

Pattern Approach for Diseases of Mesenteric Small Bowel on Barium Studies¹

Marc S. Levine, MD
Stephen E. Rubesin, MD
Igor Laufer, MD

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.

© RSNA, 2008

¹ From the Department of Radiology, Hospital of the University of Pennsylvania, 3400 Spruce St, Philadelphia, PA 19104. Received July 28, 2007; revision requested September 26; revision received September 27; accepted December 11; final version accepted December 13; final review by M.S.L. May 9, 2008. Address correspondence to M.S.L. (e-mail: marc.levine@uphs.upenn.edu).

© RSNA, 2008

The mesenteric small bowel is a long (variable in length) convoluted tube affected by a host of

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.

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.

General Techniques

SBFT is performed with a 40%–50% wt/vol barium suspension (eg, Entrobar; Mallinckrodt, St Louis, Mo) that has a density low enough to permit visualization of abnormalities en face within the barium-filled small-bowel lumen. The central role of fluoroscopy has been stressed, not only by leading experts on small-bowel radiology (2,3) but also by the American College of Radiology standards for performing SBFT (4). Periodic imaging of the small bowel should be performed with fluoroscopic guidance, with manual palpation to separate individual small-bowel loops and identify abnormalities, rather than rely on serial overhead radiographs. Manual palpation of the small bowel can be performed by using the compression cone on the fluoroscopy tower, a compression paddle, or a lead glove, depending on the preferences of the examiner. Representative spot images of the small bowel should be obtained to document both

normal and abnormal findings at fluoroscopy.

Enteroclysis (also known as a small-bowel enema) is performed by intubating the jejunum just beyond the ligament of Treitz and administering either a low-density barium suspension or an 80% wt/vol barium suspension (eg, Entero-H; E-Z-Em, Westbury, NY) followed by methylcellulose or air (3). Enteroclysis enables optimal distention of individual small-bowel loops; also, double-contrast techniques produce a trans-radiant effect for better visualization of subtle abnormalities.

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

Published online before print
10.1148/radiol.2491071336

Radiology 2008; 249:445–460

Abbreviations:

AIDS = acquired immunodeficiency syndrome
SBFT = small-bowel follow through

M.S.L. and S.E.R. are paid consultants to E-Z-Em.

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



Figure 1: Frontal spot image from enteroclysis shows normal folds in distal jejunum as thin (1–2-mm-thick) delicate structures perpendicular to longitudinal axis of bowel. There are normally four to seven folds per inch of jejunum.



Figure 2: Frontal spot image from double-contrast barium enema examination (with reflux into terminal ileum) shows carcinoid tumor as smooth, sessile, 1.5-cm-diameter polyp (black arrows) in terminal ileum. Also note multiple ileal diverticula (white arrows). (Reprinted, with permission, from reference 9.)

studies usually cause symptoms and in adults are precipitated by an underlying lesion such as a lipoma, carcinoma, metastatic melanoma (Fig 5), or in-

verted 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 intussusciens), with the coil spring representing the folds of the in-

tussusciens (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).

Figure 3

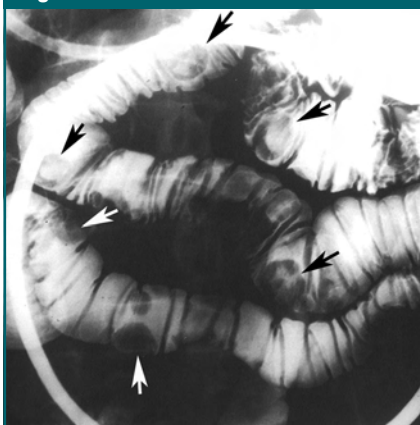


Figure 3: Frontal spot image from enteroclysis shows multiple smooth-surfaced hemispheric submucosal masses (white arrows) in small bowel; other lesions have bull's-eye appearance (black arrows) due to central ulceration. This patient had malignant melanoma with hematogenous metastases to small bowel. (Note radiopaque marker on compression paddle.)

Figure 5

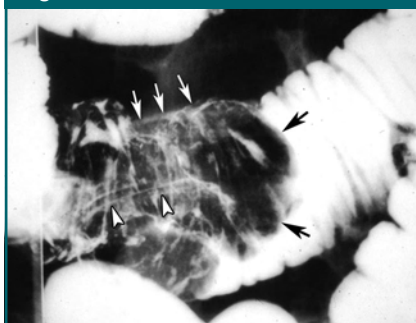


Figure 5: Frontal spot image from enteroclysis in patient with metastatic melanoma shows intussuscepting mass (black arrows) with telescoping of small bowel (intussusceptum, arrowheads) into adjacent loop (intussusciens). A "coil spring" appearance results from barium coating folds of intussusciens (white arrows).

Figure 4



Figure 4: Frontal spot image from double-contrast barium enema examination (with reflux into terminal ileum) shows lipoma as smooth, ovoid, submucosal mass (arrows) in distal ileum.

Figure 6



Figure 6: Frontal spot image from SBFT shows smooth elongated mass (black arrows) in distal ileum with telescoping of small bowel (intussusceptum, white arrow) into adjacent lumen (intussusciens), producing coil spring appearance (arrowheads) similar to that in Figure 5. This patient had inverted Meckel diverticulum acting as lead point for intussusception. (Note radiopaque cone marker on compression paddle.)

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.

Figure 7



Figure 7: Close-up view of frontal overhead radiograph from SBFT shows large ovoid filling defect (black arrows) completely obstructing distal jejunum, with barium entering gallbladder (white arrow) via cholecystoduodenal fistula. Patient had gallstone ileus with complete small-bowel obstruction by an ectopic gallstone occluding the jejunum.

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 shelflike 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 bar-

Figure 8

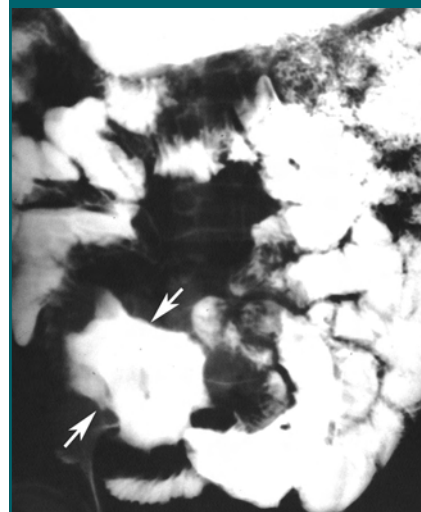


Figure 8: Frontal overhead radiograph from SBFT shows B-cell non-Hodgkin lymphoma as a giant cavitated lesion (arrows) in distal ileum, with displacement of adjacent small-bowel loops by the surrounding mass.

Figure 9

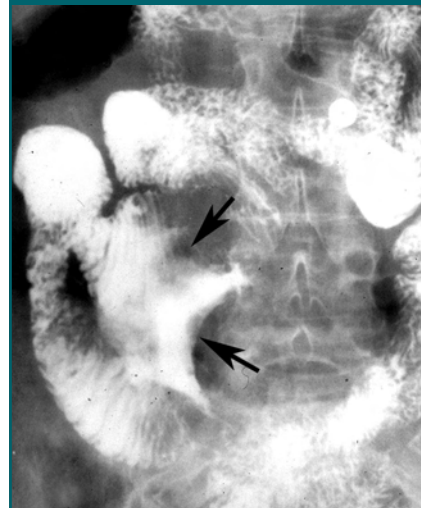


Figure 9: Frontal overhead radiograph from SBFT shows cavitated lesion (arrows) in mid-small bowel with displacement of adjacent small-bowel loops by mass. This patient had malignant gastrointestinal stromal tumor.

ium 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 sacculations 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

Figure 10

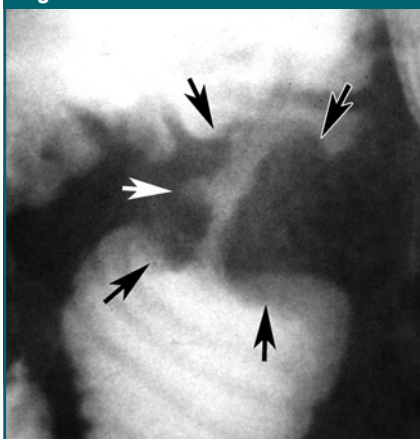


Figure 10: Frontal spot image from SBFT shows primary adenocarcinoma of small bowel as annular lesion in jejunum, with circumferential narrowing, shelflike margins (black arrows), and small central ulcer (white arrow).

Figure 12



Figure 12: Left posterior oblique spot image from enteroclysis shows partially obstructing adhesive band as vertically oriented, extrinsic, bandlike impression (arrows) traversing lumen of jejunum, with proximal dilatation.

Figure 11



Figure 11: Frontal spot image from enteroclysis shows partially obstructing small-bowel adhesion as focal area of tapered narrowing (arrow) with preserved but tethered mucosal folds.

Figure 13

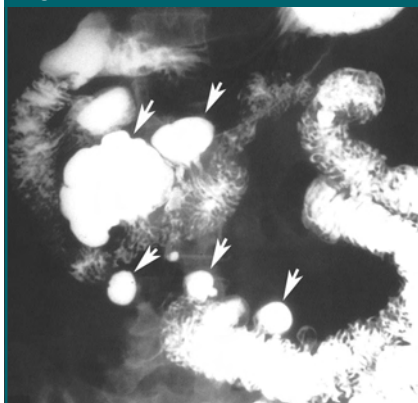


Figure 13: Frontal overhead radiograph from SBFT shows diverticula in duodenum and jejunum as smooth rounded outpouchings of varying sizes (arrows).

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 sev-

Figure 14

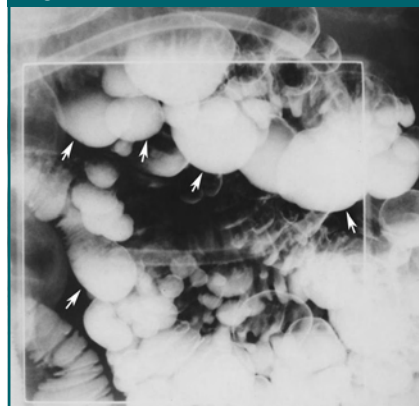


Figure 14: Frontal overhead radiograph from enteroclysis shows massive jejunal diverticulosis (arrows). Paradoxically, this degree of diverticulosis can be more difficult to detect on barium studies, because diverticula are easily mistaken for overlapping loops of small bowel.

Figure 15

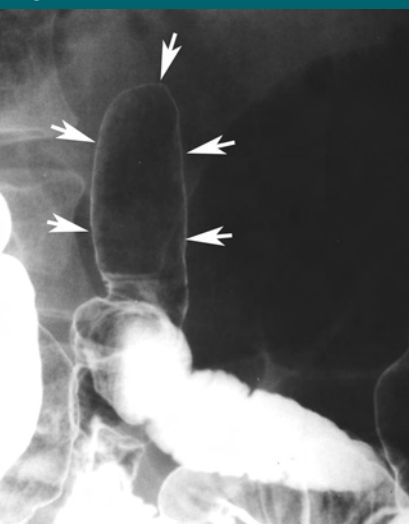


Figure 15: Left posterior oblique spot image from SBFT shows Meckel diverticulum as tubular outpouching (arrows) from distal ileum. No folds are seen in the diverticulum.

eral 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-

Figure 16



Figure 16: Frontal overhead radiograph from SBFT in patient with scleroderma shows markedly dilated duodenum and proximal jejunum, with increased number of small-bowel folds crowded together (despite luminal distention), producing the “hidebound” sign. Also note multiple outpouchings (arrows) due to asymmetric fibrosis with sacculation of opposite wall of bowel.

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.

Figure 17

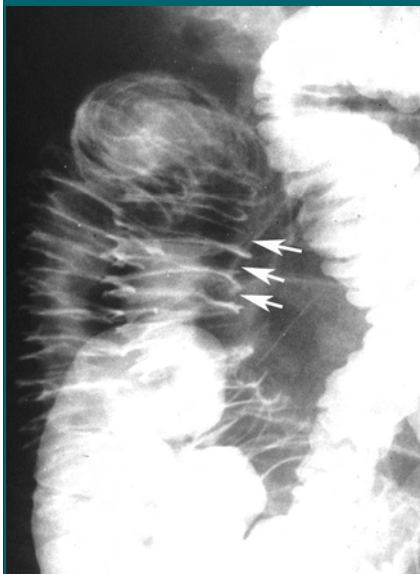


Figure 17: Frontal spot image from SBFT in patient with ischemia shows straight-segmental thickening of folds (arrows) in loop of ileum due to localized submucosal edema and hemorrhage.

Figure 19

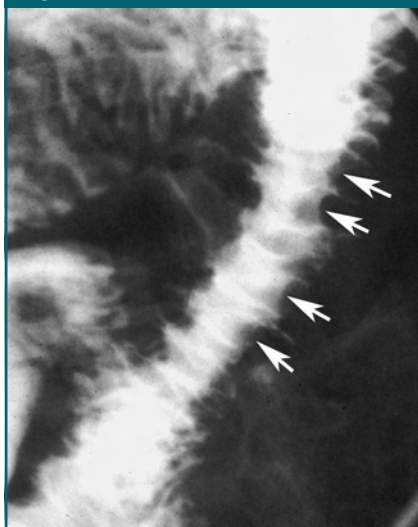


Figure 19: Frontal spot image from SBFT in patient taking warfarin sodium shows straight-segmental thickening of folds (arrows) due to localized submucosal hemorrhage from anticoagulation. Small-bowel ischemia may produce similar findings (see Fig 17).

Figure 18

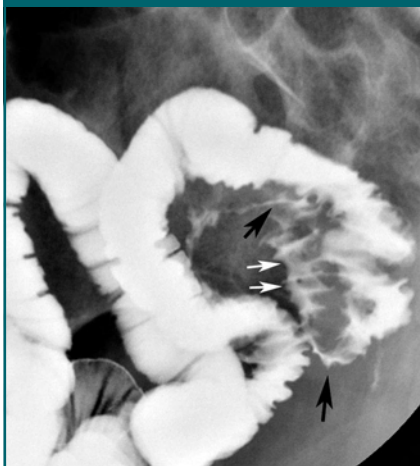


Figure 18: Right posterior oblique spot image from SBFT in patient with prior radiation therapy to pelvis shows straight-segmental thickening of folds (white arrows) in pelvic loops of ileum with narrowing, angulation (black arrows), and low-grade obstruction due to radiation serositis.

Figure 20



Figure 20: Close-up view of frontal overhead radiograph from SBFT in patient with hypoalbuminemia shows thickened straight folds due to edema of small-bowel wall. Folds were thickened throughout small bowel.

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 (31,34–37). 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

Figure 21

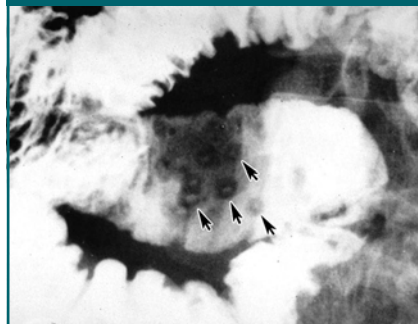


Figure 21: Frontal spot image from SBFT in patient with Crohn disease shows multiple aphthoid ulcers as punctate collections of barium surrounded by radiolucent mounds of edema (arrows).

Figure 22

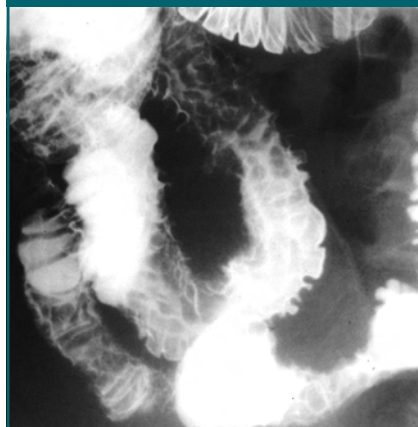


Figure 22: Frontal spot image from enteroclysis in patient with Crohn disease shows cobblestoning in distal ileum due to intersecting linear and transverse ulcers. Note separation of diseased loops from adjacent small bowel by fibrofatty proliferation in mesentery.

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

Figure 23

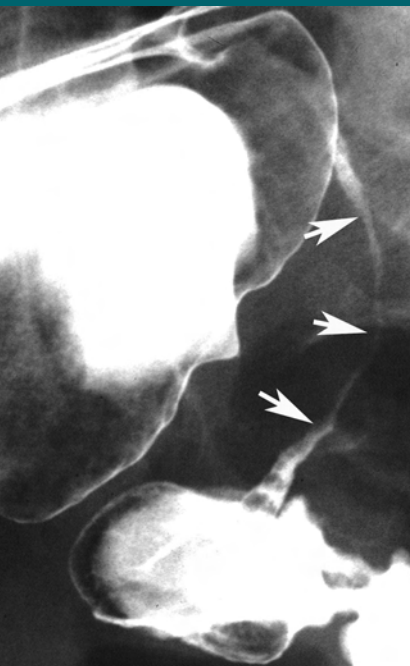


Figure 23: Left posterior oblique spot image from double-contrast barium enema examination (with reflux into terminal ileum) in patient with Crohn disease shows classic string sign with marked narrowing of terminal ileum (arrows) due to severe edema and spasm.

Figure 24



Figure 24: Right posterior oblique spot image from SBFT in patient with Crohn disease shows ileocecal fistulas (small arrows) with narrowing of terminal ileum (large arrow) near ileocecal valve.

Figure 25

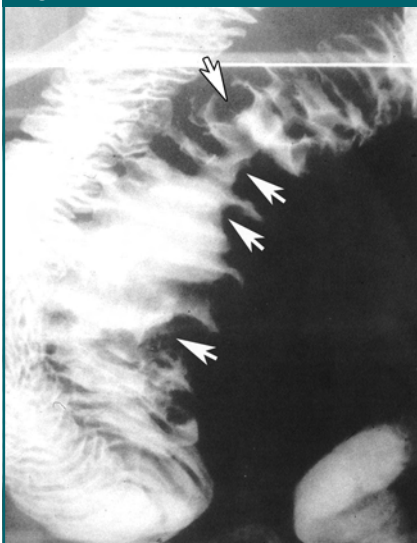


Figure 25: Left posterior oblique spot image from enteroclysis shows irregular-segmental thickening of folds in loop of mid-small bowel, with markedly thickened, lobulated folds (arrows). This patient had primary non-Hodgkin small-bowel lymphoma.

Figure 26



Figure 26: Frontal spot image from double-contrast barium enema examination shows innumerable nodules in distal ileum due to small-bowel lymphoma. Note how nodules are less uniform in size and larger than typical lymphoid follicles. This patient also had undergone subtotal colectomy with ileosigmoid anastomosis (arrows). (Image courtesy of Howard Kessler, MD, Philadelphia, Pa.)

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.—Strongyloidia-

sis 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 (floxuridine) 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-attenuation 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

Figure 27

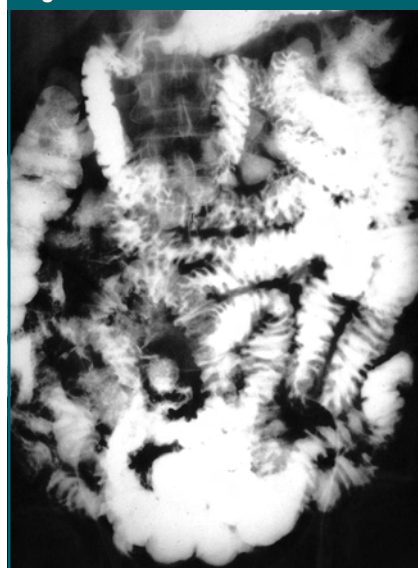


Figure 27: Frontal overhead radiograph from SBFT in patient with Whipple disease shows thickened irregular folds in jejunum and proximal ileum due to accumulation of Whipple bacilli and periodic acid-Schiff-positive macrophages in submucosa.

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

Figure 28

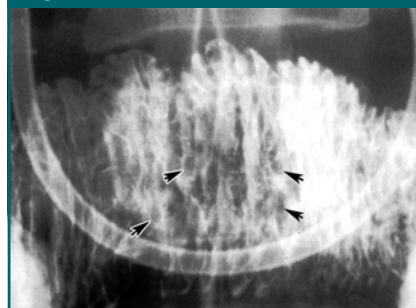


Figure 28: Frontal spot image from enteroclysis in patient with intestinal lymphangiectasia shows tiny nodules in jejunum (arrows) due to dilated lacteal vessels in lamina propria.

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

Figure 29

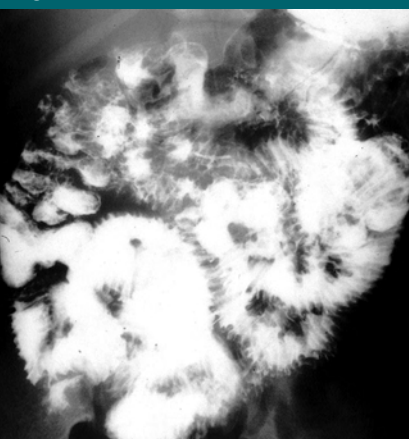


Figure 29: Frontal overhead radiograph from SBFT in patient with eosinophilic gastroenteritis shows straight- and irregular-diffuse thickening of folds in small bowel due to infiltration of small-bowel wall by eosinophils.

Figure 30



Figure 30: Frontal spot image from SBFT in patient with AIDS shows thickened irregular folds in jejunum due to opportunistic infection by cryptosporidiosis.

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-

to megalovirus 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.—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 cakes 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

Figure 31

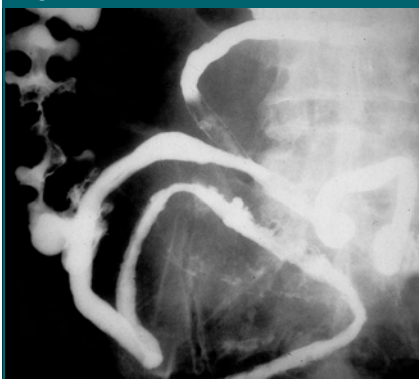


Figure 31: Close-up view of frontal overhead radiograph from SBFT shows tubular narrowing of multiple loops of distal ileum (with complete obliteration of folds) due to chronic small-bowel ischemia.

Figure 32

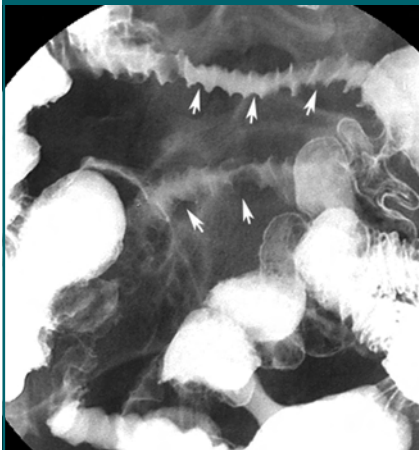


Figure 32: Frontal spot image from SBFT in patient with bone marrow transplant shows thickened and effaced folds (arrows) in several loops of distal ileum due to graft-versus-host disease.

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 contraction (Fig 16) (54). In some patients, asymmetric fibrosis of one wall causes sacculation 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 (nontropical 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

Figure 33



Figure 33: Frontal spot image from SBFT in patient with carcinoid tumor shows mass effect, angulation, and tethering of ileal loops (arrows) due to marked desmoplastic reaction incited by tumor in mesentery.

Figure 34

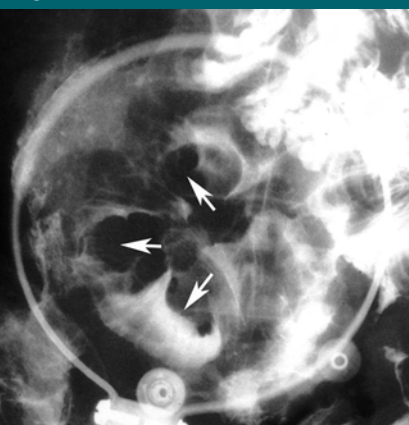


Figure 34: Frontal spot image from SBFT in patient with advanced gastric carcinoma shows areas of mass effect, angulation, and tethering (arrows) on concave (ie, mesenteric) border of distal ileum due to intraperitoneal-seeded metastases. Note resemblance to mesenteric carcinoid tumor in Figure 33.

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

Figure 35



Figure 35: Right posterior oblique spot image from enteroclysis in patient with celiac disease shows markedly decreased number of folds per inch of jejunum (black arrows) with increased number of ileal folds (white arrows), producing a "flip-flop" pattern. These findings are characteristic of celiac disease.

rare cavitory mesenteric lymph node syndrome, in which there is cavitation of mesenteric lymph nodes with fat-fluid levels on CT scans (58).

Nodules without Abnormal Folds

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 on barium studies as conglomerate patches of nonuniform large (>3-mm) nodules in the distal ileum (Fig 26) (5). Early Crohn disease occasionally may produce similar findings.

Figure 36

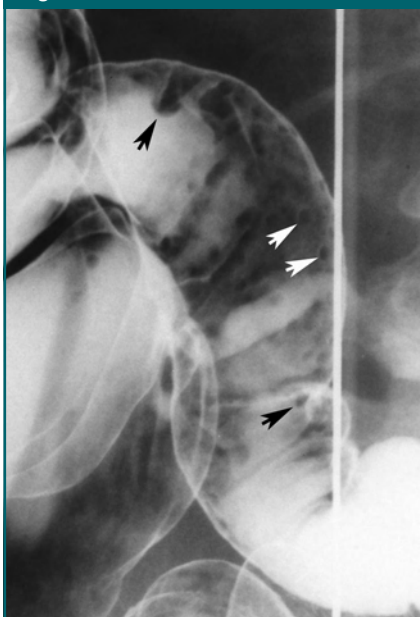


Figure 36: Left posterior oblique spot image from double-contrast barium enema examination (with reflux into terminal ileum) shows enlarged lymphoid follicles as small round nodules (arrows) separated by normal mucosa in terminal ileum.

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 (15,59).

The most common cause of mechanical obstruction is postsurgical adhesions or adhesive bands (see Annular 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, non-steroidal antiinflammatory drug strictures, anastomotic strictures, intussusception, incarcerated internal hernias, gallstone ileus (Fig 7), and strictures from chronic ulcerative jejunoileitis. 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.—Intraperitoneal 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 indistinguish-

able from those of a mechanical small-bowel obstruction.

References

- Ha AS, Levine MS, Rubesin SE, Laufer I, Herlinger H. Radiographic examination of the small bowel: survey of practice patterns in the United States. *Radiology* 2004;231:407–412.
- Maglinte DDT, Herlinger H. The small bowel follow-through and its modifications. In: Herlinger H, Maglinte DDT, Birnbaum BA, eds. *Clinical imaging of the small intestine*. 2nd ed. New York, NY: Springer-Verlag, 1999; 81–94.
- Herlinger H. Barium examinations of the small bowel. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 704–725.
- American College of Radiology. ACR practice guidelines for the performance of per oral barium small bowel examination in adults. Reston, Va: American College of Radiology, 2002; 167–169.
- Rubesin SE. Simplified approach to differential diagnosis of small bowel abnormalities. *Radiol Clin North Am* 2003;41:343–364.
- Buck JL, Harned RK. Polyposis syndromes. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 1075–1088.
- Bulow S, Lauritsen KB, Johansen A, Svendsen LB, Sondergaard JO. Gastroduodenal polyps in familial polyposis coli. *Dis Colon Rectum* 1985;28:90–93.
- Buck JL, Harned RK, Lichtenstein JE, Sobin LH. Peutz-Jeghers syndrome. *RadioGraphics* 1992; 12:365–378.
- Herlinger H, Maglinte DDT. Tumors of the small intestine. In: Herlinger H, Maglinte DDT, eds. *Clinical radiology of the small intestine*. Philadelphia, Pa: Saunders, 1989; 399–451.
- Herbsman H, Wetstein L, Rosen Y. Tumors of the small intestine. *Curr Probl Surg* 1980; 17:121–182.
- Reeder MM, Cavanagh RC. “Bull’s eye” lesions: solitary or multiple nodules in the gastrointestinal tract with large central ulceration. *JAMA* 1974;229:825–826.
- Rubesin SE, Herlinger H. Small bowel: differential diagnosis. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 884–890.
- Pantongrag-Brown L, Levine MS, Elsayed AM, Buetow PC, Agrons GA, Buck JL. Inverted Meckel diverticulum: clinical, radiologic, and pathologic findings. *Radiology* 1996;199(3):693–696.
- Lvoff N, Breiman RS, Coakley FV, Lu Y, Warren RS. Distinguishing features of self-limiting adult small-bowel intussusceptions identified at CT. *Radiology* 2003;227(1): 68–72.
- Herlinger H, Rubesin SE, Morris JB. Small bowel obstruction. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 815–837.
- Rigler LG, Borman CN, Noble JF. Gallstone obstruction: pathogenesis and roentgen manifestations. *JAMA* 1941;117:1753–1759.
- Balthazar EJ, Schechter LS. Gallstone ileus: the importance of contrast examinations in the roentgenographic diagnosis. *Am J Roentgenol Radium Ther Nucl Med* 1975;125: 374–379.
- Loren I, Lasson A, Nilsson A, Nilsson P, Nirhov N. Gallstone ileus demonstrated by CT. *J Comput Assist Tomogr* 1994;18:262–265.
- Zornoza J, Goldstein HM. Cavitating metastases of the small intestine. *AJR Am J Roentgenol* 1977;129:613–615.
- Maglinte DDT, Kelvin FM, Herlinger H. Malignant tumors of the small bowel. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 792–813.
- Yoo CC, Levine MS, McLarney JK, Rubesin SE, Herlinger H. Value of barium studies for predicting primary versus secondary non-Hodgkin’s gastrointestinal lymphoma. *Abdom Imaging* 2000;25:368–372.
- Saltz RK, Kurtz RC, Lightdale CJ, et al. Kaposi’s sarcoma: gastrointestinal involvement correlation with skin findings and immunologic function. *Dig Dis Sci* 1984;29:817–823.
- Rose HS, Balthazar EJ, Megibow AJ, Horowitz L, Laubenstein LJ. Alimentary tract involvement in Kaposi sarcoma: radiographic and endoscopic findings in 25 homosexual men. *AJR Am J Roentgenol* 1982;139:661–666.
- Levine MS, Drooz AT, Herlinger H. Annular malignancies of the small bowel. *Gastrointest Radiol* 1987;12:53–58.
- Herlinger H. Malabsorption. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 759–784.
- Lang J, Price AB, Levi AJ, Burke M, Gumpel JM, Bjarnason I. Diaphragm disease: pathology of disease of the small intestine induced by non-steroidal anti-inflammatory drugs. *J Clin Pathol* 1988;41:516–526.
- Maglinte DD, Chernish SM, DeWeese R, Kelvin FM, Brunelle RL. Acquired jejunoileal diverticular disease: subject review. *Radiology* 1986;158:577–580.
- Williams RA, Davidson DD, Serota AI, Wilson SE. Surgical problems of diverticula of the small intestine. *Surg Gynecol Obstet* 1981;152:621–626.
- Freimanis MG, Plaza-Ponte M. Radiologic diagnosis of jejunal diverticulitis. *Gastrointest Radiol* 1988;13:312–314.
- Ghahremani GG. Radiology of Meckel’s diverticulum. *Crit Rev Diagn Imaging* 1986;26: 1–43.
- Herlinger H, Furth EE, Rubesin SE. Fibrofatty proliferation of the mesentery in Crohn’s disease. *Abdom Imaging* 1998;23: 446–448.
- Nolan DJ, Herlinger H. Vascular disorders of the small bowel. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 838–854.
- Mendelson RM, Nolan DJ. The radiological features of chronic radiation enteritis. *Clin Radiol* 1985;36:141–148.
- Goldberg HI, Caruthers SB, Nelson JA, et al. Radiographic findings of the National Cooperative Crohn’s Disease Study. *Gastroenterology* 1979;77(4 pt 2):925–937.
- Caroline DF, Friedman AC. The radiology of inflammatory bowel disease. *Med Clin North Am* 1994;78:1353–1385.
- Herlinger H, Rubesin SE, Furth EE. Mesenteric border linear ulcer in Crohn’s disease: historical, radiologic, and pathologic perspectives. *Abdom Imaging* 1998;23:122–126.
- Carucci LR, Levine MS. Radiographic imaging of inflammatory bowel disease. *Gastroenterol Clin North Am* 2002;31:93–117, ix.
- Levine MS, Rubesin SE, Pantongrag-Brown L, Buck JL, Herlinger H. Non-Hodgkin’s lymphoma of the gastrointestinal tract: radiographic findings. *AJR Am J Roentgenol* 1997; 168:165–172.
- Ekberg O, Sjostrom B, Brahme F. Radiological findings in Yersinia ileitis. *Radiology* 1977;123(1):15–19.
- Brandon J, Glick SN, Teplick SK. Intestinal giardiasis: importance of serial filming. *AJR Am J Roentgenol* 1985;144:581–584.
- Dallemand S, Waxman M, Farman J. Radiological manifestations of Strongyloides stercoralis. *Gastrointest Radiol* 1983;8:45–51.

42. Kelvin FM, Gramm HF, Gluck WL, Lokich JJ. Radiologic manifestations of small bowel toxicity due to floxuridine therapy. *AJR Am J Roentgenol* 1986;146:39–43.
43. Teixidor HS, Honig CL, Norsoph E, Albert S, Mouradian JA, Whalen JP. Cytomegalovirus infection of the alimentary canal: radiologic findings with pathologic correlation. *Radiology* 1987;163(2):317–323.
44. Philips RL, Carlson HC. The roentgenographic and clinical findings in Whipple's disease: a review of 8 patients. *Am J Roentgenol Radium Ther Nucl Med* 1975;123:268–273.
45. Kingham JG, Moriarty KJ, Furness M, Levison DA. Lymphangiectasia of the colon and small intestine. *Br J Radiol* 1982;55:774–777.
46. Schulman A, Morton PC, Dietrich BE. Eosinophilic gastroenteritis. *Clin Radiol* 1980;31:101–104.
47. MacCarty RL, Talley NJ. Barium studies in diffuse eosinophilic gastroenteritis. *Gastrointest Radiol* 1990;15:183–187.
48. Tada S, Iida M, Matsui T, et al. Amyloidosis of the small intestine: findings on double-contrast radiographs. *AJR Am J Roentgenol* 1991;156:741–744.
49. Berk RN, Wall SD, McArdle CB, et al. Cryptosporidiosis of the stomach and small intestine in patients with AIDS. *AJR Am J Roentgenol* 1984;143:549–554.
50. Vincent ME, Robbins AH. Mycobacterium avium-intracellulare complex enteritis: pseudo-Whipple's disease in AIDS. *AJR Am J Roentgenol* 1985;144:921–922.
51. Jones B, Kramer SS, Saral R, et al. Gastrointestinal inflammation after bone marrow transplantation: graft-versus-host disease or opportunistic infection? *AJR Am J Roentgenol* 1988;150:277–281.
52. Meyers MA. Clinical involvement of mesenteric and antimesenteric borders of small bowel loops. II. Radiologic interpretation of pathologic alterations. *Gastrointest Radiol* 1976;1:49–58.
53. Scarnato VJ, Levine MS, Herlinger H, Wickstrom M, Furth EE, Tureck RW. Ileal endometriosis: radiographic findings in five cases. *Radiology* 2000;214:509–512.
54. Horowitz AL, Meyers MA. The "hide-bound" small bowel of scleroderma: characteristic mucosal fold pattern. *Am J Roentgenol Radium Ther Nucl Med* 1973;119:332–334.
55. Herlinger H, Maglinte DD. Jejunal fold separation in adult celiac disease: relevance of enteroclysis. *Radiology* 1986;158:605–611.
56. Bova JG, Friedman AC, Weser E, Hopens TA, Wytock DH. Adaptation of the ileum in nontropical sprue: reversal of the jejunoileal fold pattern. *AJR Am J Roentgenol* 1985;144:299–302.
57. Jones B, Bayless TM, Hamilton SR, Yardley JH. "Bubbly" duodenal bulb in celiac disease: radiologic-pathologic correlation. *AJR Am J Roentgenol* 1984;142:119–122.
58. Rubesin SE, Herlinger H, Saul SH, Grumbach K, Laufer I, Levine MS. Adult celiac disease and its complications. *RadioGraphics* 1989;9:1045–1066.
59. Megibow AJ, Balthazar EJ, Cho KC, Medwid SW, Birnbaum BA, Noz ME. Bowel obstruction: evaluation with CT. *Radiology* 1991;180:313–318.
60. Zafar HM, Levine MS, Rubesin SE, Laufer I. Anterior abdominal wall hernias: findings in barium studies. *RadioGraphics* 2006;26:691–699.
61. Ghahremani GG. Abdominal and pelvic hernias. In Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 2nd ed. Philadelphia, Pa: Saunders, 2000; 1993–2009.
62. Levine MS. Plain film diagnosis of the acute abdomen. *Emerg Med Clin North Am* 1985;3:541–562.

Radiology 2008

This is your reprint order form or pro forma invoice

(Please keep a copy of this document for your records.)

Reprint order forms and purchase orders or prepayments must be received 72 hours after receipt of form either by mail or by fax at 410-820-9765. It is the policy of Cadmus Reprints to issue one invoice per order.

Please print clearly.

Author Name _____
Title of Article _____
Issue of Journal _____ Reprint # _____ Publication Date _____
Number of Pages _____ KB # _____ Symbol Radiology
Color in Article? Yes / No (Please Circle)

Please include the journal name and reprint number or manuscript number on your purchase order or other correspondence.

Order and Shipping Information

Reprint Costs (Please see page 2 of 2 for reprint costs/fees.)

_____ Number of reprints ordered \$ _____
_____ Number of color reprints ordered \$ _____
_____ Number of covers ordered \$ _____

Subtotal \$ _____

Taxes \$ _____

(Add appropriate sales tax for Virginia, Maryland, Pennsylvania, and the District of Columbia or Canadian GST to the reprints if your order is to be shipped to these locations.)

First address included, add \$32 for
each additional shipping address \$ _____

TOTAL \$ _____

Shipping Address (cannot ship to a P.O. Box) Please Print Clearly

Name _____
Institution _____
Street _____
City _____ State _____ Zip _____
Country _____
Quantity _____ Fax _____
Phone: Day _____ Evening _____
E-mail Address _____

Additional Shipping Address* (cannot ship to a P.O. Box)

Name _____
Institution _____
Street _____
City _____ State _____ Zip _____
Country _____
Quantity _____ Fax _____
Phone: Day _____ Evening _____
E-mail Address _____

* Add \$32 for each additional shipping address

Payment and Credit Card Details

Enclosed: Personal Check _____
Credit Card Payment Details _____
Checks must be paid in U.S. dollars and drawn on a U.S. Bank.
Credit Card: ☐ VISA ☐ Am. Exp. ☐ MasterCard
Card Number _____
Expiration Date _____
Signature: _____

Please send your order form and prepayment made payable to:

Cadmus Reprints

P.O. Box 751903

Charlotte, NC 28275-1903

Note: Do not send express packages to this location, PO Box.
FEIN #:541274108

Signature _____

Signature is required. By signing this form, the author agrees to accept the responsibility for the payment of reprints and/or all charges described in this document.

Invoice or Credit Card Information

Invoice Address Please Print Clearly

Please complete Invoice address as it appears on credit card statement

Name _____
Institution _____
Department _____
Street _____
City _____ State _____ Zip _____
Country _____
Phone _____ Fax _____
E-mail Address _____

Cadmus will process credit cards and *Cadmus Journal* Services will appear on the credit card statement.

If you don't mail your order form, you may fax it to 410-820-9765 with your credit card information.

Date _____

Radiology 2008

Black and White Reprint Prices

Domestic (USA only)						
# of Pages	50	100	200	300	400	500
1-4	\$221	\$233	\$268	\$285	\$303	\$323
5-8	\$355	\$382	\$432	\$466	\$510	\$544
9-12	\$466	\$513	\$595	\$652	\$714	\$775
13-16	\$576	\$640	\$749	\$830	\$912	\$995
17-20	\$694	\$775	\$906	\$1,017	\$1,117	\$1,220
21-24	\$809	\$906	\$1,071	\$1,200	\$1,321	\$1,471
25-28	\$928	\$1,041	\$1,242	\$1,390	\$1,544	\$1,688
29-32	\$1,042	\$1,178	\$1,403	\$1,568	\$1,751	\$1,924
Covers	\$97	\$118	\$215	\$323	\$442	\$555

International (includes Canada and Mexico)						
# of Pages	50	100	200	300	400	500
1-4	\$272	\$283	\$340	\$397	\$446	\$506
5-8	\$428	\$455	\$576	\$675	\$784	\$884
9-12	\$580	\$626	\$805	\$964	\$1,115	\$1,278
13-16	\$724	\$786	\$1,023	\$1,232	\$1,445	\$1,652
17-20	\$878	\$958	\$1,246	\$1,520	\$1,774	\$2,030
21-24	\$1,022	\$1,119	\$1,474	\$1,795	\$2,108	\$2,426
25-28	\$1,176	\$1,291	\$1,700	\$2,070	\$2,450	\$2,813
29-32	\$1,316	\$1,452	\$1,936	\$2,355	\$2,784	\$3,209
Covers	\$156	\$176	\$335	\$525	\$716	\$905

Minimum order is 50 copies. For orders larger than 500 copies, please consult Cadmus Reprints at 800-407-9190.

Reprint Cover

Cover prices are listed above. The cover will include the publication title, article title, and author name in black.

Shipping

Shipping costs are included in the reprint prices. Domestic orders are shipped via UPS Ground service. Foreign orders are shipped via a proof of delivery air service.

Multiple Shipments

Orders can be shipped to more than one location. Please be aware that it will cost \$32 for each additional location.

Delivery

Your order will be shipped within 2 weeks of the journal print date. Allow extra time for delivery.

Color Reprint Prices

Domestic (USA only)						
# of Pages	50	100	200	300	400	500
1-4	\$223	\$239	\$352	\$473	\$597	\$719
5-8	\$349	\$401	\$601	\$849	\$1,099	\$1,349
9-12	\$486	\$517	\$852	\$1,232	\$1,609	\$1,992
13-16	\$615	\$651	\$1,105	\$1,609	\$2,117	\$2,624
17-20	\$759	\$787	\$1,357	\$1,997	\$2,626	\$3,260
21-24	\$897	\$924	\$1,611	\$2,376	\$3,135	\$3,905
25-28	\$1,033	\$1,071	\$1,873	\$2,757	\$3,650	\$4,536
29-32	\$1,175	\$1,208	\$2,122	\$3,138	\$4,162	\$5,180
Covers	\$97	\$118	\$215	\$323	\$442	\$555

International (includes Canada and Mexico)						
# of Pages	50	100	200	300	400	500
1-4	\$278	\$290	\$424	\$586	\$741	\$904
5-8	\$429	\$472	\$746	\$1,058	\$1,374	\$1,690
9-12	\$604	\$629	\$1,061	\$1,545	\$2,011	\$2,494
13-16	\$766	\$797	\$1,378	\$2,013	\$2,647	\$3,280
17-20	\$945	\$972	\$1,698	\$2,499	\$3,282	\$4,069
21-24	\$1,110	\$1,139	\$2,015	\$2,970	\$3,921	\$4,873
25-28	\$1,290	\$1,321	\$2,333	\$3,437	\$4,556	\$5,661
29-32	\$1,455	\$1,482	\$2,652	\$3,924	\$5,193	\$6,462
Covers	\$156	\$176	\$335	\$525	\$716	\$905

Tax Due

Residents of Virginia, Maryland, Pennsylvania, and the District of Columbia are required to add the appropriate sales tax to each reprint order. For orders shipped to Canada, please add 7% Canadian GST unless exemption is claimed.

Ordering

Reprint order forms and purchase order or prepayment is required to process your order. Please reference journal name and reprint number or manuscript number on any correspondence. You may use the reverse side of this form as a proforma invoice. Please return your order form and prepayment to:

Cadmus Reprints
P.O. Box 751903
Charlotte, NC 28275-1903

Note: Do not send express packages to this location, PO Box.
FEIN #:541274108

Please direct all inquiries to:

Rose A. Baynard
800-407-9190 (toll free number)
410-819-3966 (direct number)
410-820-9765 (FAX number)
baynardr@cadmus.com (e-mail)

Reprint Order Forms and purchase order or prepayments must be received 72 hours after receipt of form.