel to regions in which this infection is endemic. Barium studies may reveal thickened nodular folds and spasm, particularly in the duodenum and jejunum (40).

**Strongyloidiasis.**—Strongyloidiasis is a parasitic infection caused by *Strongyloides stercoralis*. Barium studies may reveal thickened or effaced folds and tubular narrowing of the duodenum and proximal jejunum, producing a classic “pipe stem” appearance (41).

**Other causes.**—Other causes of focally thickened irregular folds include 5-FUDR (flouxuridine) toxicity involving the terminal ileum or neo–terminal ileum (42), opportunistic infection of the ileum by cytomegalovirus in patients with AIDS (43), and malignant cecal tumors (including cecal carcinoma and lymphoma) directly invading the terminal ileum via the ileocecal valve.

**Irregular-diffuse Thickening**

**Whipple disease.**—Whipple disease is a rare multisystem disease caused by the Whipple bacillus (*Tropheryma whipplei*) involving the small bowel (particularly the jejunum), lymph nodes, heart valves, joints, and central nervous system (25). As a result, affected individuals may present with diarrhea, malabsorption, heart disease, arthritis, or neurologic abnormalities, depending on the predominant organs affected. Barium studies may reveal thickened, irregular folds and tiny nodules in the jejunum and, to a lesser degree, the ileum due to accumulation of the Whipple bacilli and periodic acid–Schiff–positive macrophages in the submucosa and lamina propria (Fig 27) (44). Affected patients may also have mesenteric and retroperitoneal adenopathy with fat-atenuation nodes on CT scans that are due to lymphatic obstruction and intranodal deposition of lipids (25). Because Whipple disease is an infectious condition, these patients often have a marked response to treatment with antibiotics.

**Intestinal lymphangiectasia.**—Intestinal lymphangiectasia results from obstruction to the flow of lymph in the small bowel, leading to dilatation of intestinal lymphatics. Some patients have a primary, or congenital, form, whereas others have a secondary form resulting from tumor, fibrosis, or other causes of lymphatic obstruction. These patients may present with malabsorption or a
protein-losing enteropathy. Barium studies usually reveal thickened irregular folds and tiny nodules (predominantly in the jejunum) due to dilated lacteal vessels in the submucosa and lamina propria (Fig 28) (45).

Eosinophilic gastroenteritis.—Eosinophilic gastroenteritis is characterized by eosinophilic infiltration of the gastrointestinal tract in the absence of parasitic or other extraintestinal diseases. Patients with small-bowel disease usually have associated gastric disease, but isolated small-bowel disease can occur (46,47). About 75% of patients have a peripheral eosinophilia, and 50% have an atopic history (46,47). The clinical findings depend on which portion of the small-bowel wall is infiltrated by eosinophils; mucosal disease causes a protein-losing enteropathy, diarrhea, and malabsorption, whereas intramural disease causes obstruction, and serosal disease causes eosinophilic ascites due to shedding of eosinophils into the peritoneal cavity. Barium studies may reveal thickened small-bowel folds that are irregular diffuse or straight diffuse due to infiltration of the small bowel by eosinophils (Fig 29) (46,47). Eosinophilic gastroenteritis is a chronic disease that waxes and wanes, sometimes responding markedly to treatment with steroids.

Mastocytosis.—Mastocytosis is a rare condition in which there is proliferation of mast cells in the skin (urticaria pigmentosa), bone marrow, liver, spleen, lymph nodes, and small bowel. Because mast cells mediate the release of histamine, patients can present with episodic flushing, pruritis, hypotension, and diarrhea. Barium studies may reveal thickened small-bowel folds that are irregular diffuse and are associated with innumerable tiny nodules due to accumulation of mast cells in the submucosa and lamina propria (25). Affected patients occasionally may have associated osseous disease, which manifests as multiple sclerotic foci in the bones.

Amyloidosis.—Amyloidosis is a rare systemic disease in which there is extracellular deposition of an insoluble fibrillar protein in multiple organ systems. The primary form of amyloidosis involves the heart, tongue, gastrointestinal tract, skeletal muscle, joints, liver, spleen, and kidneys. The secondary form is associated with rheumatoid arthritis, Crohn disease, and familial Mediterranean fever. Patients with small-bowel involvement by amyloidosis may present with diarrhea, malabsorption, intestinal pseudo-obstruction, or ischemia. Barium studies may reveal thickened folds that are irregular diffuse and are associated with tiny nodules resulting from ischemia or with larger (6–10-mm) nodules resulting from deposition of a fibrillar protein in the submucosa and lamina propria (48).

Waldenstrom macroglobulinemia.—Waldenstrom macroglobulinemia is a plasma cell dyscrasia in which there is deposition of immunoglobulin M in affected tissues. This condition causes hepatosplenomegaly, adenopathy, anemia, and small-bowel disease and occasionally may progress to full-blown lymphoma. Barium studies may reveal thickened irregular small-bowel folds and 1–2-mm nodules due to deposition of immunoglobulin M in the submucosa and lamina propria (25).

Opportunistic infections in AIDS.—Cryptosporidiosis is a protozoan infection that causes a potentially life-threatening secretory diarrhea in patients with AIDS. Barium studies may reveal thickened irregular folds, predominantly in the jejunum (Fig 30) (49). Mycobacterium avium–intracellulare (MAI) occurs late in AIDS when CD4 counts are less than 100. Barium studies may reveal thickened small-bowel folds that are irregular diffuse due to accumulation of the MAI bacilli and macrophages in the submucosa and lamina propria (50). CT scans may also reveal mesenteric adenopathy with necrotic low-attenuating nodes, mimicking the CT findings of Whipple disease (hence the term pseudo-Whipple disease). Cy-
Cytomegalovirus is a member of the herpesvirus group that causes severe small-bowel vasculitis, predominantly in the ileum and cecum in patients with AIDS or other immunodeficiency states. Barium studies may reveal focally thickened irregular folds, ulcers, and tubular narrowing of the ileum due to severe edema and spasm (43).

**Tubular Narrowing with Effaced Folds**

Tubular narrowing of the small bowel occurs when normal small-bowel folds become effaced or obliterated, so that the narrowed segment has a smooth featureless appearance. Other terms used to describe this appearance include ribbonlike, toothpaste, pipe stem, and lead pipe. Extensive tubular narrowing most commonly results from end-stage fibrosis of multiple small-bowel loops in patients with advanced Crohn disease, radiation enteropathy, or ischemic bowel disease (Fig 31). Strongyloidiasis may produce a reversible pipe stem appearance in the duodenum and proximal jejunum due to severe edema and spasm associated with this infection (41).

Graft-versus-host disease with or without associated cytomegalovirus enteritis may produce segmental tubular narrowing of the distal small bowel in patients who have undergone bone marrow transplantation (51). Graft-versus-host disease usually occurs 3–12 months after marrow transplantation. The most common sites of involvement are the skin, liver, and gastrointestinal tract (including the esophagus and small bowel). The ileum is much more likely to be involved by this disease than the jejunum. Barium studies may reveal thickened or effaced small-bowel folds (Fig 32), ulceration, and, eventually, tubular narrowing of ileal loops (51).

The small bowel can also have a tubular appearance in celiac disease due to loss of folds, but the small-bowel lumen is normal in caliber or dilated rather than narrowed in these patients.

**Tethered Folds**

When the small bowel is well distended, the folds normally have a perpendicular orientation in relation to the longitudinal axis of the lumen. When a desmoplastic process in the small-bowel mesentery causes traction on the small-bowel serosa, however, the affected small-bowel loops may become retracted and kinked, with angulation of folds that are tethered toward the site of retraction (5).

Intraperitoneal adhesions are by far the most common cause of tethered small-bowel folds. Nonobstructing adhesions may also manifest as fixation and marked angulation of the lumen, producing a distinctive triangular appearance. When this tethering is associated with mass effect on the bowel, the differential diagnosis includes carcinoid tumors, intraperitoneal metastases, and other less common causes.

**Carcinoid tumors.**—Carcinoid tumors are thought to constitute as many as 25% of all small-bowel tumors (20). Carcinoids are potentially malignant neoplasms that arise from enterochromaffin (enteroendocrine) cells, predominantly in the distal ileum. About 30% of patients have multiple lesions (20). Rarely, these tumors may secrete serotonin (with increased levels of 5-hydroxyindole acetic acid in the urine), leading to development of the carcinoid syndrome with flushing, diarrhea, and bronchospasm.

Barium studies may reveal one or more sessile masses in the distal ileum (Fig 2). Carcinoid tumors invading the mesentery or peritoneal surfaces may incite a marked desmoplastic reaction, manifested by angulation and tethering of adjacent small-bowel loops (Fig 33) (20). Metastases to the mesentery are visualized on CT scans as streaks of soft-tissue attenuation radiating toward a central mesenteric mass that contains calcification in more than 50% of cases (20). CT scans may also reveal ascites and omental cakcs in patients with advanced disease.

**Intraperitoneal metastases.**—Intraperitoneal metastases often involve the distal ileum due to trapping of ascitic fluid in the lower small-bowel mesentery with deposition of tumor in adjacent ileal loops. These intraperitoneal-seeded metastases manifest on barium studies as one or more areas of mass effect on the mesenteric border of the distal ileum, with a marked desmoplastic reaction characterized by tethering, spiculation, and angulation of the affected bowel loops (Fig 34) (52). This appearance is indistinguishable from metastatic carcinoid tumors on barium studies.

**Other causes.**—Other less common causes of mass effect, tethering, and
spiculation of the small bowel include retractile mesenteritis, peritonitis (especially tuberculous peritonitis), mesenteric desmoids, endometriosis, and, rarely, peritoneal mesotheliomas (53).

Abnormal Number of Folds

Scleroderma.—Scleroderma is a connective-tissue disorder involving the lungs, kidneys, heart, and gastrointestinal tract. The small bowel is the second most common site of gastrointestinal involvement after the esophagus. This disease is characterized by smooth muscle atrophy and fibrosis with deposition of collagen, predominantly in the longitudinal layer of the muscularis propria. These pathologic changes result in decreased small-bowel motility with stasis, bacterial overgrowth, diarrhea, and malabsorption. Barium studies may reveal the classic hidebound sign, which manifests as an increased number of folds crowded together in the jejunum (despite luminal distention) due to small-bowel fibrosis and traction (Fig 16) (54). In some patients, asymmetric fibrosis of one wall causes scarring of the uninvolved opposite wall. Other findings include small-bowel dilatation, intestinal pseudo-obstruction, transient intussusceptions, and a benign form of pneumatosis cystoides intestinalis.

Celiac disease.—Celiac disease (non-tropical sprue) is a gluten-sensitive enteropathy characterized by villous atrophy and crypt hyperplasia, predominantly in the jejunum. These patients usually present with steatorrhea, diarrhea, and abdominal pain that markedly improves or resolves when the patient goes on a gluten-free diet. Barium studies reveal dilated small bowel, and enteroclysis reveals a decreased number of jejunal folds (one to three folds per inch in celiac disease vs four to seven folds per inch in control subjects) due to loss of mucosal surface area (Fig 35) (55). Conversely, the ileum may have an increased number of folds as a compensatory adaptation to increase the absorptive capability of the small bowel. This phenomenon results in reversal of the normal fold pattern with an increased number of folds in the ileum relative to the jejunum (“jejunization” of the ileum), also known as a flip-flop pattern (Fig 35) (56). Barium studies may also reveal transient small-bowel intussusceptions or thickened nodular duodenal folds (ie, a “bubbly” duodenum) due to a severe form of duodenitis that occurs in these patients (57).

Patients with chronic celiac disease are at increased risk for developing small-bowel cancer or lymphoma. Unlike most lymphomas (which are predominantly located in the ileum), these tumors are T-cell lymphomas that have a marked predilection for the jejunum (58). As in other patients with lymphoma, barium studies may reveal thickened small-bowel folds that are irregular segmental. Chronic ulcerative jejunoileitis is another rare, potentially fatal complication of celiac disease characterized by thickened small-bowel folds, ulcers, and strictures (58). Despite the rarity of these conditions, small-bowel lymphoma and ulcerative jejunoileitis should be suspected when previously asymptomatic patients with celiac disease develop recurrent diarrhea or abdominal pain after initiating a gluten-free diet. Other complications of celiac disease include hyposplenism, mesenteric and retroperitoneal adenopathy, and the rare cavitary mesenteric lymph node syndrome, in which there is cavitation of mesenteric lymph nodes with fat-fluid levels on CT scans (58).
Nodular Lymphoid Hyperplasia

Enlarged lymphoid follicles may develop in the terminal ileum as a normal finding in young adults or as an immunologic response to enteric infections. Barium studies typically reveal multiple, uniform, round, 1–3-mm nodules separated by normal mucosa in the terminal ileum (Fig 36) (5). When these nodules are unusually numerous or prominent, however, they may be associated with immunodeficiency states (especially common variable immunodeficiency) and giardiasis.

Lymphoma

Small-bowel lymphoma (mucosa-associated lymphoid tissue lymphoma or mantle cell lymphoma) may manifest as conglomerate patches of nonuniform large (>3-mm) nodules in the distal ileum (Fig 26) (5). Early Crohn disease occasionally may produce similar findings.

Dilated Small Bowel

Dilated small bowel most commonly results from a mechanical small-bowel obstruction or an adynamic ileus.

Mechanical Obstruction

Barium studies (including SBFT and enteroclysis) are extremely useful for evaluating patients with low-grade or partial intermittent small-bowel obstruction. In patients with high-grade or complete obstruction, however, CT is the diagnostic test of choice, not only for confirming the presence of obstruction and determining the underlying cause, but also for differentiating simple obstruction from closed-loop obstruction and for assessing complications of strangulation, including ischemia, necrosis, and perforation (15,39).

The most common cause of mechanical obstruction is postsurgical adhesions or adhesive bands (see Anular Lesions) (Figs 11, 12) (15), and the second most common cause is external hernias. Other benign causes of small-bowel obstruction include Crohn disease, appendicitis, ischemic strictures, radiation strictures, nonsteroidal antiinflammatory drug strictures, anastomotic strictures, intussusception, incarcerated internal hernias, gallstone ileus (Fig 7), and strictures from chronic ulcerative jejunitis. Malignant causes of obstruction include small-bowel or colonic carcinoma, hematogenous or intraperitoneal-seeded metastases, and, less commonly, annular carcinoid tumors or lymphomas (24).

Incarcerated hernias.—Incarcerated inguinal or femoral hernias should be suspected when patients with small-bowel obstruction have small-bowel loops in the pelvis below the level of the pubic symphysis. Similarly, incarcerated anterior abdominal wall hernias should be suspected when patients with small-bowel obstruction have small-bowel loops extending beyond the anterior abdominal wall on lateral views, particularly if the hernia is the site of transition and the loops contained in the hernia cannot be reduced with manual compression (60). Internal hernias, which can also cause small-bowel obstruction, are beyond the scope of this article (61).

Radiation.—Radiation therapy to the pelvis (usually for carcinoma of the cervix or prostate) may cause chronic radiation enteropathy and serositis with narrowing and angulation of pelvic ileal loops, thickened, tethered folds, and a variable degree of obstruction (Fig 18).

Intraperitoneal metastases.—Intrapерitoneal metastases to the small-bowel mesentery are characterized on barium studies by multiple extrinsic masses on the mesenteric border of the small bowel associated with tethered, spiculated folds (Fig 34). These implants tend to involve distal ileal loops in the right lower quadrant, where ascitic fluid pools in the small-bowel mesentery (52). In patients who have undergone surgery for malignant abdominal tumors, it frequently is possible to differentiate intraperitoneal metastases from postsurgical adhesions on the basis of the radiographic findings.

Colonic carcinoma.—Cecal carcinoma occluding the ileocecal valve is a relatively common cause of small-bowel obstruction (62). Less frequently, left-sided colon cancers in patients with an incompetent ileocecal valve may also produce an apparent small-bowel obstruction (62). Colonic carcinoma should be suspected when small-bowel obstruction occurs in elderly patients with no history of abdominal surgery.

Adynamic ileus

The two major causes of an adynamic ileus are recent abdominal surgery and treatment with narcotics or other pharmacologic agents that decrease intestinal motility. Other causes include electrolyte imbalances, hypothyroidism, diabetes, ischemia, peritonitis, and prior vagotomy. Small-bowel dilatation may also be seen in disorders such as scleroderma and celiac disease that cause small-bowel hypotonia. For reasons that are unclear, some patients have an adynamic ileus that is confined to the small bowel and spares the colon, so the radiographic findings are indistinguishable.
able from those of a mechanical small-bowel obstruction.

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